Topic: Thyroid Eye Disease (TED)
Learning objectives: Introduction etiology, clinical features and management of Thyroid Eye Disease (TED)

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Dysthroid Ophthalmopathy

Graves disease:

- Auto immune disease characterized by excess secretion of thyroid hormones by the entire thyroid gland
- 3rd – 5th decades of life
- Male: female ratio is 8:1
- Most common cause of thyrotoxicosis

- Thyroid Ophthalmology is the most common cause of bilateral as well as unilateral proptosis.

- Proptosis is axial, uninfluenced by treatment of hyperthyroidism and permanent in 70% of cases.

Other causes:

- Toxic nodular goiter, sub-acute thyroiditis, factitious hyperthyroidism
- In 10-25% of cases, thyroid ophthalmopathy occurs in absence of clinical and biochemical evidence of thyroid dysfunction
- In Ophthalmic Graves disease, the Ophthalmic signs of Graves disease occur in a patient who is not clinically hyperthyroid.

Risk factors

- More common in females (M:F 6:1)
- HLA – DR3, HLA – B8 and the genes for CTLA4 and the thyroid stimulating hormone (TSH) receptor
- Smoking
- Personal or family history of autoimmune thyroid disease

Etiology/Pathogenesis of Ophthalmopathy

IgG antibody

Enlargement of extraocular muscles:

- Increase in glycosaminoglycans may enlarge to 8 times their normal size.

Cellular infiltration: of interstitial tissues with lymphocytes, plasma cells, macrophages and mast cells during the congestive stage. Later fibrosis occurs.

Proliferation of orbital fat, connective tissue and lacrimal glands due to retention of fluid and accumulation of glycosaminoglycans.

Clinical Manifestations

- Soft tissue involvement
- Eyelid retraction
- Proptosis
- Optic neuropathy
- Restrictive myopathy

Soft tissue involvement:

Symptoms:

Include grittiness, photophobia, lacrimation and retrobulbar discomfort.

Signs:

Periorbital and lid swelling:

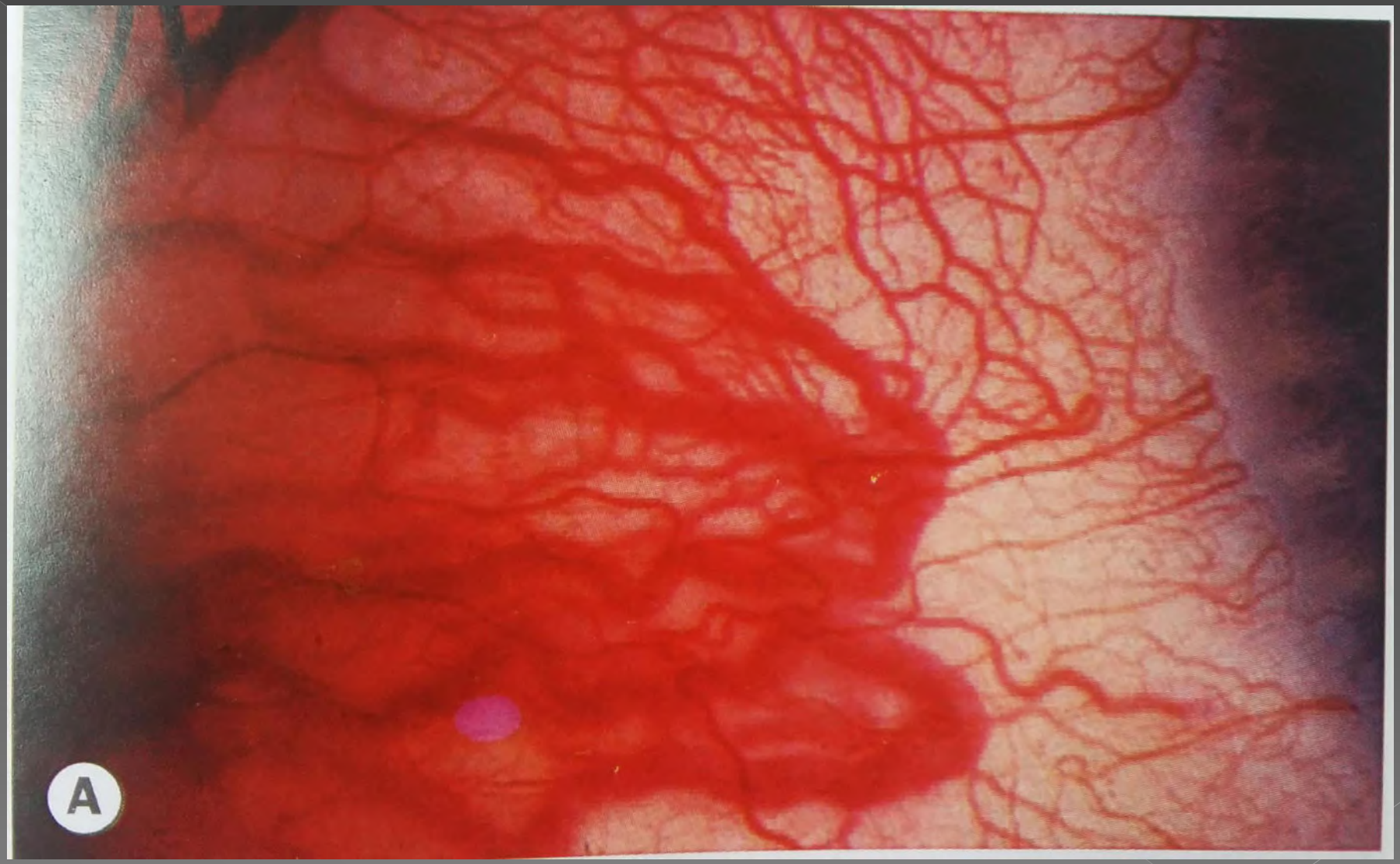
caused by edema and infiltration behind the orbital septum.

Conjunctiva hyperaemia:

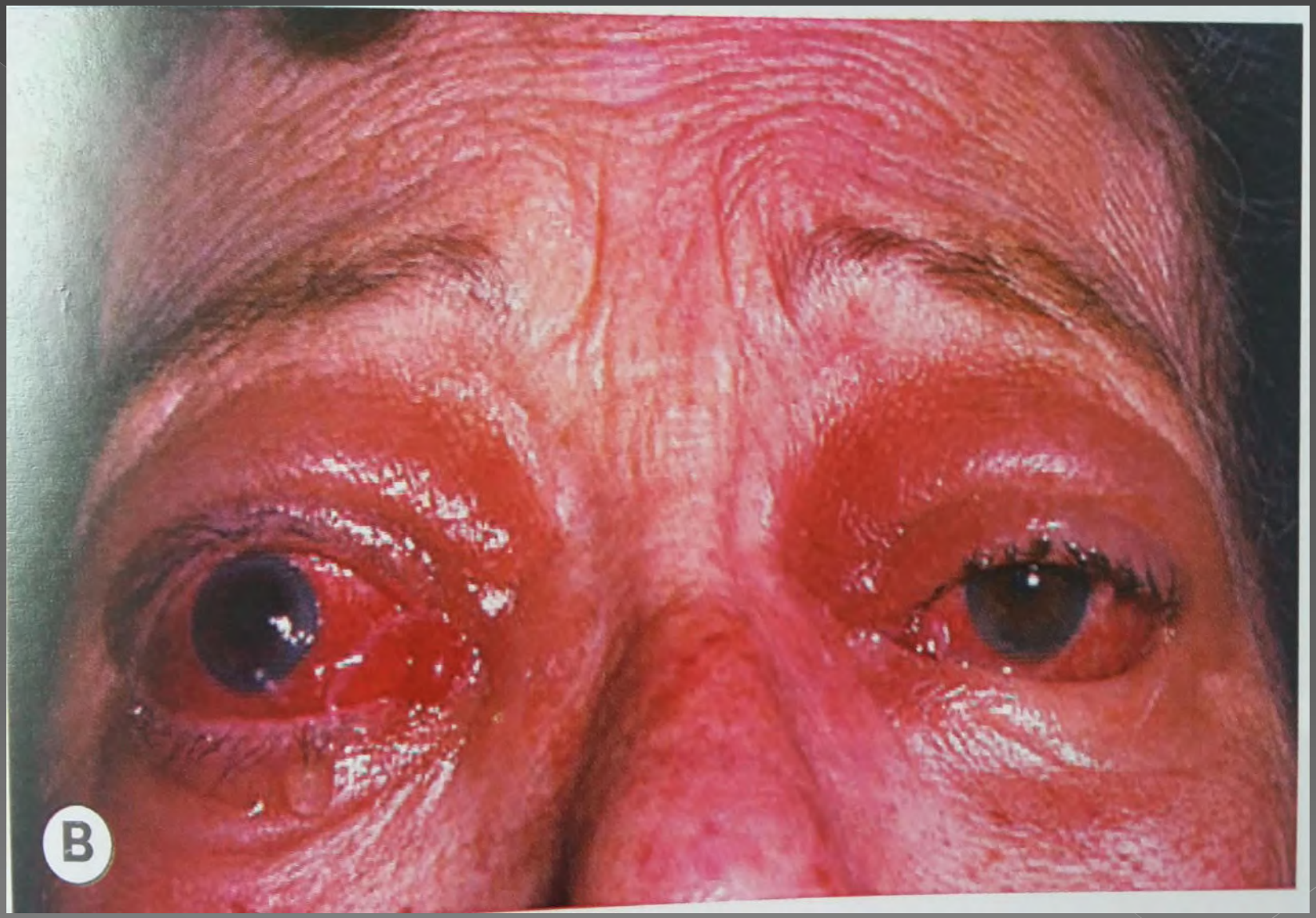
is an important sign.

Chemosis:

- Edema of the conjunctiva and caruncle.
- In severe cases, the conjunctiva prolapses over the lower eyelid



Epibublar hyperaemia overlying a horizontal rectus muscle



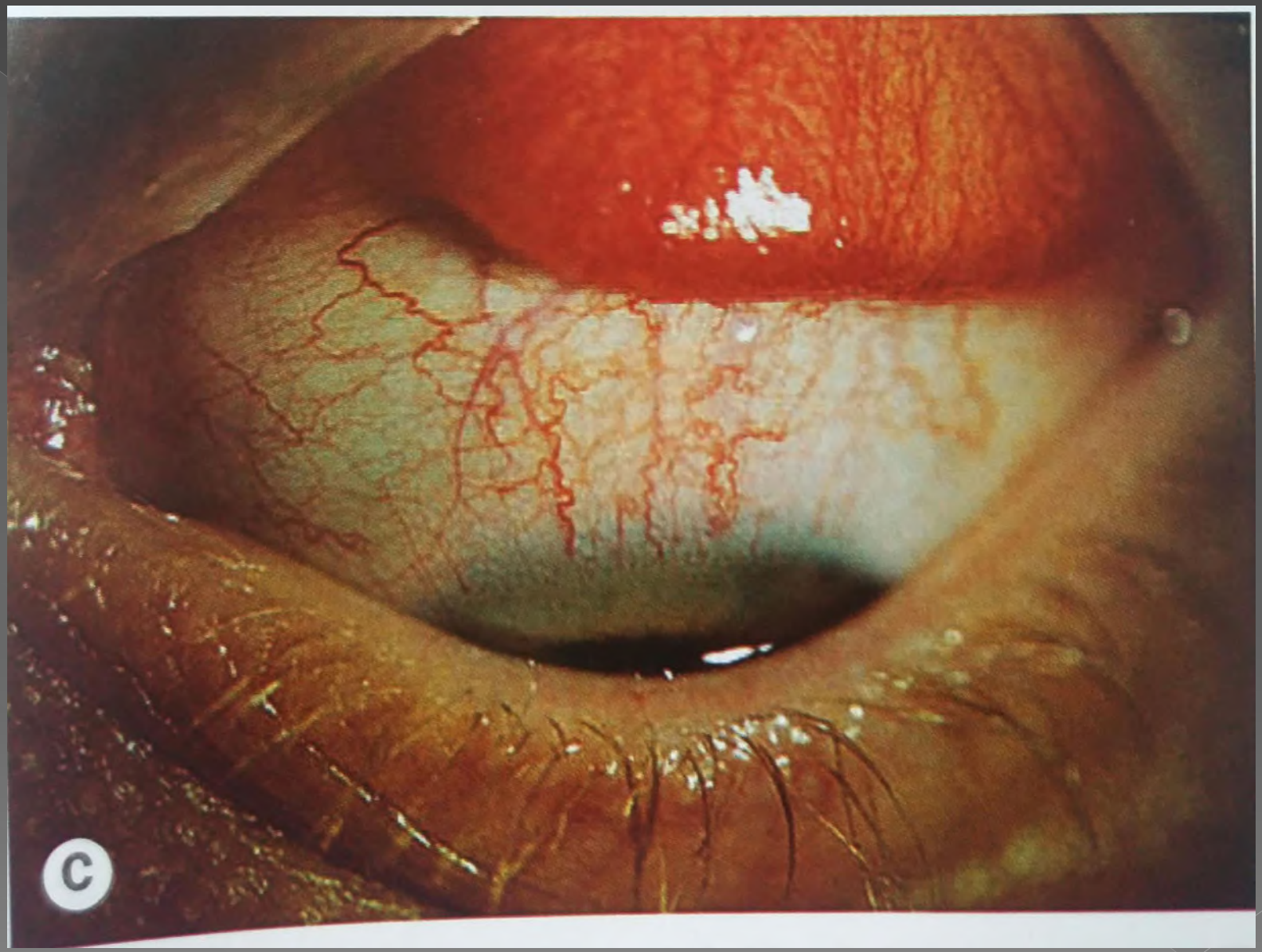
Periorbital oedema, chemosis and prolapse of fat into the eyelids

Superior limbic keratoconjunctivitis:

- Usually bilateral, asymmetrical
- Characterized by papillae on superior bulbar conjunctiva, papillary hypertrophy at the limbus, punctate epitheliopathy and corneal filaments.

Keratoconjunctivitis sicca:

- Secondary to infiltration of lacrimal glands



Superior limbic keratoconjunctivitis

Management

- Topical therapy with lubricants. Artificial tears can be used during day time and ointments during night. Patients with superior limbic K.C may require topical adrenaline 1% and acetylcysteine 5%.

- Head elevation using 3 pillows during sleep.

- Taping of the lids during sleep is specially beneficial in patients with exposure keratopathy

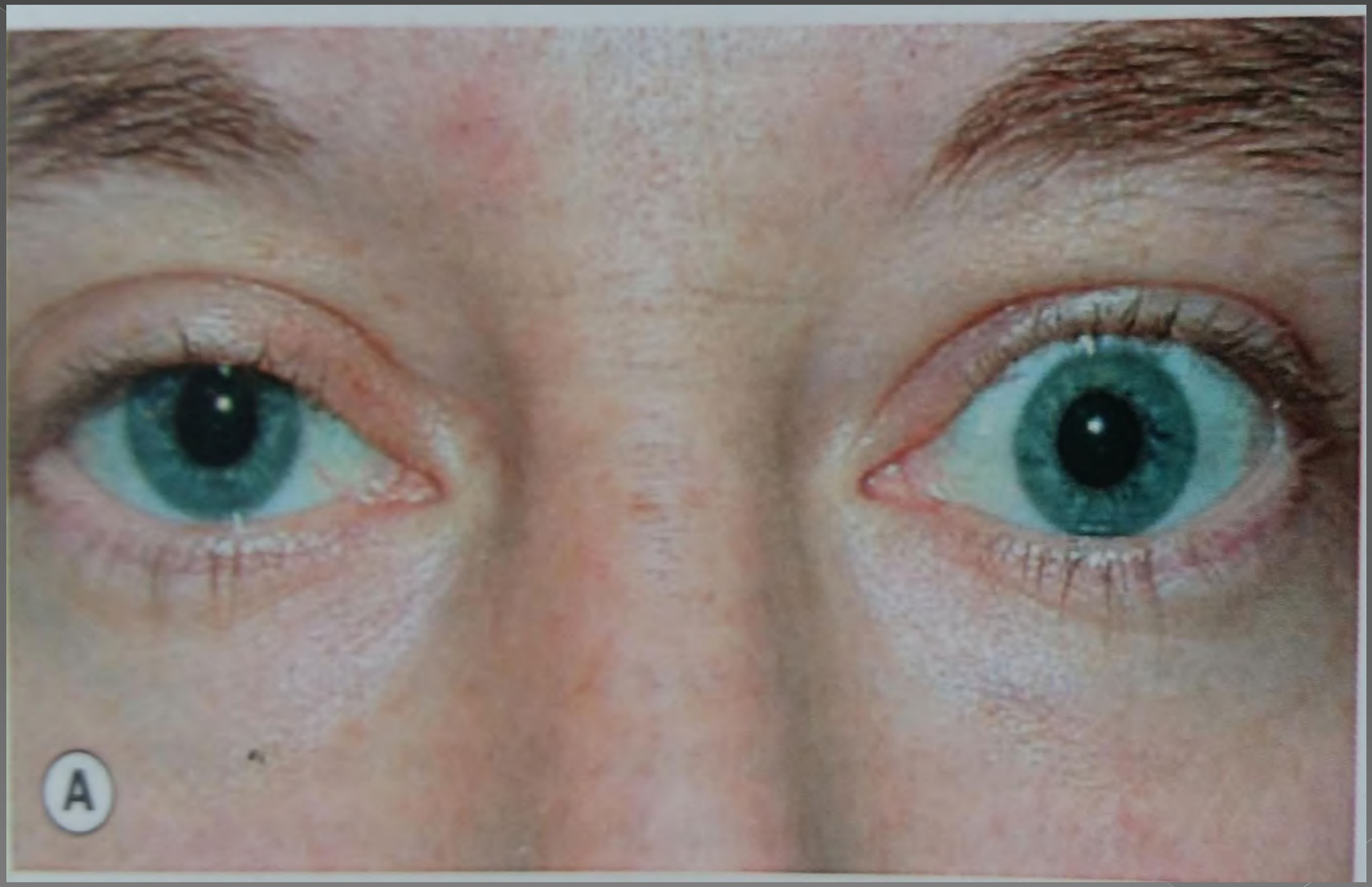
- Diuretics used at the night may reduce the morning accumulation of periorbital edema.

Eyelid retraction

- Retraction of both eyelids occurs in about 50% of patients with Graves disease.
- The postulated mechanisms are:
  - Contraction of the levator muscle associated with fibrosis and adhesions between the levator and overlying orbital tissues.
  - Worse on down gaze in the lower eyelid, fibrosis of inferior rectus may occur.

- Secondary overaction of levator – superior rectus complex in response to the hypophoria induced by fibrosis and tethering of the inferior rectus.
- There is increased lid retraction from down gaze to up gaze.

- Chemically induced over action of Muller muscle as a result of sympathetic over stimulation, secondary to high levels of thyroid hormones.
- In some patients, lid retraction may be reduced by the topical use of the sympatholytic drug Guanethidine.



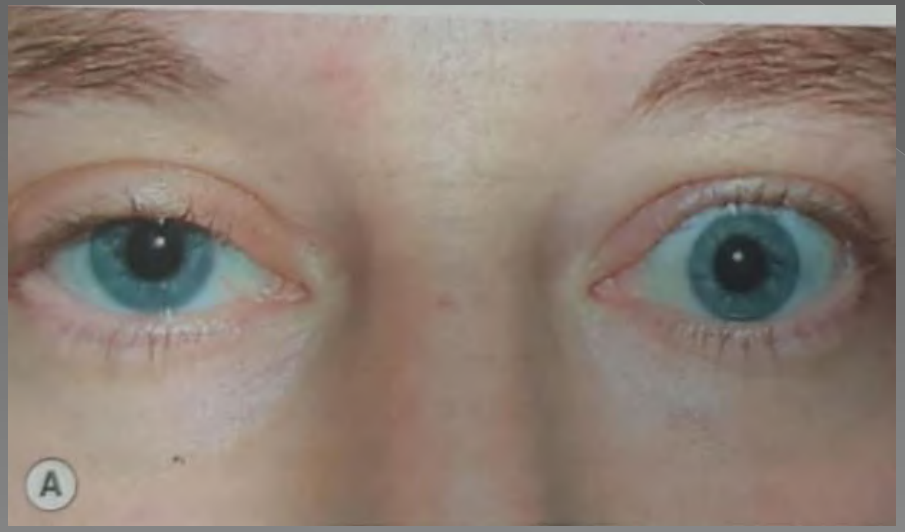
Mild left lid retraction

Clinical features (Symptoms)

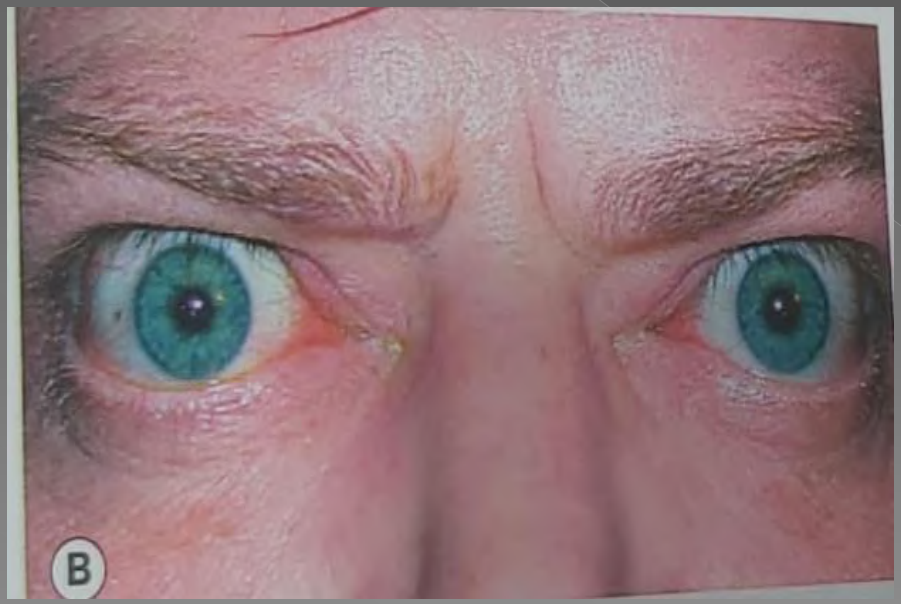
- Staring or bulging eye appearance
- Difficulty closing the eyes and
- Ocular surface symptoms

Signs

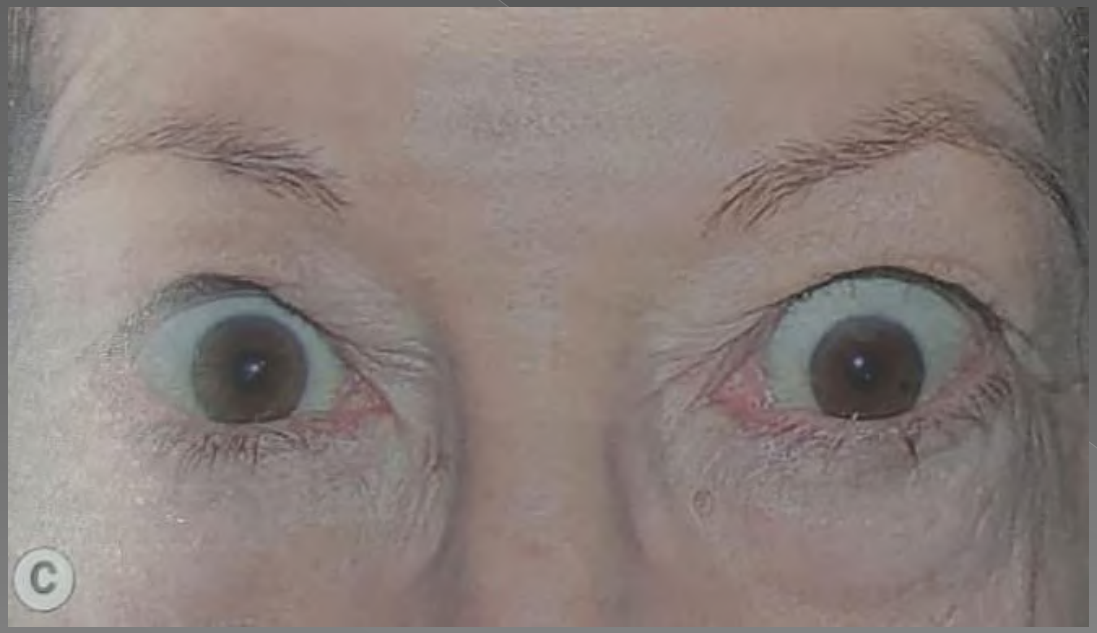
- Upper lid margin normally rests 2mm below limbus
- Lid retractiion suspected when the margin is either level with or above superior limbus, allowing sclera to be visible (scleral show)



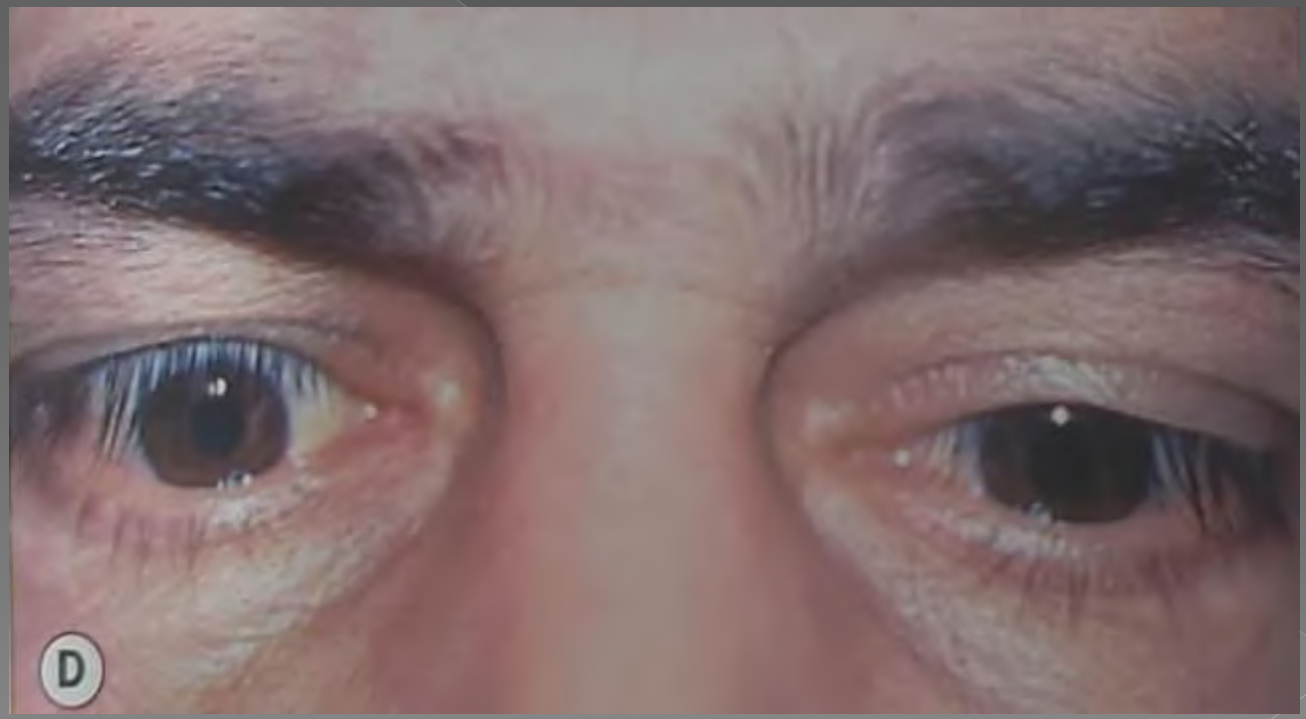
- The lower eyelid margin normally rests at inferior limbus
- Retraction suspected when sclera shows below limbus
- Dalrymple sign is lid retraction in primary gaze



- Kocher sign describes a staring and frightened appearance of the eyes, marked on attentive fixation



- The Von Graefe sign signifies retarded descent of the upper lid on down gaze



Management

- In 50% of patients, the retraction improves spontaneously.
- Treatment of associated hyperthyroidism may also improve the retraction

- Surgery is considered in patients with marked but stable lid retraction.
- Indications are:
  - Exposure keratopathy
  - Poor cosmesis

- The sequence of surgery in thyroid ophthalmopathy is:
  - Orbital decompression
  - Strabismus surgery
  - Eyelid surgery

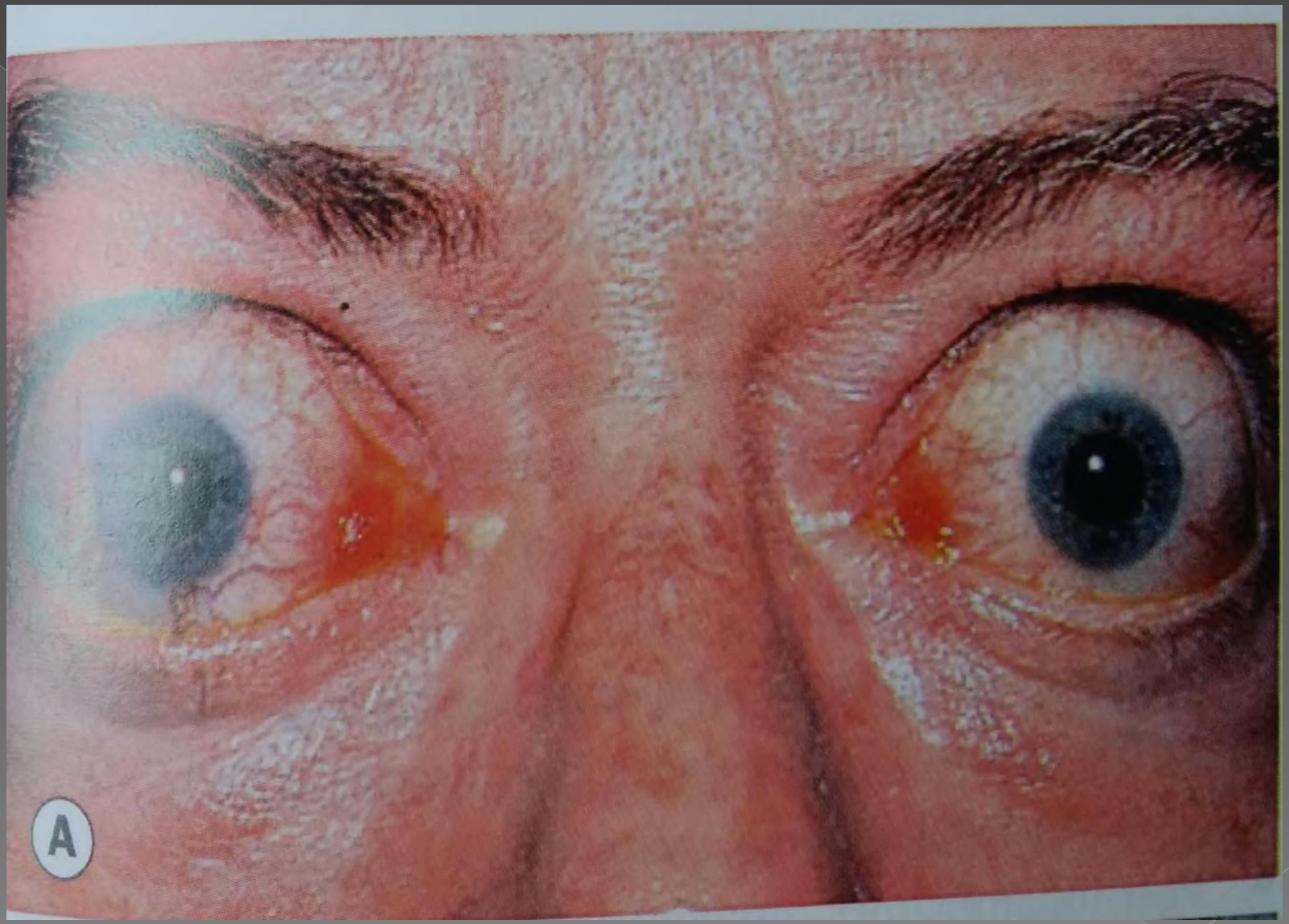
Surgical procedures

- Inferior rectus recession of 4mm is done in cases of inferior rectus fibrosis.
- Mullerectomy is done in mild cases.
- Recession of lower lid retractors with a scleral graft, when retraction of lower lid is 2mm or more.

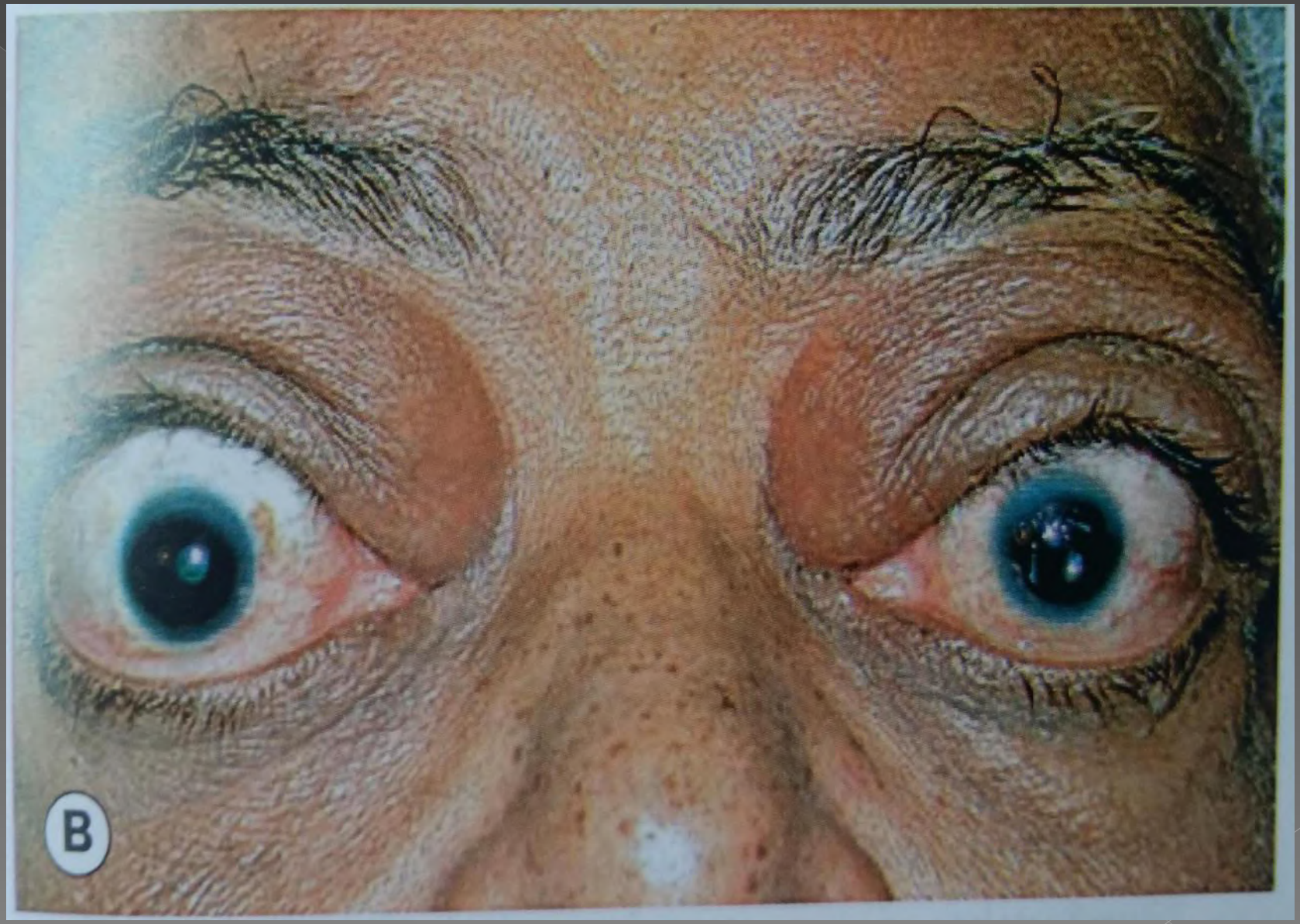
- Blepharoplasty to remove excess fatty tissue and redundant skin.
- Lateral tarsorrhaphy can be done for hiding residual proptosis following lid recession. Tarsorrhaphy should not be done as a primary procedure.

Proptosis

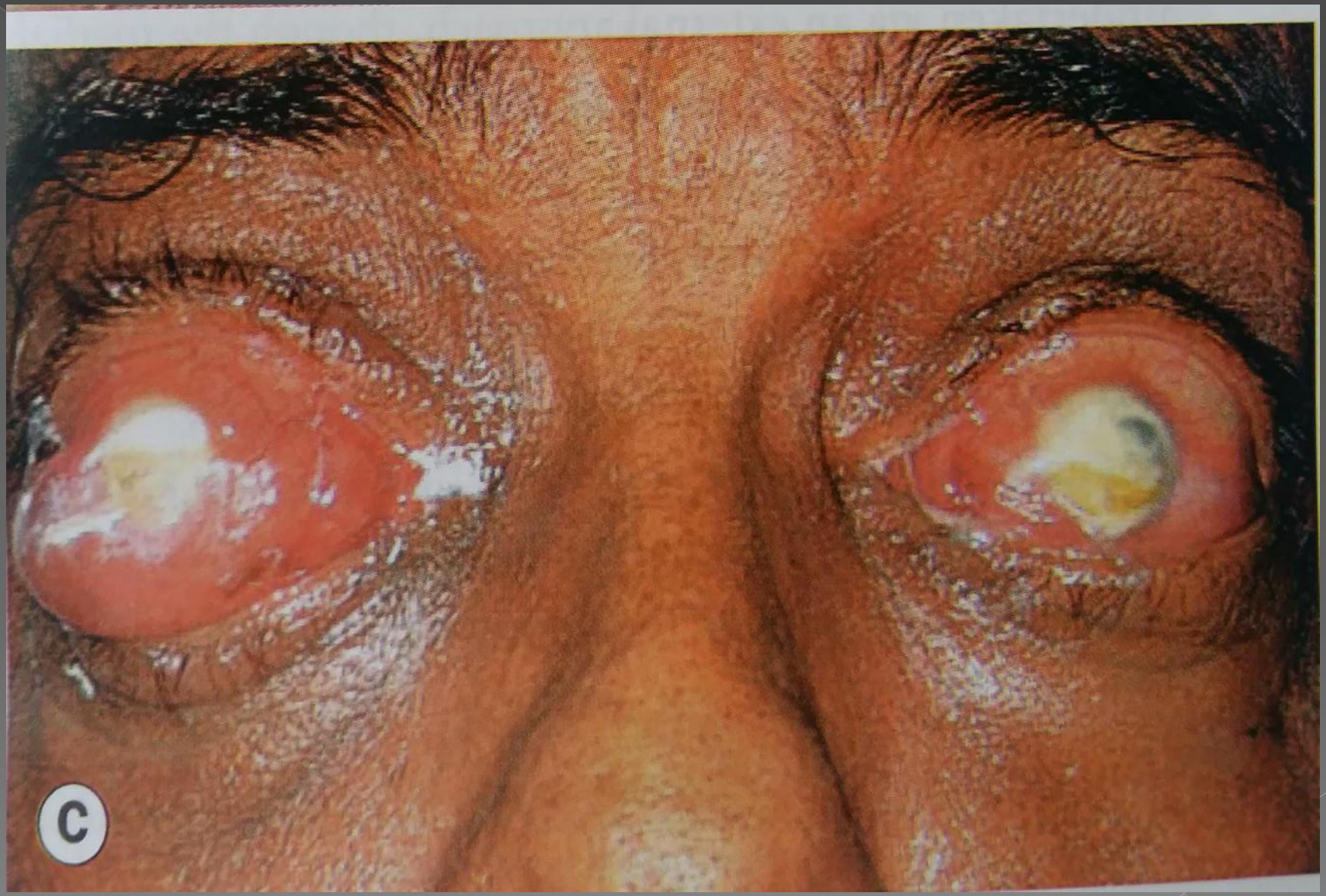
- Thyroid ophthalmopathy is most common cause of bilateral as well as unilateral proptosis.
- Proptosis is axial, uninfluenced by treatment of hyperthyroidism and permanent in 70% of cases.
- If untreated, it may lead to exposure keratopathy.



symmetrical



Asymmetrical



Bacterial keratitis due to severe exposure

Management

- Non – invasive and surgical
- Systemic steroids may be used in patients with rapidly progressive and painful proptosis, provided there is no contraindication such as TB or peptic ulcer.

- Oral prednisolone 80 – 100 mg is given initially. Dose is tapered after 48 hours, over a duration of 2-8 weeks. Addition of cyclosporine permits a lower dosage of prednisolone.

- I.V prednisolone (0.5 gm in 200 ml saline over 30 minutes) which can be repeated after 48 hours.

- Radiotherapy can be considered in patients who have any contraindication to steroids or unresponsive.
- A positive response is evident in 6 weeks, with maximal response in 4 months.
- Surgical decompression:
  - Two – wall decompression
  - Three – wall decompression
  - Four – wall decompression

Optic neuropathy

- Affects about 5% of patients
- Caused either through direct compression of optic nerve or its blood supply at the orbital apex by the enlarged recti.
- Patient presents with slowly progressive impairment of central vision, along with defective red – green color appreciation.

Signs

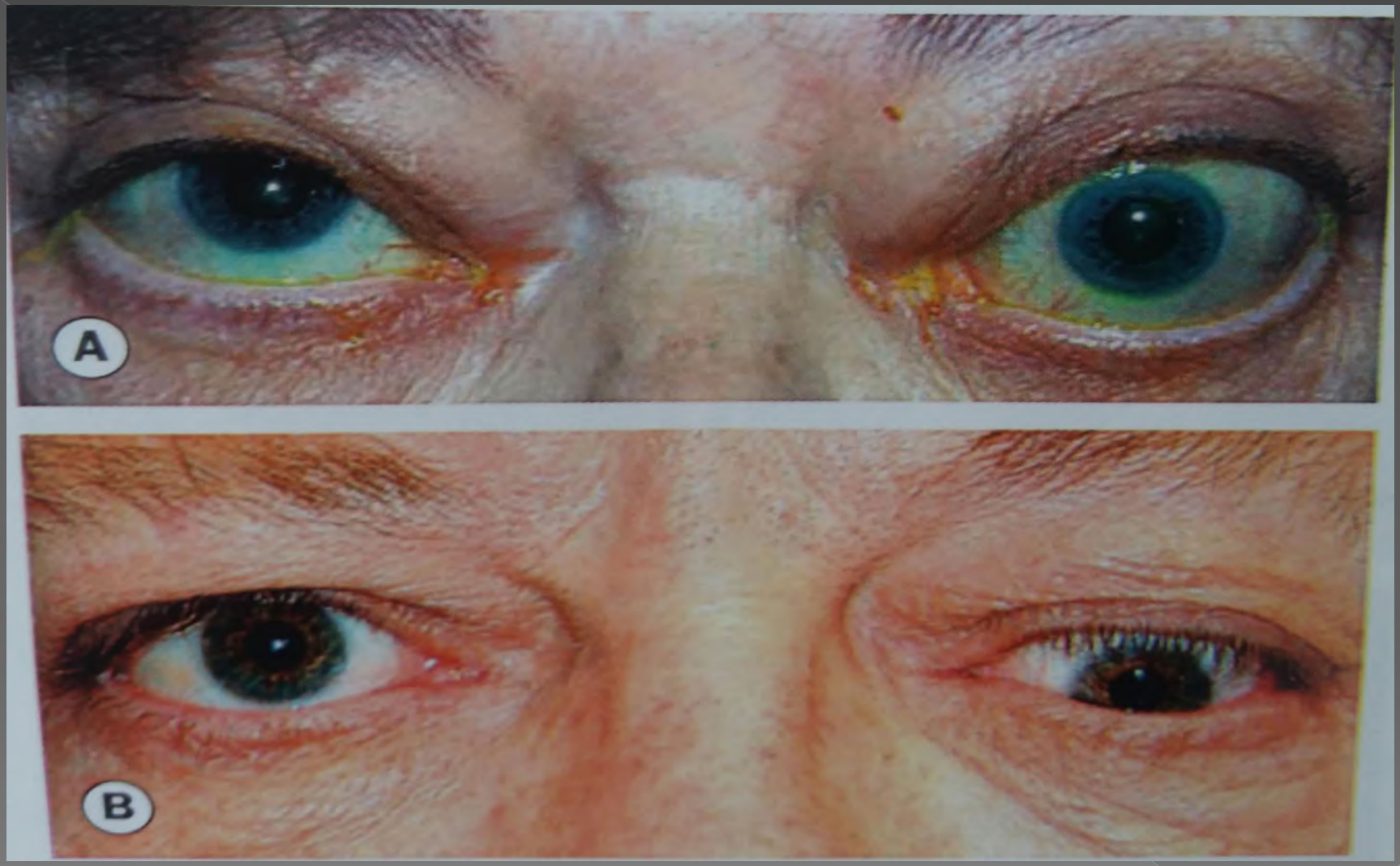
- Diminished VA may be present
- Features of optic nerve dysfunction
- Central or paracentral scotoma which may be combined with nerve fiber bundle defects
- Optic atrophy is present only in very advanced cases.
- Usually the optic nerve appears normal, although it may be swollen.

Treatment

- Initial treatment is either with systemic steroids or radiotherapy
- Orbital decompression is considered if non – surgical treatment is either ineffective or inappropriate.

Restrictive myopathy

- 30-50% of hyperthyroid patients develop ophthalmoplegia
- Diplopia is permanent in 50% of patients
- Ocular motility is restricted by edema during the infiltrative phase and later by fibrosis
- IOP increase in upgaze



(A) Defective elevation of the left eye
(B) Defective depression of the right eye

Four ocular motility defects

1) Elevation defect
2) Abduction defect
3) Depression defect
4) Adduction defect

Treatment

- Indications for surgery:

  Diplopia in the primary or reading positions of gaze.

  The angle of deviation must be stable for at least 6 months.

  No evidence of congestive ophthalmopathy indicative of active disease.

Treatment

- The goals of surgery:

  To achieve binocular single vision in the primary position of gaze and when reading.

The surgical technique:

- The most commonly performed procedure is recession of an inferior rectus and or medial rectus
- Botulinum toxin injection into the involved muscle

Investigations

1. TFTs

   usually TSH and free T4

   Biochemical investigations in TED

TFT	Hyperthyroid	Hypothyroid
TSH	↓	↑
Free T4	↑	↓

2. Thyroid auto antibodies

Immunological investigations in TED

Autoantibody	Association	
Anti – TSH receptor	>95% Graves disease	
Anti – thyroid peroxidase	80% Graves disease	90% Hashimotos thyroiditis
Anti – thyroglobulin	25% Graves disease	55% Hashiwotos thyroidits

3. Orbital imaging

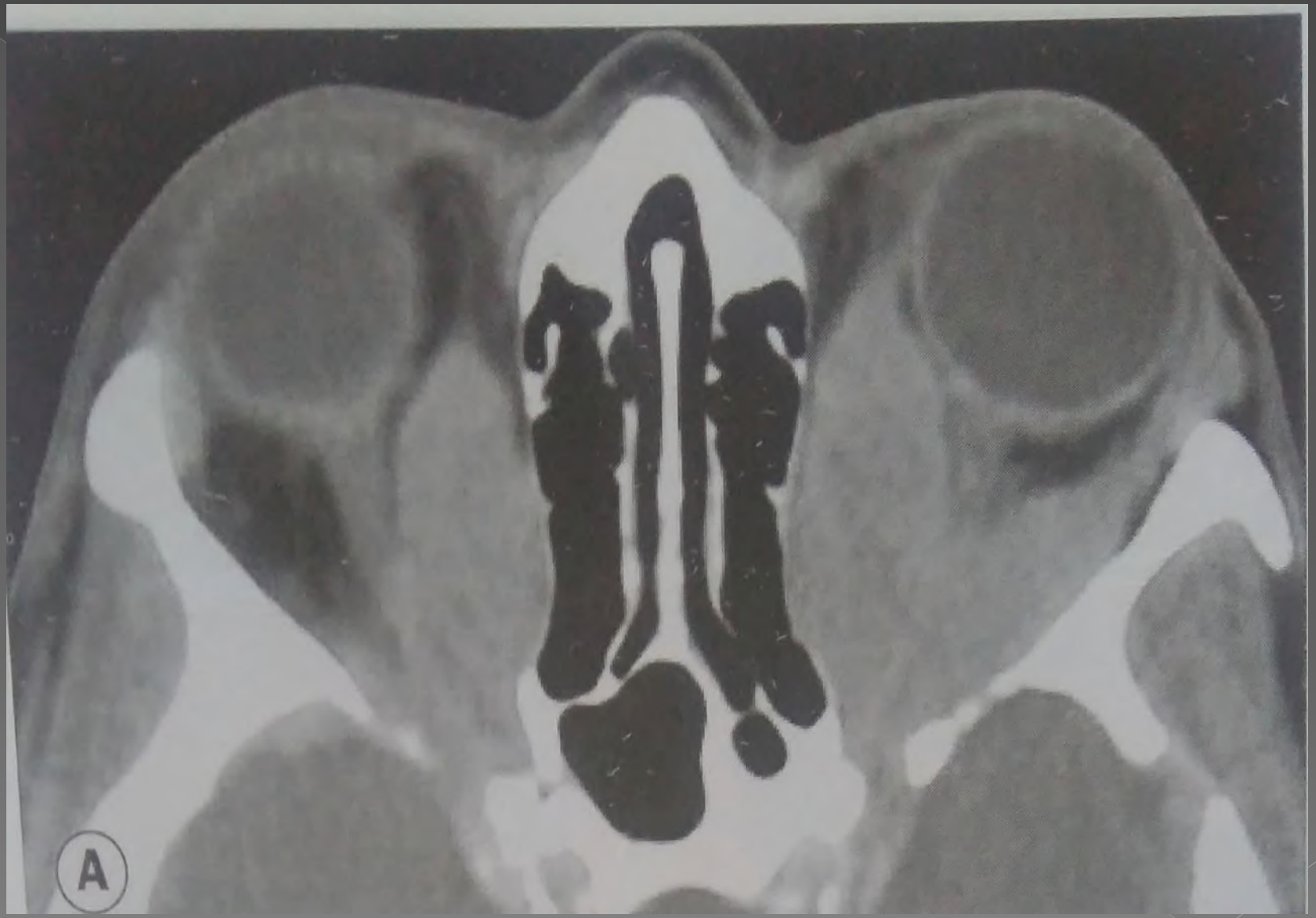
CT orbits – Better bony resolution

Preferred for planning decompression

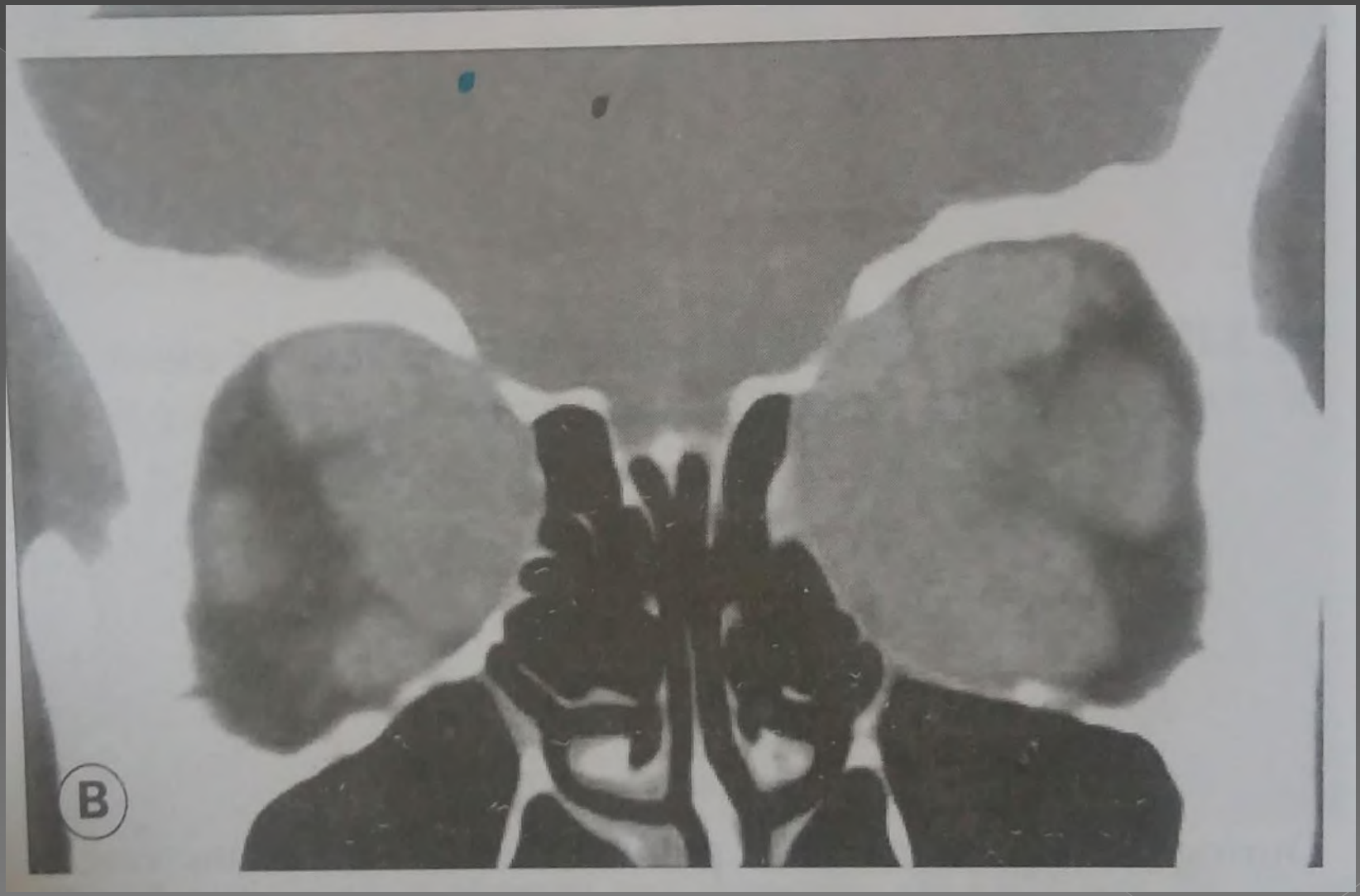
MRI (T2 – weighted & STIR)

Gives better soft tissue resolution

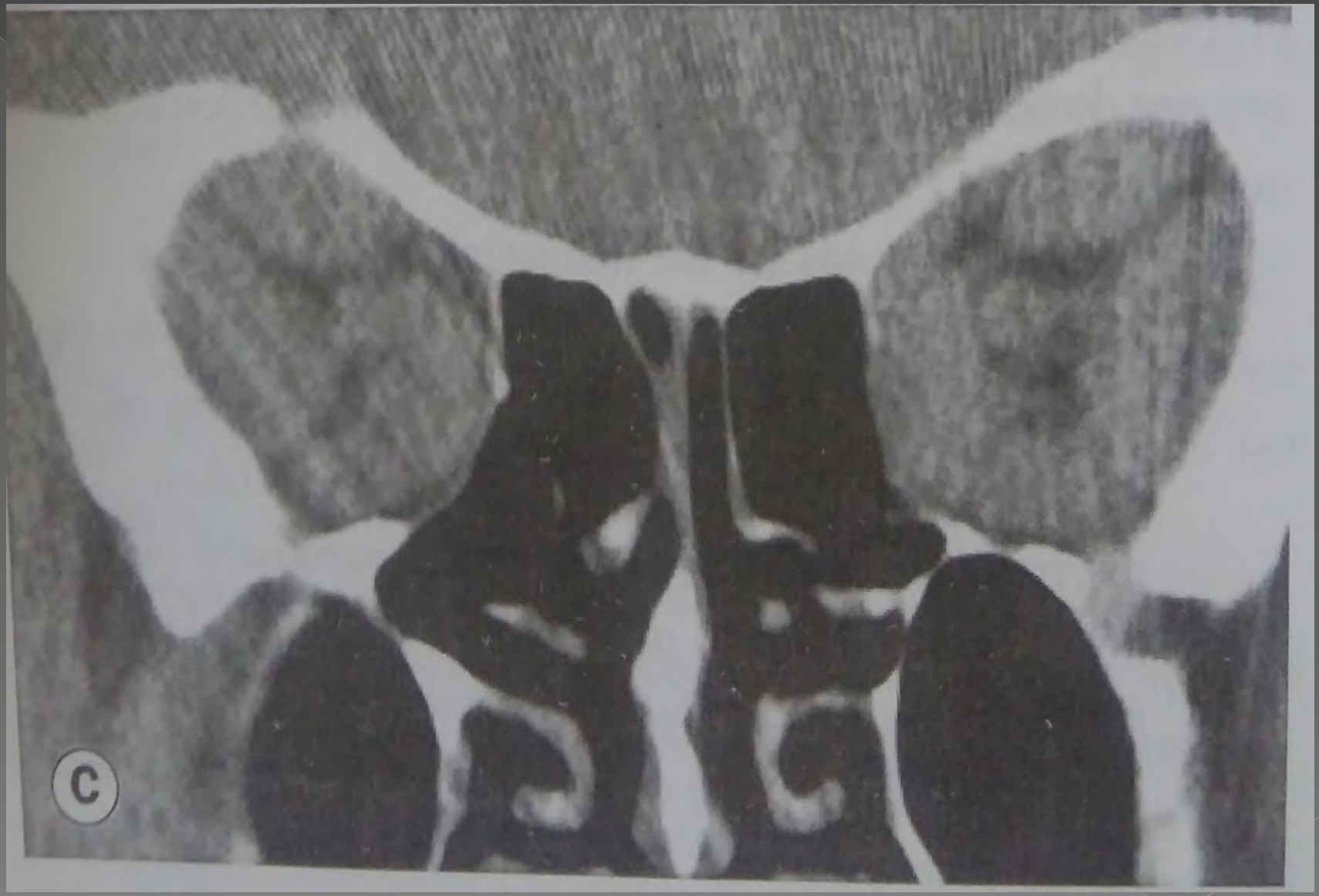
Bellies of muscles show enlargement and inflammation. Tendons spared



Axial view



Coronal view – note sparing of the right lateral rectus muscle



Coronal view shows crowding at the orbital

4. Orthoptic review

   May include – Field of binocular single vision

   Field of uniocular fixation

   Hess / Less chart

   Visual Field

General principles of management of TED

General

- Multidisciplinary input from Endocrinologist and Orthoptist.
- Supportive: counseling ocular lubricants, tinted glasses, bed head elevation. Prisms for diplopia.
- Smoking cessation

Medical

- Immuno suppression – in active disease
- Usually with systemic steroids, include ciclosporin, methotrexate, azathioprine or rituximab.
- Radiotherapy can be used, but not for sight threatening optic neuropathy.

Surgical

- For acute disease:

  Acute progressive optic neuropathy corneal exposure ➡ Emergency orbital decompression.

- For burn out disease:

  Surgery may improve function and cosmesis

  Decompression ➡ motility ➡ Lid surgery

Treatment of hyperthyroidism

Carbimazole, propylthiouracil ➡ Block production of thyroid hormones

Radioactive Iodine ➡ A single oral dose of 400 or 600MBq is given

Surgical thyroidectomy ➡ Total or subtotal preceded by radio active iodine to shrink the goitre

Treatment of hypothyroidism

- Levothyroxine

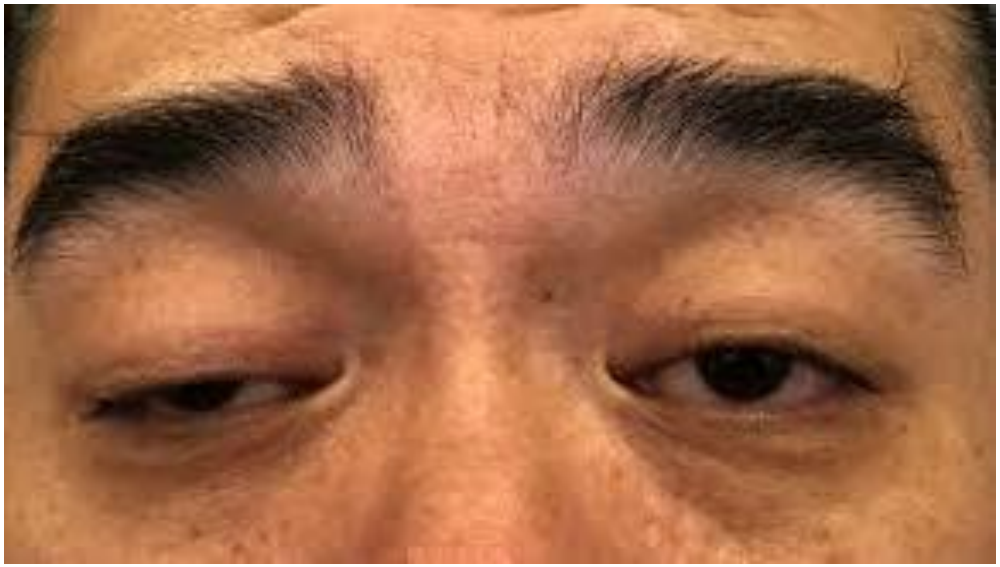
  Thyroxine replacement

Selenium and mild TED

- Antioxidant selenium
- Comparatively better quality of life
- Less ophthalmic involvement
- Reduced TED progression
- No adverse side effects.

THANKS

Myasthenia Gravis



Prof SofiaIqbal
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Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

Presentation lay out

- Introduction
- Definition
- Etiology
- Pathophysiology
- Role of thymus gland
- Types
- Classification
- Diagnosis
- Differential diagnosis
- Management

INTRODUCTION

- **A neurological / neuromuscular autoimmune disorder**
- **Error in the transmission of nerve impulses to muscles at the neuromuscular junction—the place where nerve cells connect with the muscles they control**
- Antibodies to the **acetylcholine receptor (AChR)**,nicotinic receptors found in the serum of 85% of patients
- **Affects 1 in 10,000 population**
- **Leads to weakness and fatigability**

Potential Risk Factors for Developing Myasthenia Gravis

Women 20-40 years old and men 50-80 years old

People who have rheumatoid arthritis or lupus

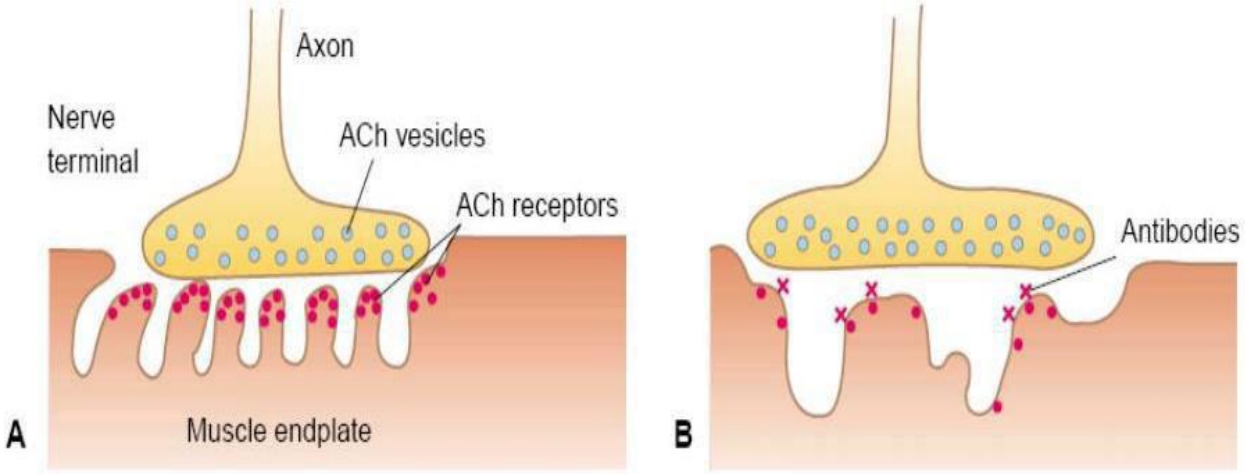
Taking certain medications for malaria, heart arrhythmia, antibiotics and psychiatric drugs

Having undergone extensive surgeries in the past

Issues with the thyroid gland

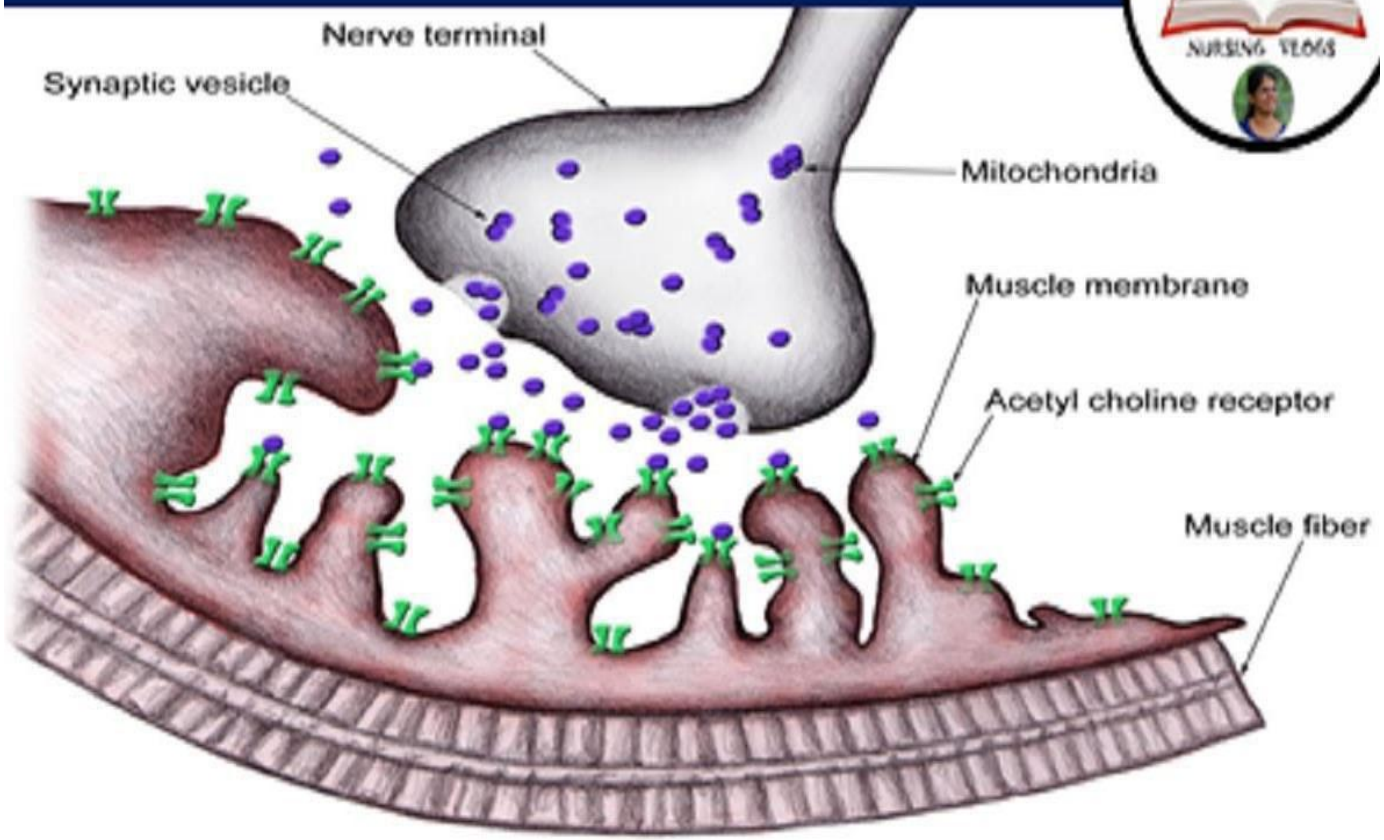
PATHOPHYSIOLOGY

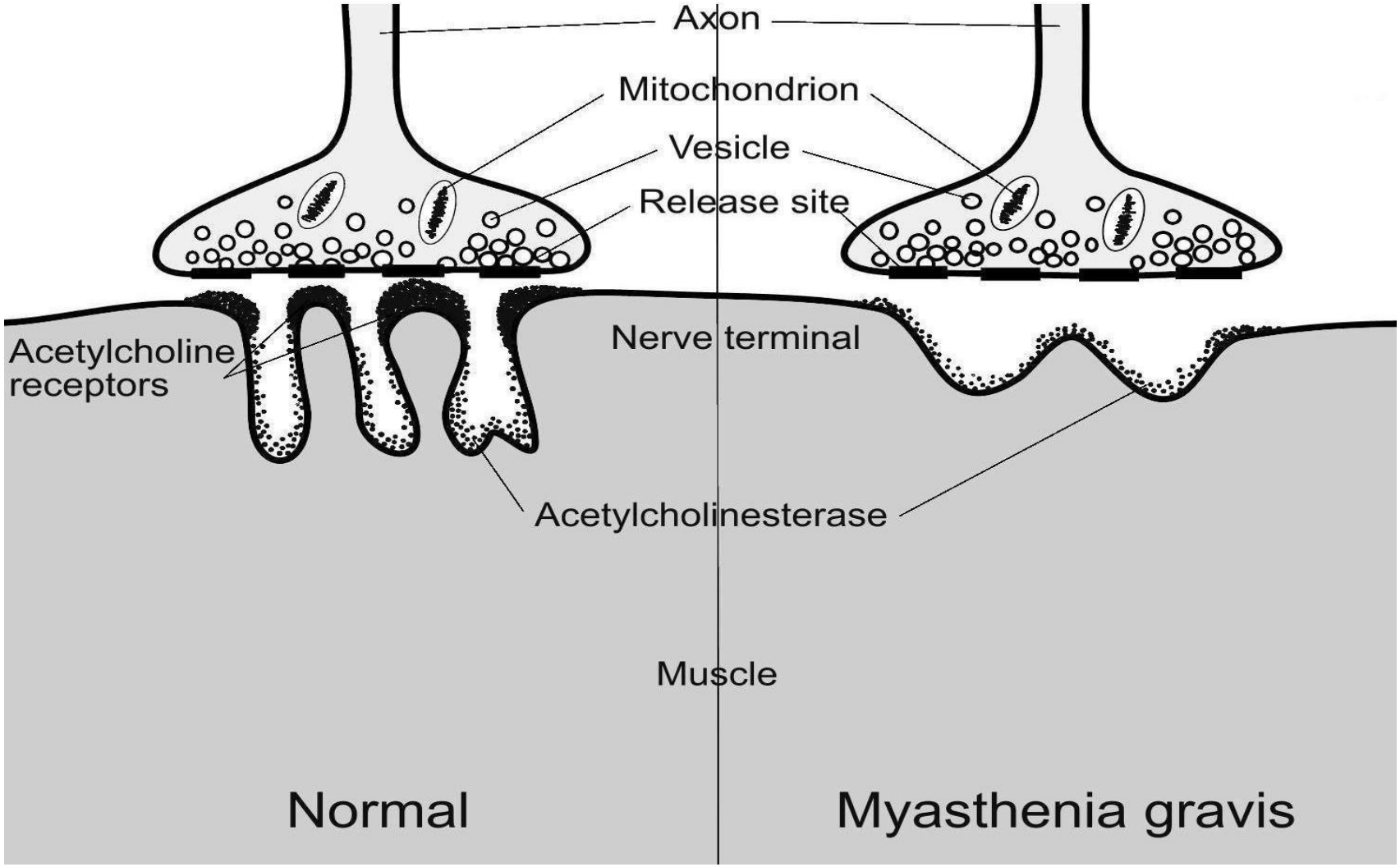
- Normally, a chemical impulse precipitates the release of acetylcholine from vesicles on the nerve terminal at the myoneural junction. The acetylcholine continuously bind to the receptor sites on the motor end plate, for muscle contraction to sustain.

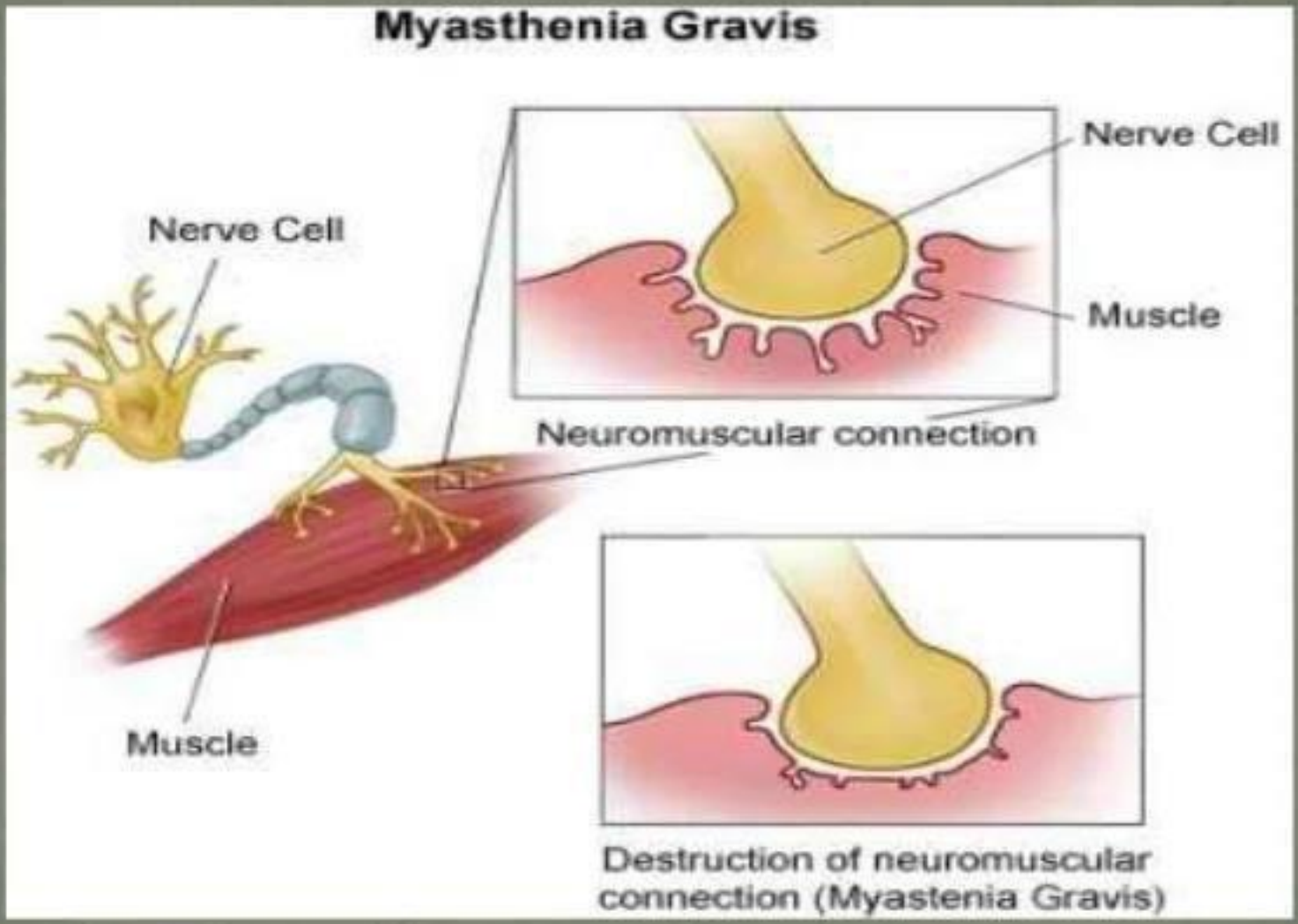


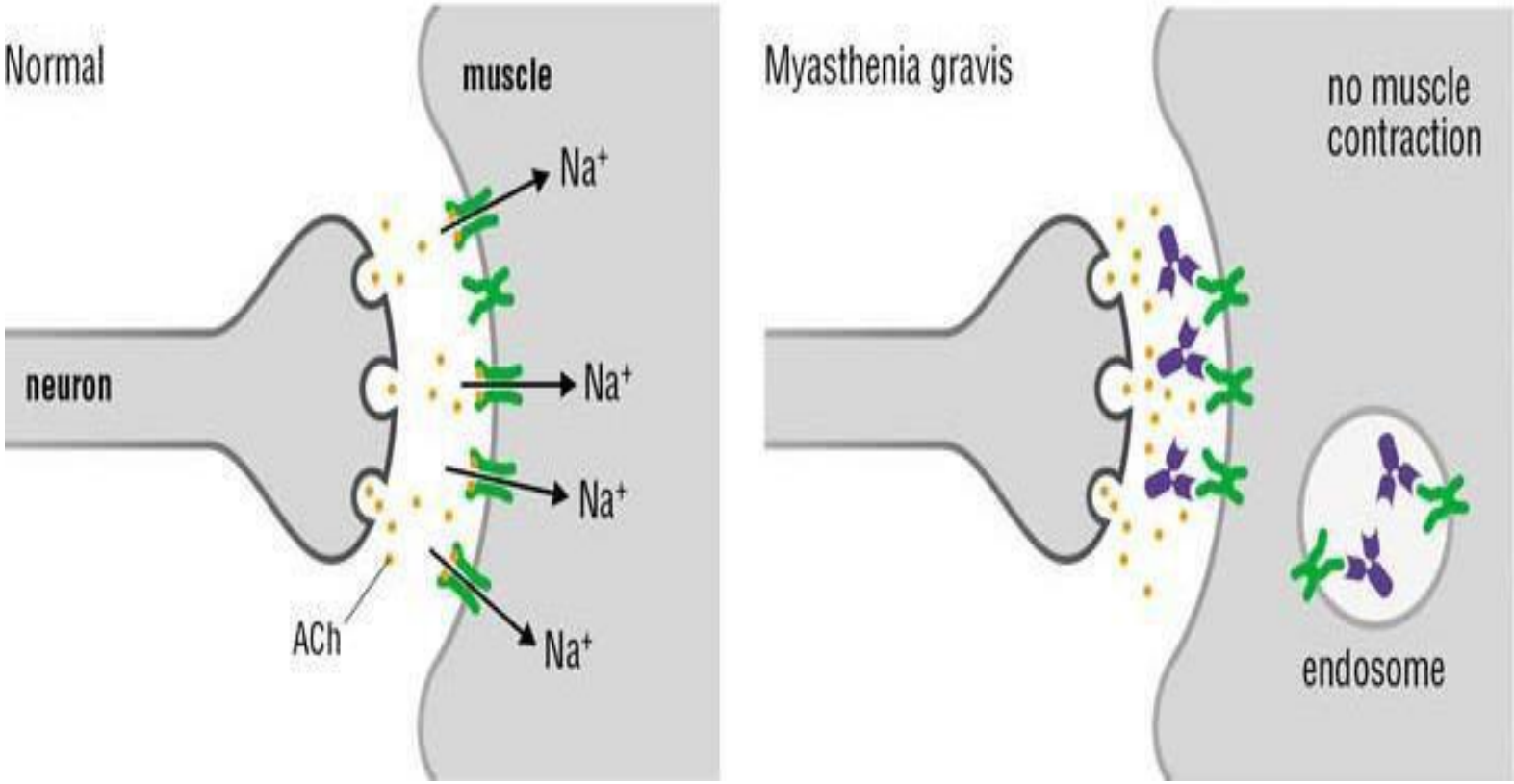
Myasthenia gravis. (A) Normal ACh receptor site. (B) ACh receptor site in myasthenia gravis.

MYASTHENIA GRAVIS



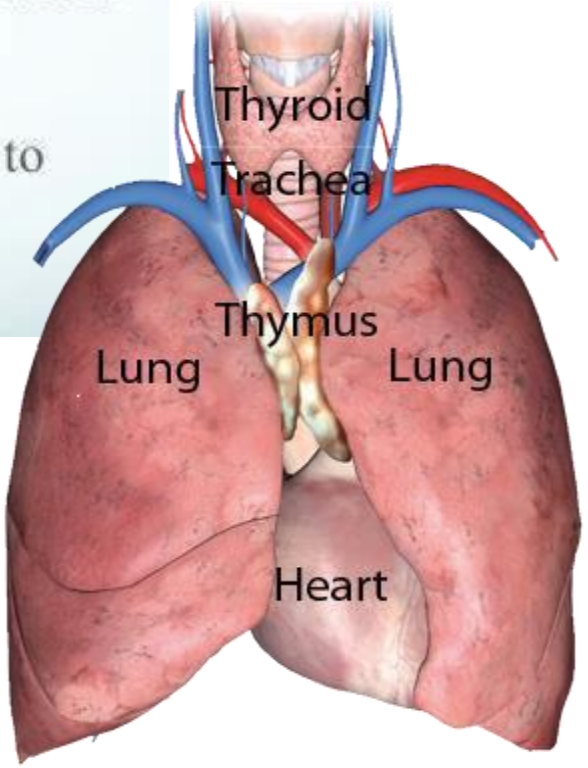

Axon
Mitochondrion
Vesicle
Release site
Acetylcholine receptors
Nerve terminal
Acetylcholinesterase
Muscle
Normal
Myasthenia gravis

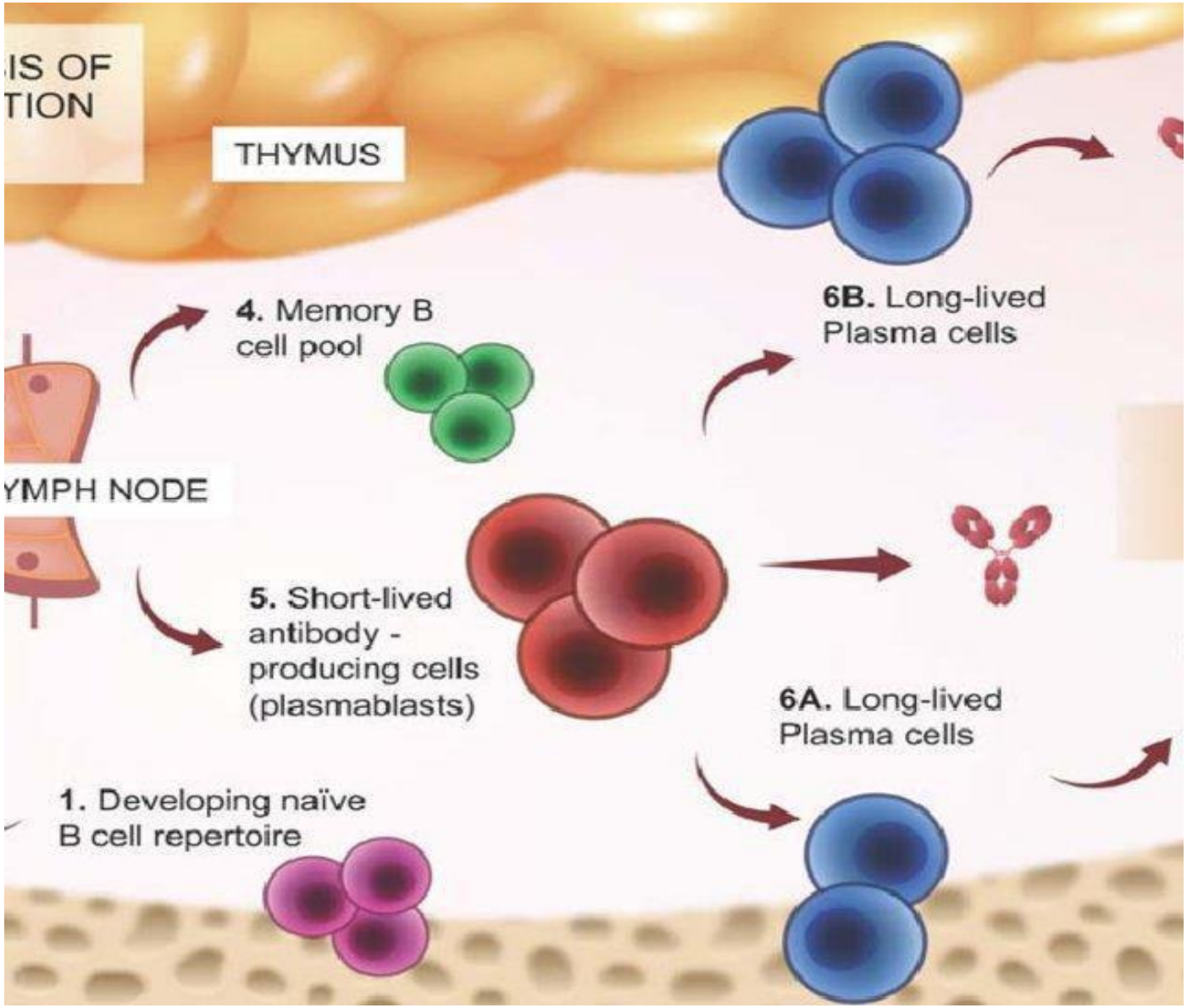

Myasthenia Gravis
Nerve Cell
Nerve Cell
Muscle
Neuromuscular connection
Muscle
Destruction of neuromuscular connection (Myastenia Gravis)


Normal
muscle
Na+
Na+
Na+
Na+
neuron
ACh
Myasthenia gravis
no muscle
contraction
endosome

ROLE OF THYMUS GLAND

- The factors that trigger the autoimmune process are not known, but the thymus gland is involved.
- The thymus lies behind the sternum and may extend down to the diaphragm or up to the neck.
- This gland plays a role in the responsiveness of T cells to foreign antigens.




IS OF
TION
THYMUS
4. Memory B
cell pool
6B. Long-lived
Plasma cells
YMPH NODE
5. Short-lived
antibody -
producing cells
(plasmablasts)
6A. Long-lived
Plasma cells
1. Developing naïve
B cell repertoire

Pathophysiology:-

- Due to etiological factors

↓

- Lymphocyte produce acetylcholine receptor antibodies that attack the post synaptic muscle membrane

↓

- Depletion of acetylcholine receptor of the neuromuscular junction

↓

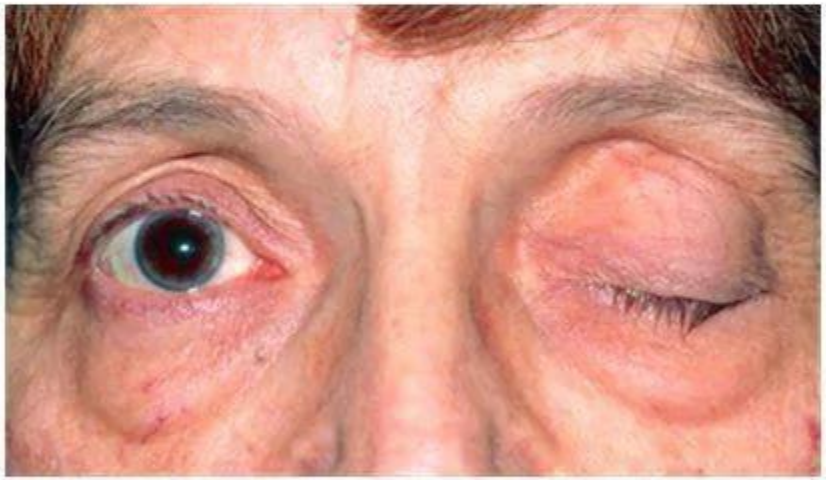
Defect in the transmission of impulse from nerve to muscle cell

↓

- Myasthenia gravis

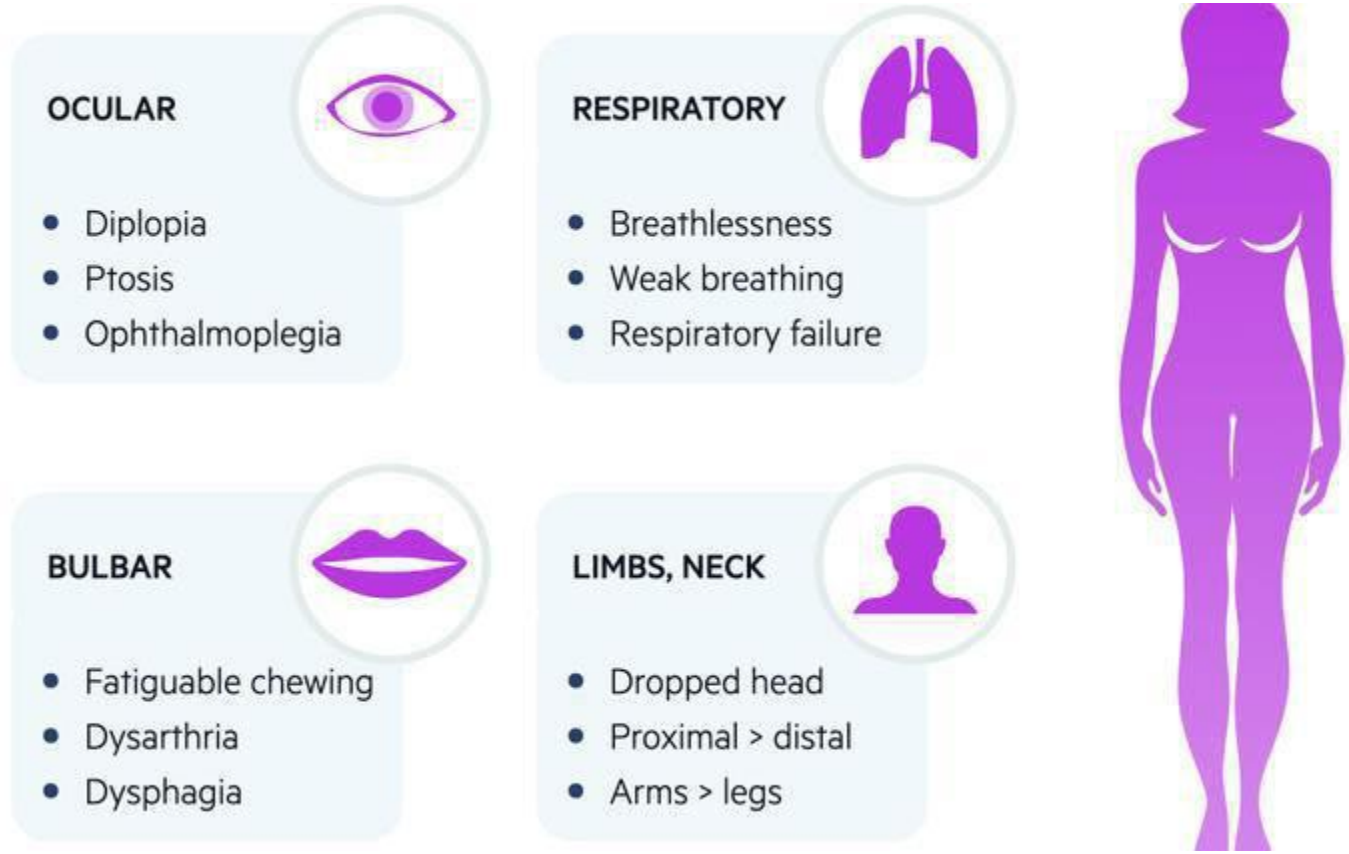
Clinical Manifestations

- Muscle weakness
- Double vision (diplopia)
- weak eyelids (unilateral ptosis)
- Difficulty speaking or smiling
- Difficulty chewing and swallowing



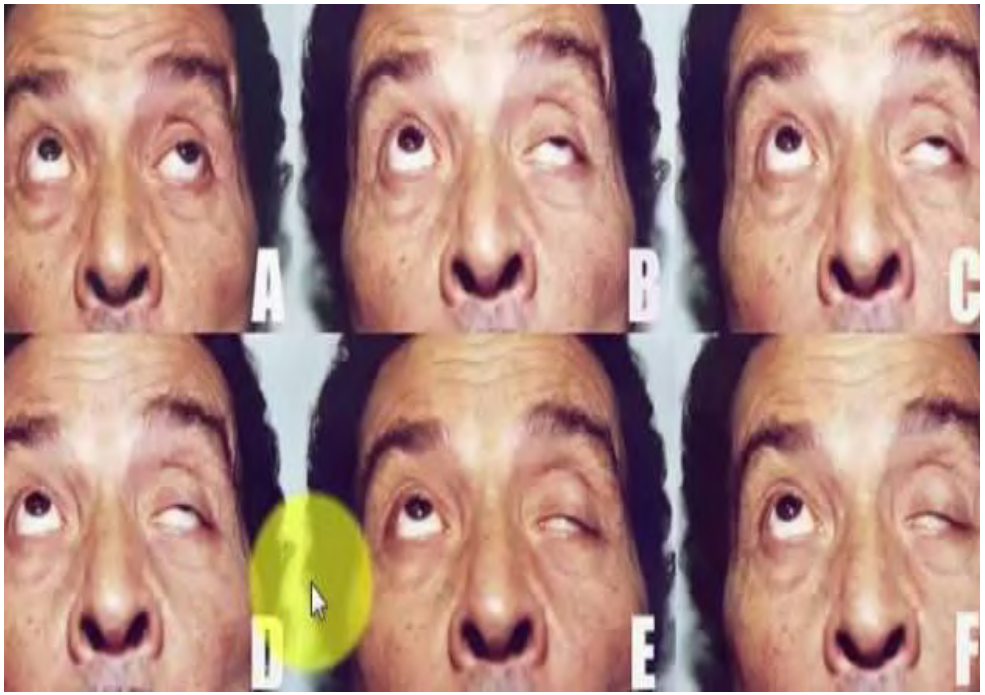
TYPES OF MG

- **OCULAR/BULBAR/GENERALIZED**



OCULAR MYASTHENIA

- **Ocular myasthenia gravis (OMG) can mimic isolated cranial nerve palsies, gaze palsies, internuclear ophthalmoplegia, blepharospasm, and even a stroke**



Strabismus types	Number of patients (%)
Vertical deviation	6 (28.6)
Exotropia and vertical deviation	5 (23.8)
Esotropia	4 (19.0)
Esotropia and vertical deviation	3 (14.3)
Exotropia	3 (14.3)
Total	21 (100.0)

Classification

- Class I: Eye muscle weakness only
- Class II: Eye muscle weakness
  + mild weakness of other muscles
- Class III: Eye muscle weakness
  + moderate weakness of other muscles
- Class IV: Eye muscle weakness
  + severe weakness of other muscles

  OR need for nasogastric feeding
- Class V: Intubation needed to maintain airway

Diagnosis: CLINCAL, SEROLOGIC AND EMG FINDINGS

1.Clinical DX:

-Bedside: ice pack test/ Edrophonium test
- Cogan sign
- Peek sign

Imaging:

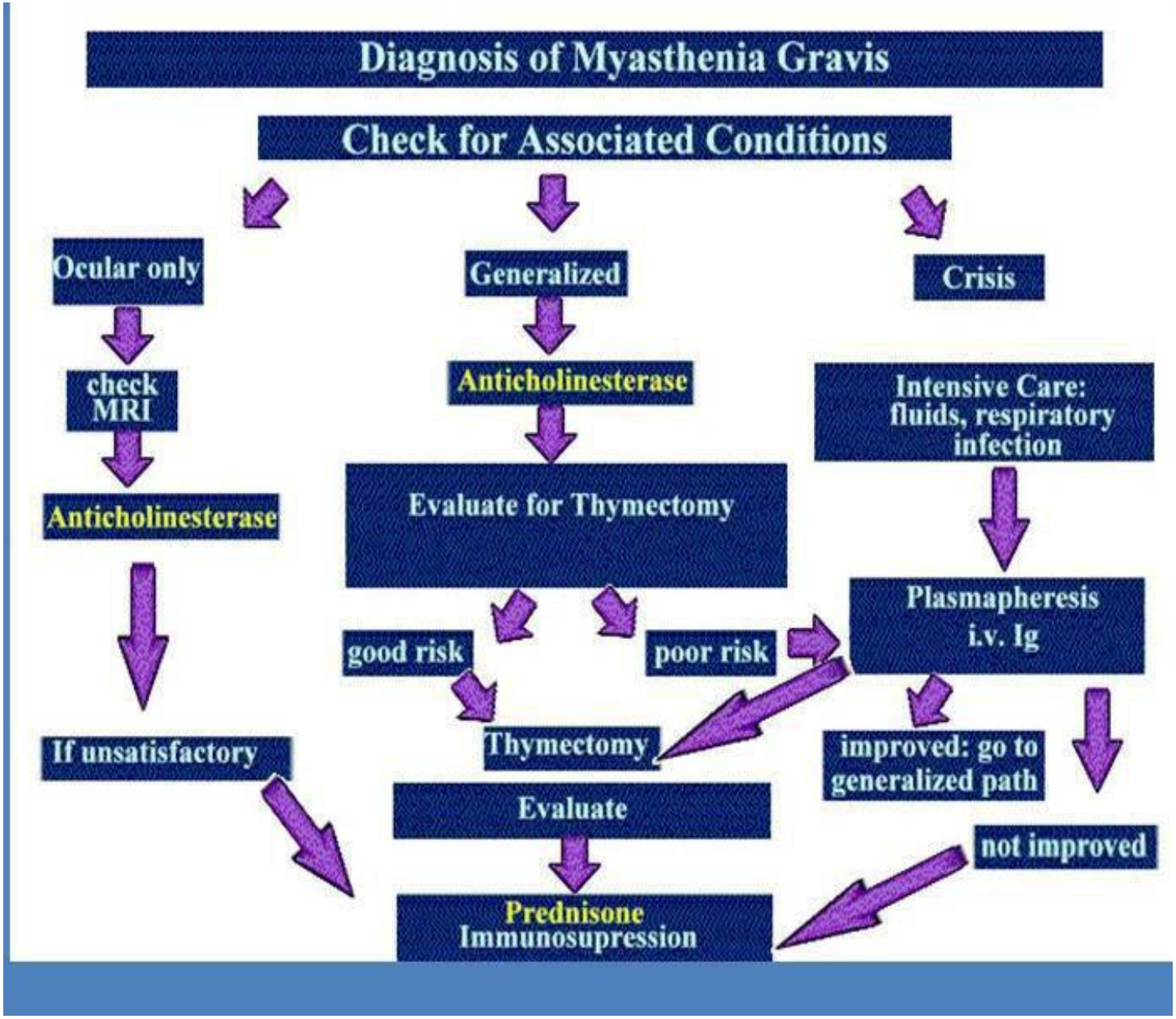
CT CHEST: Evaluate for thymoma

2.Electrophysiologic confirmation:

-Repetitive nerve stimulation
-Single fiber electromyography

3.Labs:

AchR antibodies- first step in immunologic assay
MuSK antibodies
LRP4 antibodies


Diagnosis of Myasthenia Gravis
Check for Associated Conditions
Ocular only
Generalized
Crisis
check MRI
Anticholinesterase
Intensive Care: fluids, respiratory infection
Anticholinesterase
Evaluate for Thymectomy
Plasmapheresis i.v. Ig
good risk
poor risk
If unsatisfactory
Thymectomy
improved: go to generalized path
Evaluate
not improved
Prednisone
Immunosupression

Test	Positive Result
Fatigue test	Worsening of symptoms after prolonged use
Ice test or sleep test	Improvement of ptosis after ice pack application or period of rest
Edrophonium (Tensilon or Enlon)	Improvement in symptoms within 30-60 seconds
Serologic screening	Identification of circulating AchR, MuSK or LRP4 antibodies
Electrophysiologic testing (RNS, SFEMG)	Decrease in action potential of stimulated nerves
Thyroid panel, thoracic imaging	Used to identify coexisting conditions

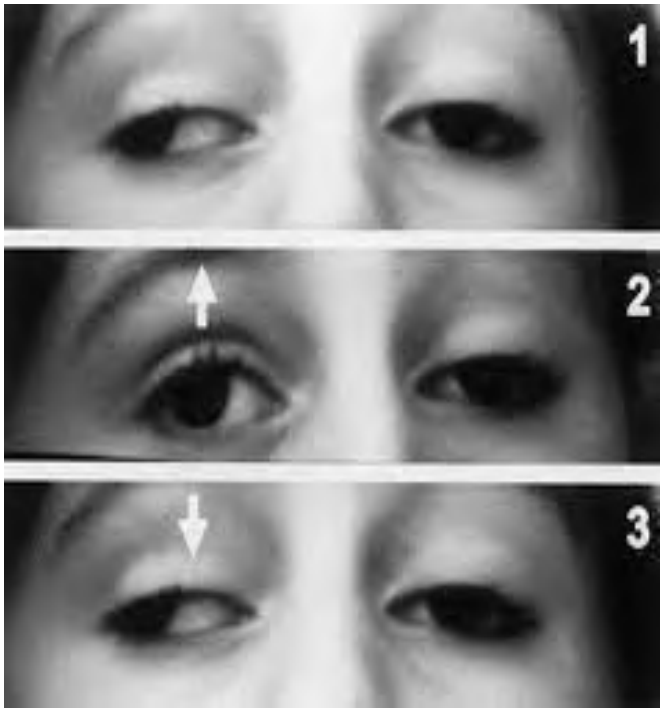
RNS = repetitive nerve stimulation; SFEMG = single-fiber electromyography

ICE PACK TEST

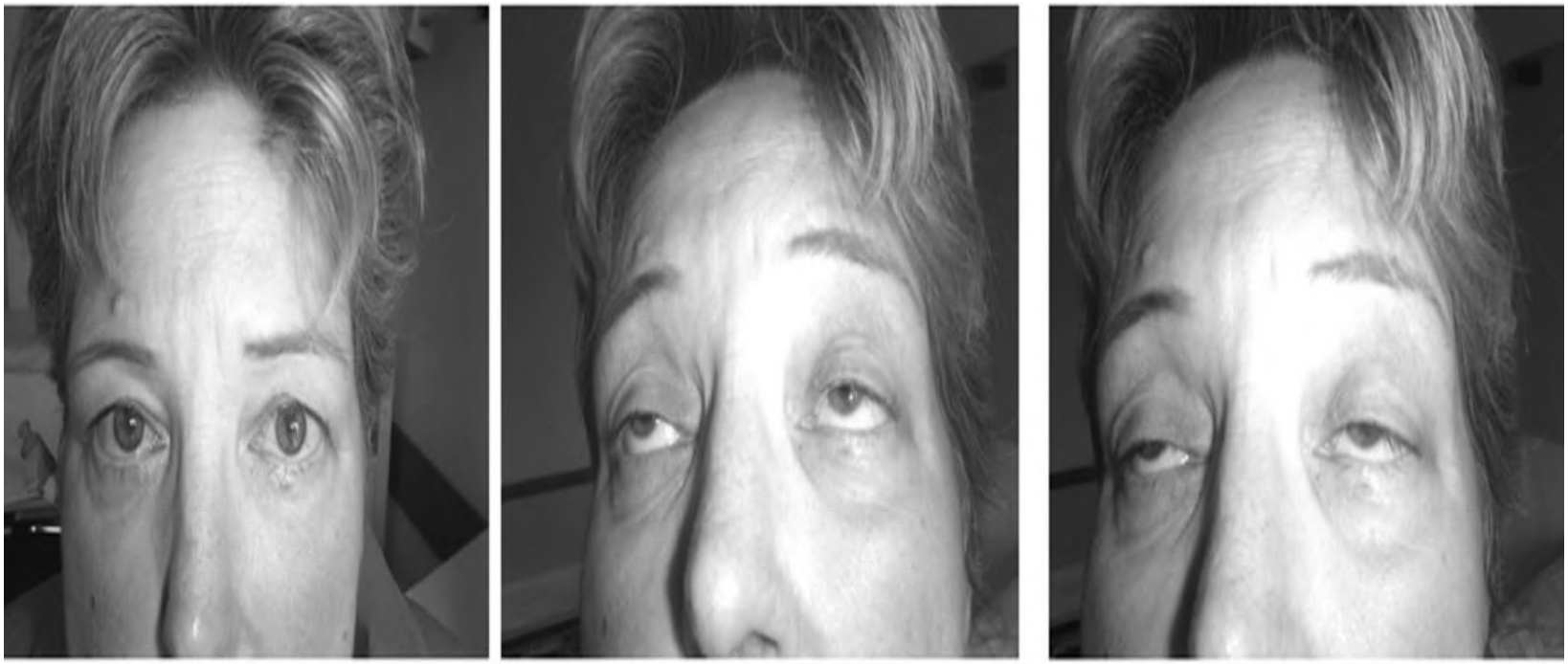
- Apply ice pack for 3 to 5 minutes
- Bed side test
- **Cold improves neuromuscular transmission**
- **Sensitivity of 85%**

Cogan's sign

- Ask the patient to gaze downward for 10–15 seconds and then returning to primary gaze
- Cogan's sign is present **when the affected lid briefly "twitches" upward on returning to primary gaze**

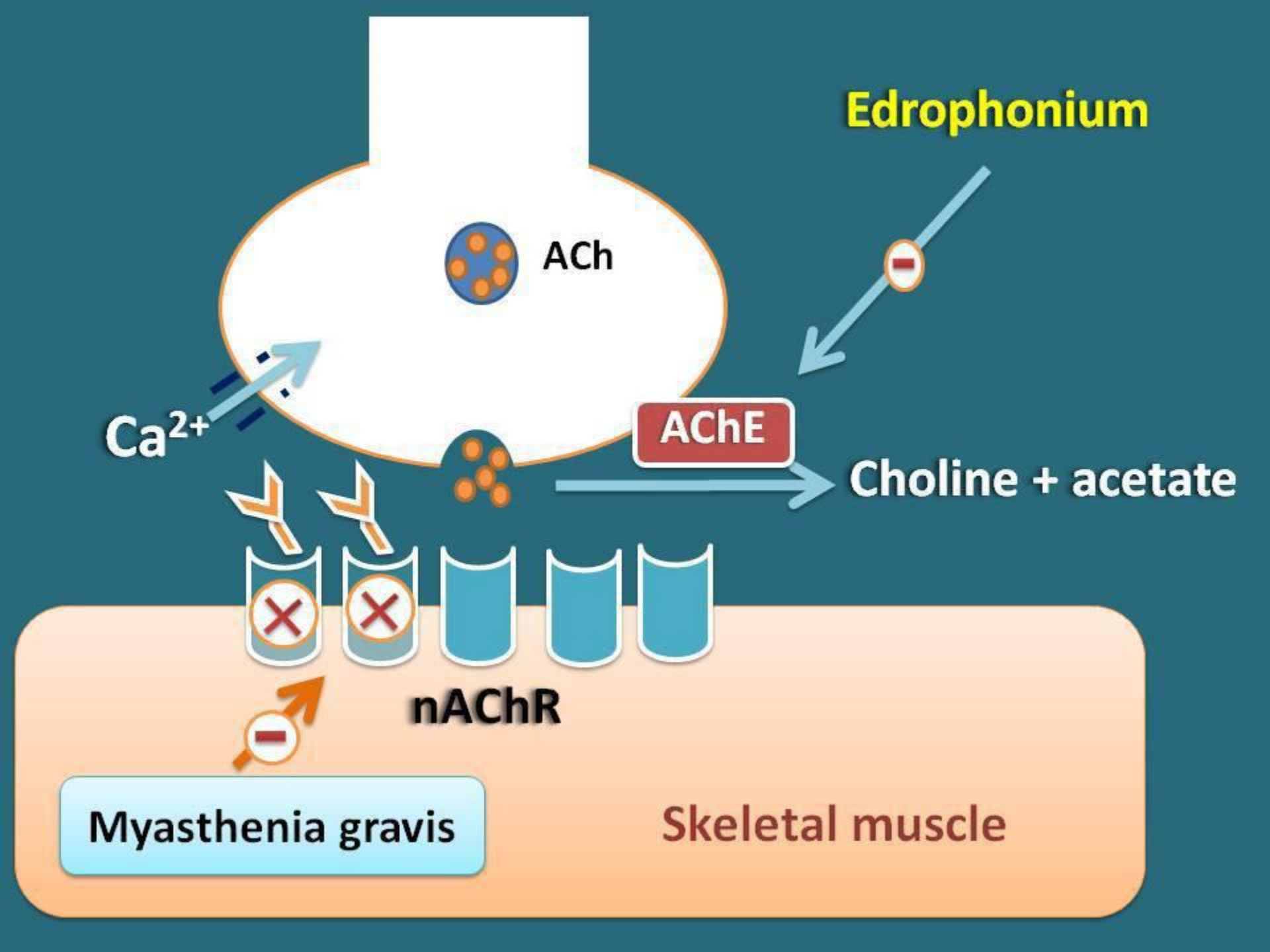


FATIGUIBILITY TEST



Tensilon Test

- Edrophonium chloride
  - Inhibits acetylcholinesterase
- Onset 30 seconds; duration 5-10 min
- NEED A CLEAR OBJECTIVE ENDPOINT
  - Works best with complete ptosis
- Compare to placebo (saline)
- Prepare atropine
- Give test dose 1-2 mg then up to 10 mg total
- SFX:
  - salivation, sweating, nausea, abdo cramping, fasciculations; hypotension & bradycardia are rare (may be as low as 0.16%)
- Sensitivity 71.5- 95%
- Specificity: not clear but can be positive in many other conditions (even ALS or normal controls)
- Not availible


Edrophonium
ACh
Ca2+
AChE
Choline + acetate
nAChR
Myasthenia gravis
Skeletal muscle

How it is given?

Initial safety check

Edrophonium

2 mg by IV

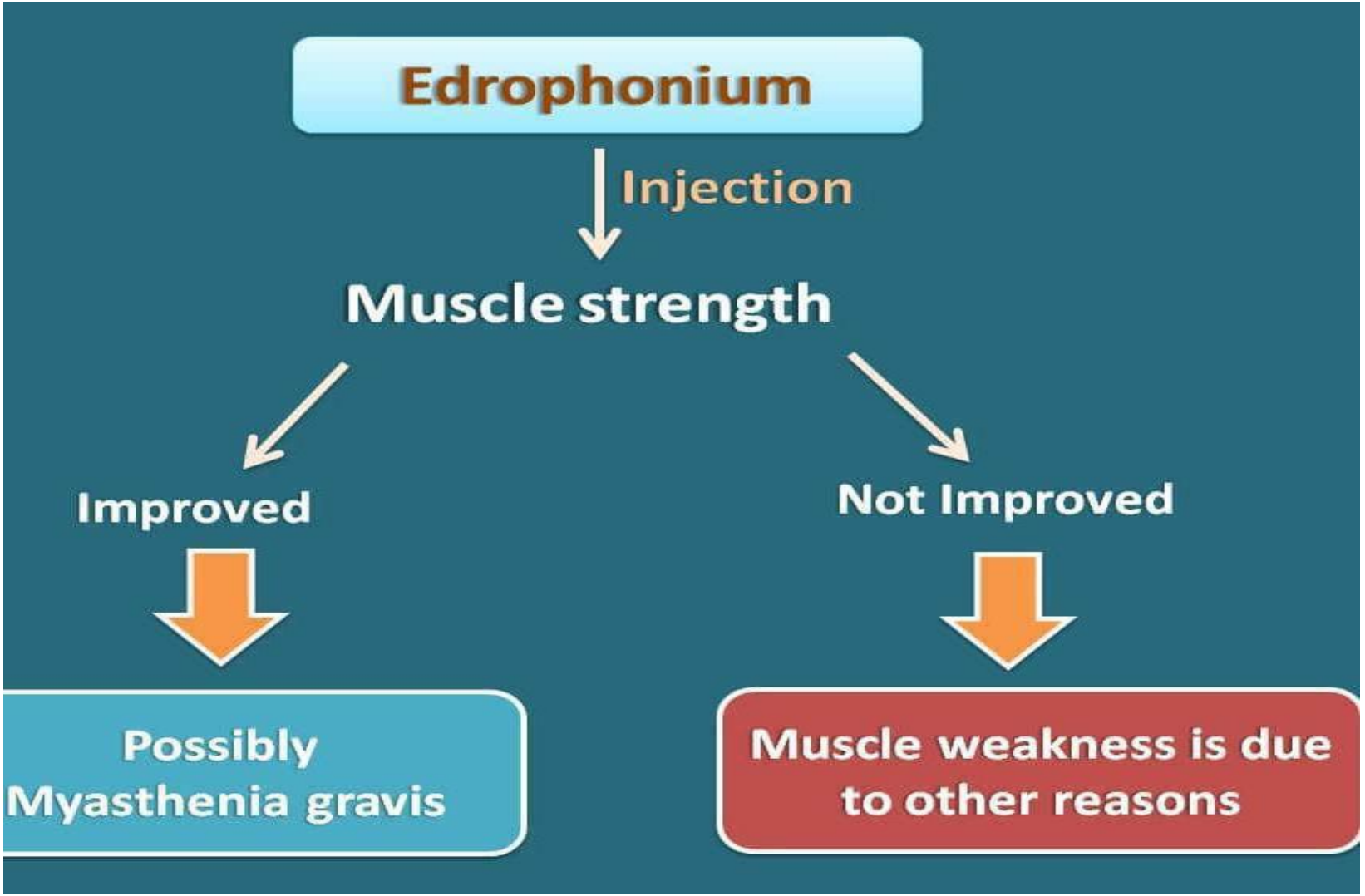
Check for any side effects

Diagnosis step

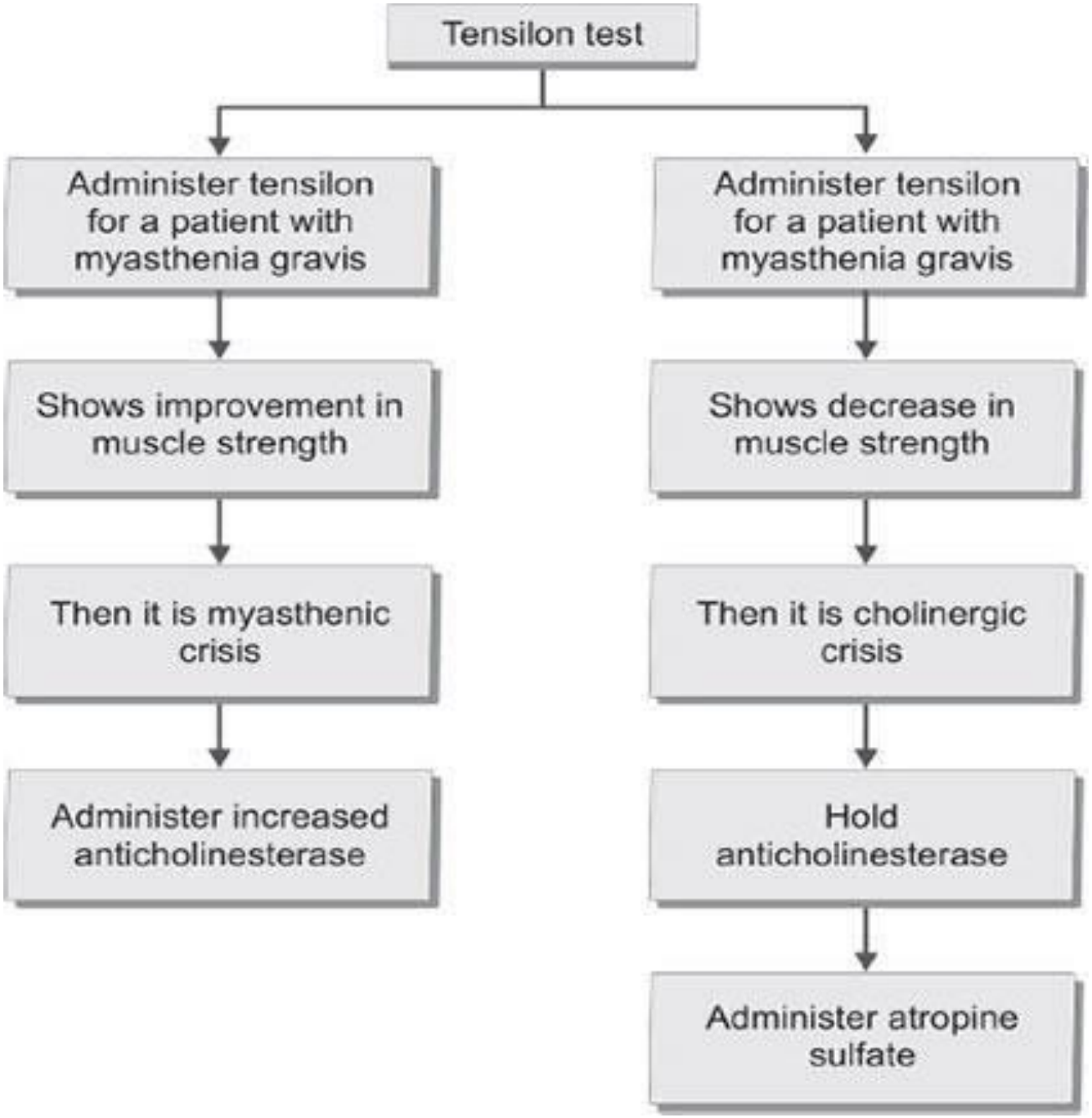
Edrophonium

8 mg by IV

Check for improvement in muscle strength


Edrophonium
Injection
Muscle strength
Improved
Not Improved
Possibly
Myasthenia gravis
Muscle weakness is due
to other reasons

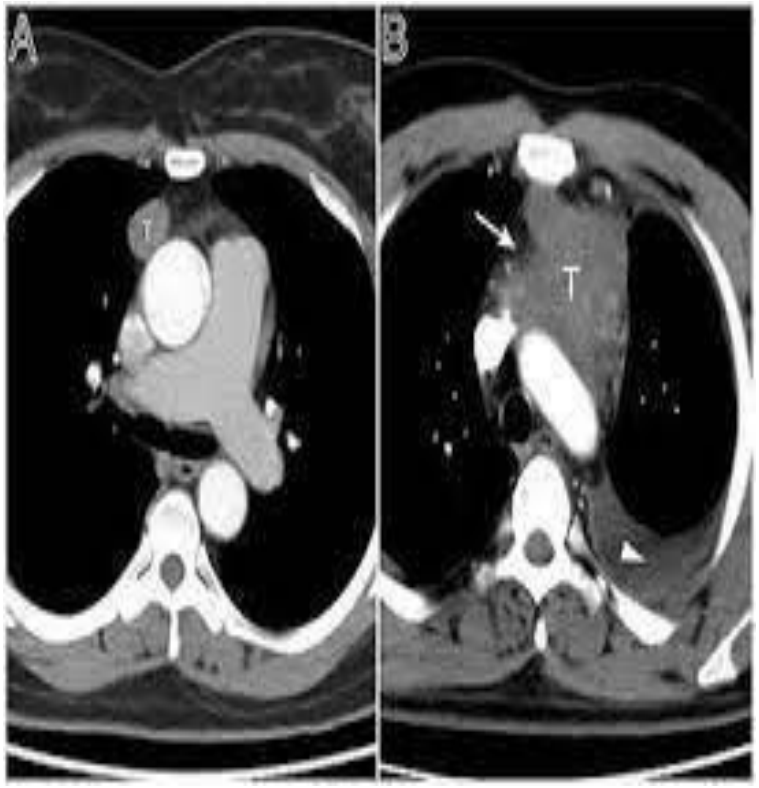
Tensilon test

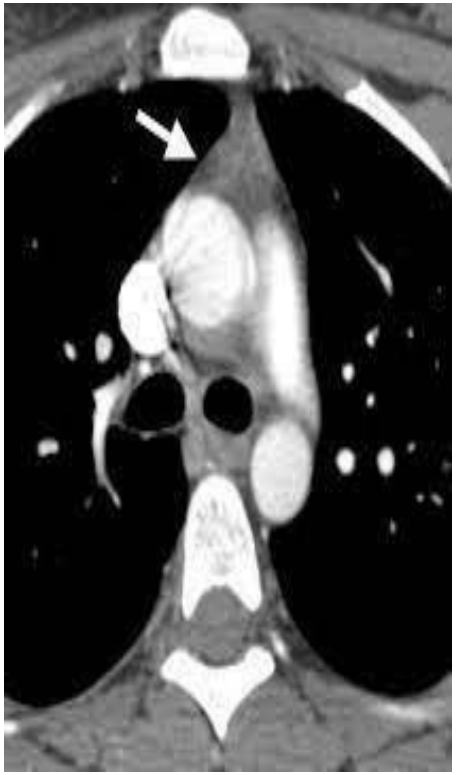

Tensilon test
Administer tensilon for a patient with myasthenia gravis
Shows improvement in muscle strength
Then it is myasthenic crisis
Administer increased anticholinesterase
Administer tensilon for a patient with myasthenia gravis
Shows decrease in muscle strength
Then it is cholinergic crisis
Hold anticholinesterase
Administer atropine sulfate

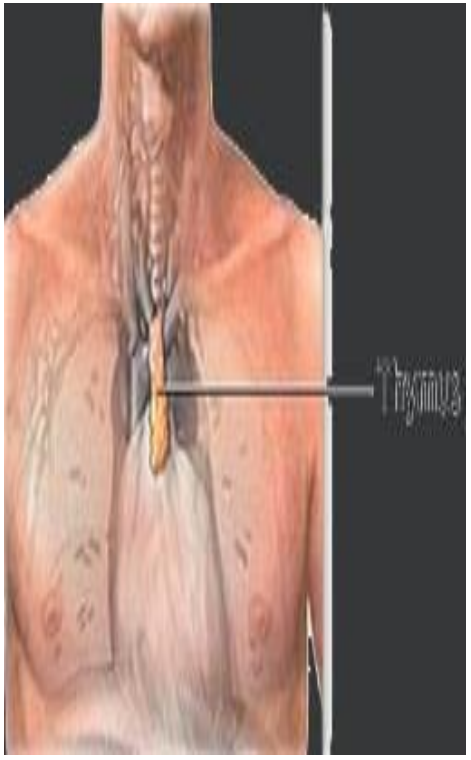
Myasthenic Crisis vs. Cholinergic Crisis

- Mysathenic Crisis
  - Serious complication where patients are unable to breathe adequately and possibly develop respiratory failure
  - Impaired swallowing and managing of secretions leading to aspiration
  - Monitory NIF, vital capacity, tidal volume
- Cholinergic Crisis
  - Due to and excess of acetylcholine at the NMJ as seen in organophosphate poisoning
  - Fasiculations, sweating, myosis, abdominal pain, bradycardia
  - Flaccid paralysis and respiratory failure
- Differentiate with edrophonium test

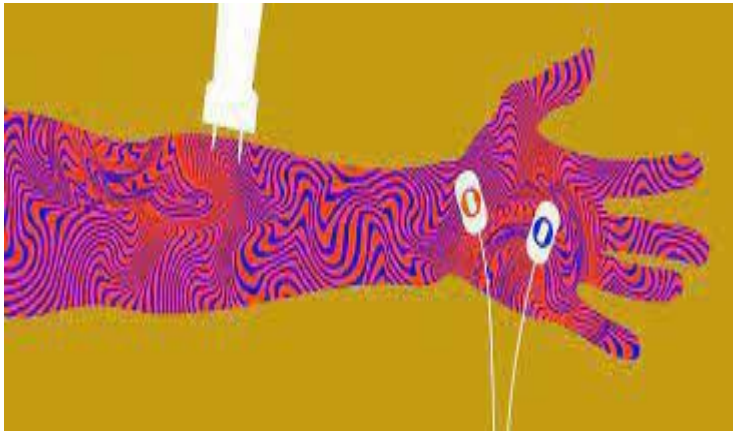
THYMOMA RADIOLOGY

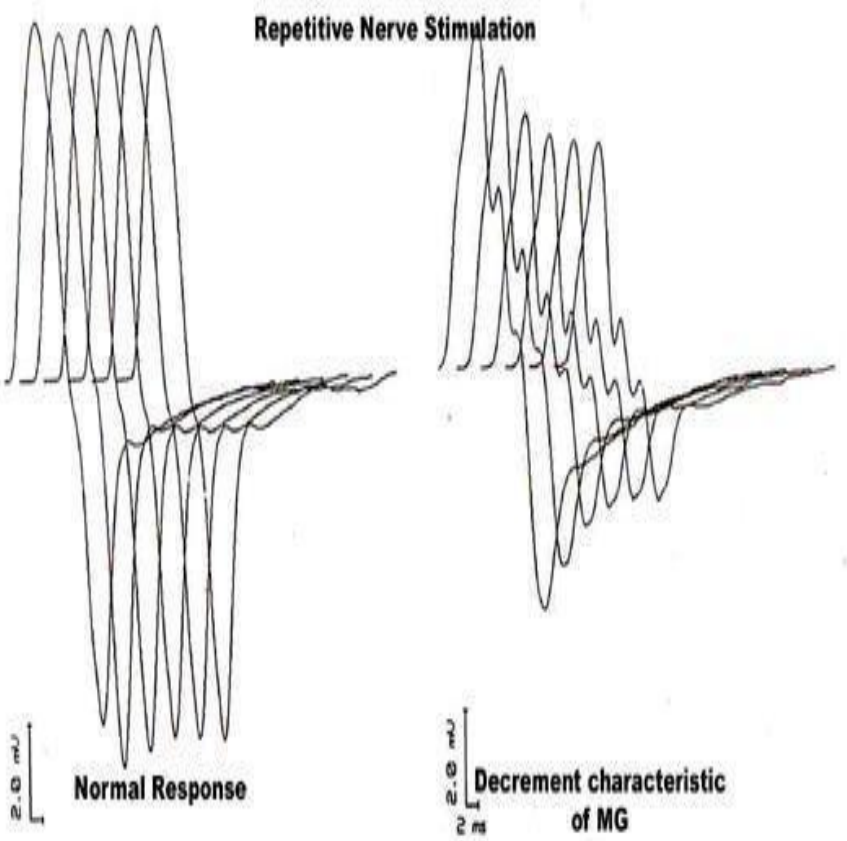




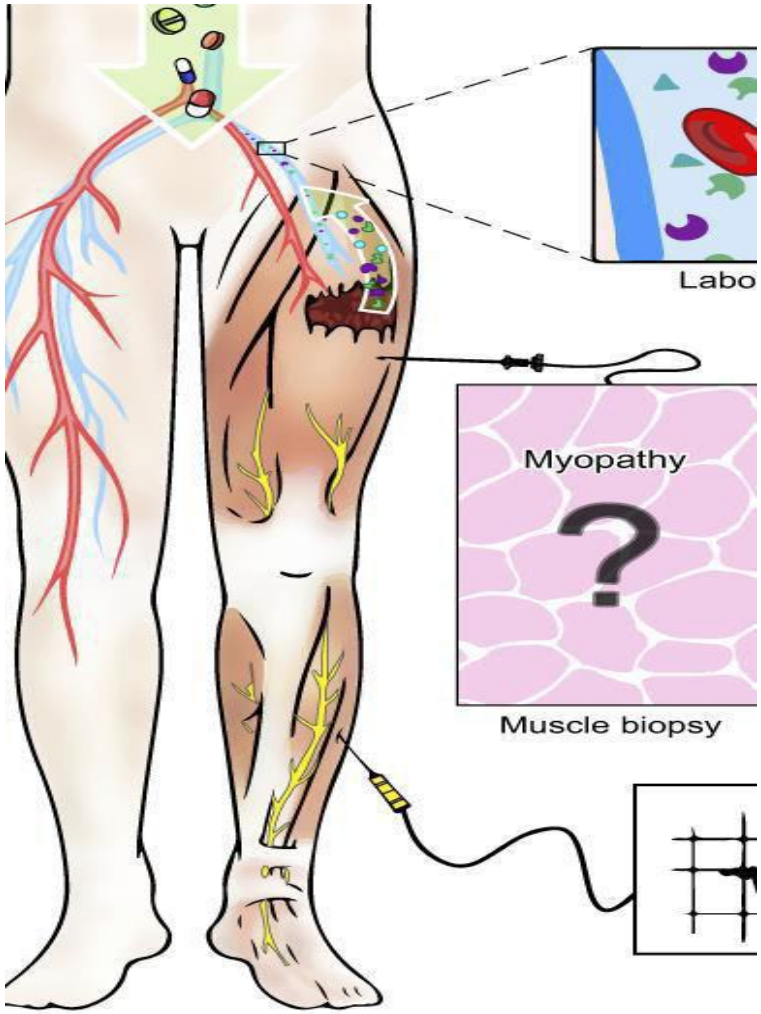


ELEECTROMYOGRAPHY
EMG Studies





MUSCLE BIOPSY



DIFFERENTIAL DIAGNOSIS

Thyroid opthalmopathy
Kearns-Sayre syndrome
Myotonic dystrophy
Brain stem/ Cranial nerve pathology
Generalized fatigue
ALS
Lambert Eaton myasthenia syndrome
Miller Fischer and PCB variants of GBS
Botulism
Penicillamine induced myastheniay

Lambert Eaton myasthenic syndrome:

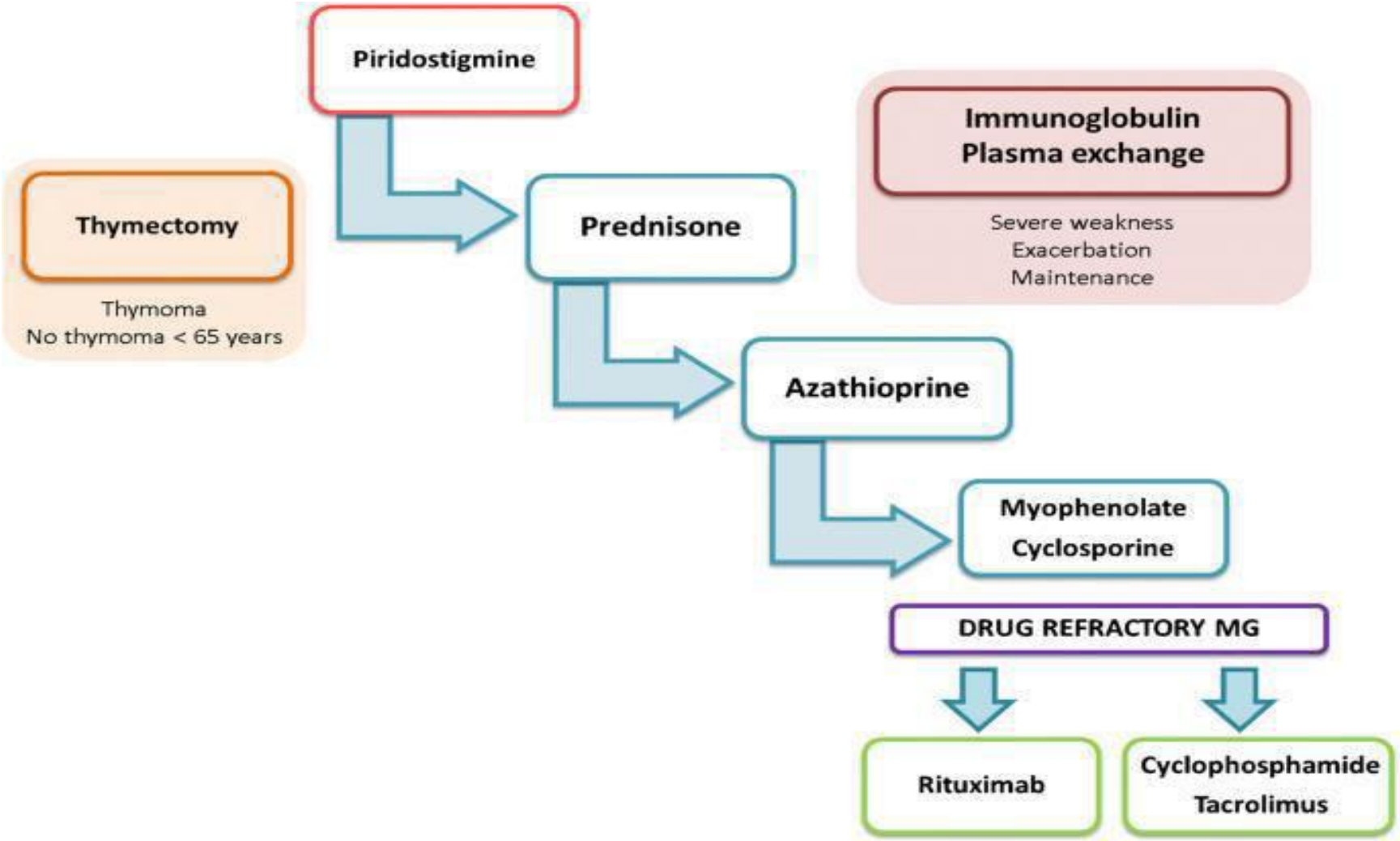
- Rare autoimmune disorder
- The immune system attacks channels that regulate calcium levels in the blood
- This causes insufficient acetylcholine to be released, leading to muscle weakness, fatigue, and other symptoms

Myasthenia gravis	Lambert Eaton syndrome
Antibody against AchR antibody	Antibody against voltage gated calcium channel
Associated with Thymic tumor	Associated with Small cell lung cancer
Weakness worsen on prolonged exercise	Weakness improves on prolonged exercise
Normal Deep tendon reflex	Decreased or absent deep tendon reflex
Autonomic dysfunction is absent	Autonomic dysfunction is present
On repeated nerve stimulation, there is decremental response	On repeated nerve stimulation, there is incremental response

Management plan

- Drug therapy
- Immunomodulation
- Surgical therapy
- Supportive therapy
- Life style modification

MANAGEMENT


Piridostigmine
Immunoglobulin
Plasma exchange
Severe weakness
Exacerbation
Maintenance
Thymectomy
Thymoma
No thymoma < 65 years
Prednisone
Azathioprine
Myophenolate
Cyclosporine
DRUG REFRACTORY MG
Rituximab
Cyclophosphamide
Tacrolimus

DRUGS USED IN MYASTHENIA GRAVIS

1) AChE inhibitors:

- Anticholinesterase inhibit Acetylcholinesterase (AchE), allowing the same Ach molecules to repeatedly interact with the available nicotinic receptors (NRs); frequency of Ach-NR interaction is increased.
- **Drugs:**
  1) Pyridostigmine bromide
  2) Prostigmine

2) Immunosuppressant medicines:

- They inhibit the immunity system, and limiting antibody production.
- **Drug:** Azothiaprine in addition to steroid medication (Prednisolone)

- **Pyridostigmine**
  Anticholinesterase with symptomatic relief

- **Rituximab (Rituxan) and eculizumab (Soliris)** are intravenous medications usually used for those who don't respond to other treatment

- **zilucoplan**, a peptide inhibitor of complement component 5 (C5 inhibitor), for the treatment of generalized myasthenia gravis in adult patients who are acetylcholine receptor antibody positive

Effects of cholinergic drugs

- CNS – enhance cognitive functions such as arousal, attention, & memory encoding – treatment for Alzheimer's disease & dementia
- Eye – pupil constriction – for surgery & treatment of glaucoma
- GI – smooth muscle stimulant – for post-op abdominal distention or paralytic ileus
- GU – urinary bladder stimulant – for post-op or postpartum urinary retention
- Musculoskeletal (indirect acting cholinergic drugs) – improve muscle tone & strength – for myasthenia gravis

Drugs that can Exacerbate Myasthenia Gravis

www.openmed.co.in

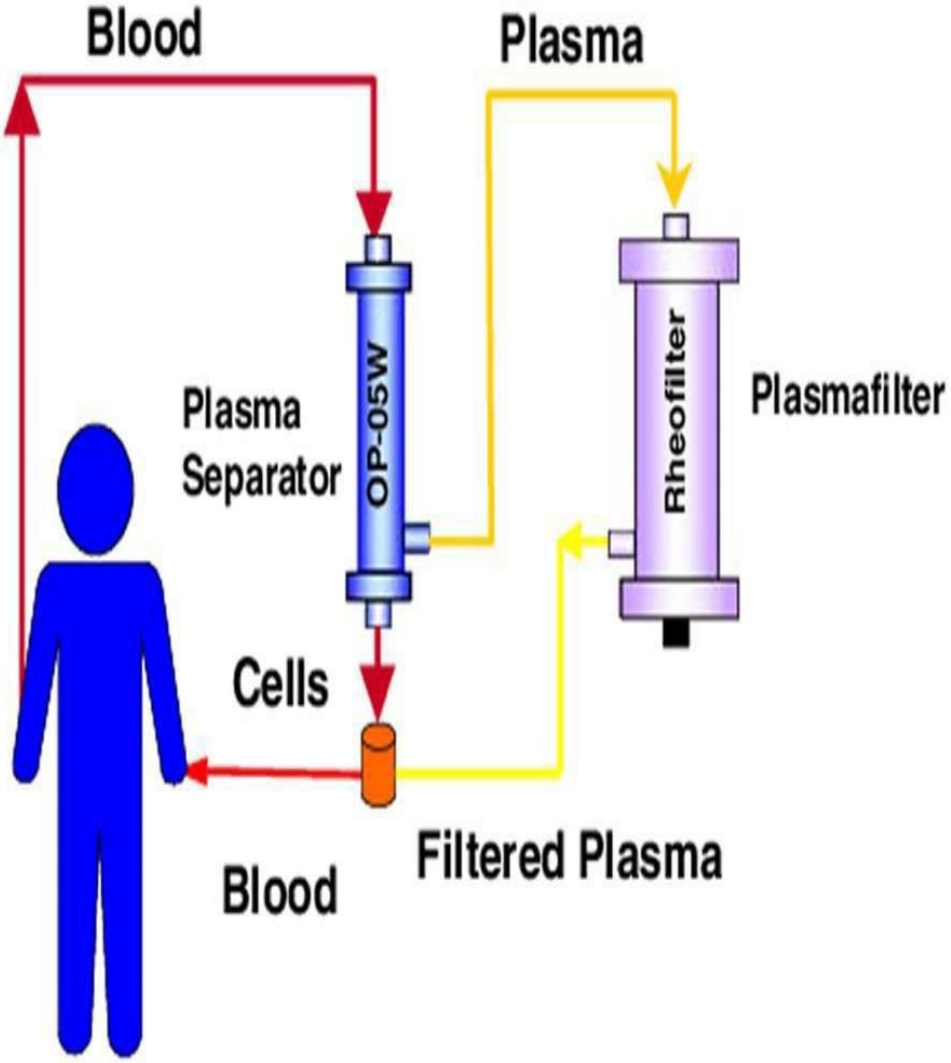
Mnemonic - *EXACERBATE*

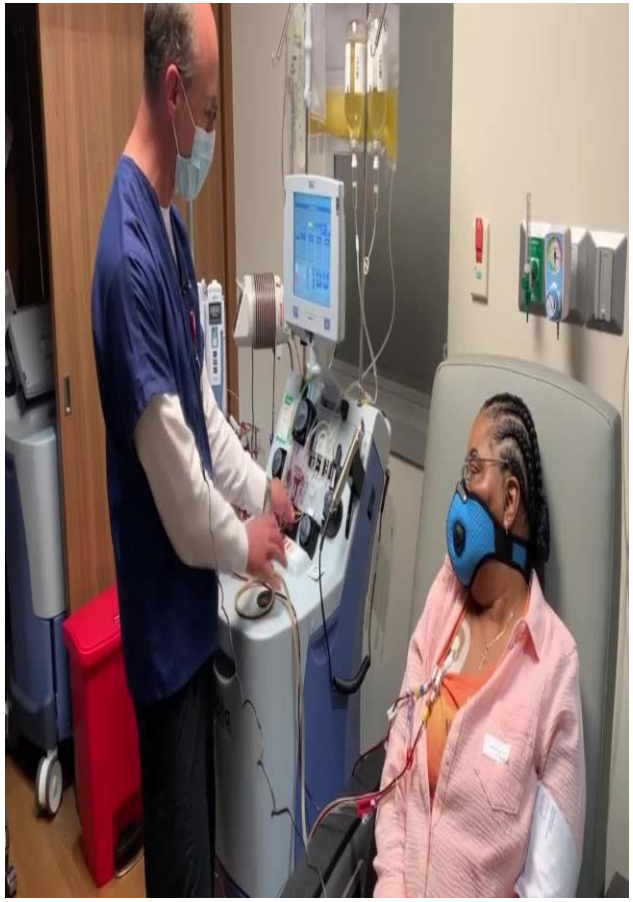
- **E**rythromycin (Macrolides)
- **X**ylocaine (Lignocaine)
- **A**minoglycosides
- **C**iproflox (Quinolones)
- **E**lectrolyte (Mg)
- **R**elaxant (Skeletal Muscle Relaxants)
- **B**otox & Beta Blocker
- **A**nti malarial (Quinine)
- **T**imolol (Eye Drops)
- **E**chothiophate (Eye Drops)

PLASMAPHERESIS

- ***A method of removing blood plasma from the body by withdrawing blood, separating it into plasma and cells, and transfusing the cells back into the bloodstream***

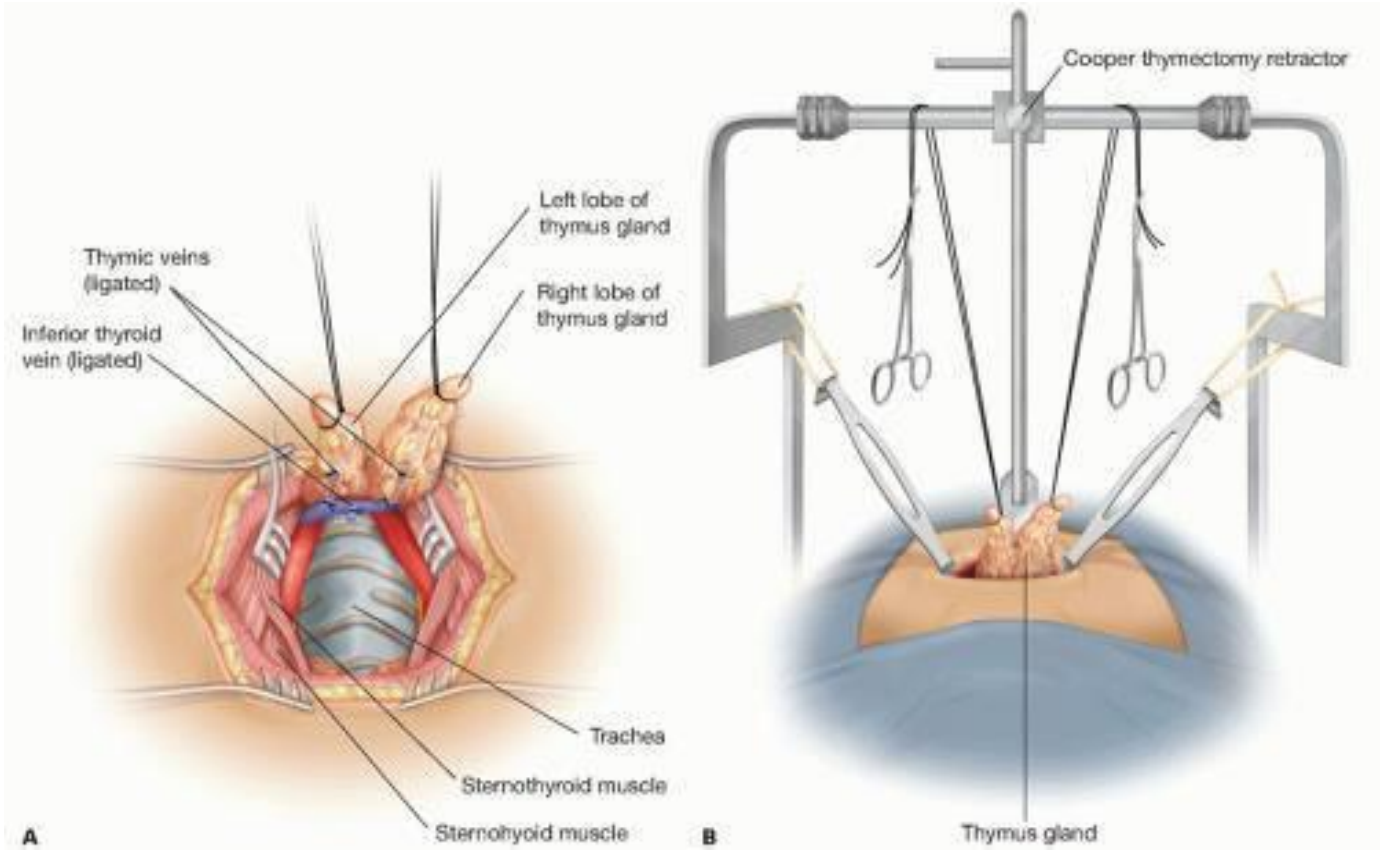
- ***It is performed especially to remove antibodies in treating autoimmune condition***


Blood
Plasma
Plasma
Separator
OP-05W
Rheofilter
Plasmafilter
Cells
Filtered Plasma
Blood

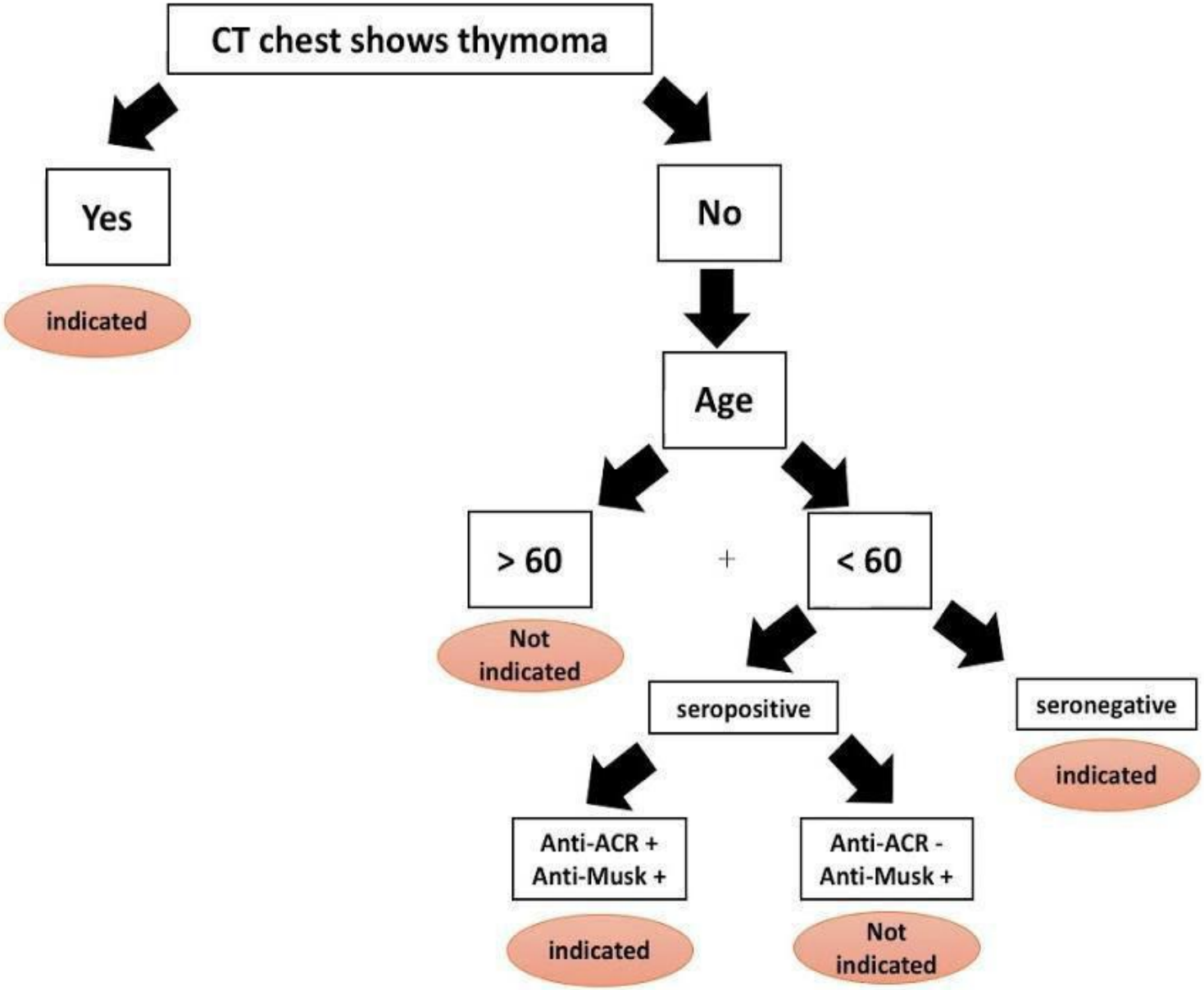


Thymectomy

- **The goal is to cause remission of the disease**
- **To allow dose reduction of harmful immunosuppressive medications**



Indications of thymectomy in Myasthenia Gravis



Life style modification

- Avoid physical exertion
- Take Plenty of Rest
- Avoid emotional stress
- Avoid exposure to extreme temperatures
- Continuous positive airway pressure therapy
- If diplopia bothers then occlusion
- Avoid medications such as muscle relaxants
- Avoid pneumonia/respiratory illness
- Avoid Low levels of potassium (diuretics and vomiting)


Myasthenia Gravis Diet Considerations
Aim to eat more small meals frequently rather than large meals
Make foods that are soft and easy to swallow or puree them
Tweak spices and temperature to boost appetite
Try out a liquid diet that includes shakes and smoothies
Incorporate thickening liquids to prevent them from being breathed into the lungs
verywell

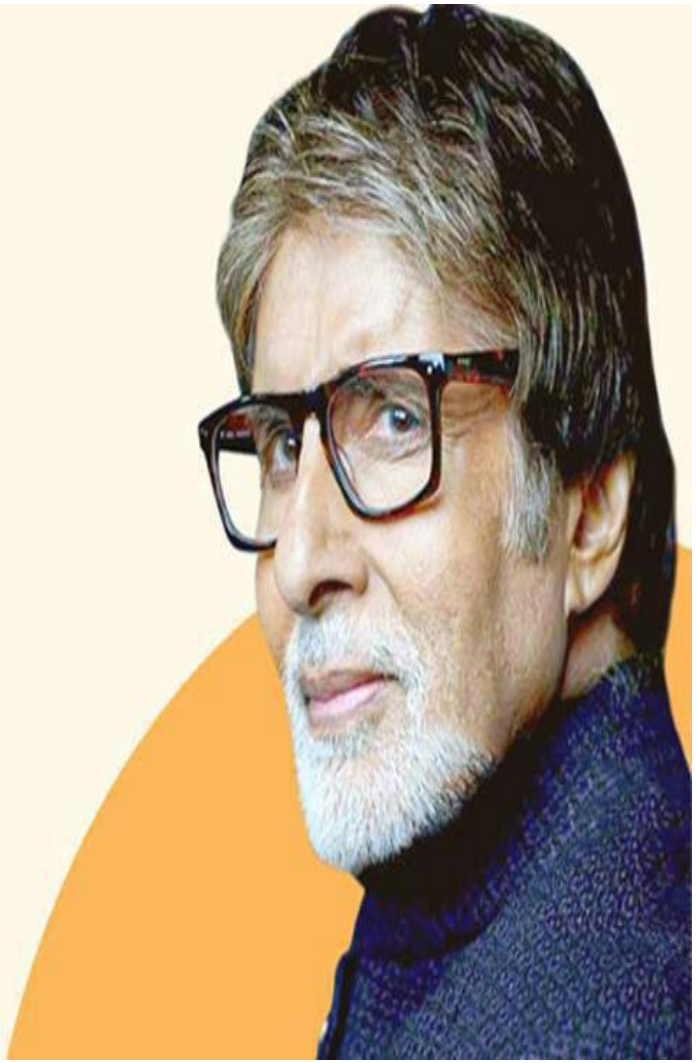
Do you know?

AMITABH BACHCHAN

is suffering from

MYASTHENIA GRAVIS

for the past 30 years!



June is....

Myasthenia Gravis Awareness Month

Many MG patients experience drastic changes in their physical appearance. For some, they may only experience changes in relation to symptoms of the MG (ex. dropping eyelids); however, others may experience changes as a side effect of medications and treatments for controlling the MG (ex. Prednisone leading to weight gain or 'moon face')

MIGRAINE

Prof SofiaIqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

Presentation lay out

- *Introduction*
- *Definition*
- *Migraine triggers*
- *Phases*
- *Classification*
- *Pathophysiology*
- *Differential diagnosis*
- *Diagnosis*
- *Goals for treatment*
- *Management*
- *Summary of prevention*
- *Conclusion and References*

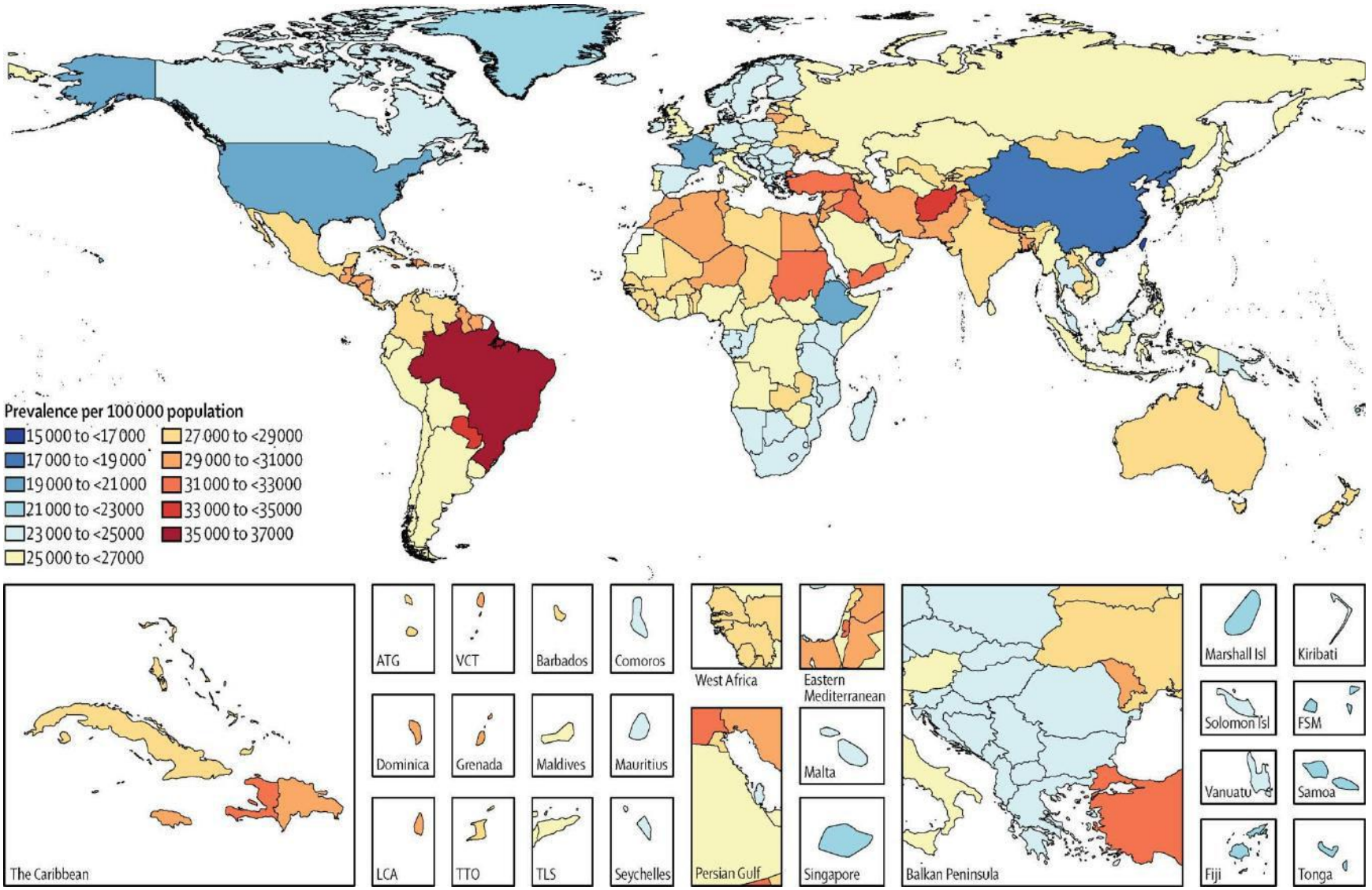
INTRODUCTION

- One of the common causes of recurrent headaches
- Constitutes 16%of primary headaches
- Migraine affects 10-20% of the general population
- It is often under diagnosed and under treated

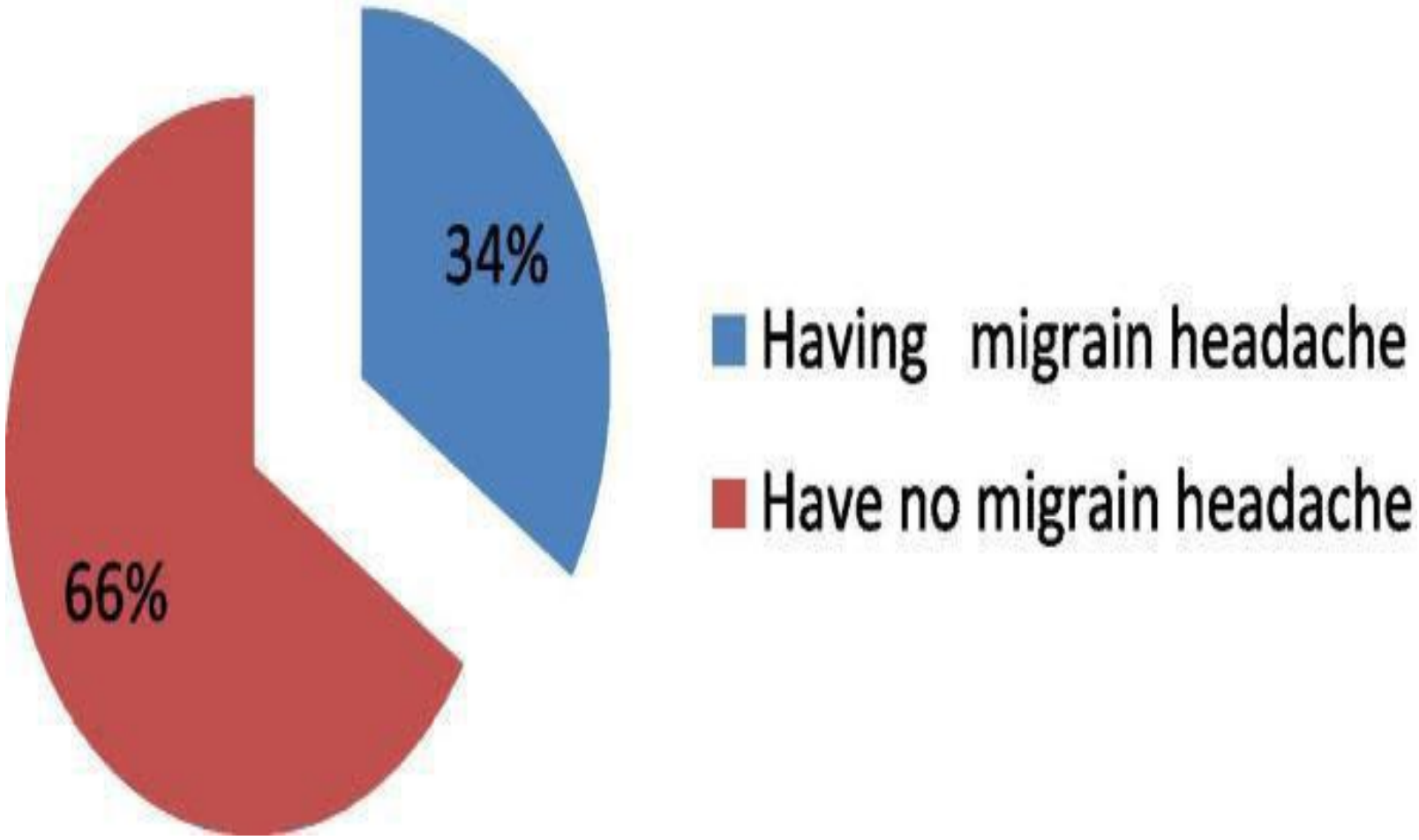
World Wide Burden

- ***It affects 18% of women and 6% of men in the US and has a worldwide prevalence of about 10%***
- ***For both men and women the prevalence rises throughout early adult life and falls after mid life***
- ***In females the rate almost triples between the age 10 and 30 years***

Global, regional and National burden



The prevalence of migrain headache



DEFINITION

- ***Originated from Greek word hemicrania meaning one side of the head***

- ***It is an episodic neurovascular phenomenon***

"Migraine is a familial disorder characterized by recurrent attacks of headache widely variable in intensity, frequency and duration. Attacks are commonly unilateral and are usually associated with anorexia nausea and vomiting"

ORIGIN OF PAIN IN THE HEAD

- Extra-cranial pain sensitive structures:
  - Sinuses
  - Eyes/orbits
  - Ears
  - Teeth
  - TMJ
  - Blood vessels
  - 5,7,9,10 cranial nerves carry pain from thes strucure
- Intra-cranial pain sensitive structures:
  - Arteries of circle of willis and proximal dural arteries,
  - Dural Venous sinuses,veins
  - Meninges
  - Dura

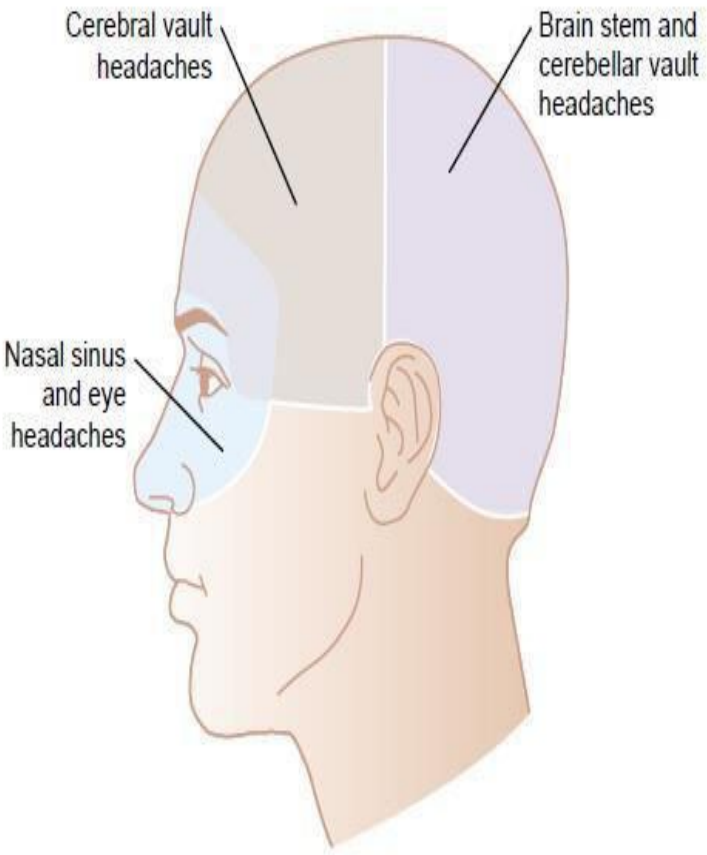


Figure 48–9

Areas of headache resulting from different causes.

Is it a migraine or type of headache?

	Migraine	Tension Headache	Sinus Headache	Cluster Headache
Location of pain	1 or both sides of head	1 or both sides of head or neck	Face, forehead, between eyes	1 side, extending from behind eye
Duration	4–72 hours	2 hours to days	Days, if untreated	30-90 minutes
Intensity	Mild, moderate or severe	Mild or moderate	Mild to severe	Severe
Treatment options	OTC medicines	Prescription from doctor	Decongestants or antibiotics	Oxygen and triptans

Primary Headache Types

	Migraine	Tension	Cluster
Pain Description	Throbbing, moderate to severe, worse w/exertion	Pressure, tightness, waxes and wanes	Abrupt onset, deep, continuous, excruciating, explosive
Associated Symptoms	Photo/phono-phobia, n/v, aura	None	Tearing, congestion, rhinorrhea, pallor, sweating

	Migraine	Tension	Cluster
Location	60-70% unilateral	Bilateral	Unilateral
Duration	4-72 hr	Variable	0.5-3 hr, many per day
Patient Appearance	Resting in quiet dark room; young female	Remains active or prefers to rest	Remains active, prefers hot shower, male, smoker

MIGRAINE TRIGGERS

- *Disturbed sleep pattern*
- *Hormonal changes*
- *Physical exertion*
- *Drugs (birth controls and vasodilators)*
- *Visual stimuli*
- *Auditory stimuli*
- *Olfactory stimuli*
- *Hunger*
- *Specific foods (alcohol and caffeine)*
- *Weather changes*
- *Psychological factors*

The role of foods and supplements in migraine

- **Skipped meals and fasting** were reported migraine triggers in over **56%** in a population-based study and **40% to 57%** in subspeciality clinic-based studies
- The mechanism by which fasting and skipping meals triggers headaches may be related to **alterations in serotonin and norepinephrine in brainstem pathways or the release of stress hormones such as cortisol.**

Clin J Pain 2009;25:446–452

External triggers

Dietary

- Caffeinated beverages
- Alcoholic beverages
- Aged cheeses
- Chocolate
- Coffee, tea, cola
- Chocolate
- Food allergens (Dairy products, yogurt)
- Ice cream

Chemical

- Monosodium glutamate
- Tyramine
- Nitrates
- Artificial sweetener (Aspartame)

Environmental

- Bright light/visual stimuli
- Odors/smells
- Weather changes
- Cigarette smoke

Behavioral

- Stress/tension
- Hunger/not eating
- Emotions
- Lack of sleep and Sleeping late/excess
- Fatigue/tiredness
- Exercise
- Hair wash or head bath

Minor head trauma

feels like CHOCOLATE

(causes of migraine)

Cheese ☑

OCP ☑

Caffeine ☑ the ones I do...

Alcohol ☑ ... ah shit.

Anxiety ☑

Travel ☑

Exercise ☑

Internal triggers

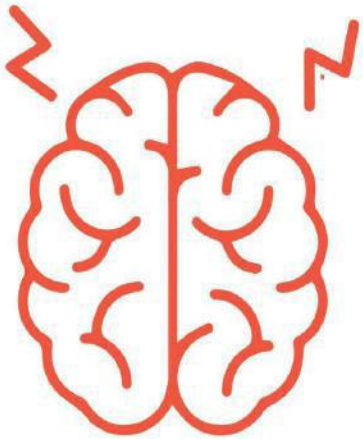
- The most common internal triggers are **sex hormones (neurosteroids and ovarian steroids).**
- The key stages of reproduction including **first menstruation, pregnancy and menopause** are associated with frequency or severity of migraine.
- Interestingly only attacks of migraine without aura occur during the perimenstrual time period and attacks of migraine with aura happen equally during the menstrual cycle.

- **P** Pulsatile
- **O** hOurs (duration of 4-72 hours)
- **U** Unilateral
- **N** Nauseating
- **D** Disabling

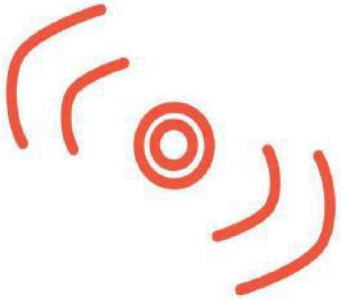
knowmedge
MEDICAL MNEMONICS

Mnemonic: "POUNDing Headache"

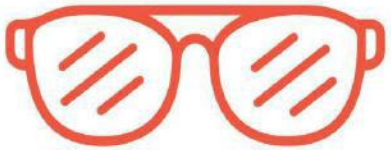
COMMON MIGRAINE SYMPTOMS



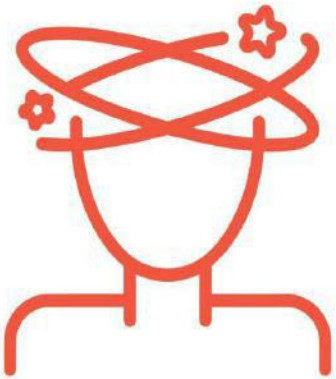
PAIN ON ONE OR BOTH SIDES OF HEAD



THROBBING OR PULSING



SENSITIVITY TO LIGHT, SOUND, OR MOVEMENT

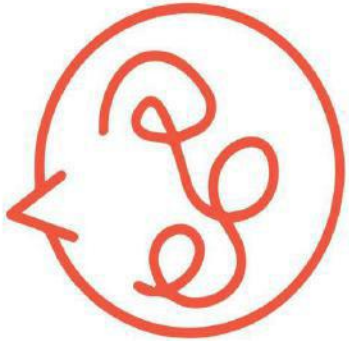


NAUSEA AND VOMITING

TEMPORARY LOSS OF VISION

ISOLATED WEAKNESS OR NUMBNESS

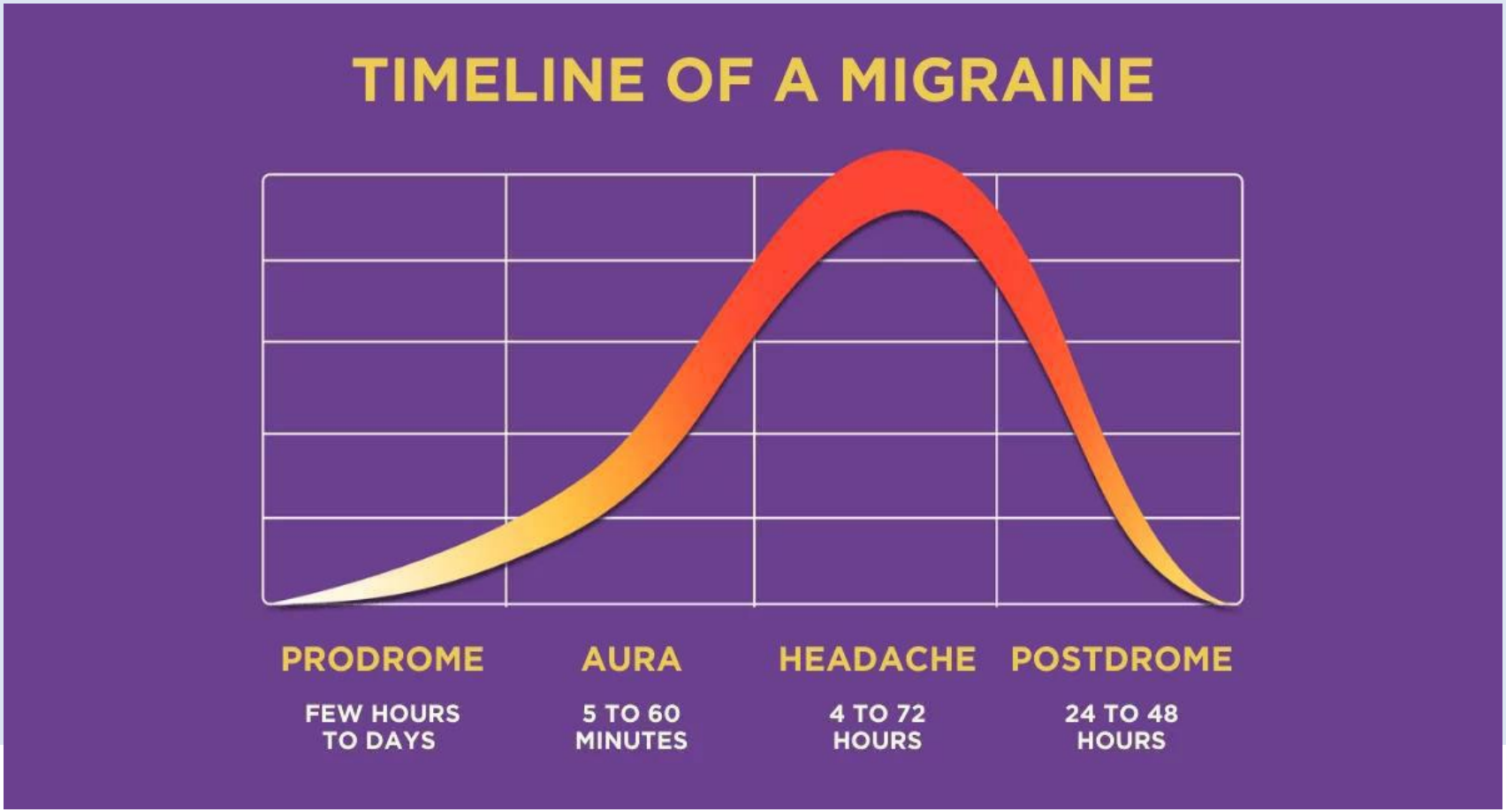
"PINS AND NEEDLES" IN EXTREMITIES



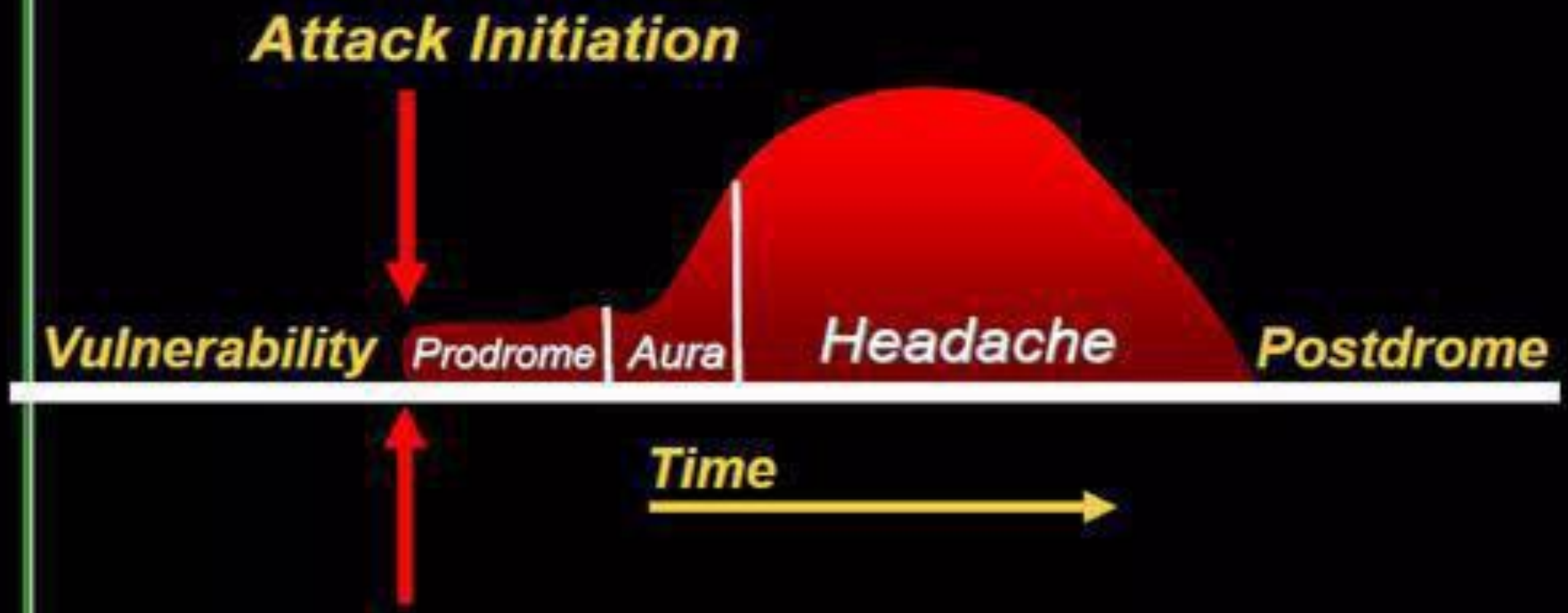
TEMPORARY TROUBLE WITH SPEECH

PHASES

- **Prodrome, Aura, Headache and Postdrome**



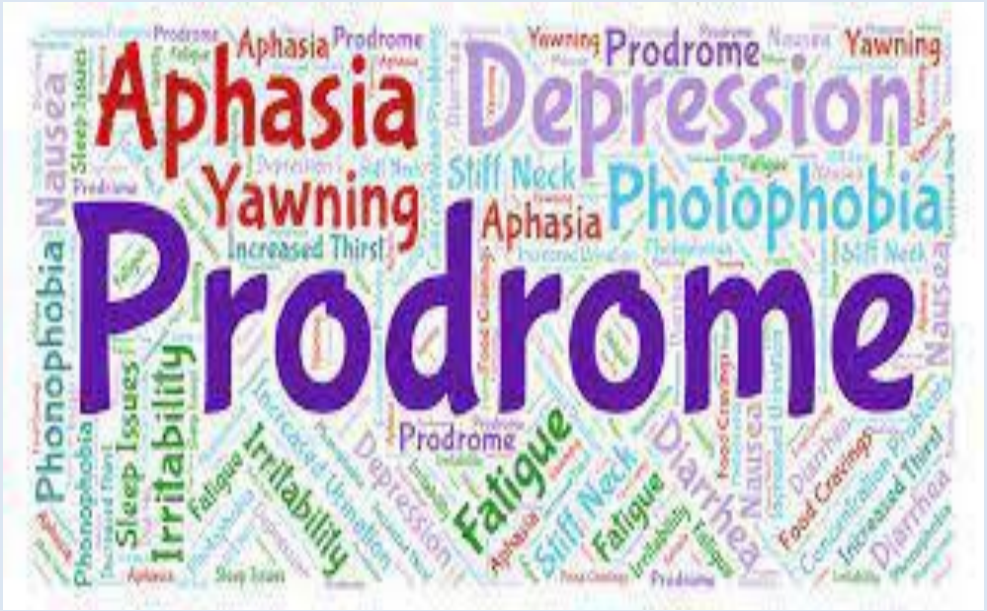
CLINICAL PHASES OF A MIGRAINE ATTACK



Blau JN. *Lancet*. 1992

PRODROME

- ***Vague premonitory symptoms that begin from 12 to 36 hours before aura and headache***



Symptoms

- Yawning
- Excitation
- Depression
- Lethargy
- Craving or distaste for various foods

AURA

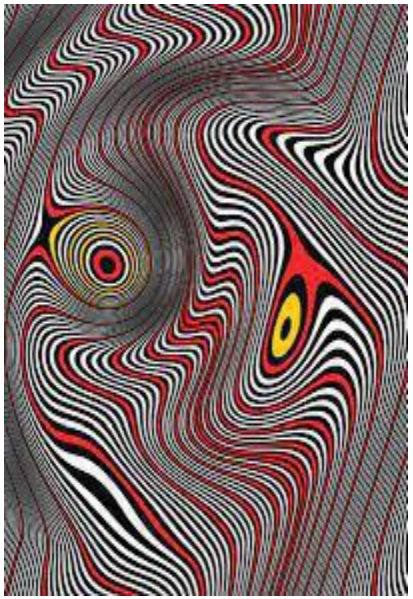
- **A warning or signal before the onset of headache**

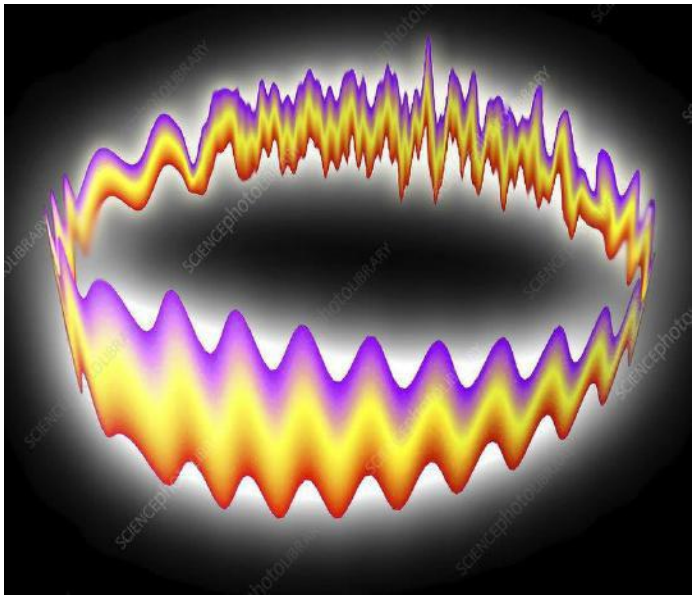
Symptoms

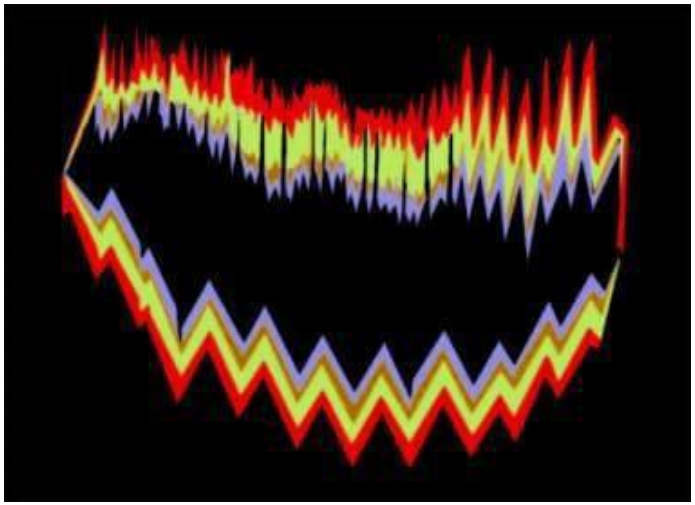
- **Flashing of lights**
- **Zig zag lines**
- **Difficulty in focusing**

Duration 15-30 min

Visual aura



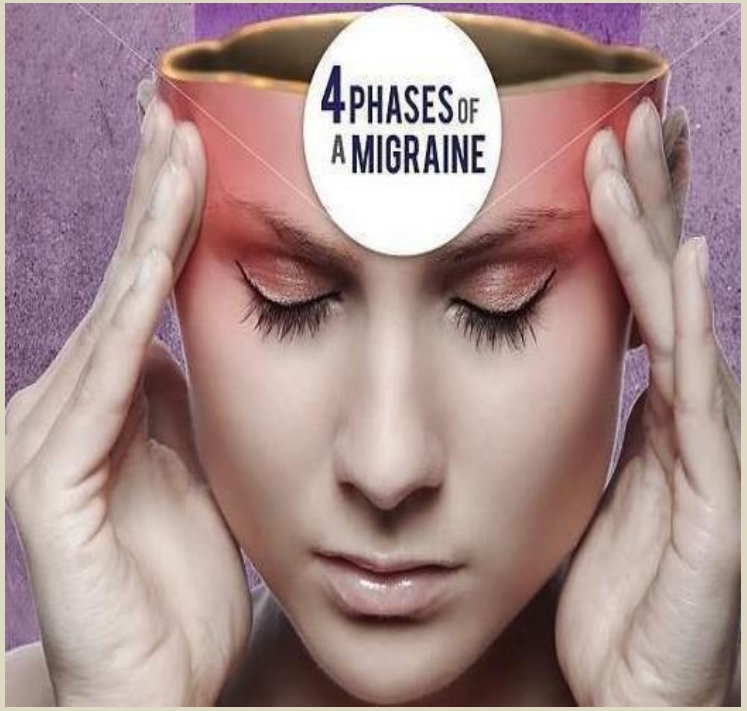




HEADACHE

- Headache is generally unilateral and is associated with ***SYMPTOMS*** like:

- **Anorexia**
- **Nausea**
- **Vomiting**
- **Photophobia**
- **Phonophobia**
- **Tinnitus**



Duration : 4-72hrs

POSTDROME

- **Following headache, patient complains of**

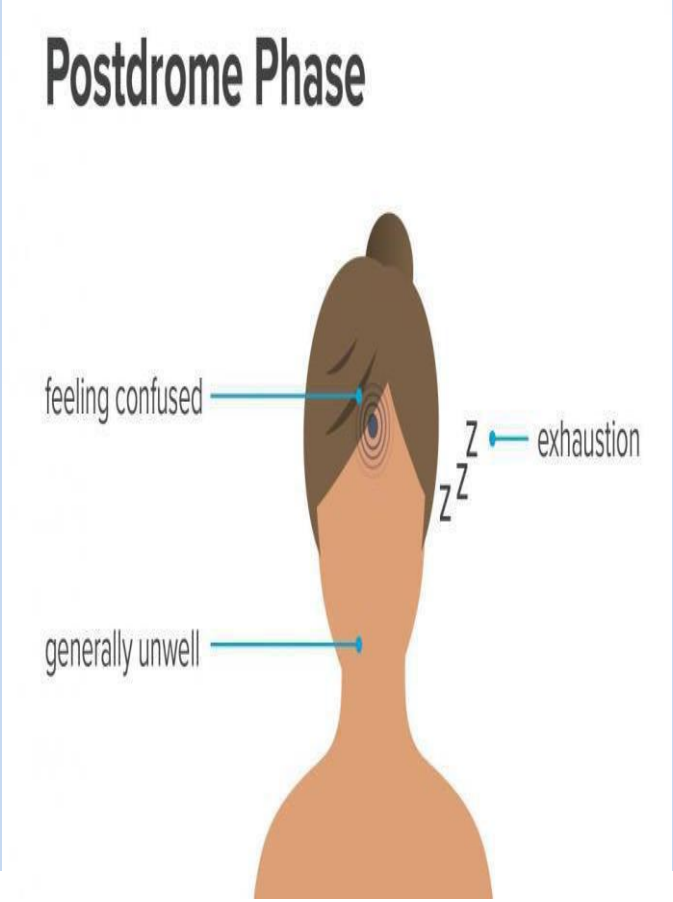
Fatigue

Depression

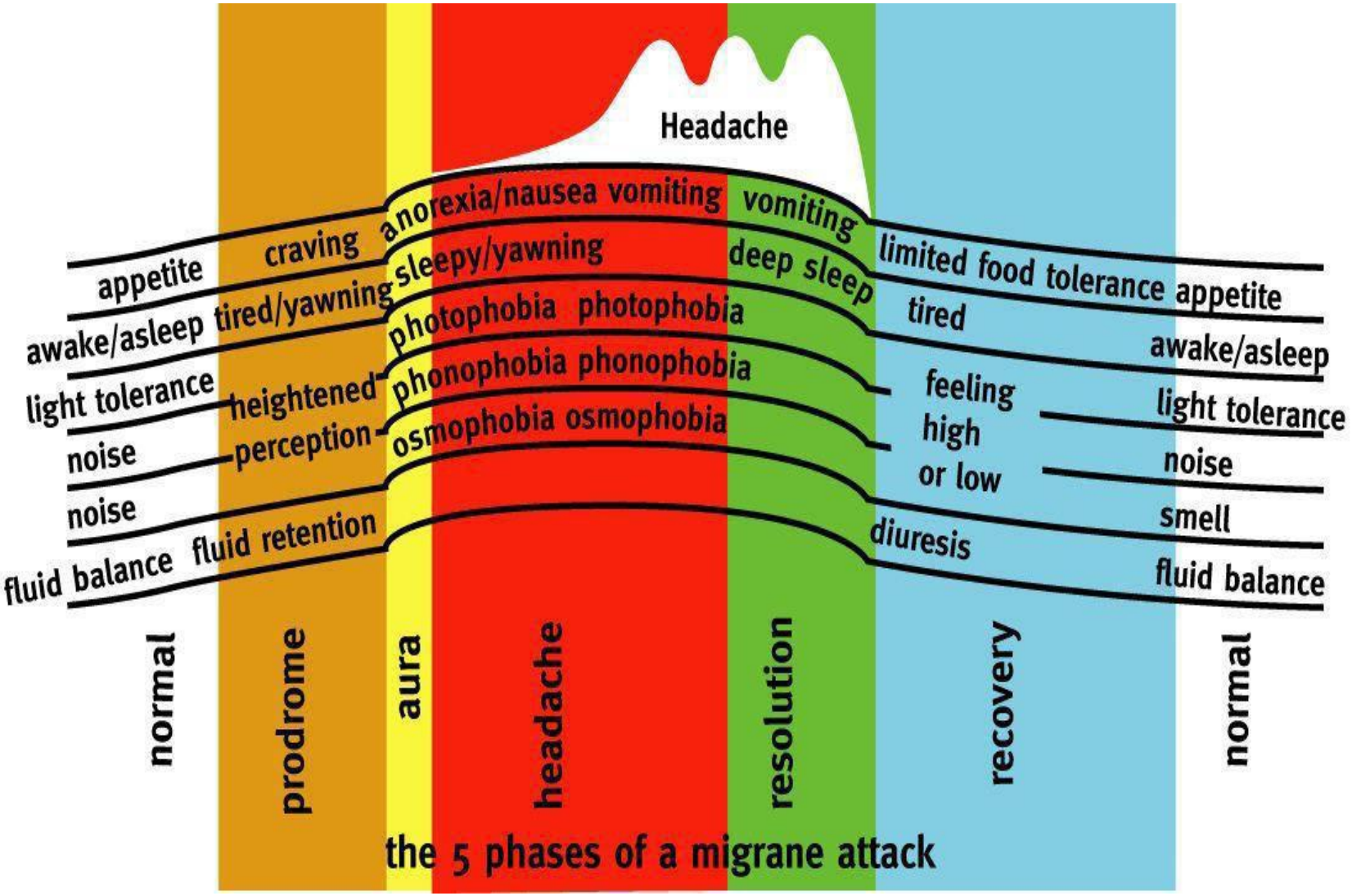
Severe exhaustion

Some patients feel unusually fresh

***Duration*: few hours or up to 2 days**



Summary of phases



CLASSIFICATION

- **According to Headache classification committee of the international Headache society migraine has been classified as:**

- **Common migraine** (without aura)
- **Classic migraine** (with aura)
- **Complicated migraine**

Types of Migraines

Migraine without aura

Headache that comes in stages

May experience nausea, fatigue, irritability, and sensitivity to light and sound

Typically on a localized spot on one side of the head

Migraine with aura

Usually causes visual disturbances for about 30 minutes prior to initial migraine symptoms

May affect speech, taste, or smell

- **Mild** (1/month, upto 8 hr)
- **Moderate** (> 1/ month, intense, 6-24 hr, nausea, vomiting associated)
- **Severe** (2-3/month, severe throbbing, 12-48 hr, vertigo, vomiting associated)

Classical migraine

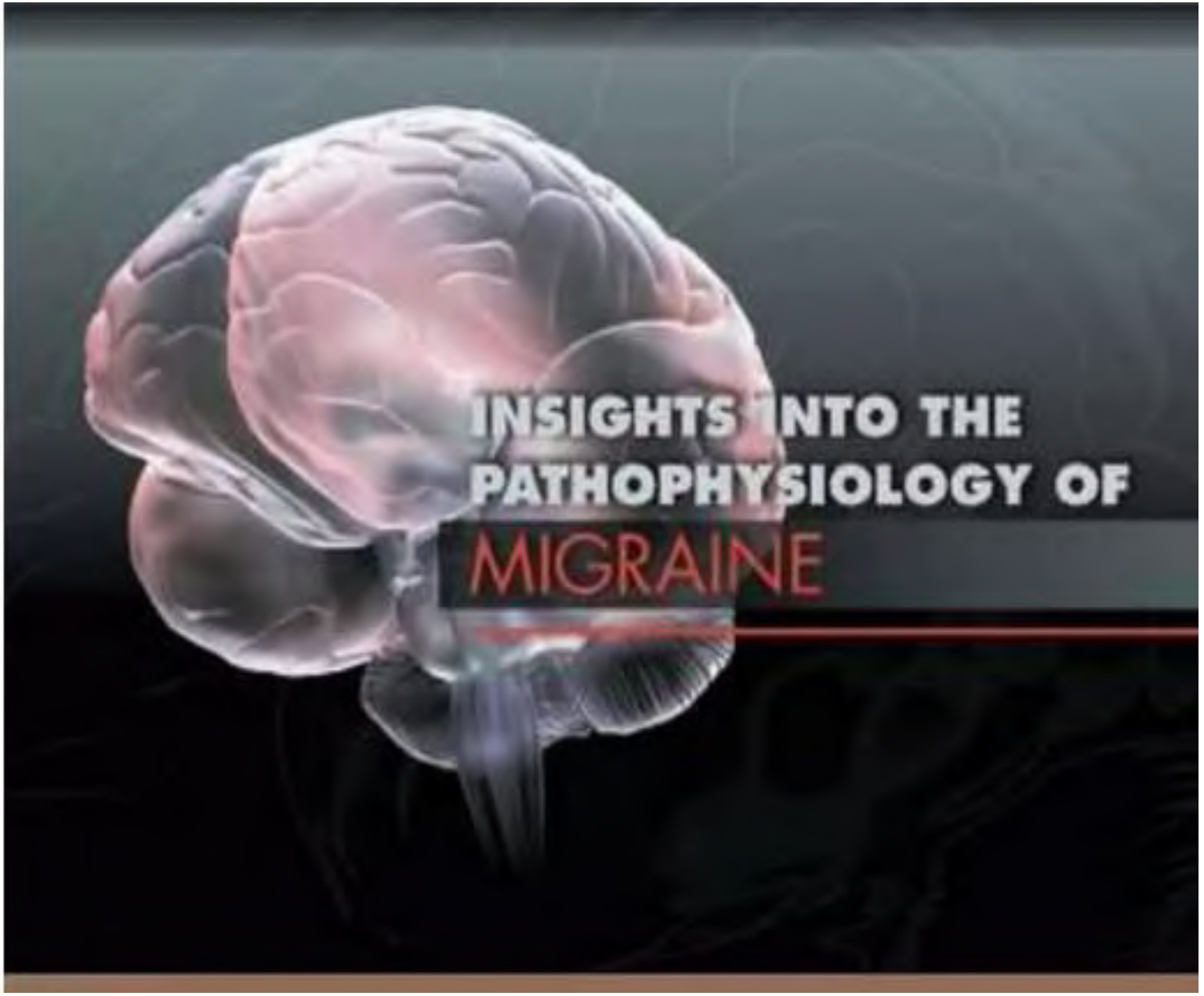
- Migraine with aura (ophthalmic, hemiplegic migraine) is defined as a recurrent disorder involving headache attacks appearing gradually over 5-20 minutes and lasting for less than 60 minutes.
- The aura encompasses focal neurological symptoms that precede or accompany at the onset of migraine attacks.
- **Aura can involve reversible visual and sensory symptoms and speech weakness.**

International Classification of Headache Disorders II. (2004). *Cephalalgia*, Vol.24, Sup.1, pp.1-160

Common migraine

- Migraine without aura (hemicrania simplex, common migraine) is a specific neurological disorder characterized by unilateral, pulsating quality, aggravation on movement, and moderate to severe headache, nausea and photophobia.
- **Most migraineurs suffer from this subtype of migraine, and there are higher frequency and more severe attacks in comparison with migraine with aura.**
- Owing to strong relationship between migraine without aura with menstrual cycle, the menstrual migraine (i.e. pure menstrual migraine and menstrually-related migraine) is categorized in this subtype.

International Classification of Headache Disorders II. (2004). *Cephalalgia*, Vol.24, Sup.1, pp.1-160


INSIGHTS INTO THE
PATHOPHYSIOLOGY OF
MIGRAINE

PATHOPHYSIOLOGY

- **VASCULAR THEORY**

Intracranial/Extracranial blood vessel vasodilation-headache

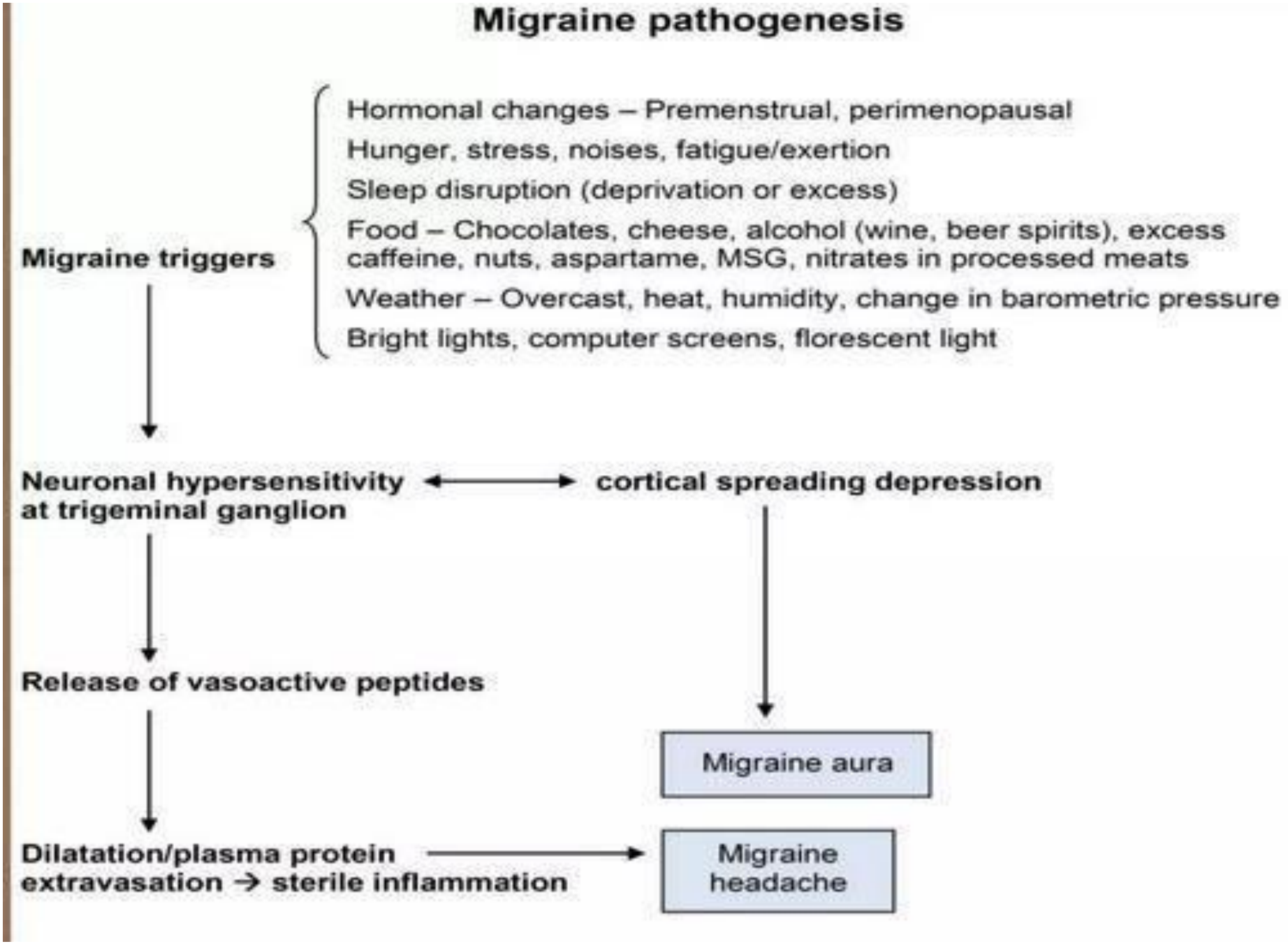
Intracerebral blood vessel vasoconstriction-aura

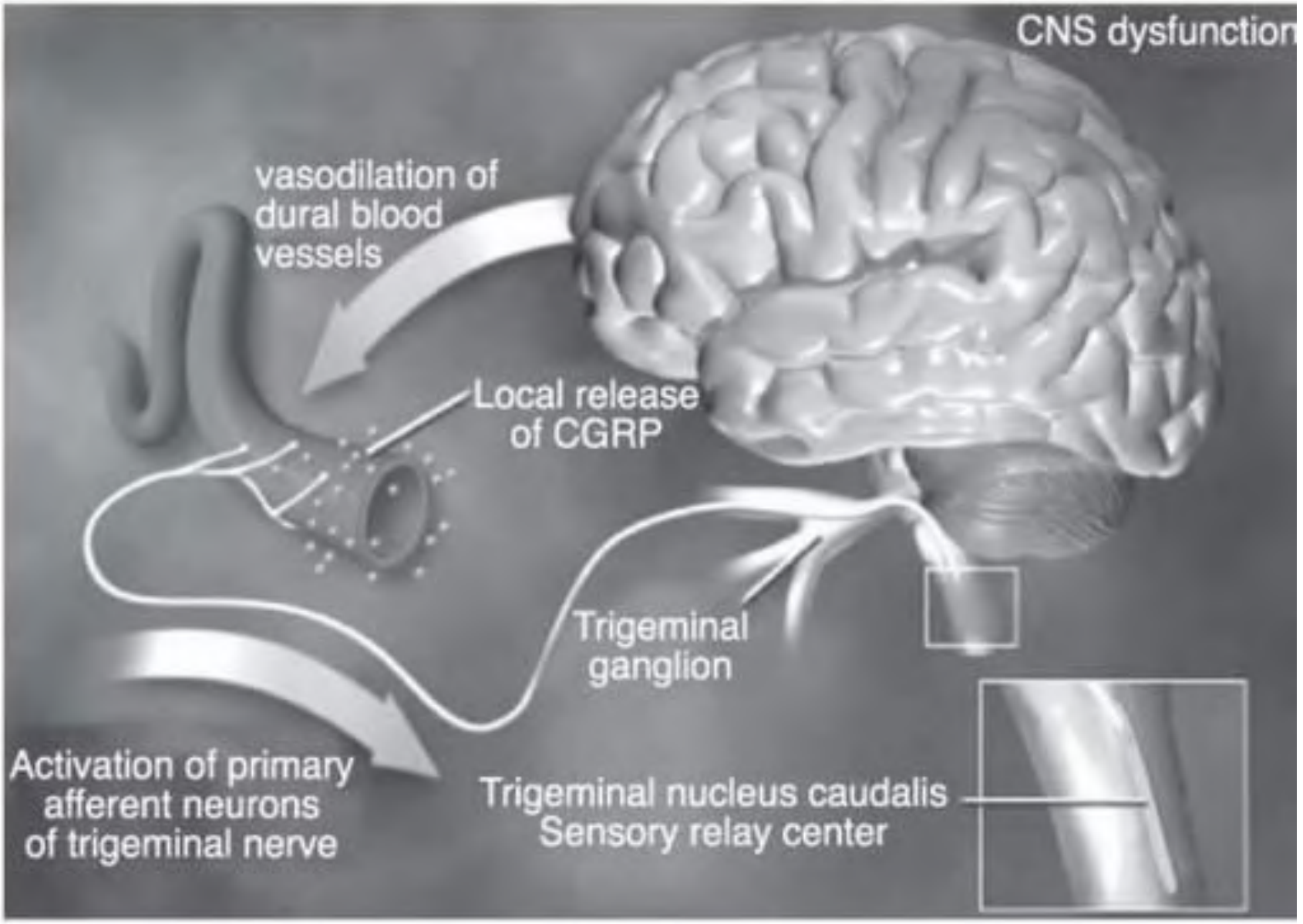
SEROTONIN THEORY

Decrease levels linked to migraine

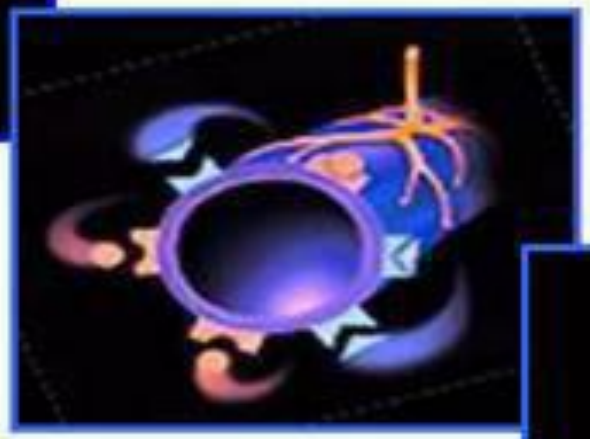
Specific serotonin receptors found in blood vessels of brain

Migraine pathogenesis




CNS dysfunction
vasodilation of dural blood vessels
Local release of CGRP
Trigeminal ganglion
Activation of primary afferent neurons of trigeminal nerve
Trigeminal nucleus caudalis Sensory relay center

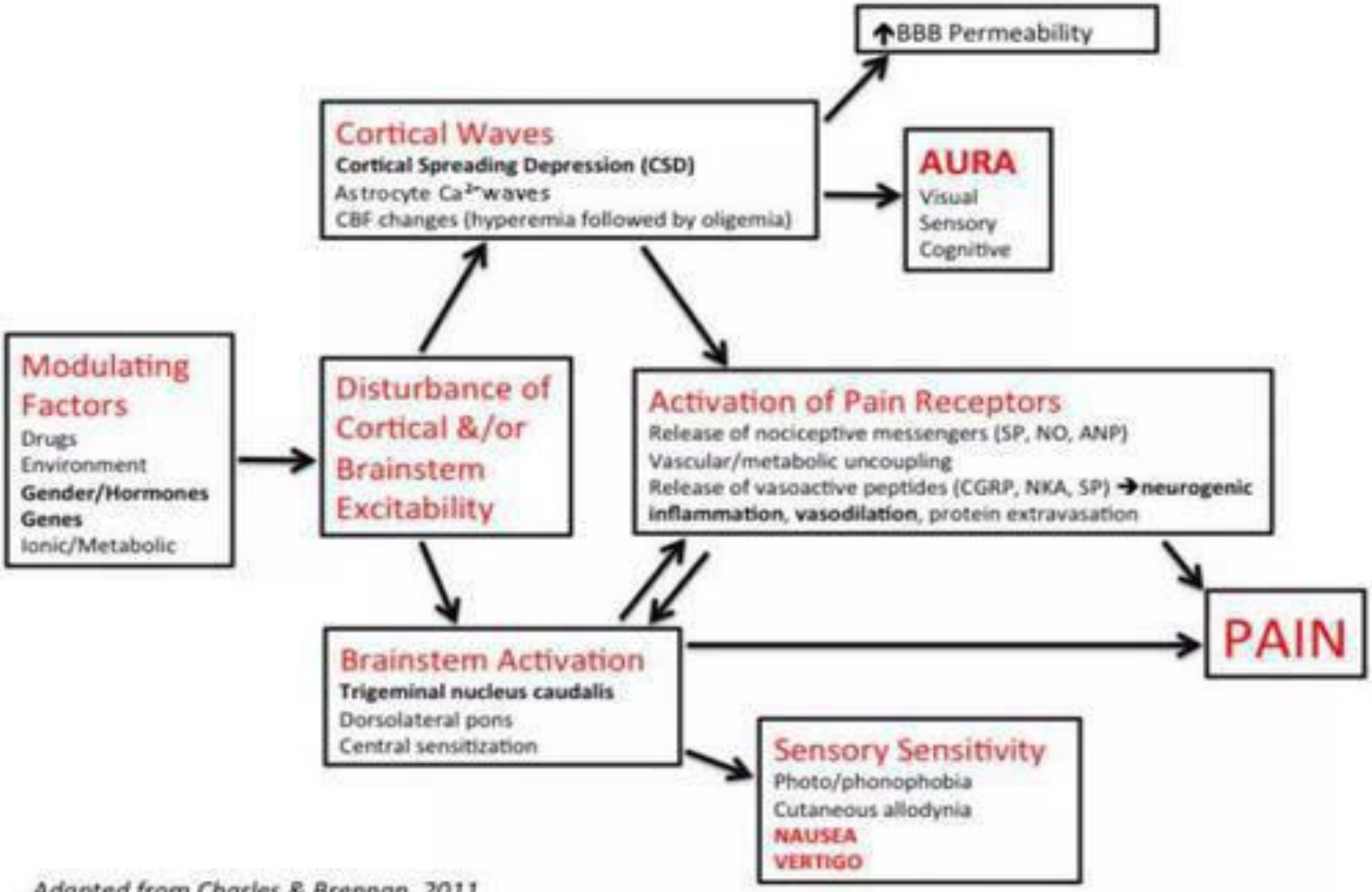
Release of
Neurotransmitter



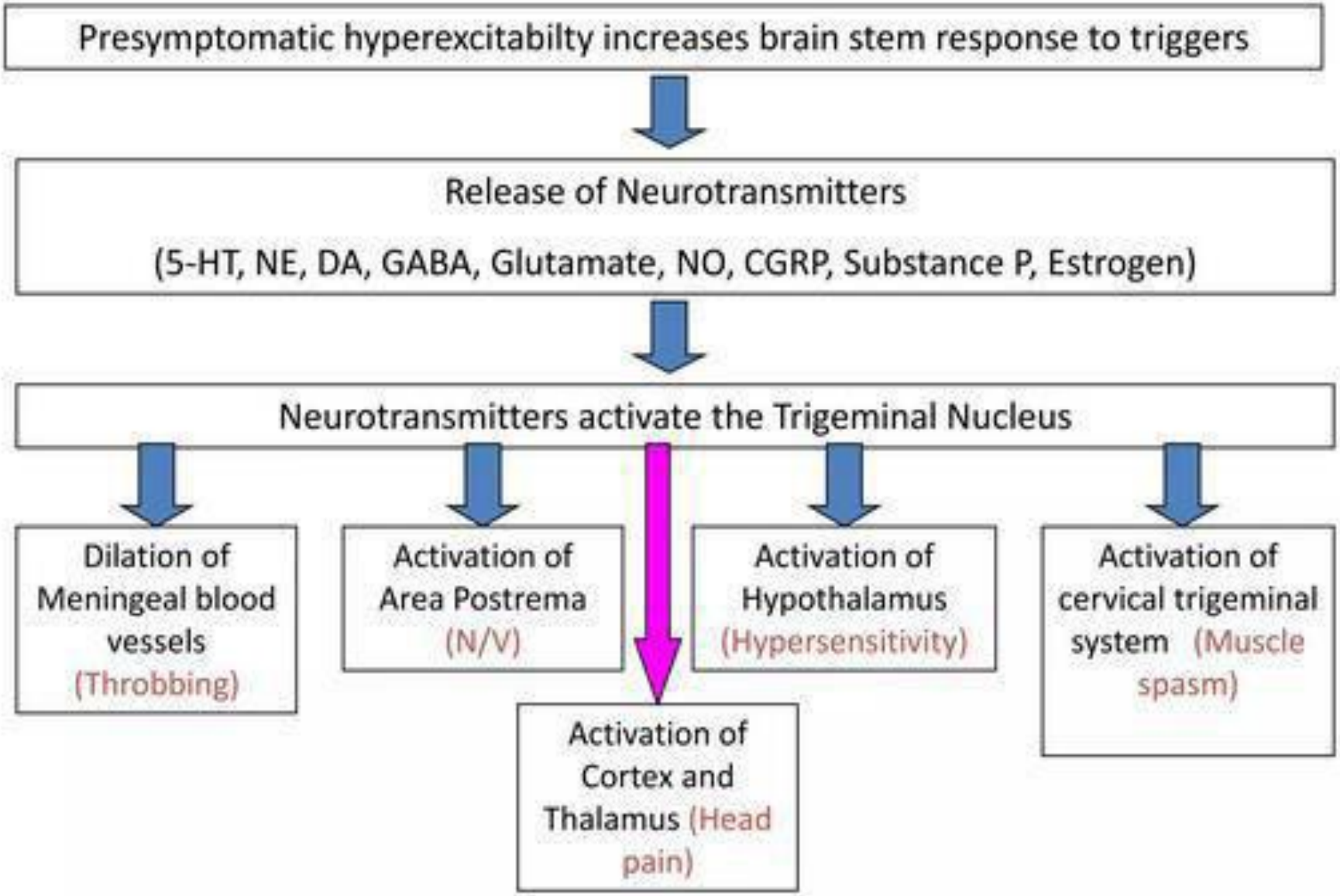
Arterial Activation

Worsening of Pain

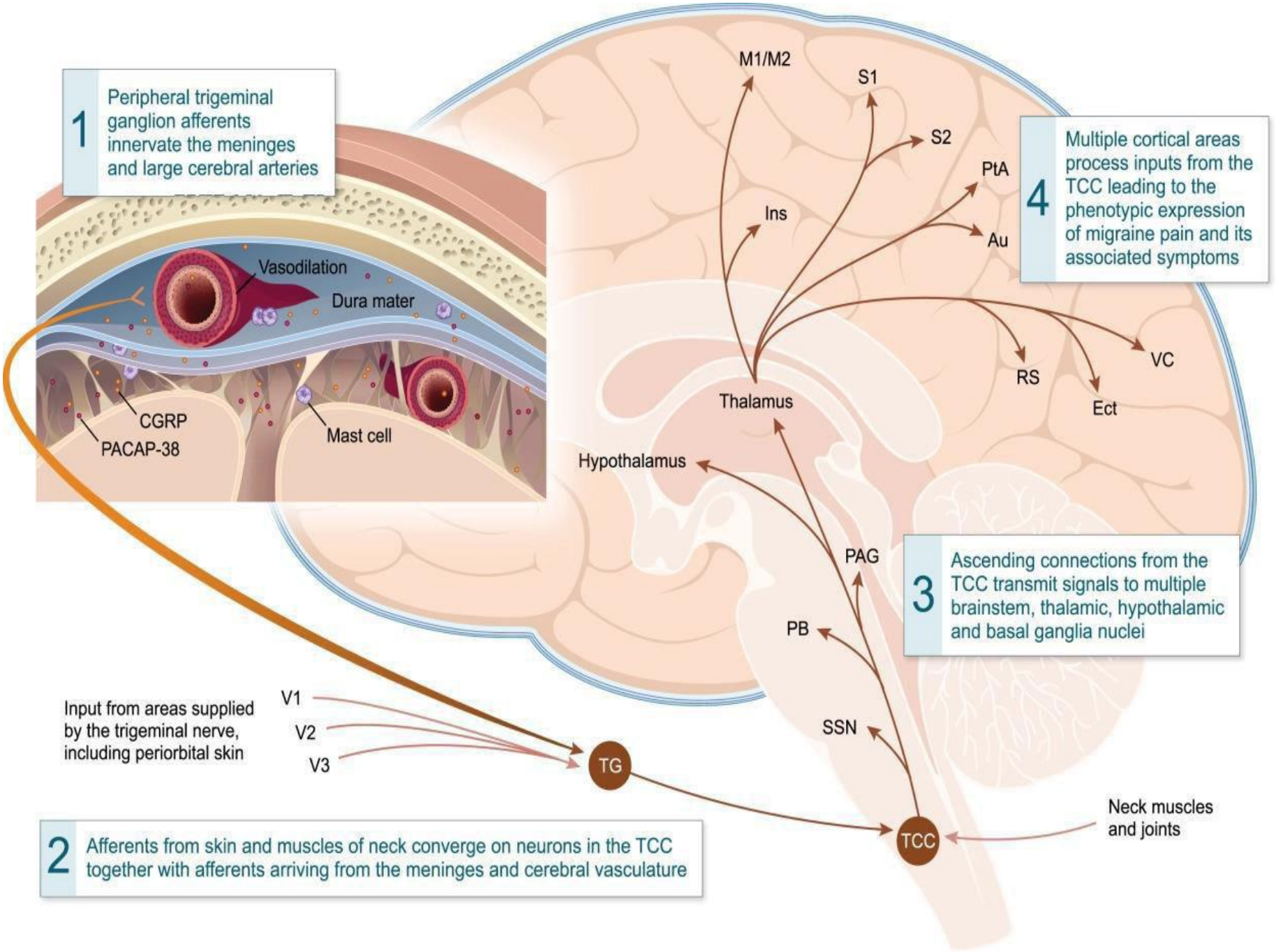
Hypothesized Sequence of Events in Migraine



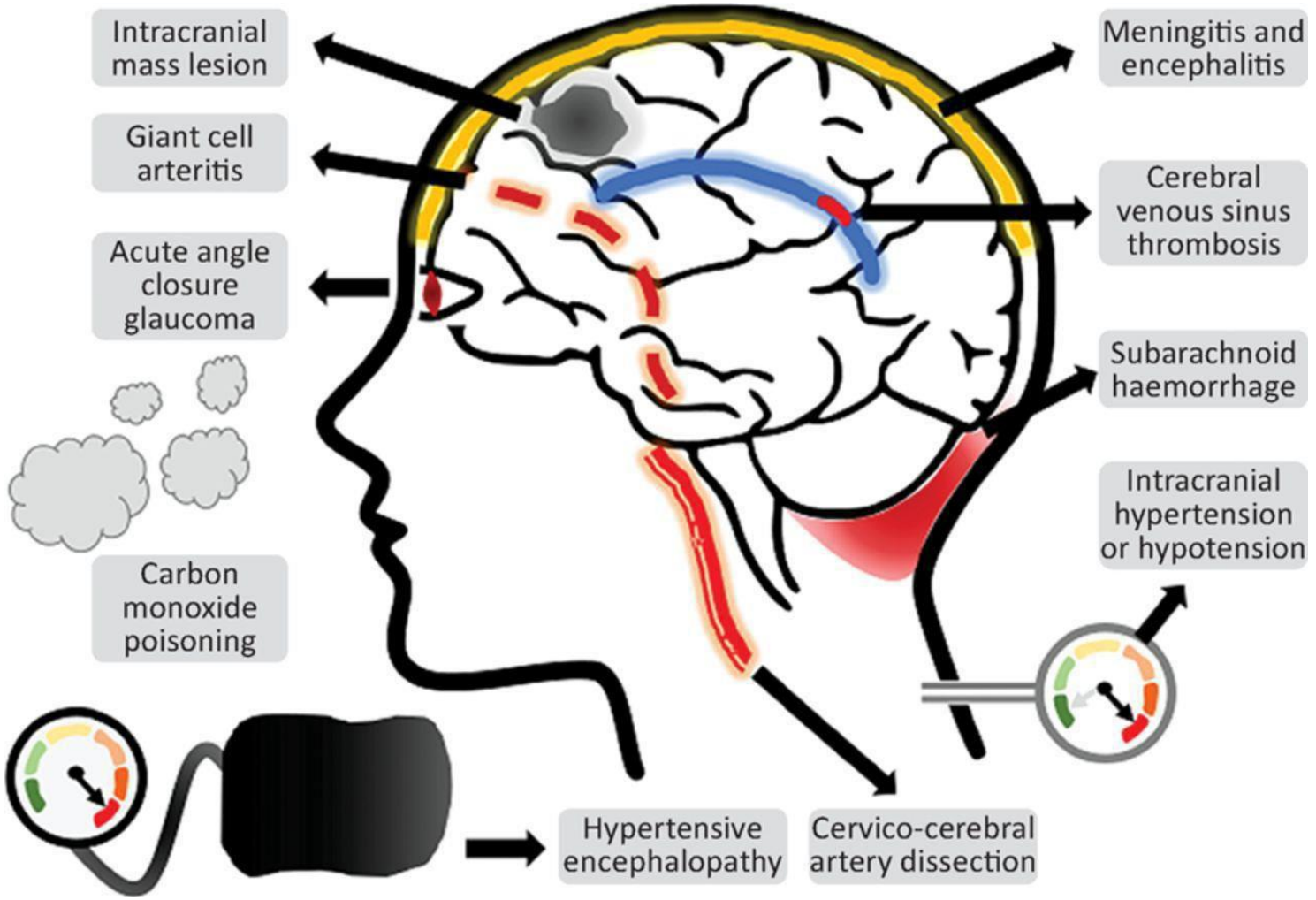
Adapted from Charles & Brennan, 2011



Marcus, DA. *Headache Simplified* 2008.


1
Peripheral trigeminal ganglion afferents innervate the meninges and large cerebral arteries
Vasodilation
Dura mater
CGRP
PACAP-38
Mast cell
M1/M2
S1
S2
PtA
Au
Ins
4
Multiple cortical areas process inputs from the TCC leading to the phenotypic expression of migraine pain and its associated symptoms
VC
RS
Ect
Thalamus
Hypothalamus
PAG
PB
SSN
3
Ascending connections from the TCC transmit signals to multiple brainstem, thalamic, hypothalamic and basal ganglia nuclei
Input from areas supplied by the trigeminal nerve, including periorbital skin
V1
V2
V3
TG
TCC
Neck muscles and joints
2
Afferents from skin and muscles of neck converge on neurons in the TCC together with afferents arriving from the meninges and cerebral vasculature

Differential Diagnosis



Sinus
pain is behind browbone and/or cheekbone.

Cluster
pain is in and around one eye.

Tension
pain is like a band squeezing the head.

Migraine
pain, nausea and visual changes are typical of classic form.

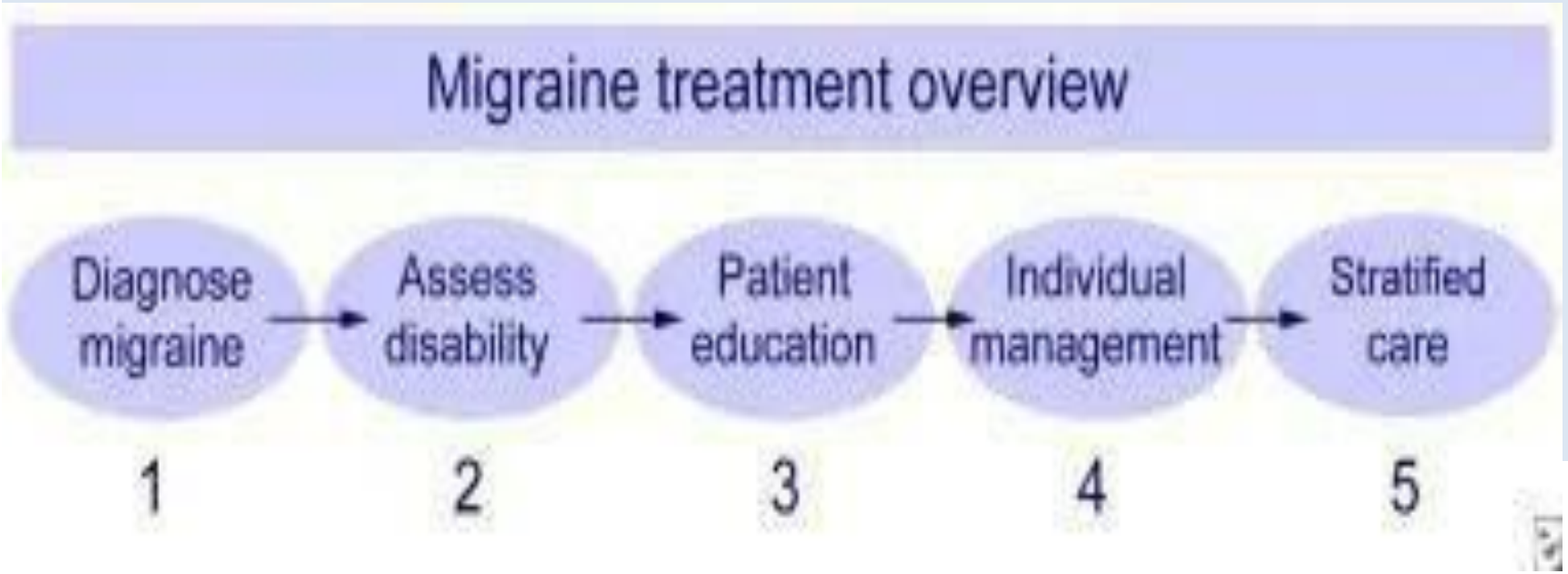
DIAGNOSIS

- **Medical history**
- **Headache history**
- **Migraine triggers**
- **Investigations**

  ECG/ CT Brain/MRI

Management of migraine

- **Not life threatening and not associated with serious illness but can make life miserable**
- **Inherited tendency of cerebral dysfunction and can not be cured completely**
- **Life style modification is important**



GOALS FOR TREATMENT

- Establish diagnosis
- Educate patient
- Discuss findings
- Establish reasonable expectations
- Involve patient in decision
- Encourage patient to avoid triggers
- Choose best treatment
- Create treatment plan

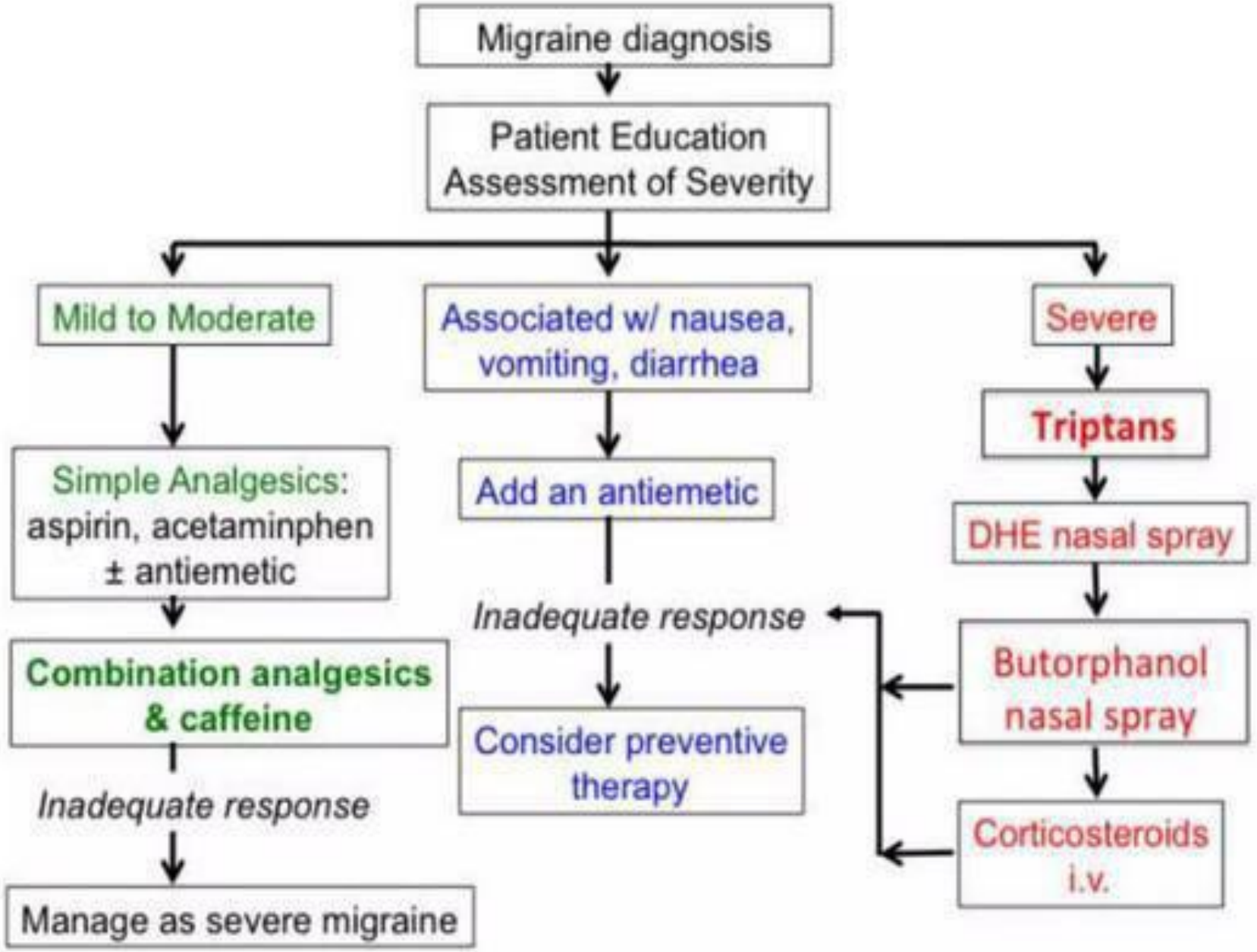
MANAGEMENT

Non-pharmacological treatment

- Triggers identification
- Meditation
- Relaxation training
- Psychotherapy

- **Pharmacotherapy**

- Abortive therapy
- Preventive therapy


Migraine diagnosis
Patient Education
Assessment of Severity
Mild to Moderate
Associated w/ nausea, vomiting, diarrhea
Severe
Simple Analgesics: aspirin, acetaminphen ± antiemetic
Add an antiemetic
Triptans
DHE nasal spray
Inadequate response
Combination analgesics & caffeine
Consider preventive therapy
Butorphanol nasal spray
Inadequate response
Corticosteroids i.v.
Manage as severe migraine
Adapted from Silberstein SD et al., 2000

ABORTIVE THERAPY

- **Non specific treatment**

DRUG	DOSE	ROUTE
Asprin	500-650mg	oral
paracetamol	500mg-4g	oral
Ibuprofen	200-300mg	oral
Diclofenac	50-100mg	Oral/IM
Naproxen	500-750mg	oral

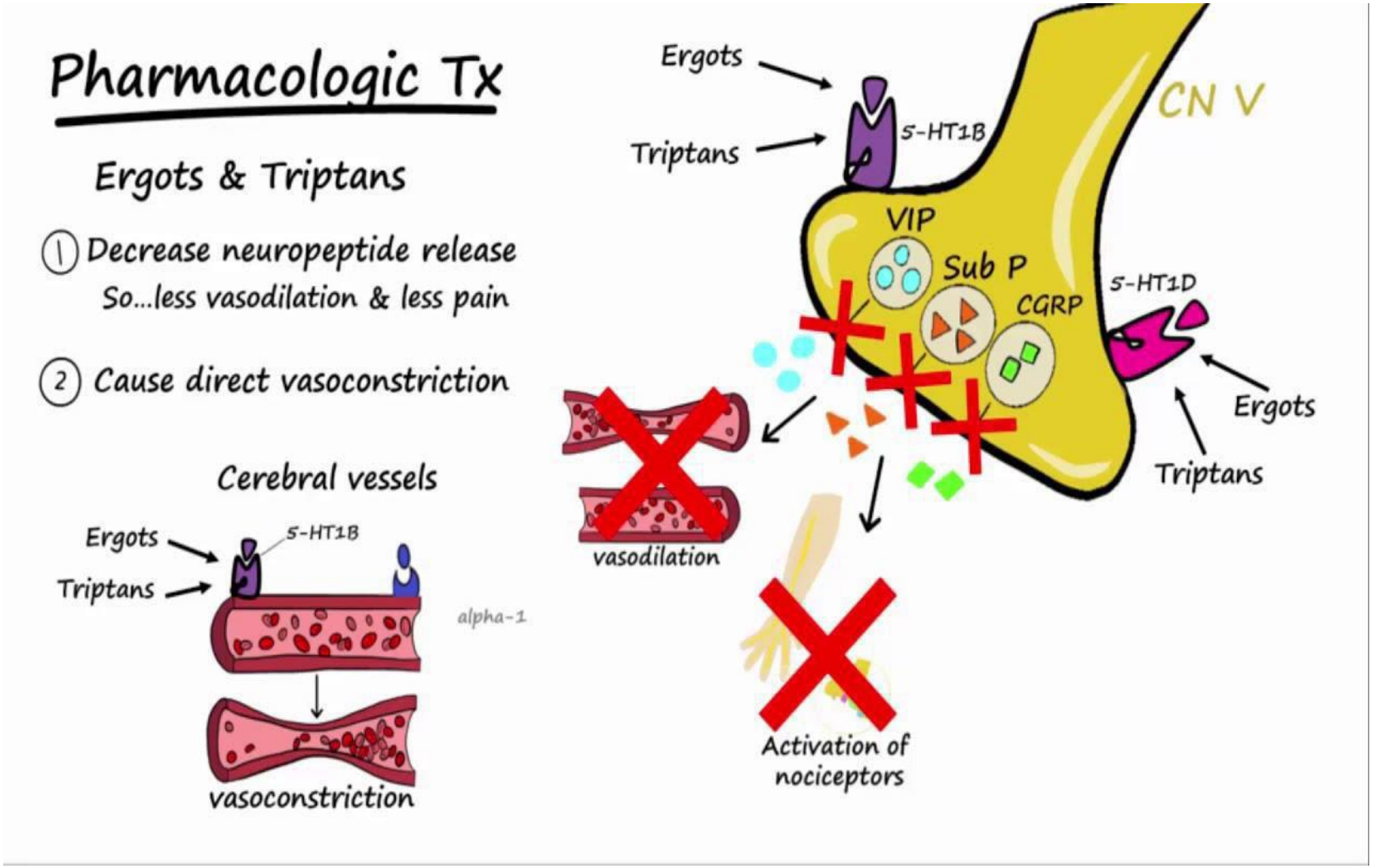
SPECIFIC THERAPY

DRUG	DOSE	ROUTE
ERGOT ALKALOIDS		
Ergotamine	1-2mg/d: max 6g/day	oral
Dihydroergotamine	0.75-1mg	SC
5HTReceptor agonists		
Sumatriptan	25-300mg	oral
	6mg	SC
Rizatriptan	10mg	oral

Ergot Alkaloids

- Ergotamine
  - Mechanism of antimigraine action
    - Exact mechanism unknown
  - Therapeutic uses
    - Drug of choice to stop an ongoing migraine
  - Pharmacokinetics
    - PO, sublingual, rectal, or inhalation
  - Adverse effects
    - Nausea/vomiting, weakness in the legs, myalgia, numbness and tingling in fingers or toes, angina-like pain, tachycardia or bradycardia

Mechanism of action



Acute Treatment - Triptans

- Reasonable first choice for patients with moderate to severe disability from migraines
- Limit use to 2-3 days per week
- Patients who fail one triptan often respond another
- Do not use one triptan within 24 hours of another

Acute Treatment - Triptans

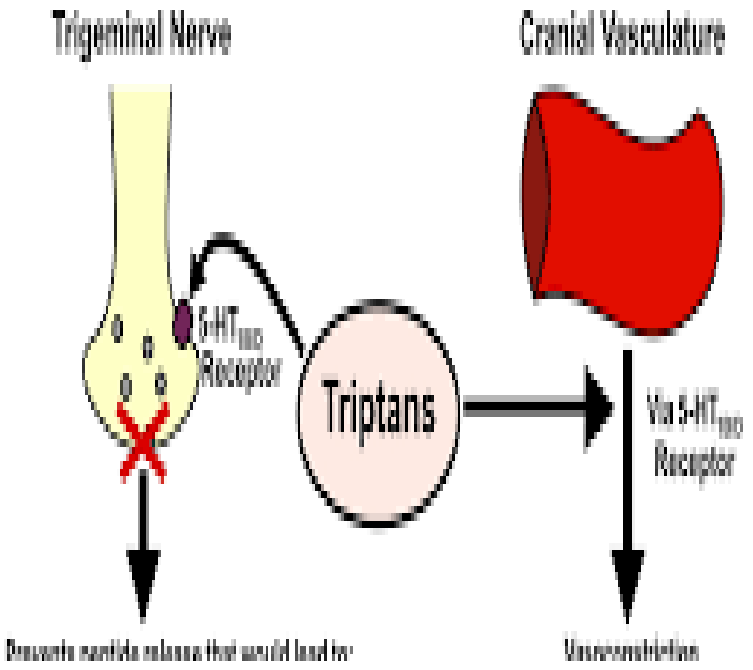
Mechanism of action

- 5HT-1B/1D agonists
- Inhibit release of CGRP & substance P
- Inhibit activation of the trigeminal nerve
- Inhibit vasodilation in the meninges

Precautions

- Ischemic heart dz or stroke
- High risk for CAD
- Pregnancy
- Hemiplegic or basilar migraine
- Ergots
- **Use w/ SSRIs?**

Proposed Triptan Mechanism of Action



Triptan	First Available	Original Brand Name	Available As					
			T	ODT	NS	SI	BAI	OF
almotriptan	2001	Axert	X					
eletriptan	2002	Relpax	X					
frovatriptan	2001	Frova	X					
rizatriptan	1998	Maxalt, Maxalt-MLT	X	X				X[1]
naratriptan	1998	Amerge	X					
sumatriptan	1992	Imitrex	X		X	X	X	
zolmitriptan	1997	Zomig, Zomig-ZMT	X	X	X			

T=tablet; ODT= orally disintegrating tablet; NS=nasal spray;
BAI=breath activated inhaler; OF=oral film

[1] Under FDA review, but not yet approved as of July, 2017.

Triptan side effects

- Flushing, feeling or warmth
- Chest pressure or heaviness
- Throat tightness
- Paresthesias
- Dizziness, fatigue, drowsiness
- Nausea
- Intolerable taste with nasal formulations

Indications for a preventive agent

- Migraine-related disability ≥ 3d/month
- Migraines last over 48 hours
- Acute treatments are contraindicated, ineffective, or overused
- Migraines cause profound disability or prolonged aura
- Patient preference

General Principles of Preventive Treatment

- Start with a low dose and increase slowly
- Use an adequate trial of 2 to 3 months
- Avoid medication interactions/contraindications
- Monitor with calendar or diary
- Monitor for medication overuse
- Consider comorbid conditions
- Consider preventive medication combinations in refractory patients
- Taper when headaches are controlled

Continuum Lifelong Learning Neurol 2012;18(4):764–782

PREVENTIVE THERAPY

	DRUGS	DOSE (mg/dl)
1	**BETA BLOCKERS**	
	Propranolol	40-320
2	**Calcium channel Blockers**	
	Flunarizine	10-20
	Verapamil	120-240
3	**TCAs**	
	Amitriptyline	10-20
4	**SSRIs**	
	Fluoxetine	20-60

Anti Migraine Drugs

- Prophylactic
  - Beta blockers
  - Valproic acid
  - Topiramate
  - Tricyclic antidepressants
  - Calcium channel blockers (e.g.,verapamil)
  - ACE inhibitors
  - Angiotensin II receptor blockers
  - Methysergide
  - Gabapentin
  - Botulinum toxin A
- Abortive
  - Triptans
  - Ergotamine
  - Dihydroergotamine
  - NSAIDS
  - Isometheptene
  - Tramadol

BETA BLOCKERS

Advantages

- Thoroughly studied and widely used
- Timolol (Blocadren) and propranolol (Inderal) are FDA approved
- Good choice for patients with HTN, CAD, tremor, or anxiety

Adverse effects of Beta Blockers

mnemonic:

BBALD FISH

B bronchoconstriction

B bradycardia

A arrhythmias

L lethargy

D disturbance in glucose metabolism

F fatigue

I insomnia

S sexual dysfunction

H hypotension

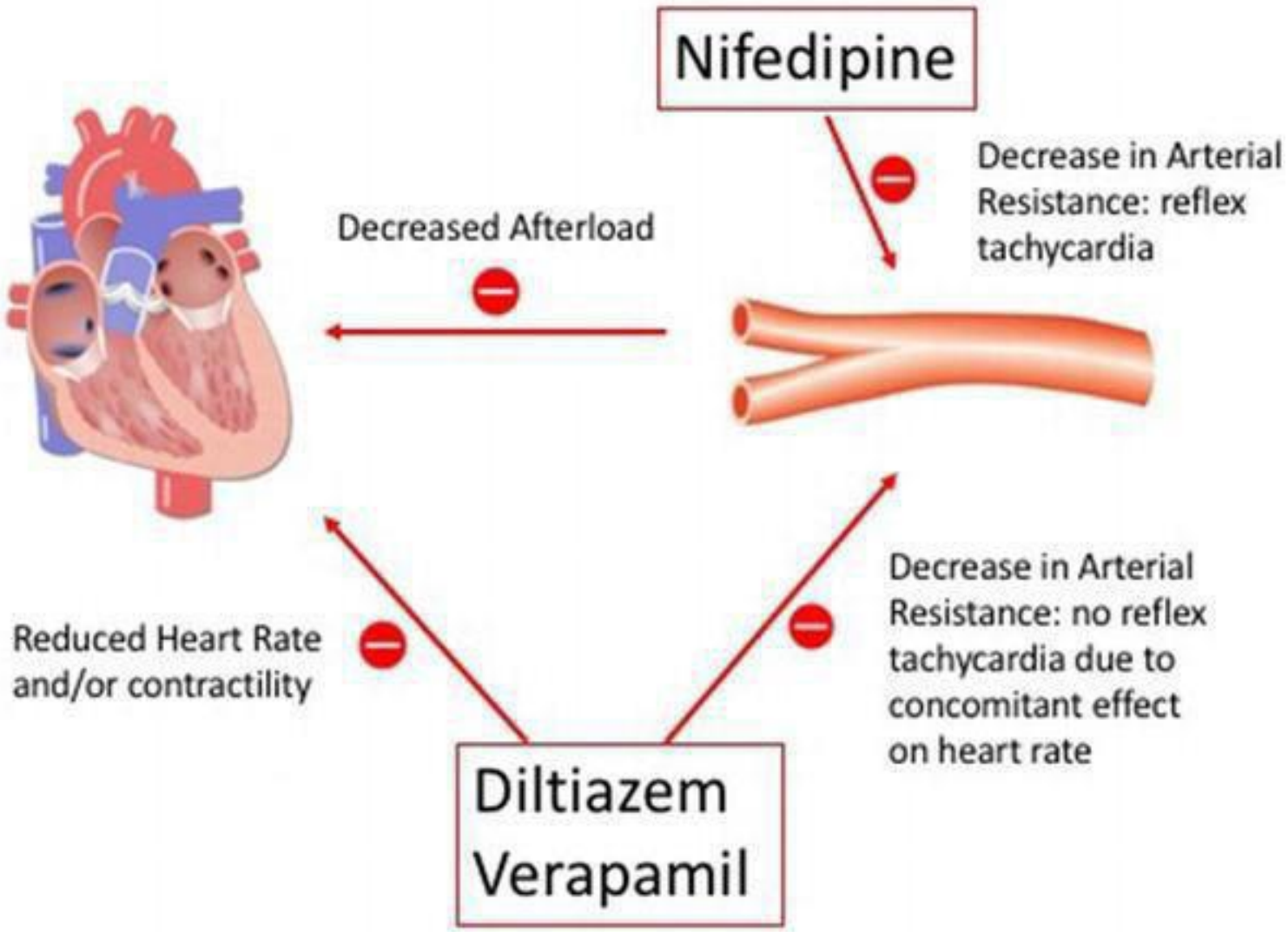
Calcium channel blockers

. Although the mechanism by which calcium channel antagonists affect migraine is not known,

. vasoconstriction , prevention of platelet aggregation and alterations in release and reuptake of serotonin.

. Several trials have indicated some benefit for verapamil and flunarizine In recurrent migraine.

. Verapamil in doses of 80 to 160 mg 3 times a day reduces the incidence of migraine with aura, but it is not as useful in migraine without aura.


Nifedipine
Decrease in Arterial Resistance: reflex tachycardia
Decreased Afterload
Reduced Heart Rate and/or contractility
Decrease in Arterial Resistance: no reflex tachycardia due to concomitant effect on heart rate
Diltiazem
Verapamil

Long term Treatment

- Reducing the attack frequency and severity
- Avoiding escalation of headache medication
- Educating and enabling the patient to manage the disorder
- Improving the patient quality of life

Take home messages/conclusion

- MIGRAINE IS A COMPLEX DISORDER OF BRAIN EXCITABILITY AND NOT SIMPLY A "VASCULAR HEADACHE"
- MIGRAINE IS EXTRAORDINARILY COMMON AND UNDERDIAGNOSED.
- THE MAJORITY OF MIGRAINE PATIENTS CAN BE EFFECTIVELY AND SAFELY TREATED WITH AN ORGANIZED PLAN OF LIFESTYLE MANAGEMENT , ACUTE THERAPY, AND PREVENTIVE THERAPY IF NEEDED
- PROMISING NEW THERAPIES ARE ON THE HORIZON

REFERENCES:

- Headache Classification Committee The International Classification of Headache Disorders. 2nd edition. Cephalalgia. 2004;24:1–160.
- Lipton RB, Bigal ME, Diamond M, Freitag F, Reed ML, Stewart WF. Migraine prevalence, disease burden, and the need for preventive therapy. Neurology. 2007;68:343–9.
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TIME
TO
RELAX

Red eye

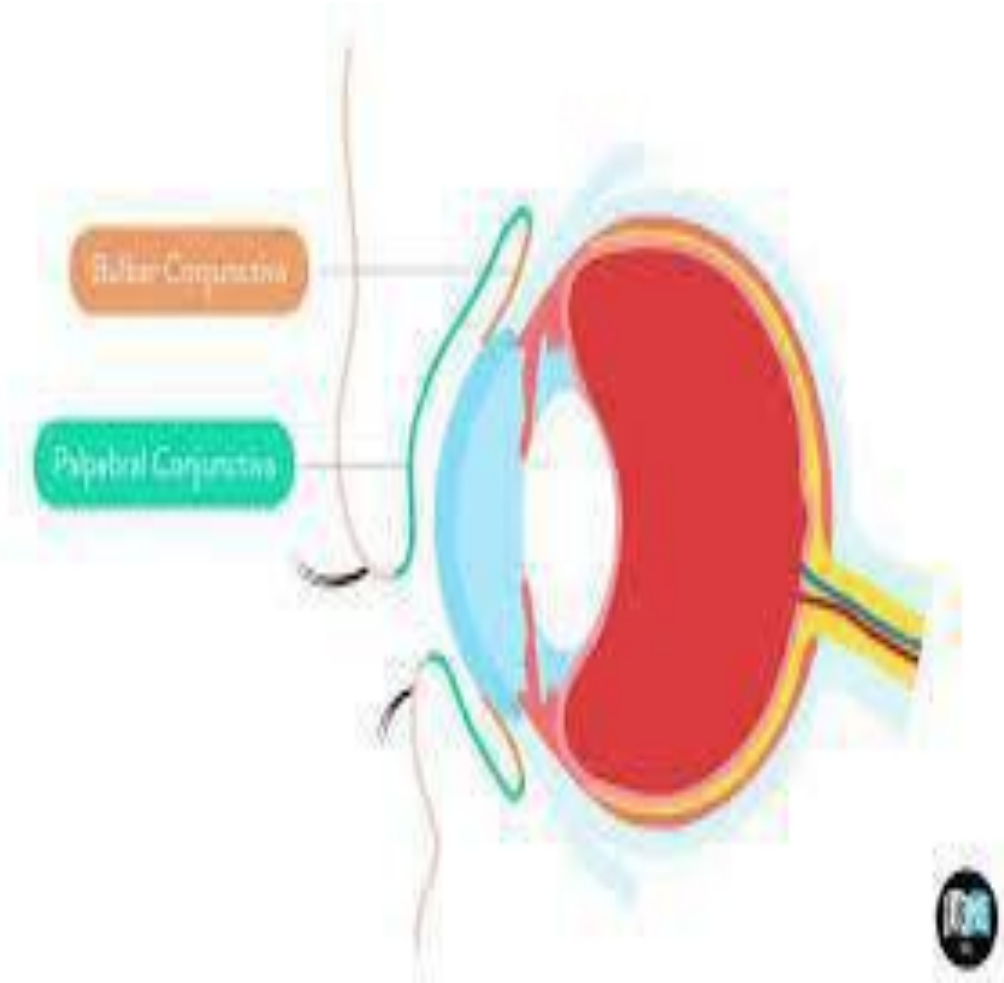
Dr Nazullah
Associate professor

Objectives

- At the end of this session the 4th yr MBB
- student should be able
- Definition
- Enlist Causes / classification
- Clinical features
- Investigation
- Treatment options
- Complications

- A “red eye” is a general term to describe red, irritated and bloodshot eyes.
- The redness happens when tiny conjunctival blood vessels under your eye’s surface get larger or become inflamed. Usually, it’s a reaction to something irritating your eye.
- Redness can affect one or both eyes. It can develop over time or appear suddenly, such as with allergies or an eye injury.

- Conjunctiva
- The surface of the eye and the
- inner surface of the eyelids are
- covered with a clear membrane
- called the conjunctiva
- *Palpebral*
- *Bulbar*



Broad division My division

- Red eye with Normal vision and no pain or mild discomfort
- Red eyes with visual loss and sever pain

Scenario

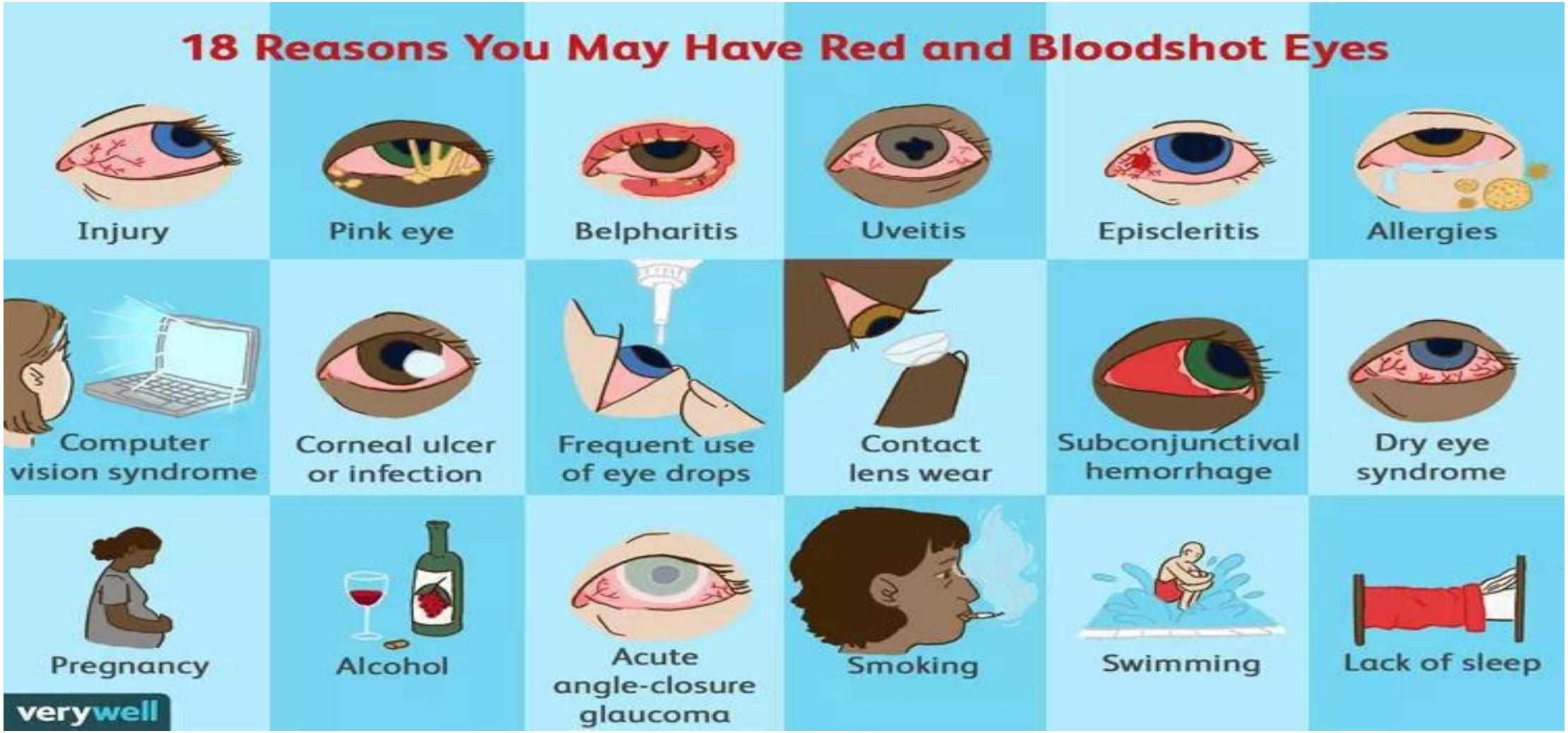
- A hypertensive patient of 52yrs comes to eye opd. He noticed redness in his Rt eye since last night. His vision is 6/6 Bes. No pain no discharge. There is mild discomfort. What is the most probable cause ,?
- A acute blepharitis
- B acute conjunctivitis
- C acute keratitis
- D sub conj- hemorrhage

Dx D

Syptoms/signs；：Depending on what’s going on, your red eye can feel:

- Completely normal. In this case, you don’t know it’s red till you see it
- Eye pain Itching. Eye discharge. Swollen eyes/Lids.
- Changes in vision, like blurred vision. Severe pain
- Light sensitivity Cold-like symptoms
- Nausea vomiting
- Blood in the AC (the colored part of the eye)

Red eye Common Causes



Conjunctivitis

s/c hg

Treatment for red eye?

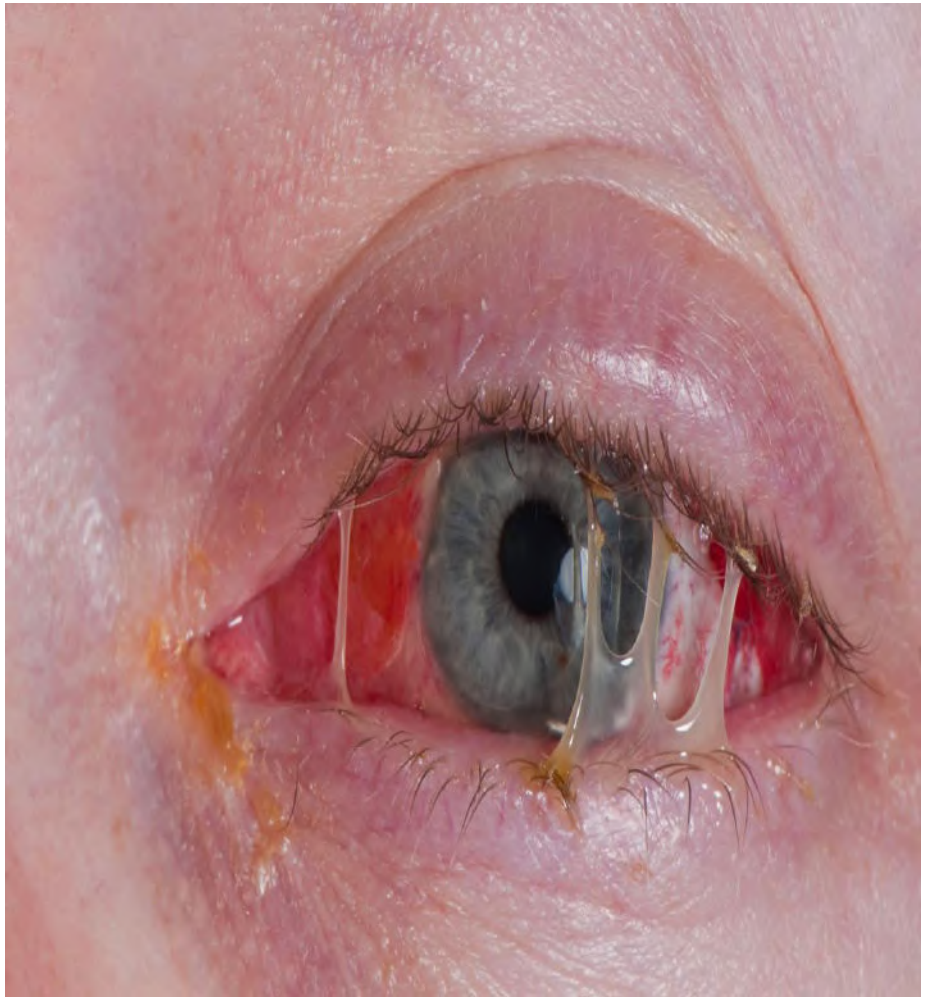
- Remedies for red eye are wide-ranging, and often they’re things you can do at home for yourself.
- Many times, the following steps can relieve symptoms:
  - Rest.
  - Cool compresses over closed eyes.
  - Lightly massaging your eyelids.
  - Gently washing your eyelids.
  - Over-the-counter eye drops. OTC med
- Other times, an eye care specialist may recommend and prescribe antibiotics, special eye drops or ointments.

Specialist opinion

- When ?
- Your eyes feel tender.
- Your vision is affected.
- Your eyes become extra sensitive to light.
- You have symptoms that continue for a week or more, or are getting worse and not better.
- Your eye is producing a lot of pus or mucus that dries into crusts.
- You have a fever or aches along with eye discomfort.

Conjunctivitis classification

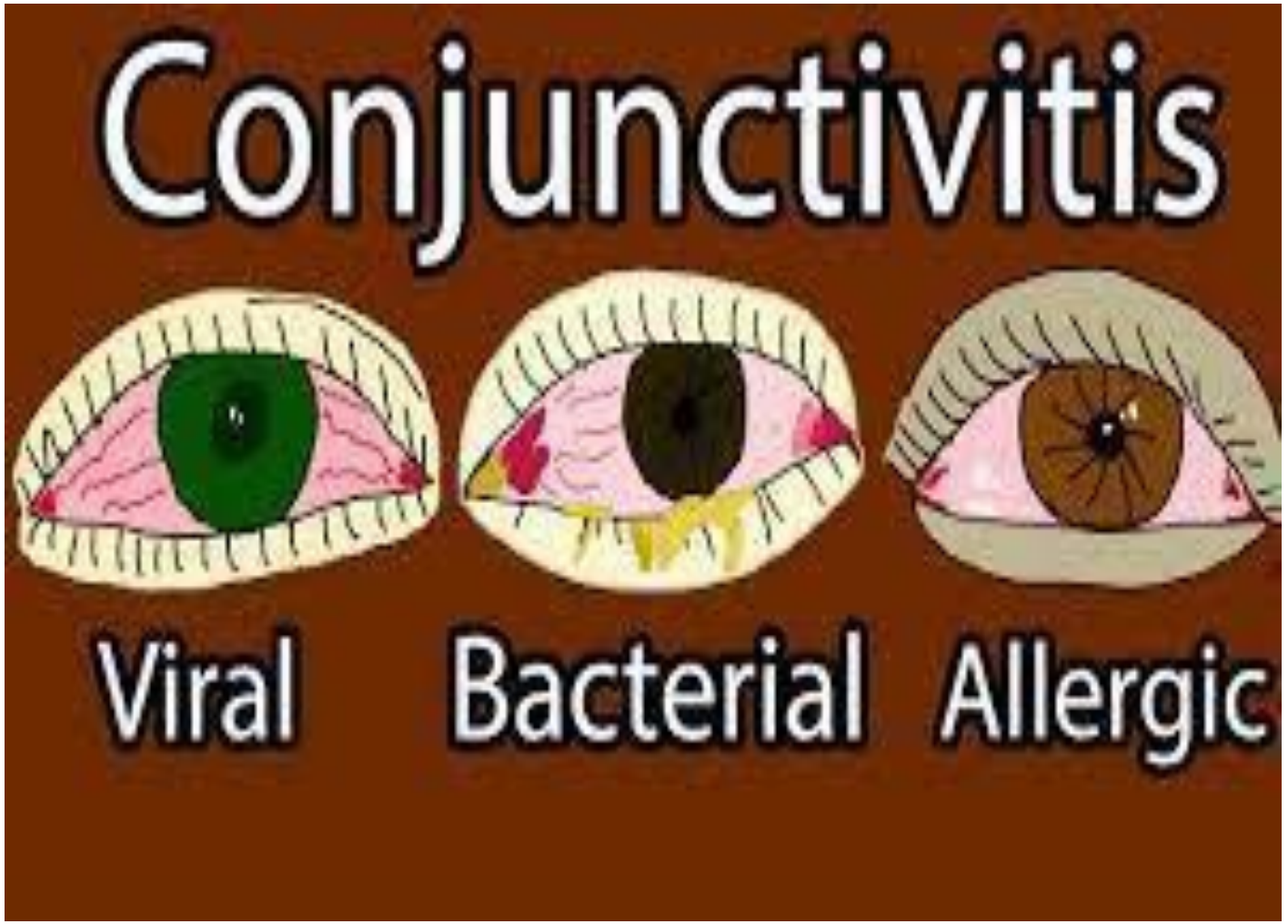
- On the basis of Aetiology(Infective)
- Bacterial
- Viral
- Chlamydial



Allergic Conjunctivitis(Non-infective)

- Seasonal allergic conjunctivitis
- Perennial allergic conjunctivitis
- Vernal keratoconjunctivitis
- Atopic keratoconjunctivitis
- Giant papillary conjunctivitis
- Keratoconjunctivitis sicca (dry eyes)
- Superior limbic keratoconjunctivitis (SLK)

. Chemical or irritative conjunctivitis


Conjunctivitis
Viral
Bacterial
Allergic

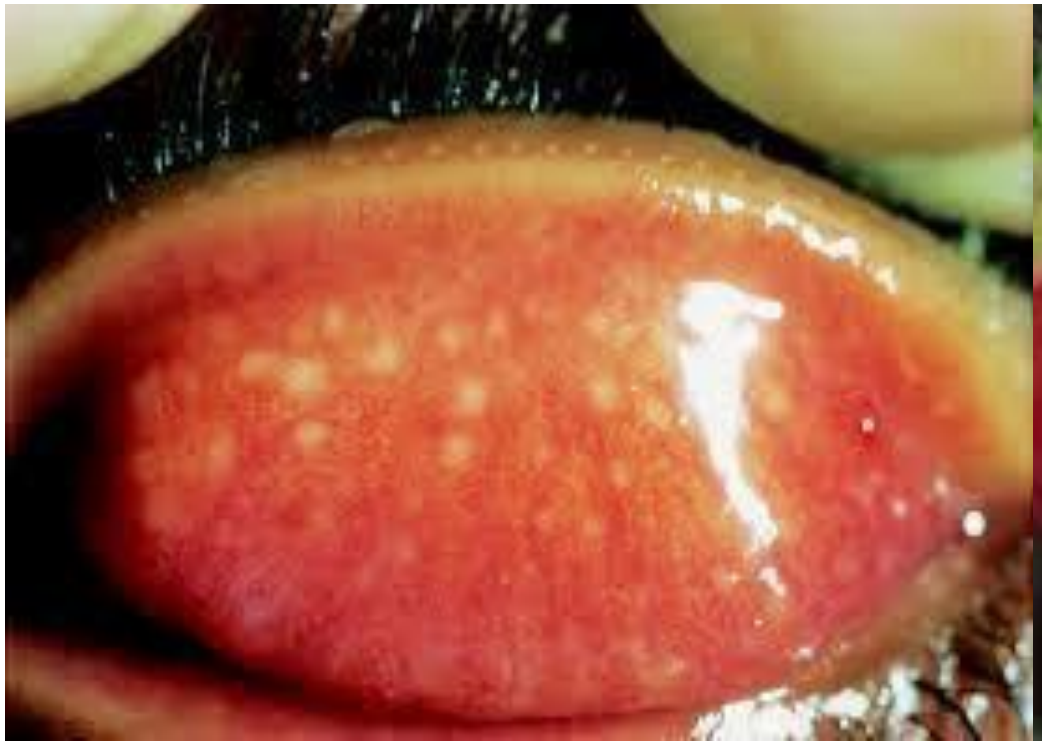
On basis of exudates

- A. Watery / Serous , Viral , Allergic , Toxic
- B. Mucoud , Chronic Allergic and Keratoconjuctivitis sicca
- C. Mucopurulent, Mild Bacterial, Chlamydial infection
- D. Purulent , Gonococcal and any severe bacterial infection

On basis of conjunctival reaction

- Follicular conjunctivitis
- Viral , Adenoviral, HSV common
- Chlamydial, Trachoma common
- Topical medicine , Epinephrine & Eserine
- Papillary conjunctivitis
- Allergic , VKC & Atopic conjunctivitis
- Autoimmune Disorder Cicatricial pemphigoid
- Chronic blephritis , Squamus blepharitis
- Chronic irritation contact lense, prosthesis, nylon suture etc

Follicule with white core



Papillae are red

Papillae

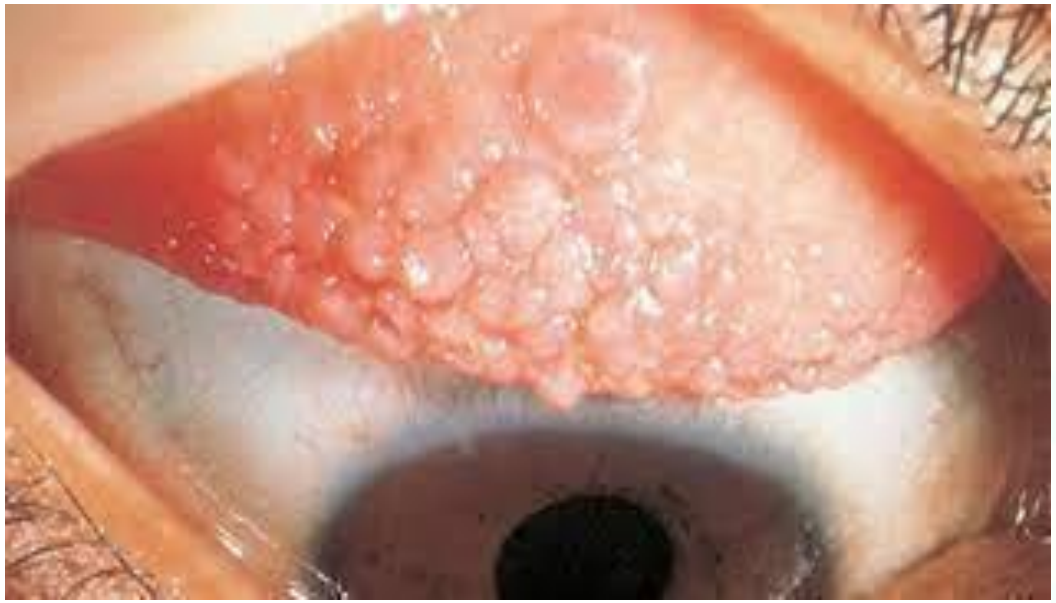
There are two important distinctions that are hallmark signs of papillae:

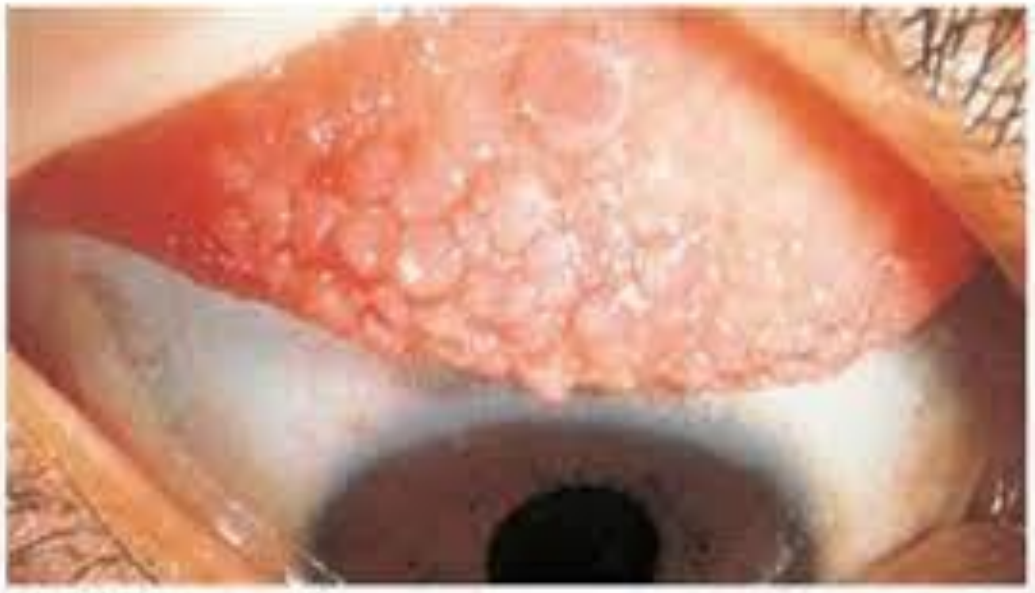
1.**Red Center** = Papillae are elevations of the vascular conjunctival tissue so there will be a red central vascular core to the lesions

2.Distinct Elevations = The conjunctival tissue has fibrous connections that limit the size and expanse of swelling. This means distinct, well separated bumps in the papillary reaction. The only time this logic doesn't fully apply is when the inflammation has been around for so long that the bumps begin to coalesce, creating the large cobblestone appearance we associate with giant papillary conjunctivitis.

- **Papillae** are raised areas of inflammation with a central blood vessel, appearing red at the surface and paler at the base. In papillae mainly vessels are involved, inflammation, engorgement due to chronic irritation & exposure.
- **Follicles** are an accumulation of white blood cells without a central vessel, and appear pale at the surface and redder at the base. In follicles the different blood cells are attracted & accumulated at the site of infection

Papillae (giant)





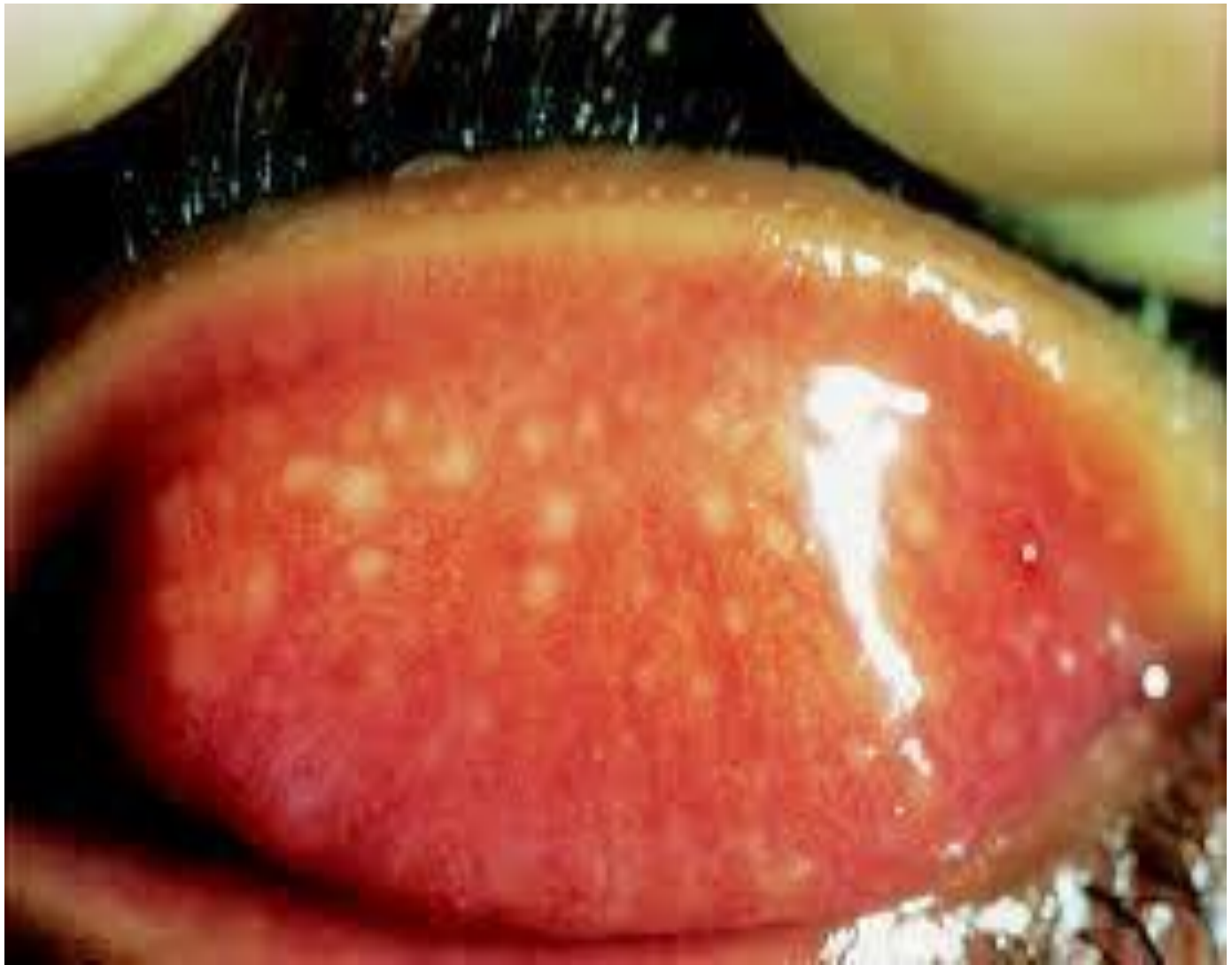
Follicle

While papillae are consistent with a local irritation, follicles are a sign of an immune reaction in the area. This finding is almost always an indication of a **viral infection** of the eye.

Follicles have a larger appearance than papillae, and have a ***white central*** core since they are really accumulations of cellular level inflammatory agents -- lymphocytes, lymphoblasts, and macrophages if you remember your microbiology. The big tell-tale sign of follicles are:

White Center = look for that inflammatory matter filling the center of the bump

Typical Follicle

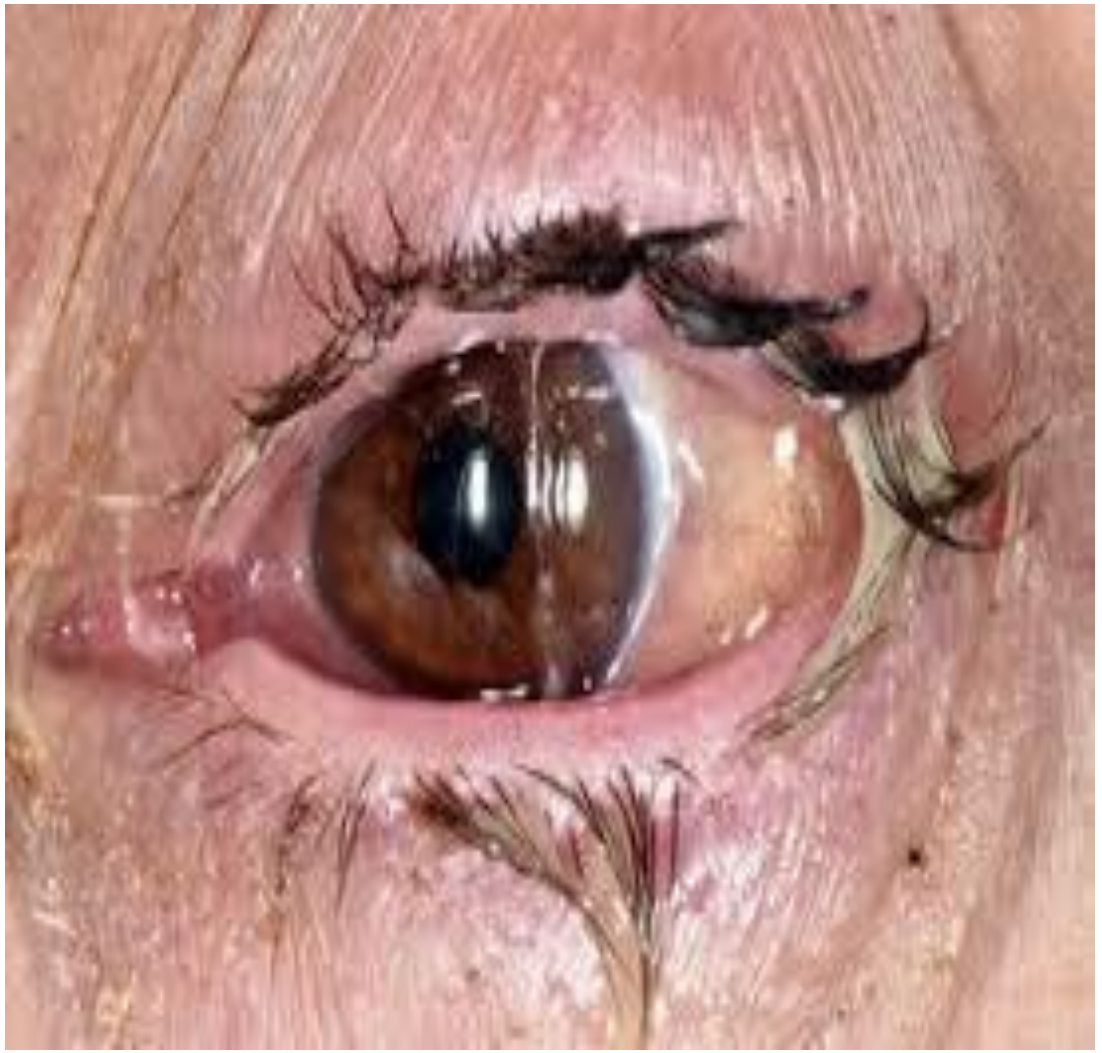


Scenario

- A 25yrs male patient come to opd. He is complaining of redness, irritation mucopurulent discharge with mild eye ache for the last few days.. On examination his vision is 6/6 Bes, there is conjunctival congestion with discharge. What can be the most probable cause.?
- A acute allergic conjunctivitis
- B acute bacterial conjunctivitis
- C acute keratitis
- D acute eye trauma
- Dx B

Bacterial Conjunctivitis

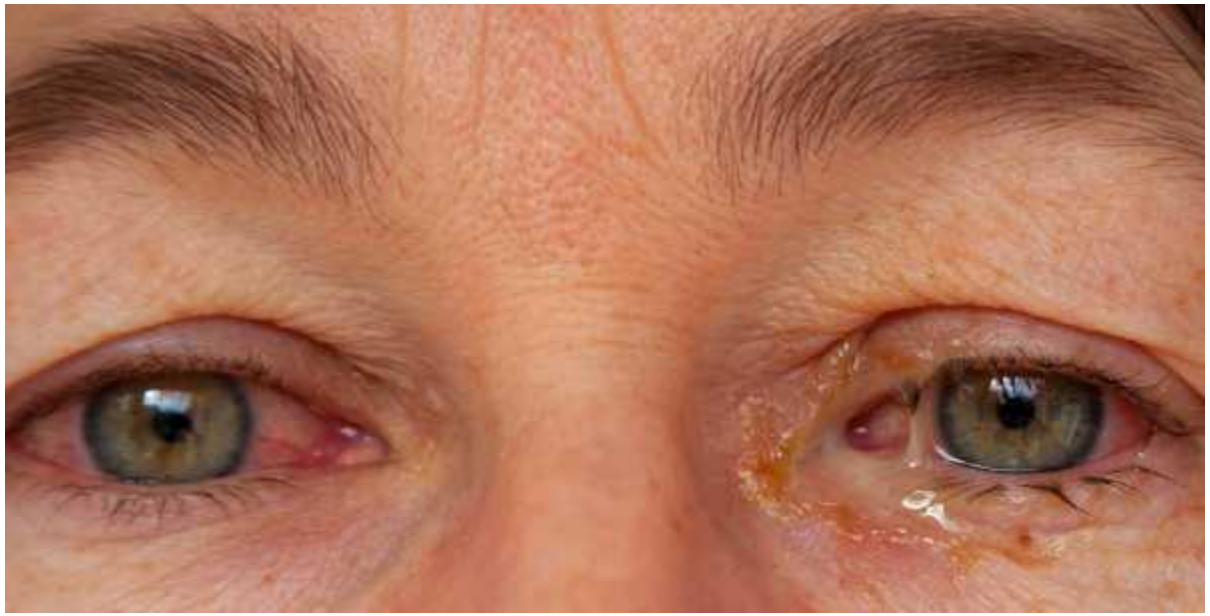
- Acute inflammation of the conjunctiva with mucopurulent discharge & redness
- Common organism are, Staph-Aureous, Strep-pneumonia & H-influenza
- Less common are Morexella lacunatea, Klebsial & Proteus species



Clinical feature

- Symptoms; Very common with acute redness, FB sensation, burning sensation & discharge
- It is usually unilateral but may be bilateral within few days
- Photophobia may or may not be there
- Signs are the redness is in the fornicial & palpebral conjunctiva.
- In severe the whole conjunctiva is hyperemic with pink coloration. S/C hg may be there.
- Discharge is mucopurulent
- Eyelashes are matted by discharge. Lids may be swollen
- Papillary reaction
- VA is normal

Bacterial conjunctivitis



Diagnosis

- Clinical evaluation
- Diagnosis of conjunctivitis and differentiation between bacterial, viral, and noninfectious conjunctivitis are usually clinical.
- Smears and bacterial cultures should be done in patients with severe symptoms, immunocompromise, ineffective initial therapy, or a vulnerable eye (eg, after a corneal transplant, in exophthalmos due to Graves disease).
- Smears and conjunctival scrapings should be examined microscopically and stained with Gram stain to identify bacteria and stained with Giemsa stain to identify the characteristic epithelial cell basophilic cytoplasmic inclusion bodies of chlamydial conjunctivitis (see Adult Inclusion Conjunctivitis) .

Treatment

- Bacterial conjunctivitis is very contagious, and standard infection control measures should be followed.
- To avoid transmitting infection, **physicians** must
- Use hand sanitizer or wash their hands properly (fully lather hands, scrub hands for at least 20 seconds, rinse well, and turn off the water using a paper towel)
- Disinfect equipment after examining patients
- **Patients** should do the following:
- Use hand sanitizer and/or wash their hands thoroughly after touching their eyes or nasal secretions
- Avoid touching the noninfected eye after touching the infected eye
- Avoid sharing towels or pillows
- Avoid swimming in pools

Treatment Contnd

- Antibiotics
- Topical like gentamycin tobramycin ofloxacin ciprofloxacin. In more sever cases Moxifloxacin 0.5% drops one hrly a day for 7 to 10 days or trimethoprim/polymyxin B 4 times a day. Single therapy or in combination can b used
- Ointment at bed time
- Systemic. In some cases systemic antibiotic can be given in addition to the topical antibiotics
- A poor clinical response after 2 or 3 days indicates that the cause is resistant bacteria, a virus, or an allergy.
-

Complications

- Are Rare.
- include corneal ulceration, abscess, perforation, panophthalmitis, and blindness.

Ophthalmia neonatorum

Ophthalmia neonatorum (neonatal conjunctivitis) results from a maternal gonococcal and/or chlamydial infection.

Neonatal conjunctivitis occurs in 20 to 40% of neonates delivered through an infected birth canal. Very rare in our society

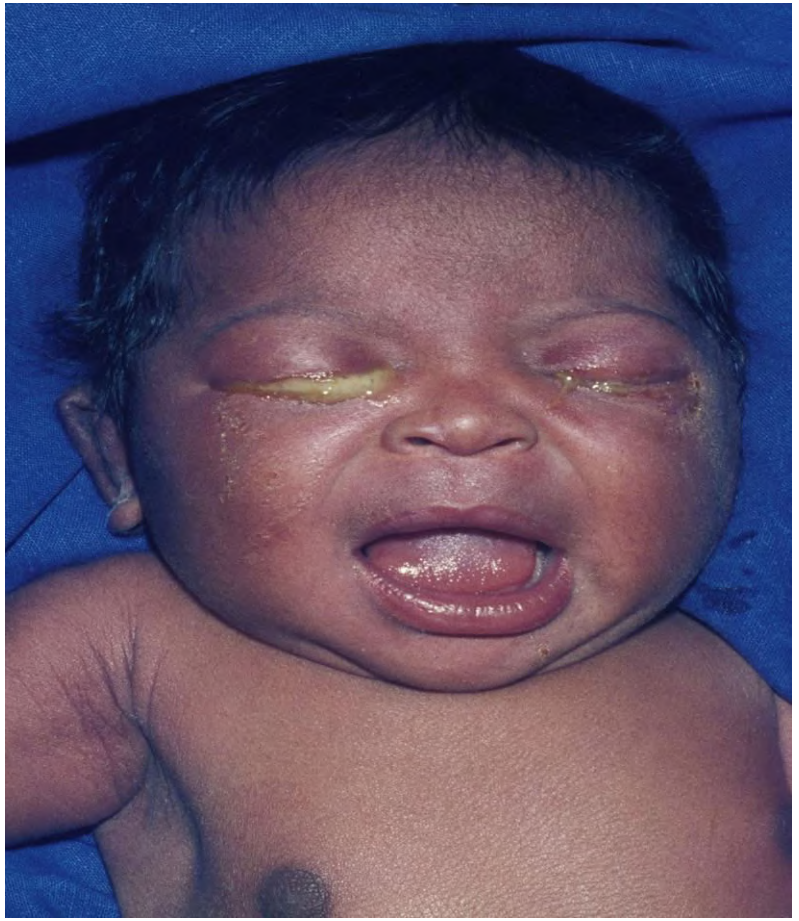
Other organism such as staphys, strep pneumonia, H Infuenza

Virus such as H simplex

Different chemical such as Silver Nitrate or antibodies used for prophylaxis can cause

Ophthalmia neonatorum

Ophthalmia neonatorum caused by gonococcal infection appears 2 to 5 days after delivery.
With ophthalmia neonatorum caused by a chlamydial infection, symptoms appear within 5 to 14 days.
Symptoms of both are bilateral, intense papillary conjunctivitis with eyelid edema, chemosis, and mucopurulent discharge.



Treatment /Prevention

- *Topical* antibiotics drops like gentamycin, Tobramycin and Quinolones one hrly is recommended. Single or combination.
- Ointment at bed/sleeping time
- Systemic For gonococcal infection, ceftriaxone 25 to 50 mg/kg IV or IM (not exceeding 125 mg) is given as a single dose.
- Chlamydial infection is treated with erythromycin 12.5 mg/kg orally or IV 4 times a day for 14 days. The parents should also be treated.
- Ophthalmia neonatorum is prevented by the routine use of silver nitrate eye drops or erythromycin ointment at birth.

Treatment(Parents)

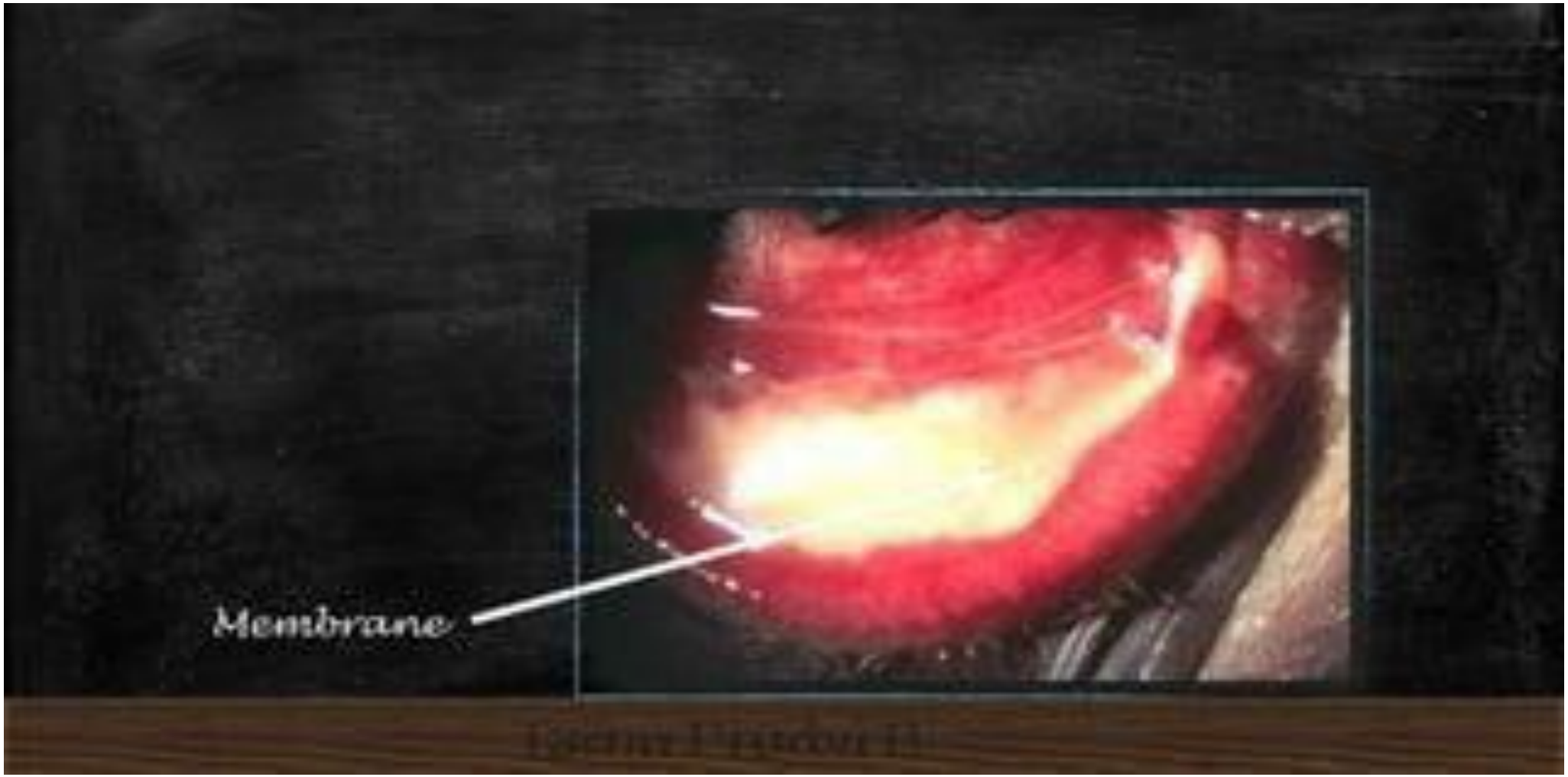
- Sex partners should also be treated. Patients need to be evaluated for other sexually transmitted infections and the local public health authorities need to be notified.
- Because of chlamydial genital infection is often present in patients with gonorrhea, adult gonococcal conjunctivitis requires dual therapy with a
- single dose of ceftriaxone 1 g IM plus azithromycin 1 g orally once (with azithromycin allergy or to treat expected chlamydial co-infection use doxycycline 100 mg orally twice a day for 7 days). Azithromycin
- Topical Quinolones recommended but because resistance is now widespread.
- Bacitracin 500 U/g or gentamicin 0.3% ophthalmic ointment instilled into the affected eye every 2 hours may be used in addition to systemic treatment.

Treatment (Chlamydial)

- Systemic Erythromycin 50mg/kg body weight divided doses for 2weeks. Azithromycin is given now a days
- Topical Suphonamide drops 10%
- Tetracyclin oint for 4weeks
- Other bacterial
- Topical antibiotics
- Systemic antibiotic

- Pead's specialist opinion

Membranous conjunctivitis



Clinical features

- There is severe conjunctival inflammation associated with deposition of fibrinous exudates forming a whitish membrane over the conjunctival surface
- Removal of this membrane is difficult & causes ulceration & bleeding
- Periocular lymph nodes are enlarged

- Is one of the serious infection in which a membrane is formed on the surface of the conjunctiva due to thick exudates
- Is caused by Mycobacterium Diphtheria
- Rare nowadays
- Very serious conjunctivitis
- Very red congested eyes with copious discharge
- Usually child age are affected
- May be fever
- With lymphadenopathy

- Bacteria such as
- Corynbacterium diphtheria, Nesseria gonorrhea, Strep pneumonia, Staph Aureus,
- Viral,
- Chemical & thermal burns

Can lead to membrane formation

Complications

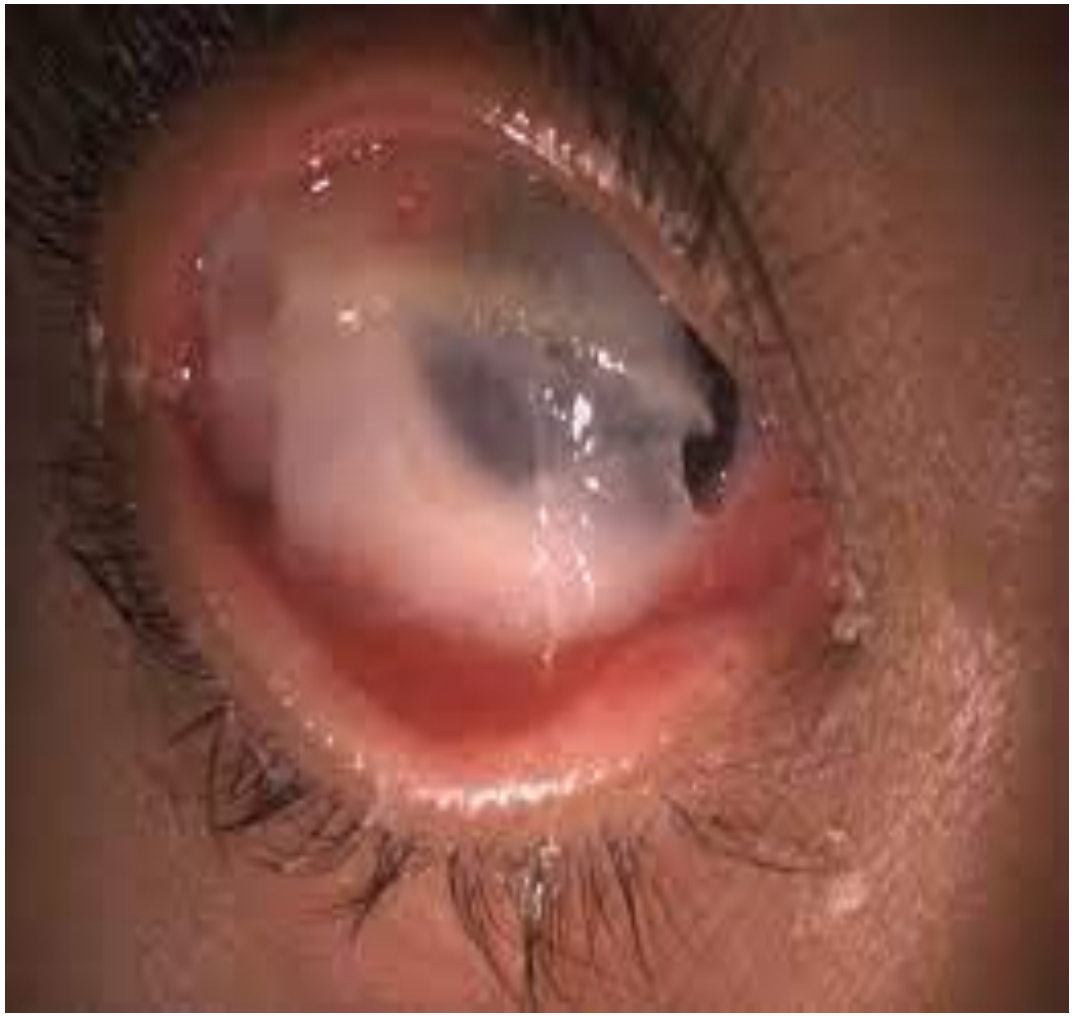
- Conjunctiva ; ulceration & cicaterization
- Xerophthalmia, Symblepharon, Entropion & trichiasis

- Cornea ; ulceration, Perforation, Blindness

- Ciliary body; accommodation paralsysis due to toxic effect

Membranous

Pseudomembranous



Membrane is made of fibrinous exudates that may or may not be firmly adherent to conjunctival epithelium. If adhered tightly means membranous and vice-versa

True membrane

Inflammatory membrane interdigitates with superficial layer of the inflamed conjunctival Surface

Removal leads to tearing & bleeding

Commonly by

N Gonorrhea

C Diphtheria

Pseudo membrane

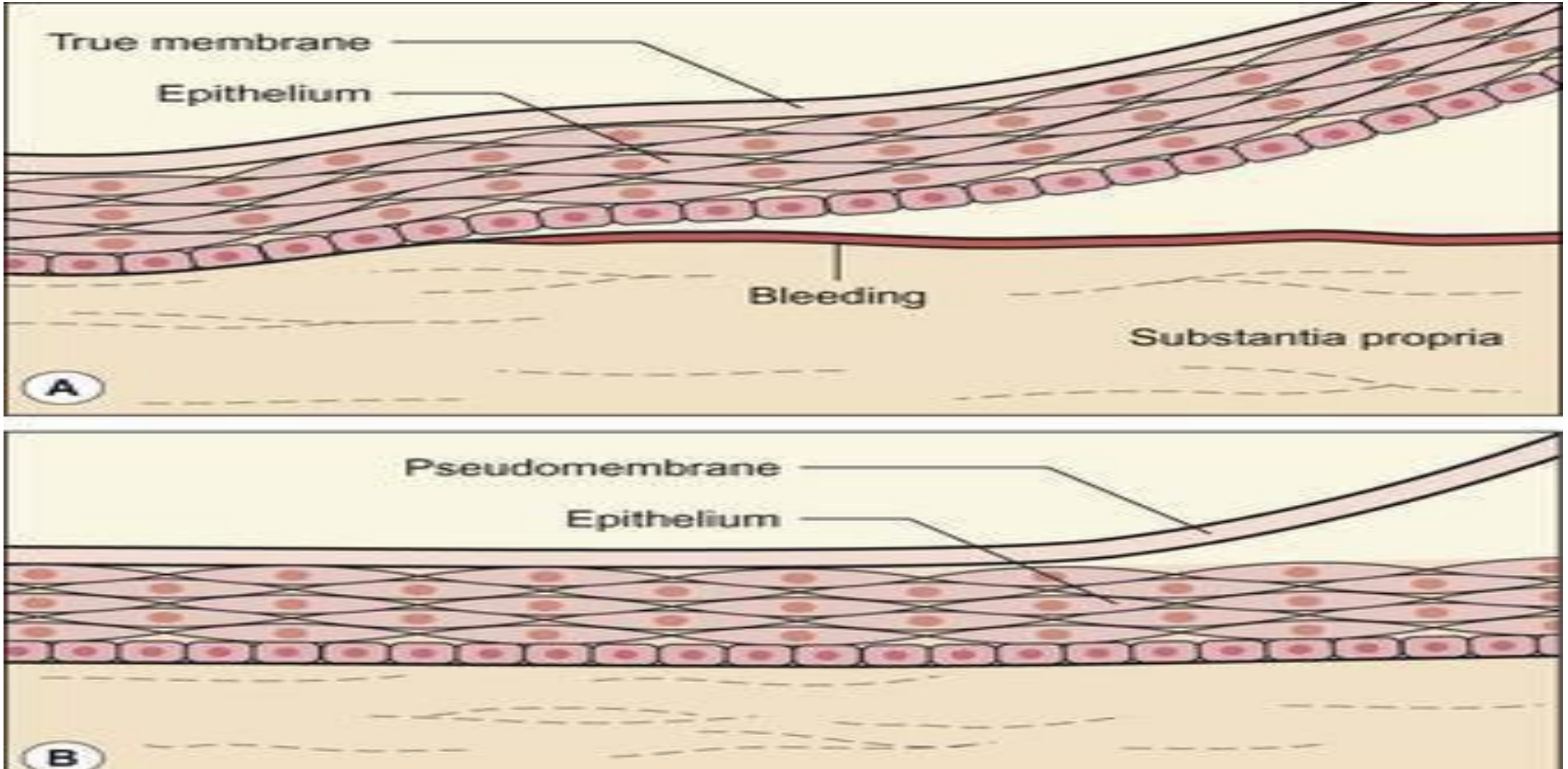
Inflammatory membrane adhere to the surface epithelium

Easily removable. No bleed on peeling

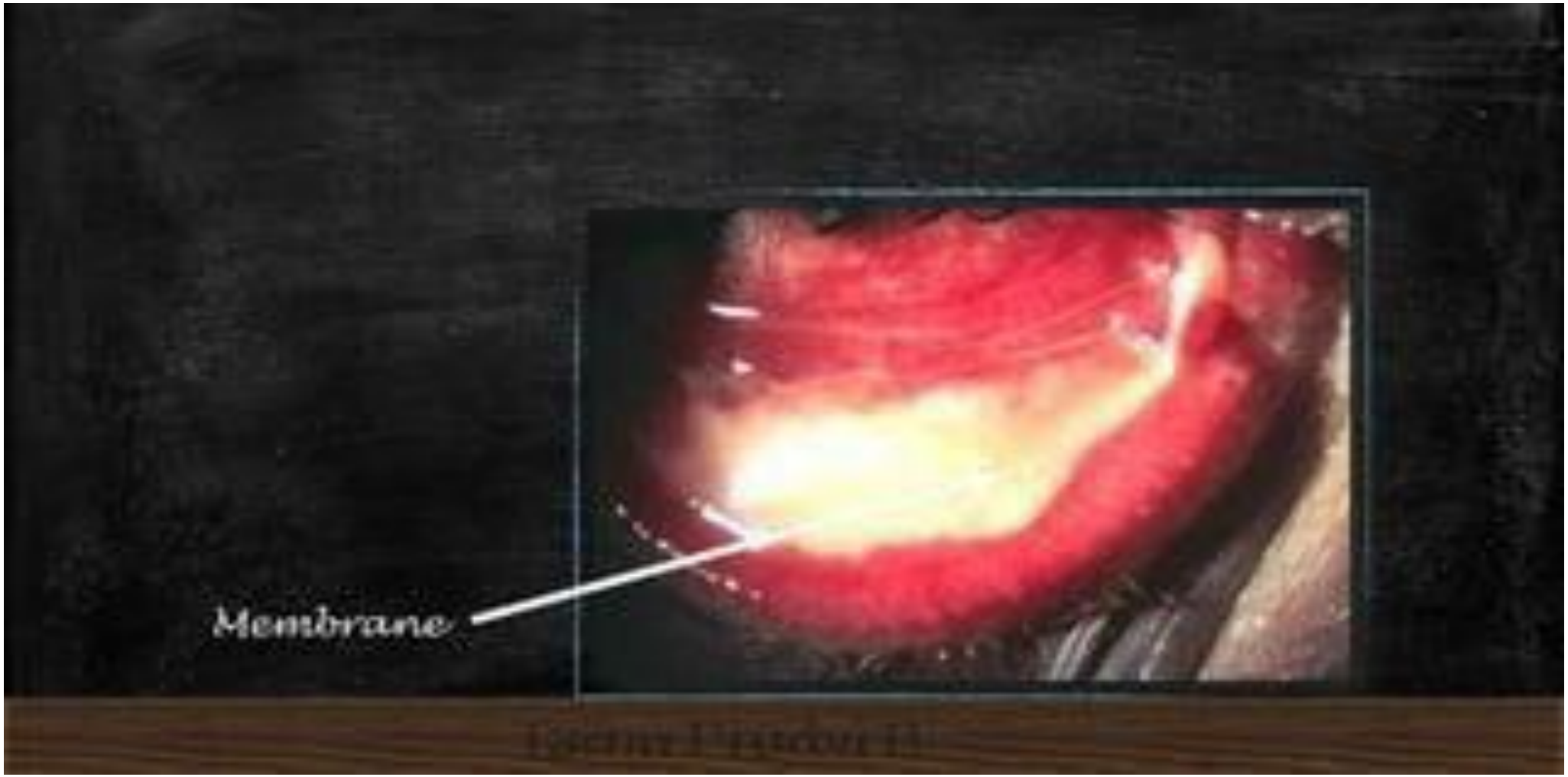
Allergic conjunctivitis, Bacterial conjunctivitis

viral conjuctivitis

Membranous VS Pseudomembranous



Membranous



Treatment

- Every child of membranous conjunctivitis should be treated as diphtheric one otherwise excluded by lab investigations . Conjunctival irrigation to remove the discharge
- Topical antibiotic; the eye is irrigated with antibiotic drops Diphtheria anti-toxin serum 4000-10000 unit along with
- Systemic antibiotic with Pencillin
  membrane peeling may be tried

- Peads opinion

Thanks

Red eye 2

Dr Nazullah

Associate professor

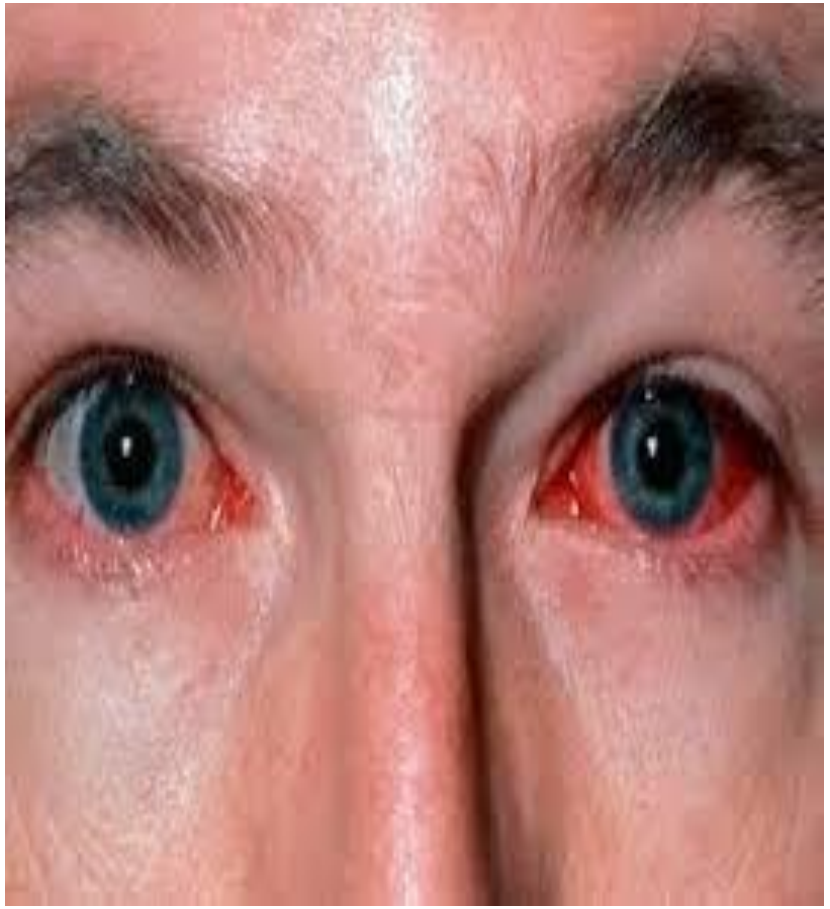
Scenario

- A 35 yrs male patient comes to opd with redness irritation for the last few days . His vision is 6/6 Bes. He is having watery discharge. Many people are affected In the area. What is most probable cause.?
- A acute bacterial conjunctivitis
- B acuta allergic conjunctivitis
- C acte viral conjunctivitisd
- D vernal conjunctivitis

- Dx C

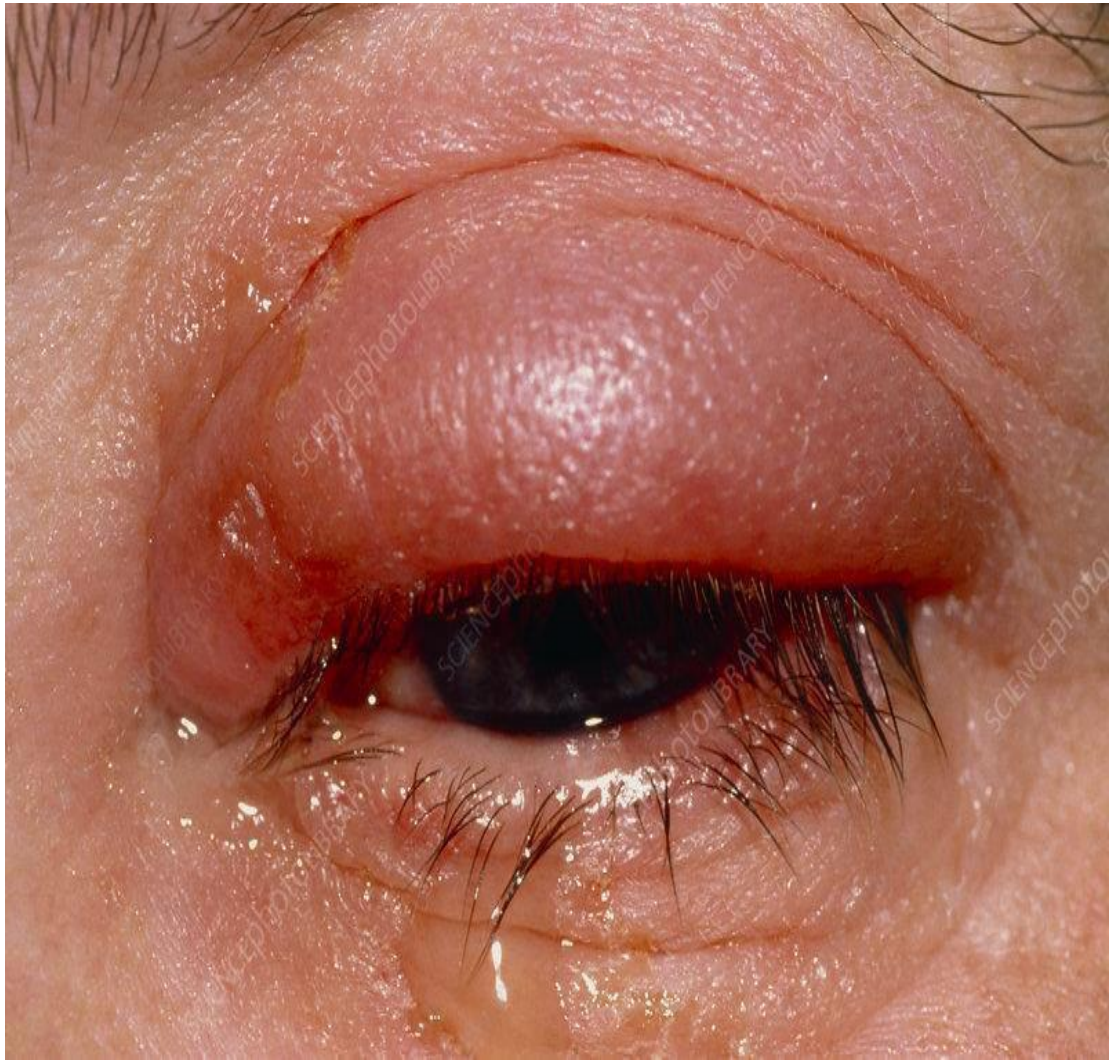
Viral Epidemic keratoconjunctivitis(EKC)

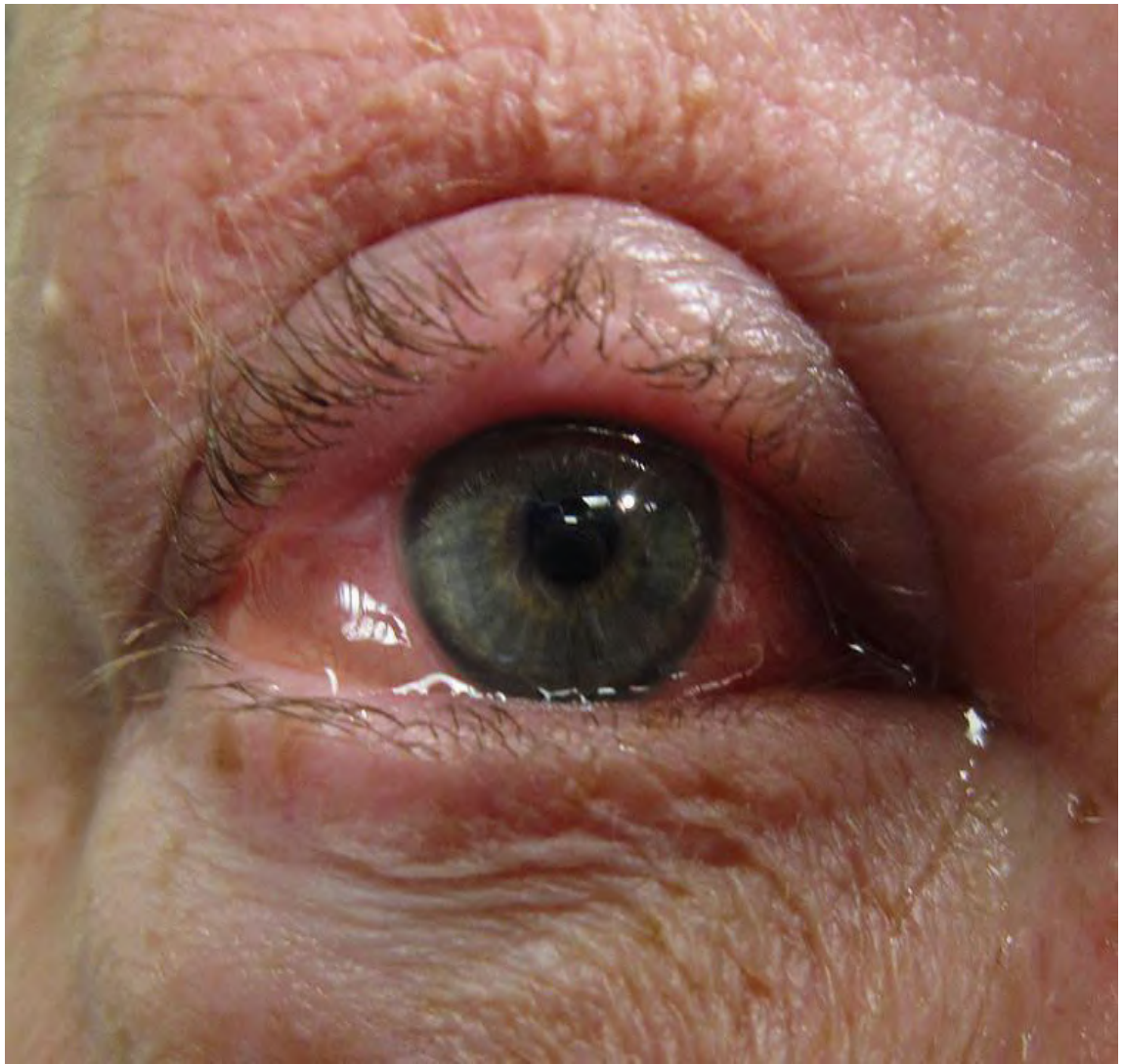
Viral conjunctivitis is a highly contagious acute conjunctival infection usually caused by an adenovirus type 8,19 &37. highly infectious .
Incubation period 7-8days
Symptoms include irritation, photophobia, and watery discharge.
Diagnosis is clinical; sometimes viral cultures or immunodiagnostic testing is indicated.
Infection is self-limited, but severe cases sometimes require topical corticosteroids.



Clinical features

- After an incubation period of about 5 to 12 days, conjunctival hyperemia, watery discharge, and ocular irritation usually begin in one eye and spread rapidly to the other.
- Transmission through conjunctival secretion
- Follicles may be present on the palpebral conjunctiva.
- A preauricular lymph node is often enlarged and painful. Many patients have had contact with someone with conjunctivitis, a recent upper respiratory infection, or both.





- In severe adenoviral conjunctivitis, patients may have photophobia and foreign body sensation due to corneal involvement (SPK).
- Lid edema & Chemosis may be present.
- Pseudo-membranes of fibrin and inflammatory cells on the tarsal conjunctiva, focal corneal inflammation, or both may blur vision. Even after conjunctivitis has resolved, residual corneal subepithelial opacities (multiple, coin-shaped, 0.5 to 1.0 mm in diameter) may be visible with a slit lamp for up to 2 years. Corneal opacities occasionally result in decreased vision and significant halos and starbursts.

- Even after conjunctivitis has resolved,
- residual corneal subepithelial
- opacities (multiple, coin-shaped,
- 0.5 to 1.0 mm
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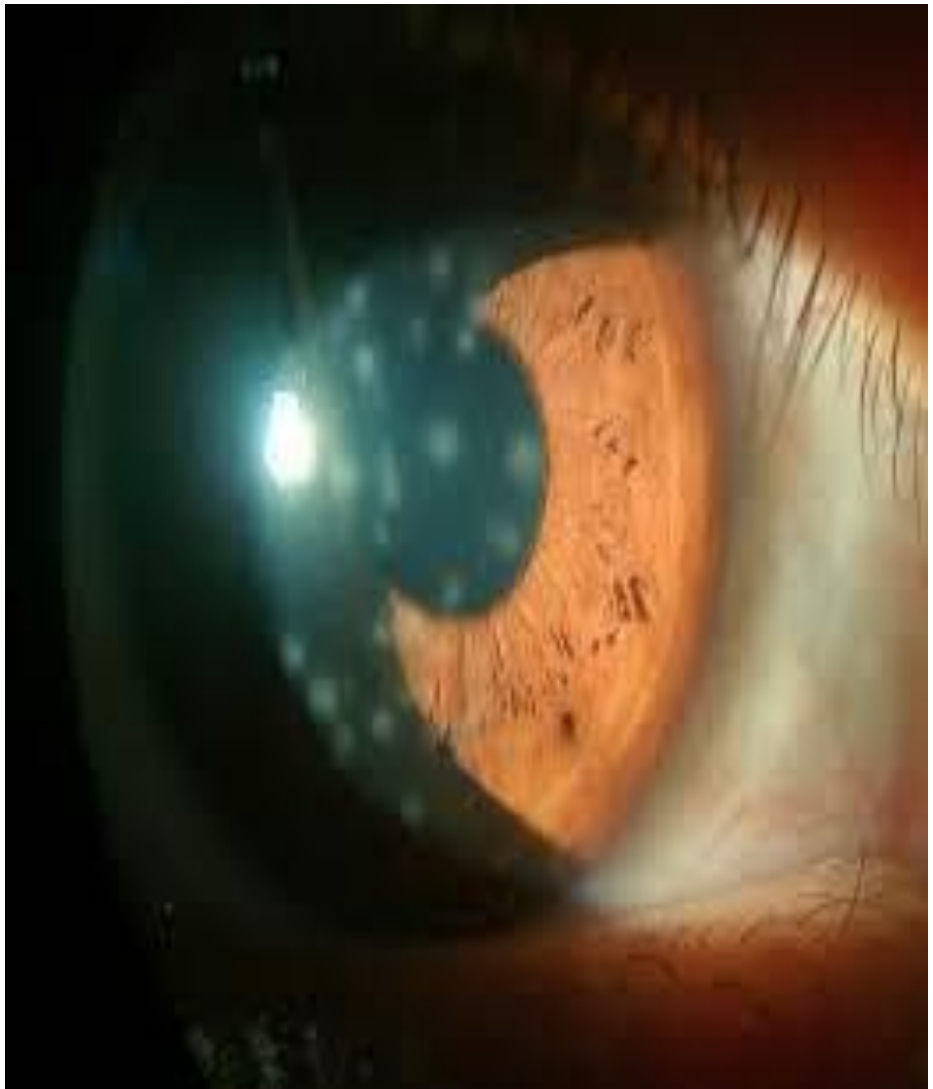


Diagnosis

- Features that may help differentiate between viral and bacterial conjunctivitis can include purulence of (a) ocular discharge, (b) presence of preauricular lymphadenopathy, and, in epidemic keratoconjunctivitis (c) flu like systemic symptoms, (d) chemosis.
- Patients with photophobia are stained with fluorescein and examined with a slit lamp.
- Epidemic keratoconjunctivitis may cause punctate corneal staining.
- Secondary bacterial infection of viral conjunctivitis is very rare. However, if any signs suggest bacterial conjunctivitis (eg, purulent discharge), cultures or other studies may be useful.

Treatment of viral Conjunctivitis

- **Preventive &Supportive measures**
- Viral conjunctivitis is highly contagious, and transmission precautions must be followed.
- To avoid transmitting infection, **physicians** must
- Use hand sanitizer or wash their hands properly (fully lather hands, scrub hands for at least 20 seconds, rinse well, and turn off the water using a paper towel)
- Disinfect equipment after examining patients

Contnd

- **Patients** should do the following:
- Use hand sanitizer and/or wash their hands thoroughly after touching their eyes or nasal secretions
- Avoid touching the noninfected eye after touching the infected eye
- Avoid sharing towels or pillows
- Avoid swimming in pools
- Eyes should be kept free of discharge and should not be patched. Small children with conjunctivitis should be kept home from school to avoid spreading the infection.
- Should sleep on the affected side

Topical antibiotic steroid combination

- Viral conjunctivitis is self-limiting, lasting 1 week in mild cases to up to 3 weeks in severe cases. It requires only cool compresses for symptomatic relief.
- ***Topical*** However, patients who have severe photophobia or whose vision is affected may benefit from topical corticosteroids (eg, 1% prednisolone acetate 4 times a day). Corticosteroids, if prescribed, are usually prescribed by an ophthalmologist.
- keratitis must be ruled out first (by fluorescein staining and slit-lamp examination) because corticosteroids can exacerbate it. Topical cyclosporin A eye drops are effective but are helpful if corticosteroid drop use is limited by adverse effects.

- Topical antibiotic for any superadded bacterial infection
- ***Sytemic***
- Antihistamine, antibiotics & sun glasses for photophobia if there.

Type	Reaction	Itching	Discharge	Lymphadenopathy	Fever sorethroat
Viral	Follicular	Minimal	Watery	Common	Present
Bacterial **Non-Gonococci** **Gonococcal**	Papillary	Minimal	Purulent Mucopurulent Hyper-Purulent	Uncommon	May or may-not
Chlamydial	Follicular	Minimal	Mucopurulent	Common	No
Allergic	Papillary	Severe	Watery mucoid	None	No

Allergic Conjunctivitis(Non-infective)

- Seasonal allergic conjunctivitis
- Perennial allergic conjunctivitis
- Vernal keratoconjunctivitis
- Atopic keratoconjunctivitis
- Giant papillary conjunctivitis
- Keratoconjunctivitis sicca (dry eyes)
- Superior limbic keratoconjunctivitis (SLK)

. Chemical or irritative conjunctivitis

Vernal keratoconjunctivitis

- **Introduction**
- Vernal keratoconjunctivitis(VKC) is an important sight-threatening, chronic, inflammatory disease of the cornea and conjunctiva characterized by recurrent flare-ups of ocular surface inflammation, causing intense ocular symptoms of itching, redness, and photophobia associated with corneal damage and impairment of visual function and quality of life.
- VKC is a clinical form of allergic conjunctivitis diseases, together with seasonal and perennial allergic conjunctivitis (AC) and atopic keratoconjunctivitis (AKC) .

- In fact, VKC shares some clinical features and pathogenic mechanisms with other forms of AC, including ocular itching, swelling, redness, and conjunctival papillary reaction associated with immunoglobulin (Ig) E-mediated release of histamine and other allergic reaction mediators from mast cells.
- However, this is likely not the only mechanism involved in VKC immunopathogenesis, as only 50% of cases of VKC show allergic sensitization. Several studies have demonstrated that the inflammatory reaction occurring in VKC also involves a lymphocyte T-helper (Th) type 2-driven reaction, a late-phase allergic reaction with eosinophil infiltration, and extracellular matrix remodeling
- In addition, the demographical, geographical, and clinical characteristics of VKC suggest that other endocrine, environmental, and/or genetic factors may play a role in the pathogenesis of this challenging condition.

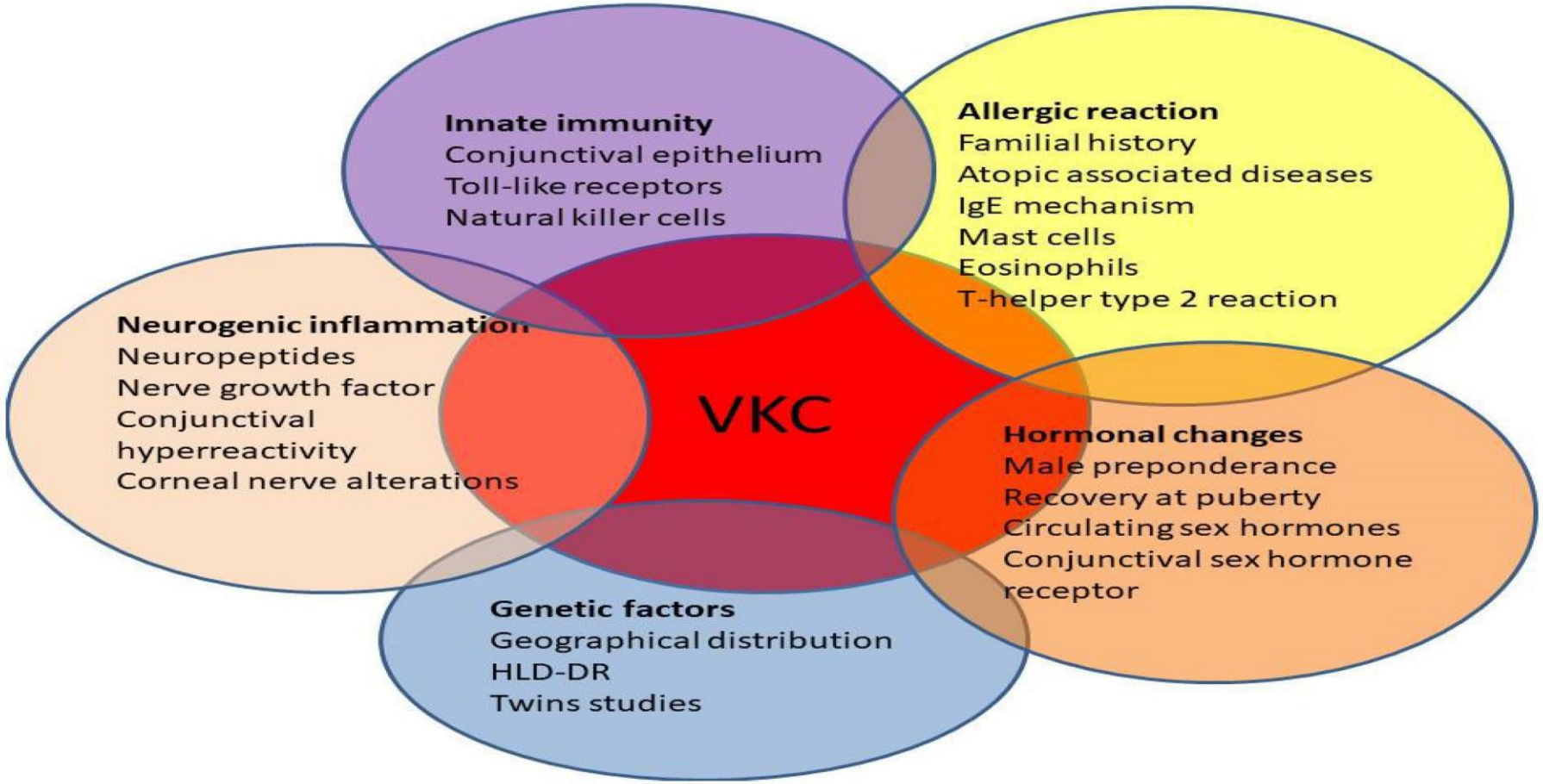
- Is an allergic conjunctivitis to an exogenous allergan which act as an antigen
- It’s a type I & type IV hypersensitivity reaction.
- Age. 5-15 yrs
- Sex. male are common as female
- Season. common in summer & spring
- Family Hx. 2/3 have family Hx

- Specifically, VKC mostly affects children, with higher propensity in boys than girls, and, in most cases, spontaneously resolves after puberty, suggesting that an imbalance of sex hormones may play a role in its pathogenesis.
- A higher prevalence of the condition is observed in warm regions, such as the Mediterranean area, Central and South America, Japan, Central and West Africa, and the Middle East, which also suggests a potential pathogenic role of genetic and/or environmental factors.
- Finally, recent studies point to a possible role of innate immunity, including toll-like receptors (TLRs) and natural killer (NK) cells, in the development and severity of

Pathology

- Exogeneous allergan act as an antigan & induce the immunoglobulin production (IgE).
- Antigen antibody reaction occur over the surface of the mast cell- causes degranulation – release of chemical mediators- serotonin, histamine & slow release substance P- causes inflammation in conjunctiva & cornea

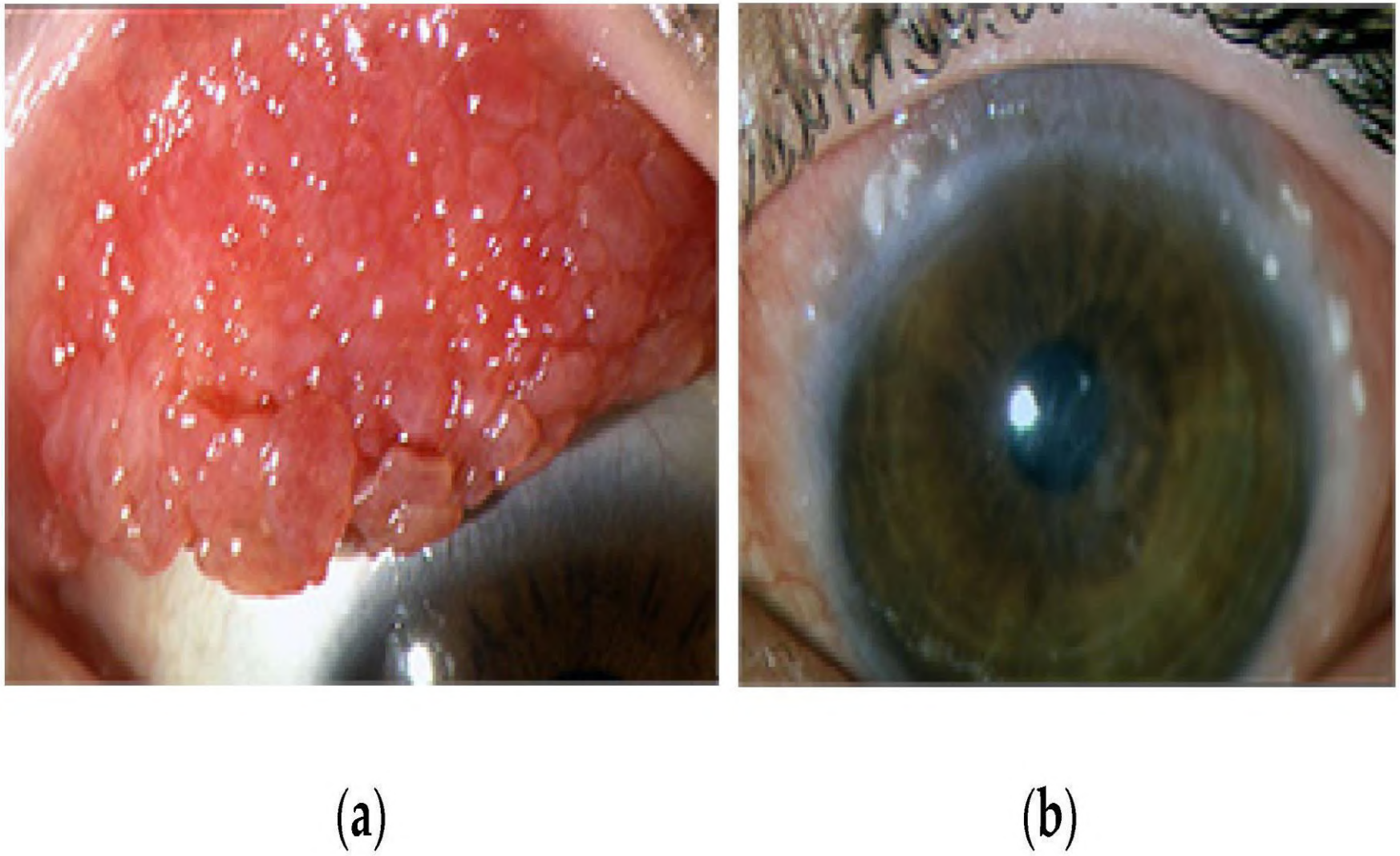
Figure. Allergic reaction represents the main pathogenic factor of VKC; however, several sources of evidence show that innate immunity and neuroinflammatory response, as well as genetic, hormonal, and environmental factors, also participate in the development and severity of VKC



Types

- Three types
- A clinical characteristic sign of VKC is the presence of a giant conjunctival papillary reaction of the upper tarsal conjunctiva and/or limbal region resulting from chronic inflammation and extracellular matrix remodeling.
- Palpebral/tarsal VKC is characterized by giant hypertrophic papillae at the upper tarsal conjunctiva with a cobblestone appearance,
- Bulbar/ limbal form is characterized by gelatinous infiltration around the cornea. in which the bulbar conjunctiva is mainly involved
- Mixed type in which both the tarsal & bulbar conjunctiva are involved & shows papillary reactions.

- Vkc
- A palpebral type
- B Bulbar type

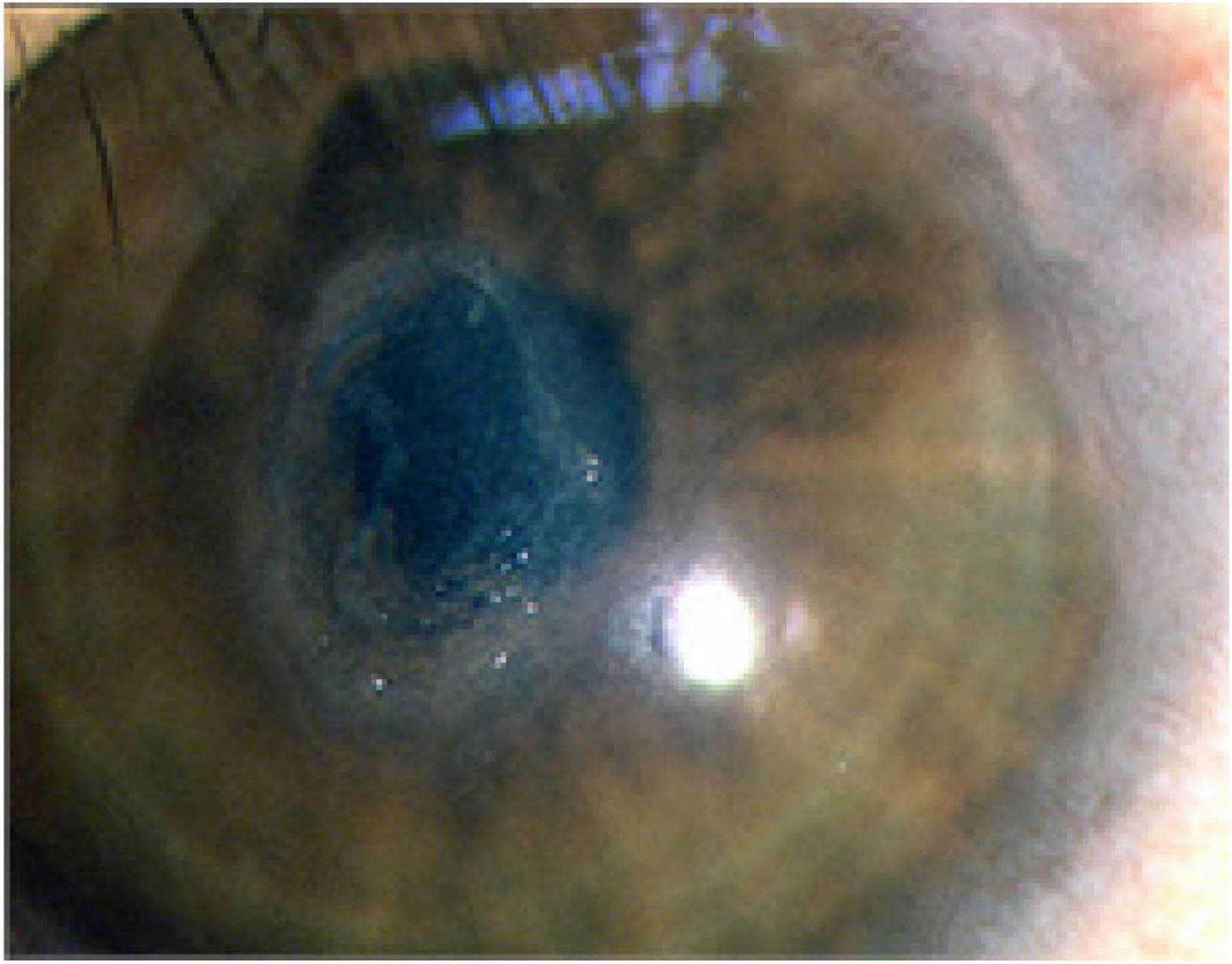


(a) (b)

- Horner-Trantas dots surrounding the cornea represent a typical sign of active inflammation and are mostly due to eosinophil infiltration.
- Corneal epithelial defect
- More severe cases, with long-standing disease, may develop corneal neovascularization and scarring, associated with permanent impairment of visual function

- Corneal ulcers are
- reported in 3–11%
- of cases and may
- cause pain and
- impairment

of visual function



Management

- A challenge for ophthalmologists.
- Cold Compressing
- **Antiallergic** eye drops such as antihistamines and/or mast cell stabilizers are effective only in very mild cases.
- Mast cell stabilizer such as sodium cromoglicate, lodxamide
- Antihistamine such as emedastine, epinastine
- **Topical steroids** The majority of patients in the active phase of ocular inflammation require the use of topical steroids and/or other immunosuppressive drugs. Topical steroid are very effective in controlling the signs and symptoms of active disease. However, their chronic use is associated with the development of severe ocular complications, such as glaucoma and cataract.

- **Immunosuppressive,** Therefore, steroid sparing agents, such as topical cyclosporine A (immunosuppressive) (CsA) or topical tacrolimus another immunosuppressive +antibiotic, are currently used for the chronic treatment of VKC
- Drops & ointment form are used
- **Supratarsal steroid injection,** in resistant & non-complianace condition is given . Dexa & triamcinolone
- Systemic antiallergic & steroid can be added

Complications

- Keratoconus
- Glaucoma may be due to steroid use
- Cataract formation may be due to steroid use
- Corneal defect like shields ulcer, scaring, vacularization

Chronic conjunctivitis

- Trachoma

Poor hygiene

overcrowding

Scenario

- A female patient of 17 yrs comes to opd. She is complaining of redness, discharge for the last 2-3months. Her vision is 6/6 Bes. There is mild conjunctival congestion with follicles on lid eversion. She took many treatment but no relief. On Hx she has poor background with many family members lives in small house. What is the most probable cause.?
- A acute allergic conjunctivitis
- B acute bacterial conjunctivitis
- C acute viral conjunctivitis
- D trachomatous conjunctivitis

- Dx D

Chlamydial conjunctivitis; Trachoma

- It is a chronic conjunctivitis caused chlamydia trachomatis
- It is usually bilateral, may be unilateral
- Caused by Chlamydia Trachomatis type A,B, & C
- Incubation period is 5-12 days
- Common in poor communities with overcrowding & poor hygiene
- Contagious with personal contacts
- Spread through conjunctival secretion
- Leading cause of preventable blindness

Transmission

- *C. trachomatis* spread through direct contact. Infected young children serve as a reservoir of infection.
- The bacteria are then transmitted by close physical contact with family members and other caregivers.
- The bacteria are also spread through shared blankets, pillows, and towels. The
- Bazaar fly *Musca sorbens* lays its eggs in human feces that can be contaminated with trachoma bacteria.
- These flies pick up bacteria on their bodies and can transmit them to humans.

- ***Certain conditions promote the spread of trachoma bacteria. These include:***
- a) poor personal hygiene
- b) poor body waste and trash disposal
- c) insufficient water supply for washing
- d) shared sleeping space with dirty hands and common face towels & clothes/shawls
- e) close association with domestic animals

The Life Cycle of Trachoma

INFECTING THE EYES

Flies carrying the micro-organism land on children's eyes, to feed on discharge.

FAMILY CONTACT

Women who take care of children also get the infection.

SPREADING OUT

Flies that breed in human feces spread the disease to others.

Dirty hands or face cloths also spread the disease.

HOW TRACHOMA BLINDS

Infections inflame and thicken the upper eyelid.

Scarred eyelids turn inward.

The lashes scratch the cornea, leading to blindness.

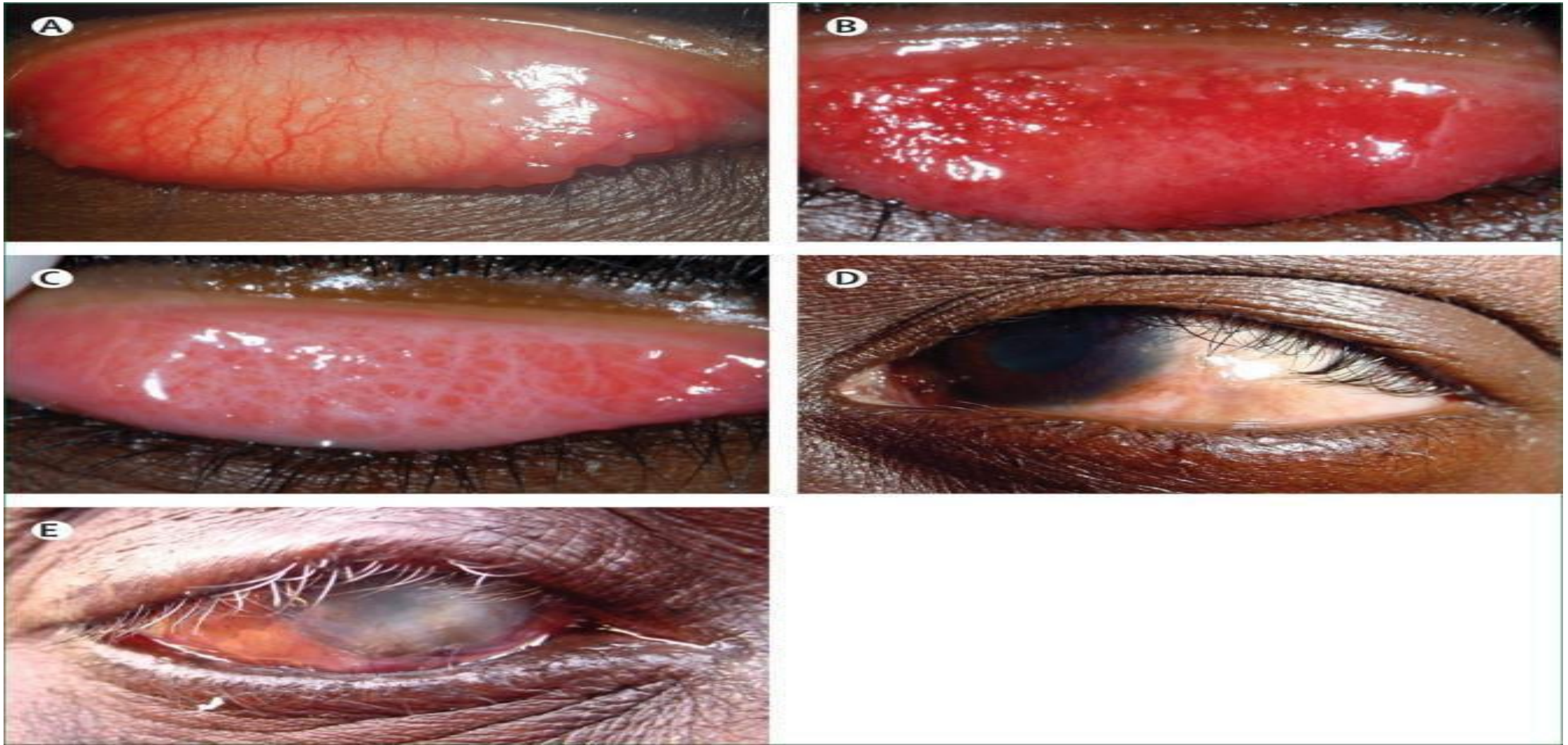
Sources: The Carter Center, International Trachoma Initiative

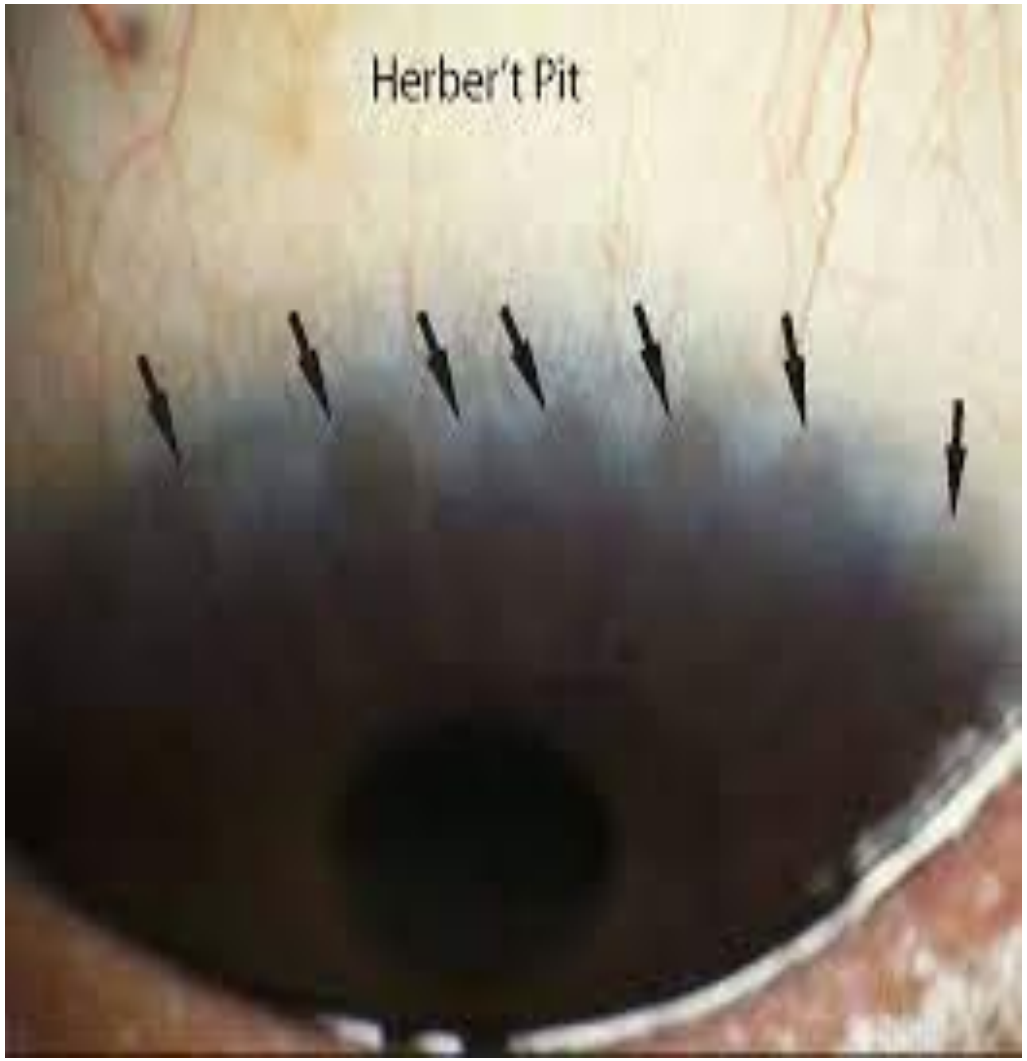
Al Granberg/The New York Times

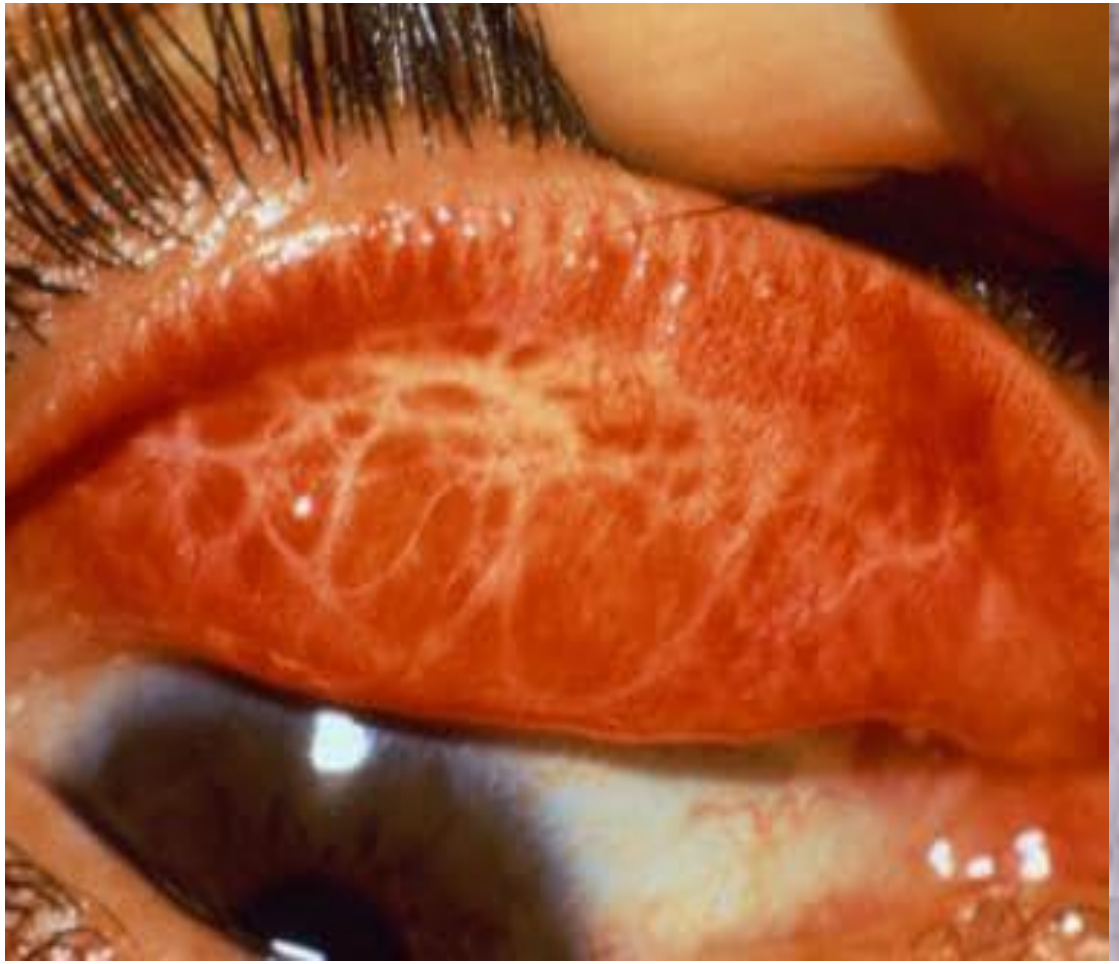
Clinical feature (WHO 1987) (FISTO)

- According to grading important
- 1. *Follicular* stage 0.5mm or more
- 2. *Inflammatory* stage with intense inflammation of the tarsal conjunctiva with more than 50% vessel obscuration
- 3. Conjunctival *Scarring*
- 4. *Trichiasis* is the stage in which eyelashes becomes misdirected touching the cornea
- 5. Scarring stage with *corneal opacity* formation leading to blurring and loss of vision

Clinical features of trachoma. (A) Active trachoma in a child with follicle formation(TF) (B) intense inflammation (TI). (B) Tarsal conjunctival scarring (TS). (C) conjunctival scarring (TS) (D) Entropion and trichiasis (TT). (E) corneal opacity CO with entropion and trichiasis (TT).




Herber't Pit



Treatment (SAFE) policy

- Surgery of the complications
- Antibiotic use topical & systemic
- Face wash means improve hygienic condition
- Environmental improvement , to increase cleanliness, water supply, to improve poverty and living condition and sanitation of the public

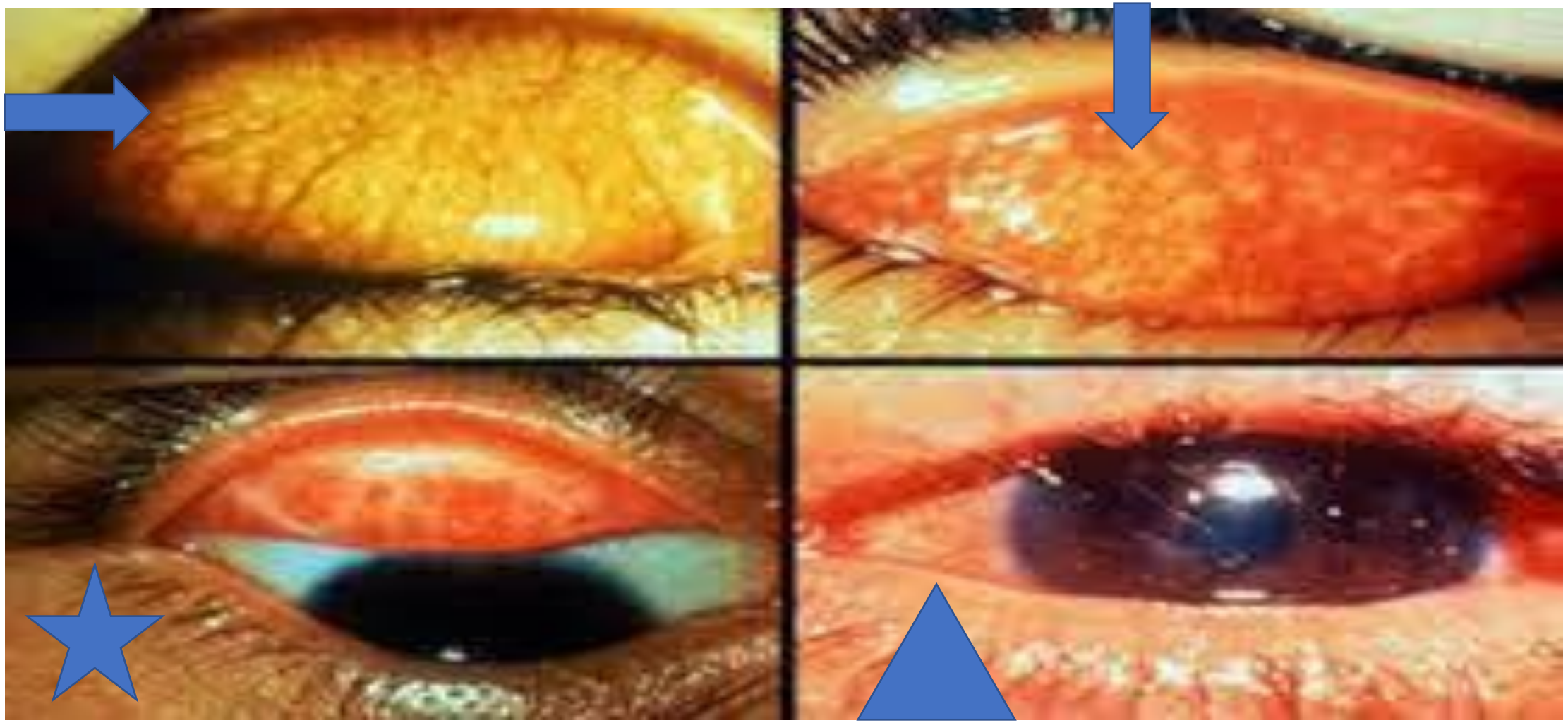
Safe strategy; surgery(S), antibiotics(A), face wash(F), environment improvement(E)

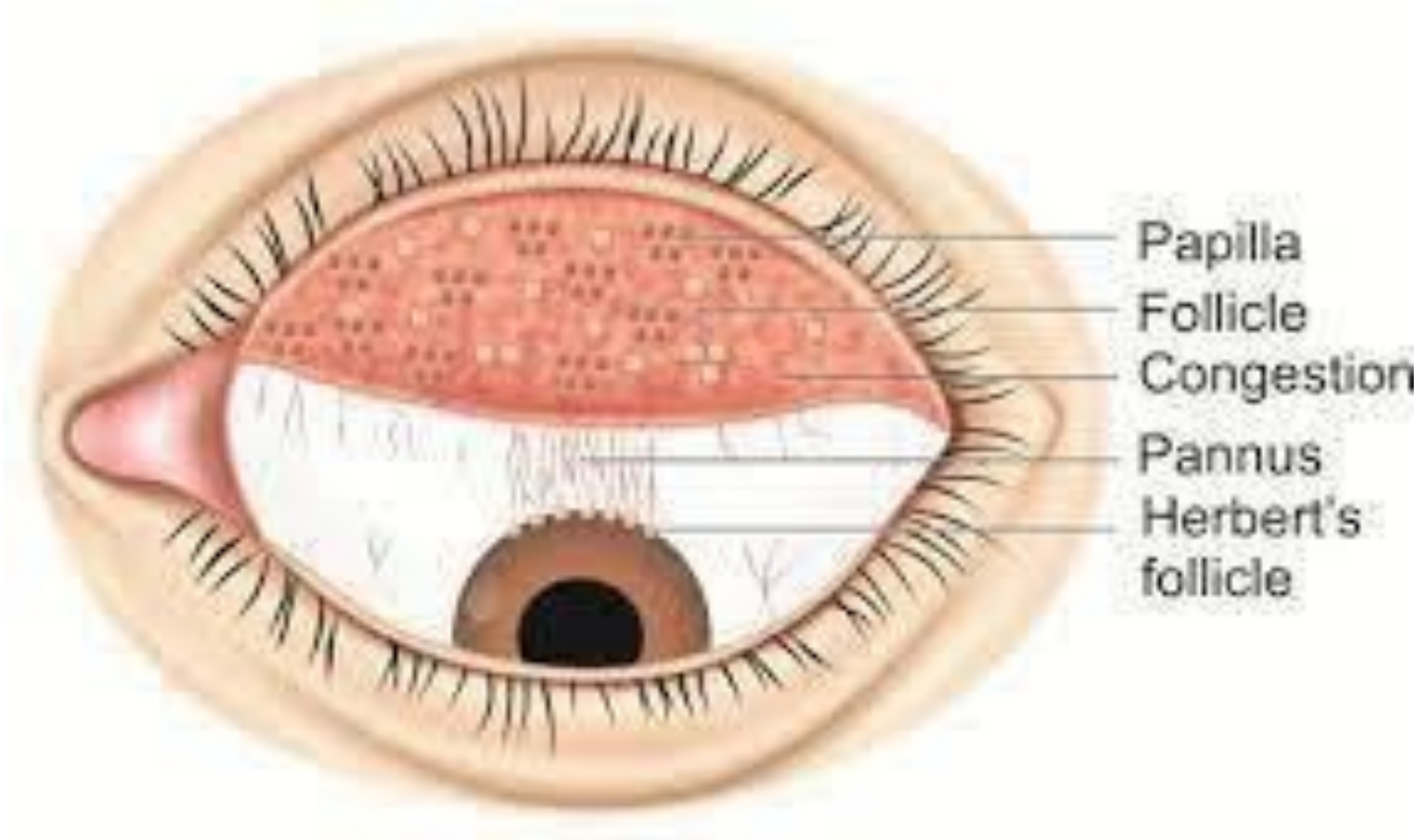
Clinical features (Mac Callan 1908)

- Four stages
- Follicular hyperplasia
- Papillary hypertrophy
- Conjunctival scarring
- Corneal involvement

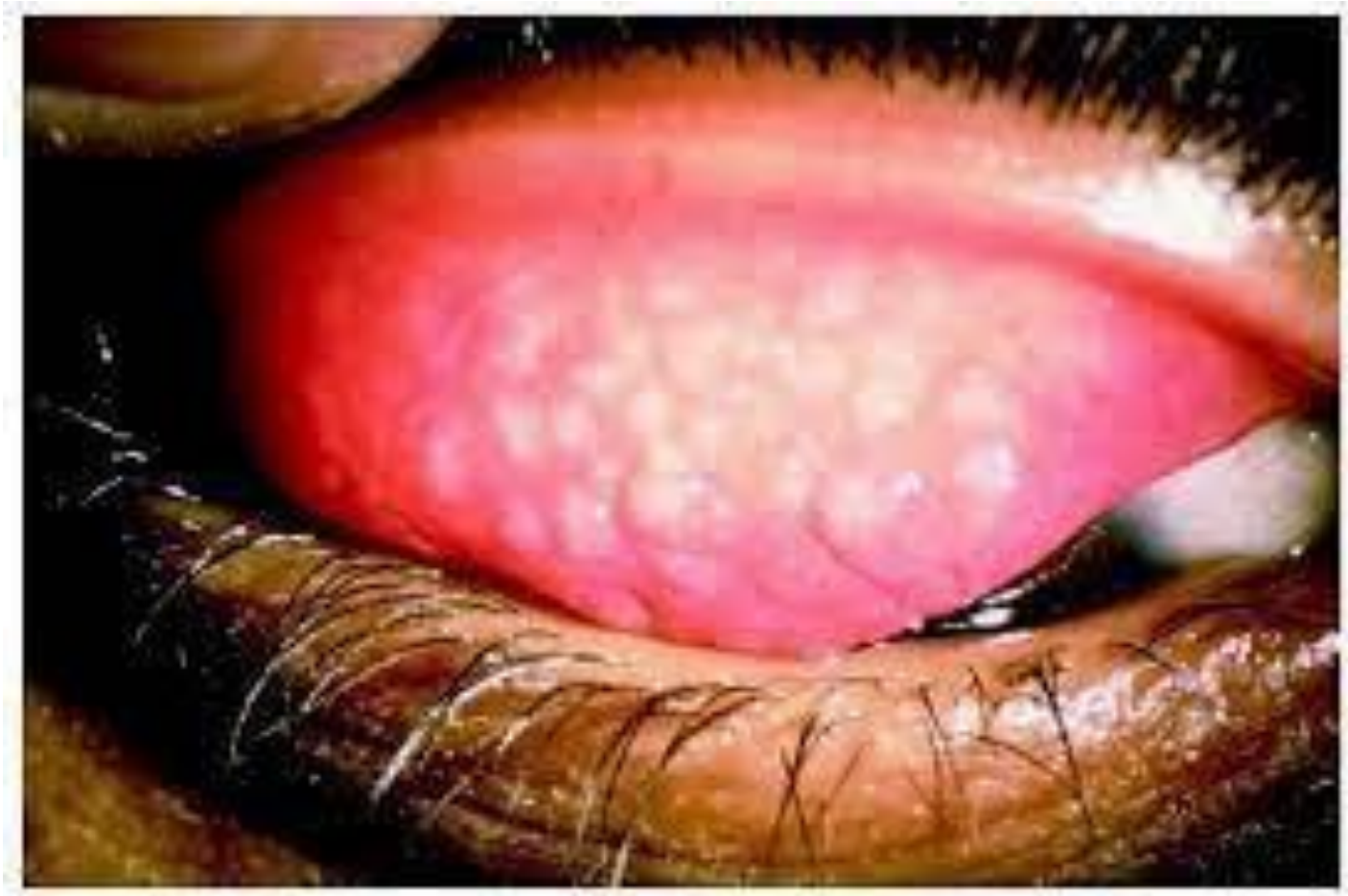
- Stage 1 with minimal symptyoms & low conjunctival reaction
- Minimal discharge
- Follicle are there in up tarsal conjunctiva & immature
- Corneal involvement +_
- No other complications
- Stage 2 with watering photophobia foreign body sensation
- Follicule formation 0.5 - 5mm
- Intense inflammation with papillary hyperplasia which may obscure the follicules
- Corneal pannus formation at the up part of the cornea
- Corneal ulceration may or may not be present

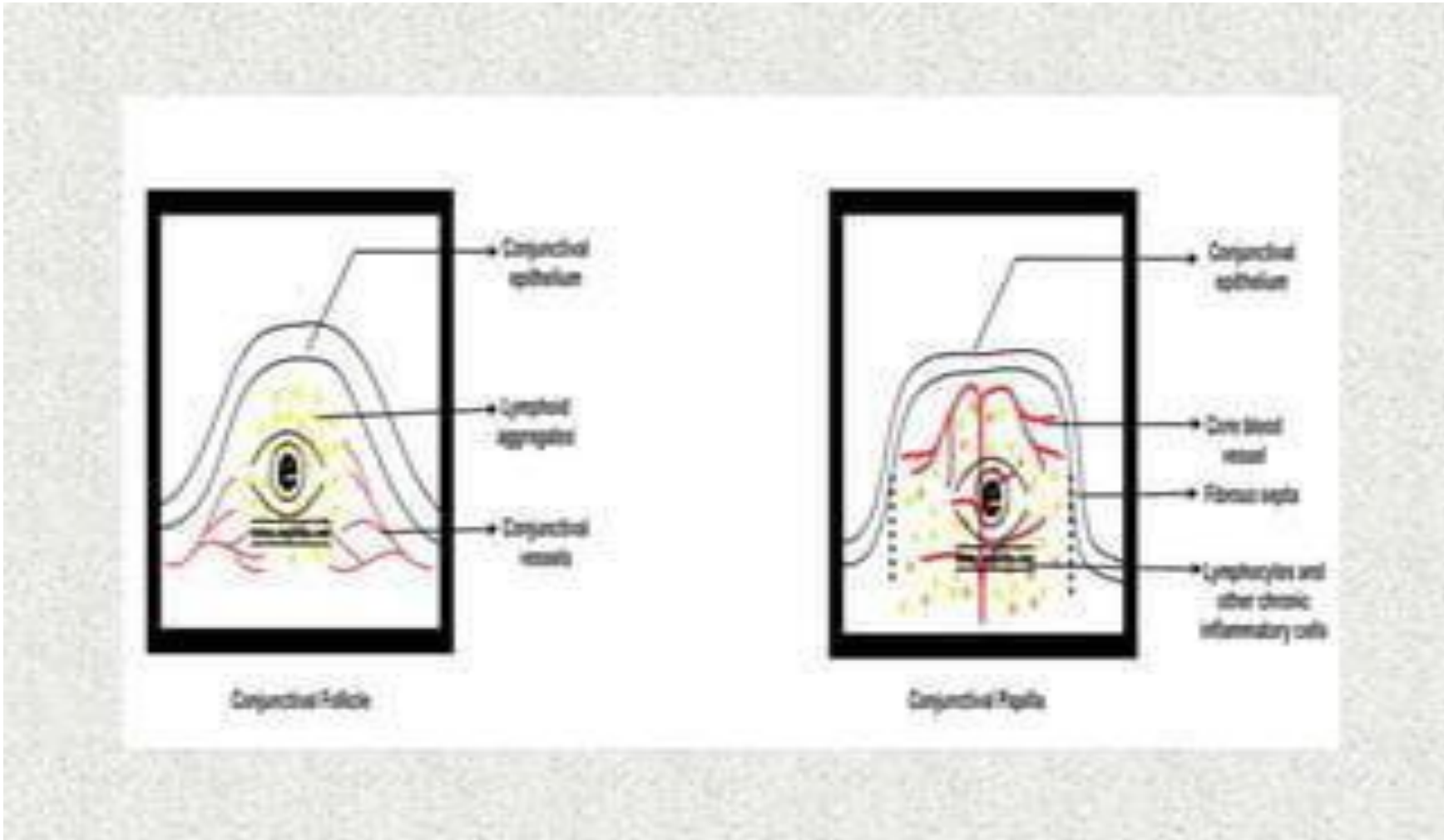
- Stage 3 with corneal scarring still an active stage but conjunctival scarring . The inflammation subsides with necrosis and the follicues ends with linear scarring called Arlets lines
- Herbert's pit are are pits at the corneal limbus due to necrosis of limbus follicules
- Regression of the corneal vascularized pannus with scar formation
- Stage 4 is the complicatins formation. The inflammation is subsided but the complications are due to cicatrization
- Corneal scaing
- Triachiasis
- Entopian formation etc etc




Papilla
Follicle
Congestion
Pannus
Herbert's follicle

- Thanks




Conjunctival epithelium
Lymphoid aggregates
Conjunctival vessels
Conjunctival epithelium
Core blood vessel
Fibrous septa
Lymphocytes and other chronic inflammatory cells

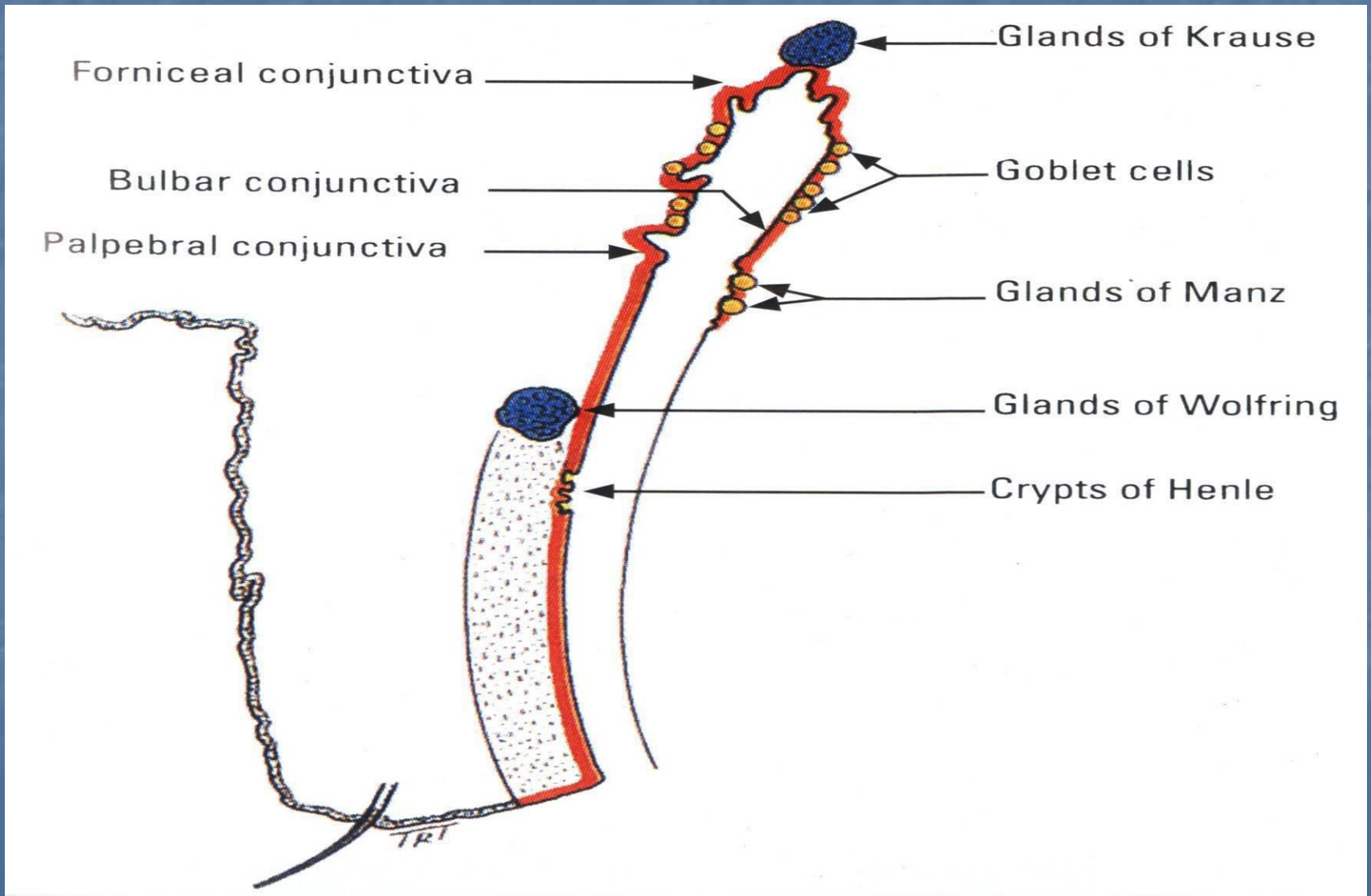
SPREAD OF INFECTIONS

- DIRECT- contact with airborne or waterborne infections
- VECTOR- flies (Musca domestica)
- MATERIAL- most important

CONJUNCTIVITIS

Dr. Afzal Qadir
MBBS, ICO, FCPS

ANATOMY

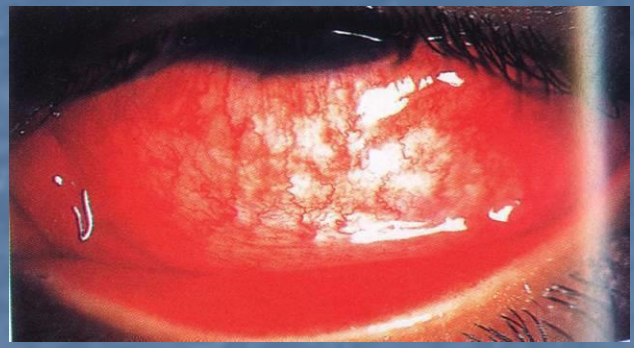

Glands of Krause
Forniceal conjunctiva
Goblet cells
Bulbar conjunctiva
Palpebral conjunctiva
Glands of Manz
Glands of Wolfring
Crypts of Henle

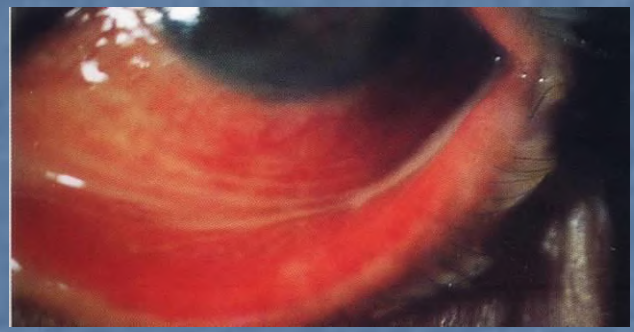
SYMPTOMS

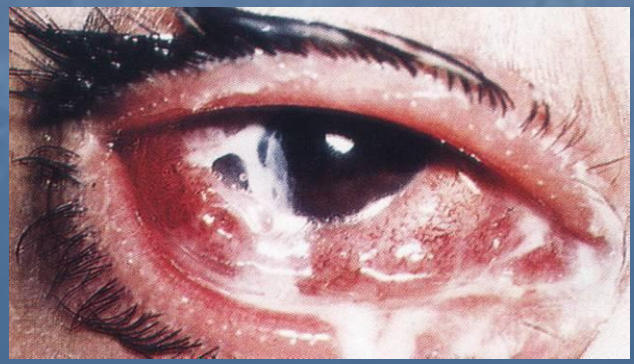
- PAIN & FOREIGN BODY SENSATION
- ITCHING
- LACRIMATION
- IRRITATION
- PHOTOPHOBIA
- STINGING & BURNING

TYPES OF DISCHARGE

- WATERY
- MUCOID
- PURULENT
- MUCOPURULENT

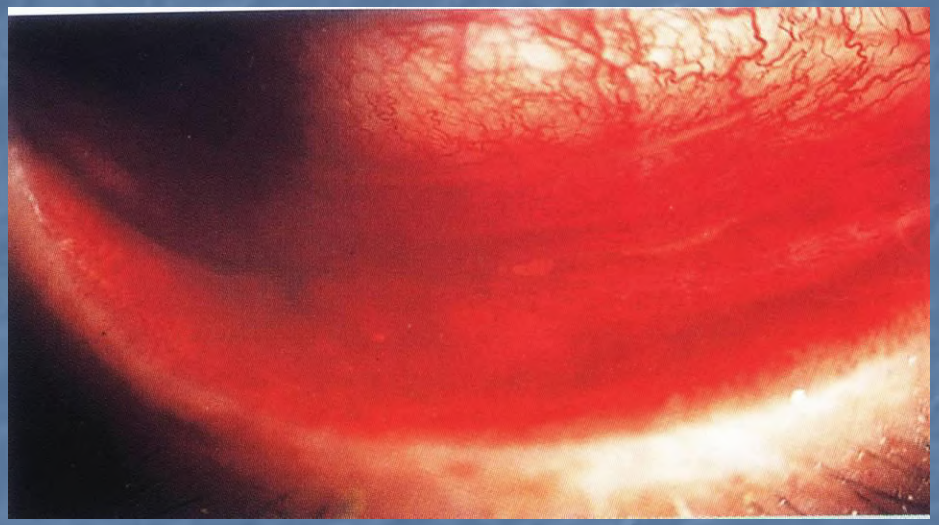




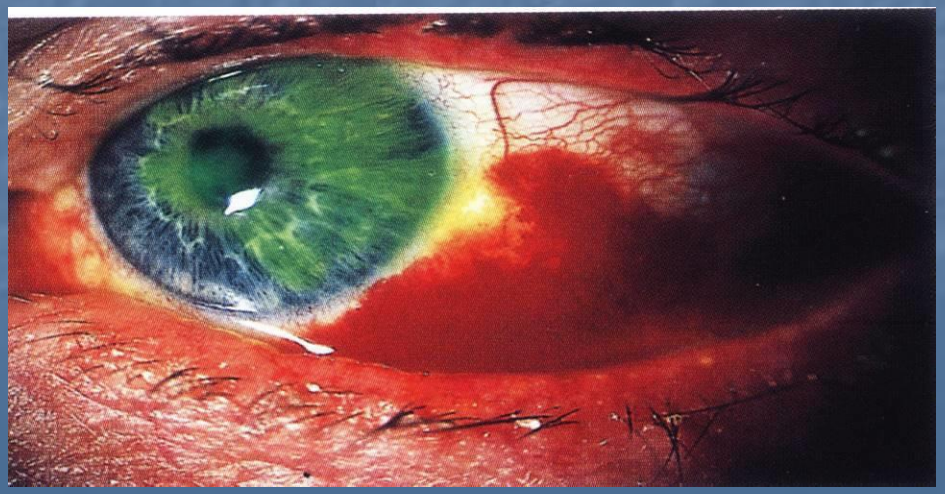


TYPES OF CONJ. REACTION

- CONJ. INJECTION

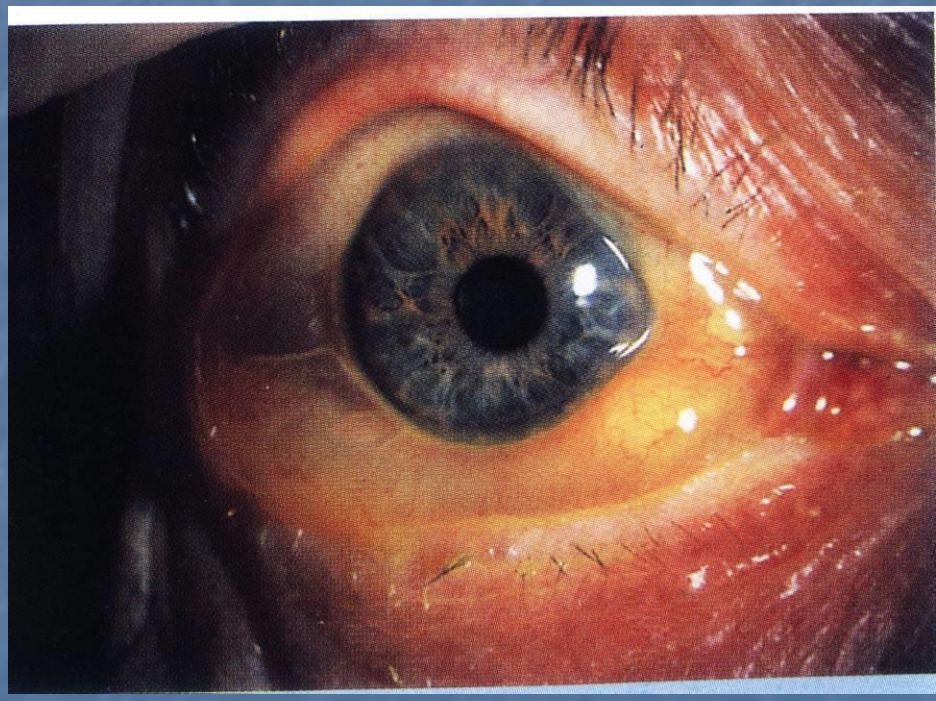


- SUBCONJ. HAEMORRHAGE

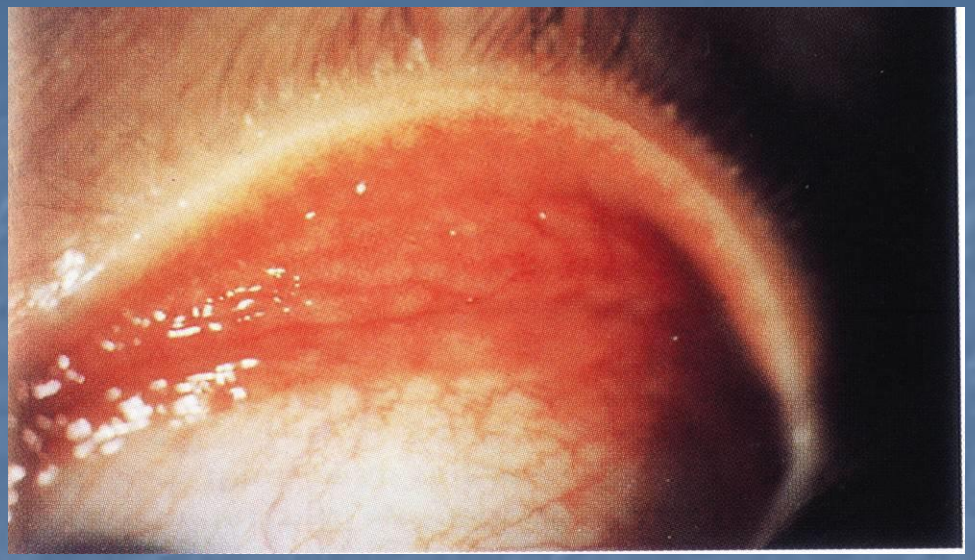


TYPES OF CONJ. REACTION

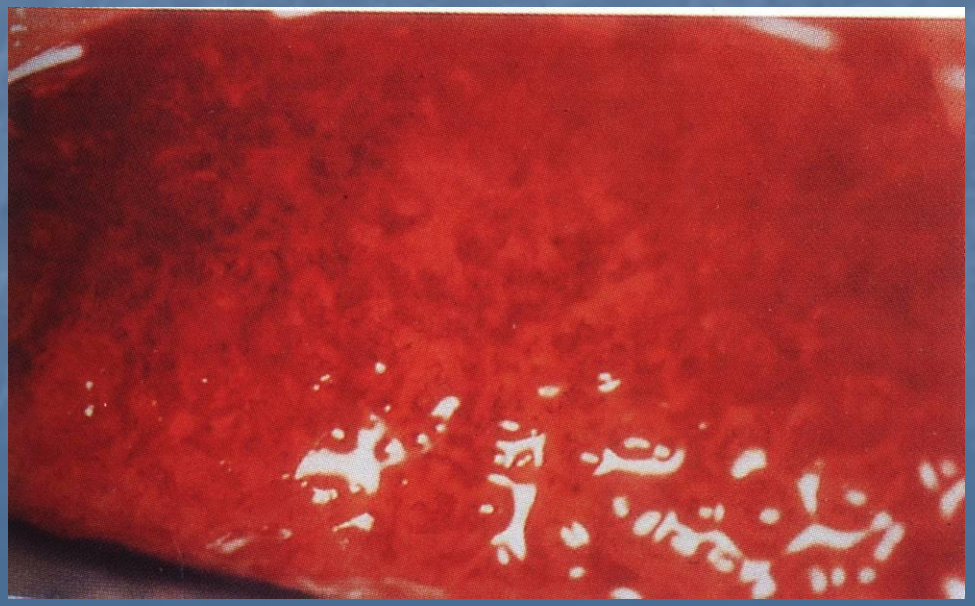
- ODEMA (CHEMOSIS)



- FOLLICULAR REACTION

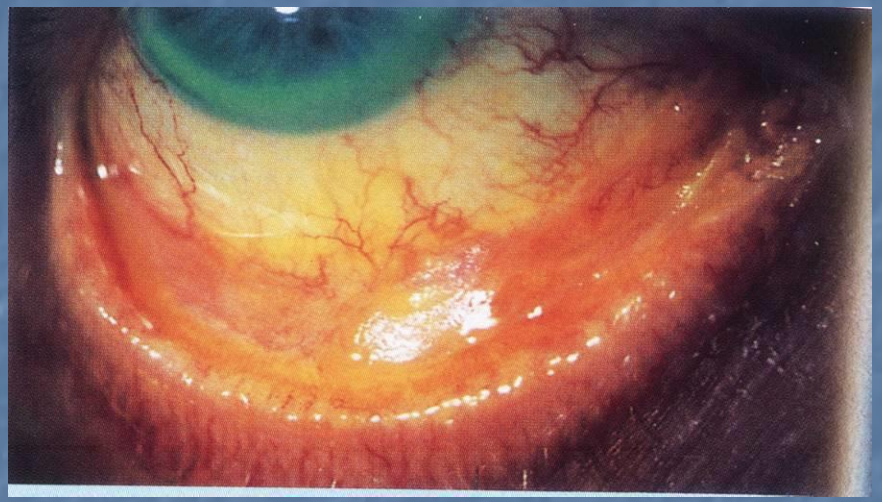


- PAPILLARY REACTION

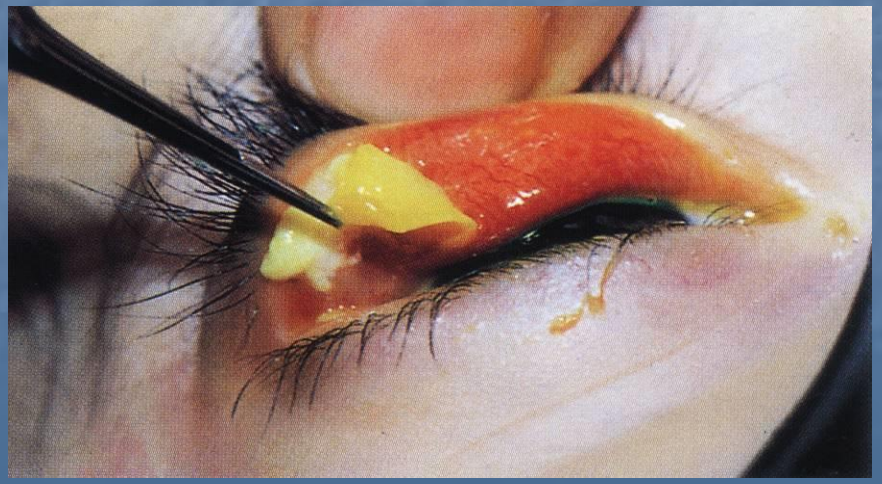


OTHER SIGNS

- CONJ. SCARRING



- MEMB. FORMATION



- LYMPHADENOPATHY

CLASSIFICATION

BASED ON DURATION OF ONSET

- ACUTE
- SUBACUTE
- CHRONIC

CLASSIFICATION

BASED ON TYPE OF EXUDATE

- SEROUS- VIRAL,ALLERGIC,TOXIC.
- CATARRHAL- ALLERGIC
- PURULENT- BACTERIAL,CHLAMYDIAL
- MEMBRANOUS- BACTERIAL
- PSEUDOMEMBRANOUS-BACTERIAL

CLASSIFICATION - AETIOLOGY

- INFECTIOUS
  - BACTERIAL
  - VIRAL
  - CHLAMYDIAL
  - FUNGAL
  - PARASITIC
- NON-INFECTIOUS
  - ALLERGIC
  - IRRITANTS
  - ENDOGENOUS OR AUTOIMMUNE
  - DRY EYE
  - TOXIC (CHEMICAL OR DRUG-INDUCED)
  - SELF INFLICTED/FACTITIOUS
  - IDIOPATHIC

INVESTIGATIONS

- CULTURE SENSITIVITY
- CYTOLOGICAL INVESTIGATION TO IDENTIFY CELLULAR INFILTRATE
- DETECTION OF VIRAL & CHLAMYDIAL ANTIGENS
- IMPRESSION CYTOLOGY
- PCR

TREATMENT

- TREAT THE CAUSE
- RESTORING NORMAL ANATOMY
- LID HYGIENE
- ANTI BACTERIAL THERAPY
- USE OF STEROIDS
- USE OF ANTI-HISTAMINES
- ROLE OF MAST CELL STABILIZERS

BACTERIAL CONJUNCTIVITIS

BACTERRIAL CONJUNCTIVITIS

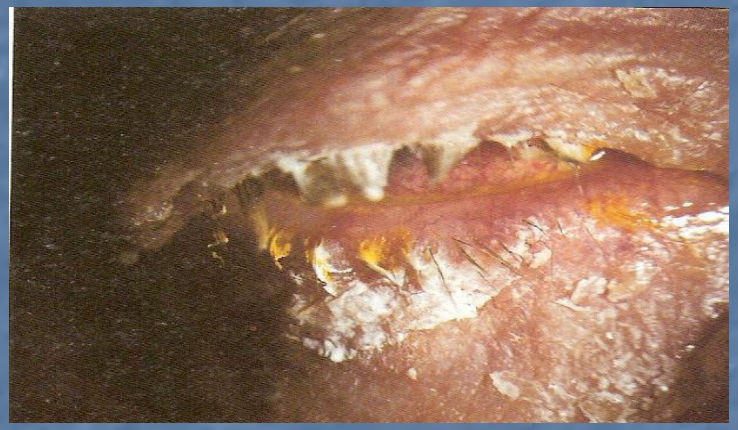
- CAUSATIVE ORGANISMS:
  - Staph Epidermidis
  - Staph Aureus
  - Strep Pneumonia
  - H Influenza
  - Moraxella Lacunata

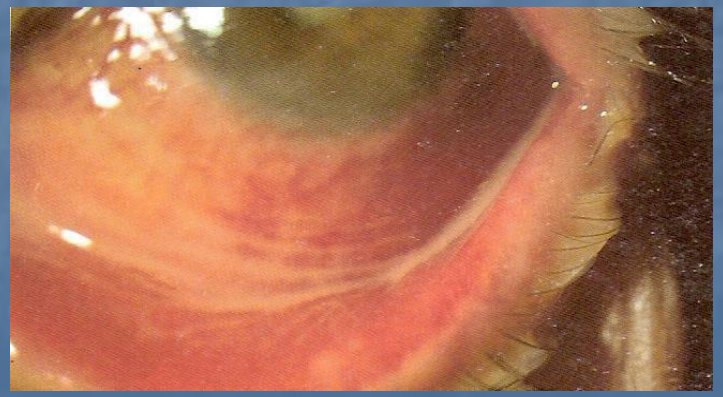
BACTERRIAL CONJUNCTIVITIS

- SYMPTOMS:
  - Common, self limiting disease
  - Redness, grittiness, burning and discharge
  - Eye lids stuck together

BACTERRIAL CONJUNCTIVITIS

- SIGNS:
  - Crusted and edematous eye lids
  - Mucopurulent discharge
  - Velvety beefy red conjunctiva
  - Inflammatory membranes in severe cases
  - PEE and peripheral corneal infiltrates (rare)





BACTERIAL CONJUNCTIVITIS

- TREATMENT:
  - Resolves within 10-14 days
  - Antibiotic drops:-
    - Fusidic acid
    - Chloramphenicol
    - Others (Ciprofloxacin, Ofloxacin, Gentamicin,Tobramycin, Neomycin)
  - Antibiotic ointments:-
    - Gives higher conc for longer durations but blurs the vision
    - Chloramphenicol, Polyfax, Tetracycline

ADENOVIRAL KERATOCONJUNCTIVITIS

- Occupational hazard of Ophthalmologists
- Transmission via respiratory and ocular secretions
- Dissemination by contaminated towels or equipment such as tonometer heads
- Incubation period is 4 – 10 days
- Following onset of conjunctivitis virus is shed for about 12 days

PREVENTION OF TRANSMISSION

- Thorough washing of hands after examining the suspected case
- Meticulous disinfection of ophthalmic instruments
- Infected hospital personnel should not come in contact with patients
- Separate towels for infected persons

CAUSATIVE VIRUSES

- PHARANGOCONJUNCTIVAL FEVER:
- Caused by adenovirus types 3, 4 and 7 and occasionally 5
- Transmitted by droplets
- Typically affects children who also develop upper respiratory tract infection
- Keratitis develops in 30% of cases

- EPIDEMIC KERATOCONJUNCTIVITIS:
- Caused by adenovirus types 8 and 19
- Infection is transmitted by hand to eye contact, instruments and solutions
- Keratitis is severe and develops in about 80% of cases

CONJUNCTIVITIS

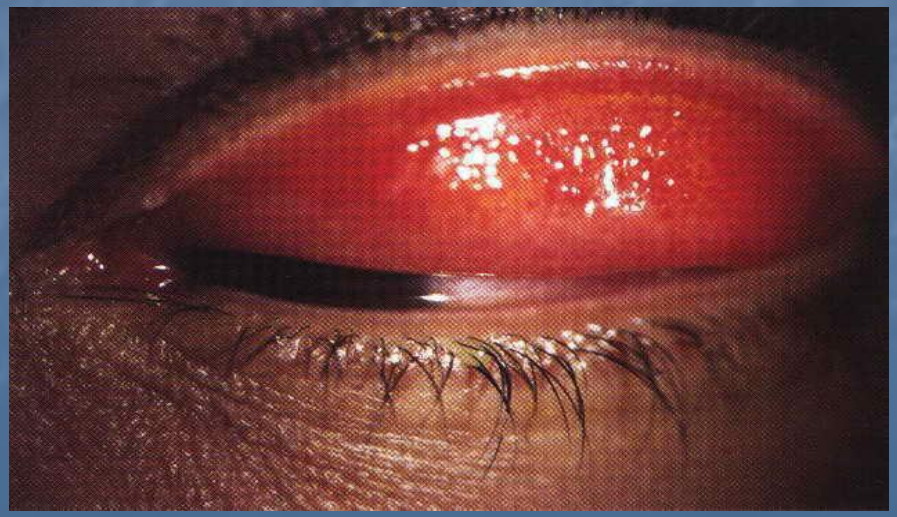
- PRESENTATION:

- ACUTE WATERING, REDNESS, PHOTOPHOBIA AND DISCOMFORT

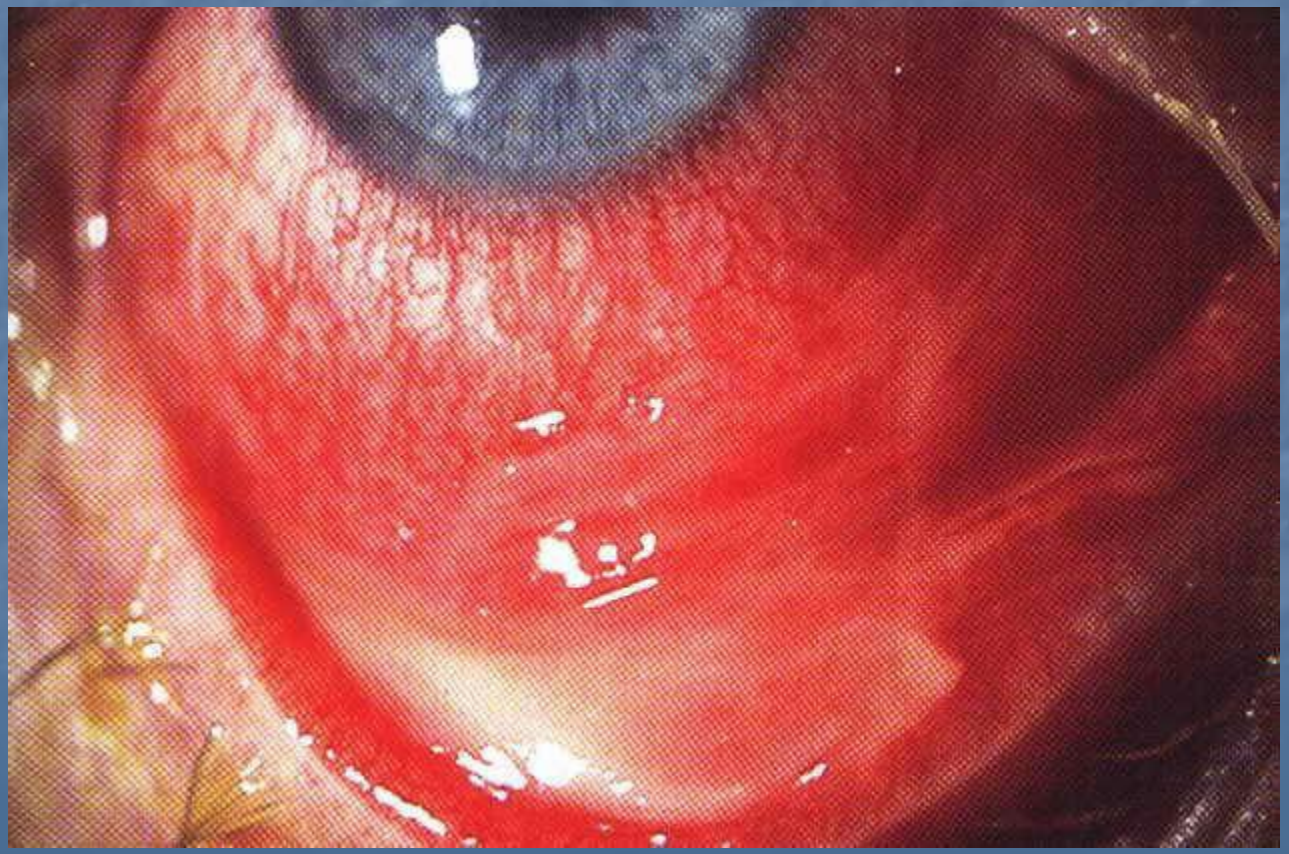
- SIGNS:

- EYELID OEDEMA

- WATERY DISCHARGE AND CONJUNCTIVAL FOLLICLES



- Subconjunctival heamorrhages
- Chemosis
- Pseudomembranes
- Tender lymphadenopathy



TREATMENT OF CONJUNCTIVITIS

- Largely symptomatic and supportive
- Spontaneous resolution occurs within 2 weeks
- Antiviral agents are ineffective and
- Topical steroids to be avoided unless infection is very severe

KERATITIS

- SIGNS:
- STAGE 1:

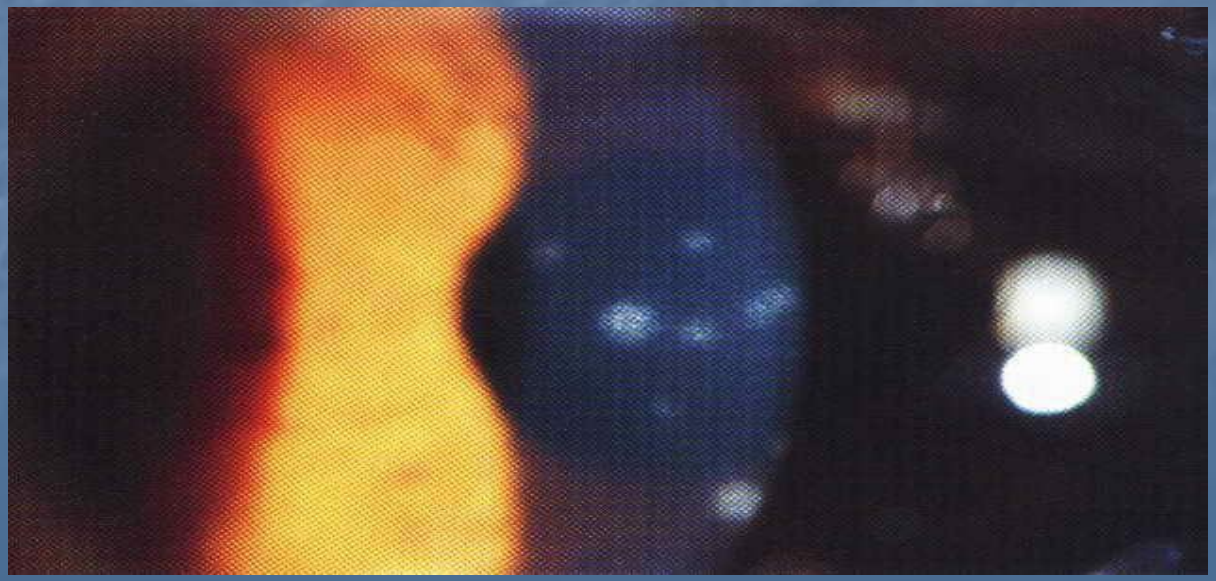
  - Occurs within 7 – 10 days of the onset of symptoms

  - characterized by a punctate epithelial keratitis which resolves in 2 weeks

- STAGE 2:

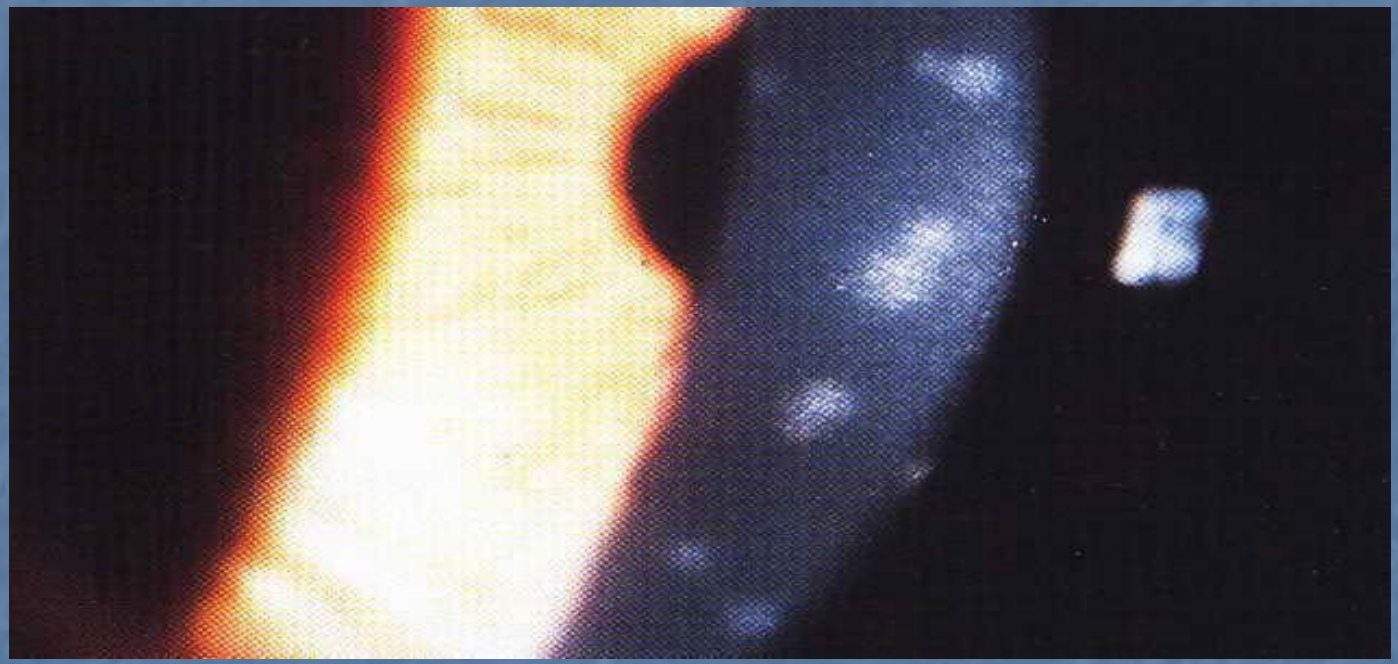
  - Characterized by focal, white, subepithelial opacities which develop beneath fading epithelial lesions

  - They are thought to represent immune response to the virus and may be associated with mild transient anterior uveitis



- STAGE 3:

  - characterized by anterior stromal infiltrates which gradually fade over months or years



TREATMENT

- With topical steroids is indicated only if eye is uncomfortable or visual acuity is reduced
- Steroids do not shorten the natural course of disease but merely suppress corneal inflammation so that the lesions tend to recur if steroid therapy is stopped prematurely


بسم الله الرحمن الرحيم

Red eye 4

Infective Corneal ulcer

Dr Nazullah

Associate professor

Classification

Microbial

- Bacterial
- Fungal
- Protozoal Acanthamoeba
- Viral
- Chlamydia trachomatis

Bacterial keratitis

- Caused by bacteria
- Common by staphy-aureus; strept-pneumonia, Gonococcus, Pseudomonas aeruginosa, Moraxella, Klebsiella & Proteus
- The infection starts when the epithelial integrity is broken either due to trauma or ocular surface disease and the organism gains access into the tissue and proliferates.
- Gonococcus diphtheria & Hemophilus can damage intact epith.
- Start with Blurred vision, pain, redness, tearing, photophobia, foreign body sensations, secretions & discharge

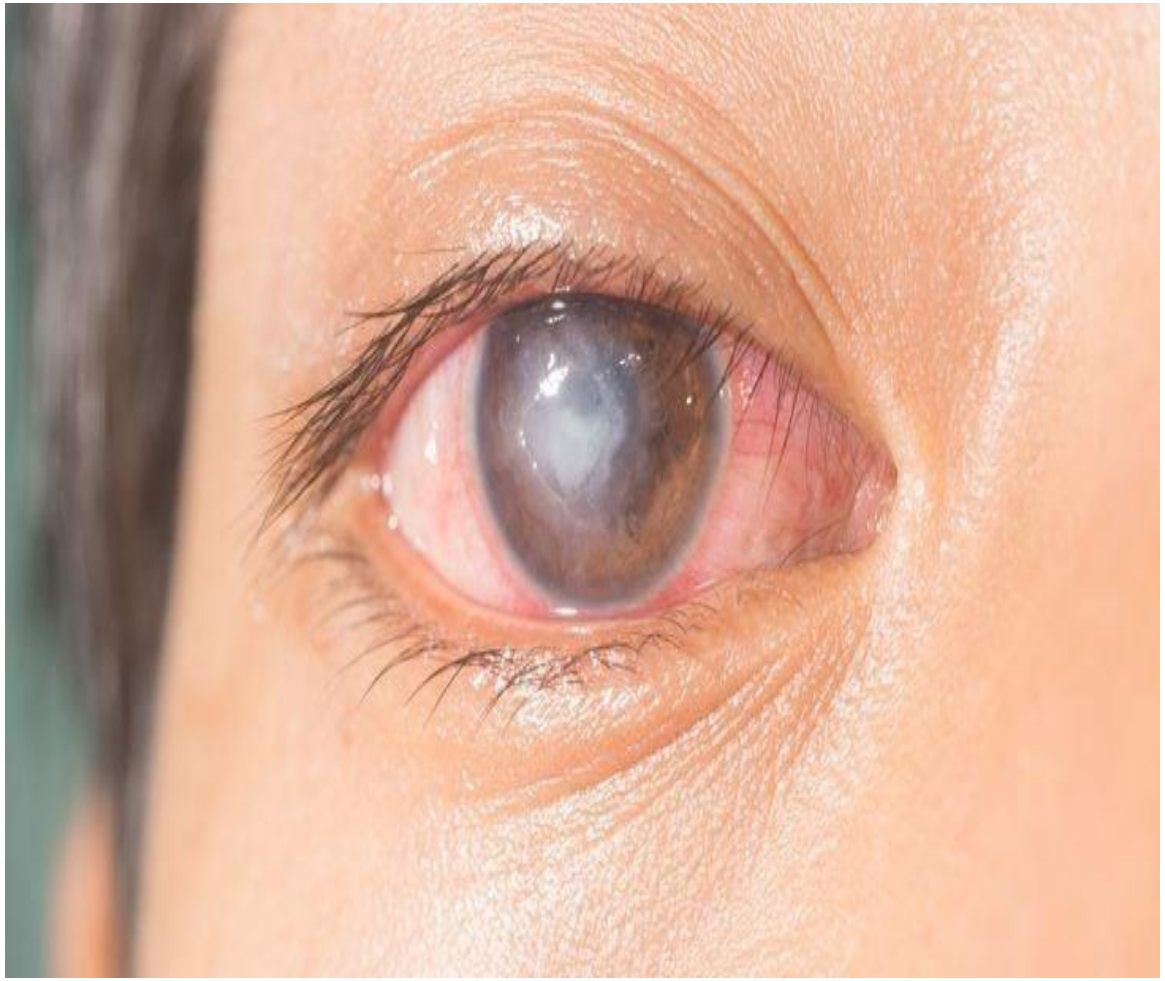
Clinical feature

- There is severe pain, redness, lacrimation, decreased vision photophobia, & blepharospasm ie the patient cannot open the eyes.
- On examination It appears as greyish white swollen cornea with necrosis at the base. Cornea stain positive with fluorescence
- Conjuctinal Hyperemia,
- Hypopyon ie pus formation & exudates in the a/c which is due to increased amount of cellular infiltrates in A/C,
- Anterior uveitis due to inflammation of iris and ciliary body due toxin & cytokines released by these inflammatory cells
- Lid edema may be there

Clinical features

- Infiltrative stage Any injury which damages the epithelium leads to polymorphonuclear attraction leading to yellow white corneal infiltrates with epithelial edema
- Active & necrotic stage There is necrosis & sloughing of the epithelium with excavation and ulcer formation. The chemical mediator are released from ulcerated area which produces the features ie congestion discharge hypopyon etc etc

- Regression There may be regression of the ulcer due to host natural protective mechanism. A line of demarcation between ulcerated & normal clear cornea.
- Healing & scarring may start by epithelialization of the ulcer leading to scarring as a result of new stromal lamellae formation by keratocyte.



Management

- Mostly treated in outpatient
- Admission may be needed if
- Large ulcer
- Resistant to the previous med
- From far flange area

Management Contd

- Proper history of any trauma specially nature & time of trauma, any associated ocular disease eg dry eyes etc & ocular medication such as steroid.
- Systemic history of any chronic illness or any medication specially of any steroid or other immunosuppressive medication should be moted.
- Clinical findings detail examination with slit-lamp. If needful then fluorescence staining should be done to confirm the diagnosis. Corneal sensitivity should be checked. The shape, size, & color of the ulcer ie height & width should be measured & documented.

Margins of the ulcer weather clear rounded , feathery margins , satellite lesion should be noted & described.

Then depth weather superficial or deep.

- Anterior chamber(AC) should be focused for depth, reaction ie cells, flare and hypopyon due to increased vascular permeability should be noted. The size ie length & width of hypopyon should be noted daily.

Investigations

- Routine blood cp with ESR urea sugar urine exam
- Swab from Discharge with can be used for examination.
- Corneal scraping specifically from margins & bed of the ulcer, with topical analgesic under microscope, is done by spatula/20 gauge syringe. Care should be taken to avoid perforation
- a) Direct examination under microscope for any fungal hyphae with KOH.
- b) Gram staining for gram positive & negative.
- c) For culture sensitivity in different culture media.

Which Medium for Which Organism?

Culture Media	Specificity
Blood agar	Most bacteria and fungi. Excludes *Haemophilus spp.*, *Neisseria spp.*, and *Moxarella spp.*
Chocolate agar	Fastidious bacteria, aerobic bacteria, *Haemophilus influenzae, Neisseria spp.,* and *Moxarella spp.*
Lowenstein-Jensen media	*Mycobacteria spp., Nocardia spp.*
Loeffler's media	Corynebacteria
Sabouraud's dextrose agar	Fungi, especially *dermatophytes*
Potato dextrose agar	Fungi
Brain-heart infusion	*Streptococci spp., Meningococci spp.*, yeast, fungi
Thioglycolate broth	Aerobic and anaerobic bacteria
Cooked meat broth	Anaerobic and fastidious bacteria
Non-nutrient agar with *E. coli*	*Acanthamoeba*
Viral transport	Viruses (e.g., HSV), *Mycoplasma spp., Ureaplasma spp.,* and *Chlamydia spp.*

- B scan for any doubtful intraocular foreign body
- X ray for any doubtful intraocular foreign body
- Ct scan can b done for any doubtful intraocular foreign body

Treatment

- A) Medical according to the pathogens involved, but broadly the following medication
- Topical antibiotics for infection control like tobramycin gentamycin ofloxacin ciprofloxacin moxifloxacin. Single or in combination, drops & in oint form. Frequency varies from half hrly to four times daily
- Cycloplegic cyclopean to relieve pain , pupil dilatation to prevent synechia formation & reduce exudation by decreasing the vascular permeability.
- Analgesic to relieve pain
- Anti glaucoma to reduce intraocular pressure (IOP) betablocker timolol etc

- Systemic
- Antibiotics oral/iv
- Analgesic oral/iv
- Antiglaucoma to reduce IOP acetazolamide AZM
- B) Bandage contact lens may b applied for mechanical support
- In resistant & non healing cases & very thin cornea

- C) Surgical in resistant & corneal thinning with threatened perforation
- Pressure bandage
- Tarsorrhaphy
- Amniotic membrane transplant AMT
- Conjunctival flape
- Tectonic graft
- PKP

Fungal keratitis

- Common pathogens
- Candida yeast
- Filamentary aspergillosis
- Mucor

Fungal keratitis

Risk factors

- Risk factors include trauma, ocular surface disease, and topical steroid use.
- In warmer climates the rule is that the most common organisms are filamentous fungi, like Fusarium spp and Aspergillus spp. With a strong relationship to trauma.
- More common in debilitated or immunocompromised patients and the causative organism being a Candida, such as yeast
- Fusarium keratitis associated with a type of contact lens solution
- Suspicion should be high in cases of trauma with vegetable matter

Clinical features

- Fungal keratitis was first described by Leber in 1879. Fungal keratitis or keratomycosis refers to an infective process of the cornea caused by any of the multiple pathologic fungi capable of invading the ocular surface. It is most typically a slow, relentless disease that must be differentiated from other types of corneal conditions with similar presentation; especially its bacterial counterpart
- Different fungi including but not limited to yeasts of Candida spp., filamentous spp Aspergillus spp., Fusarium spp., Cladosporium,spp., Curvularia, and non septated such as Rhizopus.
- Bare in mind that any agent capable of infecting humans is a potential infectious agent, especially if the host has a debilitating disease.

C/f contd

- The infection starts when the epithelial integrity is broken either due to trauma or ocular surface disease and the organism gains access into the tissue and proliferates.
- Proteolytic enzymes, fungal antigens and toxins are liberated into the cornea with the resulting necrosis and damage to its architecture thus compromising the eye integrity and function.
-

C/f contd

- Blurred vision, pain, redness, tearing, photophobia, foreign body sensations, secretions related to agr- trauma, ocular surface disease and topical steroid use are all important characteristics to ascertain in the history.
- The filamentary corneal lesions have a white/gray infiltrate with feathery borders. There might be satellite lesions with a hypopyon and conjunctival injection as well as purulent secretions.
- Candida/ Yeast ulcer are plaque-like and slightly more defined, similar to bacterial keratitis.

Management

- Hx
- Examination
- Investigation
- Treatment

Diagnosis

- Proper & detail History
- Slit lamp examination
- A high degree of suspicion from the physician accounts for early diagnosis and treatment, which are paramount for a successful resolution of the fungal keratitis.
- a) associated ocular & systemic disease, b) steroid or other immunosuppressive medication Corneal ulcers unresponsive to broad-spectrum antibiotics, c) the presence of satellite lesions, and d) scanty secretions in a large ulcer are some signs that should raise flags to the attending professional about the possibility of a mycotic agent.

Investigation

- Corneal scrapping
- For direct examination with KOH for fungal hyphae
- Gram staining
- Culture sensitivity
- B scan for any doubtful intraocular foreign body
- X ray for any doubtful intraocular foreign body

Treatment

- Topical antifungal different preparation
- Natamycin 5% drops
- Fluconazole 2% drops
- Amphotericin B 0.15% drops
- Voriconazole 1-2% drops
- Clotrimazole cream 2%
- The antifungal are fungistatic so it is used for 4 week at least
- Antibiotics for secondary bacterial infection
- Cycloplagic to relieve pain & synechiae prevention

- Systemic different preparation in Pakistan
- Ketoconazole(Nizoral)
- Fluconazole(Diflucan)
- Itraconazole (sporonox/ Icon)
- Used at least for 4-6 weeks
- Antibiotic mix with antifungal in a case of any doubt

- Mechanical debridement
- Keratoplsty

Acanthamoeba keratitis

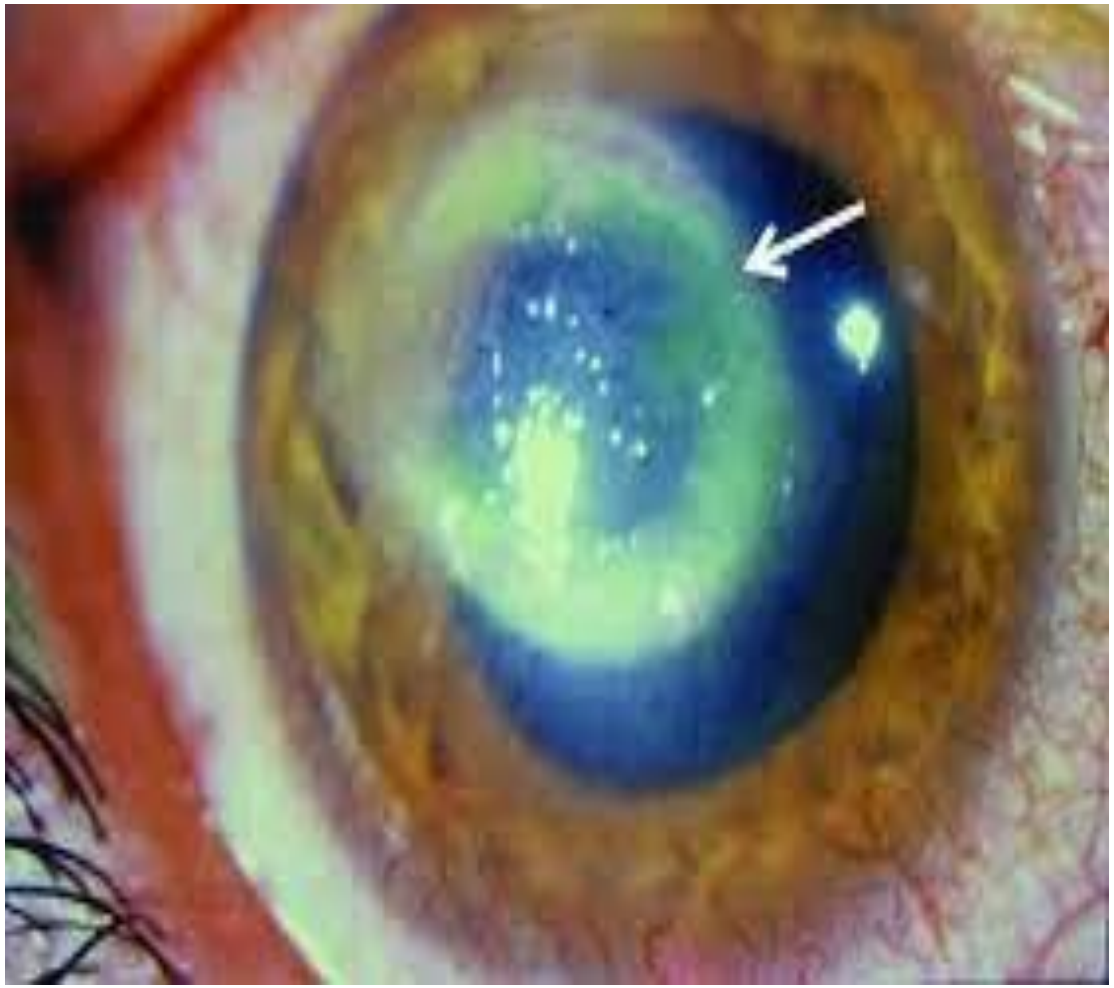
- Acanthamoeba is free living protozoa living in fresh water & soil
- Exist in active trophozoite or dormant cystic form, resistant to kill by freezing , desiccation & chlorination
- It can lead to Very serious & blinding ocular condition
- Needs urgent treatment
- Admission

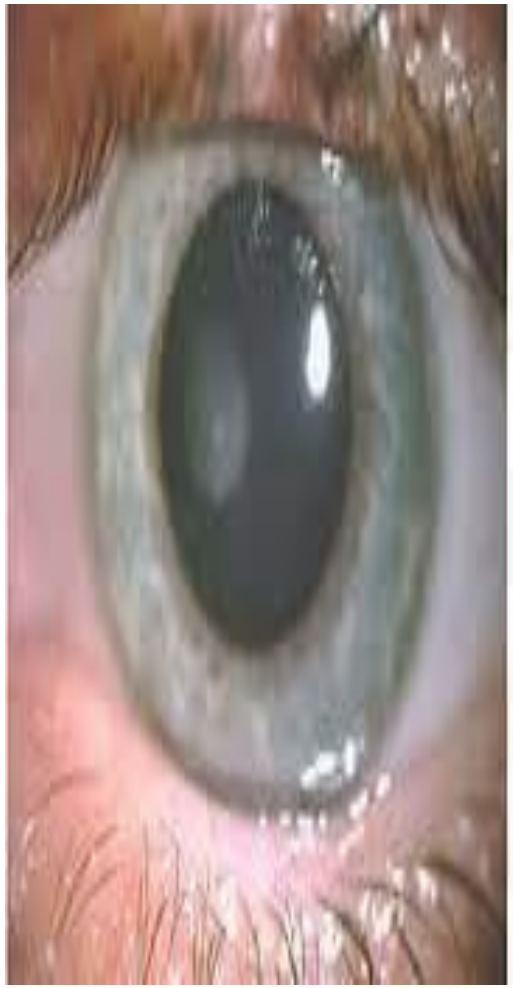
Risk factors

- Contact lens wear
- Ocular trauma
- Swimming pool

Symptoms & signs

- Blurred vision
- Severe pain as compared to the lesion ie out of proportion pain
- Epithelial keratitis diffuse or psuedodendrites like herpes
- Ring infiltrate & abscess
- Characteristic perineural radial infiltrates & abscess formation with enlargement of corneal nerves are diagnostic signs
- Corneal melting may occur





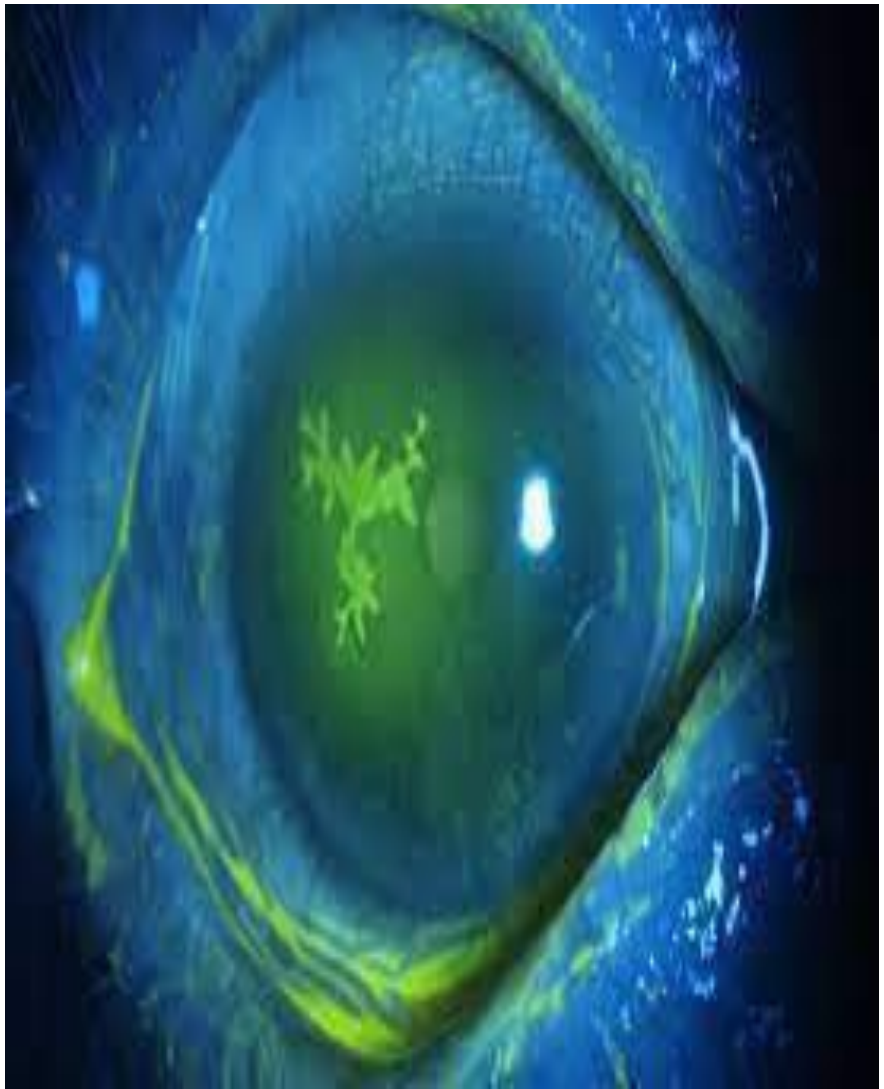
Diagnosis

- Typical history features & finding
- Investigation
- Corneal scrapping for
- A direct examination with periodic Acid Schiff(PAS) or Calcofluor white stain which have affinity for Acanthamoeba cyst & Fungai
- B gram staining cyst
- C culture in non-nutrient agar with E-coli overlay
- Corneal biopsy

Treatment

- Epithelial scraping to remove the organism
- Aminoglycoside neomycin
- Chlorhexidine 0.02%
- Voriconazole
- Polyhexaquinidine biguanide
- Propamidine isethionate(brolene)
- Keratoplasty Pkp in nonresponsive & proressive

- A male age 45 yrs come to eye opd with itching foreign body sensation watering & blurred vision Lt eye for last 15-20 days. On examination his vision is 6/12 Lt eye & 6/6 in Rt eye. There is a corneal lesion with branching pattern & conjunctival congestion. a/c is quiet. Rt is insignificant. He gives hx of fever few days back.
- What is the most probable diagnosis
- A bacterial keratitis
- B fungal keratitis
- C traumatic keratitis
- D viral keratitis



Viral keratitis

- Commonly caused by
- Herpes simplex
- Herpes zoster
- Adenoviral
- Measles
- Mumps

Herpes simplex HSV

- DNA virus
- Common cause infection
- Common cause of corneal scar
- 90% of population are seropositive to herpes simplex
- Two types
- Type i mainly affect above the waist ie face lips etc
- Acquired by droplet infection/ close contact with patients
- Type ii mainly below waist cause by sexual process spread by genital secretion

Primary HSV

- No previous exposure
- First 06month usually no infection, due by maternal antibodies
- Mild flue like & sore throat
- Periocular vesicles
- Conjunctivitis
- Eyelid blepharits

- Antiviral oint

Recurrence

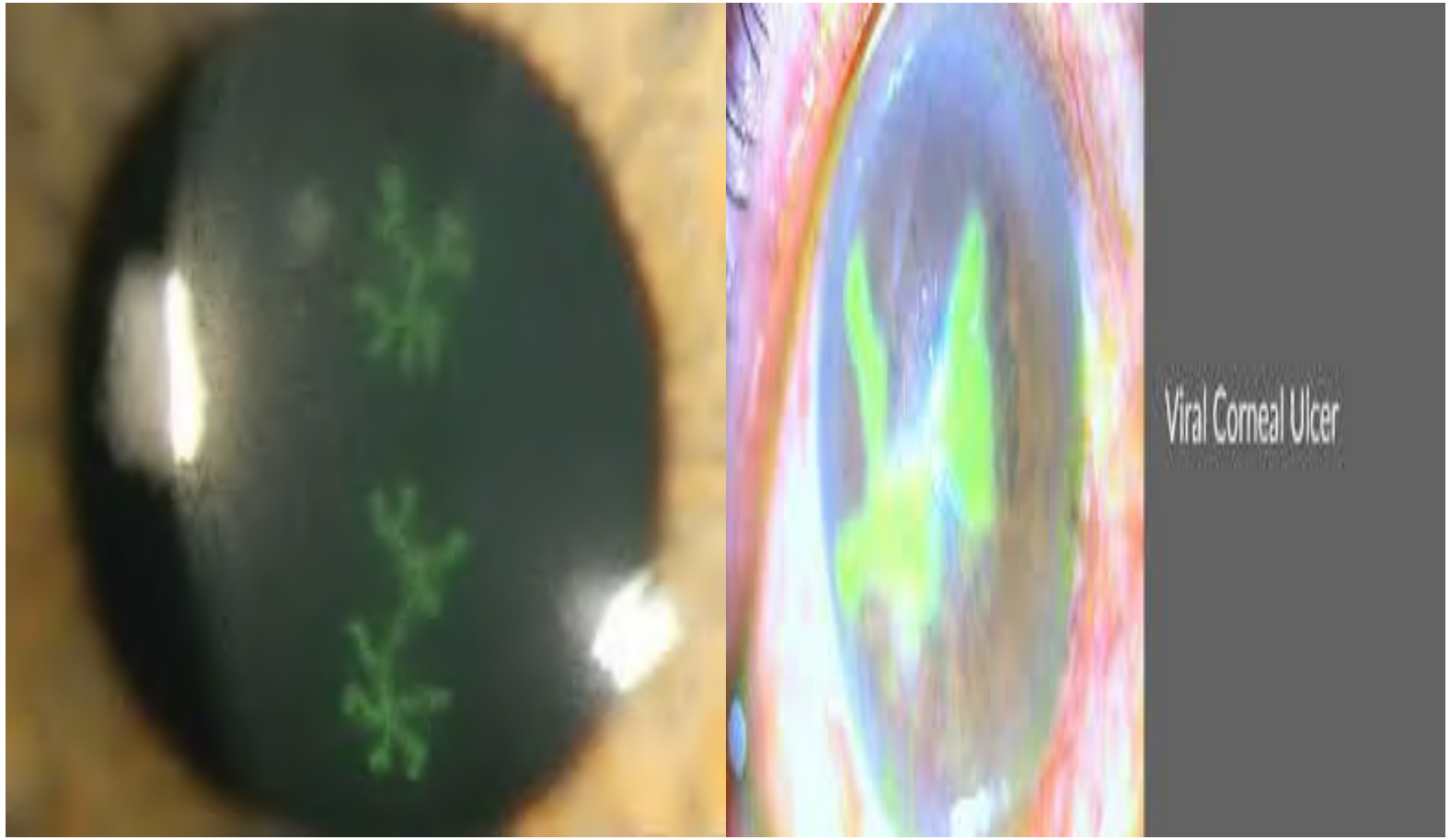
- After primary infection the virus travels along the axon of sensory nerves to the regional gangelion
- Type I to trigeminal gangelion
- Type ii to the spinal gangelion
- Recurrence
- Is due to the reactivation, replication & travels down the nerve to the target tissue causing recurrent infection.

Risk for recurrence

- Fever
- Poor health
- Xposure to sunrays/ultraviolet rays
- Psychiatric disturbance
- Use of steroid

Recurrent HSV

- Dendritic
- When it
- Widens
- It leads to
- Geographical
- Ulcer

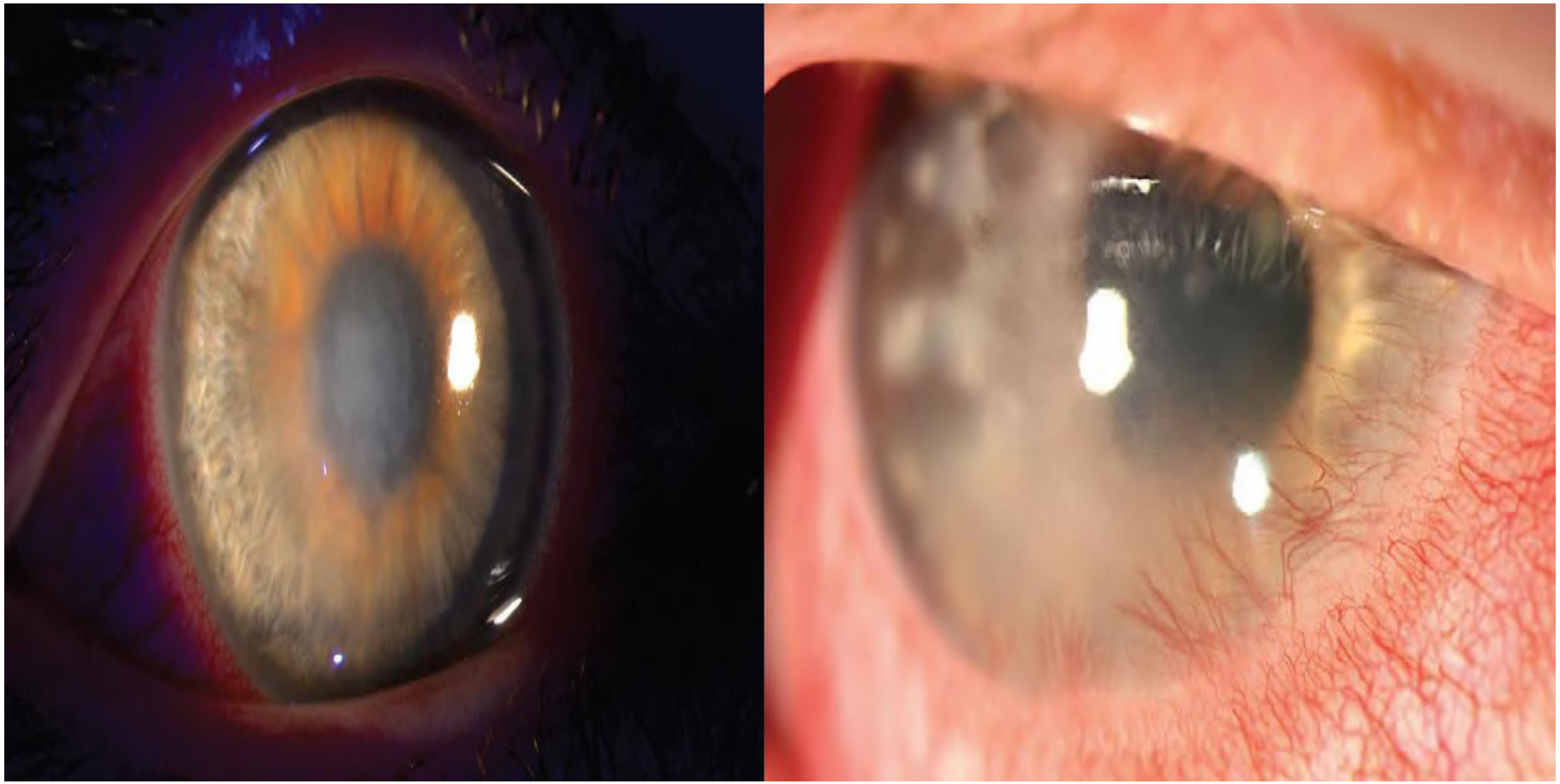


Recurrent kts

Stromal Keratitis

Necrotic stromal Keratitis

keratouvitis



- Under poor health condition the virus in the ganglion, reactivates, replicates travel to the target tissue cornea, causes rec-ulceration
- It damages the epithelial cells coalesce to form linear pattern
- Destruction of the cells--ulceration--further recurrence--further expansion of the ulcer

Clinical features typical feature

- Recurrence
- Foreign body sensation
- Blurred vision
- Mild pain
- On examination
- Branching ulcer
- Stain with fluoresce
- Corneal sensitivity is reduced

Sign

Bacterial	Fungal	Viral
Epithelial defect with large infiltrate and conjunctival injection	Grey or whitish stromal infiltrate with indistinct fluffy margins	Punctate/stellate pattern
Anterior chamber reaction	Feathery branch-like extensions	Linear branching ulcer with or without terminal buds
Hypopyon	Satellite lesions	Reduced corneal sensation
	Hypopyon	

Treatment

- Topical
- Antiviral ointment. Antivral like Acyclogaunosine Acyclovir usually here
- Triflurothimidine drops
- Adenine arabinoside drops

- Antibiotics for secondary infection
- Steroid cautious use to reduce inflammation & soothing
- Cycoplagic to reduces pain
- Systemic
- Analgesics
- Antivral Acylex tab ?? 1gm QID in case of systemic illness & weakness

Stromal keratitis

- It is a localized disc shape stromal greyish edema due to the endothelial infection of the cornea
- It is delayed type of reaction to the viral antigen
- Endothelial damage result in corneal edema due to hydration

- Blurred vision
- Stromal thickening
- Epitheliam is intact
- Corneal sensitivity is reduced
- Kps may be there
- Ant uveitis

Treatment

- Topical antiviral
- Steroid
- Cycloplagic
- Antibiotics

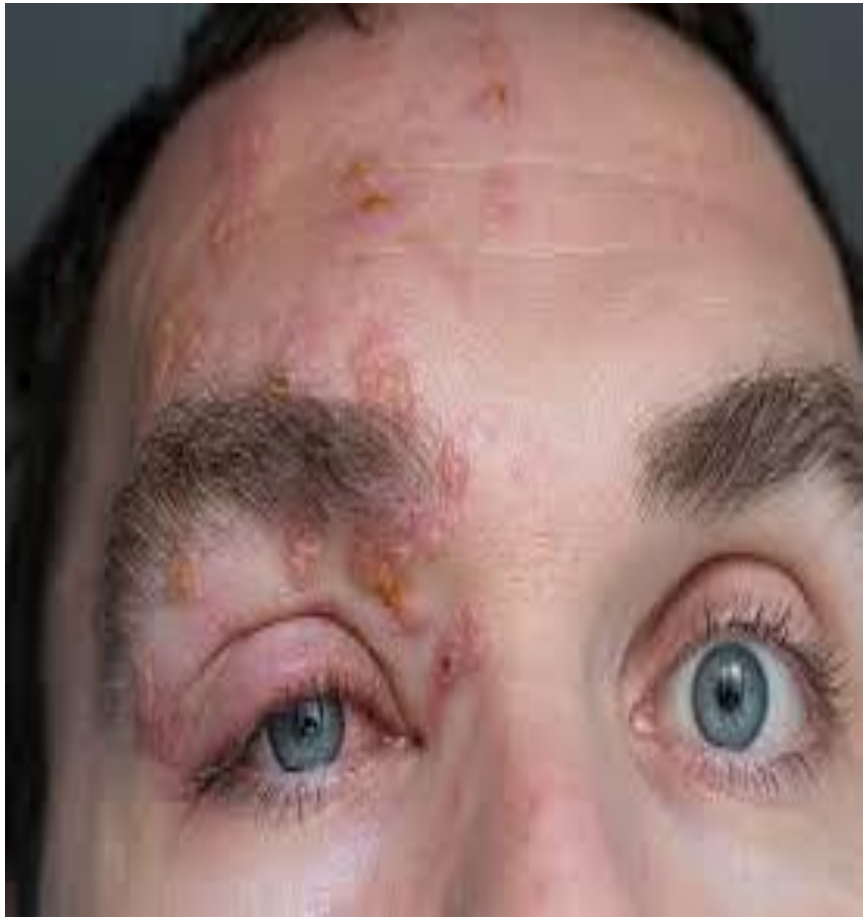
Herpes zoster ophthalmicus HZO

- Caused by the infection of the involvement of the skin of the ophthalmic branch of the trigeminal nerve
- By the human herpes virus 3
- Zoster
- Varicella

- Causing ischemic vasculitis
- Cellular infiltration
- Inflammatory Granulomatous reaction

Clinical features

- Typical features
- Pre-herpetic neuralgia with pain in the distribution of the nerve involved before the appearance of the skin rash
- Skin rash--- maculopapular rash in the painful area of the ophthalmic nerve area
- Vasicular rash develop in 24 hrs ---- leads to pustules & crustes ---- subsides in weeks in pitted scar form
- Edema is there ---in some cases to the opposite side

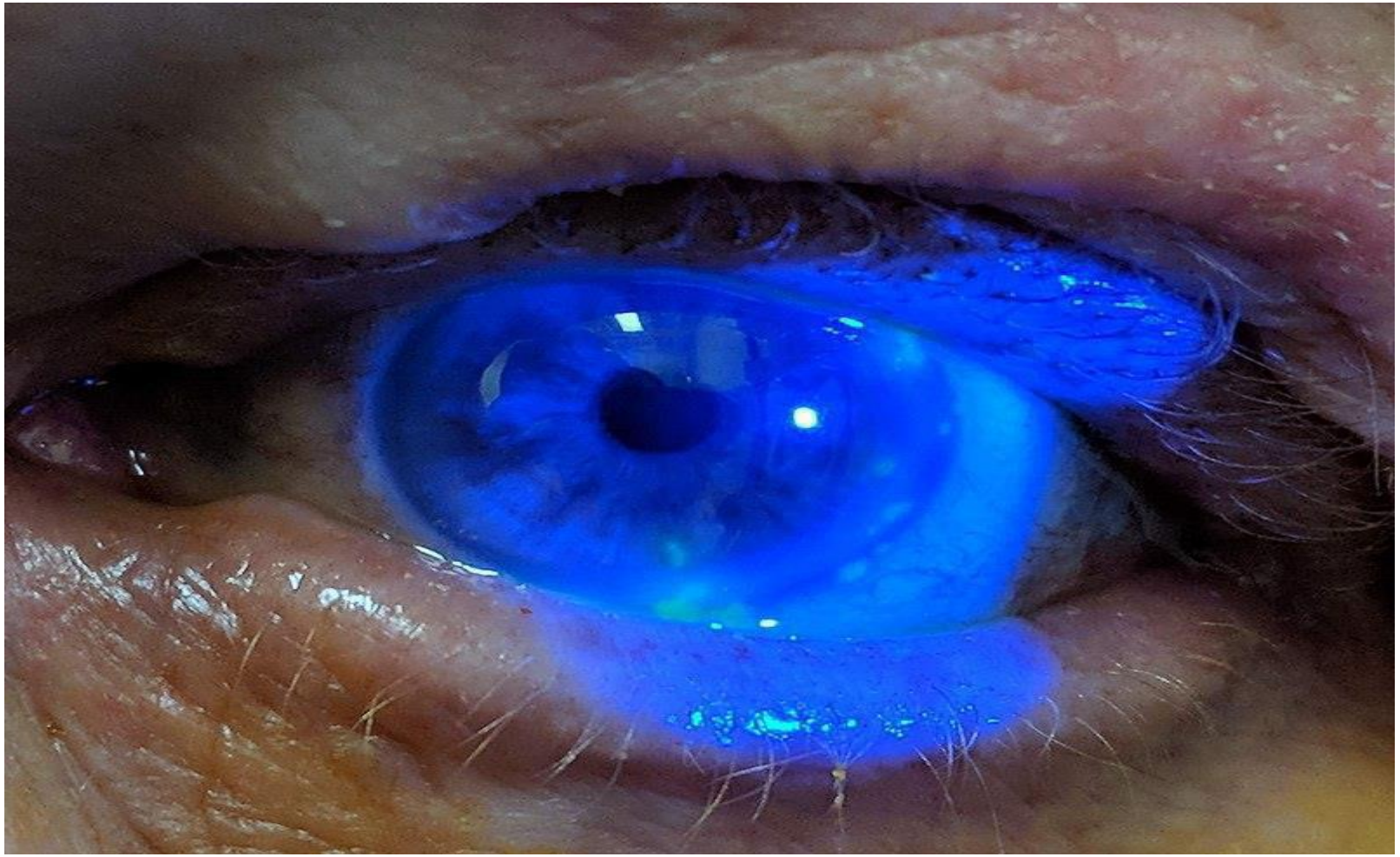



eksudativni eritem
(eryhema exsudativum)
kraste
(crusta)
mehurić
(vesikula)
otok očnog kapka
(oedema palpebrae)

- Post herpetic neuralgia after the rash is subsided pain disappears in few weeks (2-3) but there is continuous pain in some of the cases---persists for years

Ocular lesion different forms

- Keratitis epithelial in 50% of cases
- Micodendritic keratitis
- Filamentary keratitis
- Disciform keratitis
- Anterios uveitis
- Secondary glaucoma due to trabeculitis
- Conunctivitis

- 

Treatment

- Systemic oral antiviral may reduce the duration severity & post herpetic neuralgia
- Start In pre neuralgic episode 1-2 days before eruption starts
- Acyclovir (tab Acylex zovirex) common 500 5times
- Valacyclovir
- Famciclovir
- Anagesic
- Antibiotics
- Steroid

Topical

- Topical skin cream antibiotic steroid combination for skin lesion

- Antibiotic drops
- Antiviral oint
- Cyloplagic
- Antiglaucoma

Table 1: HSV KERATITIS CLASSIFICATION

HSV CATEGORY	COMMON NOMENCLATURE	BASIC TREATMENT APPROACH
Epithelial keratitis	• Dendritic keratitis • Geographic keratitis	Antiviral (topical or oral) or debridement
Stromal keratitis without ulceration	• Interstitial keratitis • Immune stromal keratitis	Topical steroid + oral antiviral prophylaxis
Stromal keratitis with ulceration	• Necrotizing keratitis	Oral antiviral in therapeutic doses + topical steroid
Endothelial keratitis	• Disciform keratitis	Oral antiviral in therapeutic doses + topical steroid

- Thanks

Dr. Yousaf Jamal Mahsood

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Fellowship in Glaucoma (Univ. of Toronto, Canada)
Advance Fellowship in Glaucoma (BPOS, UK)

Assistant Professor Glaucoma
Department of Ophthalmology
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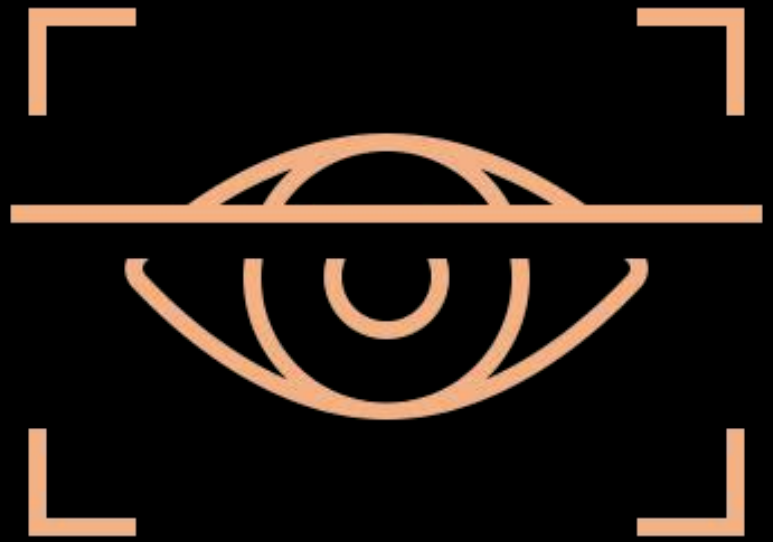


Corneal Ectasia Dystrophies & Degenerations

Learning objectives

Discuss the etiology, clinical features, investigation and management of keratoconus.

Give an overview of corneal dystrophies and degenerations.

Corneal Ectasia

Abnormal shape of the cornea.

- Keratoconus
- Pellucid Marginal Degeneration
- Keratoglobus

Keratoconus

- Keratoconus is a progressive disorder.
- Cornea assumes a conical shape secondary to stromal thinning & protrusion.
- Onset around puberty
- Both eyes are affected.

Keratoconus

- Presentation
  - Typically, during puberty
  - Unilateral impairment of vision due to progressive myopia & astigmatism.
  - 50% of normal fellow eyes progress to keratoconus within 16 years.

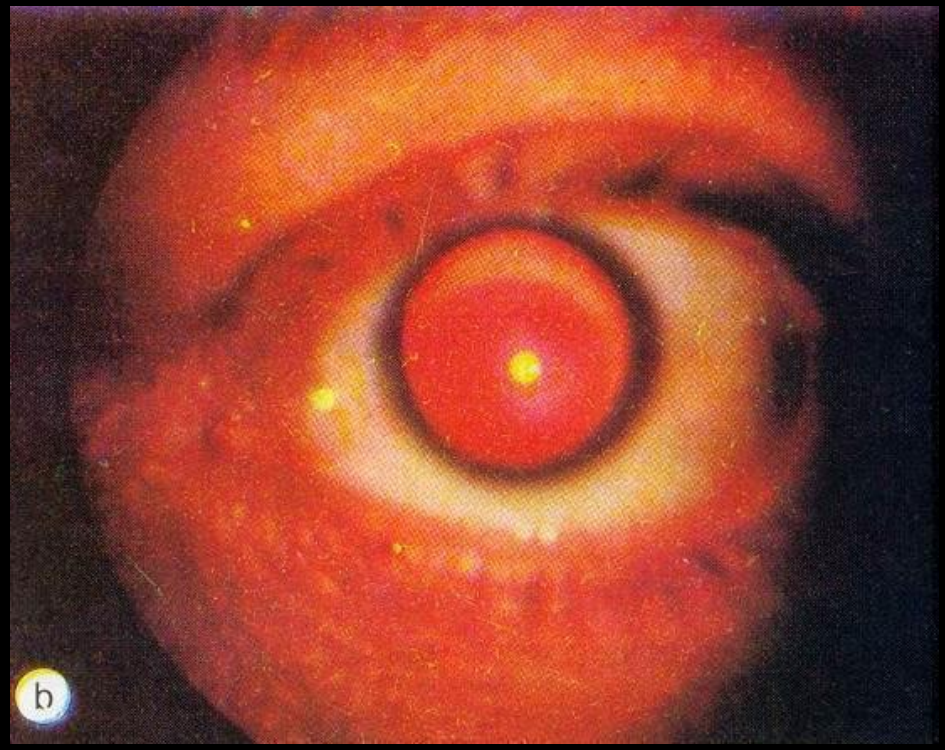
Keratoconus

Diagnosis

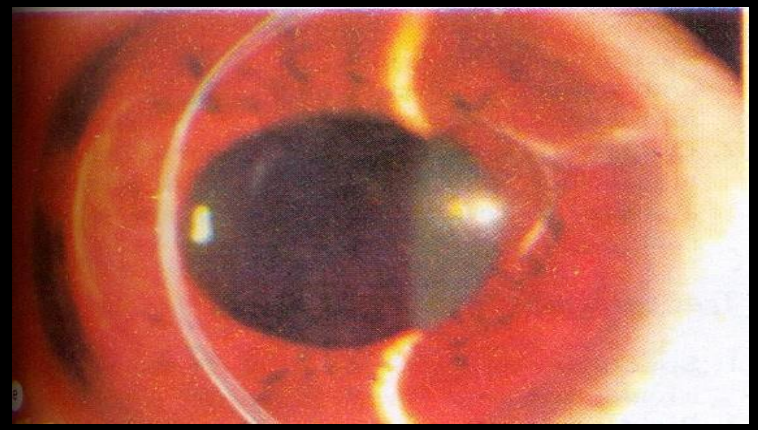
- Hallmark – central or paracentral stromal thinning, atypical protrusion & irregular astigmatism.
- Graded by keratometry according to the severity
  - Mild < 48 D
  - Moderate 48 – 54 D
  - Severe > 54 D

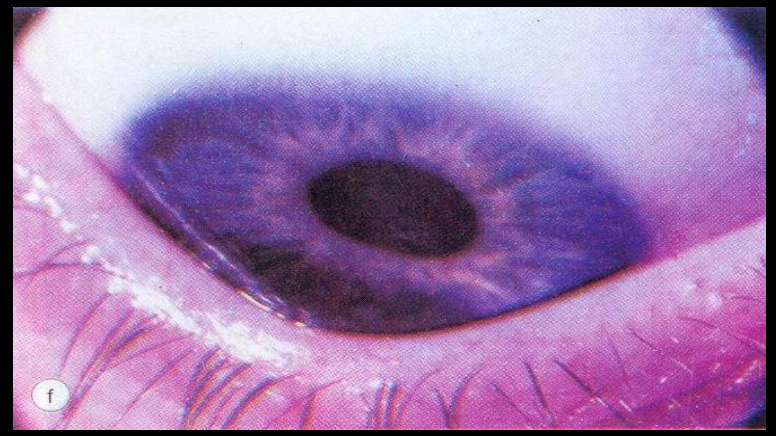
Keratoconus

- Signs
  - Direct ophthalmoscopy – oil droplet reflex
  - Retinoscopy – irregular scissor reflex
  - Slit lamp – fine vertical deep stromal striae
  - Epithelial iron deposits – surround the base of the cone (Fleischer ring)



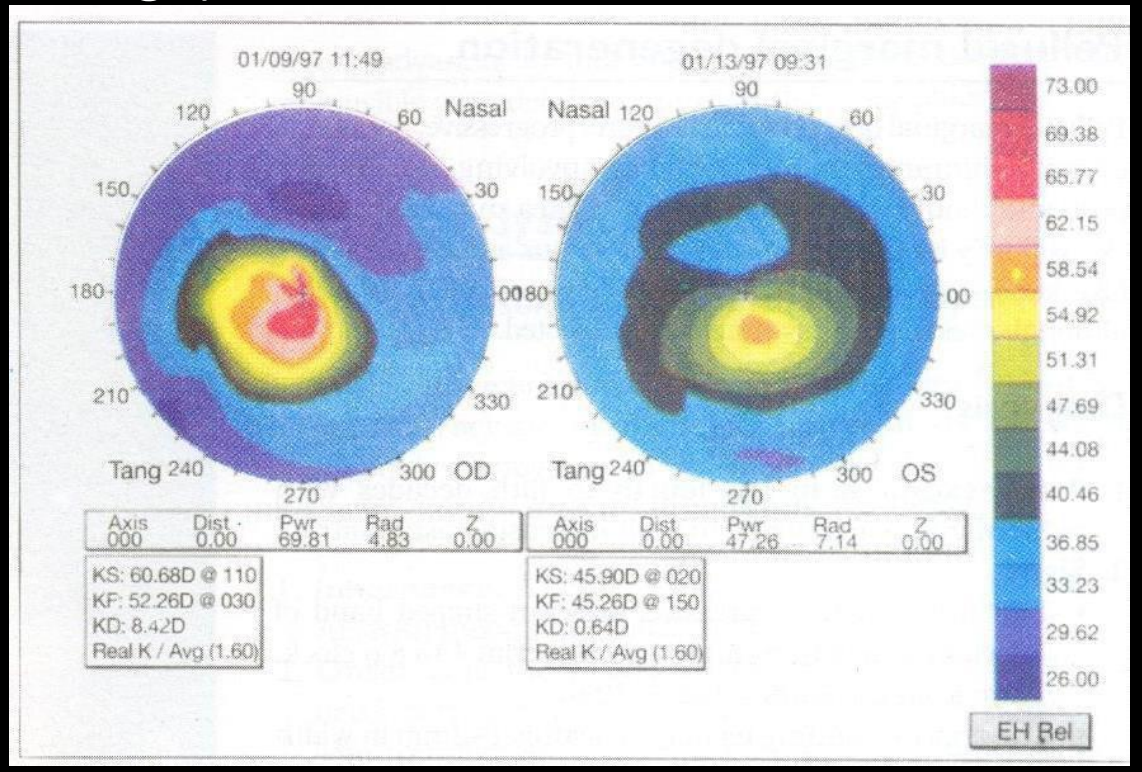
Diagnosis





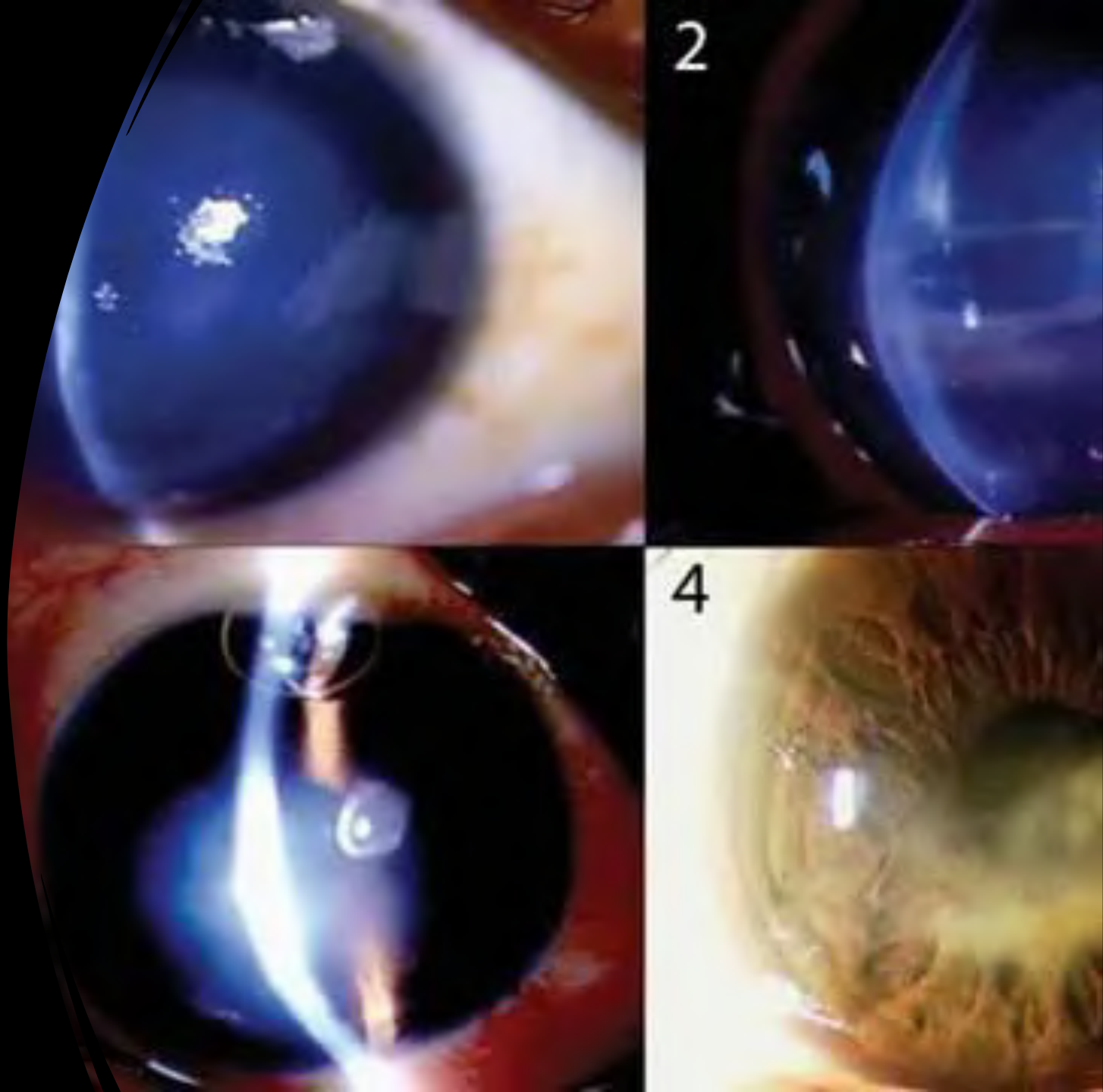
- Signs (Cont'd)
  - Progressive corneal thinning
  - Bulging of lower lid in down-gaze (Munson sign)

- Corneal Topography
  - Irregular astigmatism

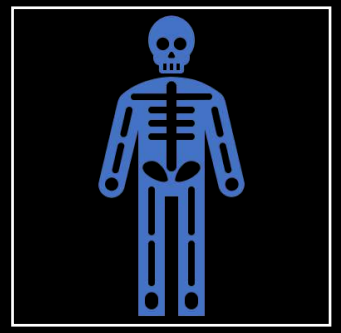


Acute hydrops

- Rupture in Descemet's membrane allows an influx of aqueous into the cornea.
- A sudden drop in visual acuity.

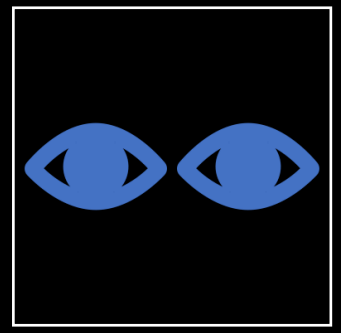


Associations



Systemic disorders

Down, Turner, Ehlers – Danlos syndrome, Marfan syndrome, Atopy.



Ocular associations

Vernal keratoconjunctivitis, blue sclera, Aniridia, ectopia lentis, Retinitis pigmentosa

Treatment

Spectacles

In early cases

Regular & irregular astigmatism

Rigid contact lenses

For higher degrees of astigmatism

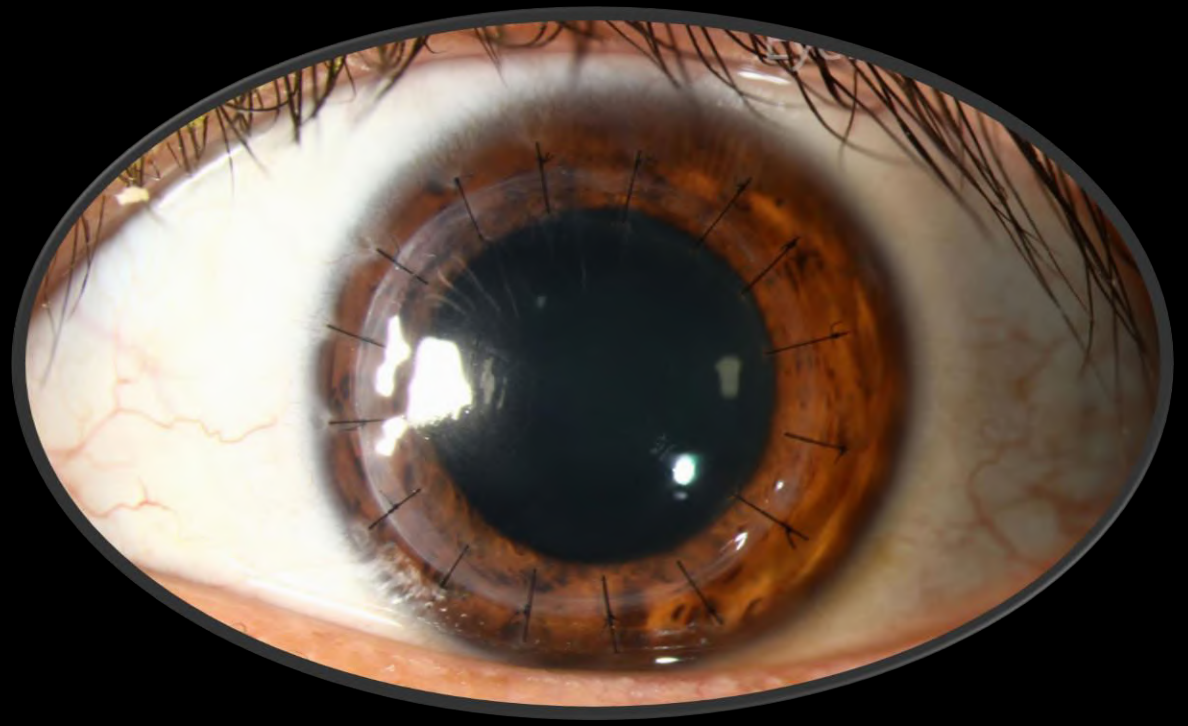
Provide a regular refracting surface

Corneal cross-linking

Intrastromal corneal rings

Treatment

- Keratoplasty
  - Penetrating or deep lamellar
  - Advanced progressive disease with corneal scarring
  - Clear grafts in 85% of cases



Corneal Dystrophies

A group of progressive disorders

Bilateral, genetically determined

Non-inflammatory opacifying disorders

Age at the presentation – 1st & 4th decade.

Classification

Based on biomicroscopic & histopathological features

Corneal dystrophies classified into

- Epithelial
- Bowman layer
- Stromal
- Endothelial

Classification (Cont'd)

Epithelial Dystrophies

- Epithelial basement membrane dystrophy.
- Meesman dystrophy
- Lisch dystrophy

Bowman layer dystrophy

- Reis Buckler dystrophy
- Thiel Behnke dystrophy
- Central Schnyder dystrophy

Classification (Cont’d)

- Stromal dystrophy
    - Lattice dystrophy 1
    - Lattice dystrophy 2
    - Lattice dystrophy 3 & 3A
    - Granular dystrophy type 1
    - Granular dystrophy type 2
    - Macular dystrophy
    - Gelatinous drop-like dystrophy
    - Central cloudy dystrophy of Francois

Classification (Cont’d)

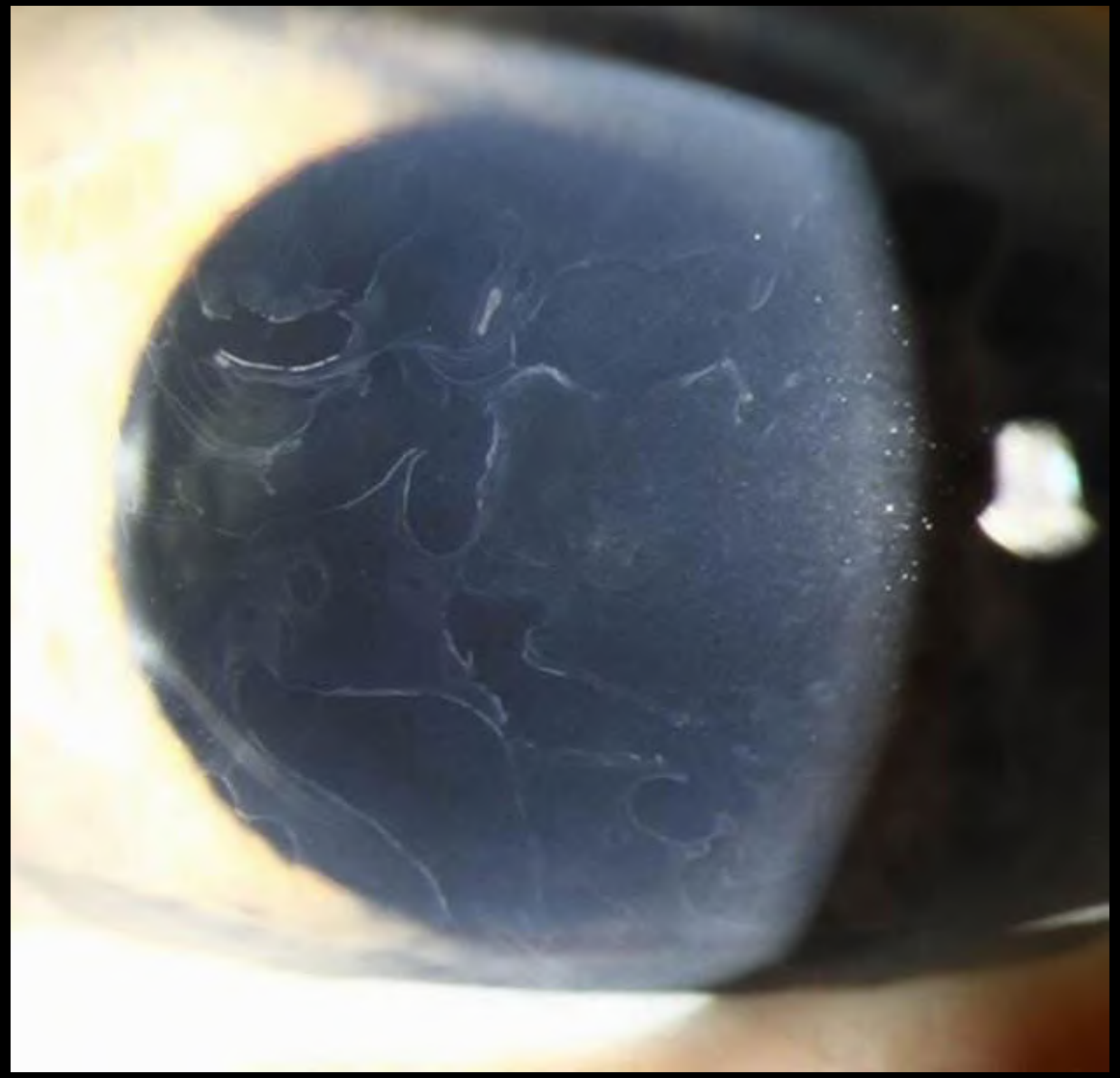
- Endothelial dystrophies
    - Fuchs endothelial dystrophy
    - Posterior polymorphous dystrophy
    - Congenital hereditary endothelial dystrophy

Epithelial dystrophy

Epithelial basement membrane Dystrophy

- Most common dystrophy
- Sporadic, rarely AD
- Onset 2nd decade
- 10% develop recurrent corneal erosion

- Sign
  - Dot-like opacities / epithelial micro-cysts
  - A sub-epithelial map-like patterns
  - Whorled fingerprint-like lines



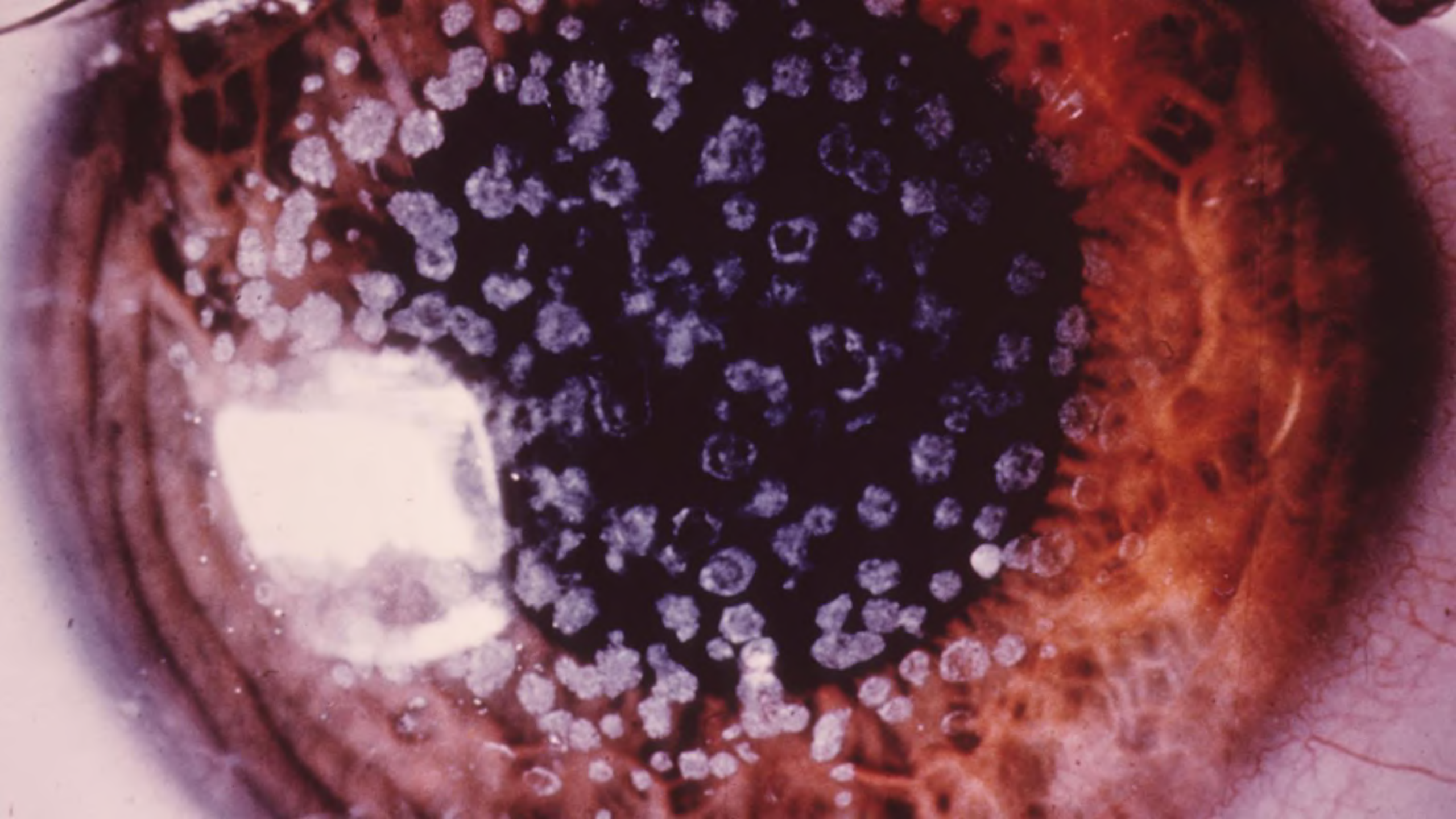
Granular dystrophy type 1

Inheritance AD

Onset – 1st decade

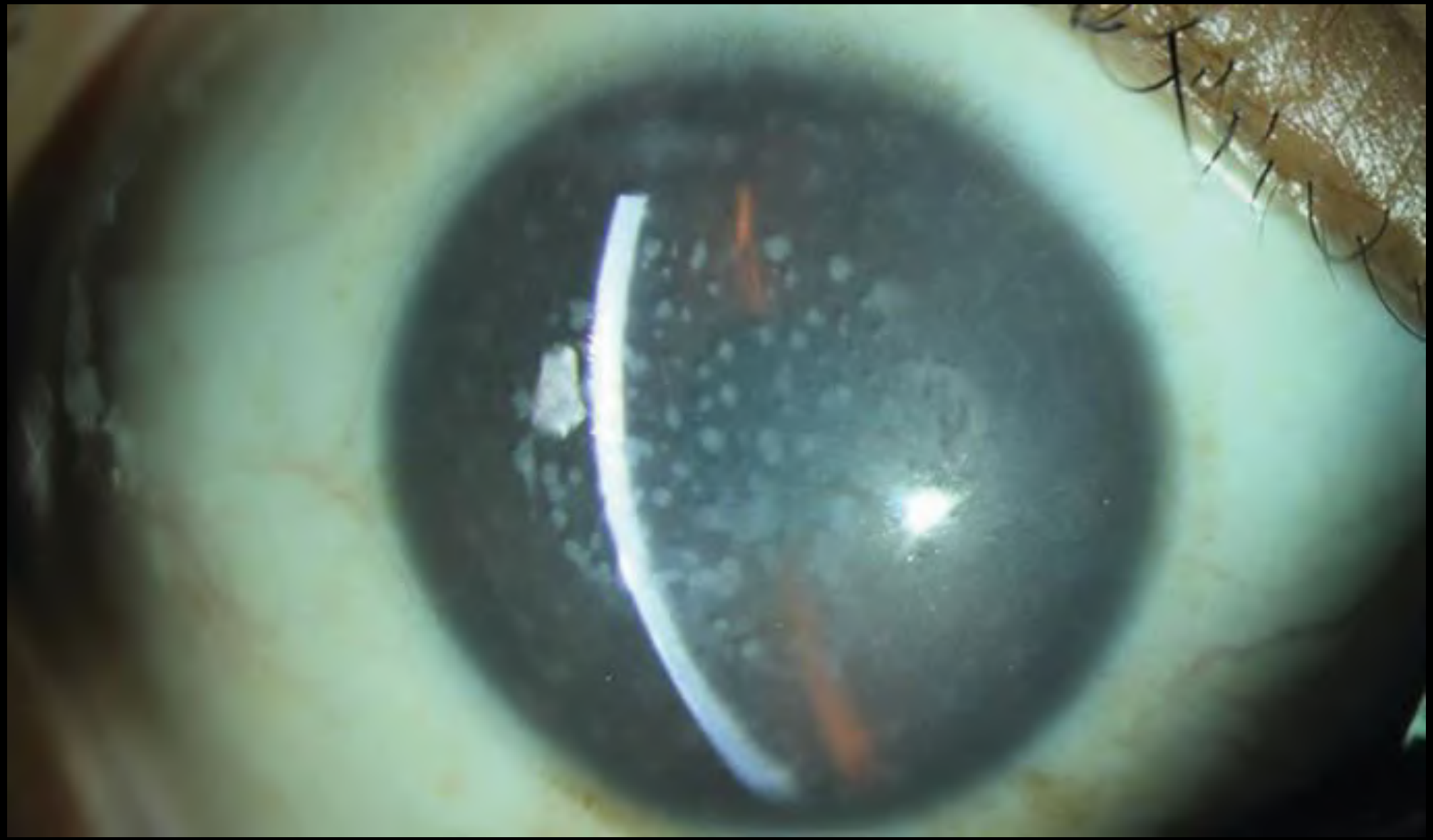
Signs

- Small, sharply demarcated deposits resembling crumbs, and rings in the central anterior stroma.
- Central stroma between opacities is clear.
- Deposits spread outward, but not reaching limbus



Macular Dystrophy

- Least common stromal dystrophy.
- Systemic inborn error of keratan sulfate metabolism
- Has corneal manifestations
- Inheritance AR
- Onset end of 1st decade with gradual impairment.



Fuchs endothelial dystrophy

- A bilateral disease
- Accelerated corneal endothelial cell loss
- AD
- Slowly progressive disease in old age

Stages

1

Cornea guttata

- Irregular warts or excrescences of Descemet's membrane are secreted by abnormal endothelial cells

2

- Endothelial decompensation – central stromal edema

3

- Persistent epithelial edema – micro-cysts & bullae (bullous keratopathy)

Treatment

- Topical
    - sodium chloride 5% drops or ointment.
- Bandage Contact lenses
    - Provide comfort by protecting exposed nerve endings.
- Penetrating Keratoplasty
    - High success rate.

Corneal Degenerations

- Any of the several tissue changes
- Occur in previously normal tissues
- As a result of prior diseases.

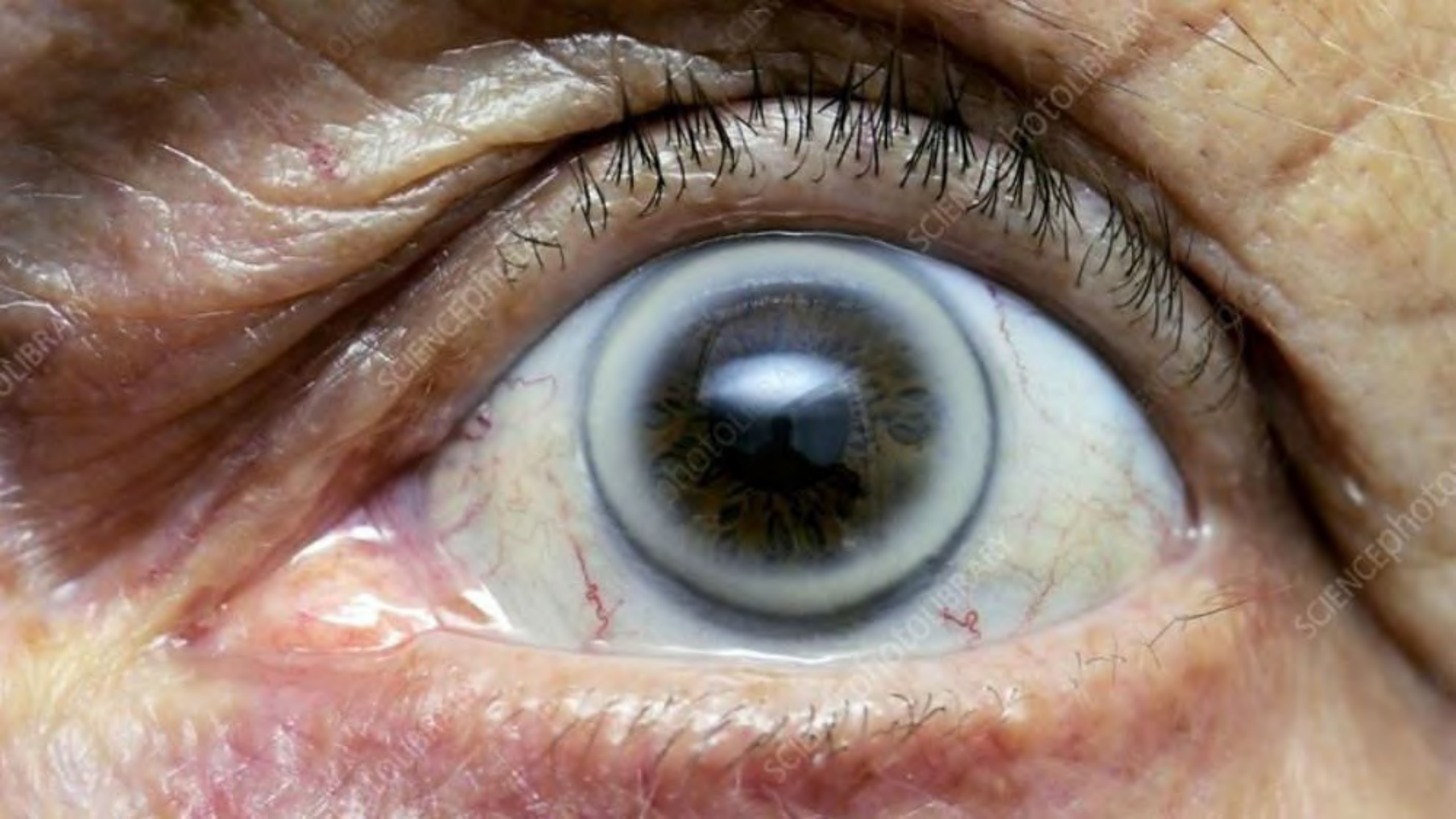
- Classification
    - Age-related degeneration
    - Arcus Senilis
    - Vogt limbal girdle
    - Corneal Farinata
    - Crocodile shagreen.

Lipid keratopathy

- Band keratopathy
- Spheroidal degeneration
- Salzmann nodular degeneration
- Terrien marginal degeneration

Arcus-Senilis

- Most Common peripheral corneal opacity
- Frequently – without predisposing systemic conditions in the elderly.
- Occasionally – Familial & non-familial dyslipoproteinemias.
  - Hyperlipoproteinemia type II
  - Less common – type III, IV & V
- Unilateral arcus – rare entity carotid disease or ocular hypotony.

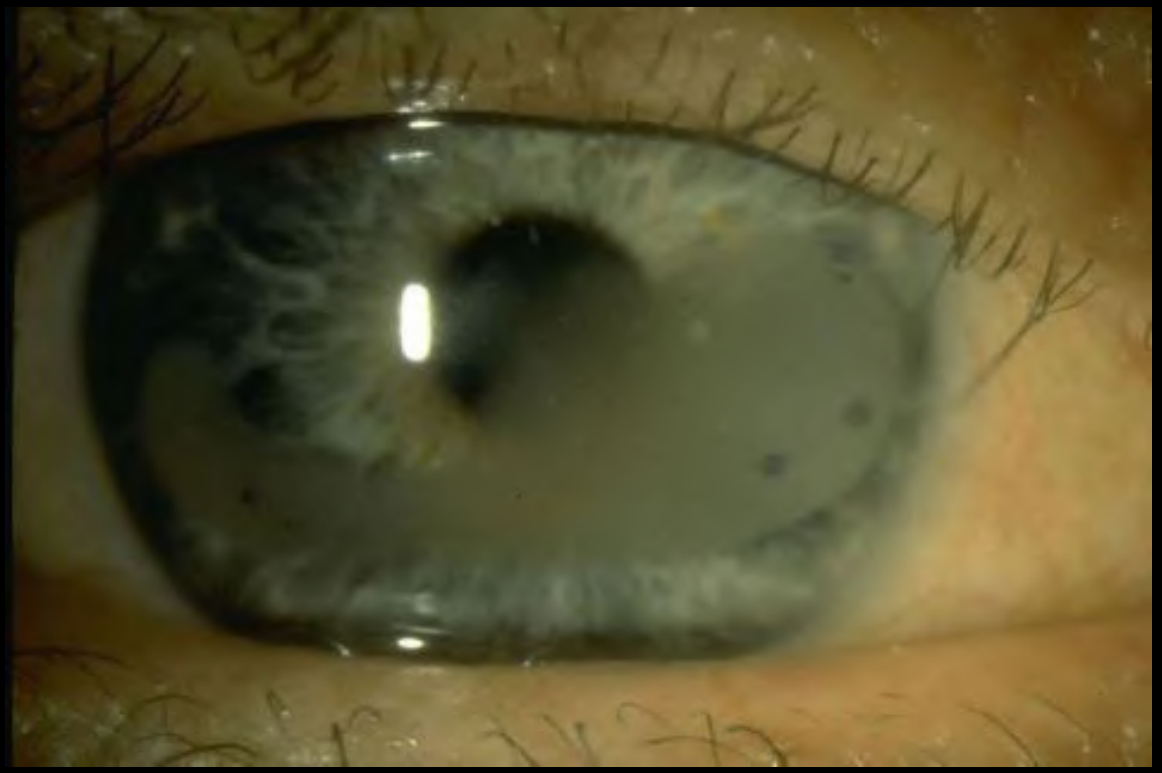


Band Keratopathy (Cont’d)

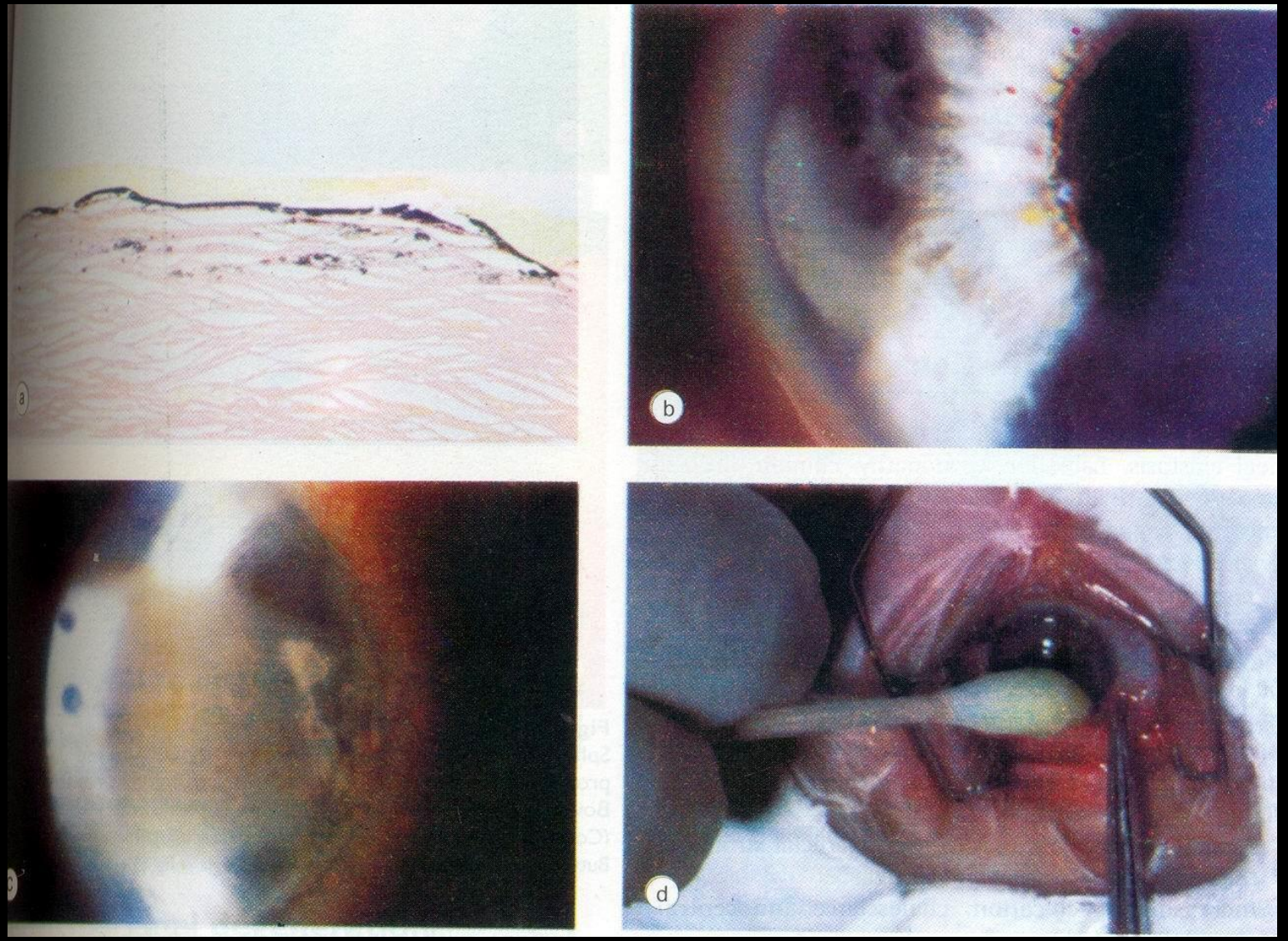
- Ocular Causes
    - Chronic anterior uveitis
    - Phthisis bulbi
- Age-related
- Metabolic Causes
    - Metastatic calcification
    - Increased serum calcium & phosphorus, hyperuricemia & CRF.
- Hereditary
    - Familial cases

Band Keratopathy (Cont’d)

- Signs
  - Peripheral interpalpebral calcification with the clear cornea.
  - Gradual central spread to form a band-like chalky plaque.
  - Contains transparent small holes



- Treatment
    - Indicated if vision is threatened.
    - Chelation
        - Effective for mild cases
        - Ethylene diamine – tetra–acetic acid (EDTA).
    - Diamond burr, Nd: YAG laser.


a
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Learning objectives

- Discuss the etiology, clinical features, investigation, and management of keratoconus.
- Give an overview of corneal dystrophies and degenerations.

Thank you


بسم الله الرحمن الرحيم

Red eye 5

Non microbial keratitis

Dr Nazullah

Associate Professor

Clinical scenario

- A female patient came to eye opd with pain redness & decreased vision in her Rt eye. On examination her vision is 6/36. There is a crescent shape peripheral corneal ulcer in nasal side with conjunctival congestion. Her left is blind because of the same problem. She has been treated in other hospital. What can b the cause.?
- A autoimmune keratitis
- B bacterial keratitis
- C fungal keratitis
- D viral keratitis


A

Mooren ulcer

- First described by Mooren in 1867. More in M as compared to F 1.6:1
- Is a chronic inflammation of the peripheral cornea
- Autoimmune in nature due to vasculitis of the limbal vessels which causes ischemia & necrosis leading to enzymatic release that play A role in ulceration
- Usually associated with Rheumatoid arthritis
- Two types; limited form which if often unilateral and mild . Occurs in old age
- Progressive form is usually bilateral & progressive . Occurs in young peoples

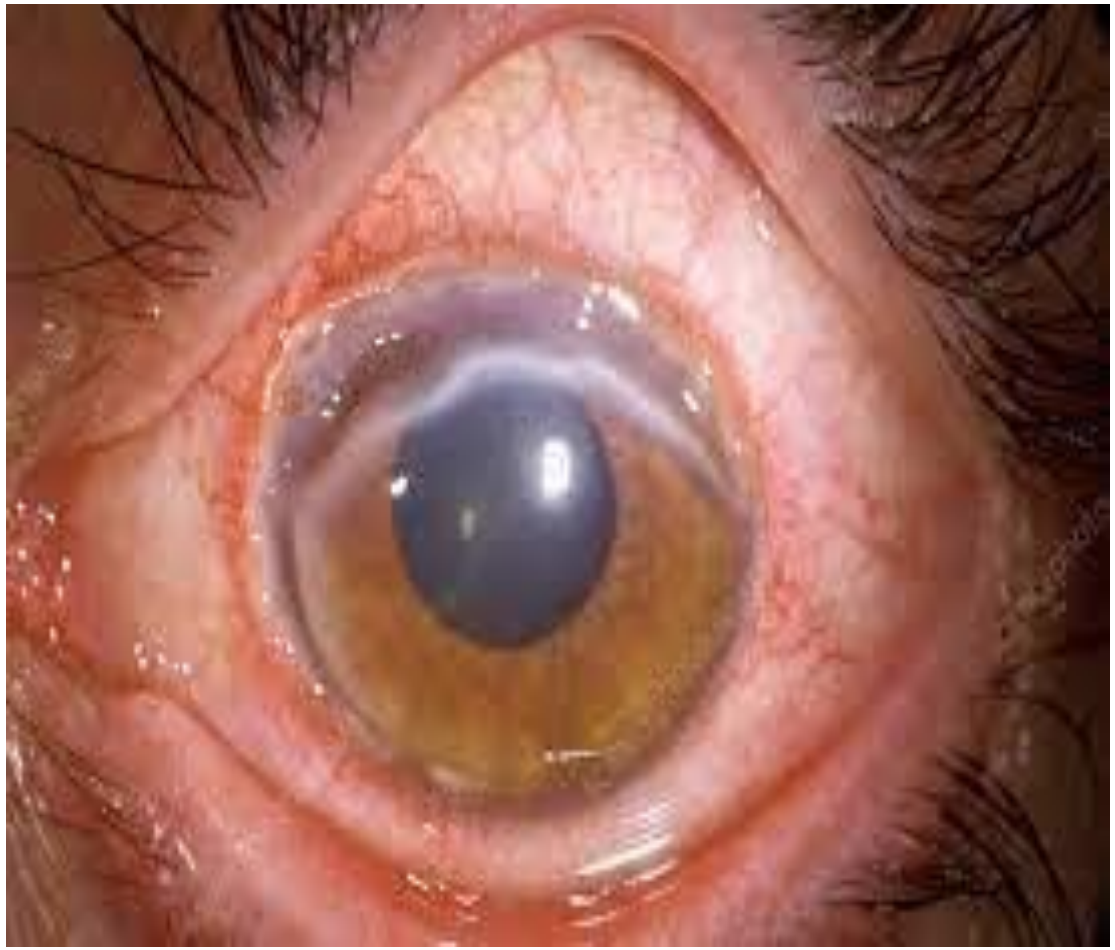
Speculation.?

- Gottsch and colleagues have suggested that this disorder can result from a host response to Calgranulin C, a normally hidden antigen expressed by keratinocytes in the corneal stroma. [6] This molecule has also been found in circulating polymorphonuclear leukocytes. [5] Receptors for this antigen have also been found on the surface of certain helminths, which has led to speculation regarding an association with helminthic infections. However, Mooren's ulcer has not been proven to be more prevalent in endemic areas of ascariasis. [4]

Clinical features

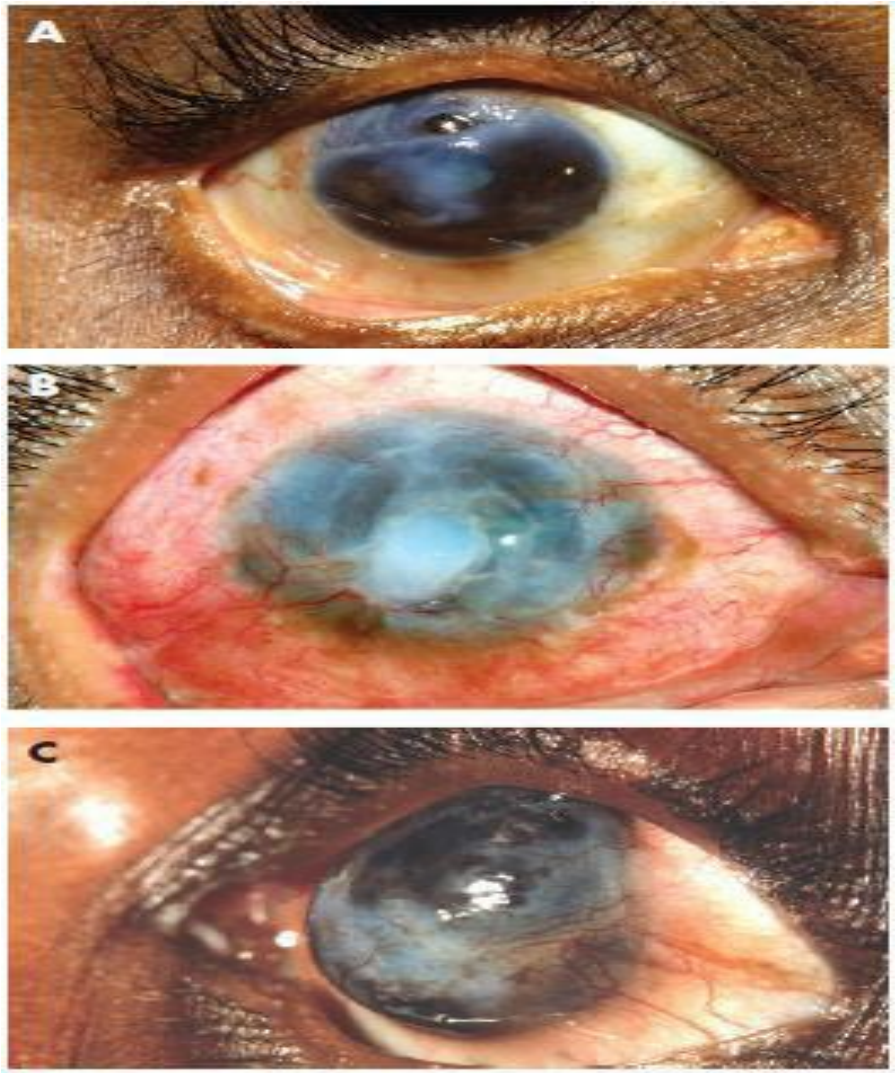
- Characterized by painful peripheral corneal ulceration of unknown etiology. The disease generally begins with intense limbal inflammation and swelling in the episclera and conjunctiva with Pain lacrimation redness & blurring of vision
- It starts spontaneously as an excavation in Corneal margin near the limbus. It progresses by undermining the corneal epith & superficial corneal lamella. It has raised margins with overhanging ridge at the advancing edge. It spreads circumferentially and centripetally & causes corneal thinning . The healed cornea is usually Descemet membrane covered with epith.
- The uninvolved cornea is clear


A



Complication

- Progressive Astigmatism
- Anterior uveitis
- Complicated Cataract & secondary Glaucoma
- Corneal Thinning
- Descemetocele formation
- Perforation
- Blindness
-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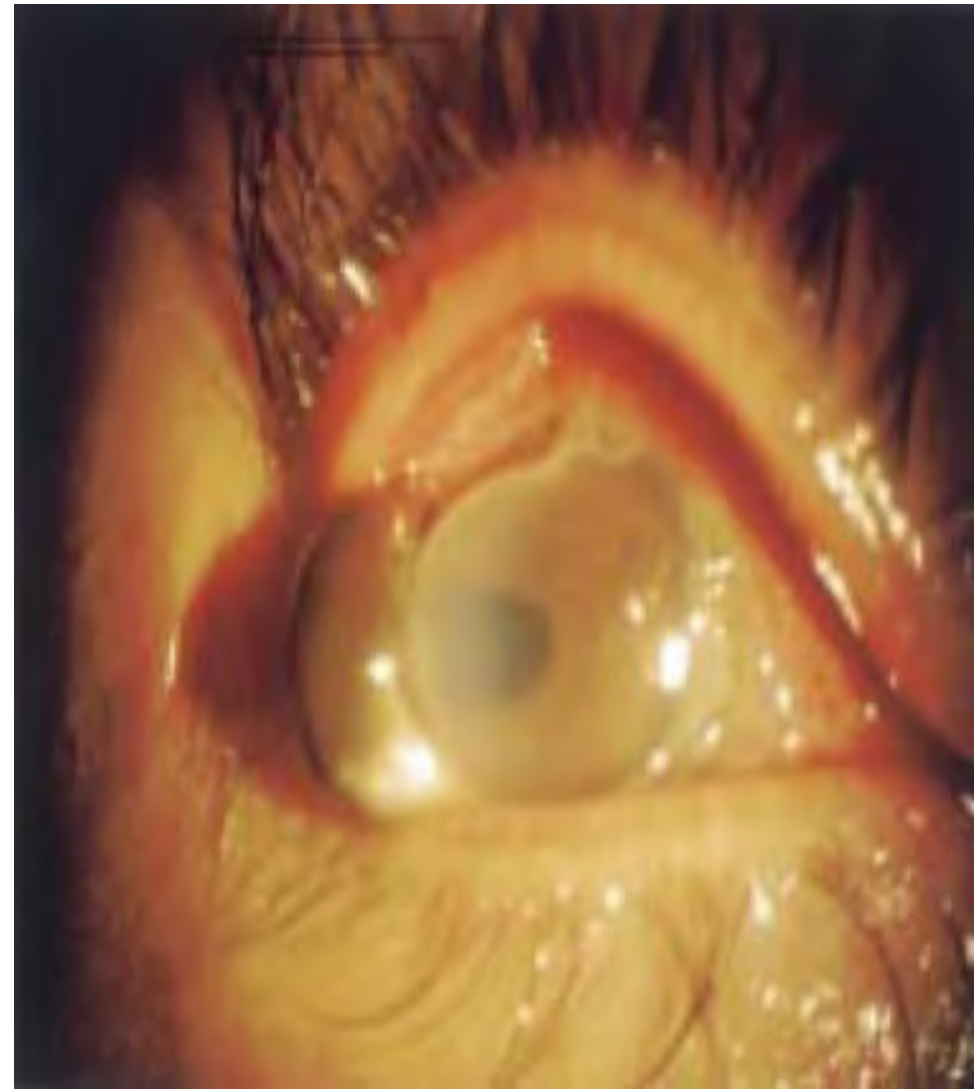


Treatment

- Med ;
- Topical; Steroid, Cyclosporin 1% or 2%, interferon alfa 2 a
- Cyclopen,
- Antibiotics
- Systemic immunosuppressive such as cyclophosphamide, azathioprine, or
- methotrexate. In more aggressive cases, oral corticosteroids, cyclosporine A
- Rituximab have also been used with some success.
- Doxycyclin; collagenase inhibitors
- Surgical
- Conjunctival resection
- Pkp

- Excision of a 3-4 mm ring of limbal conjunctiva at least 2 clock hours adjacent to a Mooren's ulcer has been shown to be an effective treatment.
- Studies have shown that when less than half of the limbus is involved, the cure rate after conjunctival excision and corneal ulcer resection is performed is 51.3%. When more than half of the limbus is involved, the cure rate after the procedure decreases to 36.8%.
- In a 1975 study where limbal conjunctival excision was performed, 8 out of 10 eyes healed, and one developed recurring ulcers which then healed upon re-treatment. It was hypothesized that the limbal conjunctiva may contain antibodies that react with antigens in the corneal stroma in addition to enzymes that destroy the corneal stroma—therefore, excision may interrupt the disease process.

- Surgical interventions can also include lamellar keratoplasty, keratoepithelioplasty, delimiting keratotomy, and conjunctival flap and patch grafts using periosteum or fascia lata.
- Resection of the corneal lesion and adjacent conjunctiva combined with lamellar keratoplasty can achieve a final healing rate of 89.6%.
- If topical 1% cyclosporine A is added, a 95% final healing rate can be achieved.
- But usually fails

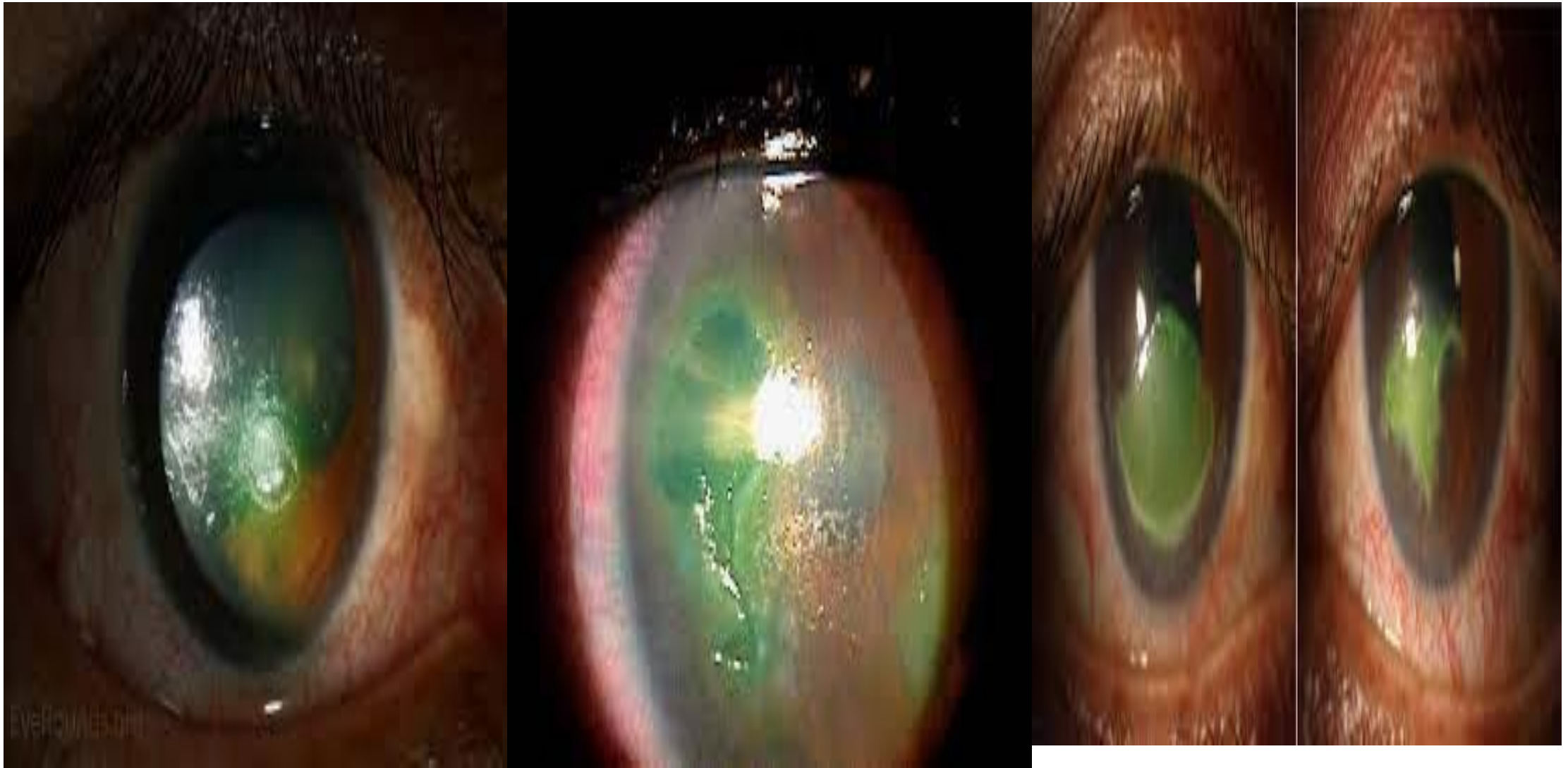
Neurotropic keratitis

- It is keratitis due to impaired Corneal innervation & sensation by ophthalmic
- division of 5th nerve → hypoesthesia → disturbed epithelial metabolism →
- accumulation of metabolites in cells → epithelial edema, exfoliation & ulceration
- Causes may be
- Surgical trauma; of trigeminal ganglion surgery or alcoholic block
- Systemic disease; such as DM is vey common, may be syphilis, leprosy etc etc
- Tumors; such as cerebellopontine tumour such as aquastic Neuroma
- Ocular problem; such as HZO
- Ocular surgery; PKP
- Refractive surgery ; LASIK etc

Clinical features

- Painless redness is the characteristic
- Va is decreased
- Decreased lacrimation & dry eyes
- Conjunctival congestion
- Corneal sensation are decreased
- Corneal dullness due edema
- Progressive sterile ulceration & melting
- Secondary infection ulceration & perforation

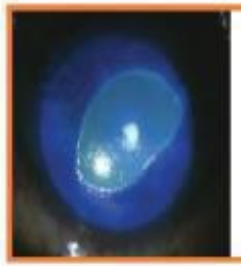
Neurotropic corneal ulcer



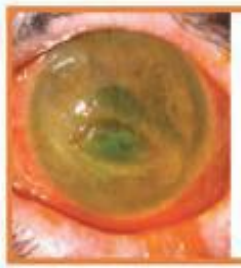
NK Is a Degenerative Disease[1]

The Mackie Classification Represents One Way to Assess NK Progression[2,3]

STAGE 1 (Mild)
Punctate keratitis



STAGE 2 (Moderate)
Persistent epithelial defect (PED)



STAGE 3 (Severe)
Corneal ulcer

- Some vision loss can potentially be seen **in all stages of NK**[3]
- If untreated, **moderate NK progresses to severe disease** with associated risks of **profound vision loss** resulting from scarring and corneal perforation[3]

1. Dua HS, Said DG, Messmer EM, et al. Neurotrophic keratopathy. *Prog Retin Eye Res*. 2018 Sep;66:107-131.
2. Semeraro F, et al. Neurotrophic Keratitis. *Ophthalmologica*. 2014;231:191-197.
3. Bonini S, Lambiase A, Rama P, et al. Phase II randomized, double-masked, vehicle-controlled trial of recombinant human nerve growth factor for neurotrophic keratitis. *Ophthalmology*. 2018;125:1332-1343.

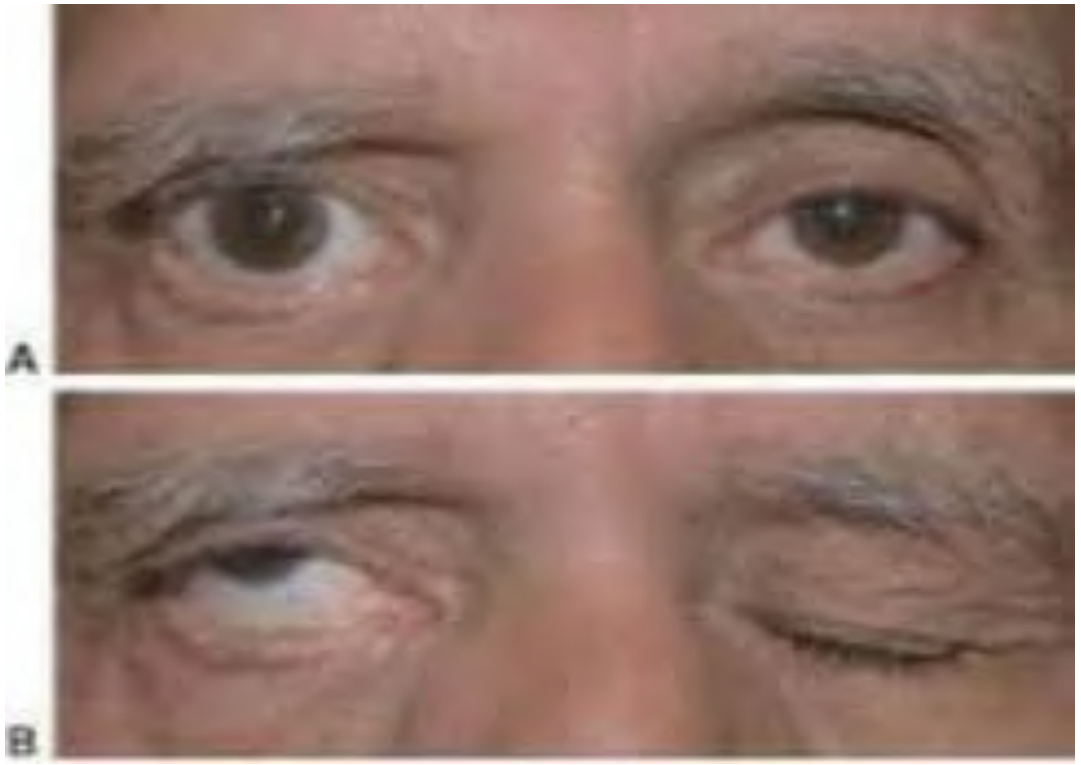
Treatment

- Topical lubricant such as tears plus drops frequently
- Topical neurotonic/ nerve growth factors drops
- Avoid drops containing toxic preservative such as benzalkonium
- Autologus serum may be helpful
- Anticollegenase such as tetracyclin doxycycline
- Taping of the eyelids
- Botox injection to induce ptosis
- Silicon contact lenses
- AMT, Conj-flap, Tarsorrhaphy if no healing

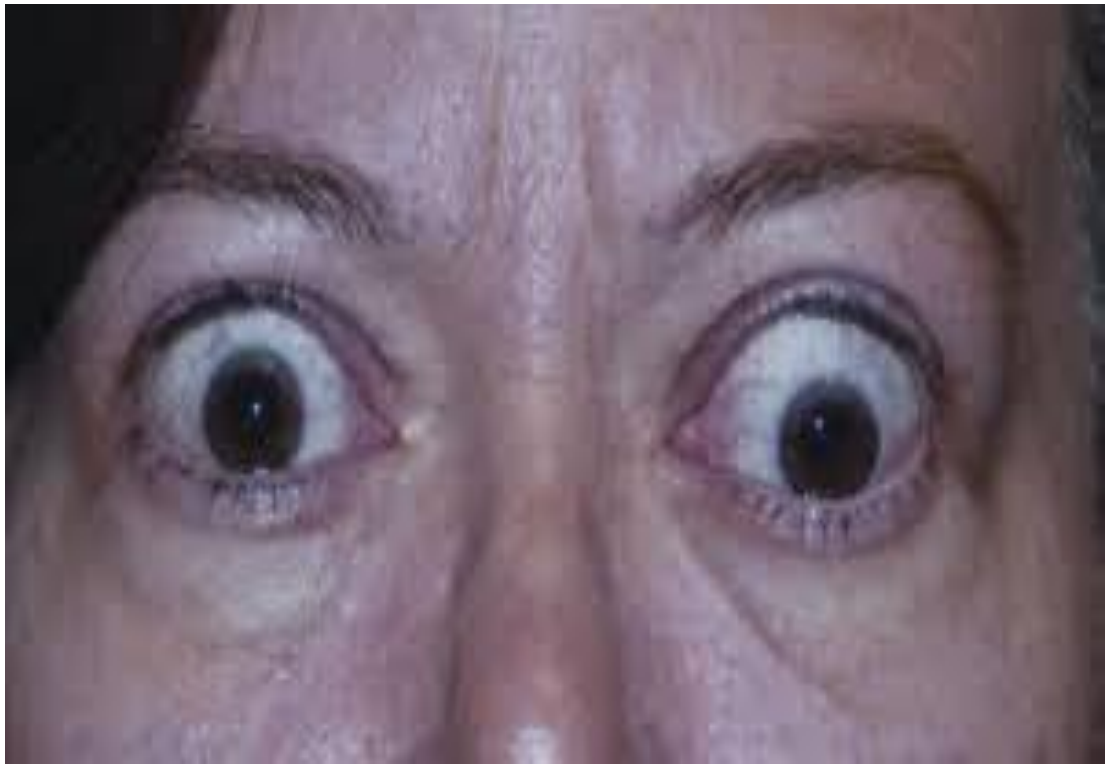
Exposure keratitis

- Normally the lid constantly moistens the cornea by frequent blinking and covered the eye during sleep
- Is due to incomplete closure of the lid which results into dryness of the eye leading to damage of the corneal epithelium and ulceration
- Causes Due to
- Neurogenic; 7^{th} n paralysis
- Proptosis; due to thyroid eye disease, orbital tumours
- Eyelid abnormalities; such as entropian, coloboma
- Blepharoplasty

7th n palsy



thyroid eye disease (TED)



Unilateral proptosis

Clinical feature

- Foreign body sensation, lacrimation, redness
- Vision is decreased
- Marked conjunctival congestion
- Epithelial erosions inferior 1/3 of the cornea
- Large defect corneal ulceration
- Secondary infection

Treatment

- Med; Topical lubricants
- Antibiotic
- Treat the cause TED etc
- Surgical; tarsorrhaphy
- Entropion surgery
- Orbital tumour surgery
- Thyroid orbital decompression


بسم الله الرحمن الرحيم

Dry eyes

Dr Nazullah

Associate professor

Ophthalmology kgmc/hmc

Dry eyes/ Keraocunjuctivitis sicca

- Is a condition in which there is insufficient secretion of aqueous tear to maintain the normal tear film

- Is a condition in which pre corneal tear film is deficient due to its decreased production or increased evaporation, leading into unstable tear film and ocular surface diseases

Contd two main types

- Can be divided in two main types

- Aqueous production deficiency

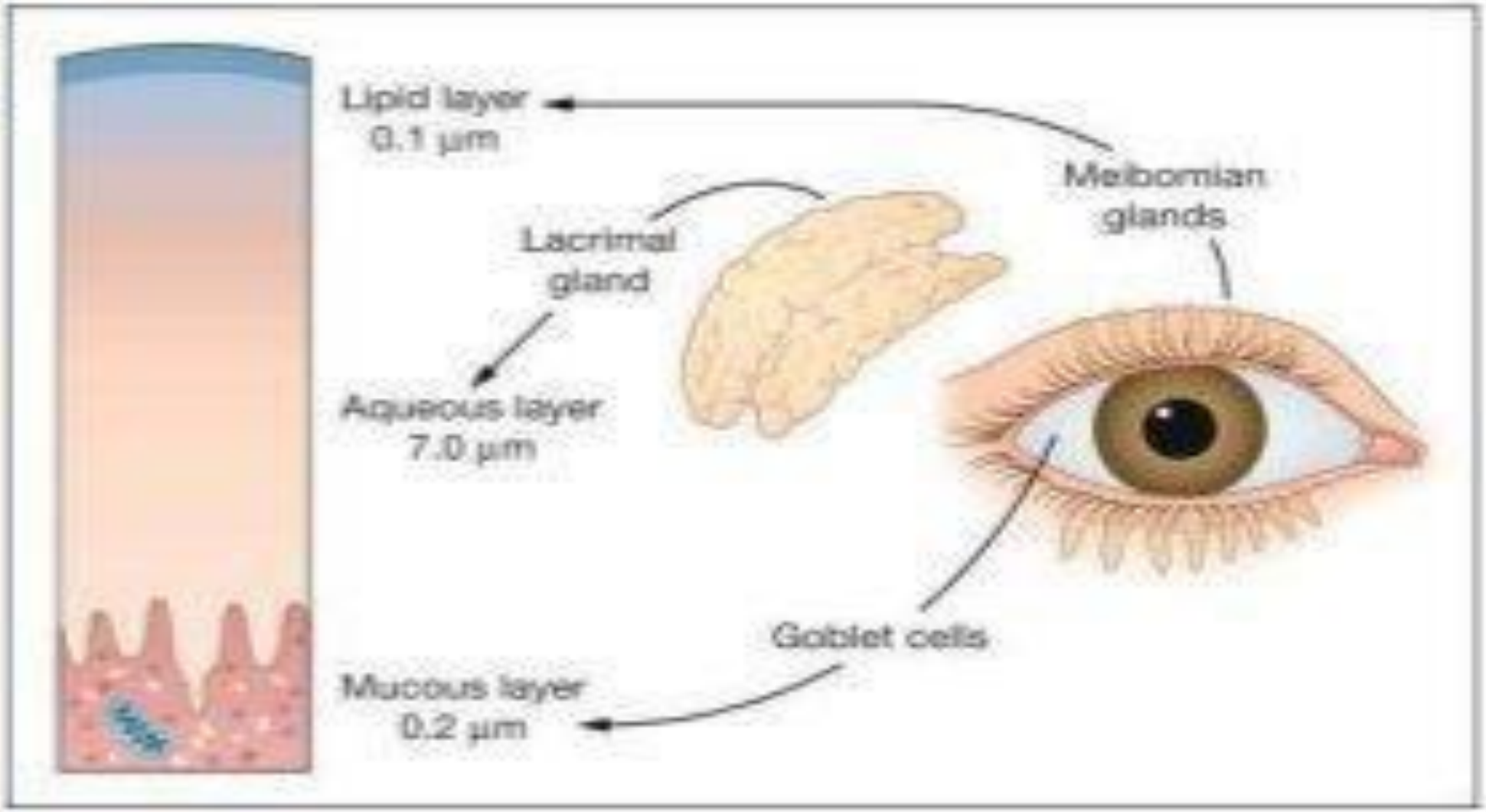
- Evaporative Tear dysfunction

Tear production

- Main lacrimal gland; main tear production 90-95% is by the main lacrimal gland . Basic production is by the accessory lacrimal glands of Krause & Wolfring, present in the conjunctival sac.

- Accessory lacrimal glands are in the conjunctiva. It produces the basic tears in the resting condition
- Normal volume 3.4 -10.7 microliter
- Normal rate in resting 1.2-2.2 microliter/mint

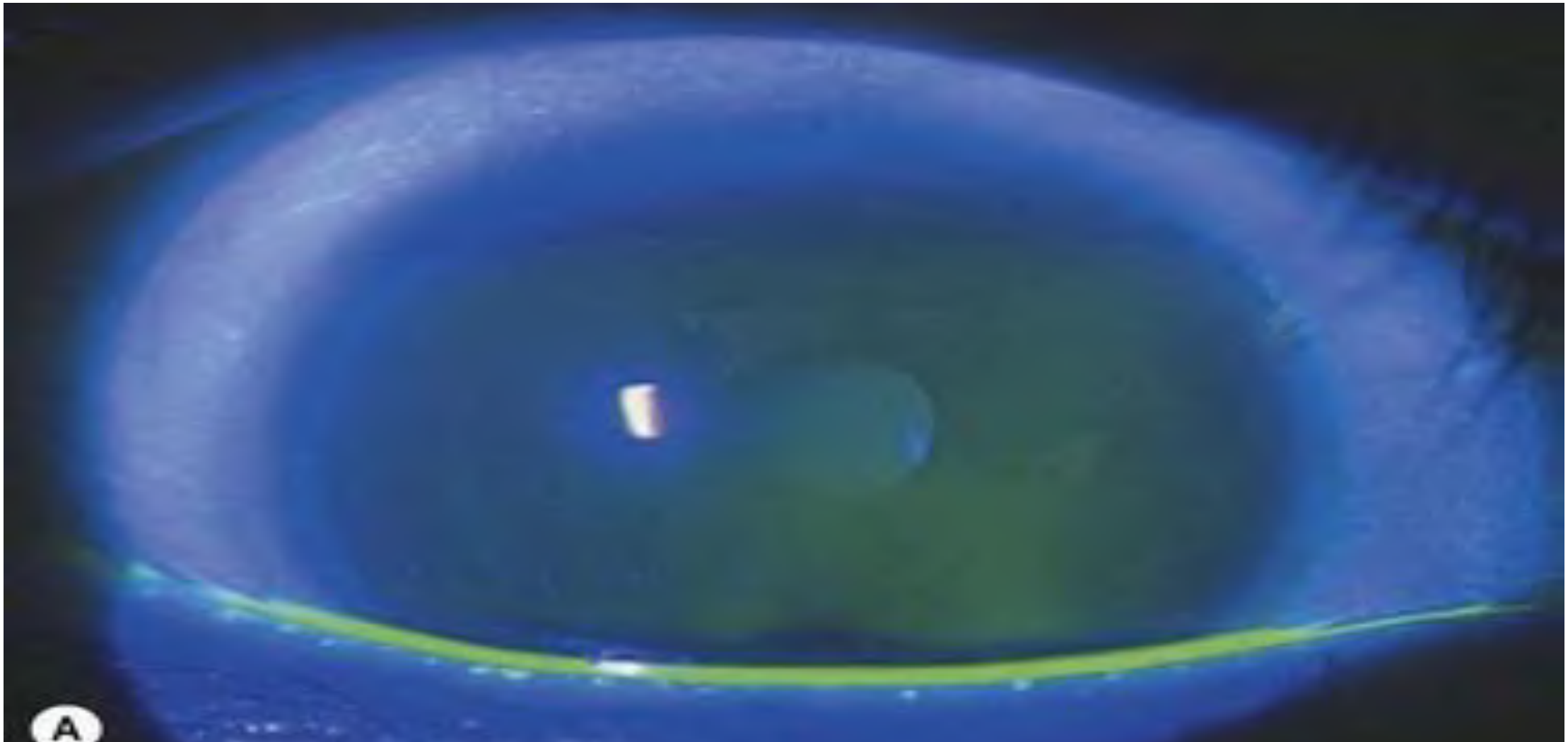
Tear film


Lipid layer
0.1 µm
Meibomian glands
Lacrimal gland
Aqueous layer
7.0 µm
Goblet cells
Mucous layer
0.2 µm

Layers of the tears

- Lipid layer; it is the outer/superficial layer of the tears which prevents the evaporation of tear film. Meibomian glands are responsible for its production.
- Aqueous layer; which is the main bulky layer 70-80% of tear film. It is the central main part of the tears and does the main function of it. It is secretd by the lacrimal gland(main +accessory gland). Major constituent is the water & minerals. It gives nutrition's and oxygen flow to the corneal avascular surface.
- Mucin layer; is the third and inner most layer of the tear. It is produced by the goblet cells/gland of the conjunctiva. It maintains attachment of the tear to the cornea by making the corneal surface sticky.

Normal precorneal tear film



A. Aqueous production deficiency

Keratoconjuctivitis sicca

- Pure Sicca syndrome; in which only the lac glands are involved
- Congenital Alacrimia
- Denervation hyposecretion such as surgery on Trigeminal gangelion
- Idiopathic hyposecretion

Sjogren syndrome

- Primary, Is an autoimmune condition in which antibodies are produced against the lacrimal gland and salivary glands leading to its inflammation & destruction without any systemic associations, and to decrease secretion of tears and leading to dry eyes & dry mouth syndromes
- Secondary, when the dry eyes are associated with systemic autoimmune disorder such as
- Rheumatoid arthritis
- Systemic lupus erythmatous

Non Sjogren sydrome

- Is a dry eye condition caused by non autoimmune conditions such as
- Trauma, Chemical thermal radiation
- Infection, trachoma
- Inflammation, like sarcoidosis, thyroid eye diseases
- Hypersensitivity, like steven Johnson syndrome
- Tumor, benign & malignant tumors of the Lacrimal gland
- Secondaries, like Leukemias and Lymphomas.

B. Evaporative causes

- Post blepharitis
- Atopc keratoconjuctitis sicca
- Sever proptosis
- Facial nerve palsy
- Eyelid scarring following blepharoplasty
- Contact lens wear

Clinical features

Photophobia, dryness, grittiness, & Foreign body sensation that becomes worse in sun , windy and hot climates

Pain with blinking & stringy mucus discharge & some blurring of vision

Examination will show Foreign bodies

Very thin tear film, almost absent.

Flouresene stain will shows very thin tear film

Rose bengal stain positive cornea with mucin filament attached to the corneal surface

Dry Eye Severity Grading Scheme

Dry Eye Severity Level	1	2	3	4*
Discomfort, severity and frequency	Mild and/or episodic; occurs under environmental stress	Moderate episodic or chronic stress or no stress	Severe, frequent or constant without stress	Severe and/or disabling and constant
Visual symptoms	None or episodic mild fatigue	Annoying and/or activity limiting episodic	Annoying, chronic and/or constant, limiting activity	Constant and/or possibly disabling
Conjunctival injection	None to mild	None to mild	+/-	+/++
Conjunctival staining	None to mild	Variable	Moderate to marked	Marked
Corneal staining (severity/location)	None to mild	Variable	Marked central	Severe punctate erosions
Corneal/tear signs	None to mild	Mild debris, ê meniscus	Filamentary keratitis, mucus clumping, é tear debris	Filamentary keratitis, mucus clumping, é tear debris, ulceration
Lid/meibomian glands	Meibomian gland disease (MGD) variably present	MGD variably present	Frequent	Trichiasis, keratinization, symblepharon
Tear film break-up time	Variable	≤ 10 seconds	≤ 5 seconds	Immediate
Schirmer score (per five min.)	Variable	≤ 10mm	≤ 5mm	≤ 2mm

* Must have signs *and* symptoms.

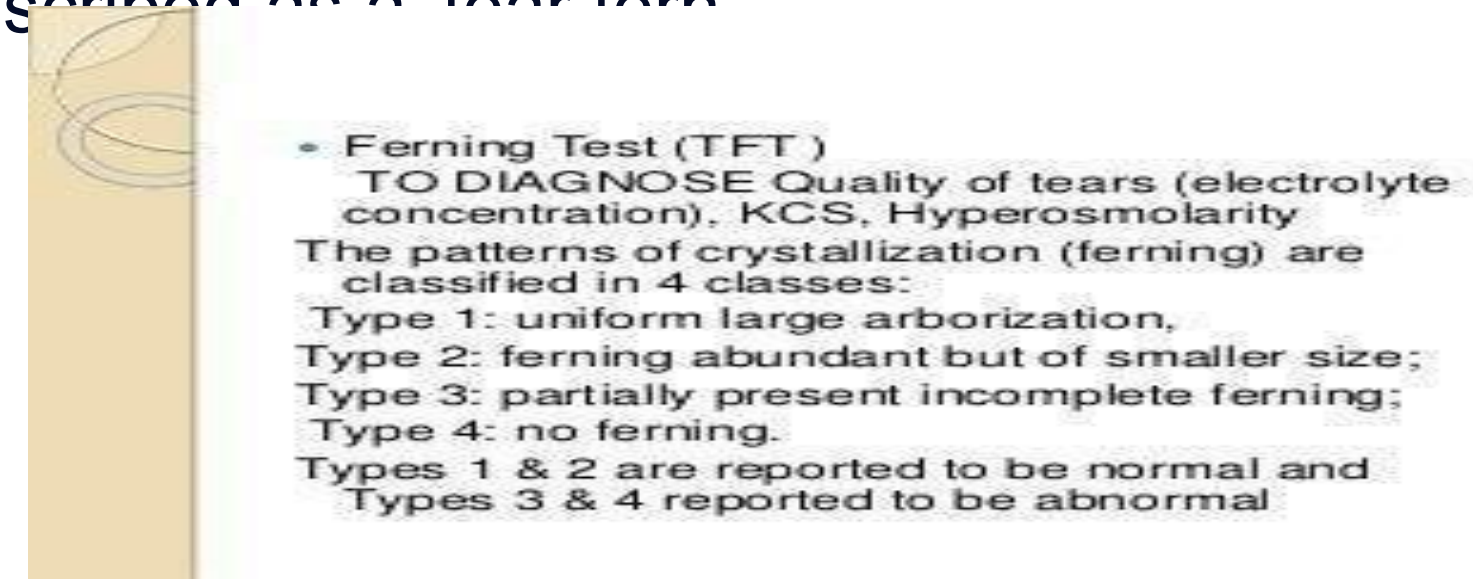
Source: Behrens A, Doyle JJ, Stern L,et al; Dysfunctional tear syndrome study group. Dysfunctional tear syndrome: a Delphi approach to treatment recommendations. Cornea. 2006 Sep;25(8):900-7.

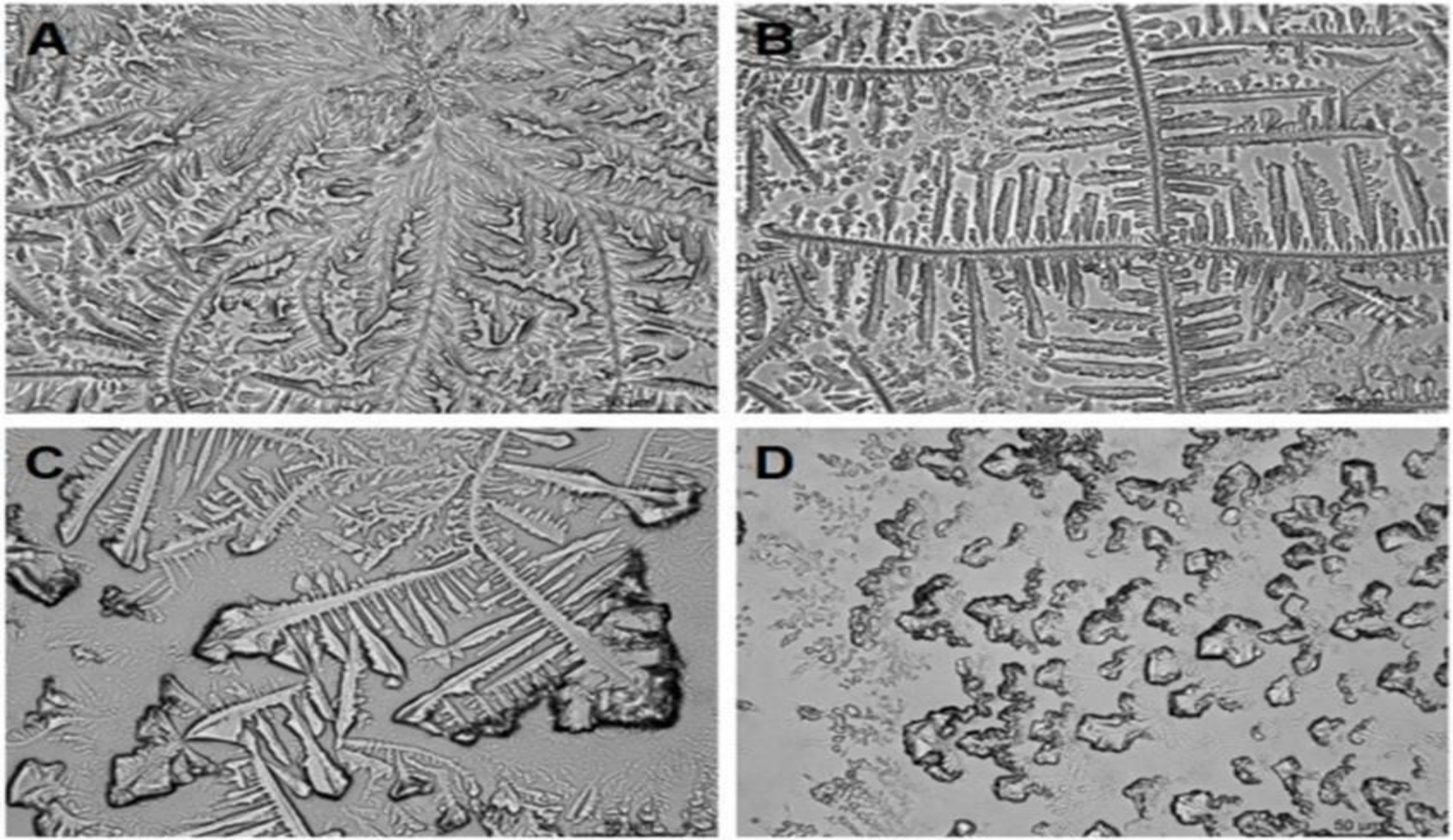
Diagnosis / investigation

- Slit lamp examination;
- Tear film break up time, will be reduced. Normal is 15-20 seconds.
- will show absent/ thin tear film, filaments attached to cornea
- Flourosene stain. Very thin tear film
- Rose bengal stain will shows mucus filament attach to the cornea
- Schirmer test shows very mild cases of dry conditions . In this condition whatman filter paper is placed in the conjunctival sac for 05 mints. Normal 15mm, moderate is 6-10mm. In dry eyes it is less then 06 mm
- Impression Cytology , detects the goblet cells which are reduced in dry eyes

Fern test

- The tear ferning test is a laboratory test but it has the potential to be applied in the clinic setting to investigate the tear film in a simple way. Drying a small sample of tear fluid onto a clean, glass microscope slide produces a characteristic crystallisation pattern, described as a 'tear fern'
-




A
B
C
D

Tear ferning test classification

- (Rolando's classification).
- Type 1: uniform arborization in the entire field of observation without spaces between the ferns. Single ferns are big and closely branched (A).
- Type 2: Arborization is abundant, but the single ferns are smaller and have a lower frequency of branching than in grade 1; empty spaces appear between the ferns (B).
- Type 3: Single ferns are little and incompletely formed with rare or no branching (C).
- Type 4: No ferning is present; mucus may appear in clusters and threads (D).
-

- Impression Cytology

-

- Brush Cytology Technique

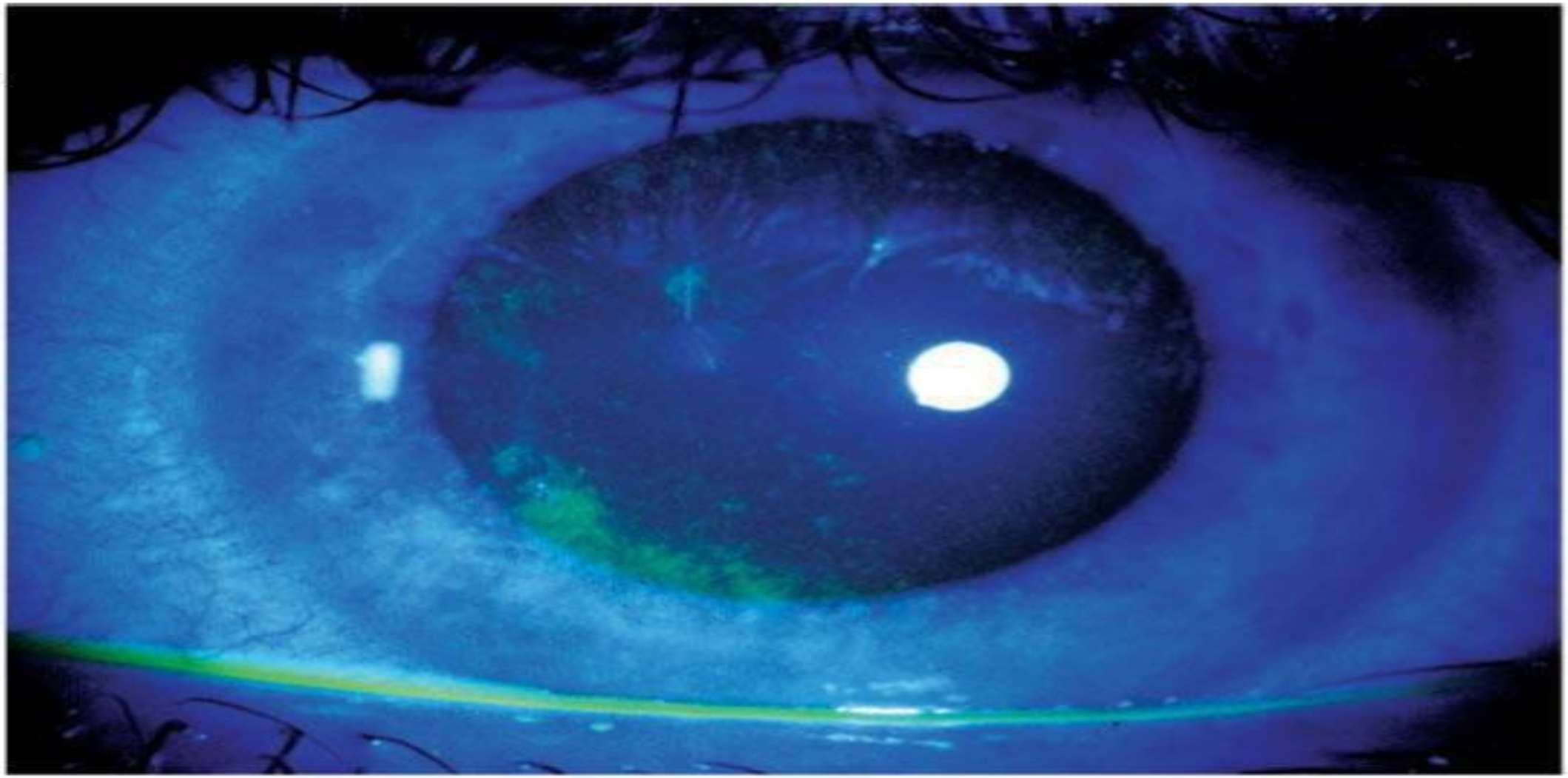
1) squamous metaplasia,
2) detecting inflammatory cells
3) expression of several surface markers on the ocular surface epithelium

- Flow cytometry in impression cytology

HLA DR expression by epithelial cells, gold standard for inflammatory assesment

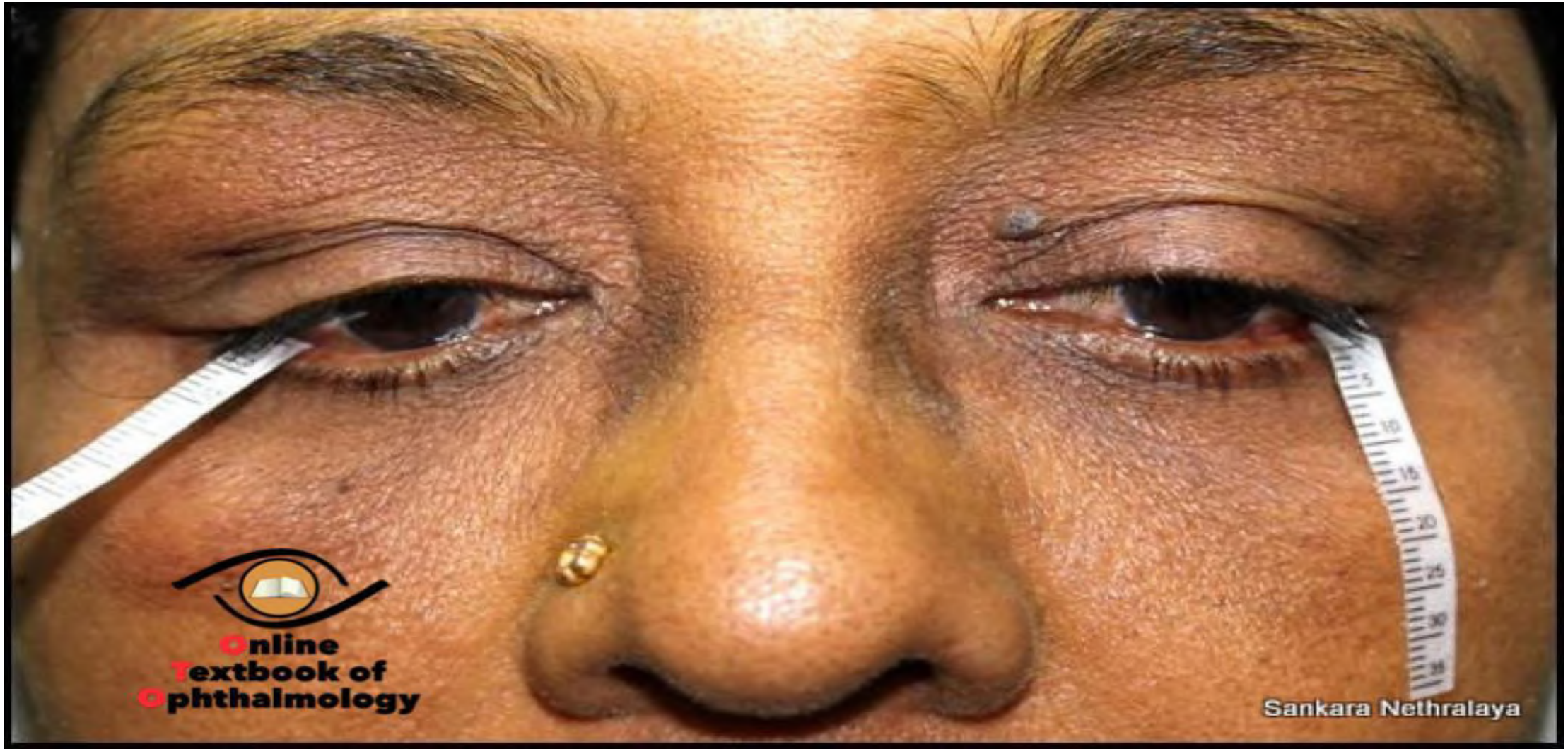
- The technique of impression cytology was established by Egbert et al in 1977 for studying goblet cells.
- The basic principle of impression cytology is the application of cellulose acetate filter paper to the ocular surface for the collection of superficial layers lining the ocular surface following which histological, immunohistological, or molecular analysis of the cells can be done.
- Impression cytology is a very useful, relatively non-invasive tool for assessing ocular surface in various dry eye disorders, such as keratoconjunctivitis sicca (KCS), cicatricial ocular pemphigoid, and vitamin A deficiency.

J. Daniel Nelson, MD



Punctate epithelial erosions in an eye stained with fluorescein.

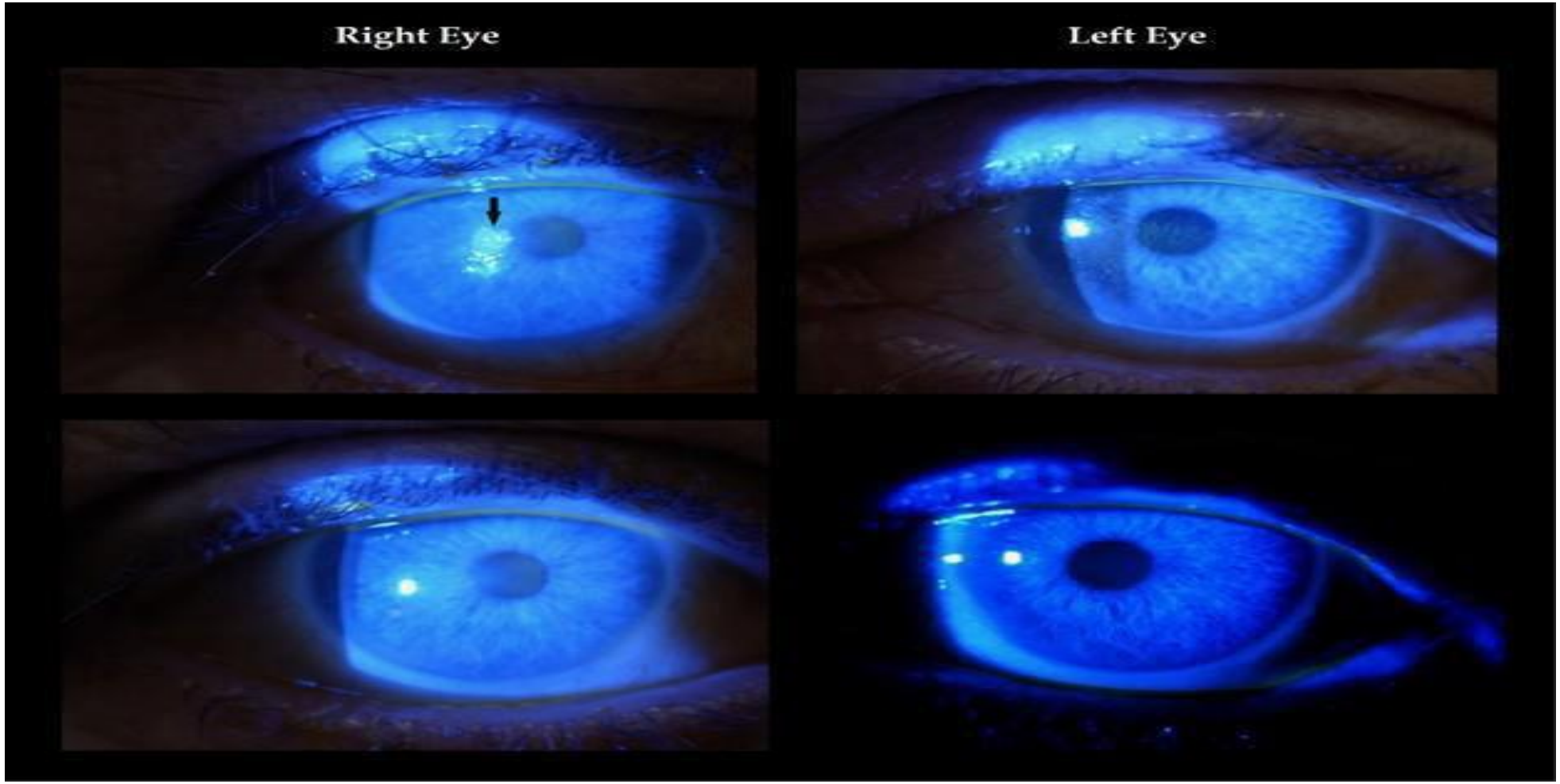
Schirmer test



Treatment, Conservative/ Surgical

- Conservative
- Tear substitute eye drops mainstay of treatment such as
- Methyle cellulose, polyvinyle alchohol, sodium hyalorunat
- Topical steroid & topical mucolytic such as Acetylcystein for dispersing mucus threads
- Autologus serum, which contains growth factor & vitamin A etc etc
- Topical cyclosporin eye drops
- Soft contact lenses, which traps the fluid behind it & make the surface smooth

Fingerprick autologous blood for dry eyes and persistent epithelial defects. Top: fluorescein-stained PED (arrow) of the right cornea and punctate staining of the left cornea at presentation. Bottom: healed PED of the right cornea and resolution of punctate staining on the left on day 4 of FAB treatment.



- Top: fluorescein-stained PED (arrow) of the right cornea and punctate staining of the left cornea at presentation. Bottom: healed PED of the right cornea and resolution of punctate staining on the left on day 4 of FAB treatment.\
- PED Persistent Epithelial Defect
- FAB finger prick Autologous Blood

Normal tear film

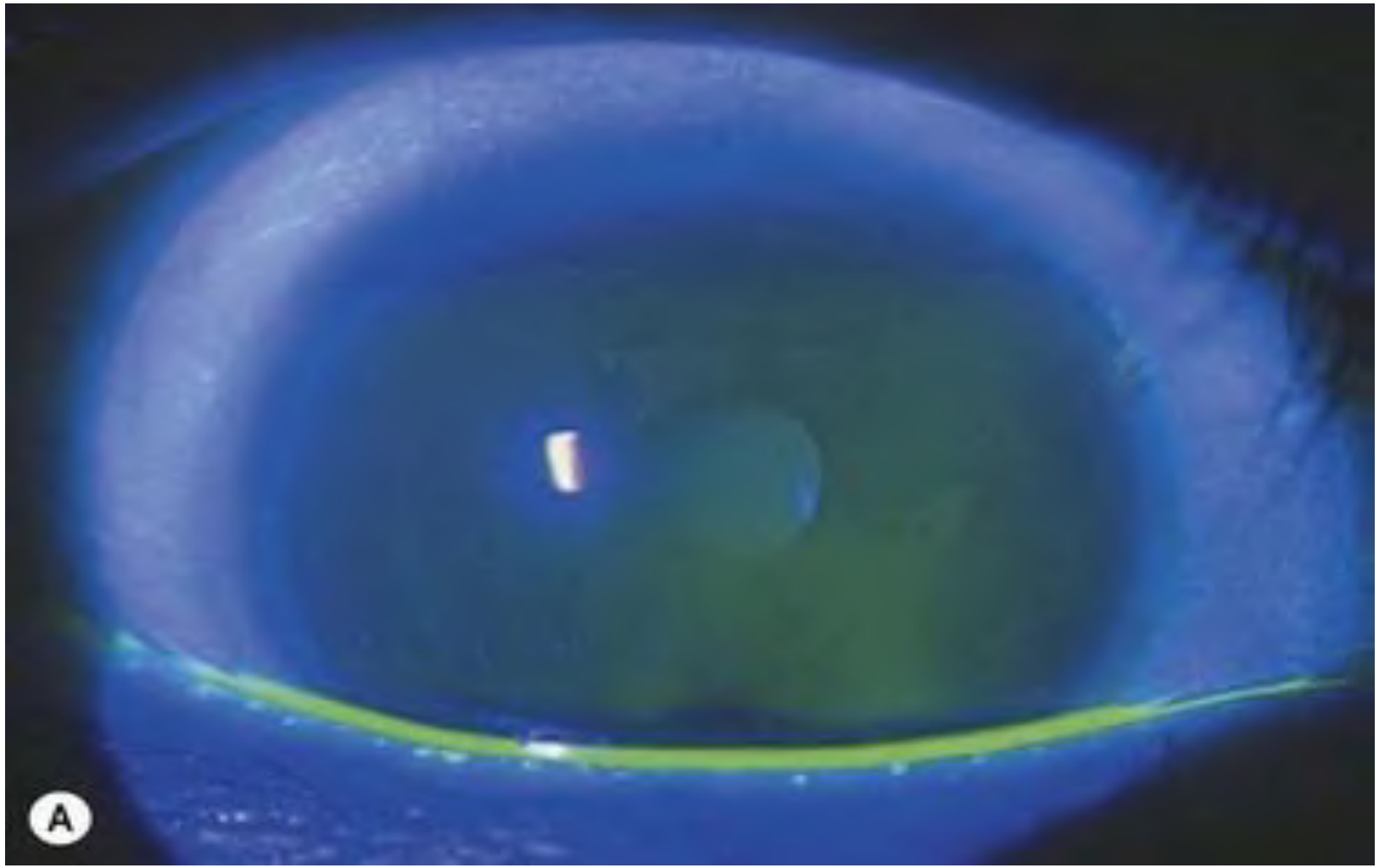
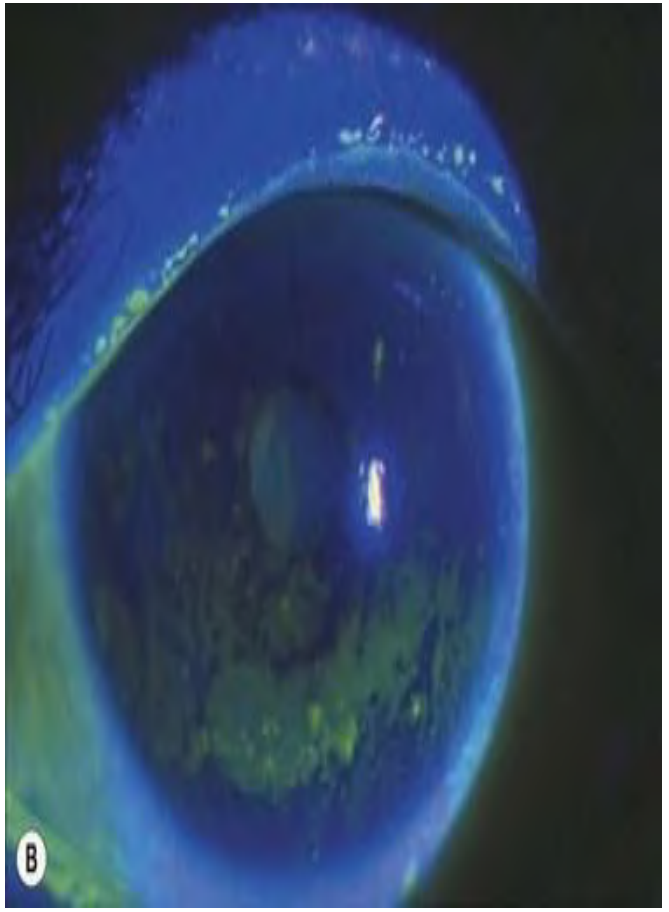
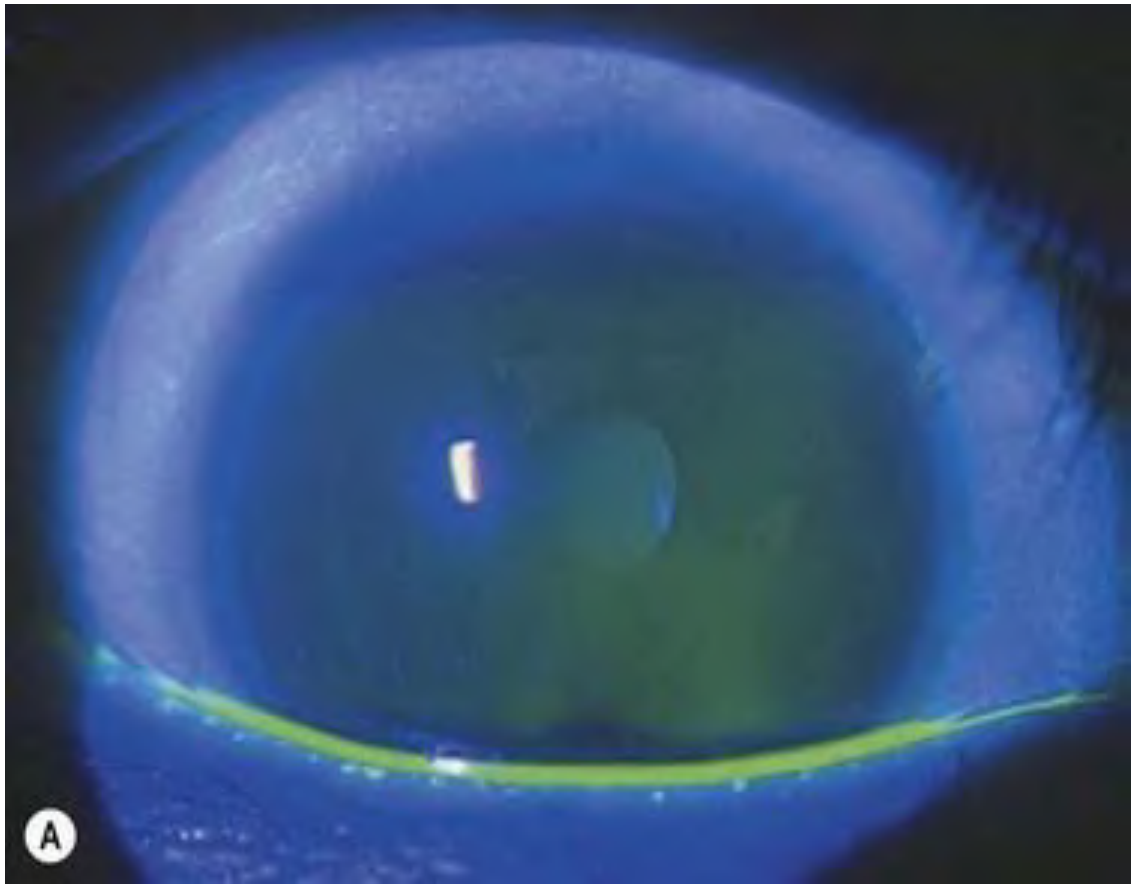


FIGURE 3.1 Slit lamp photographs with fluorescein staining of a representative dry eye patient and a normal subject. **(A)** Twenty-six-year-old male normal subject. Estimated tear film thickness was 6.4 μm. **(B)** Thirty-six-year-old female dry eye patient with Sjögren syndrome. Estimated tear film thickness was 2.4 μm. Am J Ophthalmol 2011;151:18–23.e1.)

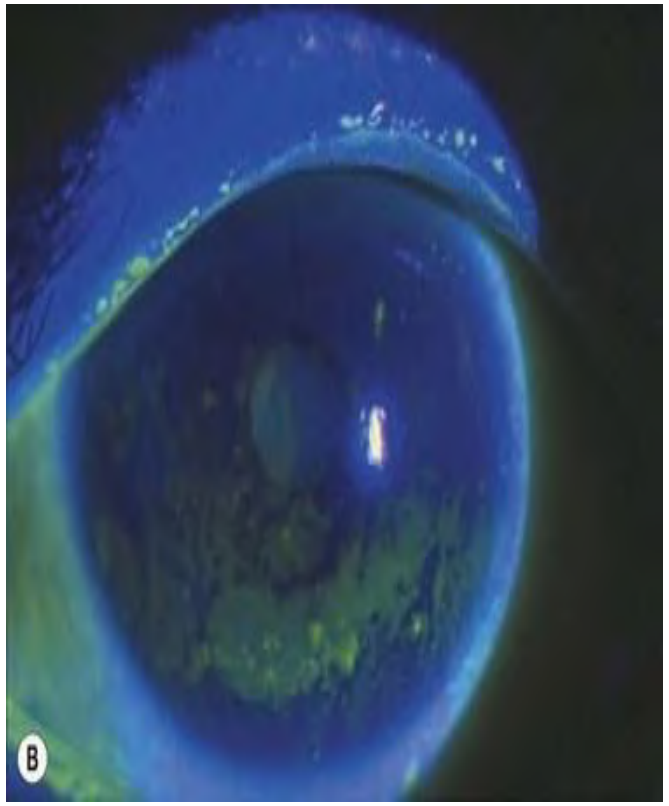


- Tear film

- Normal 6.4micron
-



- Dry eyes
- Tear film 2.5 micron



- Surgery
- Punctal Plugs to reduce the tear drainage
- Surgical transplantation of parotid duct into conjunctival sac

- Tear plugs

B. Tear Retention
1. Punctal Occlusion

Types

- absorbable and nonabsorbable. The former are made of collagen or polymers and last for variable periods of time (3 days-6 mnths).
- The nonabsorbable "permanent" plugs include silicon plugs, consists of a surface collar resting on the punctal opening, a neck, and a wider base
- Herrick plug is shaped like a golf tee and is designed to reside within the canaliculus.
- cylindrical Smart plug: expands and increases in diameter in situ, due to thermodynamic properties of its hydrophilic acrylic composition.

Vitamin A deficiency Xerophthalmia

- It is a spectrum of ocular conditions caused by vitamin A deficiency.
- The leading cause of childhood blindness
- Responsible for 20,000 to 100,000 new cases of blindness in the world, each year
- It may be caused by Malnutrition, Malabsorption, Chronic diarrhea loose motion, Chronic alcoholism and or due highly selected diet.

WHO Report

- Vitamin A Deficiency is among the leading causes of blindness
- worldwide, estimated to blind half a million children each year.
- Although VAD is rarely seen in developed countries, it remains a
- public health concern in more than half of all countries, mostly
- affecting young children in impoverished regions.

- The World Health Organization (WHO) estimates that 228 million
- children have VAD, causing 1-3 million childhood deaths and 5-10
- million cases of eye disease.
-

- VAD is especially prevalent in Africa and South-East Asia, where young children and pregnant
- women in low-income countries are disproportionally affected.
- In the United States, VAD is rare. In 2013, it was estimated at 0.3%. VAD usually involves a malabsorptive process, such as
- inflammatory bowel disease or post-gastric bypass surgery, or a
- severely restrictive diet.

Reduced intake of Vitamin A	Impaired absorption of Vitamin A	Reduced Storage of Vitamin A
•Inadequate food supply •Chronic alcoholism •Highly selective dieting •Dysphagia •Mental illness	•Crohn's disease •Celiac disease •Pancreatic insufficiency •Short bowel syndrome •Chronic diarrhea •Inflammatory bowel disease •Upper gastrointestinal surgery •Giardiasis •Abetalipoproteinemia	•Liver disease •Cystic fibrosis

Mechanism

- Vitamin A is a fat-soluble vitamin that humans derive primarily from diet. It has several essential functions in the body, including cell development, metabolism, immune function, vision, and reproductive function.
- The columnar epithelium of the mucus membrane undergoes squamous metaplasia with the loss of goblet cells, which results in dryness of conjunctiva, cornea & corneal ulceration leading to keratomalacia and perforation and blindness.
- In retina there is reduced formation of photoreceptor visual pigment resulting into night blindness.

- In the eye, Vitamin A is essential for maintenance of conjunctival and corneal epithelia as well as night vision.
- VAD causes metaplasia and keratinization of mucus-secreting epithelium, which can cause conjunctival and corneal xerosis, corneal ulcers, keratomalacia, and corneal scarring.
- Rods are the retinal photoreceptor that is responsible for night vision. Rods have a singular photopigment, rhodopsin. Retinol is a vitamin A-derived cofactor that is required for the formation of rhodopsin; thus, VAD leads to impairment of rod function and causes nyctalopia, or night blindness due to the eye's inability to adapt from light to dark[7].
-

Normal daily requirement of Vitamin A

- The recommended dietary allowance of vitamin A is 700ug/day in females and 900ug/day in the males.
- For children and pregnant or lactating women, the recommended amount is 300-900, 770, and 1300ug/day, respectively.
- Children aged 1-5 years old require a minimum of 200ug/day to prevent symptomatic VAD[

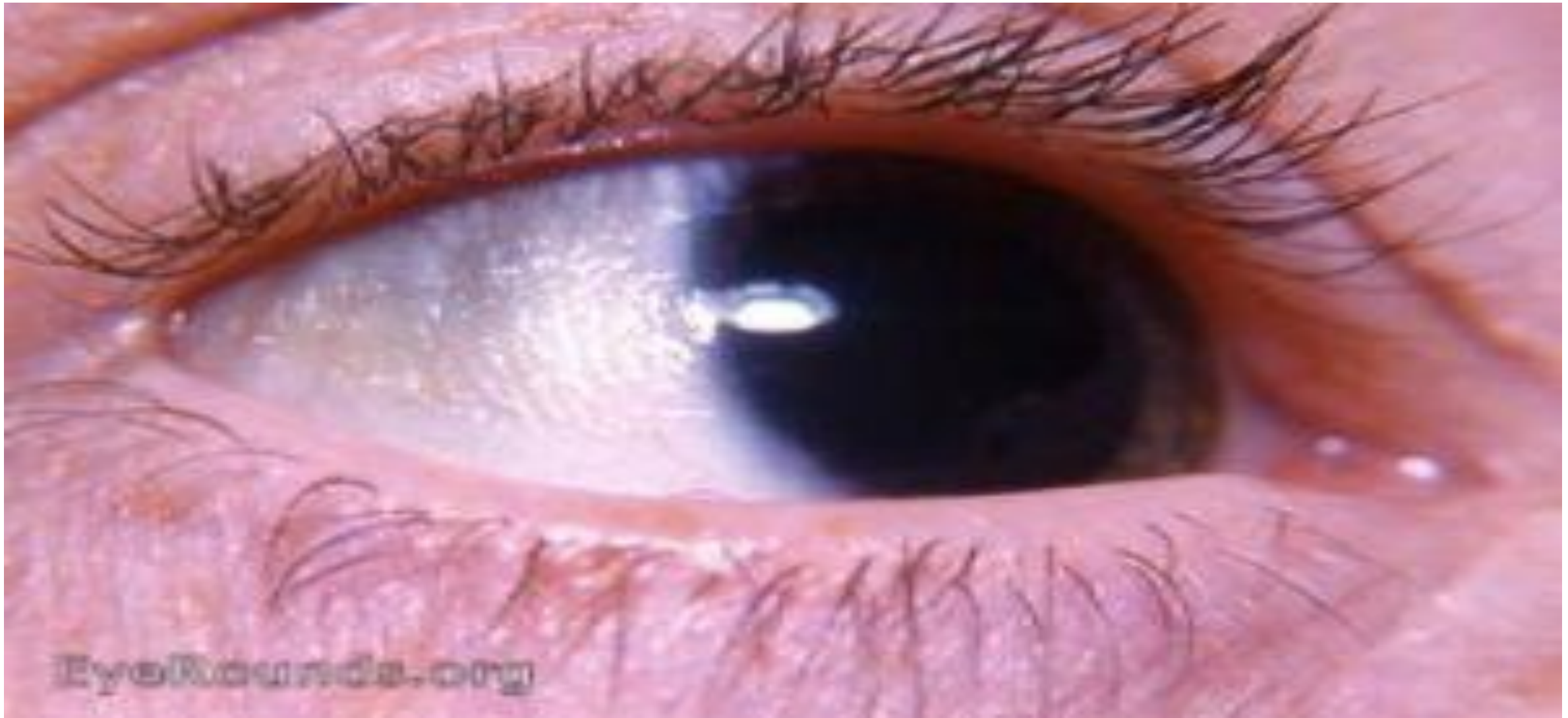
- Dietary sources of preformed vitamin A include dark leafy greens, orange-colored vegetables, fish liver oils, liver, egg yolks, butter, and vitamin A-fortified dairy products. A variety of other foods contain beta-carotene and other provitamin carotenoids, which get converted into vitamin A. These include green leafy and yellow vegetables, carrots, and deep- or bright-colored fruits.
- 80-90% of vitamin A is stored in the Liver

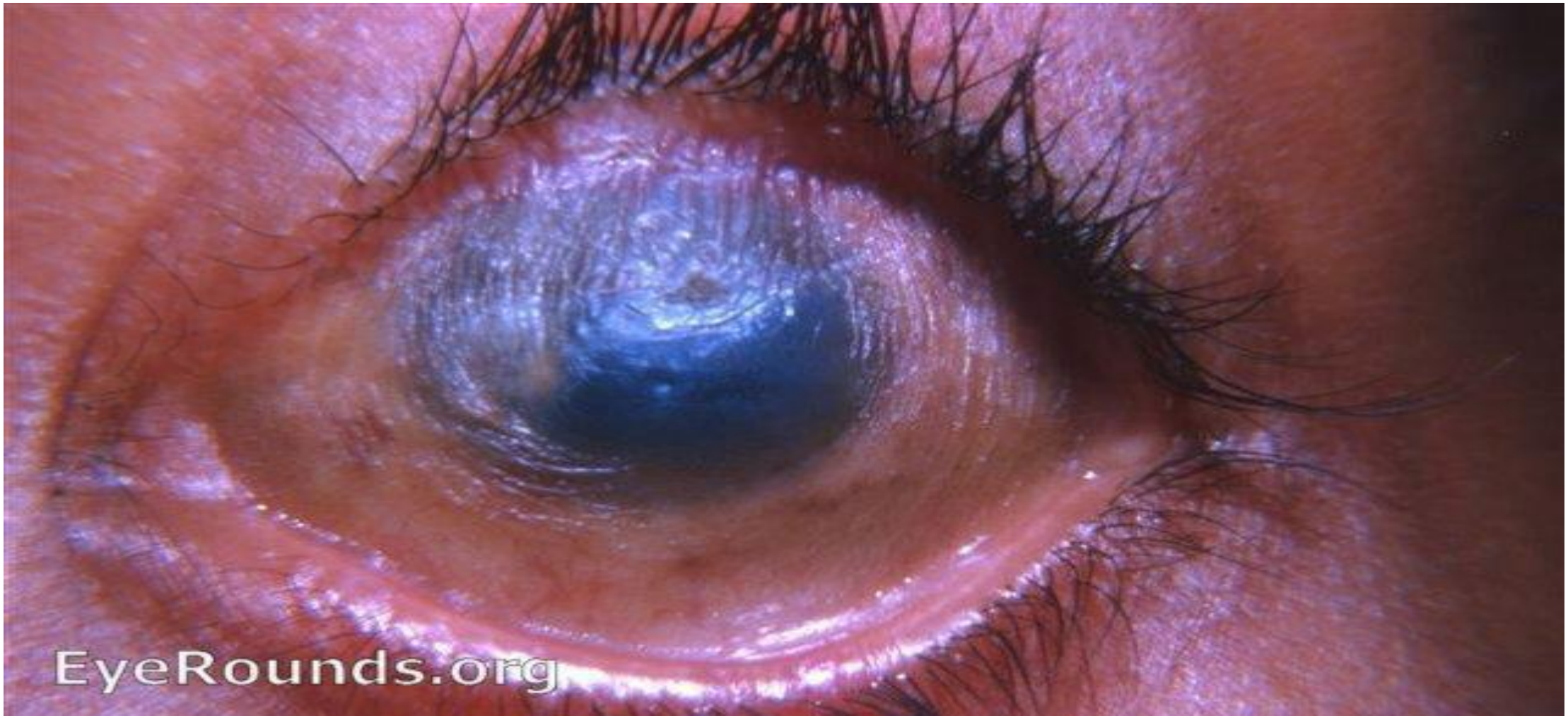
Clinical features

- Night blindess is the earliest symptom
- Dryness & Ocular foreign body sensation
- It appears as soft yellowish subconjunctival mass near the outer canthus with lusterless appearance(bitot’s spot)
- The surface is keratinized & may exhibit hair,

Grading system WHO

Grade of xerophthalmia	Peak age group (years)	Type of deficiency
XN: Night blindness	2-6; adult women	Longstanding. Not blinding
X1A: Conjunctival xerosis	3-6	Longstanding. Not blinding
X1B: Bitot's spots	3-6	Longstanding. Not blinding
X2: Corneal xerosis	1-4	Acute deficiency. Can be blinding
X3A: Corneal ulcer <1/3 cornea	1-4	Severe acute deficiency. Blinding
X3B: Corneal ulcer/keratomalacia1/3 cornea or greater	1-4	Severe acute deficiency. Blinding
XS: Corneal scarring (from X3)	>2	Consequence of corneal ulceration
XF: Xerophthalmos fundus	Adults	Longstanding. Not blinding. Rare


EyeRounds.org


EyeRounds.org

Different stages

- 5-year-old boy with severe autism and an extremely poor diet (only bacon, an occasional blueberry muffin, and Kool Aid) presented with bilateral corneal ulceration.
- **Figure 1** Exam revealed generalized hyperkeratosis and lash hypertrichosis,
- **Figure 2** Bilateral diffuse Rose Bengal staining with Bitots spots at the superior limbus of the left eye,
- **Figure 3** shows numerous yellow flecks in the peripheral retina at the level of the retinal pigment epithelium
- **Figure 4** A conjunctival biopsy showed keratinized conjunctival epithelium
- ***Figure 5*** days later, he developed a corneal descemetocele in the right eye despite intensive antibiotic drop therapy.
- He was managed with a penetrating keratoplasty and tarsorrhaphy as well as punctal occlusion.
- Vitamin A palmitate, 100,000 USP units, was given intramuscularly. A further 50,000 USP units was given 2 months later. 3 months post-operatively, the corneal graft was clear and the ocular surface appeared normal.[16] Images courtesy of Thomas L. Steinemann, MD.

Figure 1

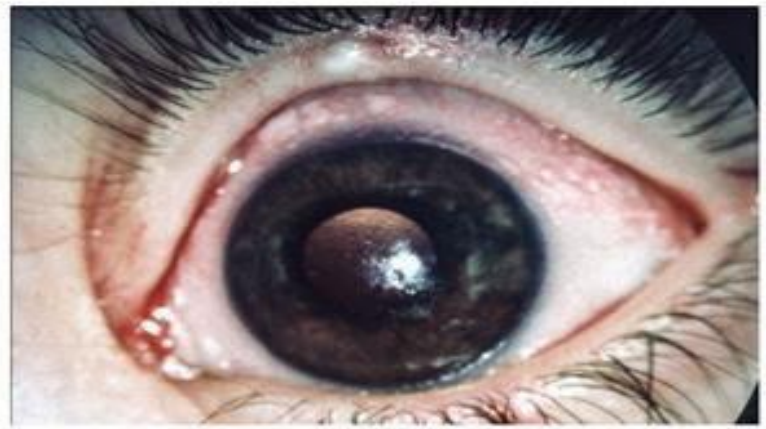


Figure 2

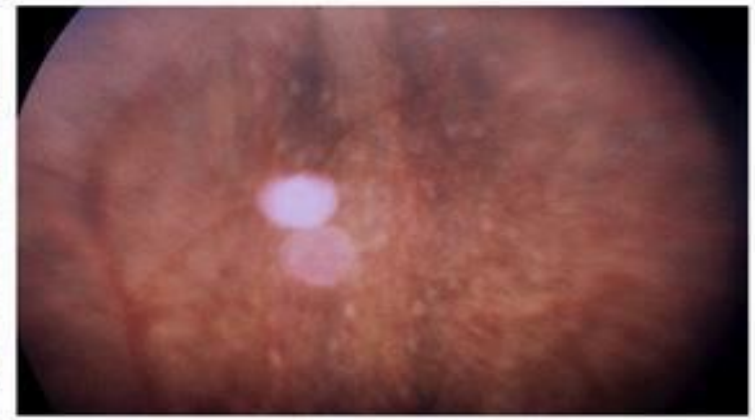


Figure 3

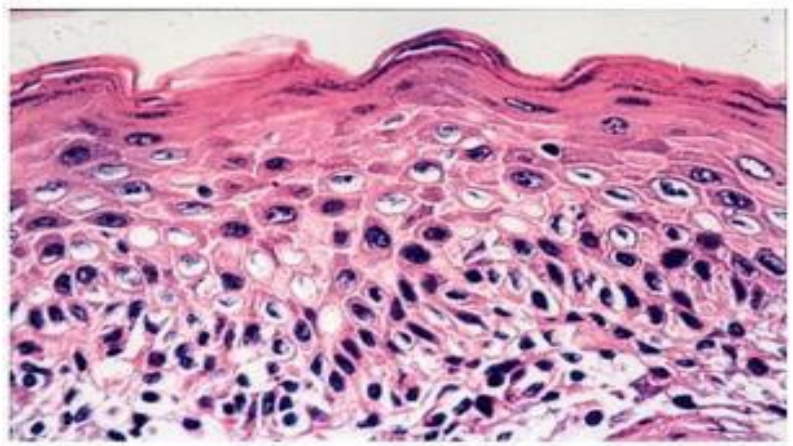


Figure 4

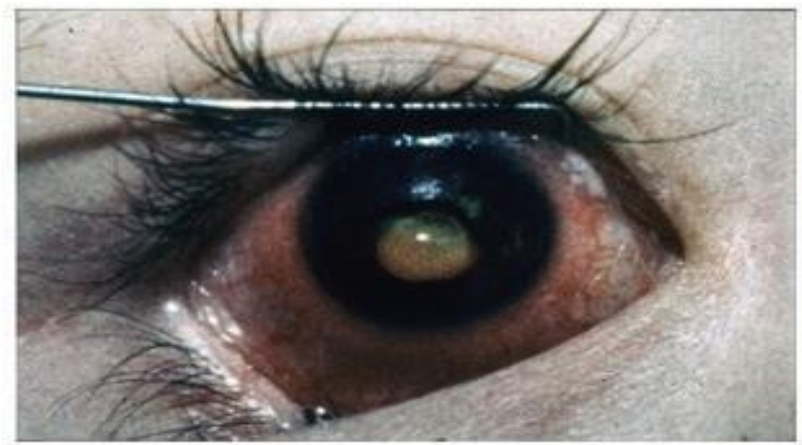


Figure 5

Management

- Keratomalacia should be treated as a medical emergency, as it is an indicator of very severe VAD. High-dose vitamin A is the treatment for all patients, and treatment can either be oral or intramuscular. Recommended Vitamin A deficiency treatment regimens are described in the following table[13]. Treatment can be adjusted as needed based on regular serum retinol level monitoring

Management

Vitamin A dosage (IU)	
Young infants 0-5 mo[1]	50,000
Older infants 6-11 mo[1]	100,000
Children (males: 12 mo or more;females 12 mo to 12 y and 50 y or more)[1]	200,000
Women (13-49 y) withnight blindness and/or Bitot's spots	10,000 every day or 25,000 every week for at least 3 mo
Women (13-49 y) with activecorneal lesions	200,000 on days 1, 2, and 14

Schedule of Vitamin A doses

- Sever malnutrition, Day 1
- Measles , Day 1& Day 2
- Xerophthalmia, day1 day 2 day 14

- Serum vitamin A/retinol normal range: 20-60 mcg/dL. These levels can be normal due to maintenance of circulating retinol levels by hepatic stores.
- VAD-related ocular symptoms have been shown to develop at concentrations <10mcg/dL.
- Serum retinol binding protein (reference range: 30-75 ug/ml).
- Serum zinc (reference range: 75-120 mcg/dL)
-


بسم الله الرحمن الرحيم

Red eye 3

CORNEAL ULCER General

Dr Nazullah

Associate professor

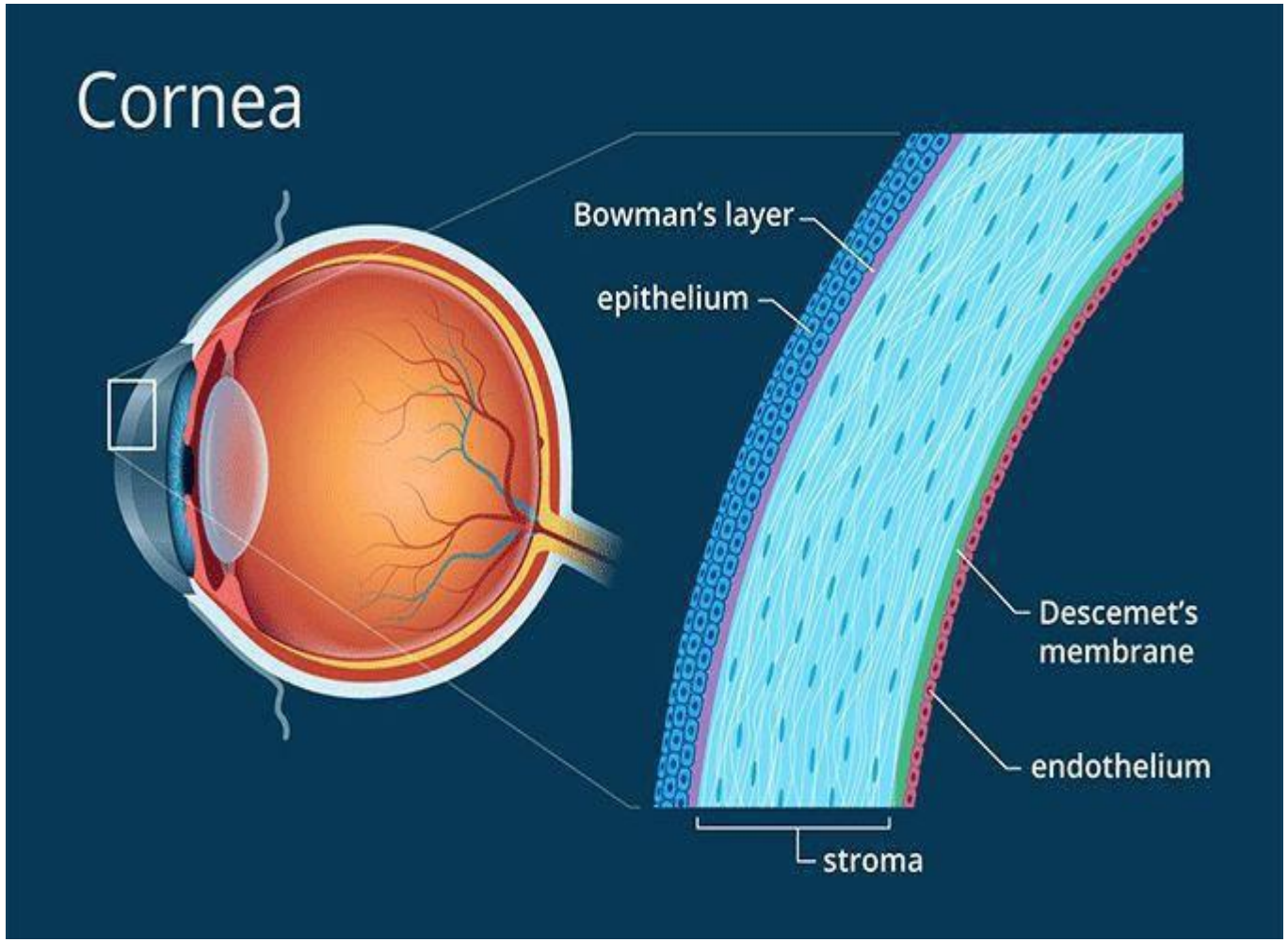
Cornea rounded convex transparent structure forming the anterior 1/6 of the eye globe

- Functions
- Refraction/ vision
- Shape to the globe
- Protection of the underlying structures

Layers .???

Layers

- Epithelium
- Bowmens layer
- Stroma
- Descemet membrane
- Endothelium
- Total thickness
- Central 500- 550
- Periphral 600-650



Extra 6th layer

- The Dua's layer was discovered by Harminder Singh Dua in 2013. It is found between the stroma and Descemet's membrane. It is made up of five to eight thin and compact layers of lamellae of type I and type VI collagen. The layer may be sparsely or not at all populated with keratocytes.
-

Scenario

- A patient age 50 years comes to eye emergency unit. He is complaining of severe pain redness and loss of vision Rt eye for the last few days. On examination he cannot open his Rt eye. His vision is HM & 6/6 in the Lt eye. There is a rounded lesion with whitish margins in the Rt eye cornea, pus in the a/c, severe conjunctival congestion and watering. Left eye is grossly normal. What is the most cause. ?
- A acute anterior blepharitis
- B acute anterior uveitis
- C acute bacterial conjunctivitis
- D bacterial corneal ulcer
- E viral corneal ulcer

- 

- A male age 45 yrs come to eye opd with itching foreign body sensation watering & blurred vision Lt eye for last 15-20 days. On examination his vision is 6/12 Lt eye & 6/6 in Rt eye. There is a corneal lesion with branching pattern & conjunctival congestion. a/c is quiet. Rt is insignificant. He gives hx of fever few days back.
- What is the most probable diagnosis
- A bacterial keratitis
- B fungal keratitis
- C traumatic keratitis
- D viral keratitis

Keratitis / Corneal ulcer

- It is the inflammation of the cornea
- The local defect & excavation of the corneal surface produced by the sloughing off, of the inflammatory tissues

Classification/Types

- Two main types
- Microbial
- Bacterial
- Viral
- Fungal
- Protozoal Acanthamoeba
- Chlamydia trachomatis

Non-microbial

- Allergic (hypersensitivity) such as vkc (Type I) & Phlyctenular Type IV
- Autoimmune such as Mooren ulcer & Peripheral ucer in collagen disease
- Neuroparalytic such as seventh n palsy
- Neurotropic such as lesion of ophthalmic branch of 5^{th} nerve & DM
- Exposure keratopathy such proptosis
- Nutritional such as vit A deficiency & protein deficiency
- Tear film deficiency such as k-c-sicca & xerophthalmia
- Chemical burns such as acid & alkali burns
- Eyelid abnormalities such as trichiasis & entropion

Clinical features

- Pain
- Redness
- Lacrimation
- Photophobia
- Blurred vision
- Fb sensation
- Discharge

Signs

- Corneal epithelial erosions
- Corneal Epithelial edema
- Subepithelial infiltrates
- Corneal filaments
- Corneal ulceration
- Corneal vascularization
- Folds & breaks in Descemet membrane
- Descemetocele

Clinical feature

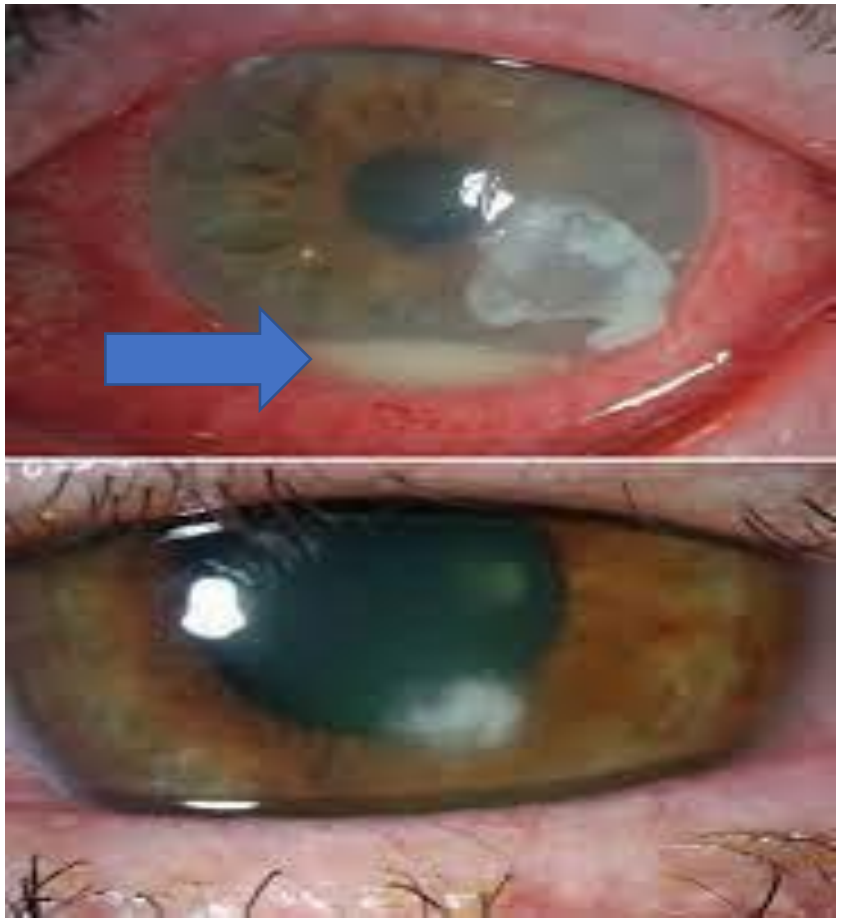
- There is severe pain, redness, lacrimation, decreased vision photophobia, & blepharospasm ie the patient cannot open the eyes.
- On examination It appears as greyish white swollen cornea with necrosis at the base. Cornea stain positive with fluorescence
- Conjuctinal Hyperemia,
- Hypopyon ie pus formation & exudates in the a/c which is due to increased amount of cellular infiltrates in A/C,
- Anterior uveitis due to inflammation of iris and ciliary body due toxin & cytokines released by these inflammatory cells
- Lid edema may be there

Clinical features

- Infiltrative stage Any injury which damages the epithelium leads to polymorphonuclear attraction leading to yellow white corneal infiltrates with epithelial edema
- Active & necrotic stage There is necrosis & sloughing of the epithelium with excavation and ulcer formation. The chemical mediator are released from ulcerated area which produces the features ie congestion discharge hypopyon etc etc

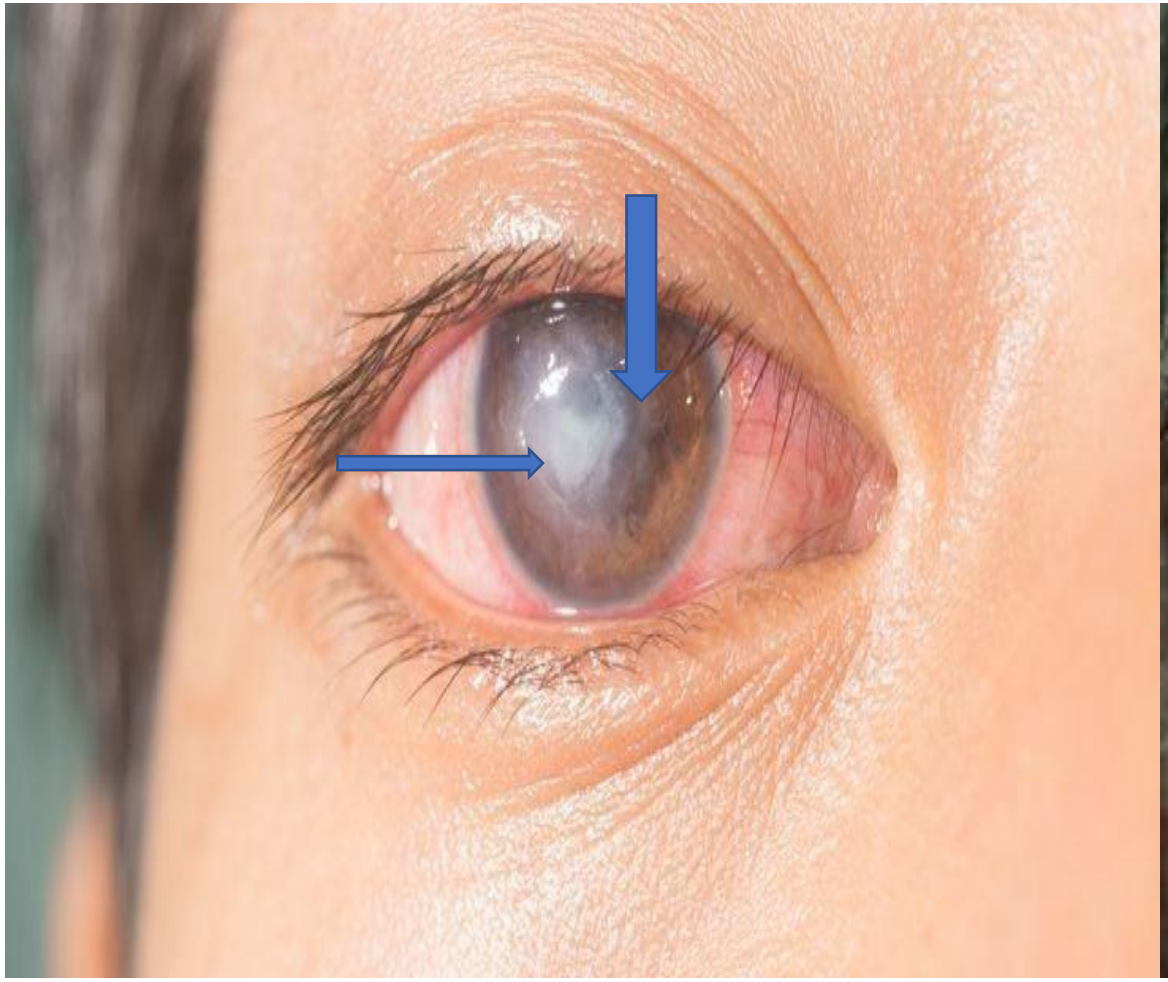
- Regression There may be regression of the ulcer due to host natural protective mechanism. A line of demarcation between ulcerated & normal clear cornea.
- Healing & scarring may start by epithelialization of the ulcer leading to scarring as a result of new stromal lamellae formation by keratocyte.

Corneal ulcer with hypopyon



fluorescein stain +

Regression



Healing/Scarring

Management

- Mostly treated in outpatient
- Admission may be needed if
- Large ulcer
- Resistant to the previous med
- From far flange area

Management Contd

- Proper history of any trauma specially nature & time of trauma, any associated ocular disease eg dry eyes etc & ocular medication such as steroid.
- Systemic history of any chronic illness or any medication specially of any steroid or other immunosuppressive medication should be moted.
- Clinical findings detail examination with slit-lamp. If needful then fluorescence staining should be done to confirm the diagnosis. Corneal sensitivity should be checked. The size, shape & color of the ulcer ie height & width should be measured & documented. Then depth weather superficial or deep, margins of the ulcer weather clear rounded , feathery margins , satellite lesion should be described & noted.
- Anterior chamber(AC) should be focused for depth, reaction ie cells, flare and hypopyon should be noted. The size ie length & width of hypopyon should be noted daily.

Investigations

- Routine blood cp with ESR urea sugar urine exam
- Swab from Discharge with can be used for examination.
- Corneal scraping specifically from margins & bed of the ulcer, with topical analgesic under microscope, is done by spatula/20 gauge syringe. Care should be taken to avoid perforation
- a) Direct examination under microscope for any fungal hyphae with KOH.
- b) Gram staining for gram positive & negative.
- c) For culture sensitivity in different culture media.

Treatment

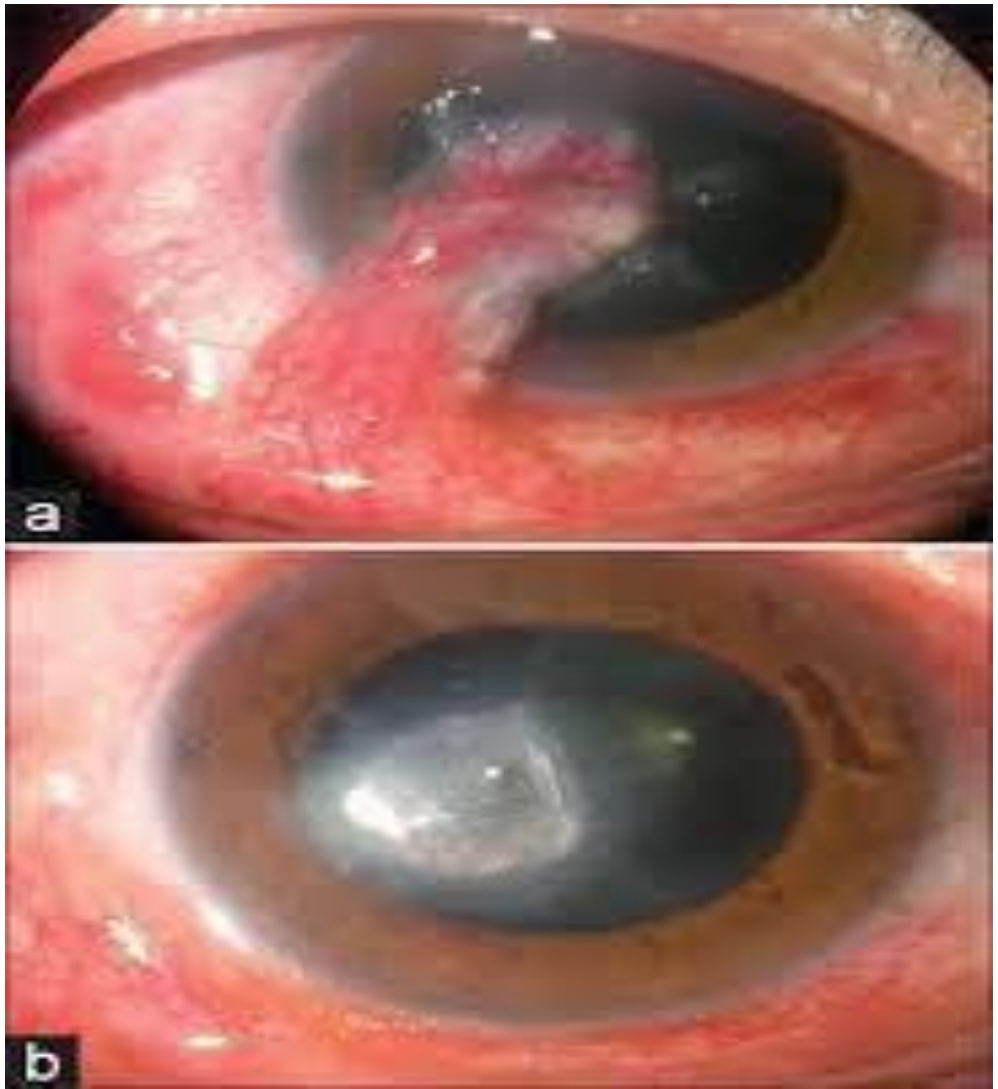
- A) Medical according to the pathogens involved, but broadly the following medication
- Topical antibiotics for infection control like tobramycin gentamycin ofloxacin ciprofloxacin moxifloxacin. Single or in combination, drops & in oint form. Frequency varies from half hrly to four times daily
- Cycloplegic cyclopean to relieve pain , pupil dilatation to prevent synechia formation & reduce exudation by decreasing the vascular permeability.
- Analgesic to relieve pain
- Anti glaucoma to reduce intraocular pressure (IOP) betablocker timolol etc

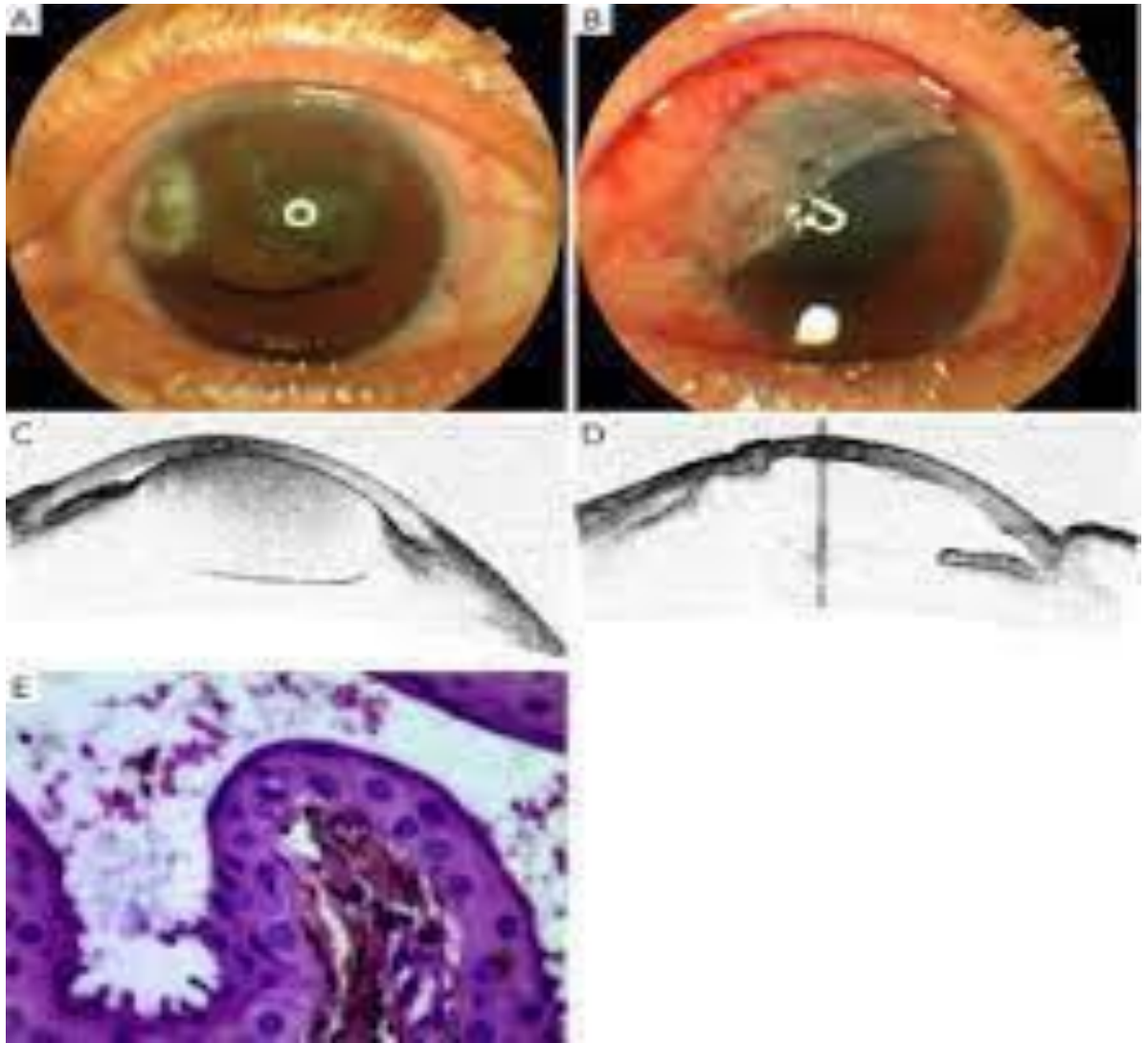
- Systemic
- Antibiotics oral/iv
- Analgesic oral/iv
- Antiglaucoma to reduce IOP acetazolamide AZM
- B) Bandage contact lens may b applied for mechanical support
- In resistant & non healing cases & very thin cornea

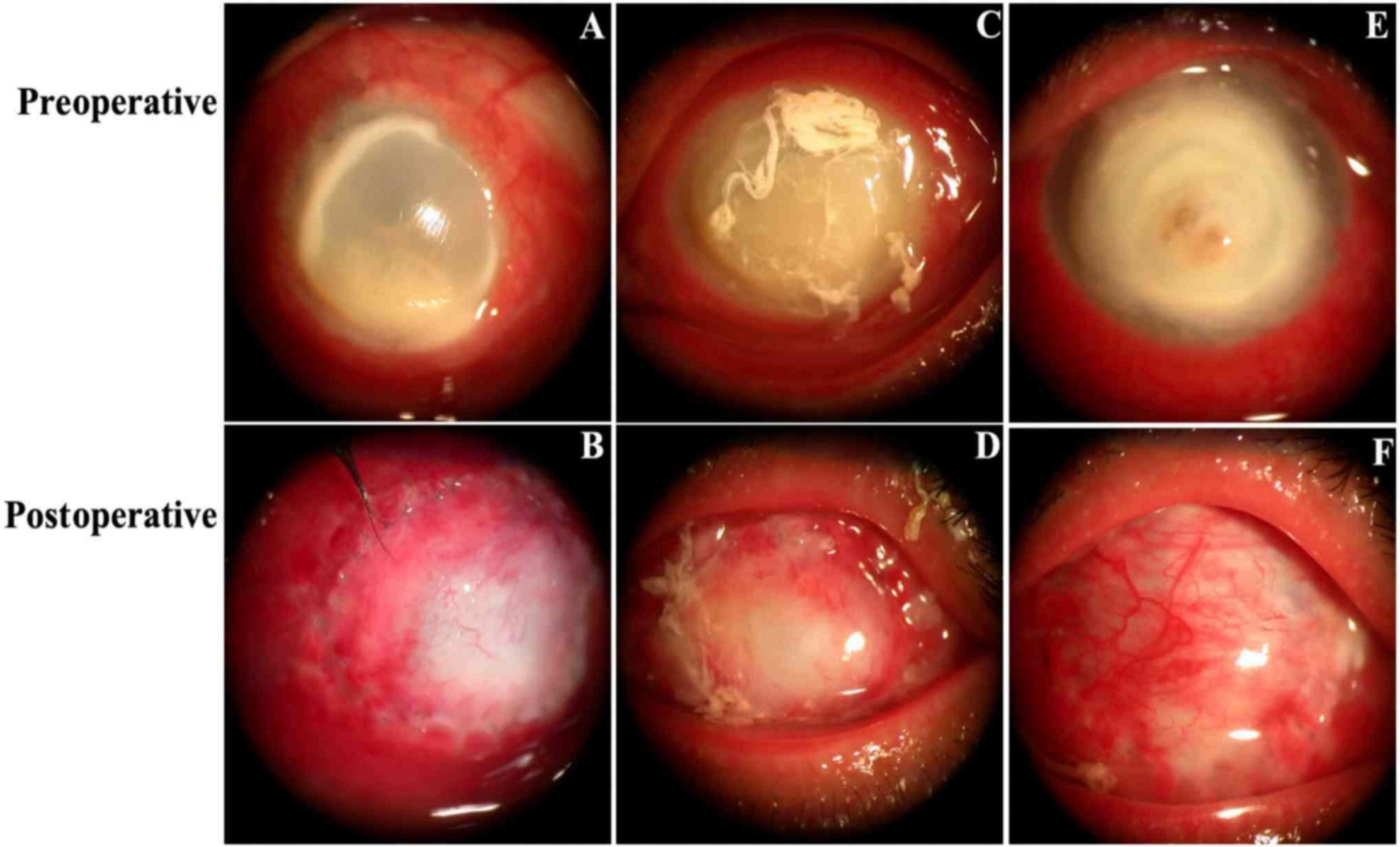
- C) Surgical in resistant & corneal thinning with threatened perforation
- Pressure bandage
- Tarsorrhaphy
- Amniotic membrane transplant AMT
- Conjuctival flape
- Tectonic graft
- PKP

BCL

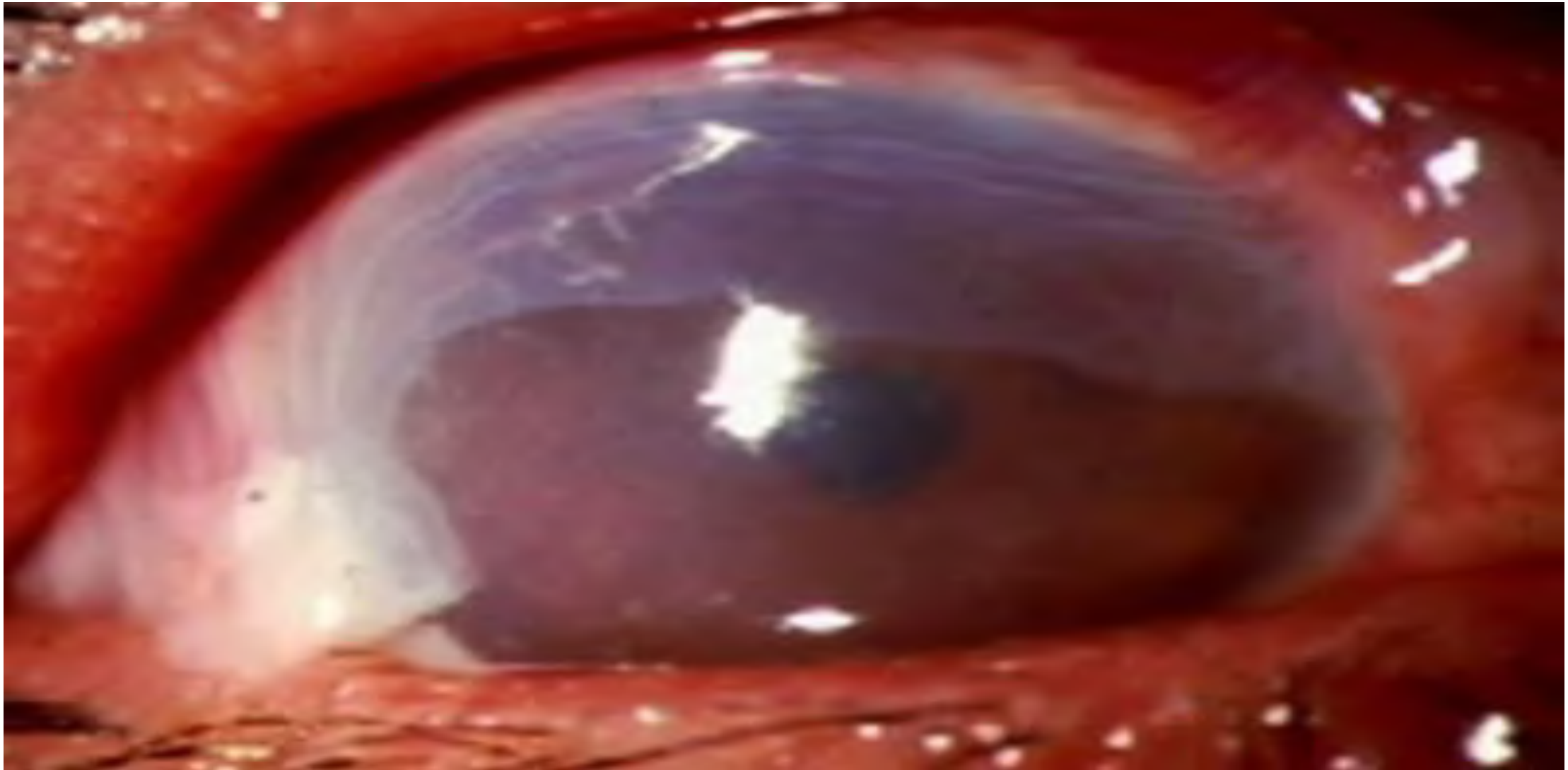
Coj flape






Preoperative
Postoperative
A
B
C
D
E
F

AMT for corneal ulcer

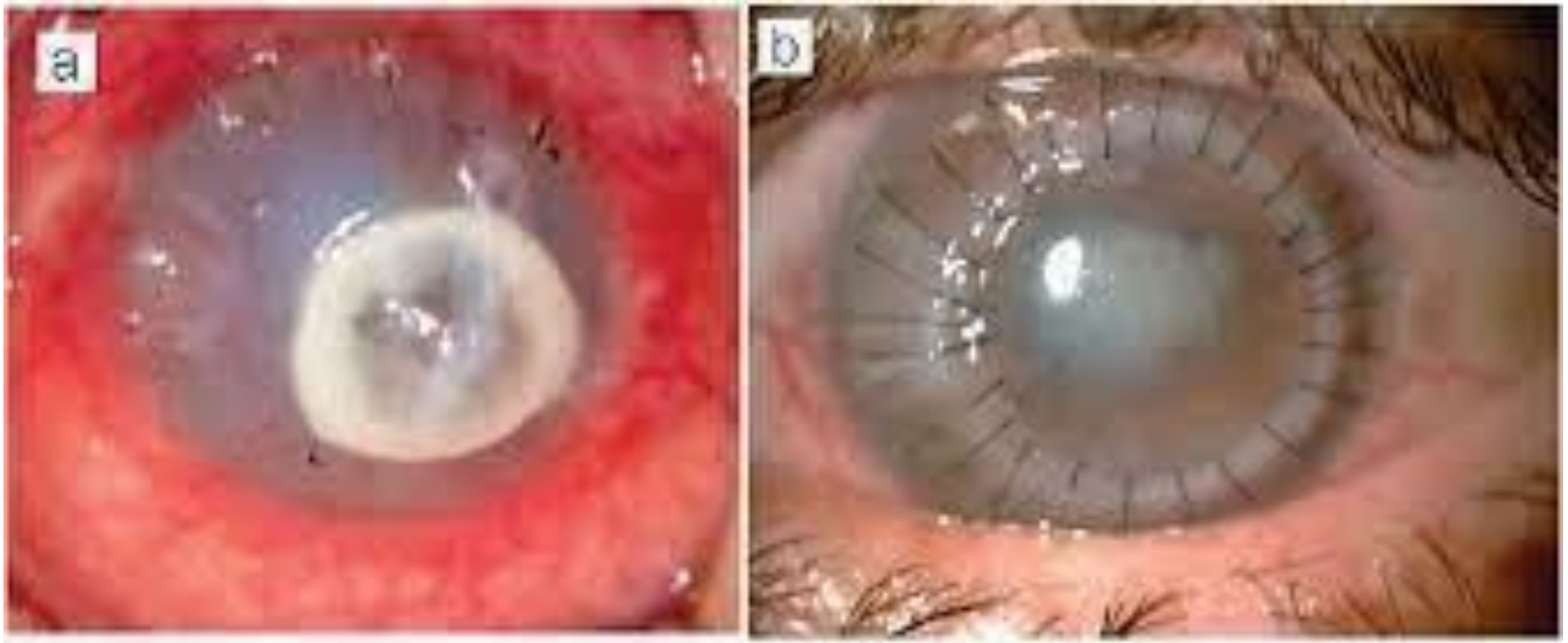


Tarsorrhaphy

Tectonic graft

Figure 1: Right Eye Corneal perforation with iris prolapse and a shallow anterior chamber

Extensive corneal ulcer with full PKP graft

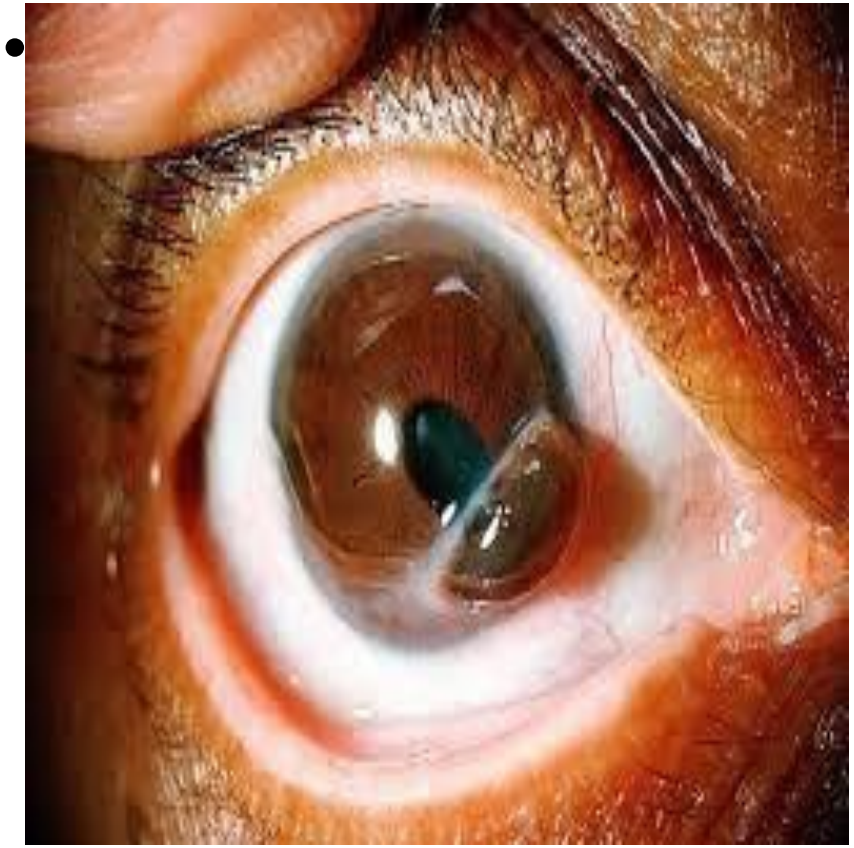


Complication

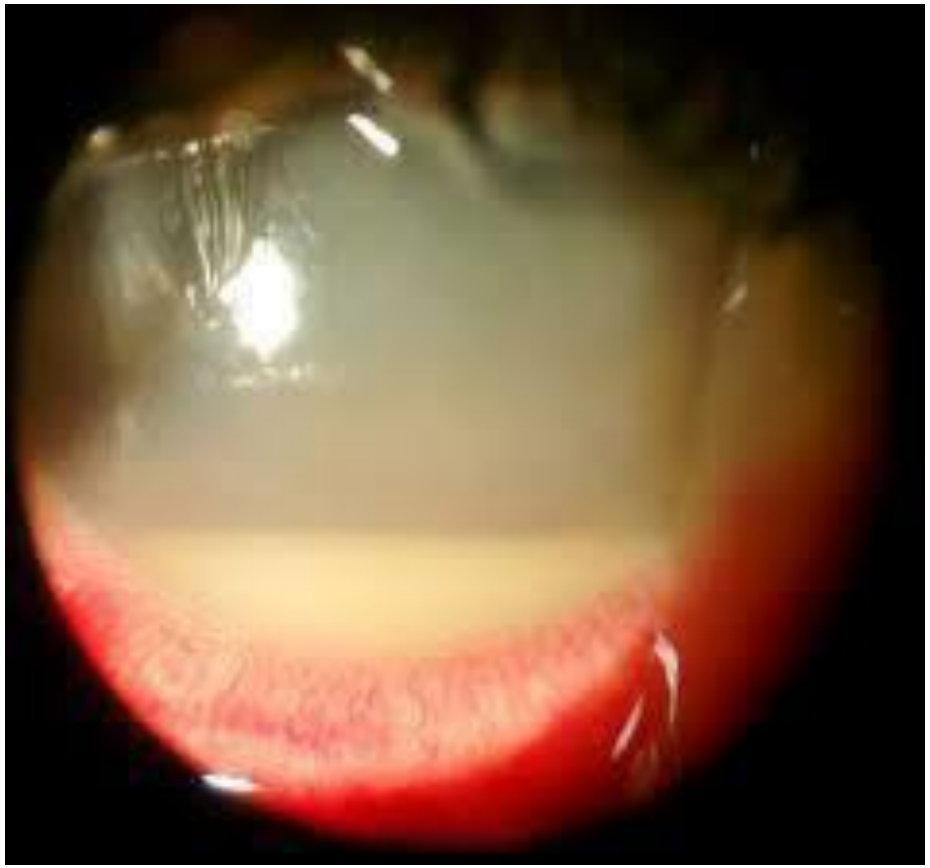
- Corneal opacity formation. Nebula, macula & leucoma according to opacity density
- Corneal thinning descemetocele formation
- Corneal astigmatism / corneal ectasia
- Anterior staphyloma abnormal anterior protrusion of the cornea with uveal tissue
- Uveitis due spread of the infection & inflammation to the iris
- Secondary glaucoma due exudate blocking the angles
- Corneal perforation

- Iris prolapse through the cornea
- Flat anterior chamber
- Synechia formation anterior & posterior
- Lens subluxation
- Cataract formation
- Endophthalmitis
- Panophthalmitis
- Intraocular hg
- Retinal detachment

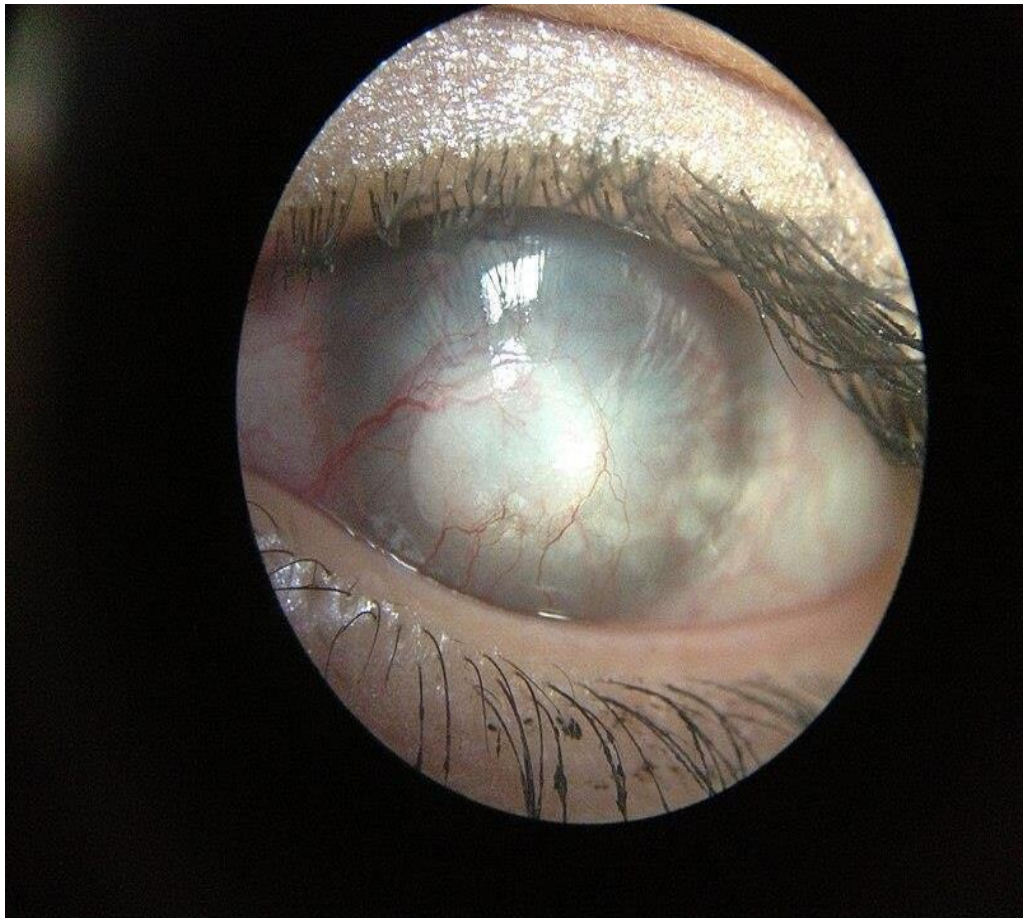
Iris prolaps



Endophthalmitis

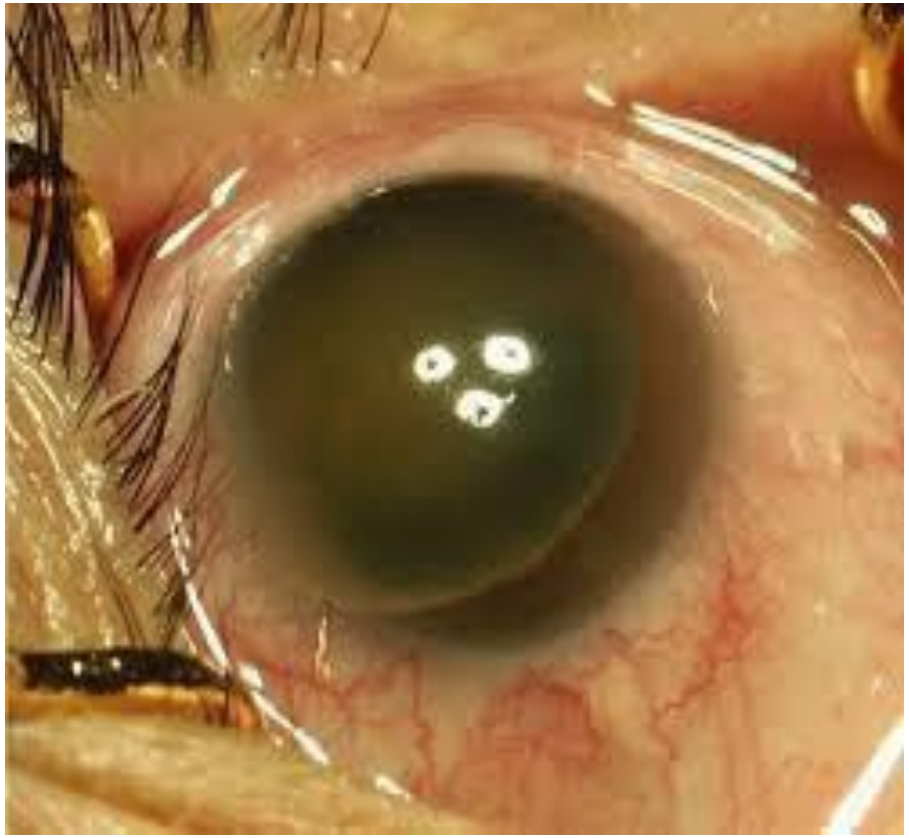


Opacity



Secondary glaucoma

Lens dislocation



Subluxation of the lens in Marfan's syndrome.

Fig. 7.23 The lens is displaced superiorly and medially. As the zonule fibers are intact, a certain measure of accommodation is still possible.

- Thanks


بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

Red eye/Blepharitis

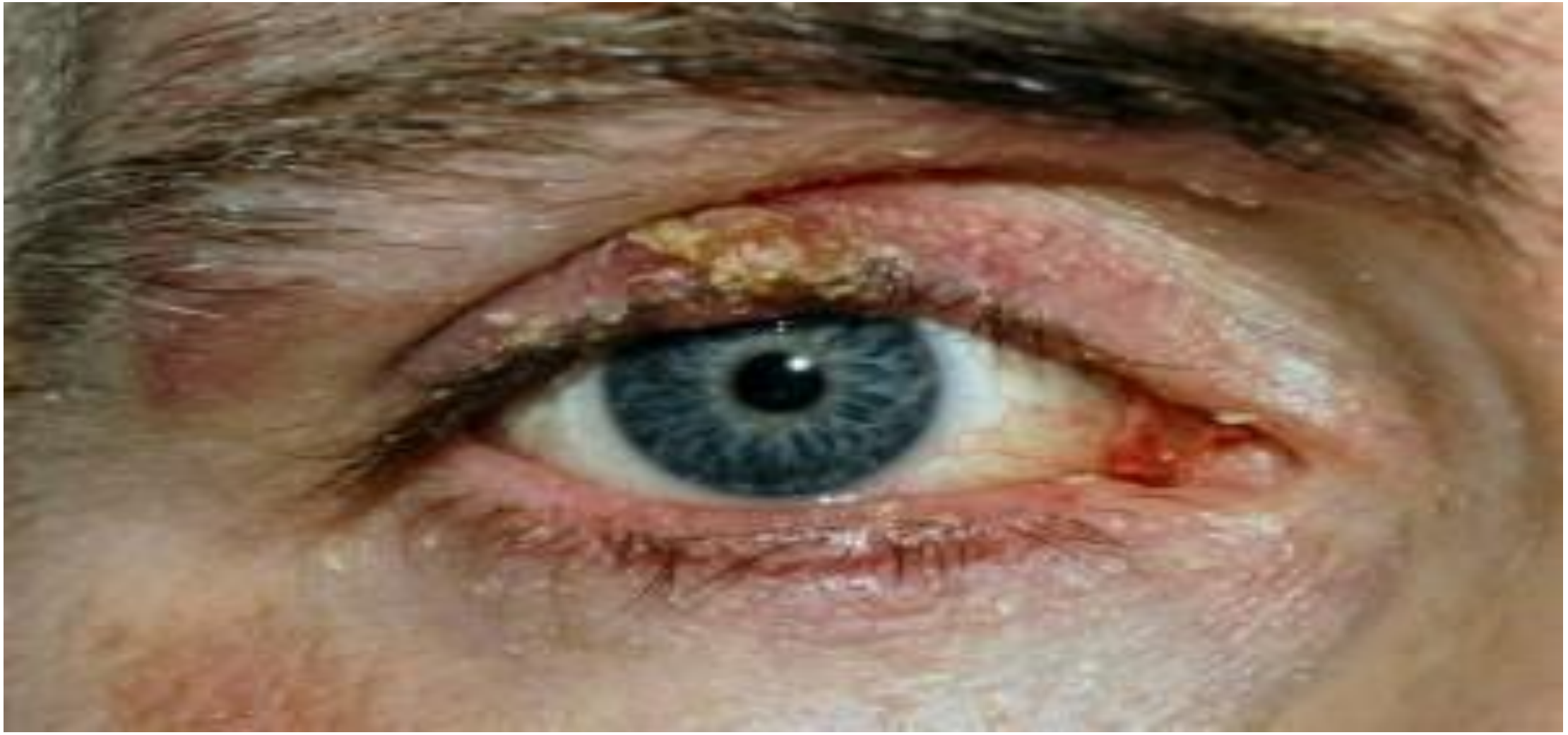
Dr Nazullah

Associate professor

Ophthalmology HMC/KGMC

Scenario

- A patient of age 25 yrs come to eye opd with itching foreign body sensation watering Bt eye for last few yrs. On examination his vision is 6/6 BEs. He is also complaining of recurrent chalazian formation.
- What is the most probable diagnosis
- A allergic conjunctivitis
- B vernal conjunctivitis
- C anterior blepharitis
- D anterior uveitis



- It is the subacute/ chronic inflammation of the eye lid margins.
  Is a common cause of ocular discomfort and irritation in all age and ethnic groups.
  While generally not sight-threatening, it can lead to permanent alterations in the eyelid margin or vision loss from superficial keratopathy, corneal neovascularization, and ulceration.

Lid margins

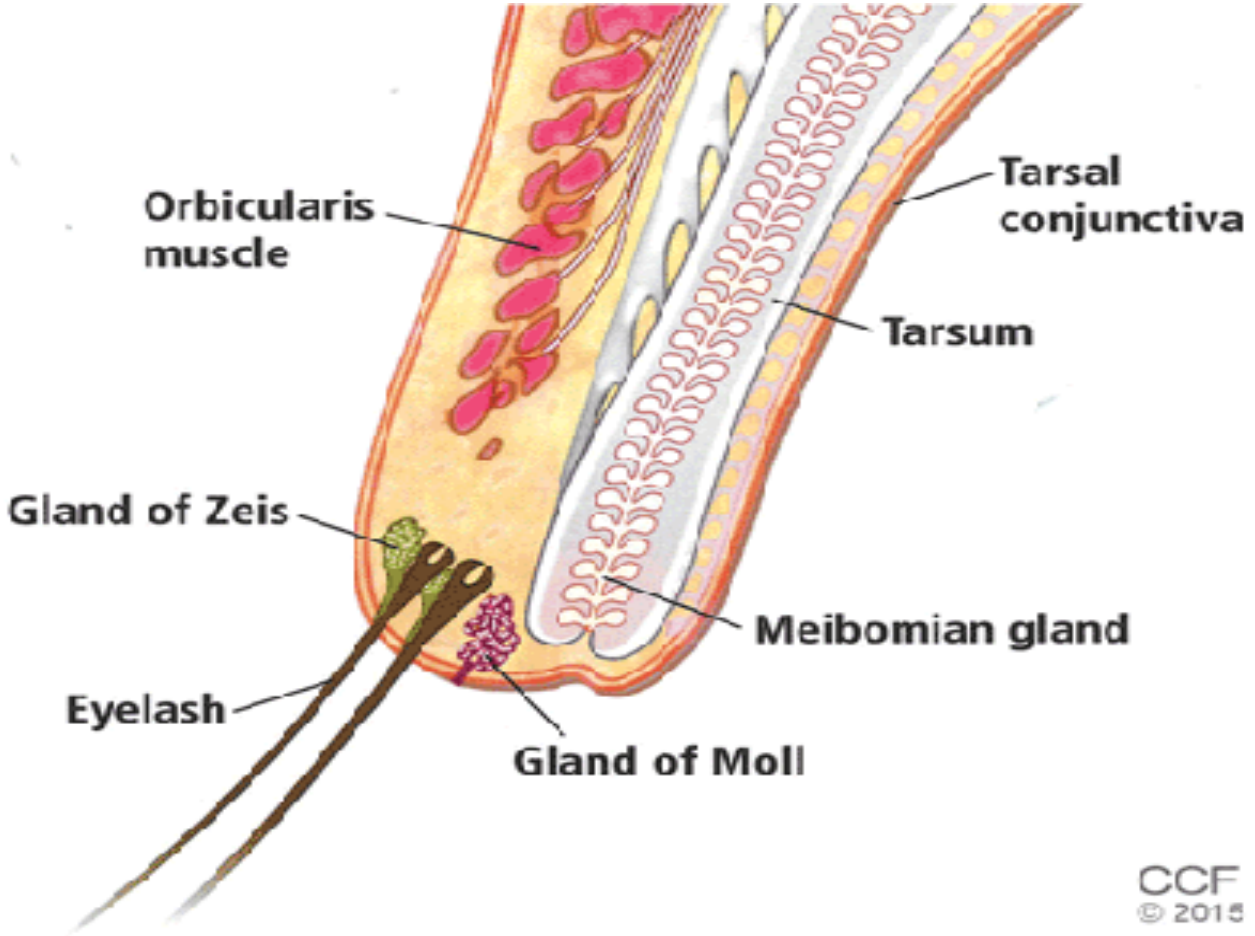
- Gross anatomy. ??

- Types,??

Lid margin types

Hordeolum an acute abscess within an eyelid gland, usually staphylococcal in origin

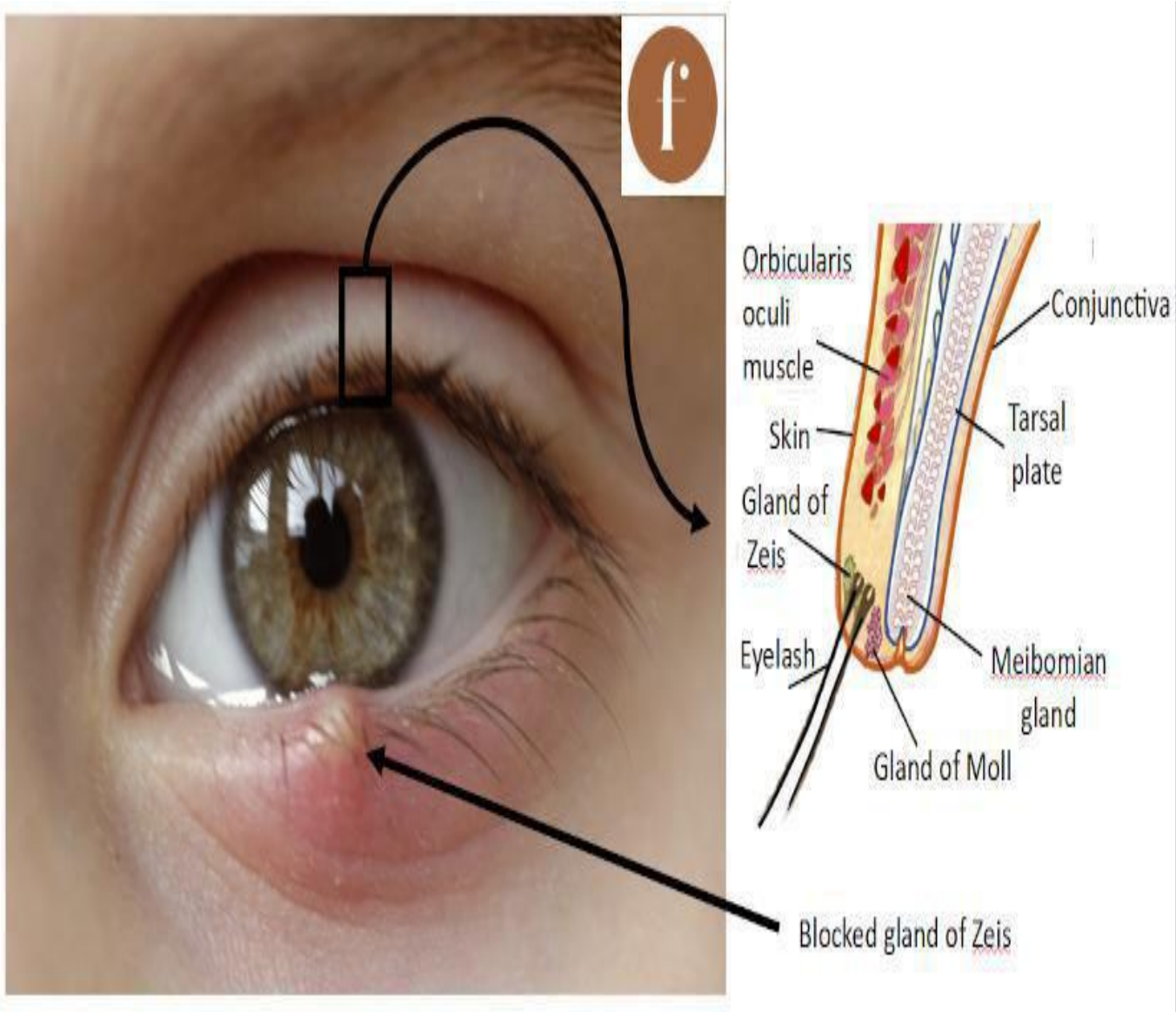
- When it involves a
- meibomian gland
- it is termed an
- *internal hordeolum*,
- and when it involves
- the gland of Zeis or
- Moll it is termed an
- *external hordeolum*

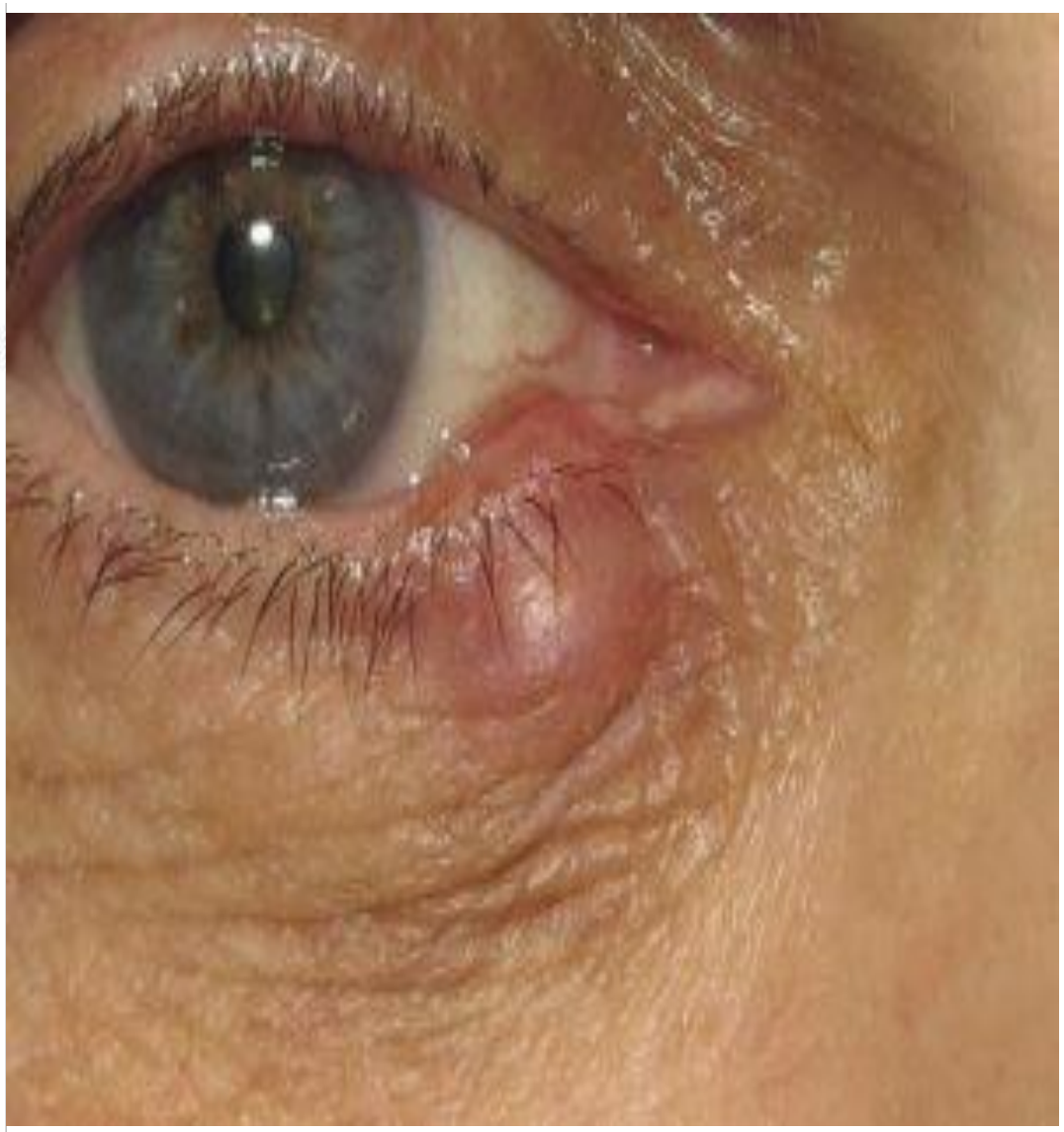


Chalazion

- A is a chronic
- lipogranuloma
- due to leakage of
- sebum from an
- obstructed
- meibomian gland.

ɔ

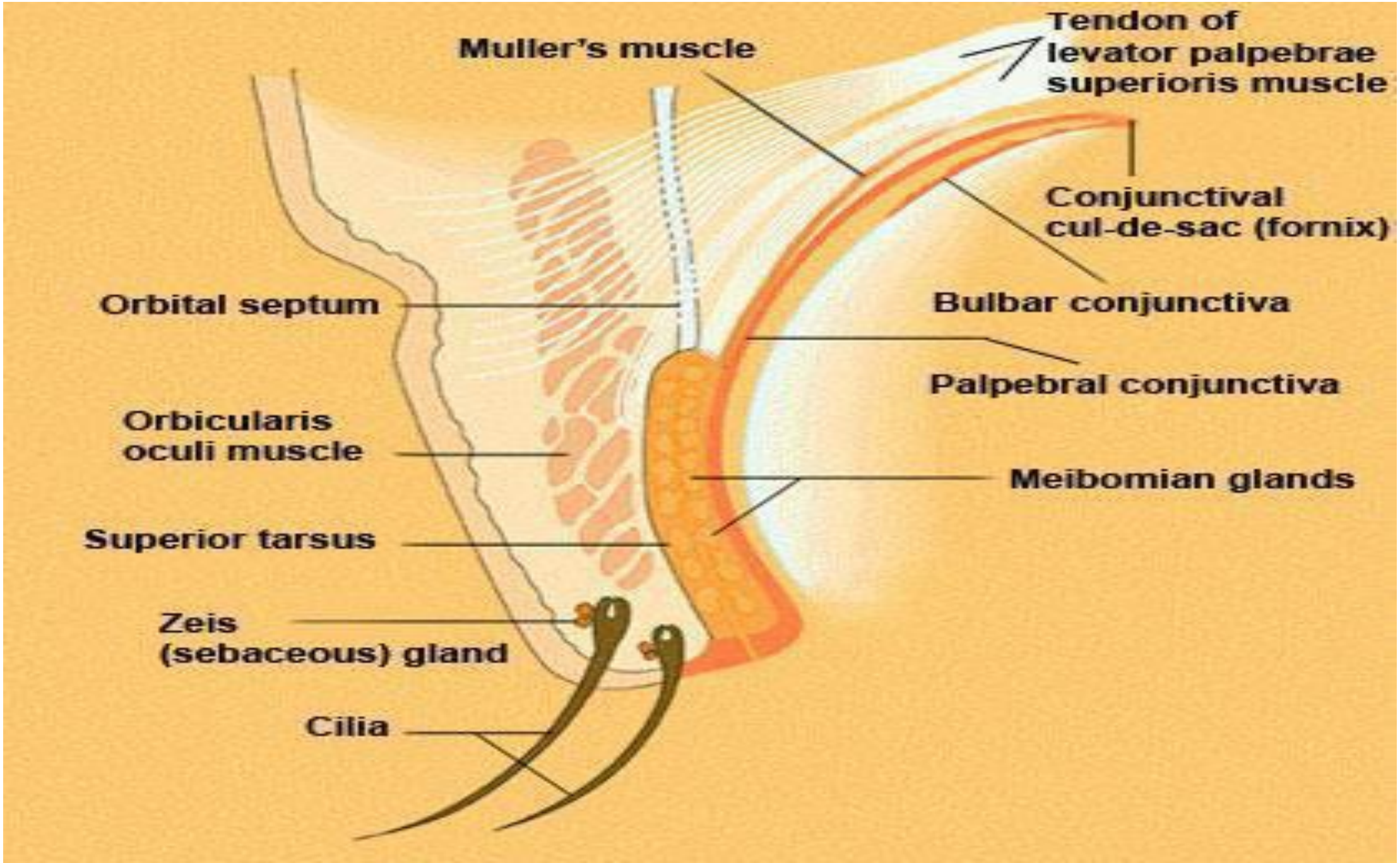

f
Orbicularis oculi muscle
Skin
Gland of Zeis
Eyelash
Conjunctiva
Tarsal plate
Meibomian gland
Gland of Moll
Blocked gland of Zeis



Layers of lid

- Anterior lamella
- Skin
- Orbicularis muscle

- Posterior
- Tarsal plate
- Conjunctiva


Muller's muscle
Tendon of levator palpebrae superioris muscle
Conjunctival cul-de-sac (fornix)
Orbital septum
Bulbar conjunctiva
Palpebral conjunctiva
Orbicularis oculi muscle
Meibomian glands
Superior tarsus
Zeis (sebaceous) gland
Cilia

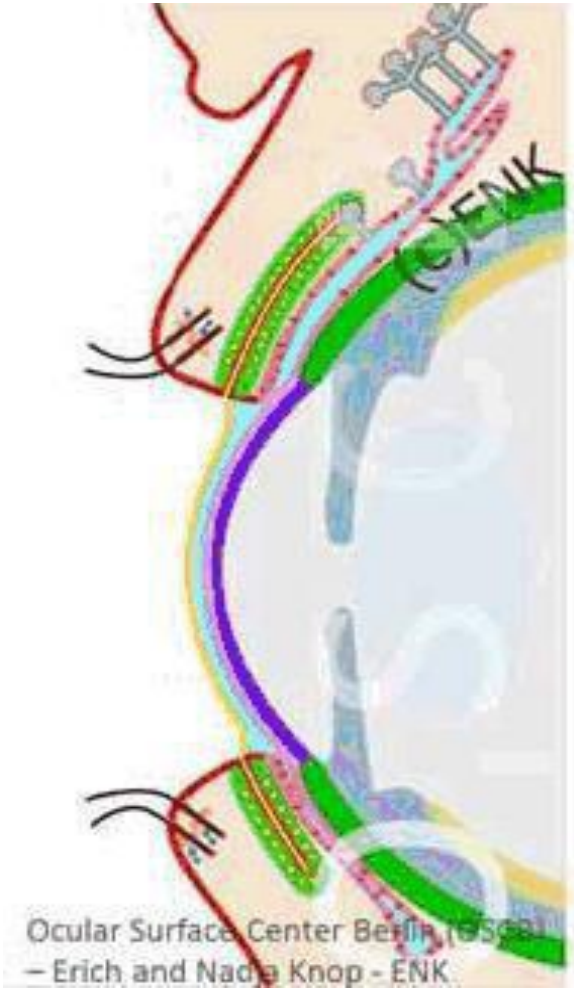
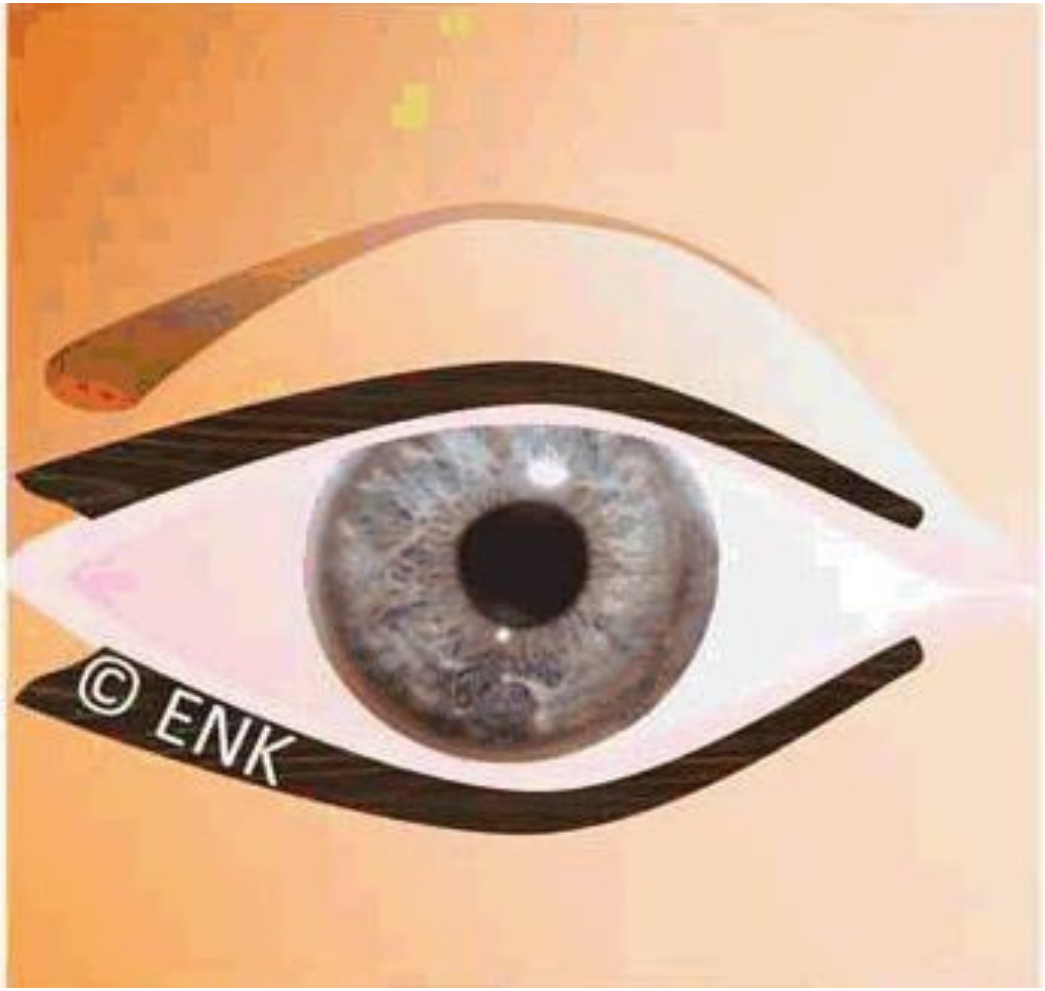
Functions Of Eyelids:

I. Act to protect the anterior surface of the globe from local injury.

II. Aid in regulation of light reaching the eye.

III. Tear film maintenance by distributing the protective optically important tear film over the cornea during blinking.

IV. Tear flow by their pumping action on the conjunctival sac and lacrimal sac.

- 
© ENK
Ocular Surface Center Berlin (OSCB)
– Erich and Nadja Knop - ENK

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Cont...

- Functions:
  - Forms hydrophobic barrier at the margin of the eyelid,preventing spillage of tears at the lid margin
  - Forms oily layer of tear film over cornea &bulbar conjunctiva

Retards evaporation of tears.

- Eyelids acts as shutters protecting the eye from injuries and excessive light
- Help in spreading tear film over cornea and conjunctiva via blinking and also helps eliminate tears from lacrimal lake
- Contribute to facial features of the individual
- Relay information regarding the state of wakefulness and attention of the person

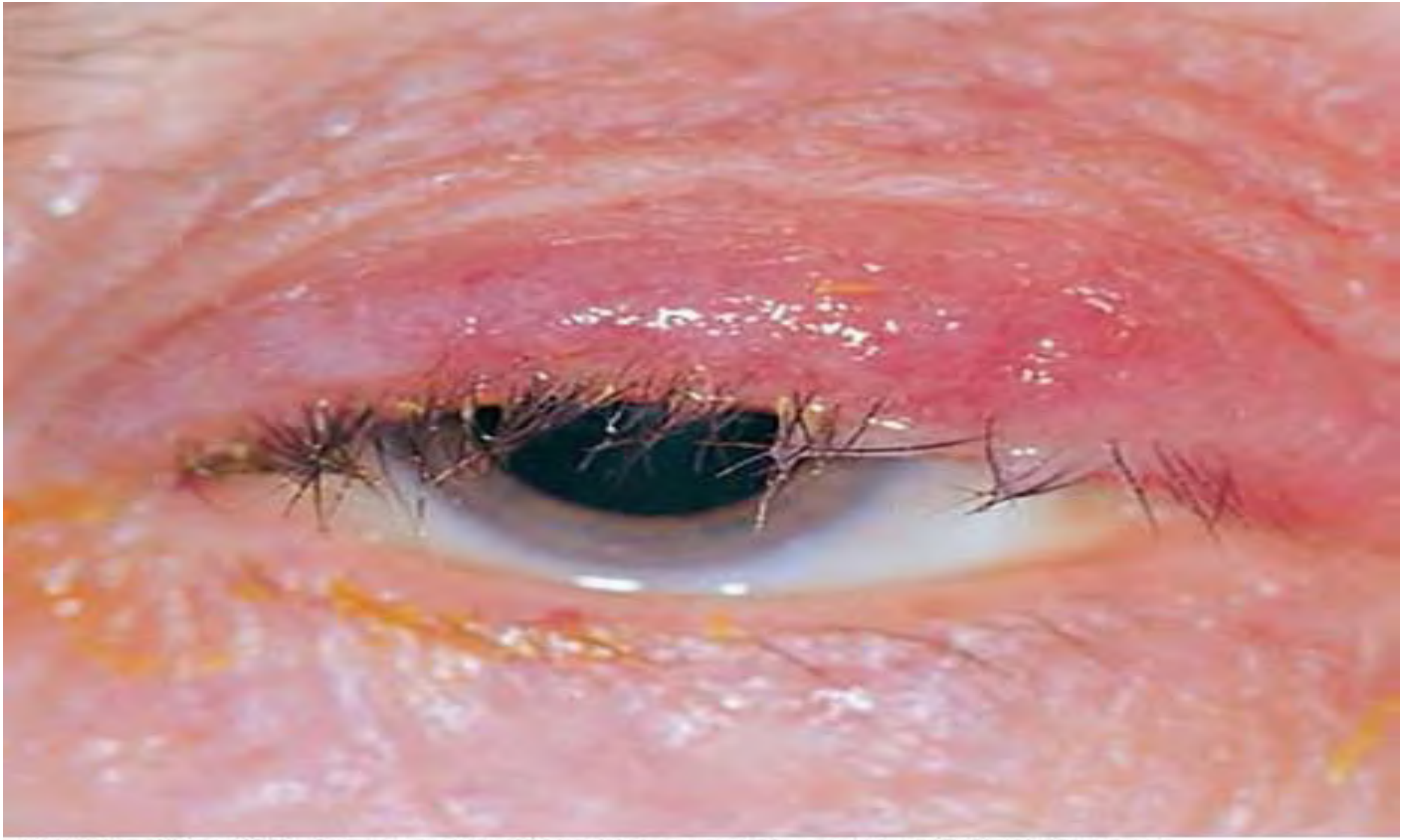
Eyelids Function

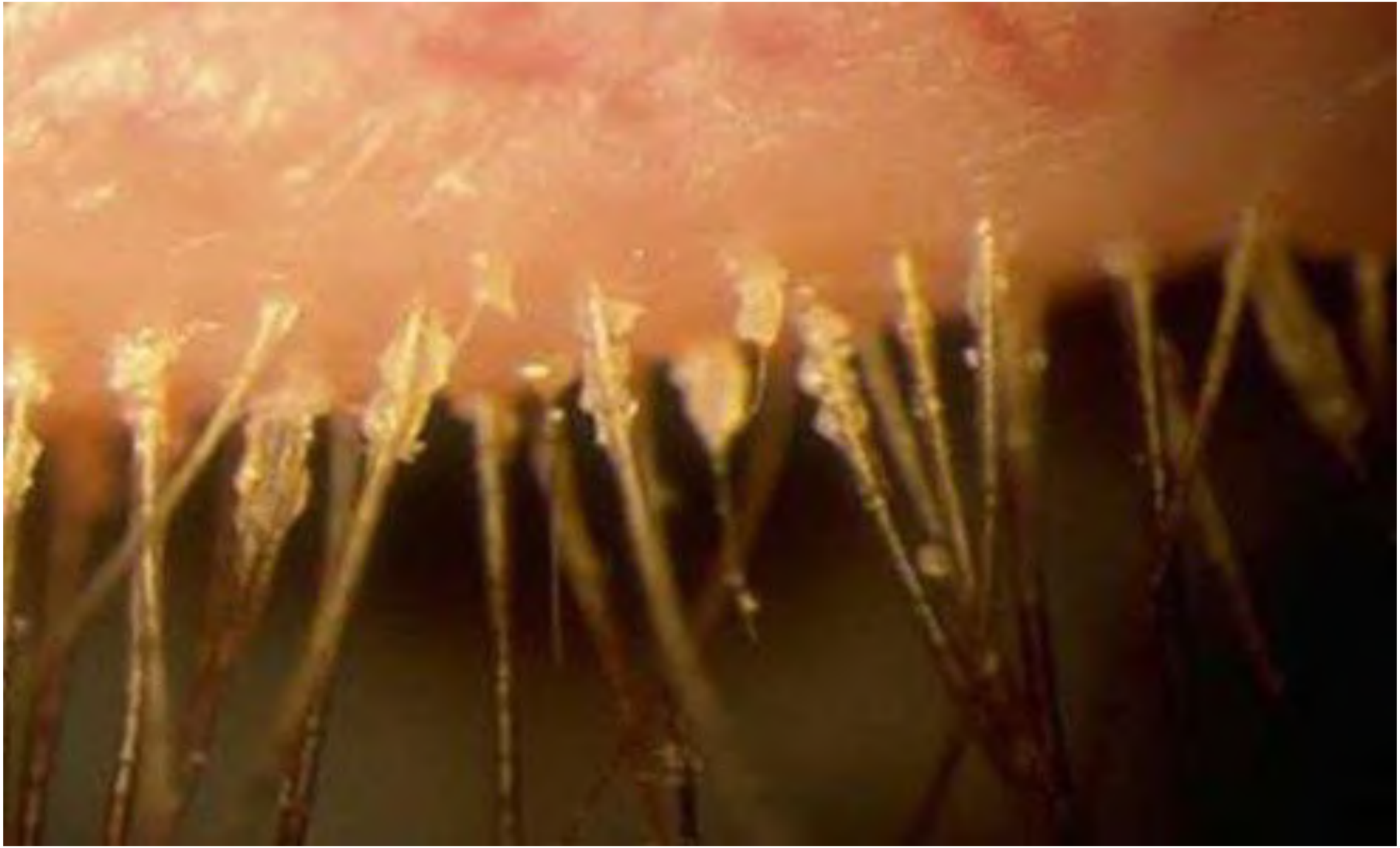
- Mechanical defense The lid, via the tarsal plate and muscles, forms a
- shield and mechanical barrier from the external world. This serves to
- protect the globe from injury and/ or excess light. This is an example of
- “native immunity” by acting as a barrier.
- Optical; Keeps the tear layer smooth via blinking. This maintains the
- optical clarity by resurfacing tears, which is important for visual acuity and
- contrast sensitivity.
- Assist immune system Via blinking, the lid removes pathogens in the tears
- It also replenishes immunological substances which inhibit bacterial
- growth, such as Lysozyme, lactoferrin, beta lysine
- Immunoglobulins (IgG, E, M)

- Facilitate corneal metabolism
- With blinking, oxygen
- is distributed and waste products (carbon dioxide and lactic acid) are removed.
- Reduce visual stimuli
- Decreased light with closure

- Blepharitis can be divided into anterior and posterior according to anatomic location, although there is considerable overlap and both are often present.
- Anterior blepharitis affects the eyelid skin, base of the eyelashes, and the eyelash follicles and includes the traditional classifications of staphylococcal and seborrheic blepharitis.
- Posterior blepharitis affects the meibomian glands and gland orifices and has a range of potential etiologies, the primary cause being meibomian gland dysfunction MGD


Anterior Margin
Posterior Margin





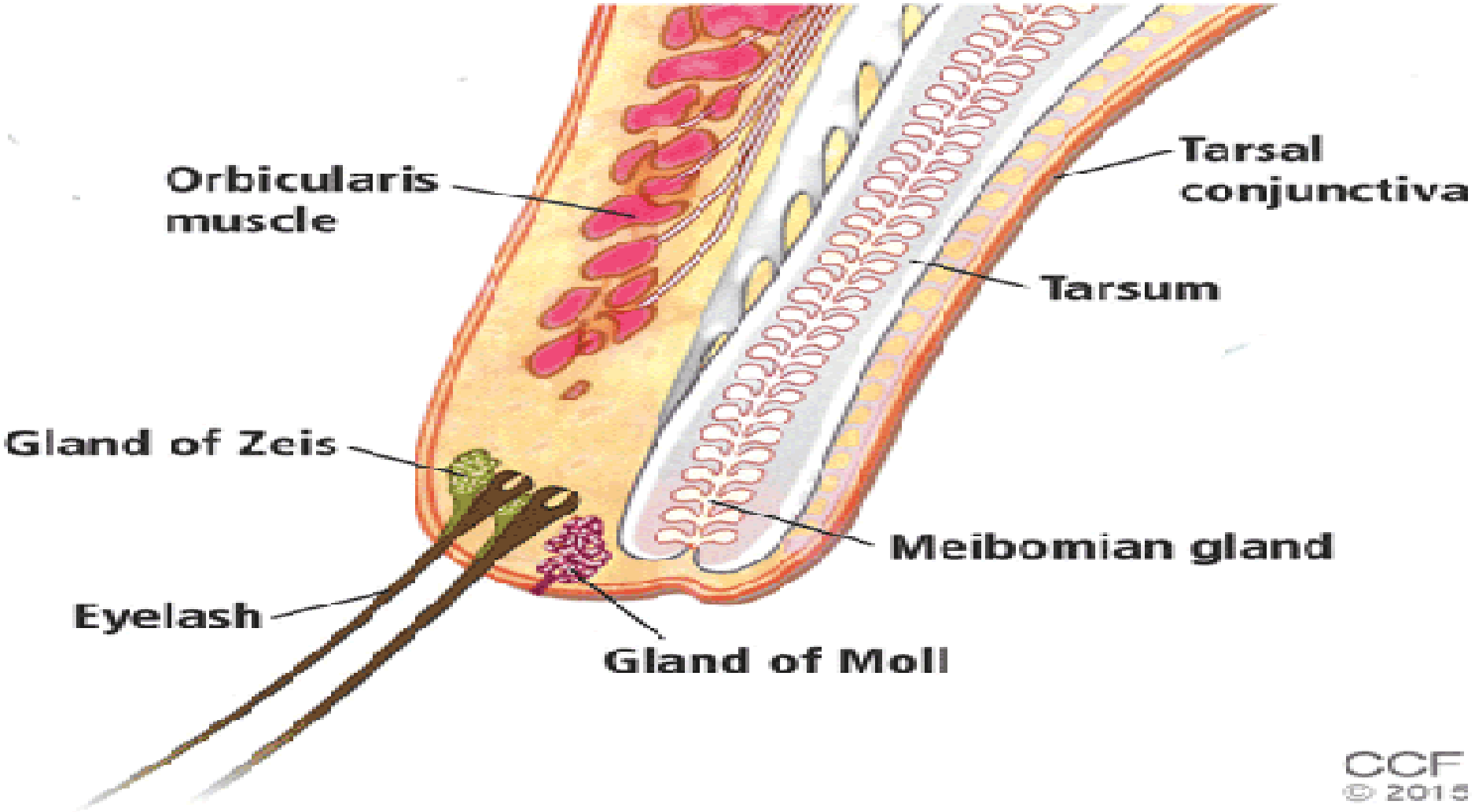
Types

- **Anterior;** it involves the area surrounding the basis of eye lashes.
- May be
- A) Infective/ulcerstive
- B) Non-infective/seborrhic blepharitis

- **Posterior**/Meibomianitis

Anterior ulcerative blepharitis

- It is the blepharitis in which there is acute & chronic infection and inflammation of lash follicle associated with gland of Zeiss and Moll
- Etiology bacterial infection mainly
- Staphylococcus aureus, staphylococcus epidermidis
- **Clinical features;**
- History of chronic irritation, redness, itching, lacrimation, glueing of lashes and photophobia & chalazion formation
- It is usually bilateral

- 
Orbicularis muscle
Tarsal conjunctiva
Tarsum
Gland of Zeis
Meibomian gland
Eyelash
Gland of Moll
CCF
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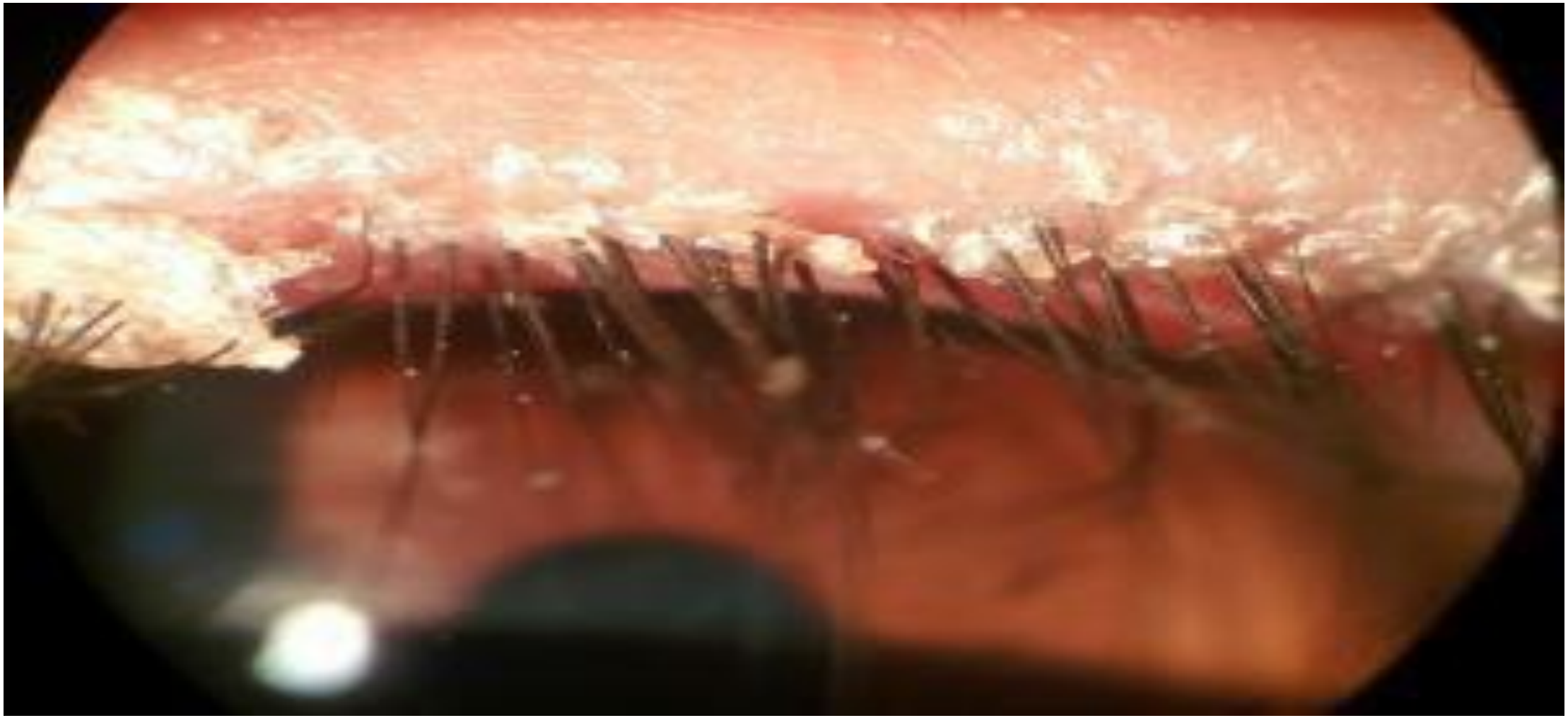
Contd

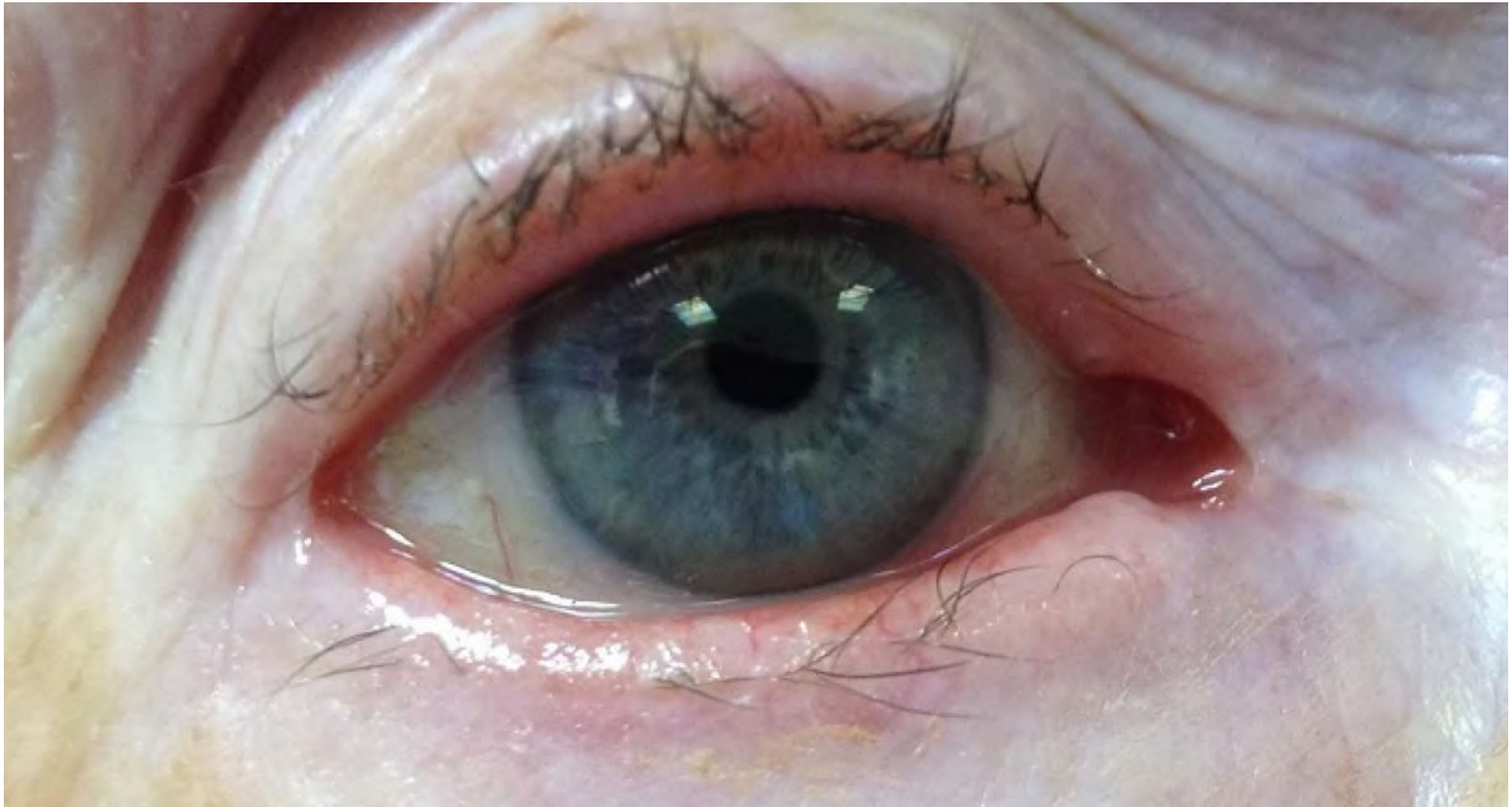
- Signs;
- Lid margins are swollen, red & inflamed.
- Yellow pus secretion of suppurative lesion which when dries up leads crusts formation
- Crusts tend to be centred around the bases of lashes. When you remove it there will be tiny ulcer behind.
- Eye lashes involvement is more in infective than in seborrheic type.
- Eyelashes may be matted together



Complications

- Lids; it can lead to
- Misdirected eyelashes Trichiasis,
- Loss of lashes Madarosis,
- Whitening of lashes poliosis,
- Recurrent stye & chalazion and ptosis due to edema
- Conjunctiva Recurrent attacks of mild bacterial conjunctivitis and papillary conjunctivitis
- Cornea Marginal Keratitis and toxic punctate keratitis
- Due to toxin released by the staphylococcus bacteria

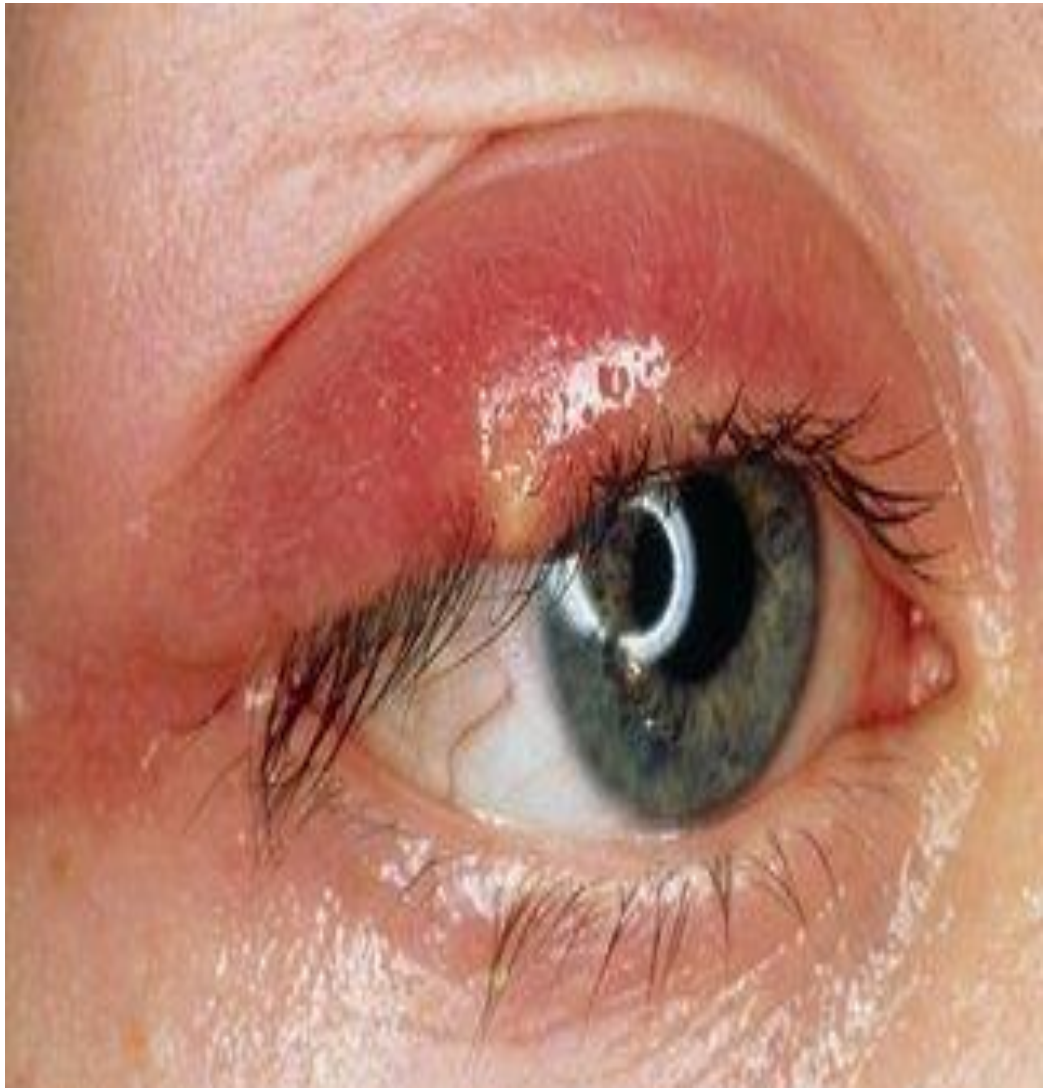
Triachiasis



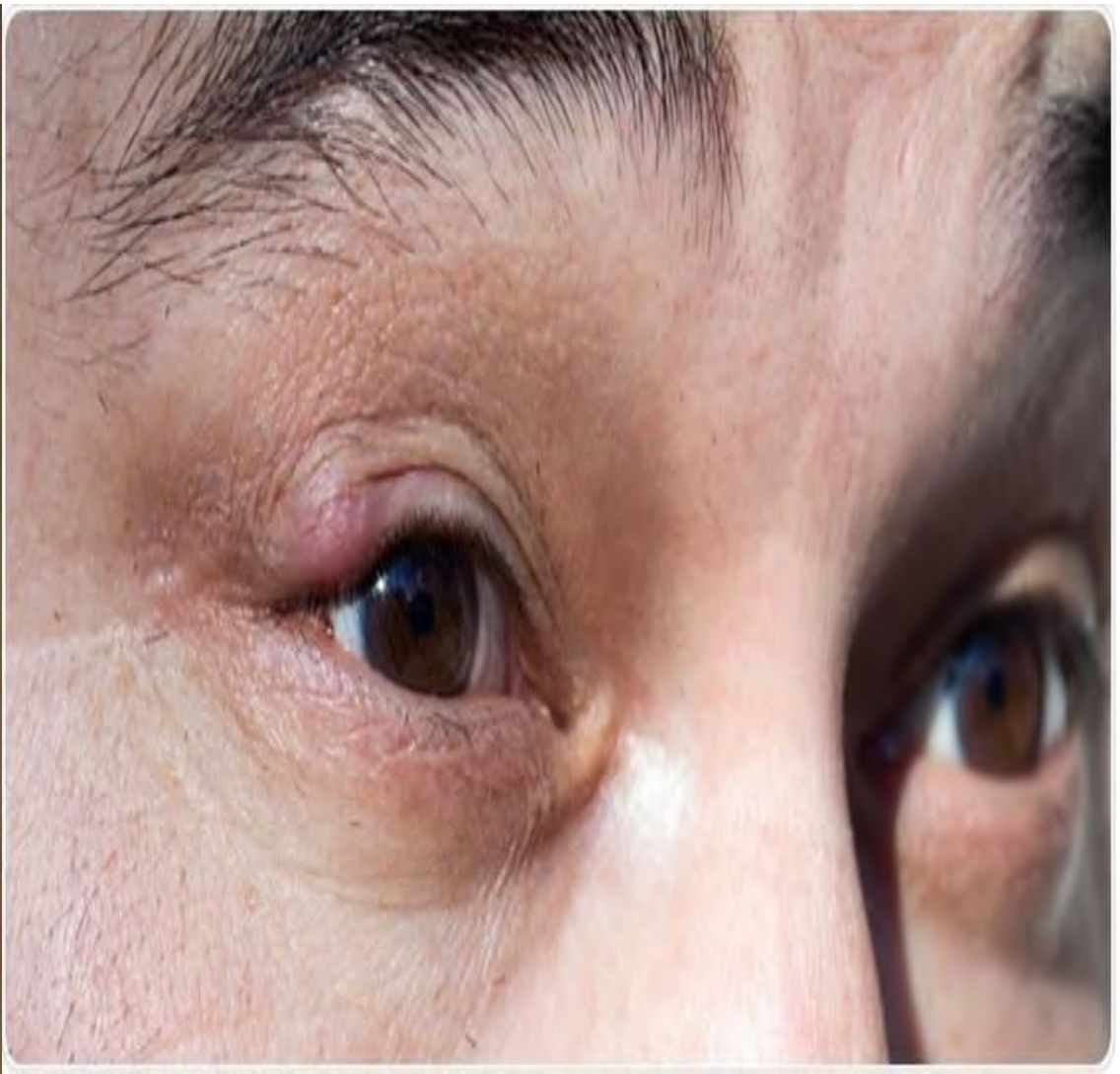
Poliosis

Medarosis

Stye & Chalazion



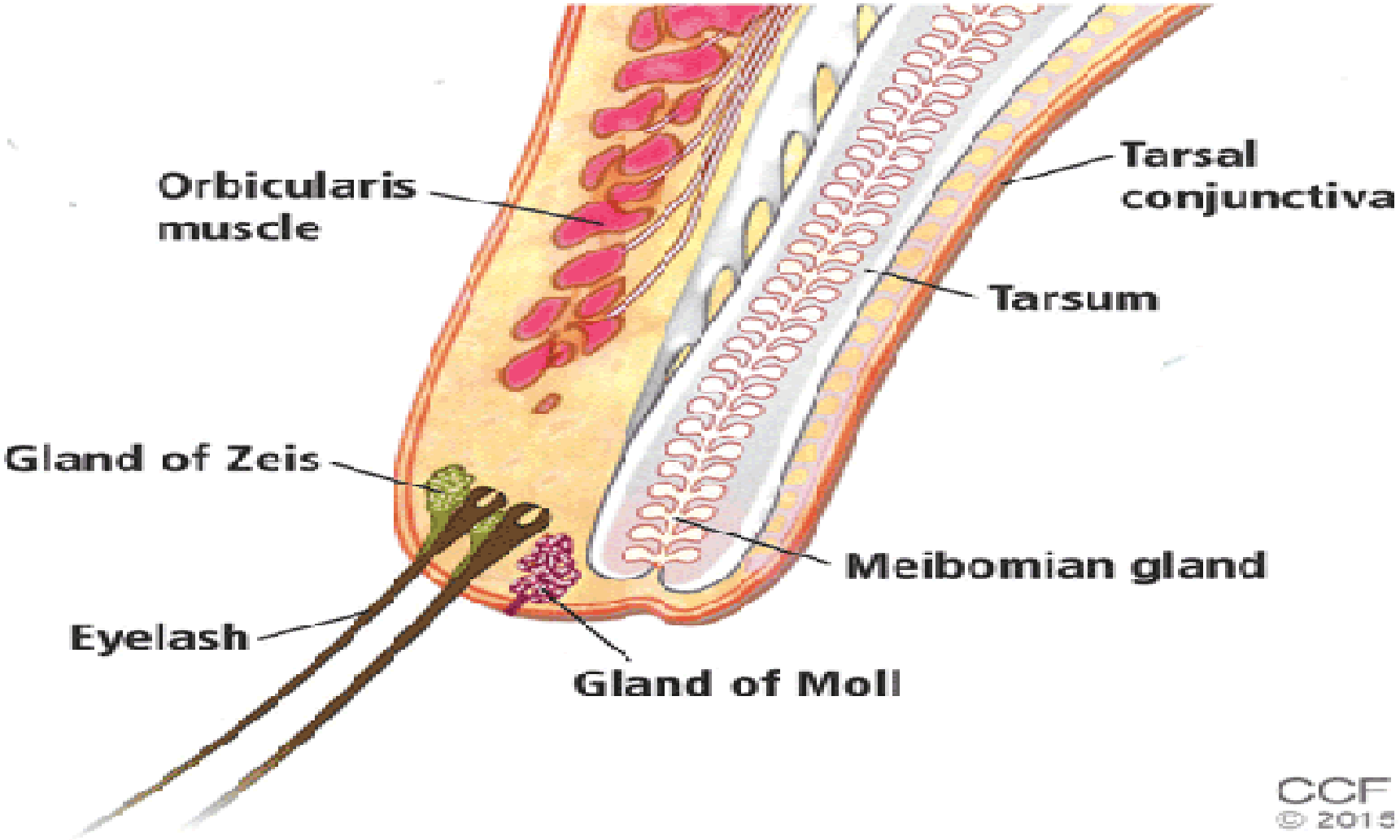
Chalazion & Ptosis




A
B
C
D
E
F

Ant Seborrheic /Squamous blepharitis

- It is the type of blepharitis In which there is problem in the glands of Zeis & Moll.
- Aetiology is not exactly known
- Some metabolic disorder,?
- Associated with seborrheic dermatitis, Rosacea and Atopic dermatitis
- The gland of Zeis secretes abnormal excessive neutral lipids which are then split into irritating free fatty acid by the Corynebacterium acne.

- 
Orbicularis muscle
Tarsal conjunctiva
Tarsum
Gland of Zeis
Meibomian gland
Eyelash
Gland of Moll
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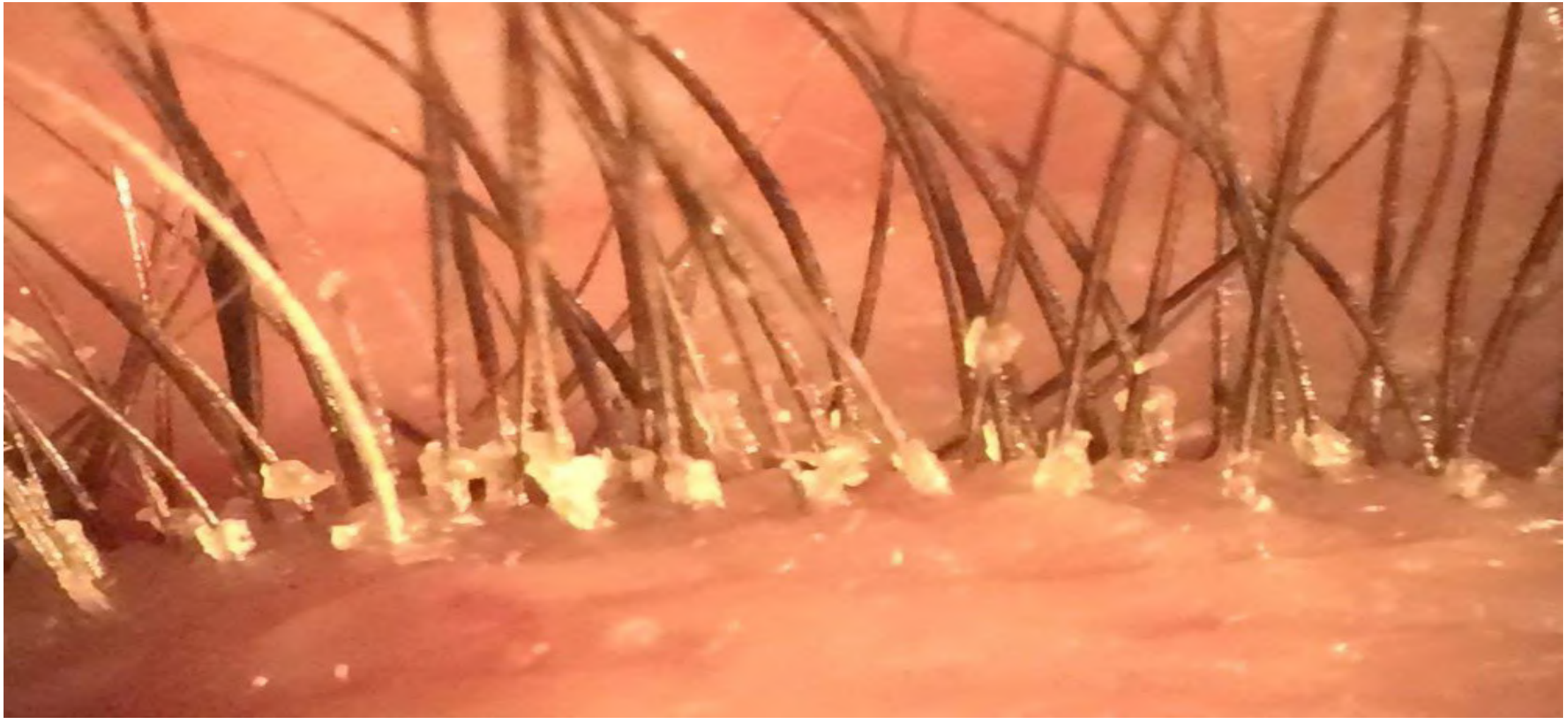
Clinical features

- Symptoms,
- Discomfort, irritation, redness,
- Watering and
- Falling of eyelashes

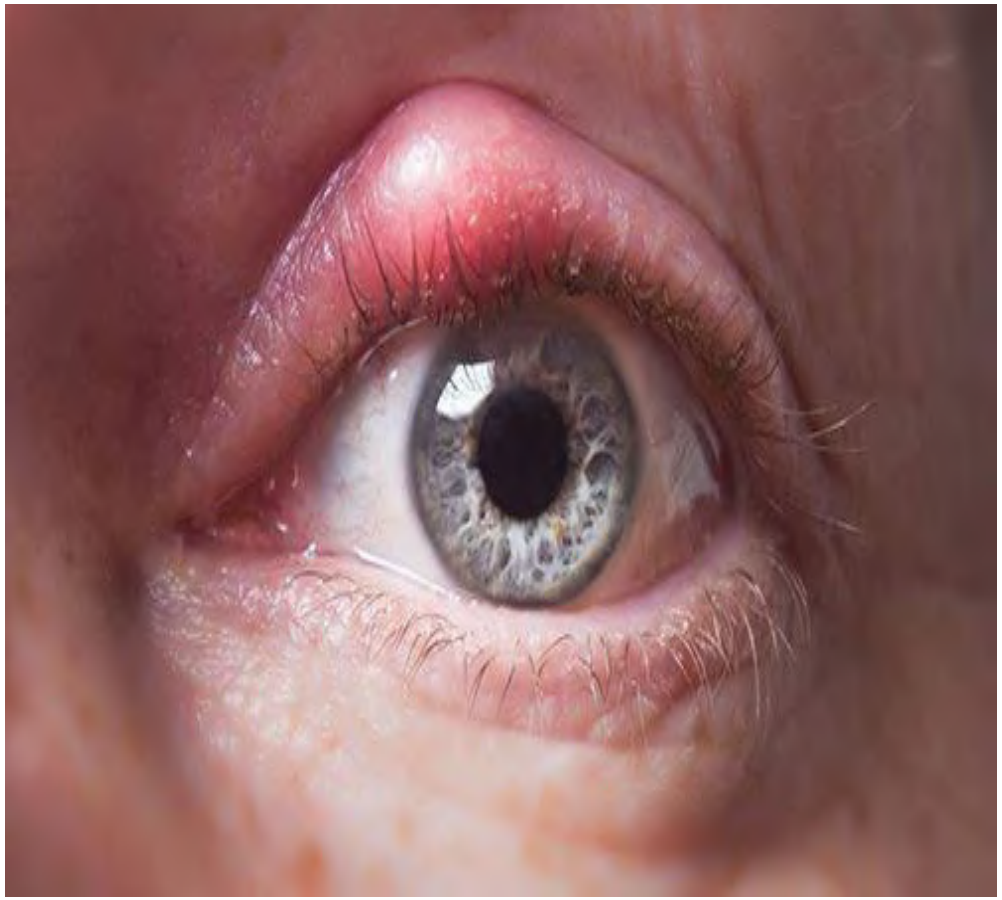
- Signs
- Lid margins have a shiny and waxy appearance
- Erythema and telangiectasia
- White yellow dandruff like scaly material which is the desquamation of the epidermis, located at the eyelashes and lid-margins
- Scales are soft easy to remove with no ulcer
- Lashes are greasy
- In sever cases Lid margins are swollen



Complications

- Trichiasis
- Loss of lashes (madarosis)
- Whitening of lashes (poliosis)
- Conjunctivitis
- Tear film instability in 25-30%
- Chalazion formation

Blepharitis chalazian

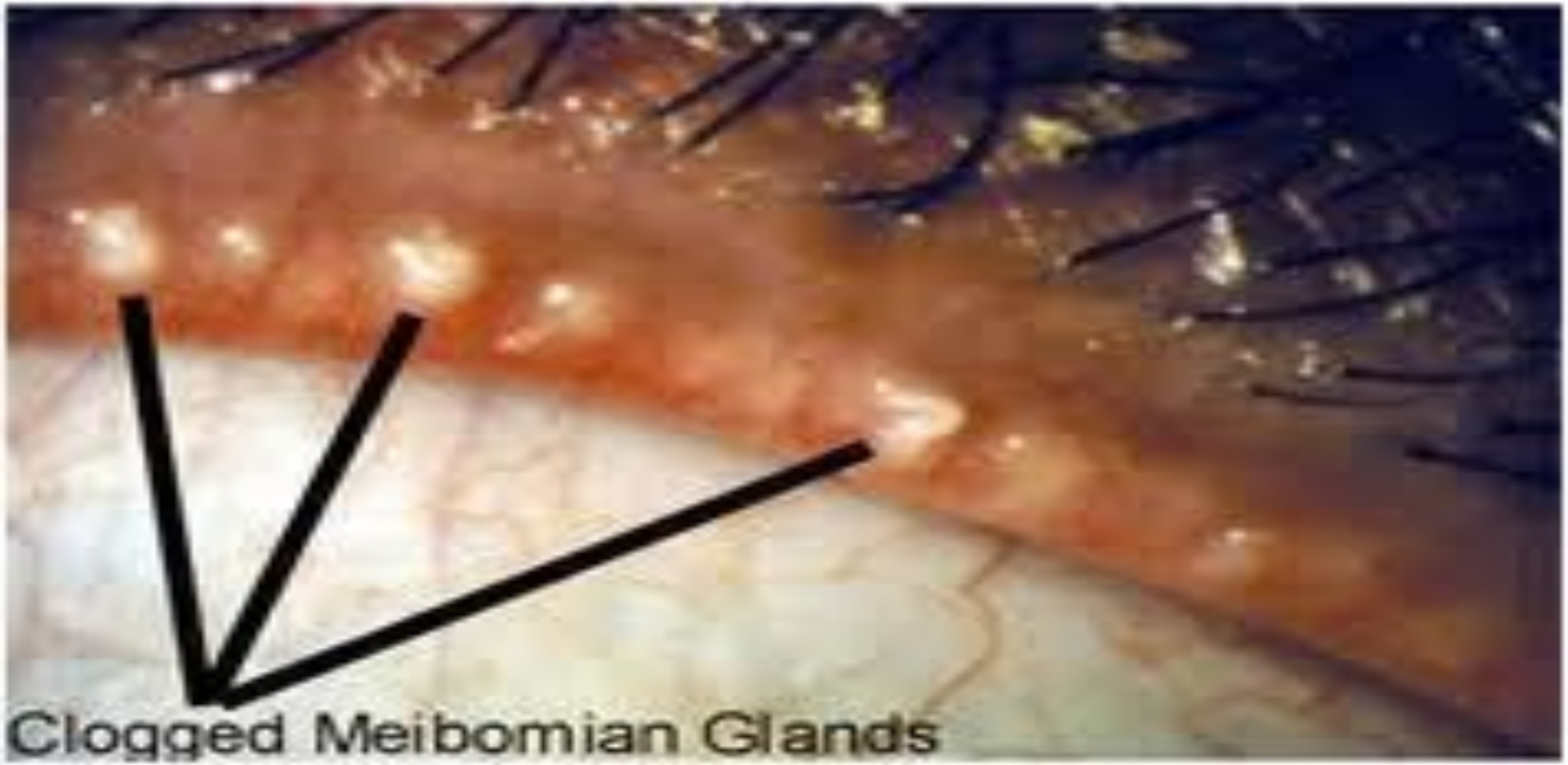


Post blepharitis/ Meibomitis

- It is the type of blepharitis in which mainly the post margin of the lid margin is involved leading to diffuse inflammation of meibomian glands and hypersecretion of gland

Clinical features

- Burning sensation, irritation redness due to excessive secretion of the fatty acid
- White frothy foam like secretion at lid margins and canthi
- The orifices of the gland are pouting and are capped with small oil globule
- On lid eversion the M-glands ducts are yellow white lines through the conjunctiva
- On massage, oily secretion come out
- Duct obstruction lead to chalazion formation


Clogged Meibomian Glands

Complications

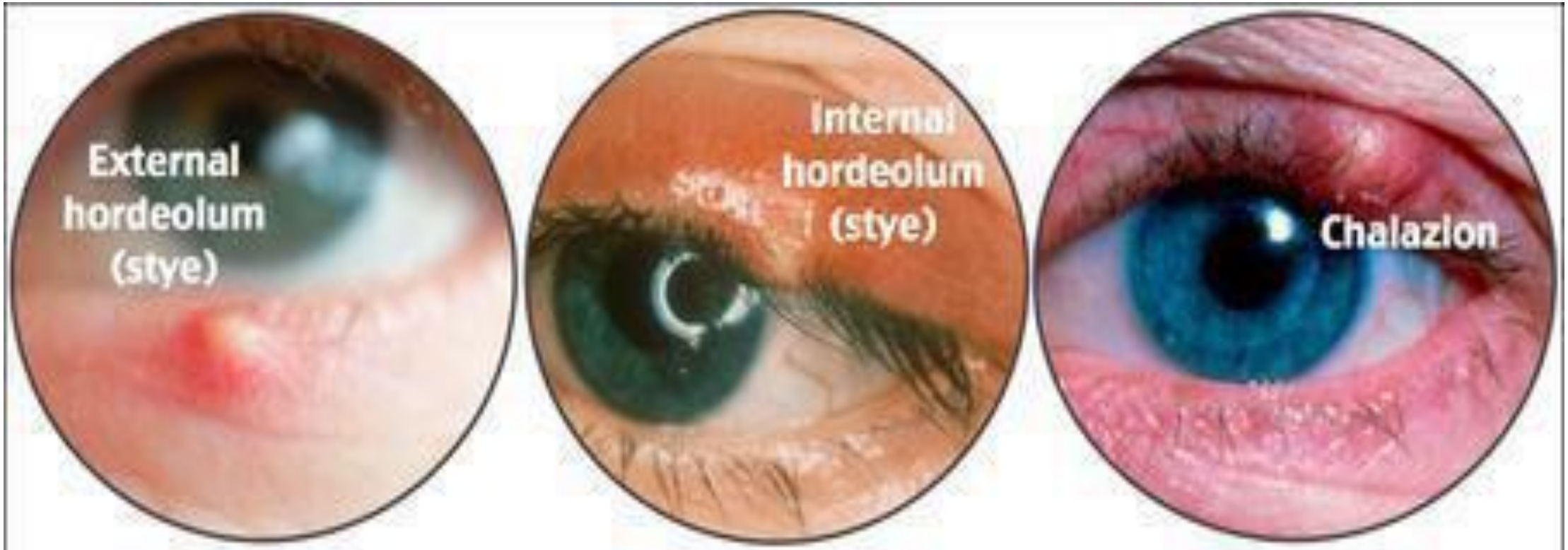
- Chalazion formation
- Papillary conjunctivitis
- Tear film instability 25-30 %
- Cornea Punctate epithelia erosions

Treatment

- Local Lid hygiene
- Topical , Antibiotics & steroid drops
- Artificial tears
- Systemic antibiotic
- Tetracycline
- Doxycycline
- Erythromycine

Categorization of blepharitis

	Infectious	Seborrheic	Meibomian gland dysfunction
Location	Anterior eyelid	Anterior eyelid	Posterior eyelid
Loss of lashes	Frequent	Rare	None
Lid margin	Hard, fibrinous scales with matted crusts	Oily or greasy	Unusual
Lid ulceration	Occasionally	None	None
Conjunctivitis	Papillary with occasional purulent discharge	Follicular or papillary tarsal reaction with mild hyperemia	Papillary tarsal reaction with mild-to-moderate hyperemia
Keratitis	Inferior punctate erosions, marginal infiltrates, vascularization, phlyctenules	Inferior punctate erosions	Inferior punctate erosions, marginal infiltrates, vascular pannus
Tear film disruption	Occasional	Occasional	Occasional
Rosacea	Negative	15%–25%	40%–50%



	Hordeolum (Stye)	Chalazion
Location	Most commonly found at or near an eyelash follicle	Most commonly found above the eyelashes on the upper lid
Cause	Bacterial infection either at the root of the eyelash follicle or in the oil gland of the lids	A blocked oil gland (Meibomian or Zeiss)
Symptoms	Tenderness, swelling	Firm, painless lump
Treatment	Spontaneous drainage, warm compresses	Warm compresses, antibiotic eyedrops, surgery

Phtheriasis Palpebrarum

- Eye lashes infestation with phthirus pubis (Crab lice)

- Transmitted through
- Sexual activity
- Closed contact with infected person
- Use of contaminated clothes towels & beddings

Clinical features

- Symptoms
- Blepharitis like features redness foreign body sensation
- Severe itching and irritation are the main symptoms

- Signs on slit lamp examination with red lid margins
- Numerous nits & eggs are adherent to the lashes root
- Adult lice are stick to eyelashes
- Blood stained debris may be present

- Diagnosis is made by slit lamp examination

Figure. The adult louse, along with the nits, is attached to the eyelash.

Treatment

- Lid hygiene improving
- Mechanical removal of the lice & nit with forceps
- Eyelashes shaving and removal
- Petroleum jelly twice daily for 14 days
- Yellow mercuric oxide 1% oint 4 times 14 days
- Laser therapy
- Cryotherapy
- Permithrin/ Malathion anti lice shampoo 1% twice

- Thanks


بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

Scenario

- A pt age 50 yrs comes to eye with redness, foreign body sensation in Rt eye for the last so many times. On examination there is a red lesion near his medial canthus Rt eye. His vision is 6/12 & 6/6 in Rt & Lt eye respectively.
- What is the most probable cause.?
- A Allergic Conjunctivitis
- B Marginal keratitis
- C Pterygium
- D Phlyctenule

Pterygium

Dr Nazullah

Associate professor

Ophthalmology KGMC/HMC

Pterygium

- It can be defined as triangular conjunctival fibrovascular tissue over riding/overgrows the cornea
- It is a degenerative condition of the conjunctiva
- Usually occurs in the interpalpebral fissure
- Nasal is the common site

Aetiology

- Exactly is not known
- Probable Can be
- Drying of this area is the primary theory
- Ultraviolet radiation and damage of the corneal epithelium, bowman membrane and the underlying stroma
- The following conditions can precipitate
- Chronic conjunctivitis
- Dusty environment

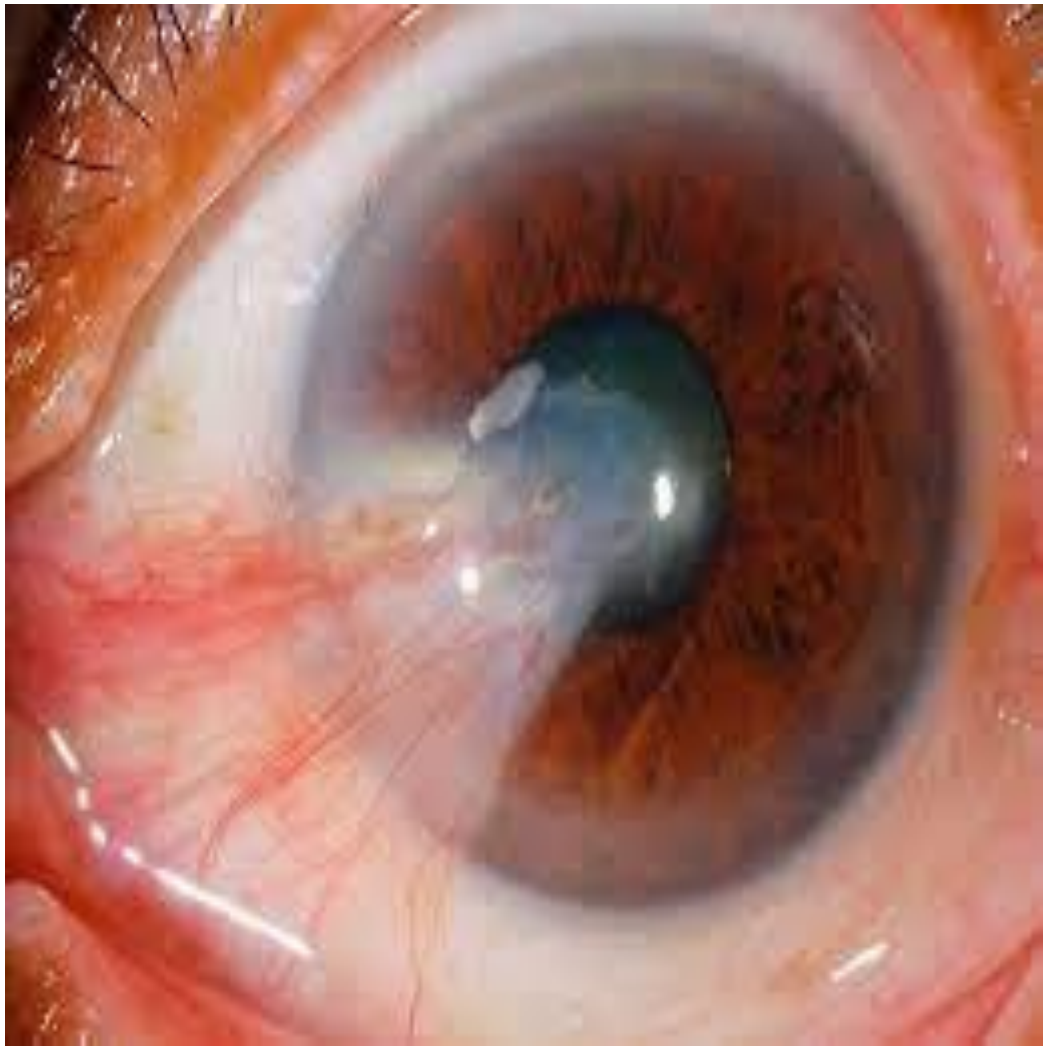
Mechanism

- It’s a degenerative , inflammatory & hyperplastic condition
- Sub-conjunctival tissue undergoes elastotic degeneration & proliferate as vascularized granulation tissue , under the epithelium, which ultimately encroaches the cornea
- Corneal epithelium, bowman membrane and underlying stroma are damaged.
- So simultaneous changes occur in conjunctiva & cornea

Types of pterygium

- Progressive type; It is a thick vascularized fleshy tissue with infilterate at the anterior end called the Cap. It progresses with time.
- Stationary type; its not progresses with time & remains in the same stage
- Regressive type; in this condition the tissue is very thin , atrophic & with very little vascularity almost avascular
-

Progressive



Regressive

Stages of pterygium

Stage 0: This stage is when the lesion is posterior to the limbus (border of the cornea that is contact with the white of the eye (sclera)), specifically called pinguecula.
A pinguecula is a yellowish patch or bump on the conjunctiva that occurs due to a deposition of protein, fat or calcium on the tissue.
In this case there is no vascularity and conjunctival and corneal ectasia are seen.

- **Stage 1:** In this the lesion involves limbus. Minimal papillary response is seen and conjunctival and corneal tissues are flat.
- Extends upto 1mm
- **Stage 2:** In this stage the lesion appears just on the limbus. The vascularity is normal but a minimal elevation is observed on conjunctival and corneal tissues. 1-2mm

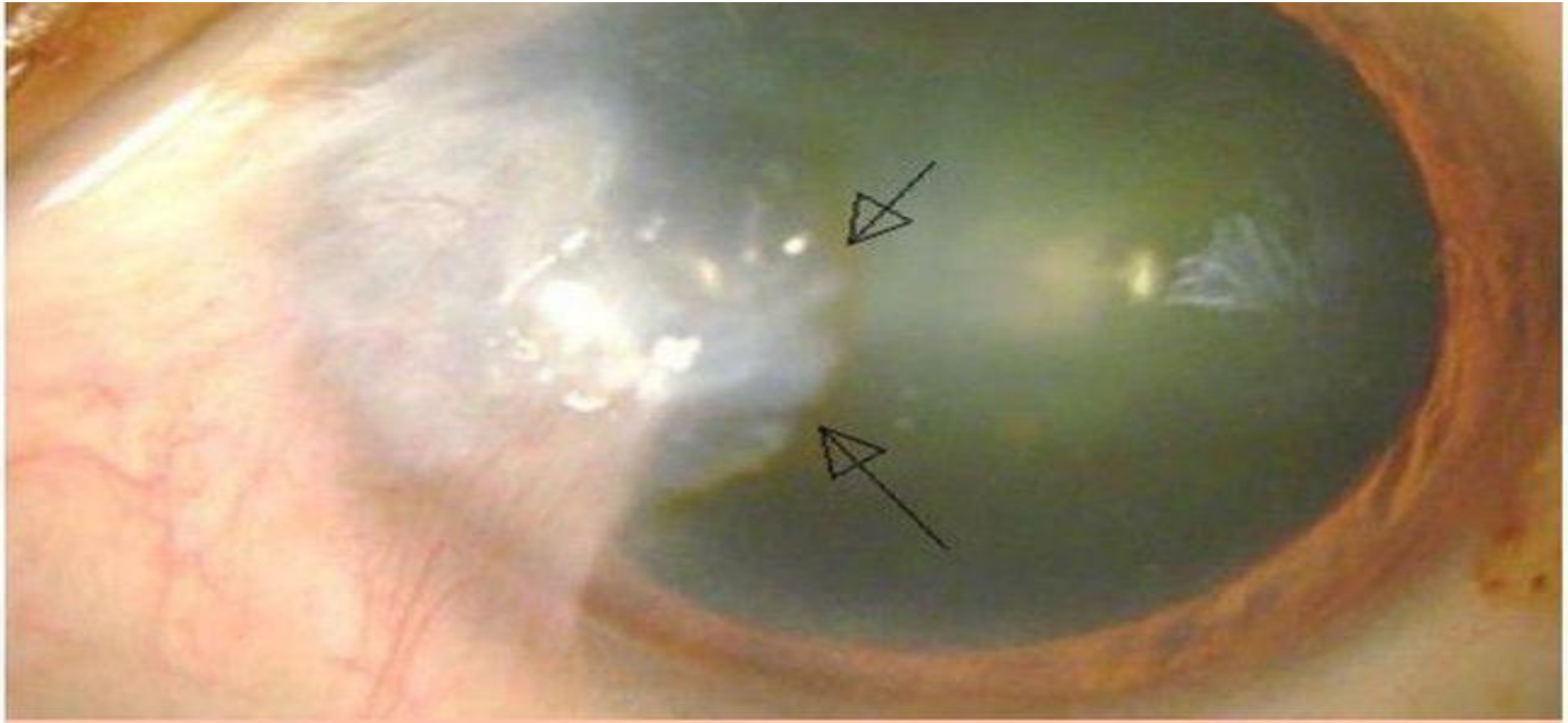
- **Stage 3:** The pterygium covers the area between the limbus and pupillary margin. Moderate vascularity with vessel congestion is seen and the lesion Extends upto 4mm.
- May cause astigmatism
- **Stage 4:** In this case the lesion is central to the pupillary margin. It extends to more than 4 mm. This is a severe form of pterygium with vessel congestion and dilation. It is of dense and deep color and may involve areas of vision (visual axis).
- This is associated with increase in astigmatism and can even lead to vision problem & eye movement.

Difference between

	Progessive pterygium	Atrophic pterygium
Appearance	Thick and fleshy	Thin and membranous
Blood vessels	Very prominent	Very few blood vesssels giving a pale appearance
Cap in front of the head	Present	Absent
Progression	Continues to advance further into the cornea	Static after an initial period of growth

Parts of the pterygium

- A pterygium appears as a wing shaped growth on the visible part of the sclera in the horizontal meridian, which is seen to be invading into the adjacent cornea. The color depends on the degree of prominence of blood vessels - it may be red, thick and fleshy in a progressive pterygium, and pale, thin and membranous in an atrophic pterygium.
- A line of iron deposition known as Stocker’s line is often seen in front of the head of long standing pterygia.
-

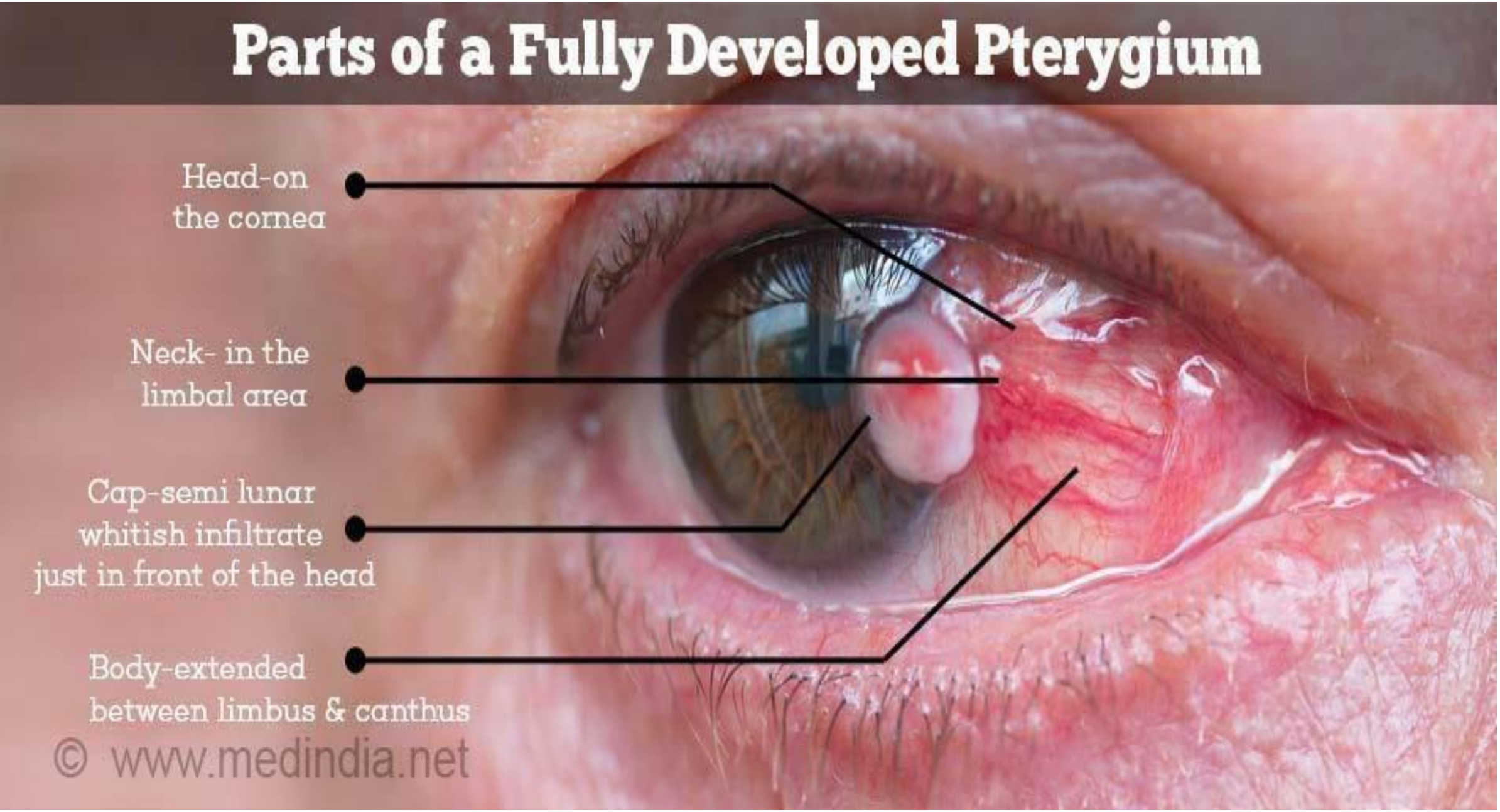


Stocker's Line. Iron deposition line in the corneal epithelium, located at the corneal leading edge of a pterygium.

Parts contd

- Head: Part of the pterygium in the cornea
- Cap: opaque infiltration seen in front of the head of progressive pterygia
- Stocker’s line - A line of iron deposition adjacent to the head of the pterygium. It appears in case of chronic pterygium.
- Neck: the part that overlies the junction between the cornea and sclera
- Body: the part that lies in the conjunctiva overlying the sclera

Parts of a Fully Developed Pterygium



Clinical features

- With Irritation, redness, foreign body sensation
- Typically develops in hot, windy & dry climates
- Asymptomatic In early stages
- More common nasal side than on temporal side
- Appears as fibrovascular wing shape lesion grows in the limbus with apex towards the cornea
- Diminished vision - Either due to the growth altering the shape of the cornea producing astigmatism, or due to obstruction of the visual axis.
- Diplopia may be due to limitation of movement

- Usually pterygium is asymptomatic apart from its appearance.
- Sometimes a pterygium can give rise to the following symptoms -
- **Dryness, grittiness or foreign body sensation -** Due to rapid tear evaporation because of an uneven ocular surface.
- **Redness and pain -** If the pterygium is inflamed.
- **Diplopia -** This is very rare, and occurs in very large recurrent ptergia due to restriction of ocular movments.
-

Treatment

- Non surgical

- Surgical

Non surgical

- Tear substitute to control the dryness
- Topical steroid for inflammation
- Sunglasses to avoid sun & ultraviolet exposure

Surgical

- Indication are
- Optical for visual restoration, due to Astigmatism and or mechanical obstruction of visual axis
- Cosmetics somebody may feel cosmetic problem
- Irritation & redness

Different procedures

- Bare sclera; with simple excision with 80% recurrence rate
- To prevent recurrence
- Simple excision with mitomycin-C
- Excision with conjunctival auto-graft
- Excision with amniotic membrane graft
- Laser with Argon laser
- Radiation with Beta rays

Surgical Complication

- Recurrence is the common problem
- So to prevent it

Conjunctival autograft

- This method might not be necessary in every case, but it's the least likely to lead to recurrence. It's also the technique I perform most often.
- To perform a conjunctival autograft, first measure the conjunctival epithelial defect and how much bare sclera you need to cover. Then, harvest the conjunctiva approximately 90 degrees or 3 to 4 clock hours away from the resected site, usually in the superior globe, with Wescott scissors. Dissect the conjunctiva free from the underlying Tenon's capsule to an extent that matches the surface area of the pterygium.

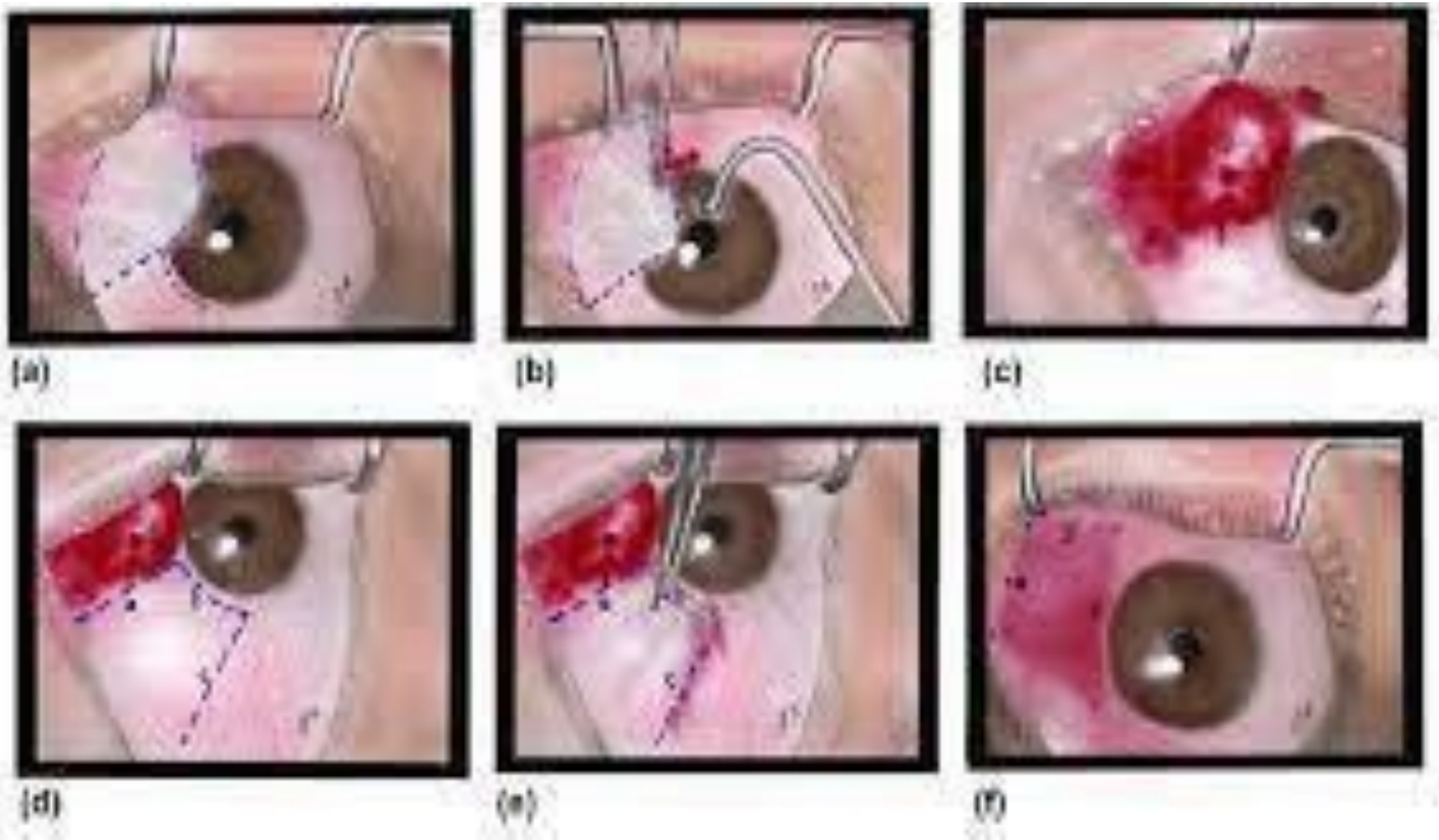
- Glue or suture the flap to the bed with 8-0 vicryl. If using glue, aim for as little glue as possible. Postoperatively, prescribe topical antibiotic drops such as fluoroquinolone q.i.d. for a week, and a steroid drop such as prednisolone acetate q.i.d., tapered over one to three months.

- Recurrence the least one

Amniotic membrane transplant

Amniotic membrane, which can be placed and glued or sutured over the area of the defect, is a very effective method. We prefer to use glue, since it’s fast and simple. Amniotic membrane makes patients more comfortable and contributes to the healing of the tissue. However, it’s not quite as effective in discouraging recurrence as the third option.

Recurrence is low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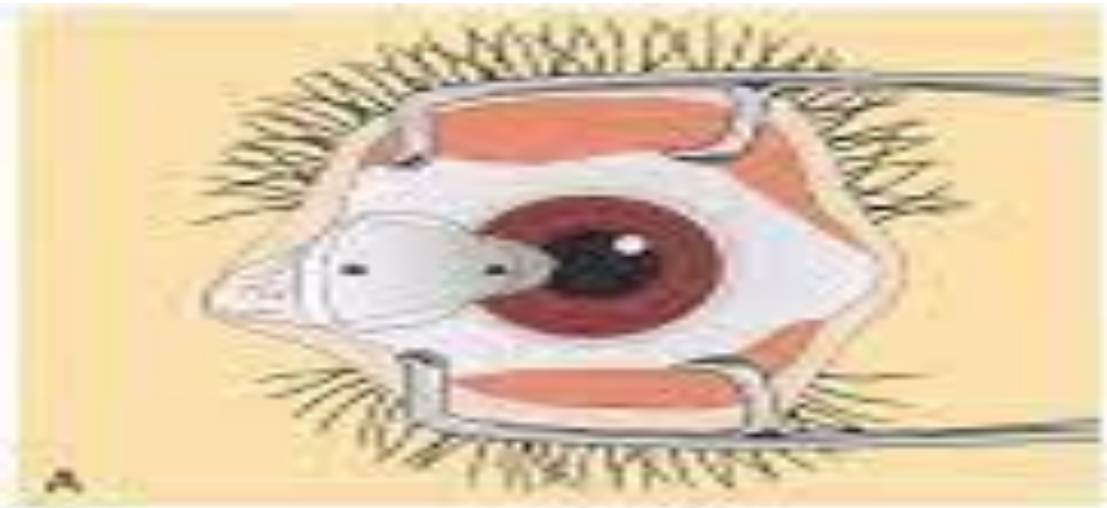
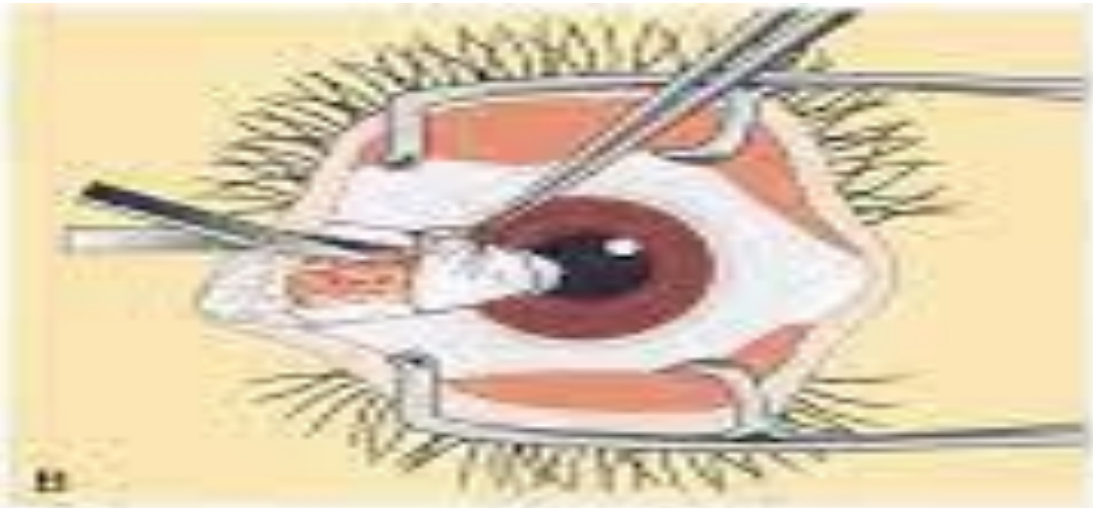

A


B


C

Pseudo pterygium

- An abnormal fold of conjunctiva attached to cornea at an abnormal location . Other than the nasal area
- Causes
- Corneal injuries & burns
- Peripheral corneal ulcers

Clinical features

- A conjunctival adhesion to the corneal periphery
- In any location/quadrant
- Its neck is free and a probe can be passed behind it
- Can cause diplopia

Pterygium vs pseudopterygium

	Pterygium	*Pseudopterygium*
1. Etiology	Degenerative process	Inflammatory process
2. Age	Usually occurs in elderly persons	Can occur at any age
3. Site	Always situated in the palpebral aperture	Can occur at any site
4. Stages	Either progressive, ssive, regressive or stationary	Always stationary
5. Probe test	Probe cannot be passed undemeath	A probe can be passed under the neck

Treatment

- Surgical excision

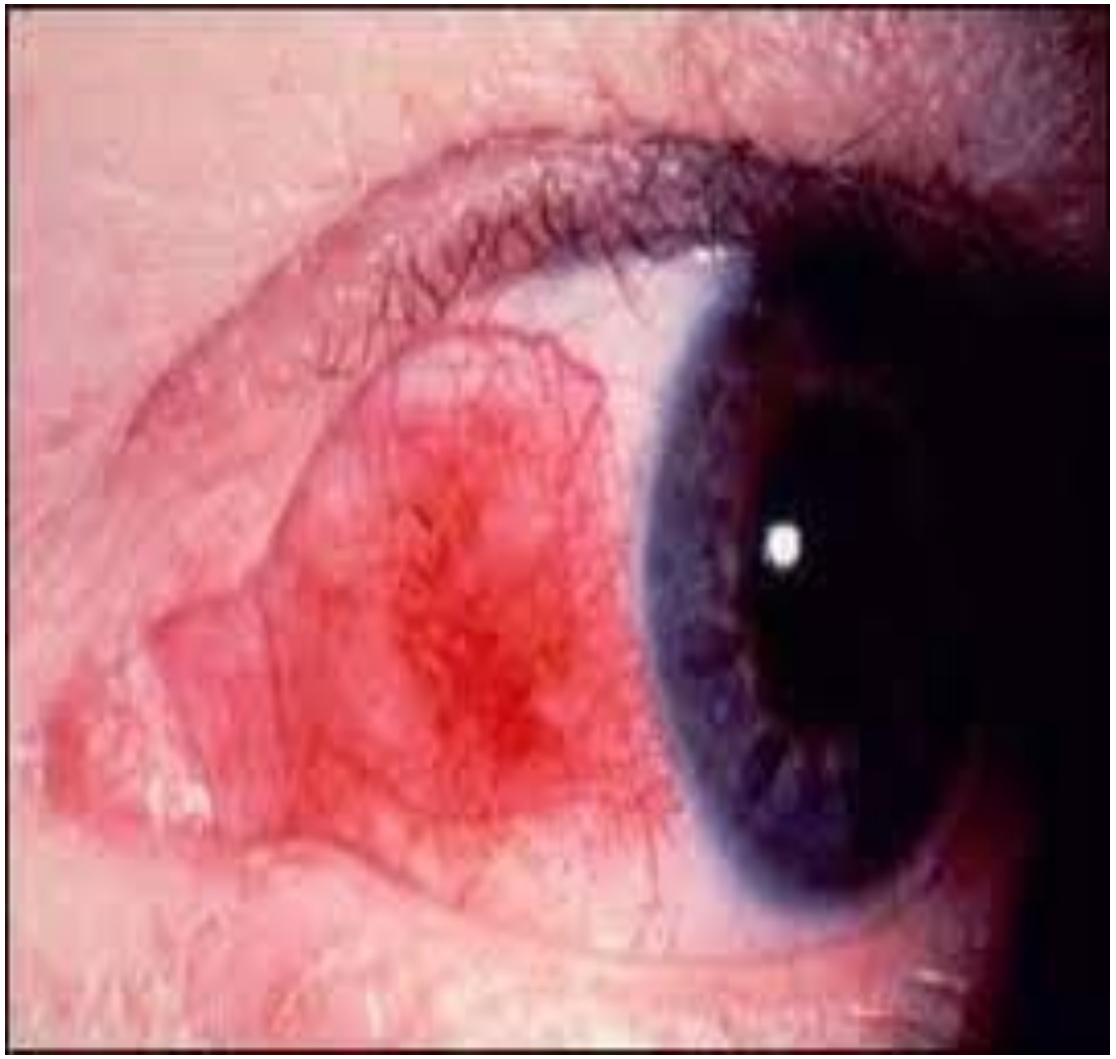
Episcleritis

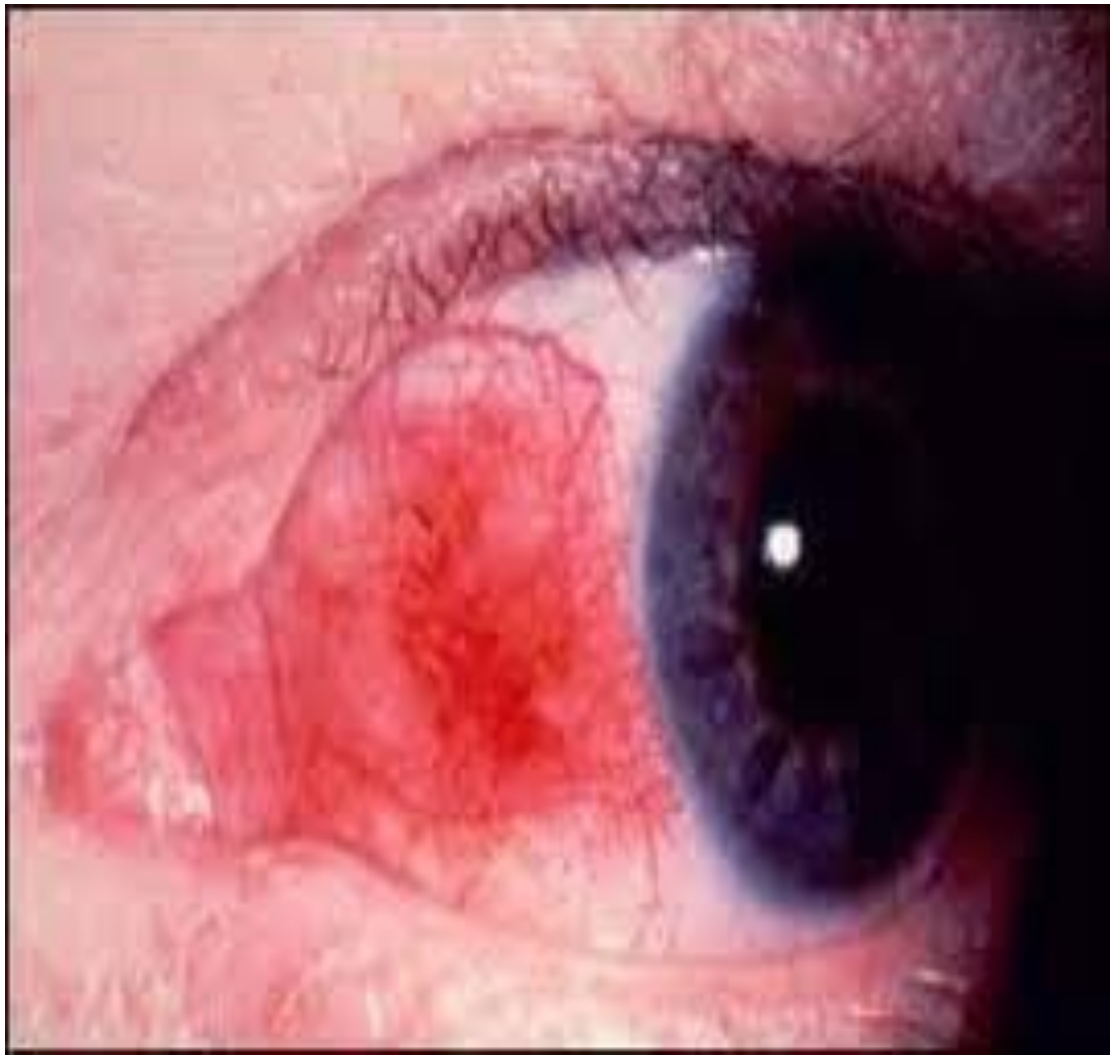
- Episcleritis is an inflammatory condition affecting the episcleral tissue between the conjunctiva (the clear mucous membrane lining the inner eyelids and sclera) and the sclera (the white part of the eye) that occurs in the absence of an infection.
- It is a benign self limiting condition

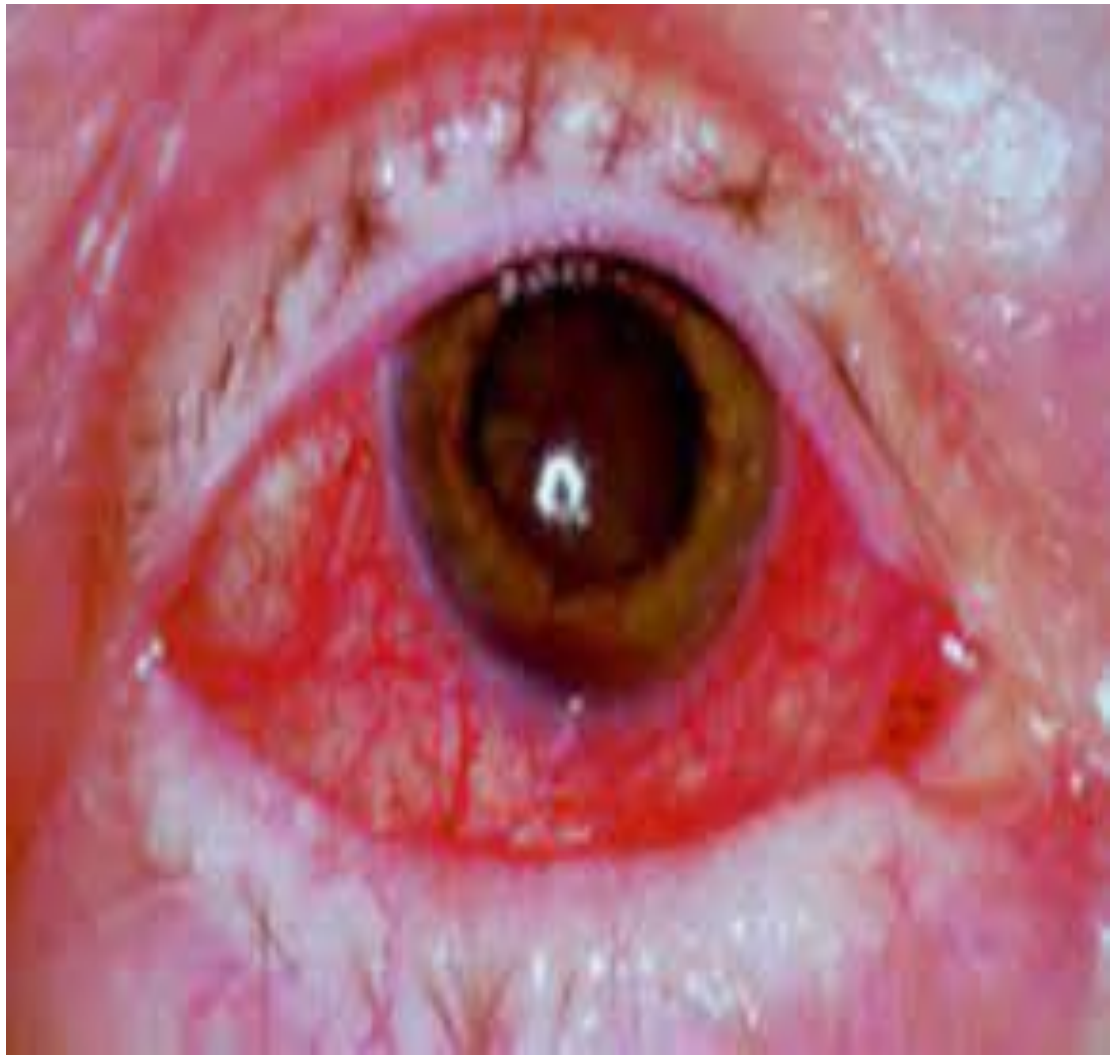
Scenario

- A young pt age 25-30 yrs come to eye opd with redness foreign body sensation. On examination there is redness in the lateral side of the globe . His vision is 6/6 Bes. No pain no discharge. He is also having arthritis.
- What is the most probable cause.?
- A Conjunctivitis
- B Episcleritis
- C Keratitis
- D Uveitis

- Ans B
-







Aetiology

- Idiopathic in many conditions
- May be associated with
- Rheumatoid Arthritis
- Inflammatory bowl disease
- Atopy , Gout . Acne Rosacea
- TB, Syphilis
- May be infective associated with Herpes zoster ,TB ,Syphilis

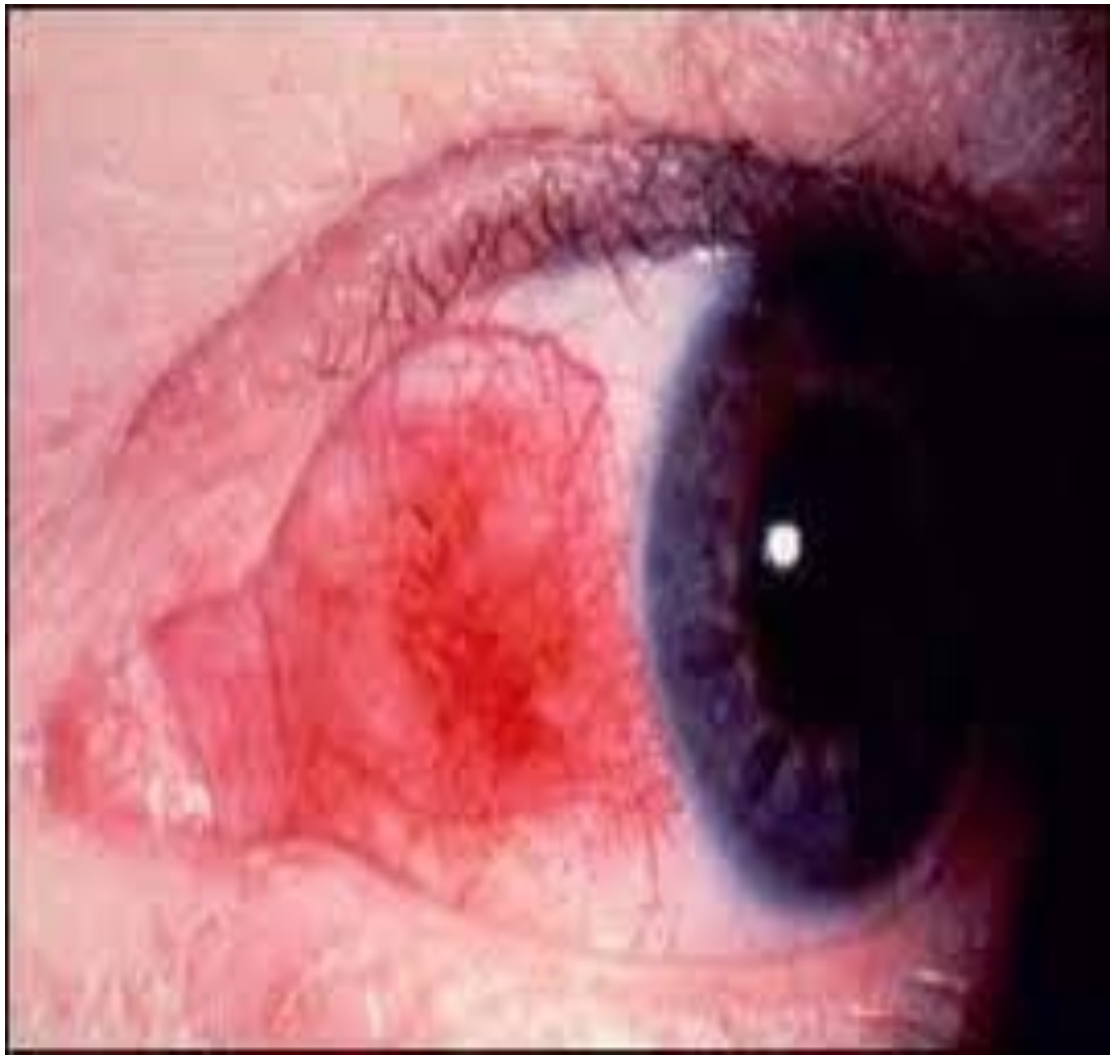
Types

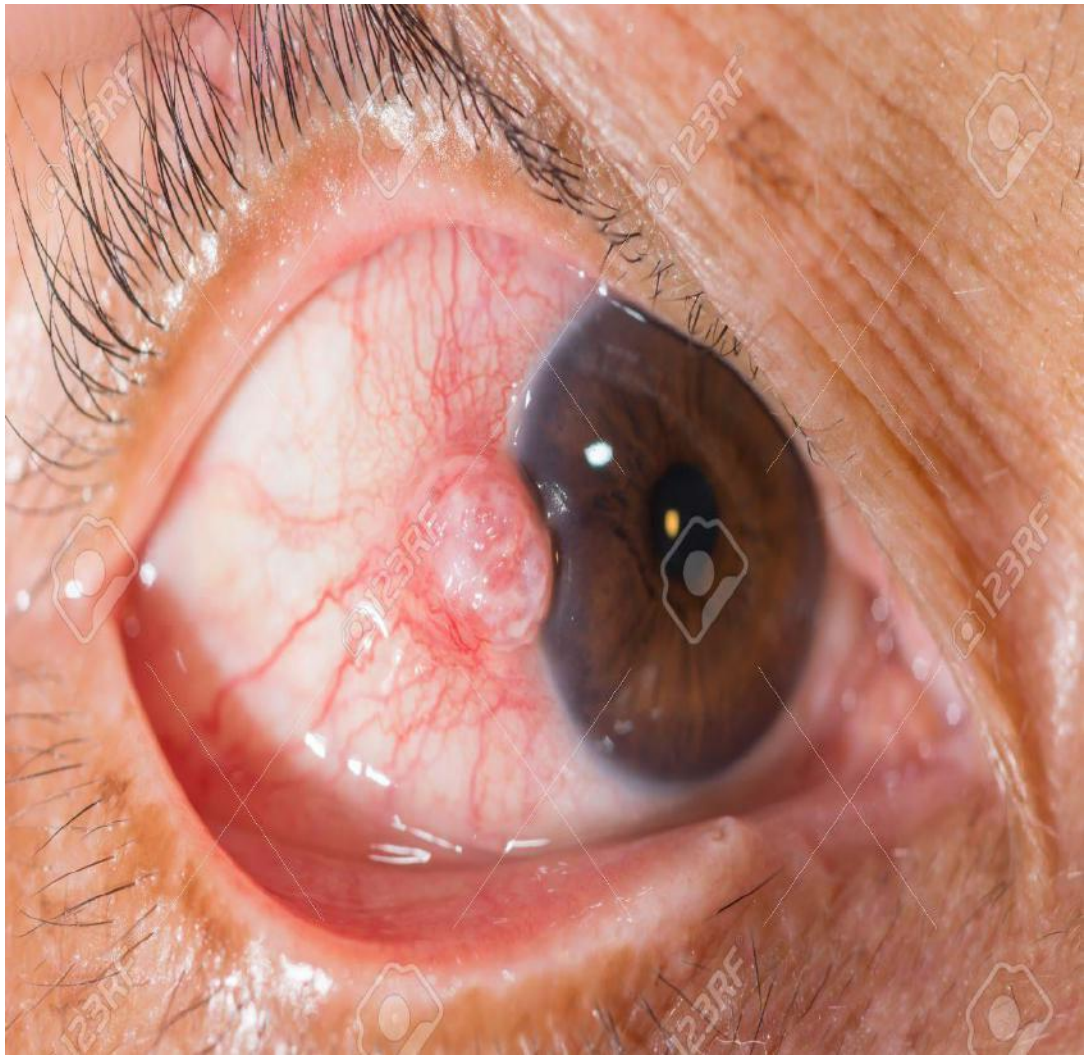
- Simple episcleritis
- Sectorial 70% in which only section is involved
- Diffuse 30% whole episcleral is involved

- Nodular episcleritis is with a localized movable nodule

- Pathology this is a non-granulomatous inflammation with vascular dilatation with perivascular lymphocytes & plasma infiltration

Simple vs Nodular type





Clinical features

Diffuse Scleritis

- Commonest type
- Sectoral /diffuse redness.
- Resolves spontaneously in 1 weeks

Nodular Scleritis

- Localized, raised, congested nodule
- Takes longer time to resolve
- Sclera not swollen
- Sclera appears translucent

Clinical features

- Symptoms
- Usually sudden onset
- Female >male
- 20-35 yrs of age
- Sectorial involvement is common in 70%. Redness usually occur in one sector
- Hotness, pricking sensation, & or discomfort is common

Signs

- Redness varies from mild to severe pink colour
- Sectoral/ diffuse
- Straight inflamed vessels goes posteriorly from the limbus
- The lesion over the deep tissue with application of cotton buds

Diagnosis

- With clinical features
- Topical phenylephrine 2.5% application will blanch the superficial episcleral vessel but not the deep scleral one
- D/D
- Conjunctivitis
- Scleritis

Treatment

- Self limiting; generally within 1-2 weeks
- Topical artificial tears
- Topical vasoconstrictor to reduce the redness
- Topical weak steroid
- Topical NSAID may be required such as diclofenac, naproxen flurbiprofen etc etc

Scleritis

- An inflammatory condition of the sclera
- About 50% are associated with systemic immunological disease
- So systemic problem should be searched out & treated

Aetiology

- Inflammatory with systemic disease such as
- Rheumatoid Arthritis; most common about 40% of cases
- Systemic lupus erythematosus
- Wegners granulomatosis
- Poly arthritis nodosa, Ankylosing spondylitis, Giant cell artritis, inflammatory bowel disease syphilis
- Infective ; such as
- Bacterial TB, syphilis
- Viral such as Herpes zoster

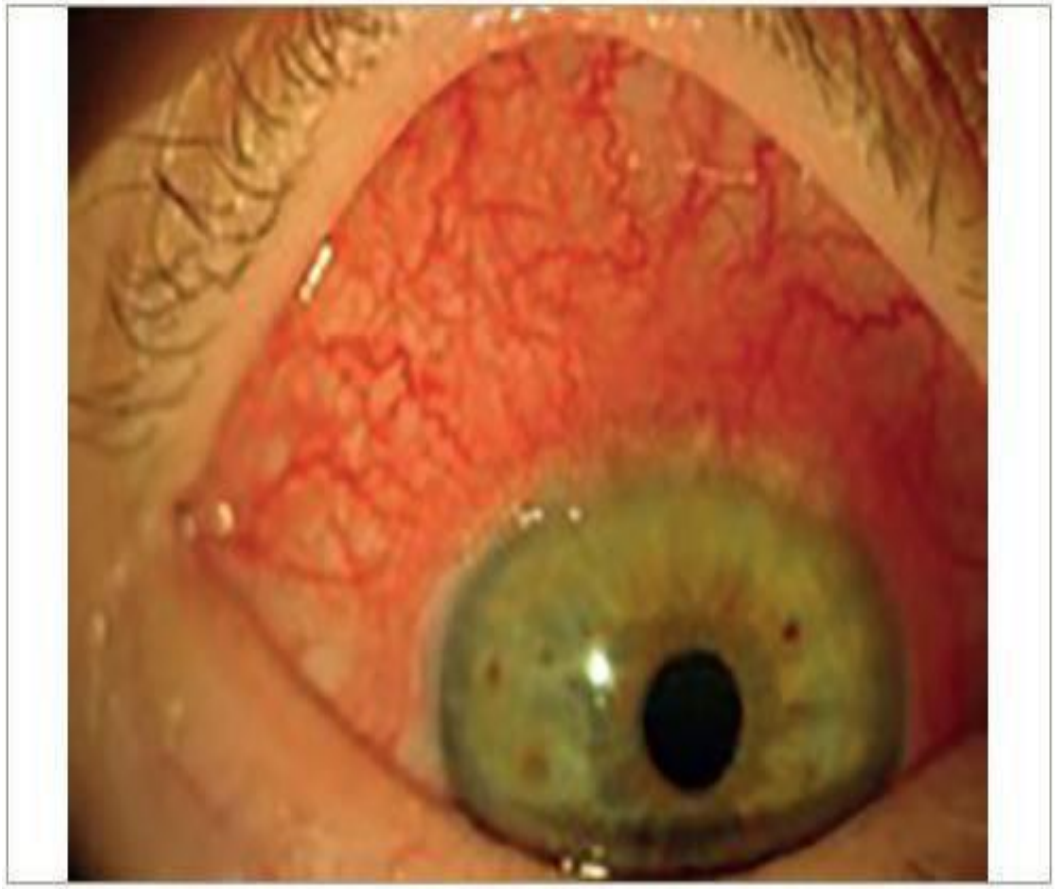
Pathology

- It is an immune mediated vasculitis that destroys the sclera

Classifications

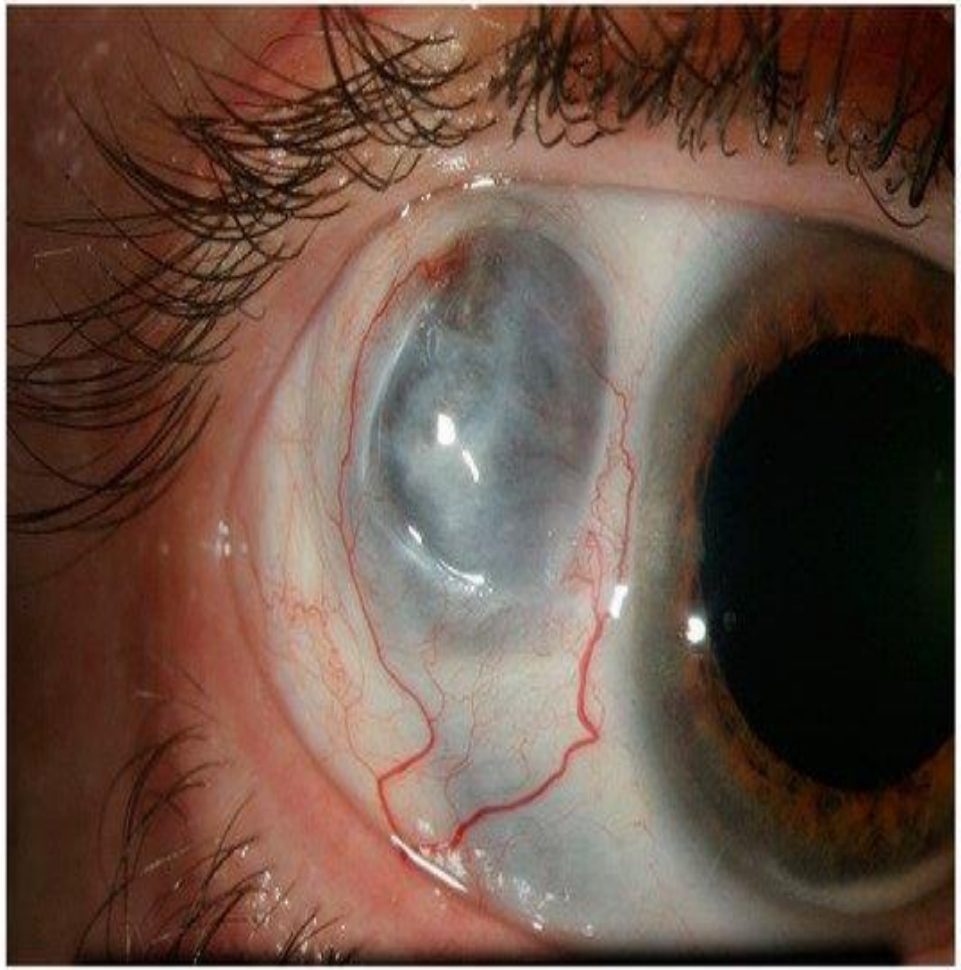
- A Anterior very common . About 98%
- 1 Non narcotizing further
- Diffuse
- Nodular
- 2 Necrotizing further
- With inflammation
- Without inflammation
- B Posterior less common about 2%

Figure 1: Slit-lamp photograph depicting diffuse anterior scleritis



Note the dilated blood vessels and generalized inflammation.

Figure Necrotizing scleritis without inflammation, or scleromalacia perforans



Anterior non-necrotizing

- DIFFUSE
- Relatively benign condition with mild or no visual loss
- Pain is mild
- Rednes is sectoral or diffuse
- Distortion of the normal radial vessel pattern is maintained

- NODULAR
- Pain is moderate
- Red nodule is visible
- Vessel cant be moved over the lesion
- No vascular blanching with 2.5% phenylephrine
- Visual loss in 25% of cases

Anterior Necrotizing type

- WITH INFLAMMATION it painful, initially mild with time becomes severe, cant even sleep
- Onset is gradual
- Bilateral in 60%
- Associated with systemic diseases is common
- Mortality rate is 25% within 5 yrs
- Visual prognosis is poor

- SIGNS on examination the deep vascular complex is congested
- Signs of scleral necrosis with avascular patch due vascular distortion & occlusion
- Conjunctival ulceration over the scleral necrosis
- After resolution thin sclera appears bluish due to underlying uvea

- WITHOUT INFLAMMATION also called as scleromalacia perforin
- Bilateral usually
- Common in women
- No pain is there
- On examination yellowish necrotic patch of sclera in uninflamed area
- Progressive with increasing melting & thinning

Posterior scleritis

- It is the inflammatory condition of sclera posterior to the equator
- Pain & vision loss are common
- Lids may become edematous
- Proptosis
- Ophthalmoplegia
- Anterior segment may be normal
- *Fundus examination may show*
- Swollen disc & retinal detachment

- Choroidal detachment & folds
- Macular edema
- *Diagnosis*
- Detail fundus examination with dilated pupil
- Ultrasonography show T sign due to thickening of the sclera & fluid in the Tenon space
- CT scan shows thick posterior sclera

Investigation

- Complete blood count
- Erythrocyte sedimentation rate ESR
- Circulating immune complex
- Seological test such as
- RA factor (Rheumatoid factor)
- ANA (antinuclear antibodies)
- Ant DNA antibodies (Double stranded DNA antibodies)
- ANCA antineutrophil cytoplasmic antibodies

- Angiotensin converting enzymes(ACE)
- VDRL & FTA- AB
- Uric acid level
- Chest x ray
- Mantoux test

Complications

- Corneal common 37% such as sclerokeratitis, stromal keratitis, marginal corneal melting
- Anterior uveitis
- Glaucoma
- Cataract
- Staphyloma formation

Course

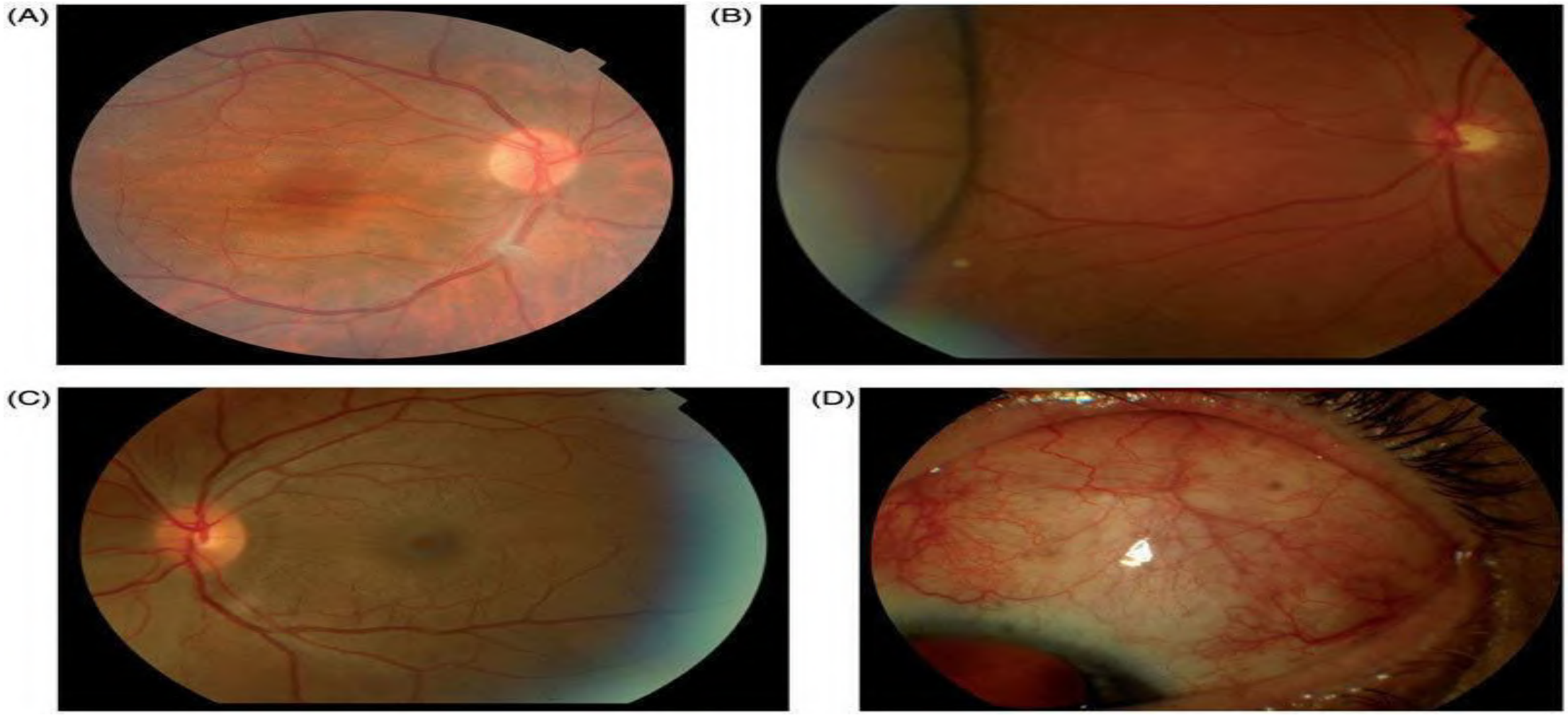
- Complications occur in late stage
- Visual loss in 27-37%
- Uveitis in more than 30%

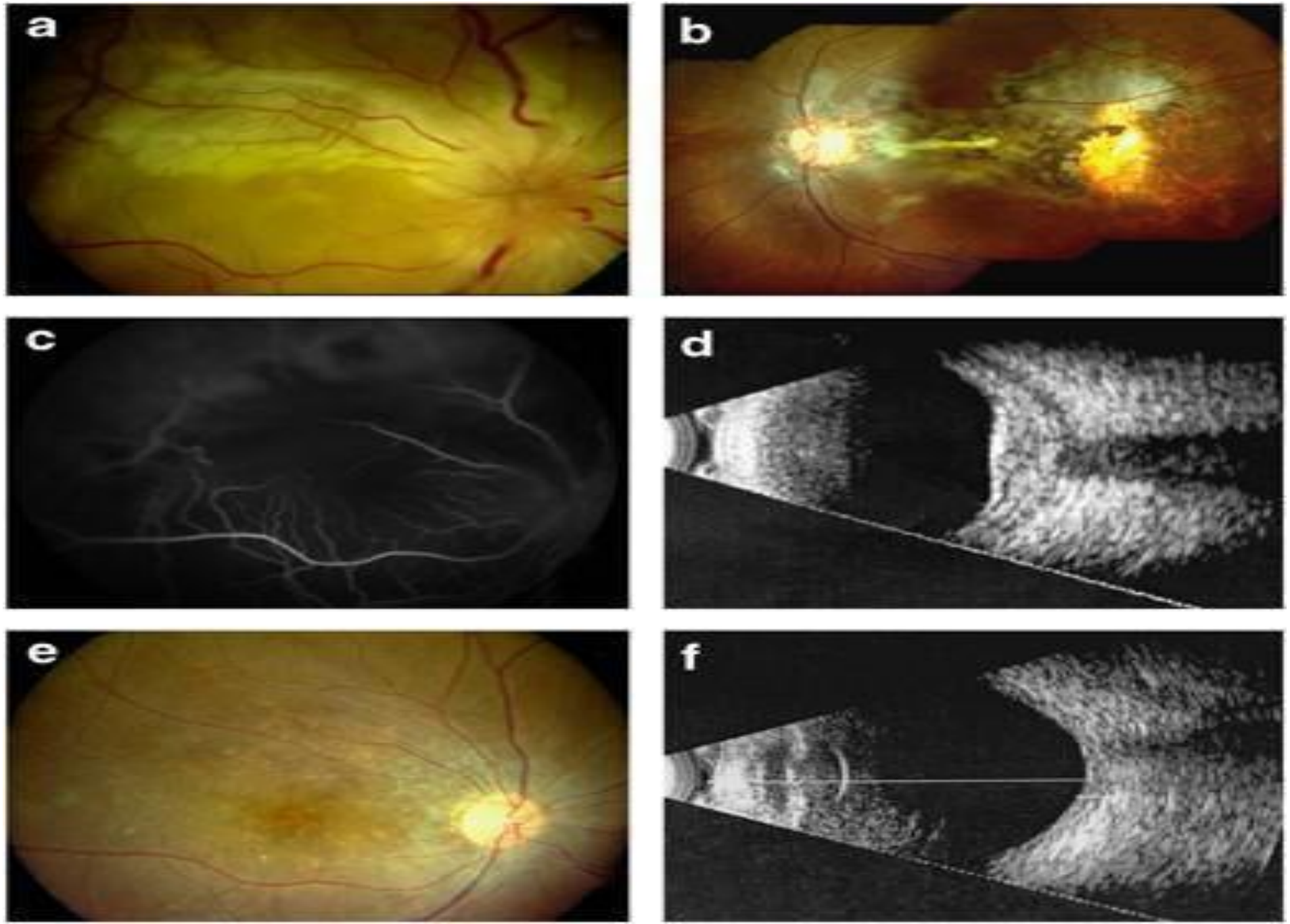
Treatment

- Treat the systemic disease
- Treat the associated ocular problem like cataract glaucoma
- Treatment of scleritis
- Topical steroid
- Systemic NSAID effective also for systemic problem as well as for for ocular

- Systemic steroid for Anterior necrotizing & posterior scleritis
- Systemic immunosuppressive drugs such as cyclosporin, methotrexate cyclophosphamide when steroid are not affective or contra indicated
- Sub-conjunctival steroid injections such as triamcinolone-acetonid for Anterior necrotizing & non-necrotizing scleritis

Photographs of posterior scleritis showing presence of choroidal folds (A), choroidal detachment (B), retinal striae (C), and associated anterior scleritis (D).




a
b
c
d
e
f

Angiographic, and ultrasonographic presentation, and post-treatment resolution of posterior scleritis with retinal artery occlusion and exudative detachment

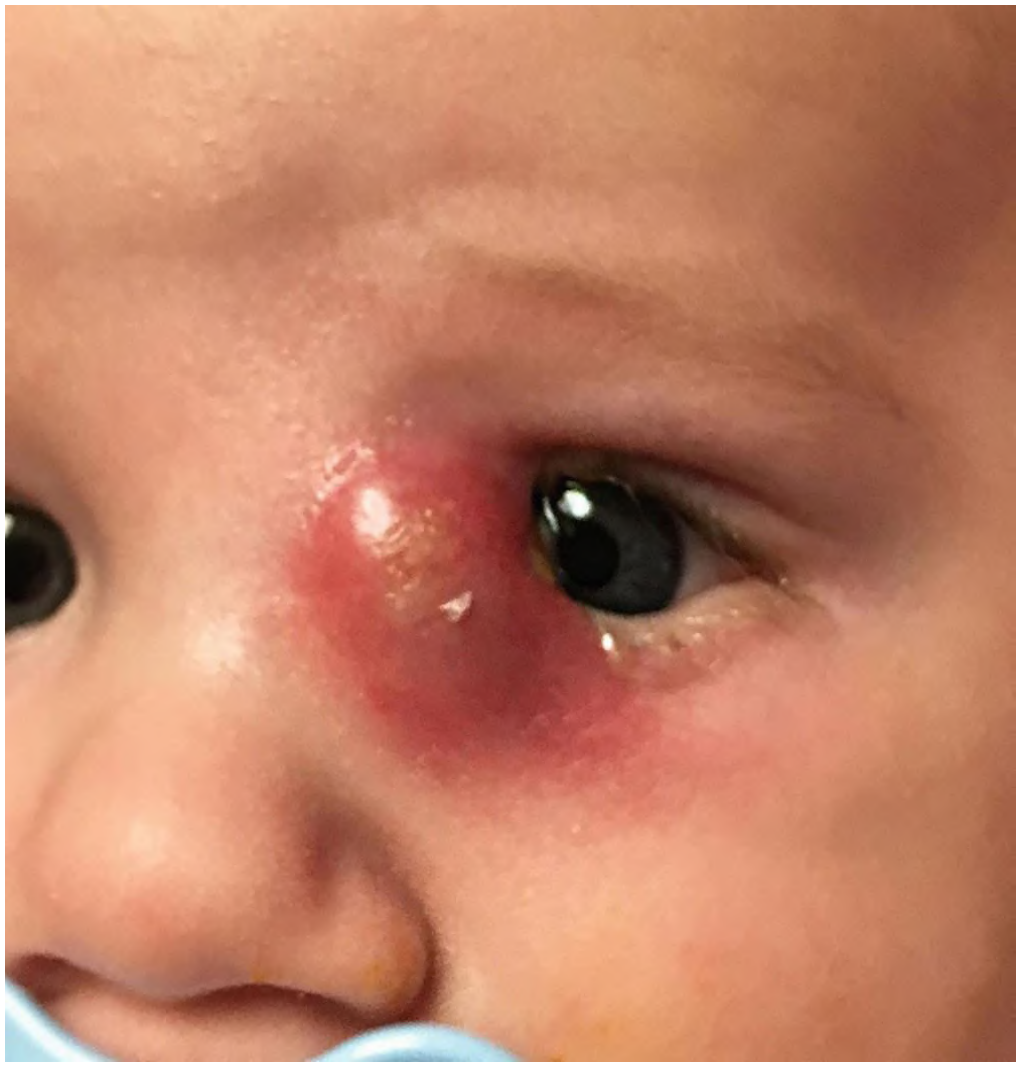
- (a) Fundus view of the right eye, showing optic disc oedema, venous dilatation, serous retinal detachment, and segmental pallor along the superotemporal arcade, rendered less prominent by the turbidity of the subretinal fluid.
- (b) Left fundus showed the evidence of resolved inflammation with severe chorioretinal scarring, retinal pigment epithelial atrophy, and pigment migration.
- (c) Mid-phase fluorescein angiogram shows delayed transit of dye from the superotemporal branch retinal artery, with empty corresponding capillaries and vein. The faint choroidal hyperfluorescence is probably indicative of associated inflammatory activity.
- (d) B-scan ultrasound through the right optic nerve shows gross hyper-reflective thickening of the retina-choroid-sclera complex (5.06 mm), fluid in the sub-Tenon's space (T-sign), and squaring of the optic nerve shadow. (e) 4 months later, the fundus shows complete resolution of the inflammatory and vascular sequelae, with residual granular pigmentary stippling of the posterior pole. (f) B-scan confirms the resolution of scleral thickness (1.39 mm) and sub-Tenon's fluid, with normalized optic nerve shadow.
-

بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

Clinical scenario

- A child of 6months is brought to you in out patient c/o watering & discharge from his Rt eye. On examination there is watering & discharge from the eye . Eye ball is normal . Lt eye is normal
- What is the most probable cause

- Birth trauma
- Buphthalmas
- Congenital NLD block
- Punctal atresia
- Conjunctivitis

- 


Theme Red eye

- NLD obstruction

- Dr Nazullah
- Associate Professor
- Kgmc/Hmc

NASOLACRIMAL DUCT OBSTRUCTION

Congenital

Acquired

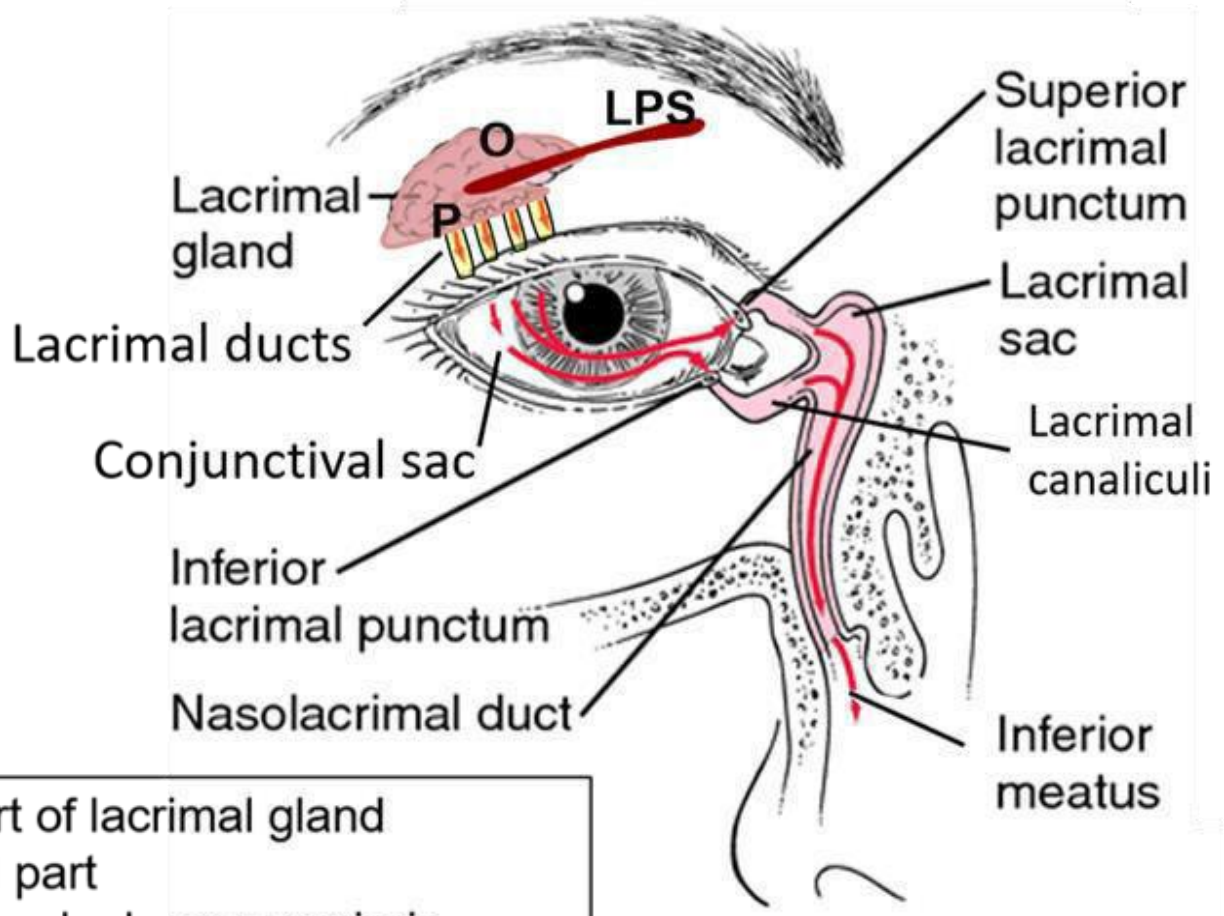
- Congenital

Anatomy of the lac system. ???

- Secretary portion ???

- Excretory portion ???

Anatomy of lacrimal system

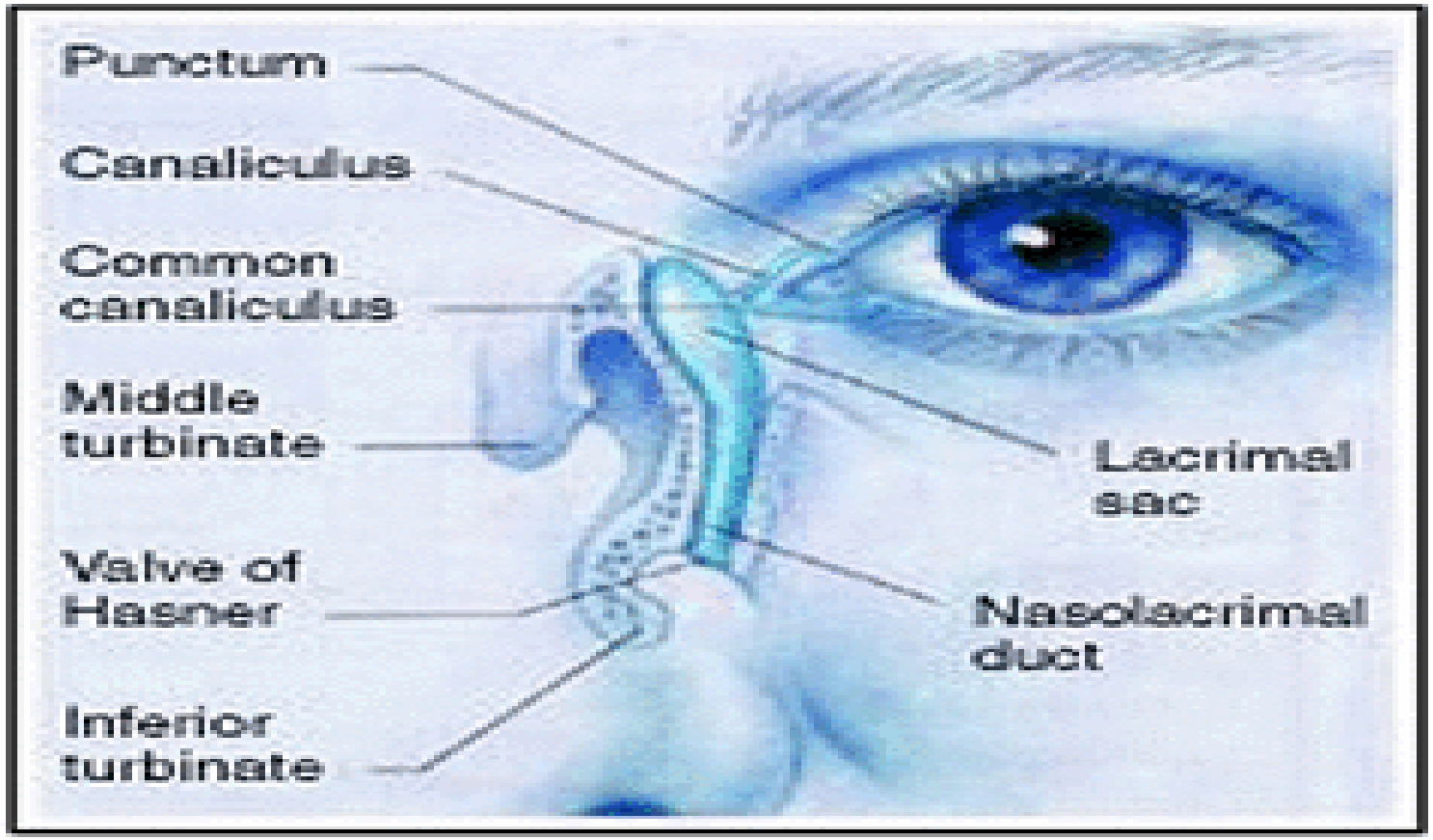


O- orbital part of lacrimal gland
P- palpebral part
LPS- levator palpebrae superioris

Lacrimal gland
↓
Lacrimal ducts
↓
Conjunctival sac
↓
Lacrimal punctum
↓
Lacrimal canaliculi
↓
Lacrimal sac
↓
Nasolacrimal duct
↓
Inferior meatus of nose

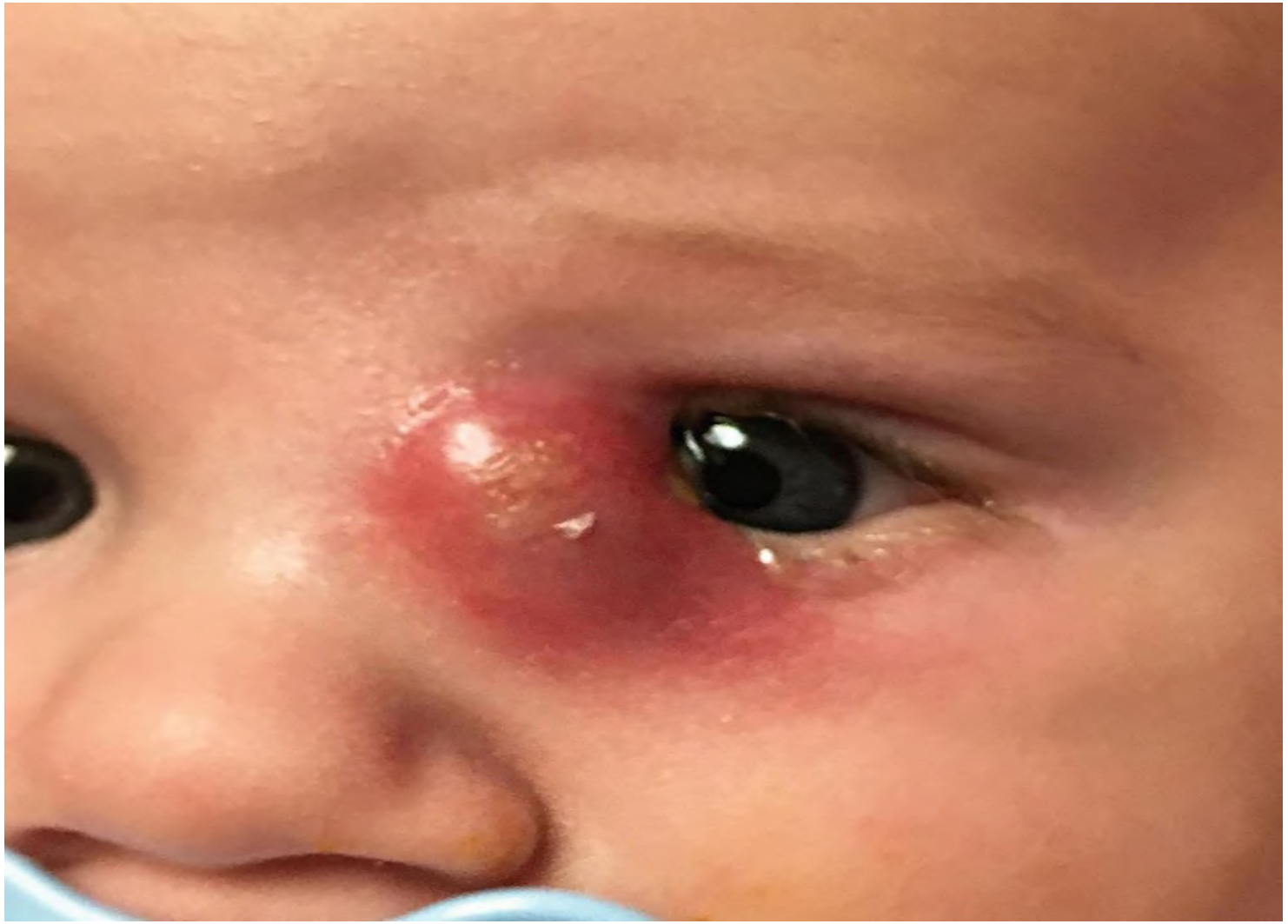
Lacrimal drainage portion

- The lacrimal system
- Punctum .3mm
- Canaliculus, vertical 1-2mm
- Horizental 6-8mm
- Common caliculus
- Lac sac 12-15mm
- Nasolacrimal duct 15-18mm



Congenital Watering/Epiphora, causes

- Congenital NLD block
- Punctal atresia
- Conjunctivitis
- Birth trauma
- Congenital glaucoma
- Foreign body

- 

Objectives

- At the end of this session the 4th yr MBBS student should be able

- Enlist Different causes
- What are Clinical features
- What are Different Treatment options
- How Regurge test is performed

Etiology/Causes;

- Is common congenital lacrimal problem
- Occurs about in 5% children
- Due to non-canalization of the membrane (Hasner valve) at the lower
- end of the nasolacrimal duct
- Which usually opens spontaneously within few weeks of life
- Failure to open will cause watering and infection

Clinical features;

- Symptoms occurs typically within 3-4 weeks of life
- There is watering with with sticky mucoid or mucopurulent discharge

- It is usually uniocular but may be bilateral
- Regurge is positive by giving gentle pressure over the lac sac area

Complications

- Conjunctivitis
- Acute dacryocystitis
- Chronic dacryocystitis
- Mucocele
- Fistula formation

Differential Diagnosis

- Punctal atresia
- Conjunctivitis
- Birth trauma
- Congenital glaucoma
- Foreign body
- Keratitis / uveitis

Contnd Management

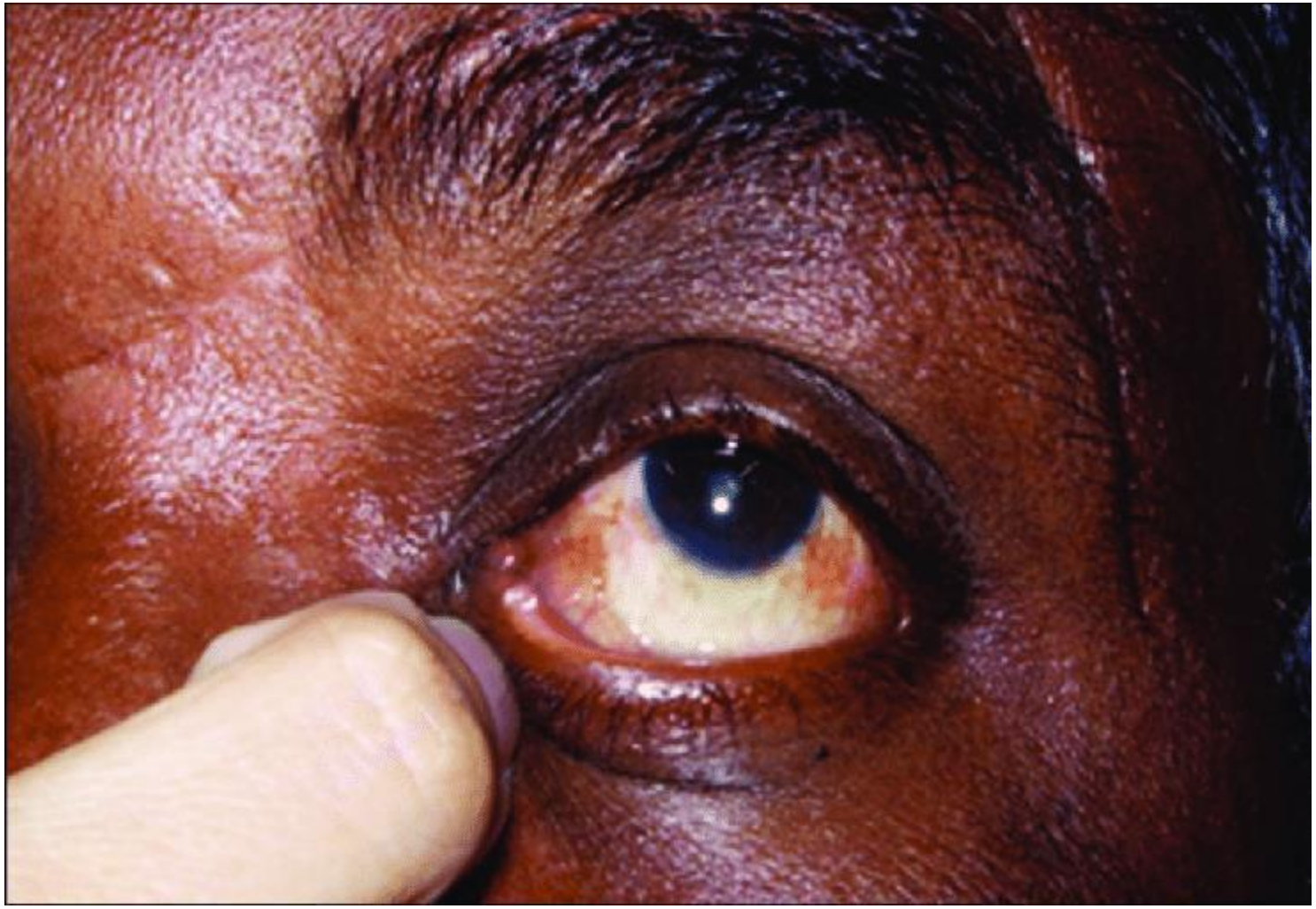
- Conservative
- Massage & Topical Antibiotics
- By applying Digital pressure over the lac sac will increase the hydostatic pressure and may rupture the membranr
- About 10 stroke 4-5 times daily
- Topical & systemic antibiotic to prevent and treat secondary infection
- Results ; about 90-95% in 6-9 months of age

Difference between ??? Audience ???

- Regurge test

- Lacrimal massage

Regurge



Positive regurge

- 

Lacrimal massage

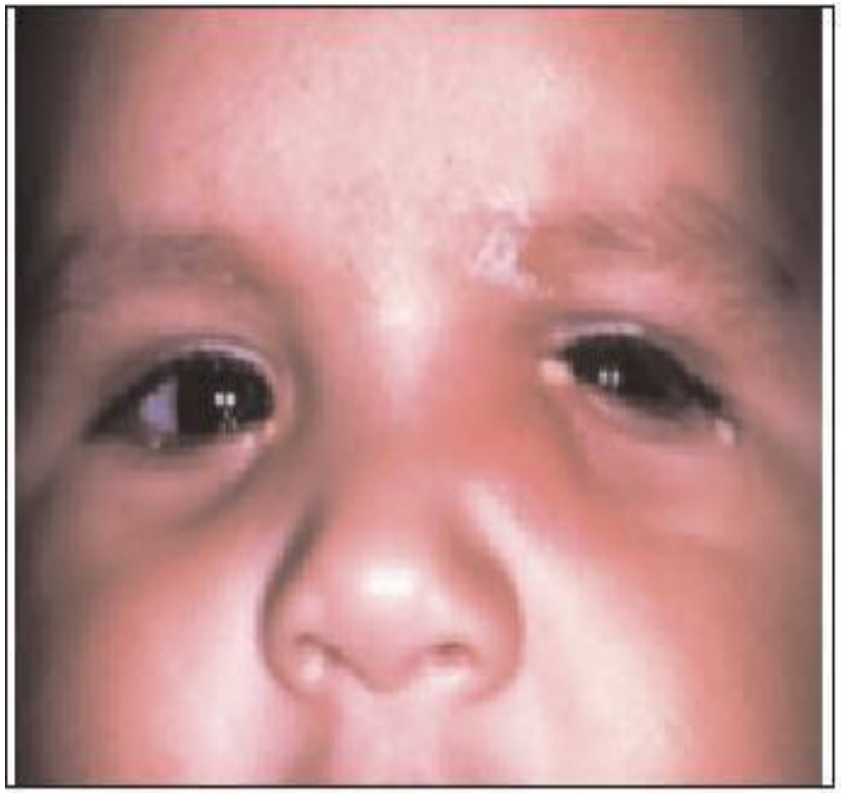
- 

Figure 1: BNLD before massage.

Figure 2: Method of massaging.

Treatment Contd

- Probing should be delayed 8- 12 months of age
- Done UGA
- A probe is passed through puctum, canaliculus, lac sac into the nasolacrimal duct and nose
- Results The results of probing are excellent. 90% of cases are cured by the 1st probing and about 6% in the 2nd probing

Probing

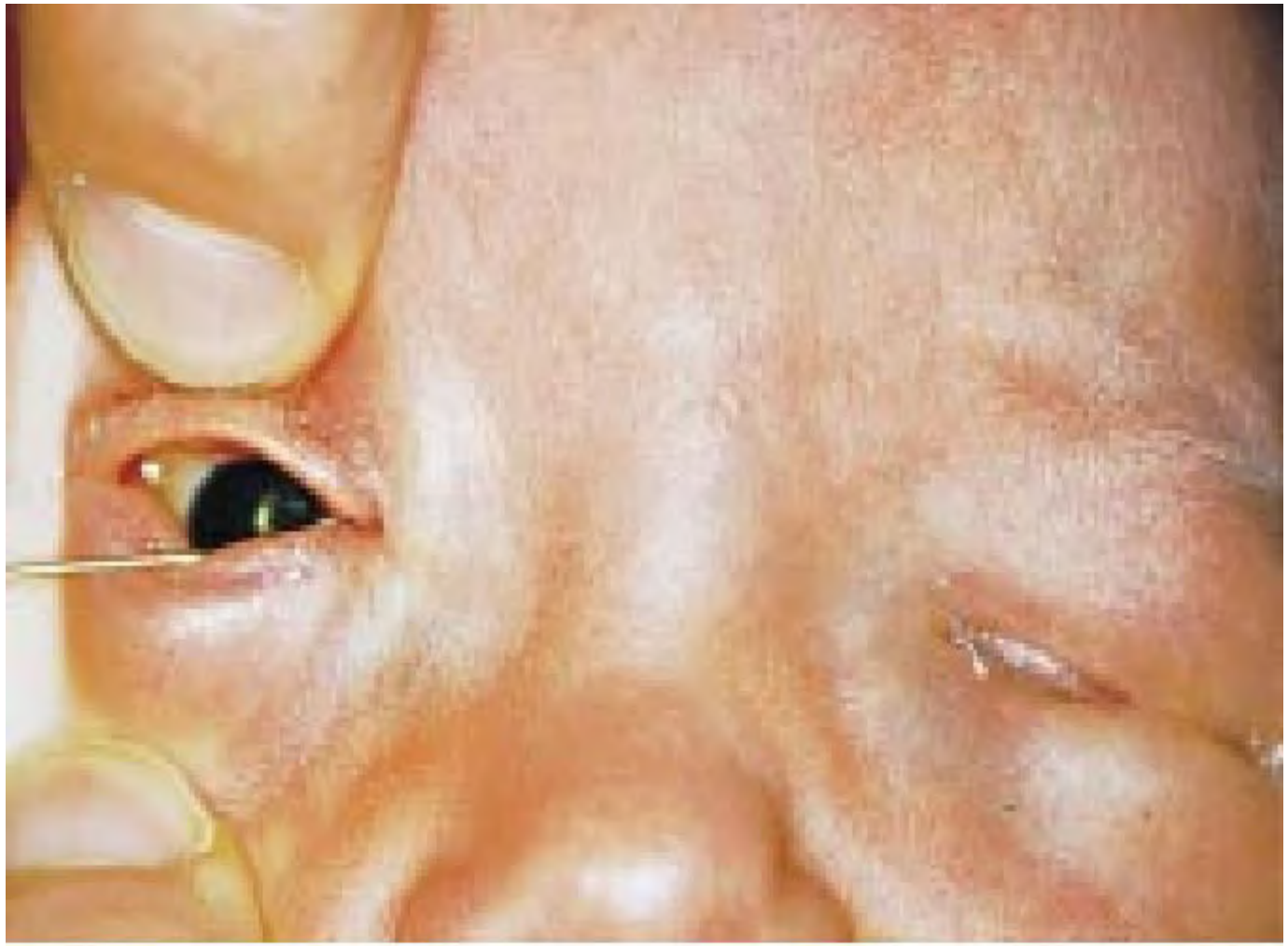
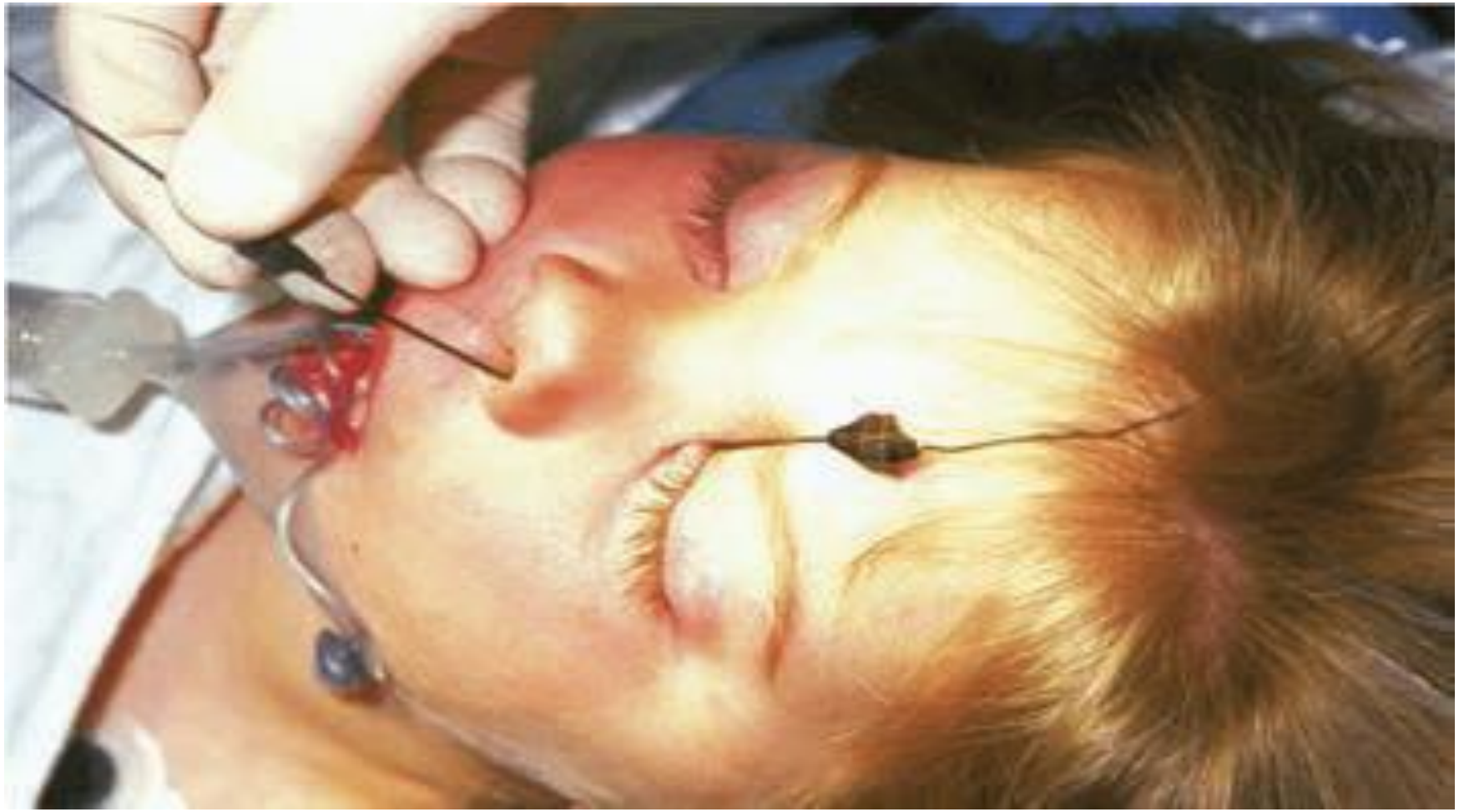
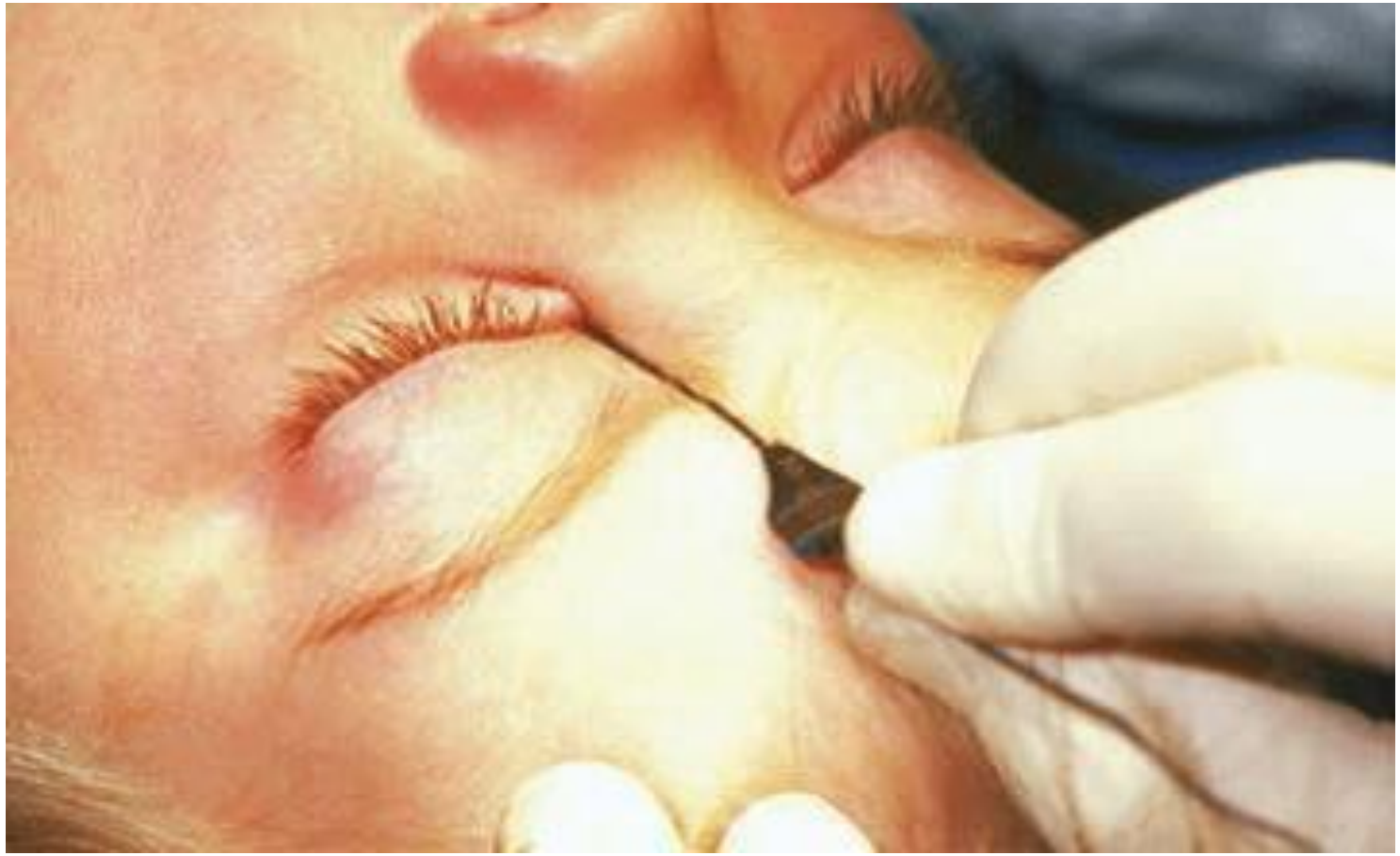


Figure 5 : Sondage de la portion horizontale de la voie lacrymale

Probing



Probing

- 

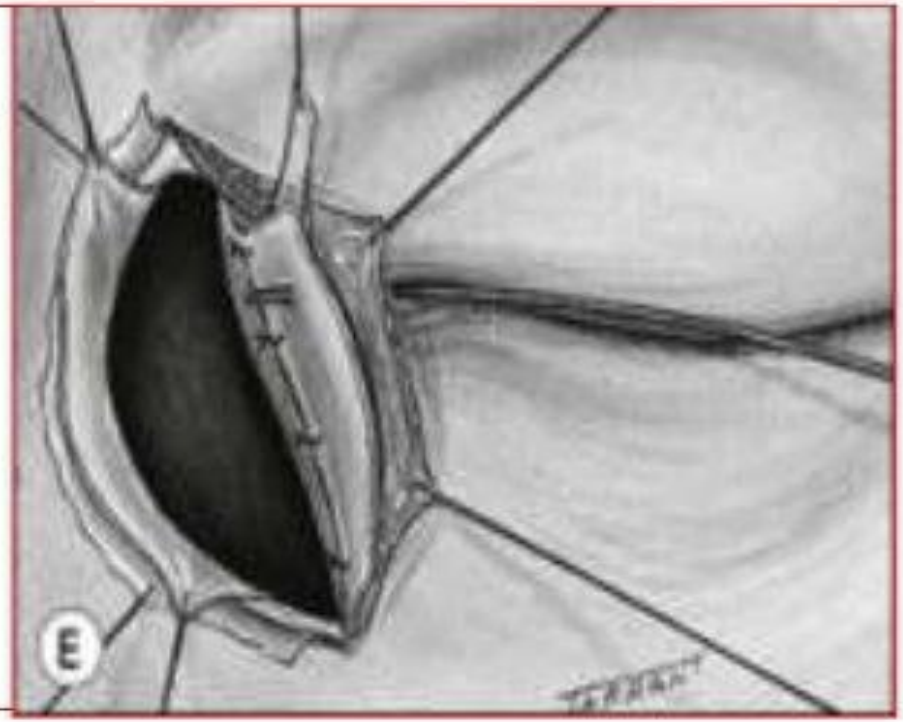
- Intubation without Dacryocystorhinostomy (DCR)
- Upto 2-3 yrs tube is passed in the lacrimal passages without DCR.
- Stays for 5-6 months

- Dacryocystorhinostomy
- After 4-5yrs of age dcr is done
- Fistula is formed between the lac sac and the nose by cutting the lac sac, the bone and nasal mucosa

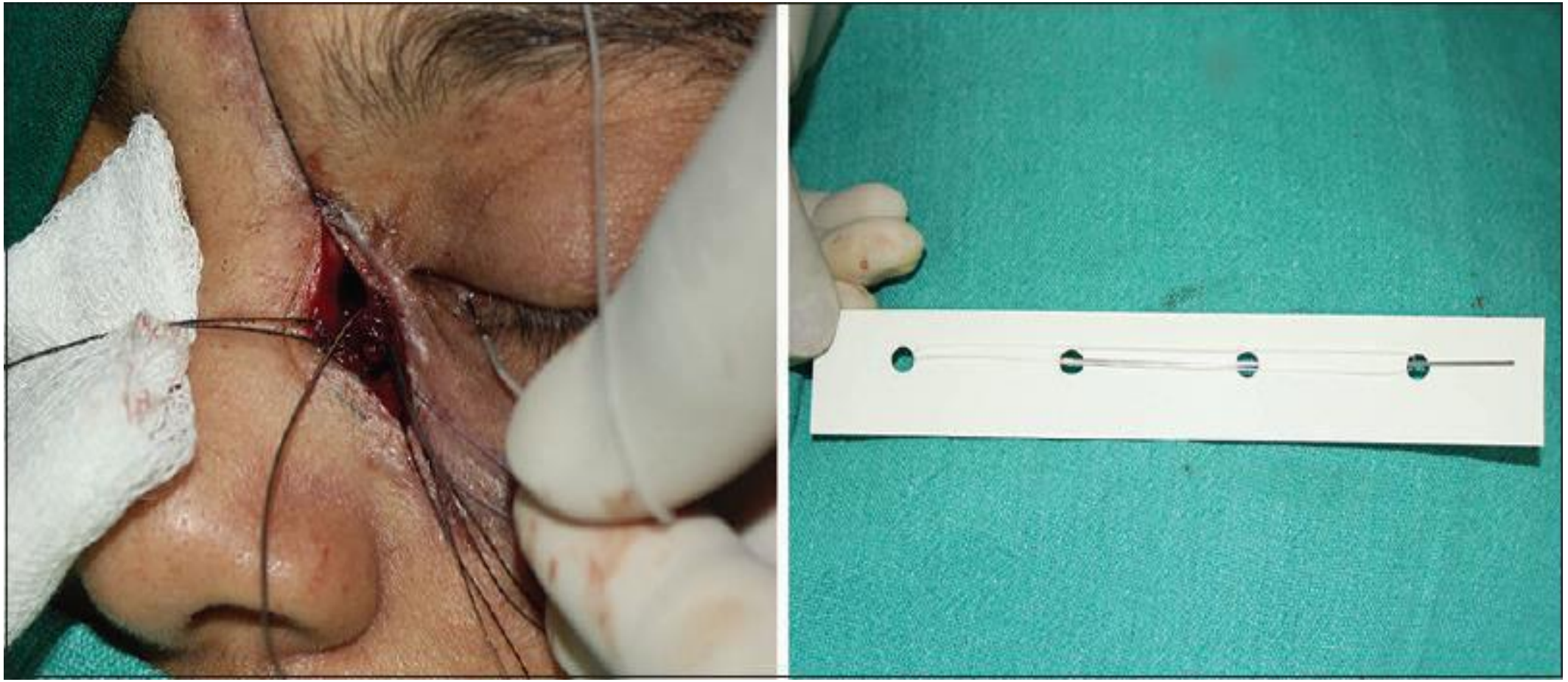

Probing
LVPEI
Dr Javed Ali

DCR

- The posterior flaps are sutured (Fig. 2.18E).
- Silicone intubation may be performed.



DCR



Clinical scenario

- A child of 6months is brought to you in out patient with CLDO with watering & discharge from his Rt eye. On examination there is watering & discharge from the eye . Eye ball is normal. Lt eye is normal
- Where is the usual site of obstruction.?

  - **Canaliculus**
  - **Common canaliculus**
  - **Lac sac**
  - **Lower end of NLD**
  - **Punctum**

Adults nasolacrimal duct obstruction

- CAUSES:
- Involutional stenosis, most common cause
- Inflammatory disease such as sarcoidosis, Wagner's granulomatosis
- Trauma , Tumors of nasopharynx
- Dacryolith etc etc.
- Clinical features;

Watering with mucoid and mucopurulent discharge

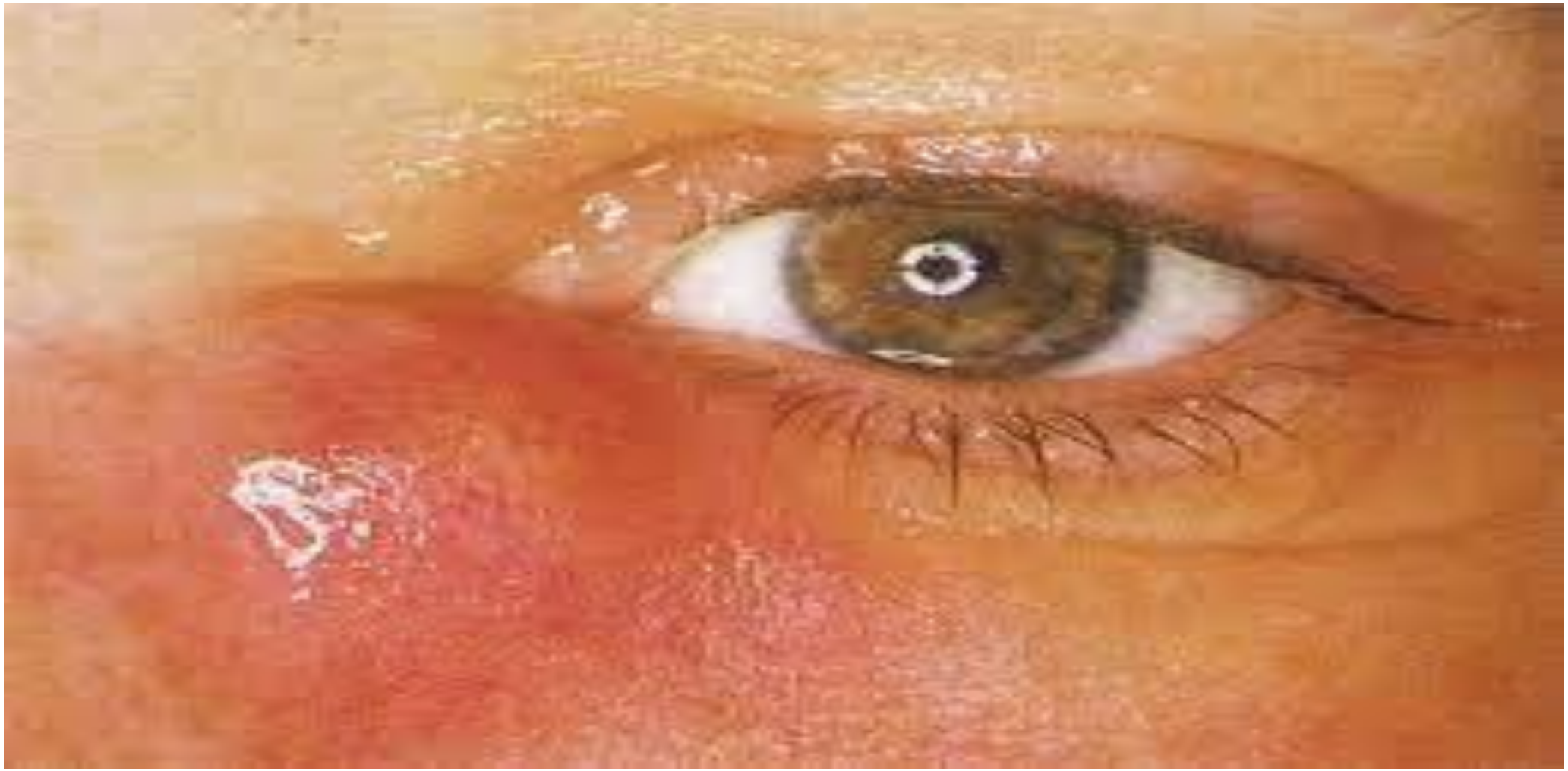
Recurrent Attacks of Dacryocystitis

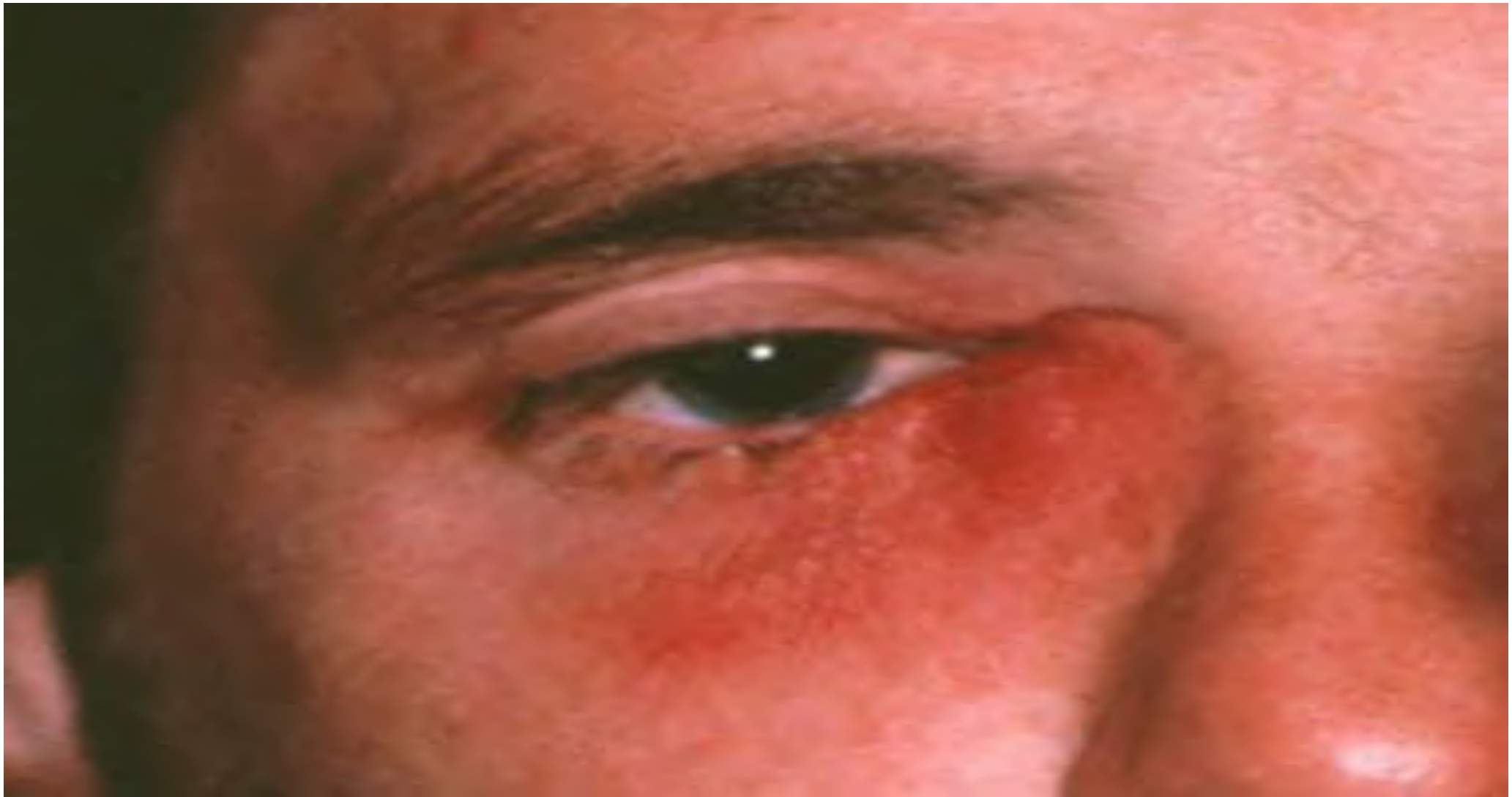
Conjunctivitis

Dacryocystitis

- Acute Dacryocystitis
- Chronic Dacryocystitis

Acute





Causes/Aetiology

- It may arise denovo or as a secondary infection of already obstructed nasolacrimal duct
- Organism like Bacteria such as Staphylococcus, Streptococcus and Pseudomonas invasion etc etc are the usual causes

Clinical features

- Painful swelling, redness and watering in the medial canthal area
- Examination shows swelling in the medial canthus, red and tender to touch
- Difficult to examine
- Regurge is difficult to perform, painful
- Discharge purulent mucopurulent
- Abscess formation may occur

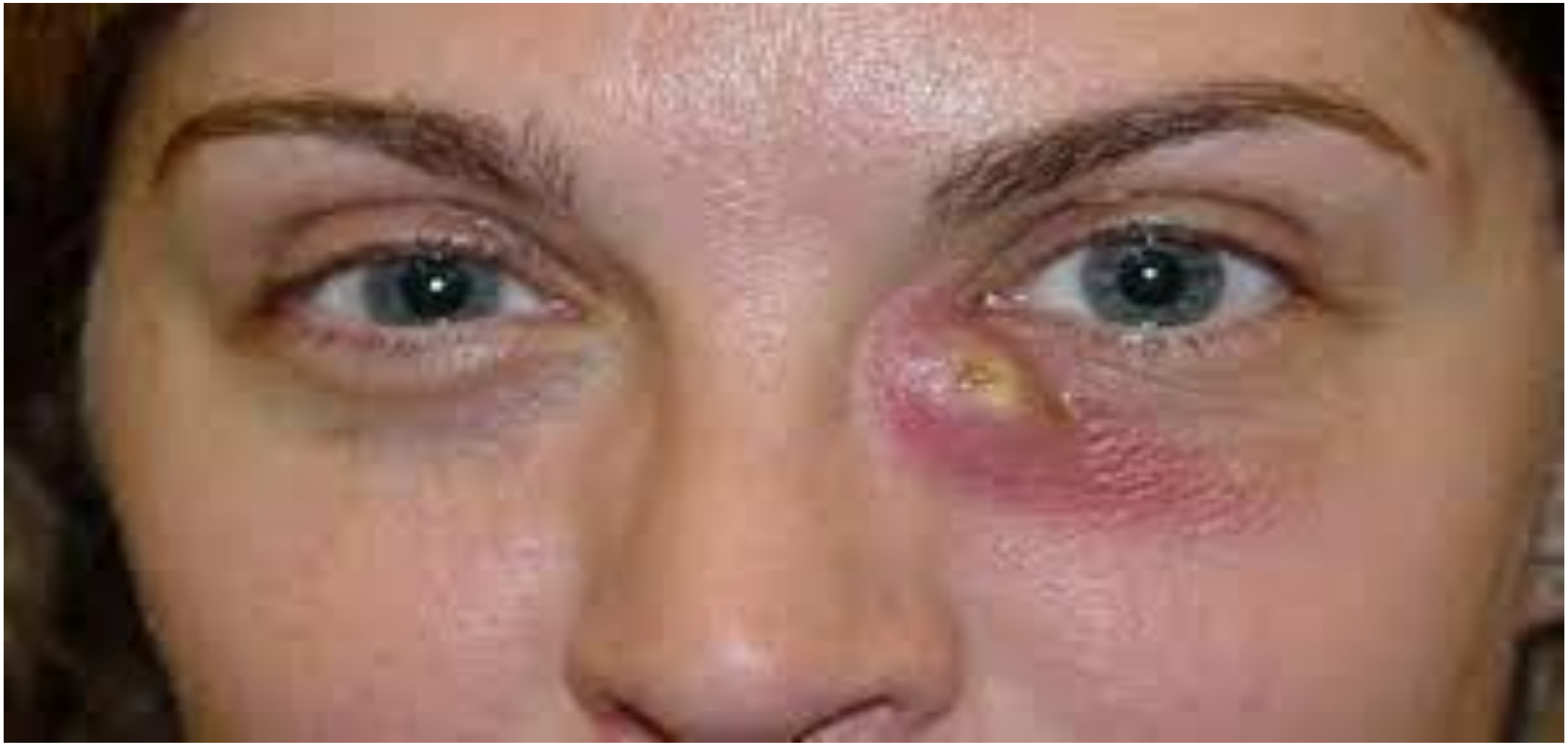
Complications

- Preseptal cellulitis
- Fistula formation

Cellulits/Abscess

Cellulitis

Fistula



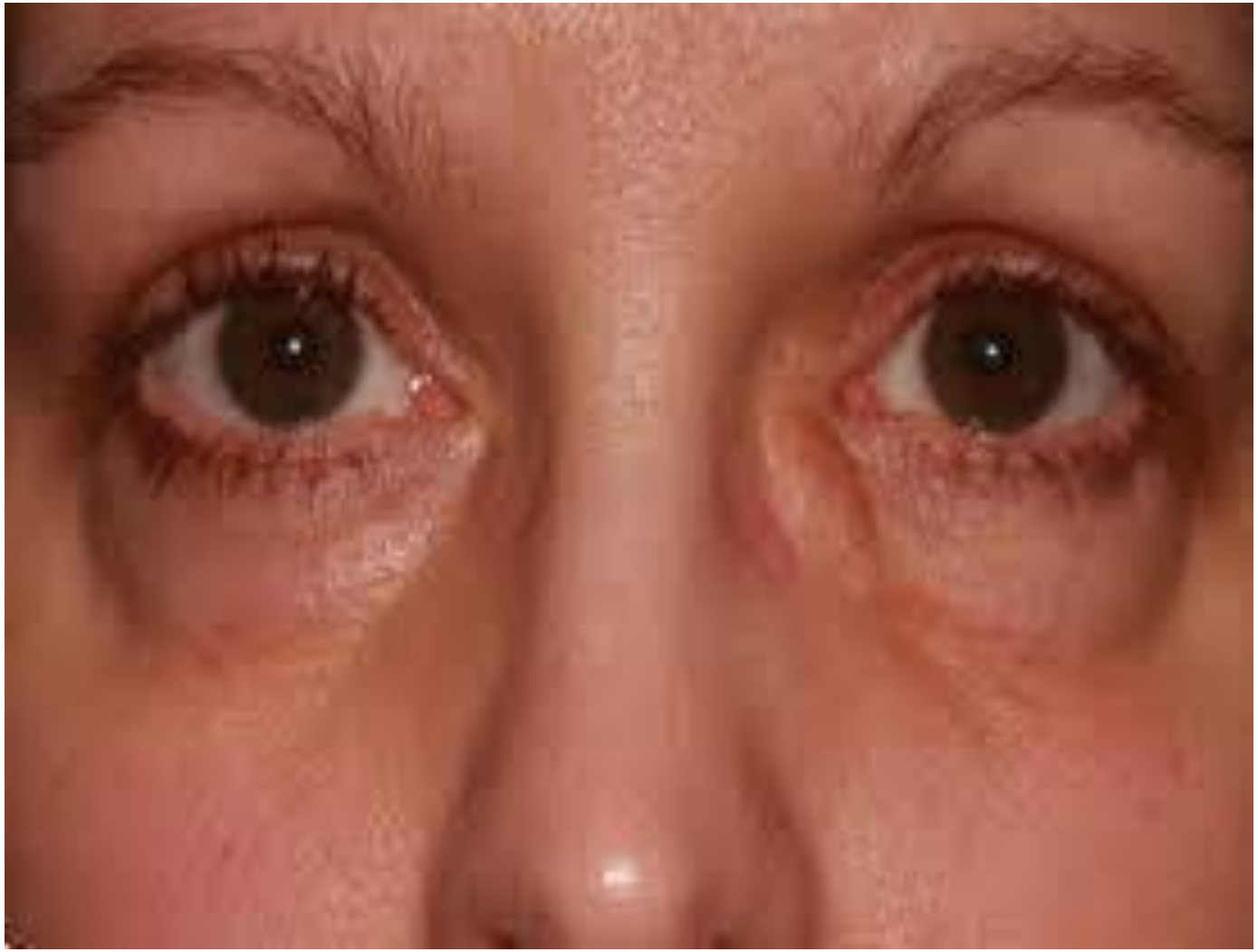
Treatment

- Antibiotic
- Analgesic

- Systemic and Local

- Hot compression

Chronic Dacryocystitis



Aetiology

- Is the chronic inflammation of the lac sac
- The impaired of the sac leads to the stasis of the tear flow which eventually leads to secondary infection by low virulent organism

Clinical features

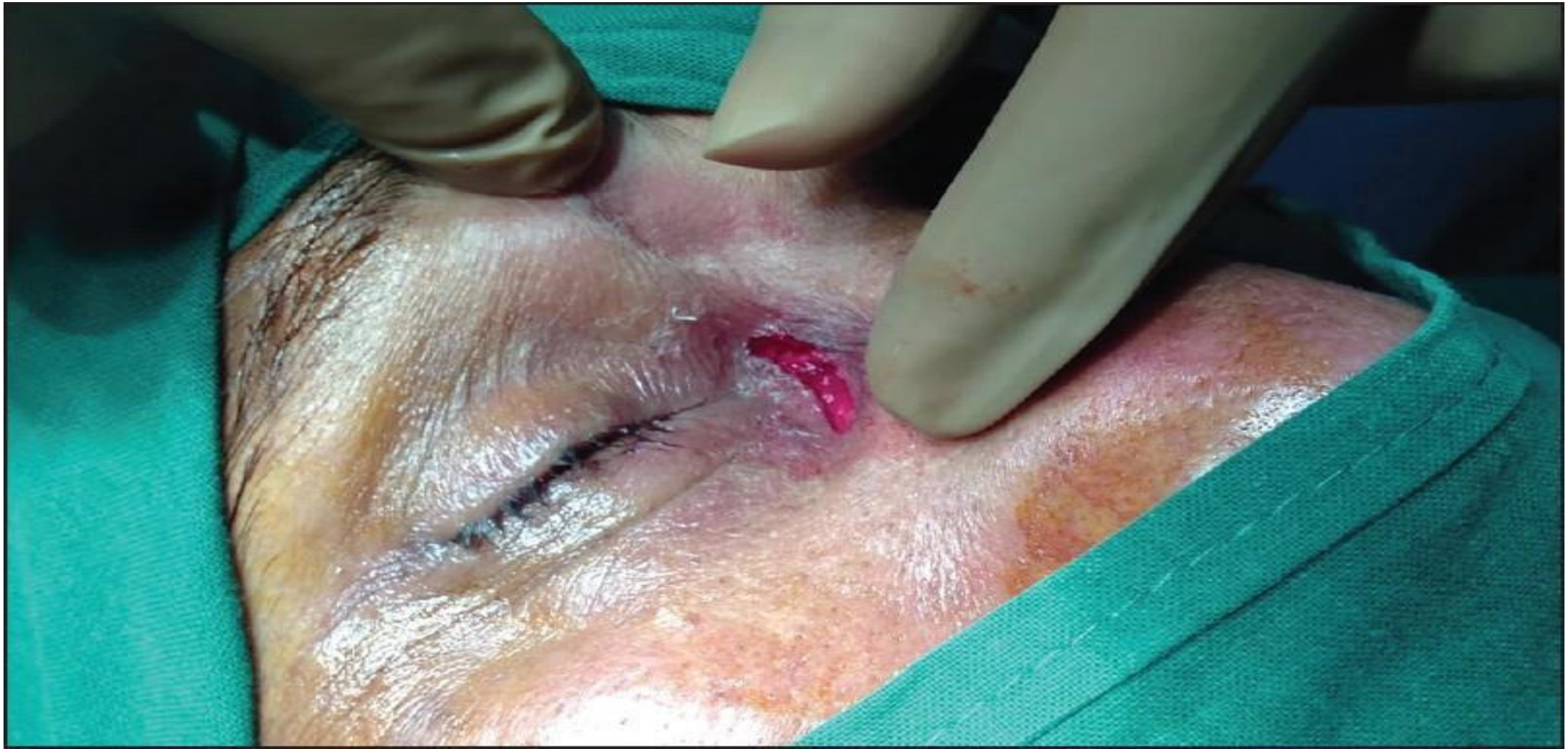
- More common than acute cdc
- C/o watering & discharge
- May be there is swelling in the medial canthal area
- Unilateral may b bilateral
- Regurge is positive with reflux of water, mucoid and mucopurulent material from the punctai.
- Mucocele formation
- May b associated with ch-conjuctivitis

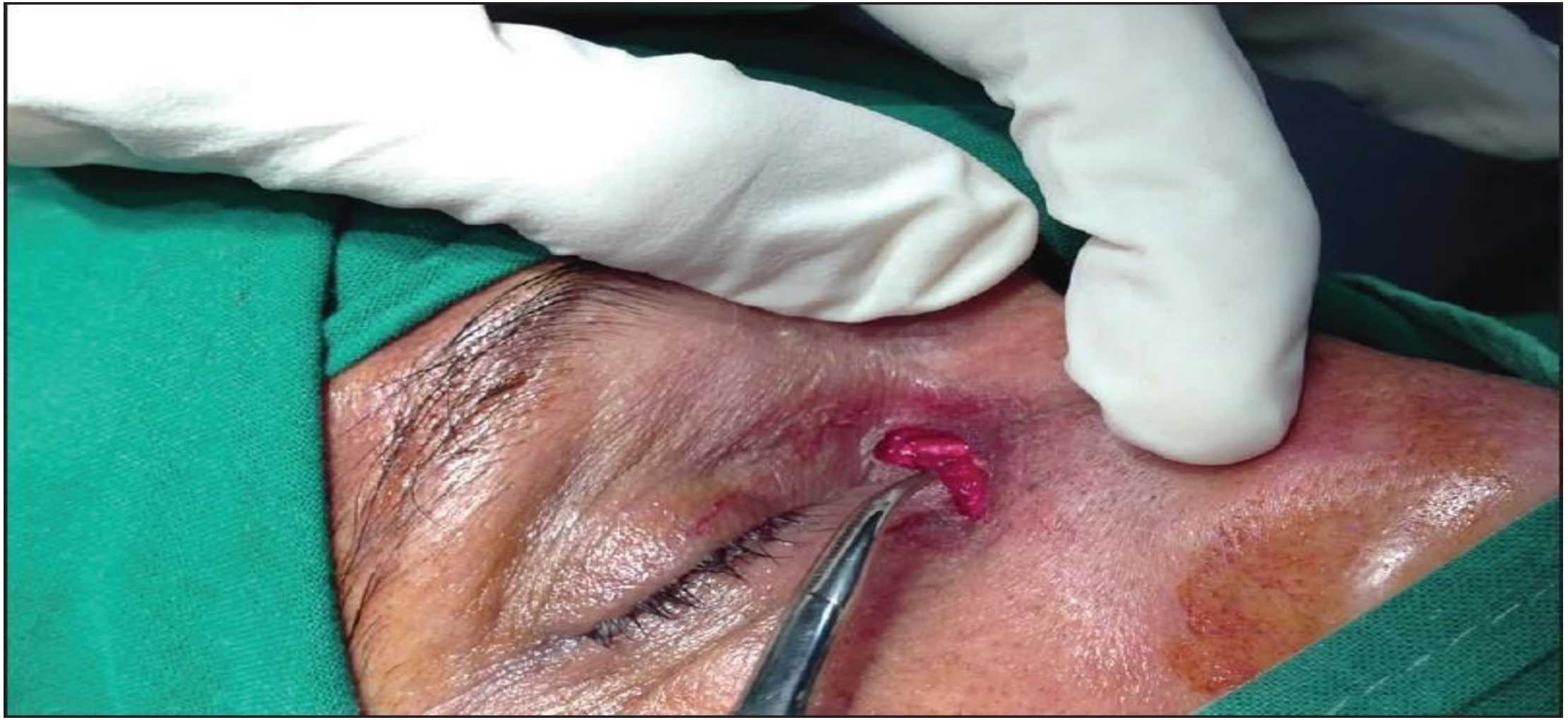
Probing syringing

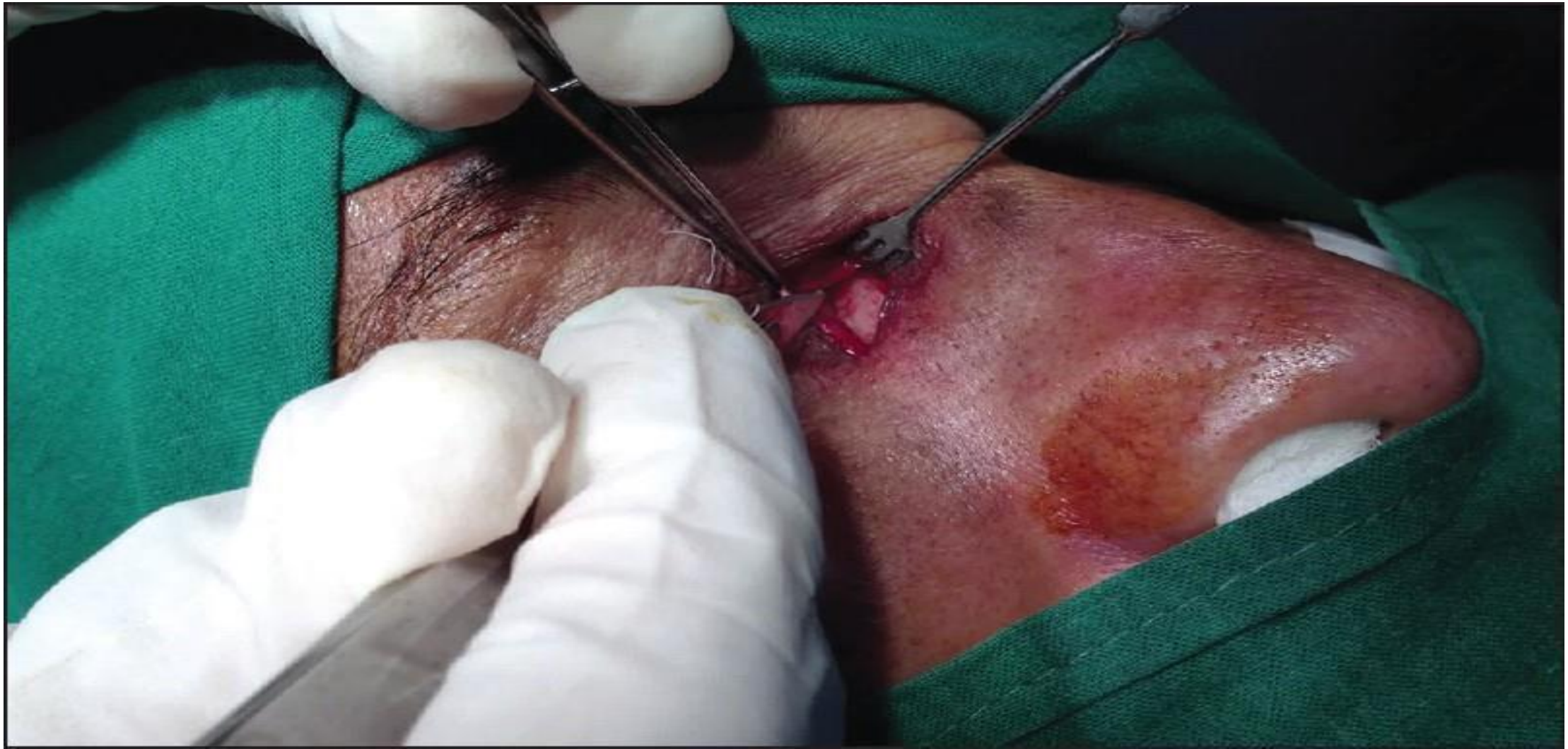
- Diagnostic/therapeutic; in adult it is done not only for therapeutic purposes but mainly for diagnostic purposes
- To relieve the obstruction
- To determine the level of obstruction

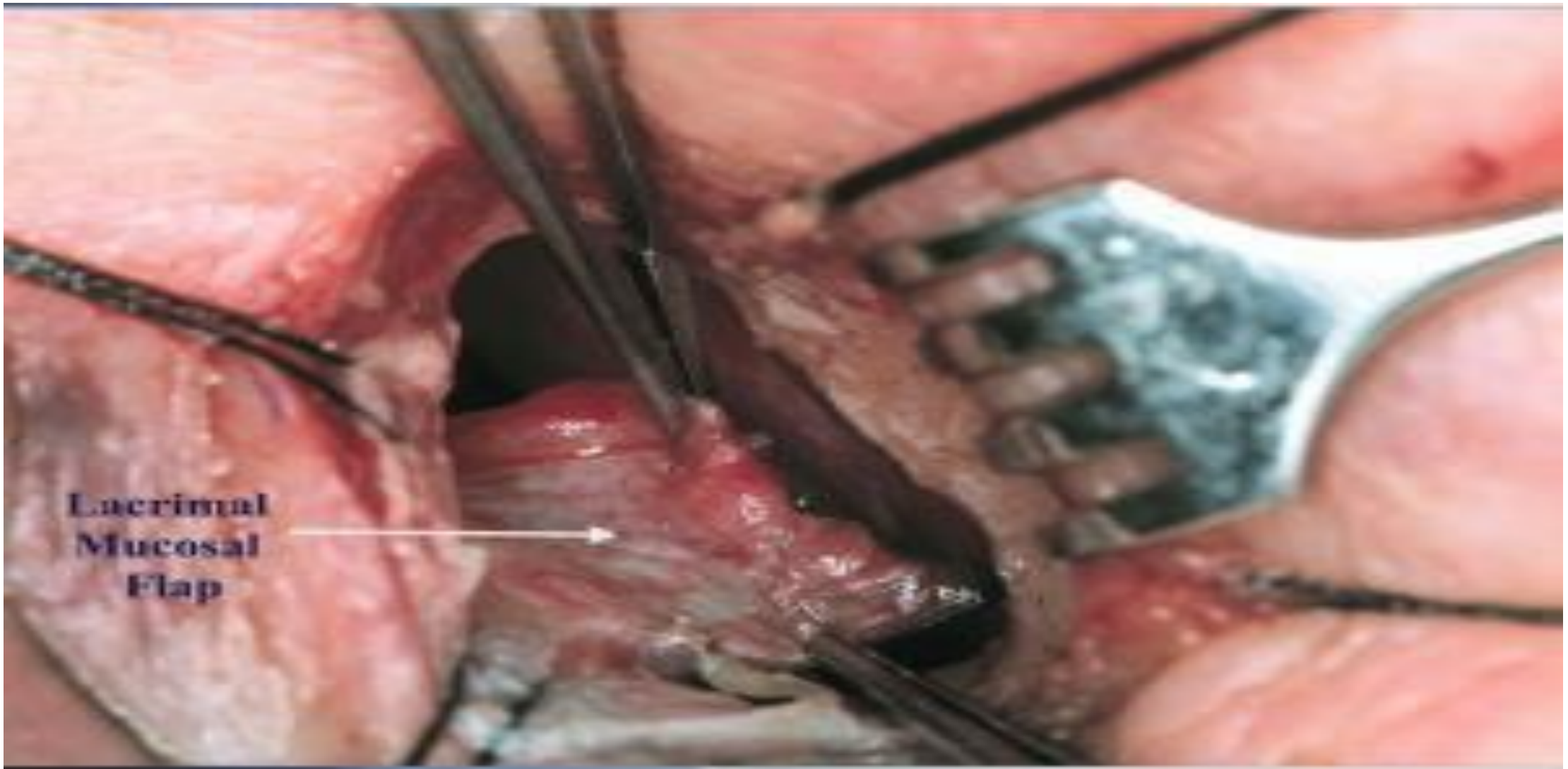
Treatment; Dacryocystorhinostomy(DCR)

- To make a communication between lac sac & nasal cavity
- Skin Incision
- Skin, muscle separation,
- Bone & Lac sac exposed
- Bone punching/cutting
- Nasal mucosa exposed
- Cutting and stitching of sac+mucosa with each other
- Muscle and skin stitches








Lacrimal
Mucosal
Flap

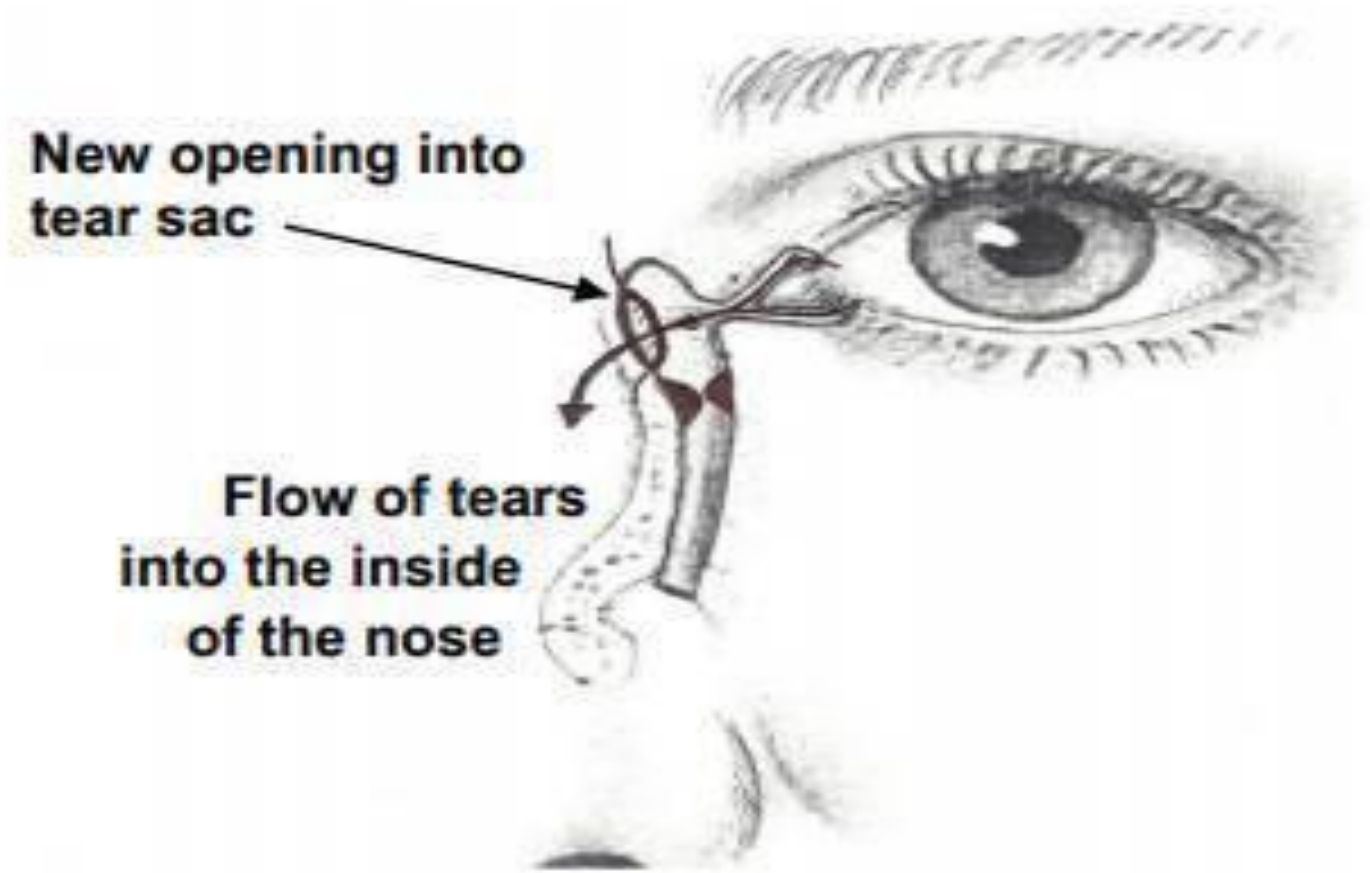
- Success rate is 90-95 %

DCR endoscopic+ Diode laser

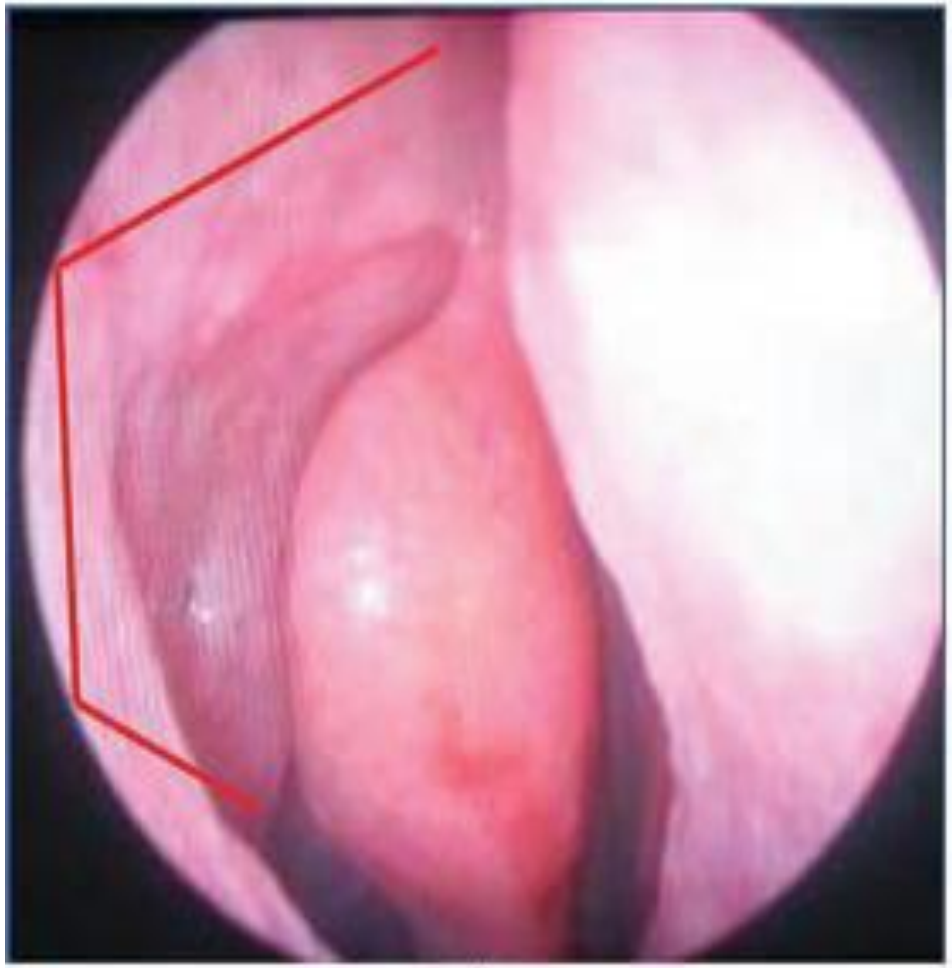
- No skin incision
- Through endoscope the small cut is given from the nasal side
- Dcr tube Badkin tube/Silicon tube is passed

- No skin incision
- Through Diode laser in probe, the cut is given to the bone
- Dcr tube Badkin tube/Silicon tube is passed
- No scar

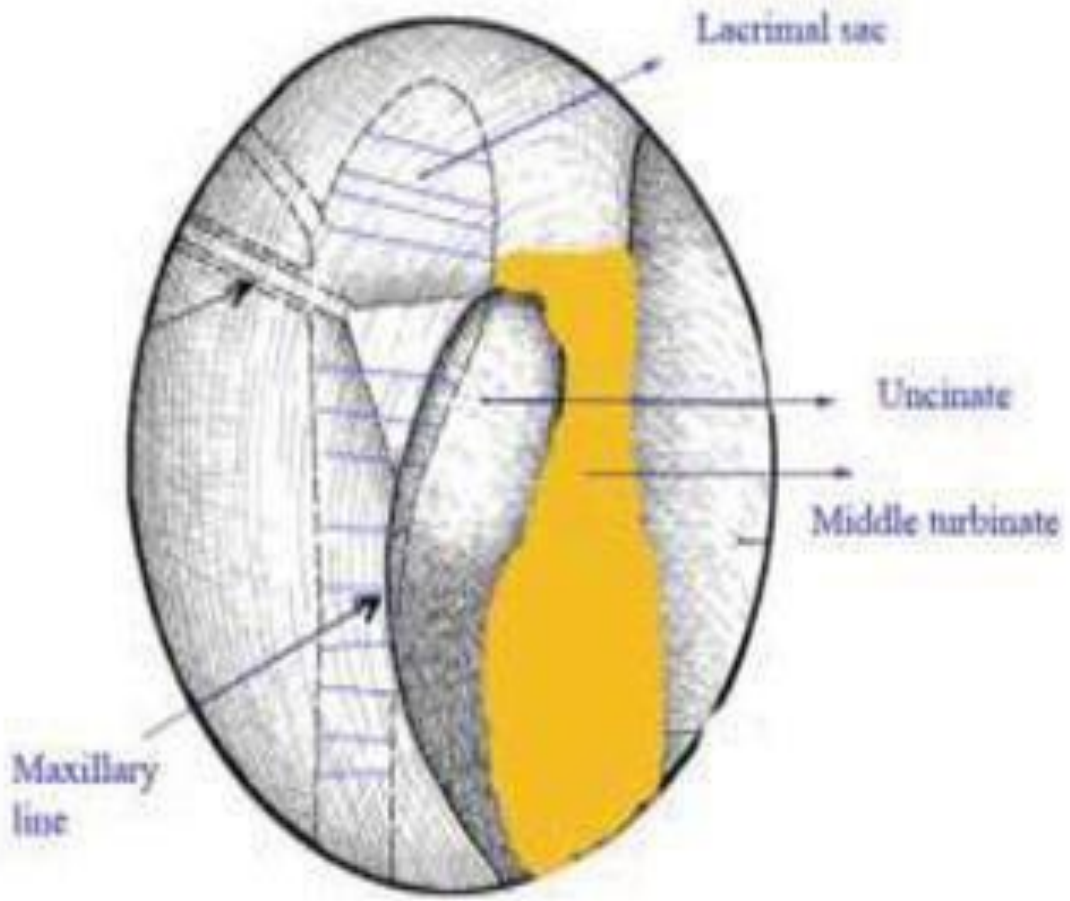
Diagram 3:


New opening into
tear sac
Flow of tears
into the inside
of the nose

Endoscopic dcr



A



B

- But the Failure rate is high

- Thanks

- DR NAZULLAH
- ASSOCIATE PROFESSOR
- OPHTHALMOLOGY
- KGMC/HMC

Objectives

- At the end of this session the 4th yr MBBS student should be able

- Enlist Different causes
- What are Clinical features
- What are Different Treatment options
- How Regurge test is performed

Learners competence level

- 4th year MBBS students of Khyber Girls Medical College

- Subject . ophthalmology

Competence level, Common competencies are.

- Knowledge.
- Skill.
- Professionalism.
- Personal grooming with leadership qualities.
- Community health.

PMDC 7 Stars Doctors, have the competencies;

- knowledge
- Skill
- Critical thinker
- Manager
- Researcher
- Lifelong learner
- Community health provider

Competencies selected

- Knowledge
- Skill

Cognitive Domain, different causes are

- Enlist the causes of watering in a small 6months baby
- Congenital NLD block
- Conjunctivitis
- Birth trauma
- Congenital glaucoma
- Foreign body
- Punctal atresia

- Watering
- Purulent discharge
- Sticky eye lids
- Swelling in the medial canthal area

Table of specifications. Cognitive Domain

CONTENTS	COGNITIVE LEVEL	TIME	MIT/TOOL	VENUE	ASSESSMENT	
ENLIST THE CAUSES OF EPIPHORA	C1	01	INTERACTVE LECTURE	LECT HALL/EYE WARD	OSPE	
DESCIBE CLINICAL FEATURE	C2	01	INTERACTVE LECTURE	LECT HALL/EYE WARD	OSPE	
WHAT ARE TREATMENT OPTIONS	C3	01	INTERACTVE LECTURE	LECT HALL/EYE WARD	OSPE	
					OSPE	

Psychomotor Skills, How regurge test performed

- Observe P1
- Assist P2
- Perform under supervision P3
- Perform independently P4

Table of Specifications. Psychomotor skills

CONTENTS	SKILL LEVEL	TIME	MIT/TOOL	VENUE	ASSESSMENT	
PERFOM REGURGE TEST	OBERVE P1	01	DIERCT PATIENT	EYE OPD	OSPE	
PERFOM REGURGE TEST	ASSIST P2	01	DIERCT PATIENT	EYE OPD	OSPE	
PERFOM REGURGE TEST	UNDER SUPERVISION P3	01	DIERCT PATIENT	EYE OPD	OSPE	
PERFOM REGURGE TEST	PERFORM INDEPENDENTLY P4	01	DIERCT PATIENT	EYE OPD	OSPE	

Eye Injury

Muhammad Tariq Khan

Eye Injury

- An insult to the eye
  - Physical
  - Chemical
- Can affect or impair vision
- Can result to blindness or potential blindness

Eye Injury – Extent

- Can range from minor bruises to lacerations
- Chemical burns
- Globe injuries may be associated with intra-ocular foreign bodies
- Globe injuries may be associated with fractures

Ocular Injury

- Non - trauma
- Trauma
  - Blunt trauma
  - Penetrating trauma

- Etiological Classification of Trauma : 1. Accidental trauma.
  2. Self inflicted trauma.
  3. Occupational trauma.
- Classification on the basis of Nature:
  - 1. Physical trauma
    - a. Perforating
    - b. Non perforating
  - 2. Chemical trauma
    - a. Acid
    - b. Alkali
    - c. Dye (Salt of acid or alkali)

Mechanical Trauma

- Closed Globe
  - Burn
    - Chemical Burn
    - Radiation/Thermal Burn.
  - Laceration
- Open Globe
  - Rupture
  - Laceration
    - Penetrating
    - Perforating





Ocular Injuries

- **Closed globe injury:** No full-thickness wound of eye wall,but there is intr-ocular damage.
- **Open globe injury:** It refers to the full thickness injury of the eye wall and the intra-ocular structures.
- **Contusion:** It is a result of direct energy delivary to the eye by a blunt object.injury may be at the site of impact or at a distant site.
- **Lamellar laceration:** Partial-thickness wound of the eyewall. • Laceration Full-thickness wound of the eyewall, caused by a sharp object.
- **Penetrating injury:** is an injury where a foriegn object has been embedded in the eye. It is usually a full thickness wound & it has a site of Entrance. Perforating injury has both an Entrance and exit wounds.

Ocular Injuries

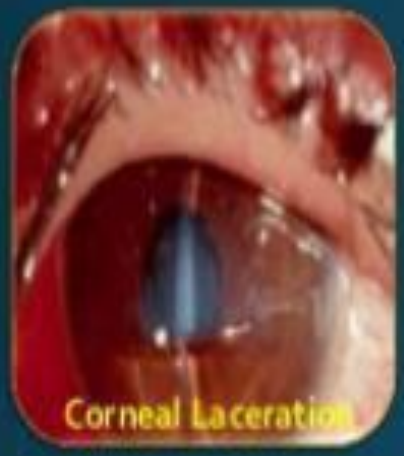
PENETRATING INJURY : Usually by a sharp and pointed instruments like needles,sticks,pencils,knives arows,pens,glass and any object with sharp edges.

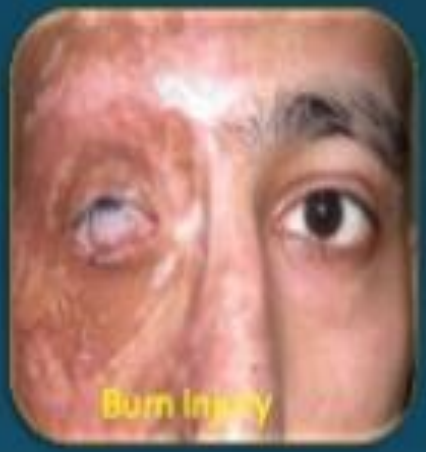
- The most common causes of penetrating ocular injuries are due to trauma caused by wood, metal and stone .Most of the injuries occurred during chopping or cutting wood, hammering metals or nails and carving stone.
- These are associated with professions such as farming, garage work and carpentry in adults.
- Children, on the other hand, mostly sustain accidental injuries by rubber bands, needles, pencils, sticks while playing with others.

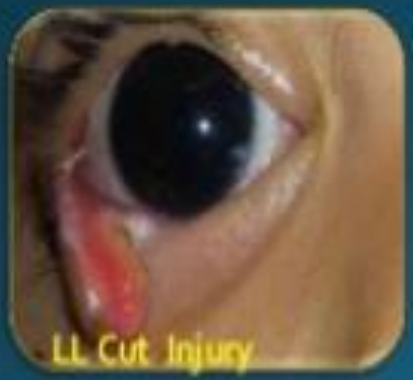
EFFECTS OF PENETRATING OCULAR INJURIES

Laceration of the conjunctiva,corneal lacerations,Vitreous haemorrage,rupture of globe,retinal tears and detachments,scarring which leads to cataract and glaucoma & Intra ocular foriegn bodies, iridocyclitis or Endophthalmitis .

Common Ocular Injuries

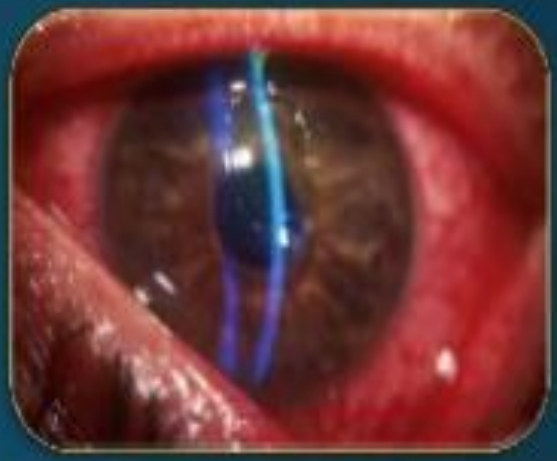






Corneal Abrasion

H/O: Blurring of vision ,
redness, Pain , photophobia,
FB sensation, watering,
swelling eyelid.

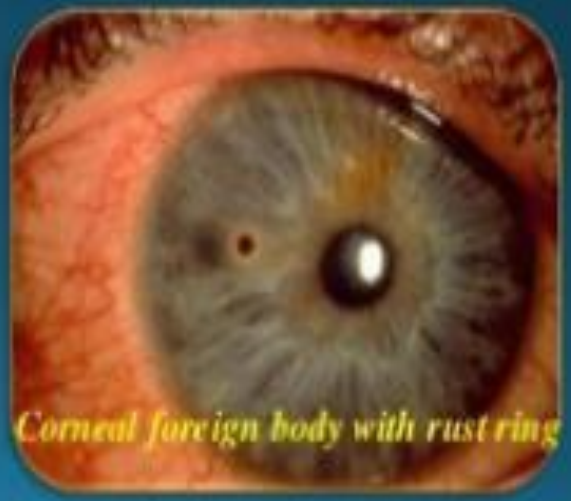


Epithelial staining defect with fluorescein

Rx:
Erythomycin ,Ciprocin,Contact Lens,Patching.

Corneal Foreign Bodies

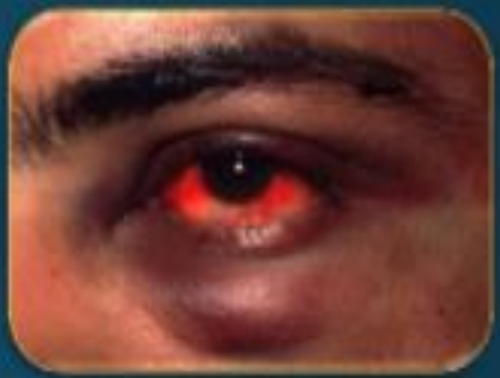
A corneal foreign body is an object (eg, metal, glass, wood, plastic, sand) either superficially adherent to or embedded in the cornea of the eye.

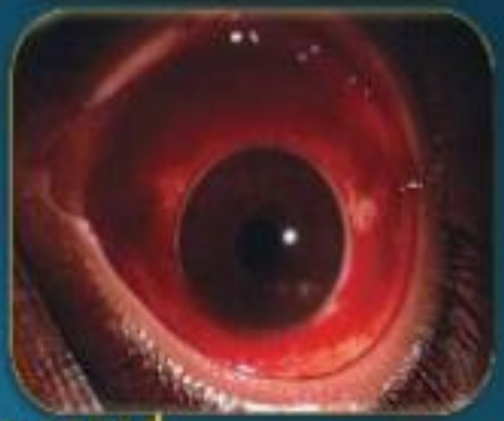


Rx:
Removal FB, CT-Scan Orbit.

Subconjunctival Hemorrhage

Subconjunctival bleeding, also known as subconjunctival hemorrhage, is bleeding underneath the conjunctiva. The conjunctiva contains many small, fragile blood vessels that are easily ruptured or broken. When this happens, blood leaks into the space between the conjunctiva and sclera.





H/O : Redness, Discomfort or Burning sensation.

Blackish shadow around side the eye.

Rx:

The elective use of aspirin and NSAIDs is typically discouraged.
Artificial tears may be applied four to six times a day.

Traumatic Hyphaema

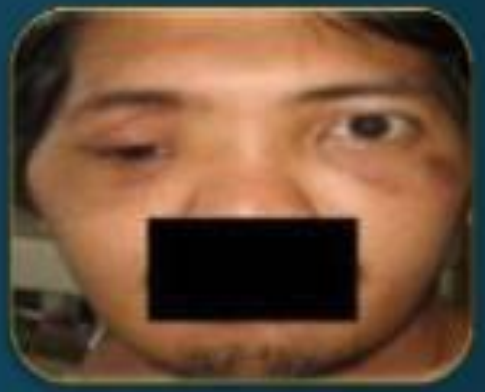
- Blood in anterior chamber,Hyphema can occur after blunt or lacerating trauma, after intraocular surgery,
- Loss of vision,Severe Pain,Redness,Photophobia.

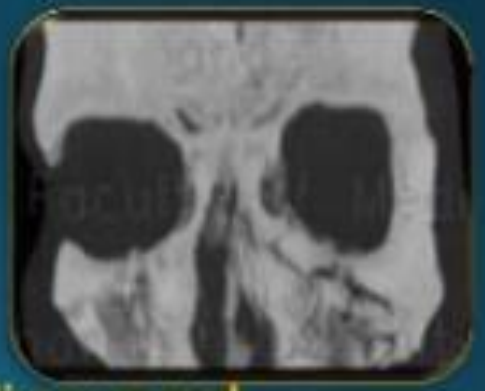
Rx:

- Topical cycloplegics (e.g. cyclopentalate 2% tid,) will dilate the pupil and prevent synechiae to the lens.
- Topical steroids (e.g. prednisolone acetate 0.125-1% qid) are used to decrease inflammation.

Orbital Fractures

Orbital fractures are breaks of the facial bones surrounding the eye. An orbital blowout fracture is a break in the thin bone that forms the floor of the orbit and supports the eye (orbital floor fracture).





ORBITAL FRACTURES Types :

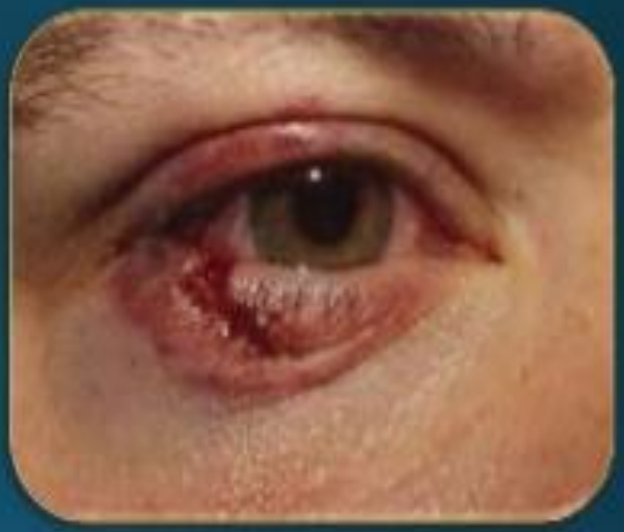
- Blow-out orbital floor fracture
- Blow-out medial wall fracture
- Roof fracture
- Lateral wall fracture

Rx:

- The elective use of aspirin and NSAIDs is typically discouraged. Artificial tears may be applied four to six times a day.
- Reconstruction of Orbital fractures .

Lid Lacerations

- **Causes:** Sharp or blunt trauma.
- **H/O :** Full thickness cut injury BUL / LUL medial canthus side.
  Redness, Watering, pain, Bleeding
- Vision may dimness.

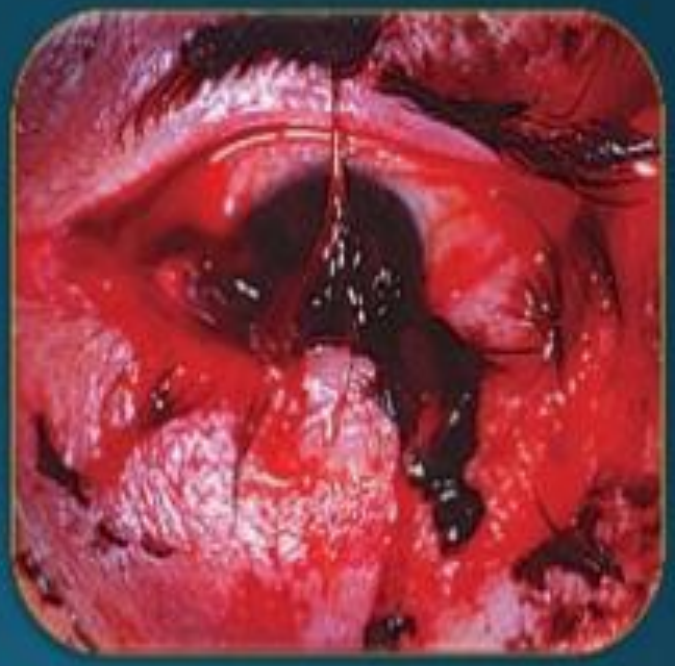


Rx:

- Eyelid Laceration Repair
- Topical antibiotics & Artificial tears .

Penetrating / Ruptured Globe

- Corneal or scleral lacerations.
- Hypopyon (not always present).
- Severe chemosis & hemorrhage.
- Intraocular FB may present.
- Limitation of extraocular motility.
- Irregular pupil.



Rx:

- Repair globe injury / Laceration
- Evisceration / Enucleation

Chemical Burns

- **Alkali-Based Chemical.**

Lime, Cement, Whitewash, Metal Polishes, Ammonia.

- **Acid-Based Chemical:**

Cleaning Solutions , Battery Acid (H_2SO_4, HCL), Acetic Acid.

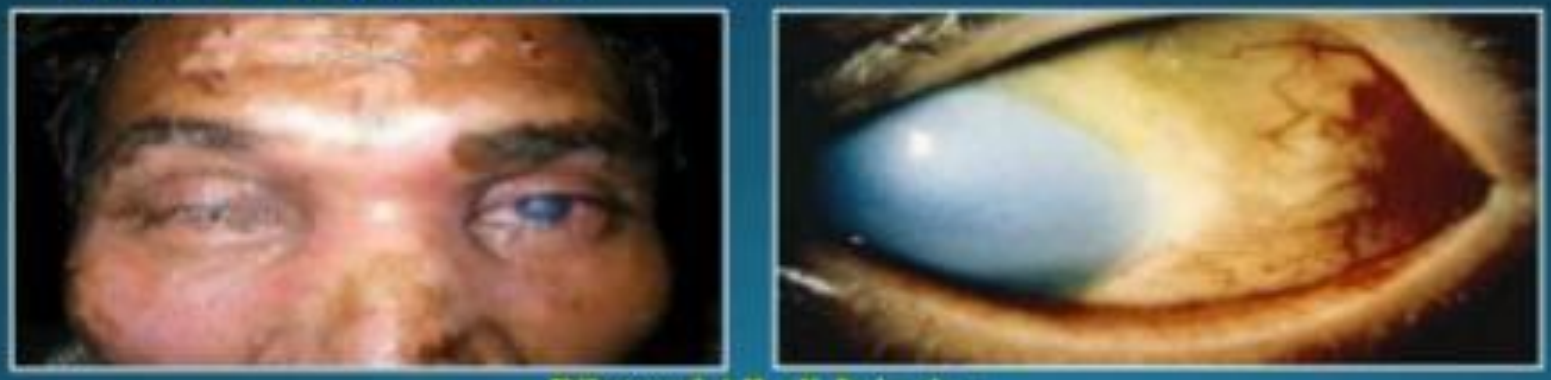
Severity: The severity of the chemical burn depends on Concentration of the chemical substance, affected area, & duration of exposure.

N.B : Alcali burn are more damaging than Acid burn.

Chemical Burns

Both acid and alkali burns can be blinding

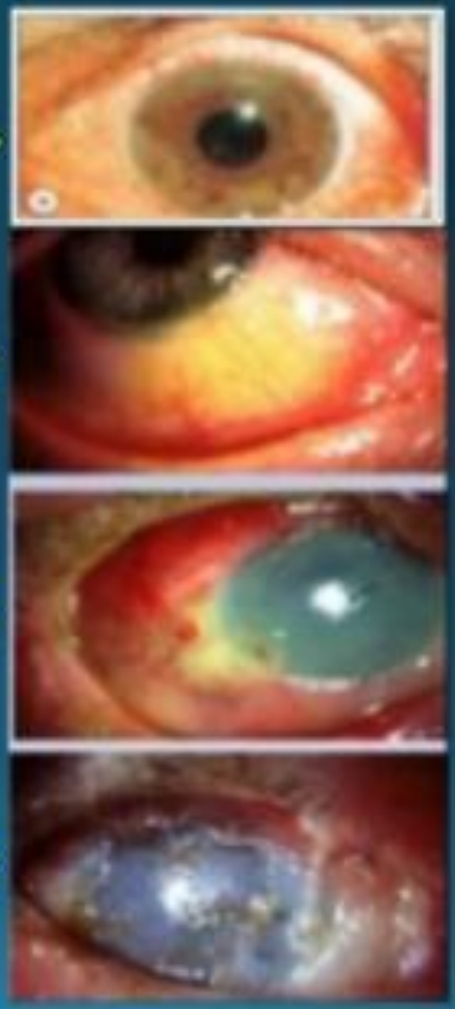
- ***- Acid burns tend to coagulate proteins, necrosis of conjunctiva, epithelial defect & limiting the depth of penetration.***
- ***- Alkali burns can rapidly penetrate the cornea , stromal necrosis & thinning , raised IOP, and Causing damage to intraocular structures.***



Bilateral Alkali Injuries

GRADING OF SEVERITY OF CHEMICAL INJURIES

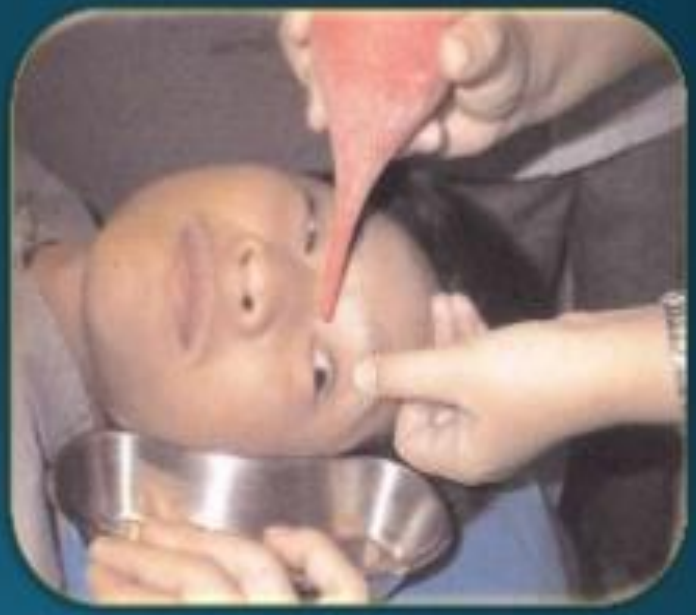
- Grade I (excellent prognosis) •Clear cornea •Limbal ischaemia - nil
- Grade II (good prognosis) •Cornea hazy but visible iris details •Limbal ischaemia <1/3
- Grade III (guarded prognosis) •Hazy cornea with no iris details •Limbal ischaemia 1/3 to 1/2
- Grade IV (very poor prognosis) •Opaque cornea •Limbal ischaemia >1/2.



Chemical Burns & Our Activitis

Immediate copious irrigation with a minimum of 1-2 L of saline or until pH is normalized (7.3-7.7)

- Instill a topical anesthetic.
- Use eyelid retractor.
- Double eversion of the eyelids.



Rx Of Chemical Injuries

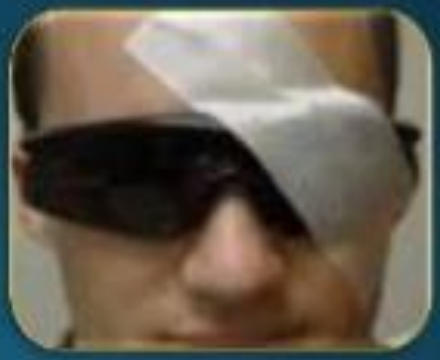
- 1.Copious irrigation (15-30 min) – to restore normal pH
- 2.Topical steroids (first 7-10 days) – to reduce inflammation
- 3.Topical and systemic ascorbic acid – to enhance collagen production
- 4.Topical citric acid – to inhibit neutrophil activity
- 5.Topical and systemic tetracycline – to inhibit collagenase and neutrophil activity
- 6.Cycloplegia – to improve comfort

Acute Eye Conditions

Emergency	Very Urgent	Urgent
(Immediately)	(Within a few hours)	(Within one day)
Retinal arterial occlusion	Perforation	Orbital cellulitis
Chemical burns	Ruptured Globe	Orbital injury
	Acute glaucoma	Corneal ulcer
	Sudden congestion proptosis	Corneal abrasion
		Hyphema
		Intraocular FB

Primary Rx for Ocular Injuries

- Every eye injury should be given medical attention; do not touch, rub or try to remove any object in the eye. If the eye has been cut or there is an object in the eye, rest a protective shield – such as a paper cup – on the bone around your eye
- In minor cases of trauma, such as a black eye from a sports injury, applying cold to the affected area can help bring swelling down, and allow the affected area to heal faster.
- In general, if a person is not sure if they have a serious eye injury, they should call an ophthalmologist or see an emergency-medicine doctor, preferably at a large hospital that has an ophthalmologist on call, for advice and/or treatment.



Take Home Messages

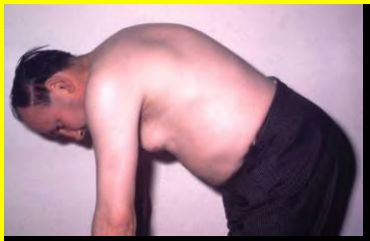
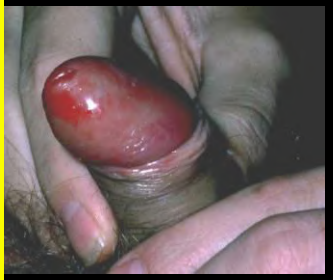
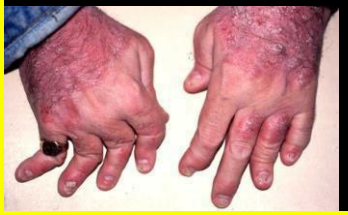
- Wear protective eyewear during risky activities.
- Wear goggles when exposed to chemicals. Supervise your child's use of tools.
- Protect your eyes while doing yardwork.
- Keep children away from flying debris.
- Use caution with chemicals and cleaners.
- Be careful when cooking or using hot objects.
- Keep sharp kitchen tools and utensils away from small children.
- Use car seats. Avoid certain children's toys.
- Wear protective eyewear during sports.
- Keep small children safe around dogs.

Thank You

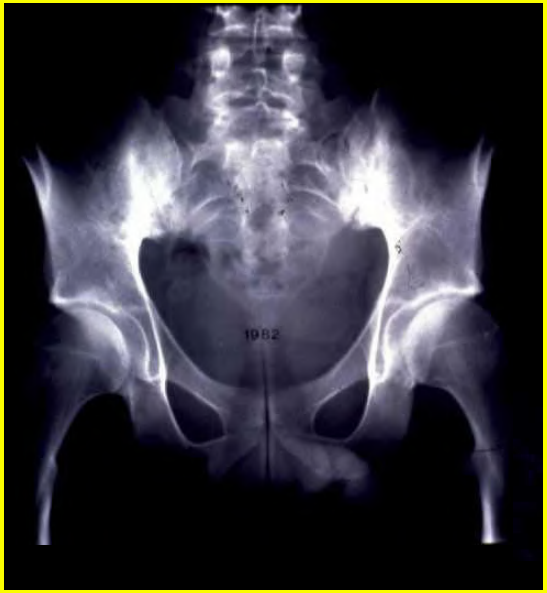
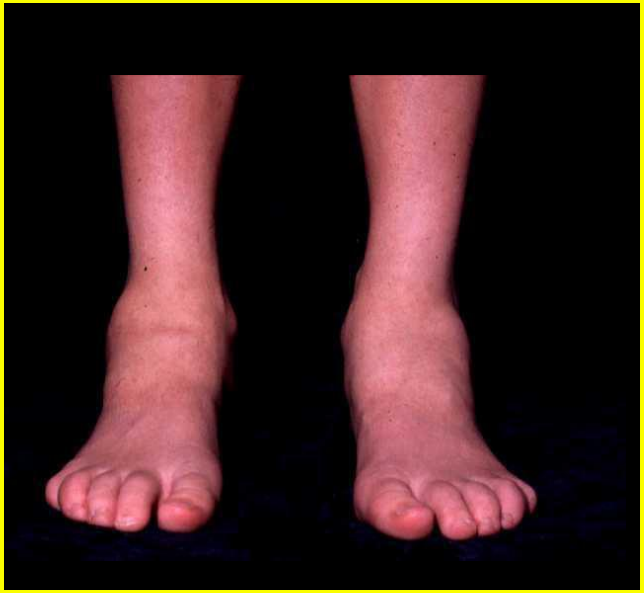
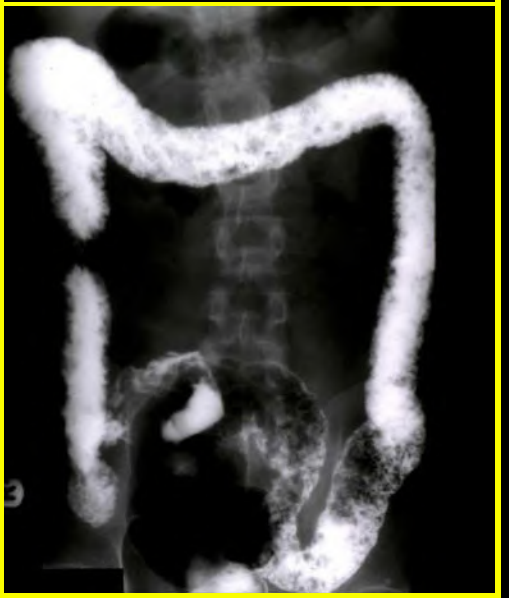
IMPORTANT SYSTEMIC ASSOCIATIONS OF UVEITIS

1. **Spondylarthropathies**
2. **Juvenile idiopathic arthritis**
3. **Sarcoidosis**
   - Systemic features
   - Ocular features
4. **Behçet disease**
   - Systemic features
   - Ocular features
5. **Vogt-Koyanagi-Harada syndrome**
6. **Inflammatory bowel disease**
   - Ulcerative colitis
   - Crohn disease
7. **Tubulointerstitial nephritis and uveitis**

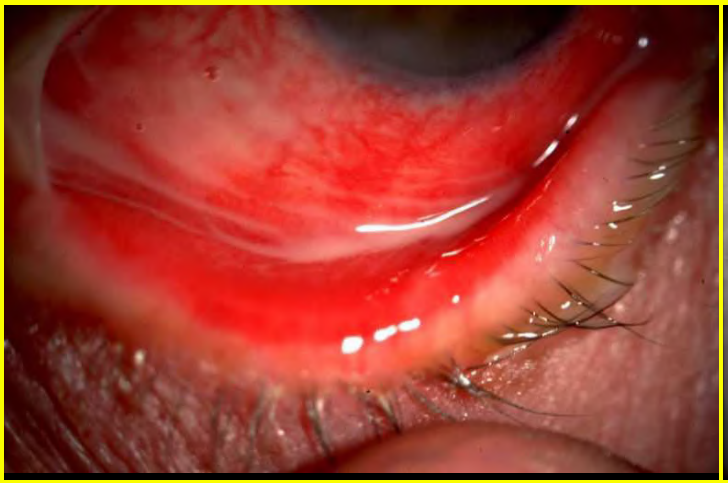
Spondylarthropathies

	Gender	HLA-B27	Acute iritis
 Ankylosing spondylitis	70% males	95%	30%
 Reiter syndrome	90% males	60%	20%
 Psoriatic arthritis	equal	30%	10%

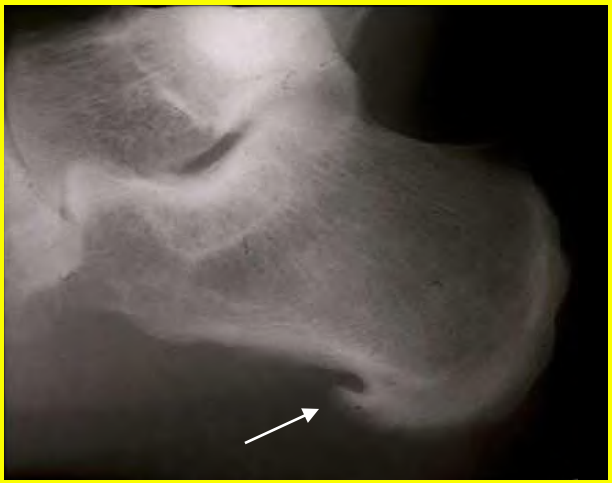
Spondylarthropathies

	Sacroiliitis	Peripheral arthritis	Bowel inflammation
			
Ankylosing spondylitis	100%	20%	Common
Reiter syndrome	60%	100%	Uncommon
Psoriatic arthritis	30%	100%	Occasional

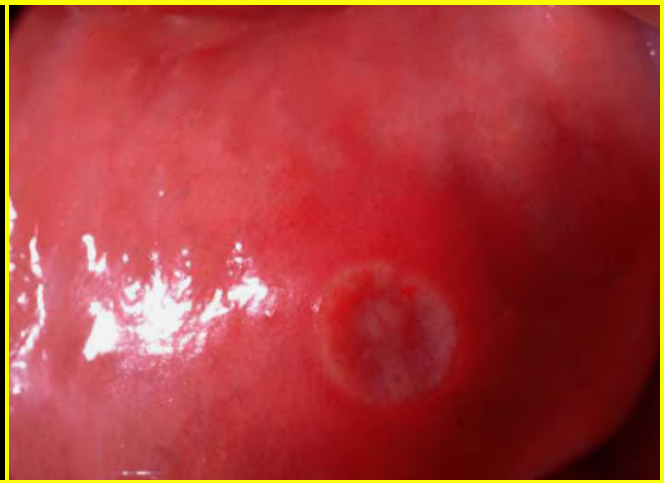
Clinical features of Reiter syndrome



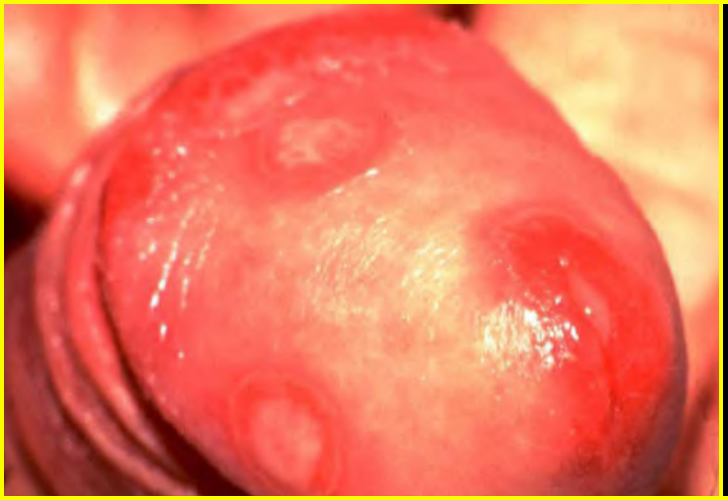
Conjunctivitis



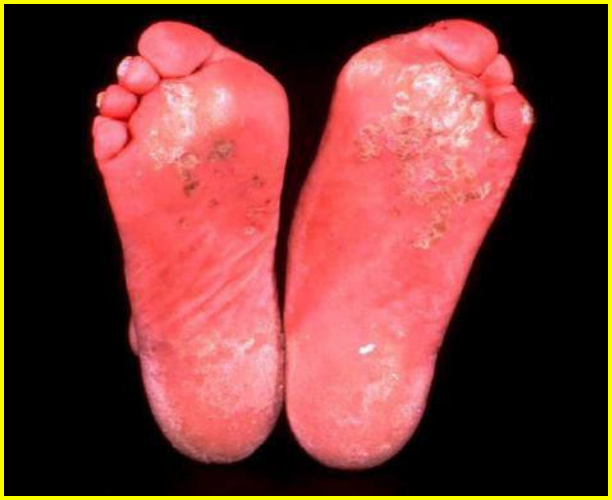
Plantar fasciitis



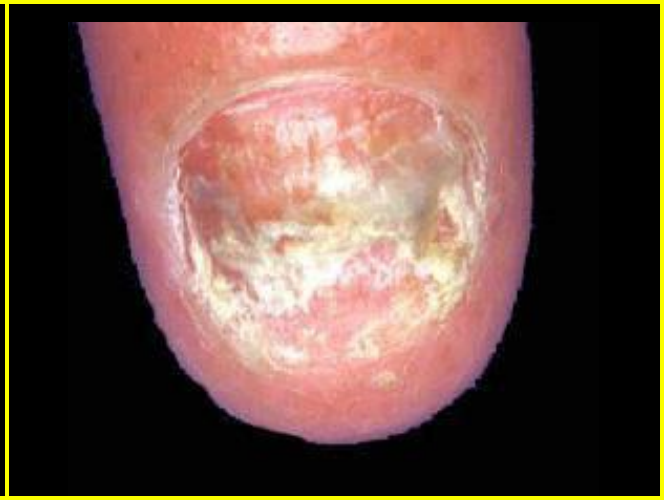
Painless oral ulceration



Urethritis and circinate balanitis

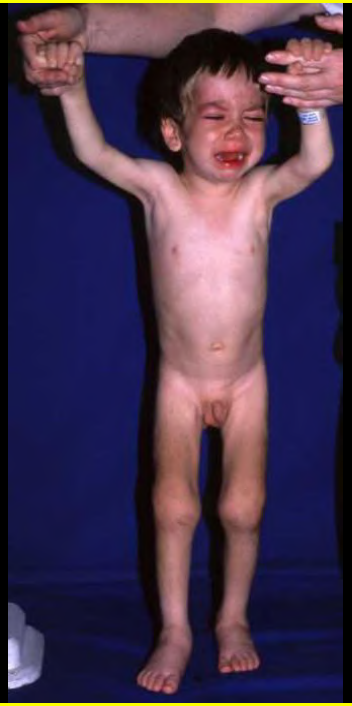
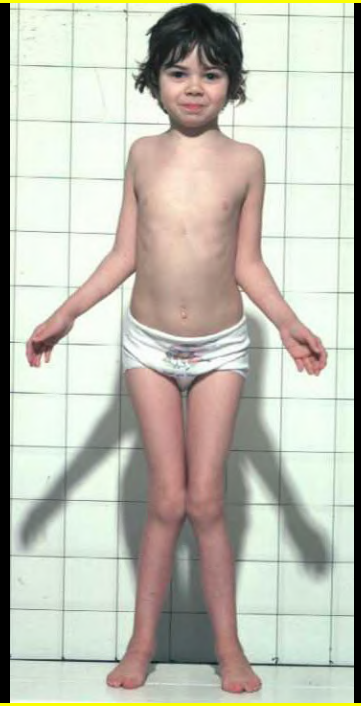
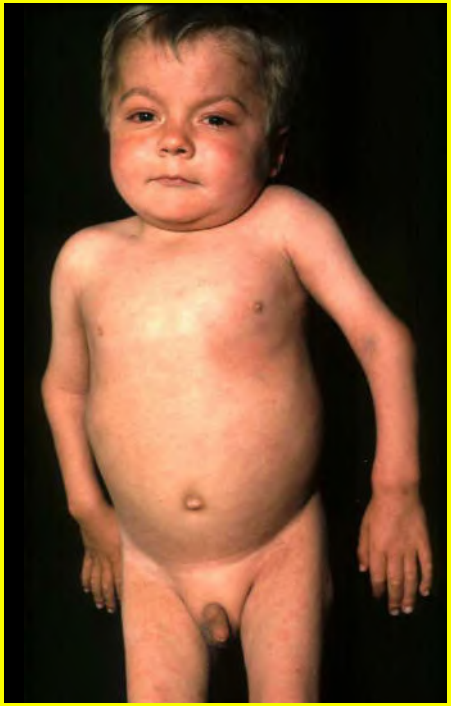


Keratoderma blenorrhagica

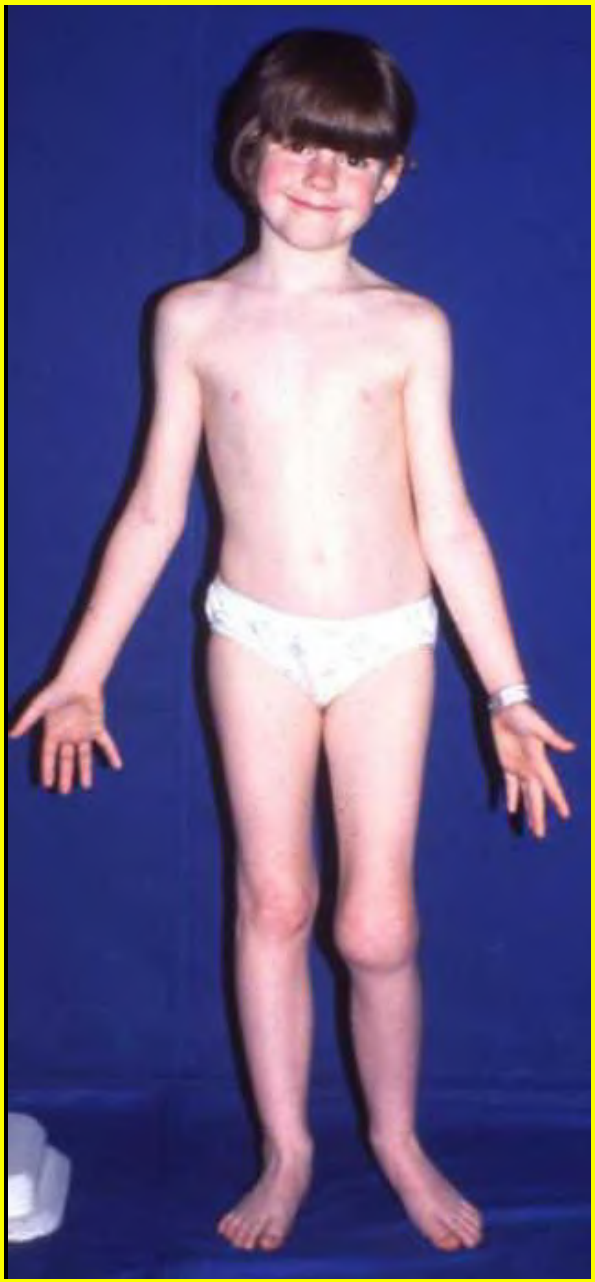


Nail dystrophy

Juvenile idiopathic arthritis

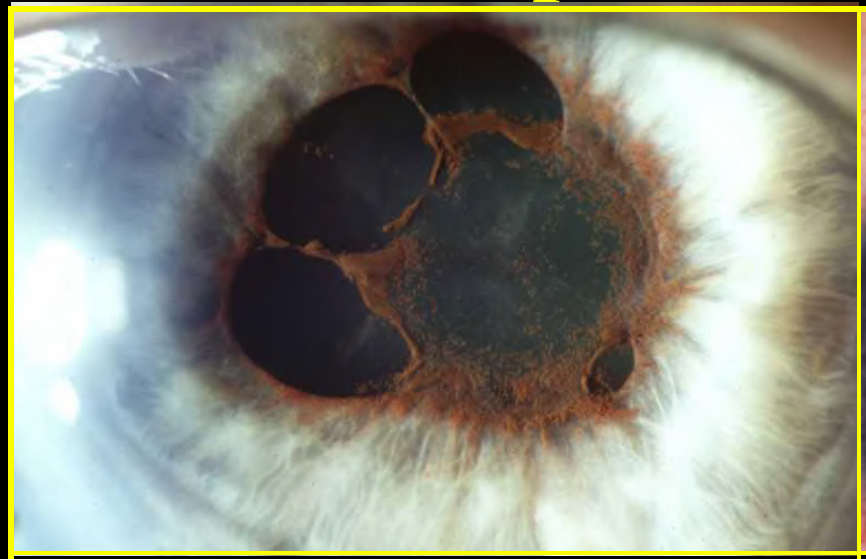
	Pauciarticular (60%)	Polyarticular (20%)	Systemic (20%)
			
Joints no.	< 5	> 4	Variable
Onset	< 6 years	Variable	Variable
Systemic features	Absent	Mild or absent	Severe
Positive ANA	75%	40%	10%
Iridocyclitis	20%	5%	Absent

High risk factors for uveitis in JIA

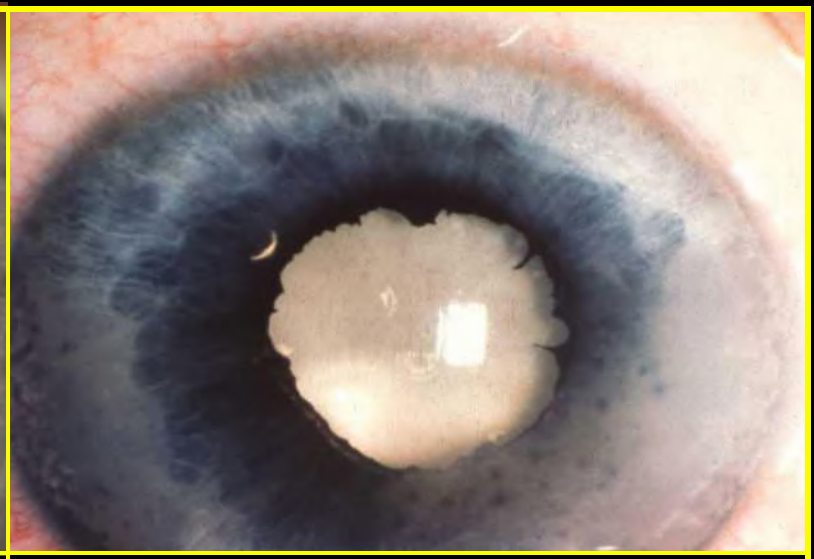


- Girls
- Early onset
- Pauciarticular onset
- ANA
- HLA-DR5

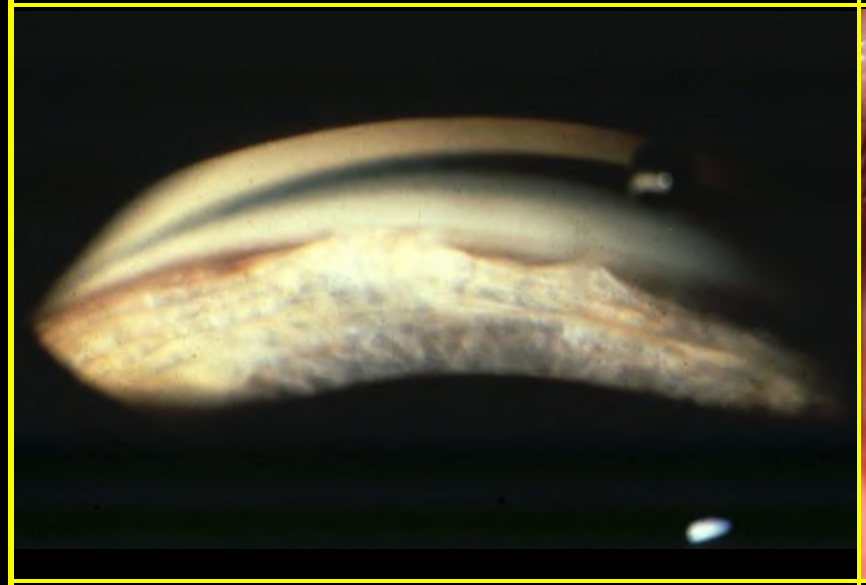
Complications of uveitis



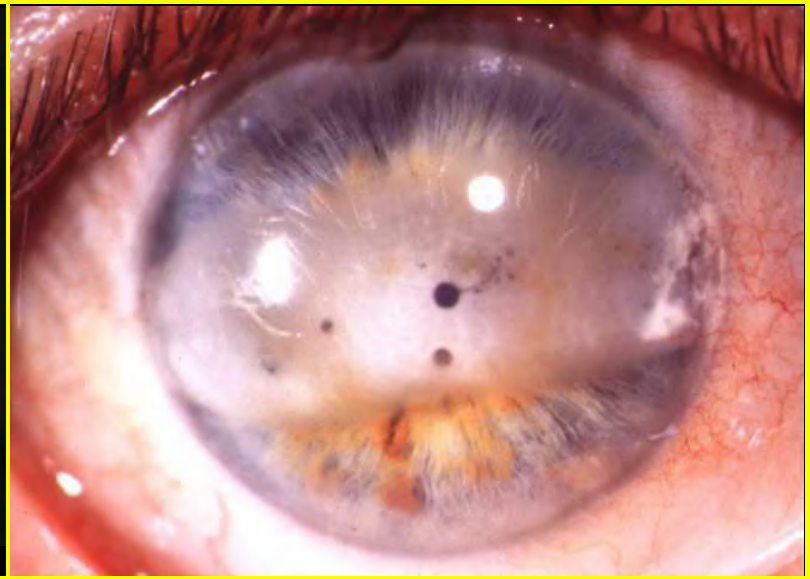
Posterior synechiae - 30%



Cataract -20%



Glaucoma due to PAS - 15%

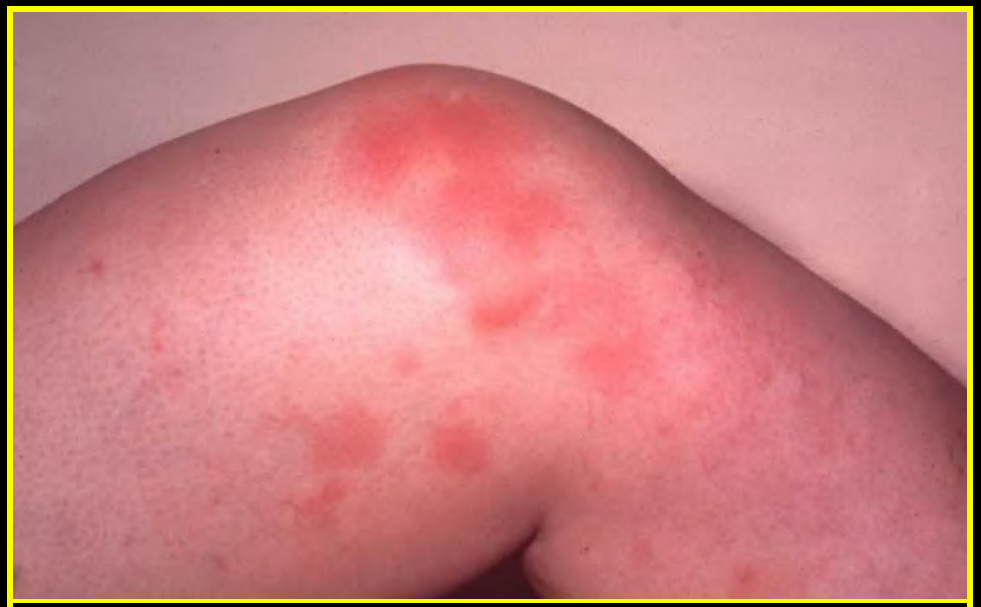


Band keratopathy - 10%

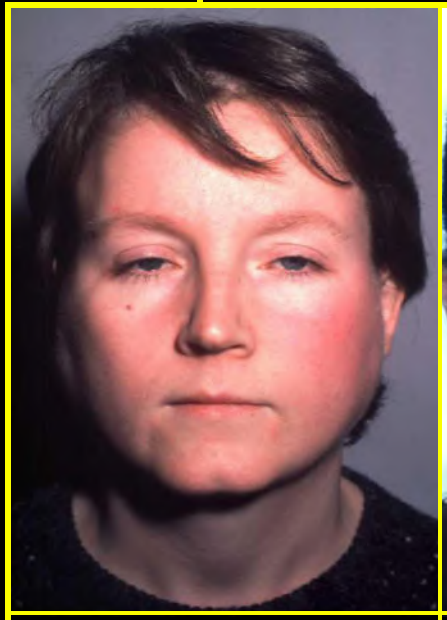
Systemic Features of Sarcoidosis

1. Idiopathic, multisystem non-caseating granuloma
2. More common in blacks than whites
3. Presentation
   - Acute - third decade
   - Insidious - fifth decade
4. Organ involvement
   - Lungs - 95%
   - Thoracic lymph nodes - 50%
   - Skin - 30%
   - Eyes - 30%

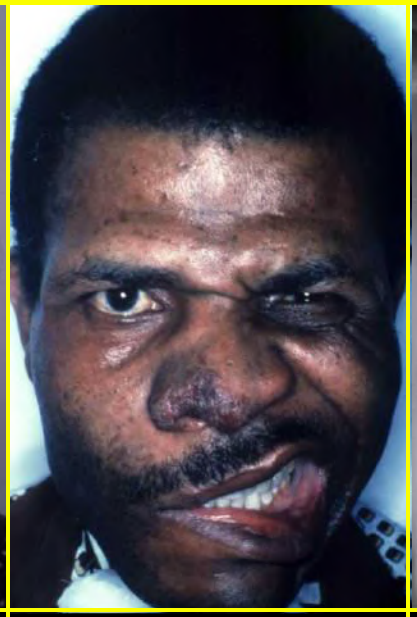
Acute sarcoidosis



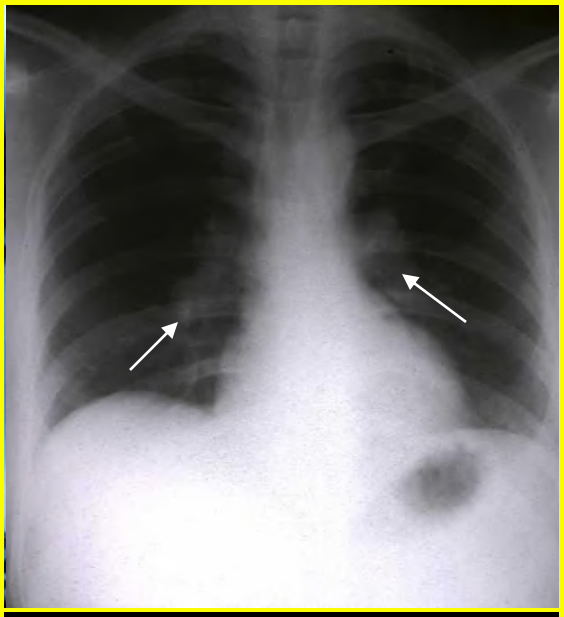
Erythema nodosum



Parotid enlargement

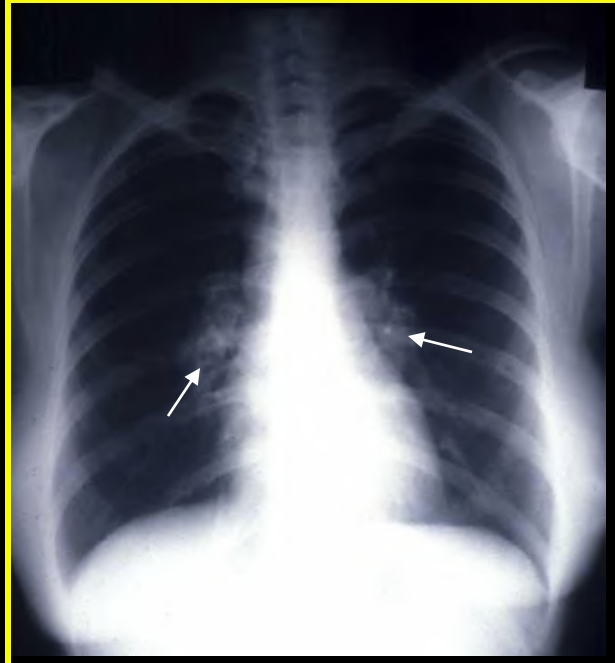


Facial palsy

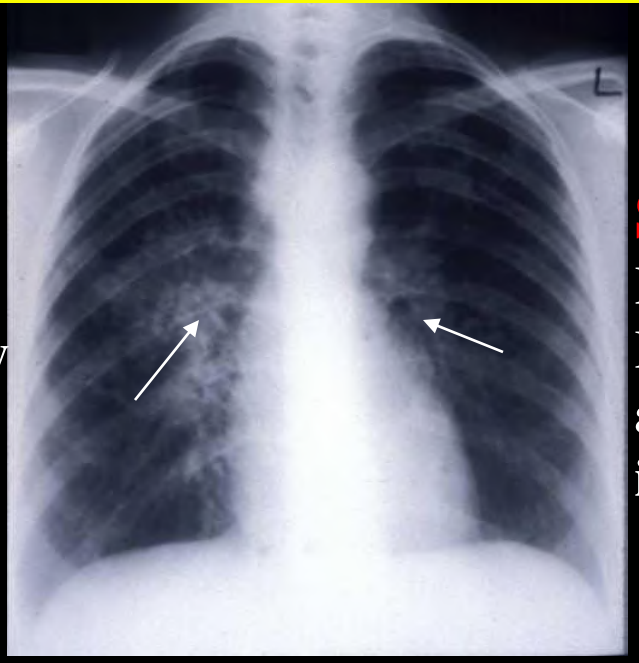


Hilar lymphadenopathy

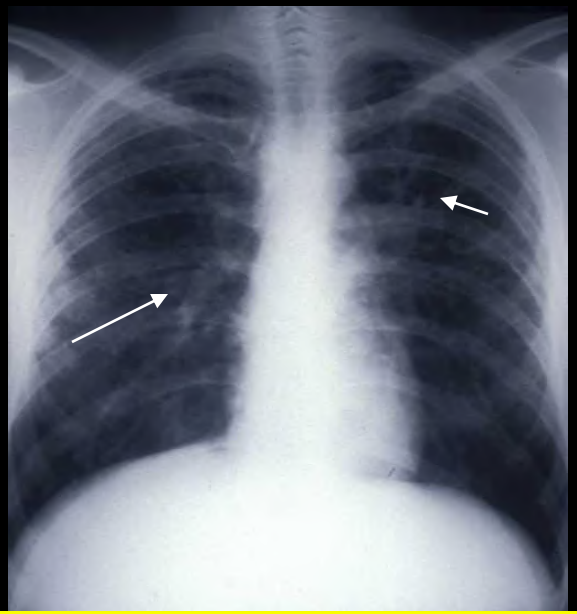
Classification of sarcoid lung lesions



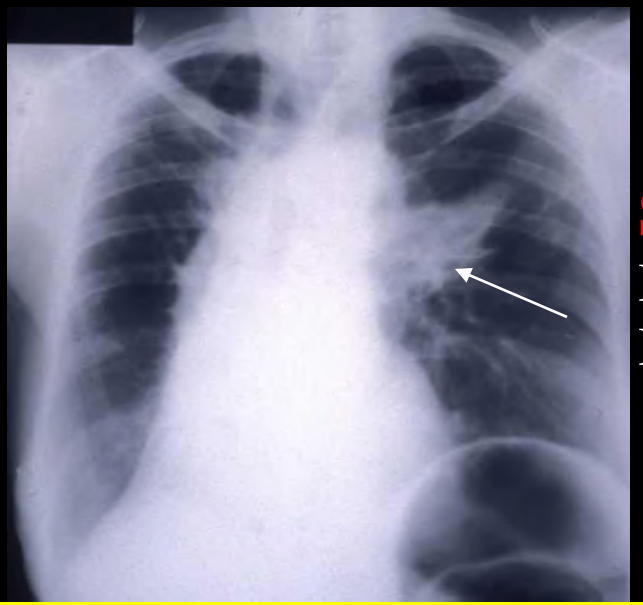
Stage 1
Hilar
Lymphadenopathy



Stage 2
Hilar
Lymphadenopathy
and parenchymal
infiltrates

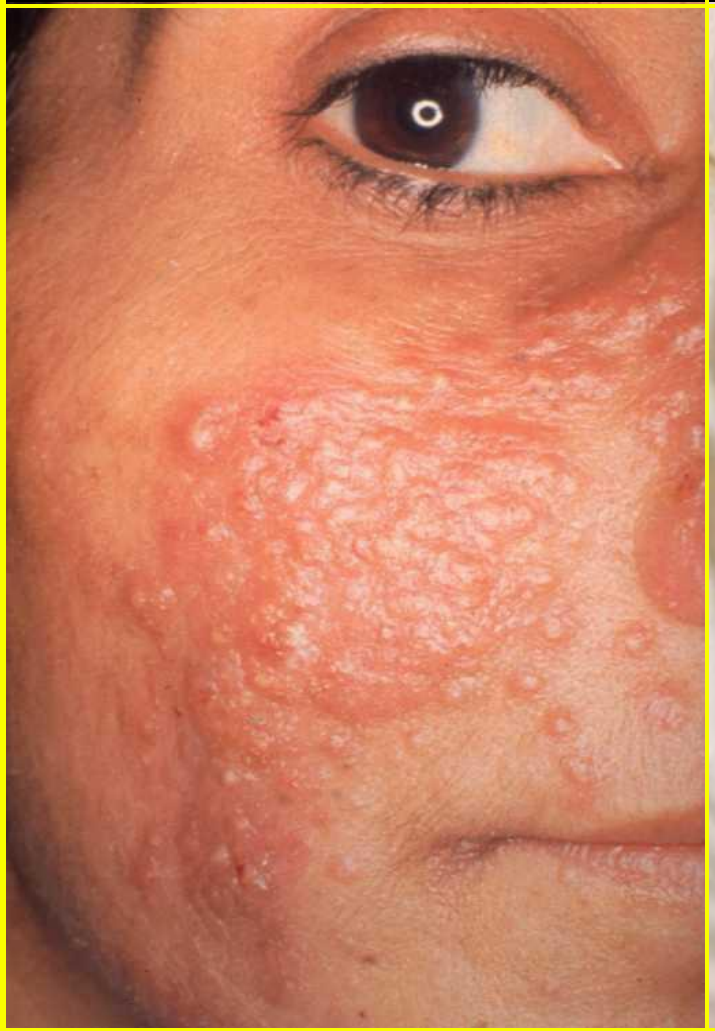
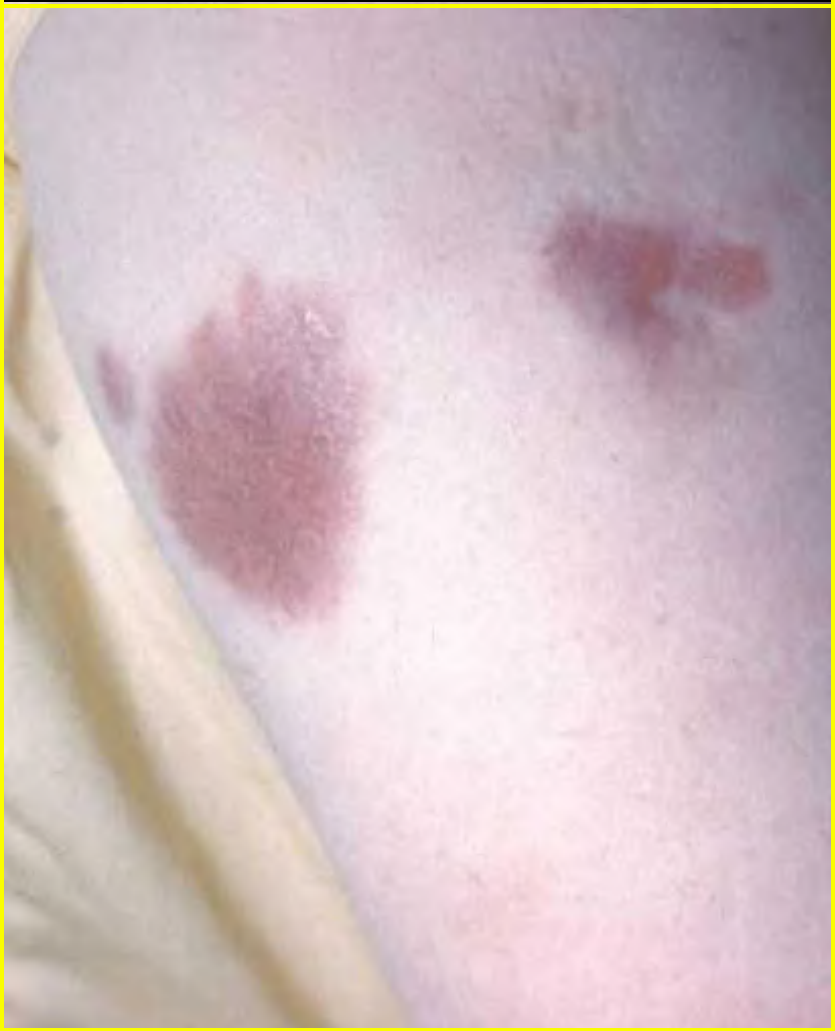


Stage 3
Parenchymal
Infiltrates alone

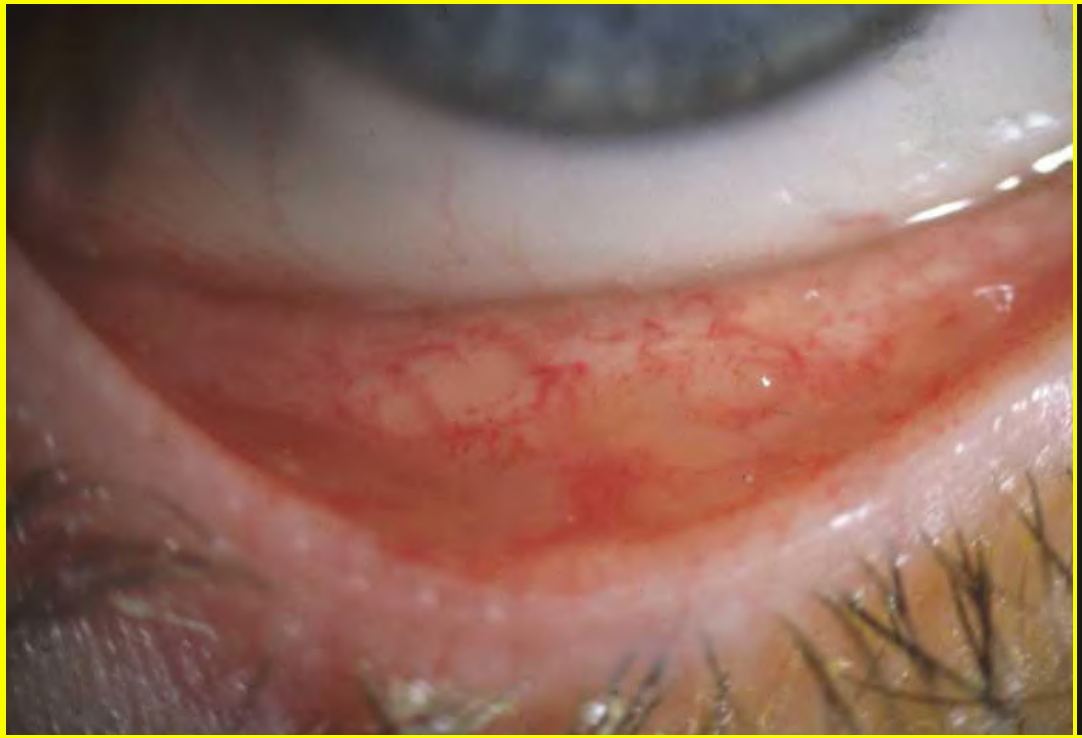


Stage 4
Fibrosis and
Bronchiectasis

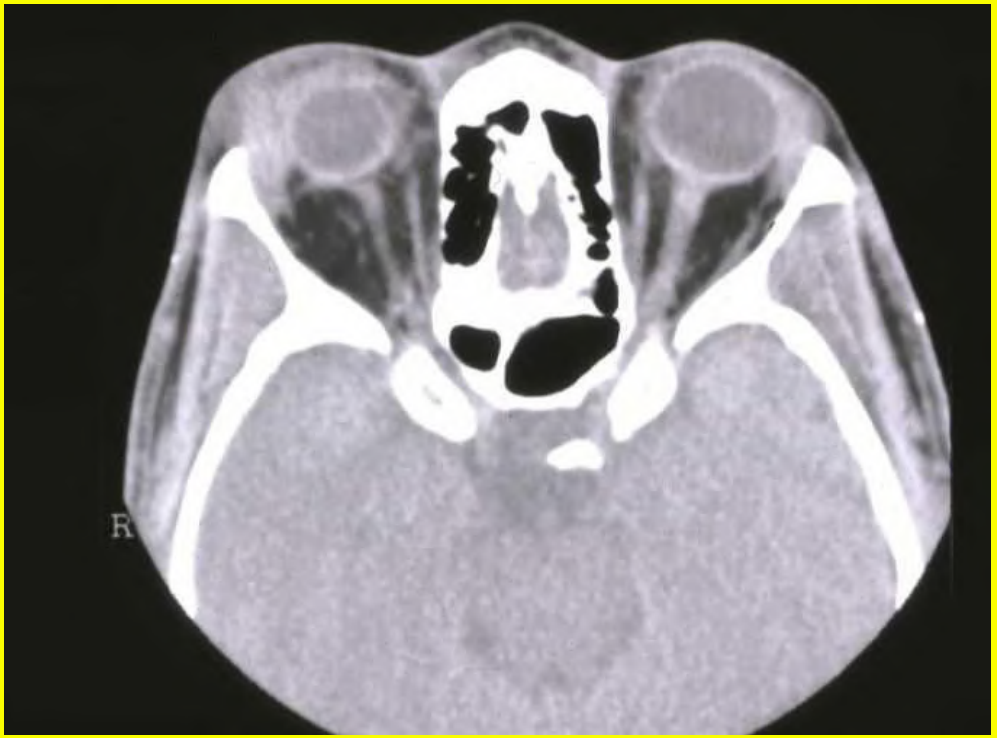
Sarcoid skin lesions

Granulomata	Lupus pernio
	
On face, buttocks and extremities	Indurated, purple-blue lesions

Anterior segment lesions in sarcoidosis

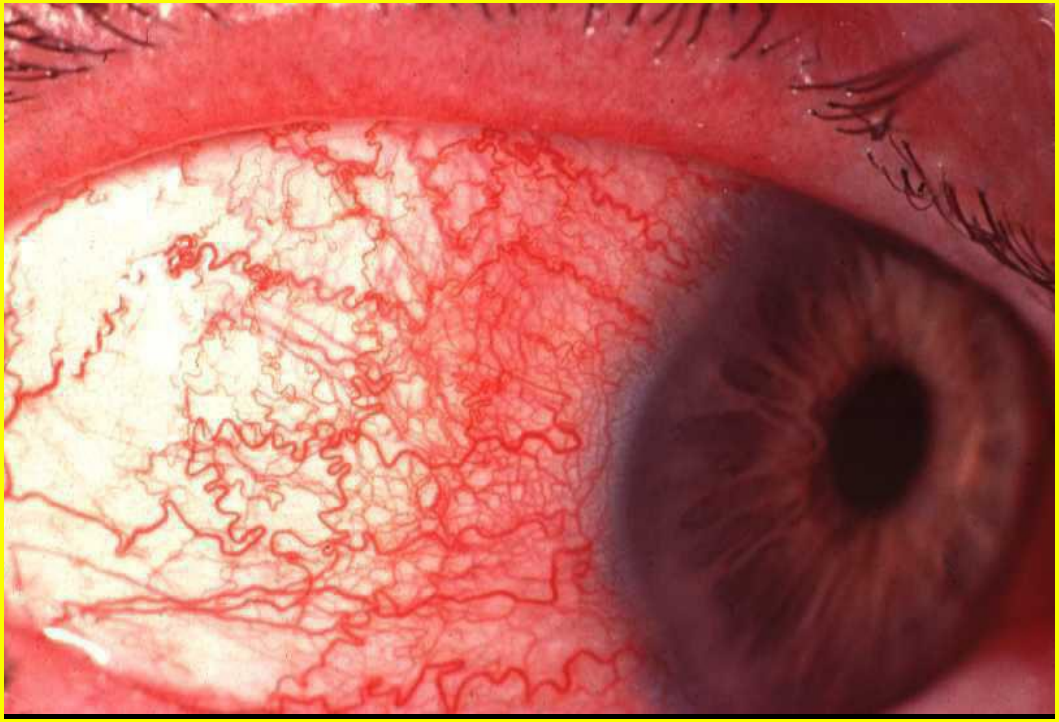
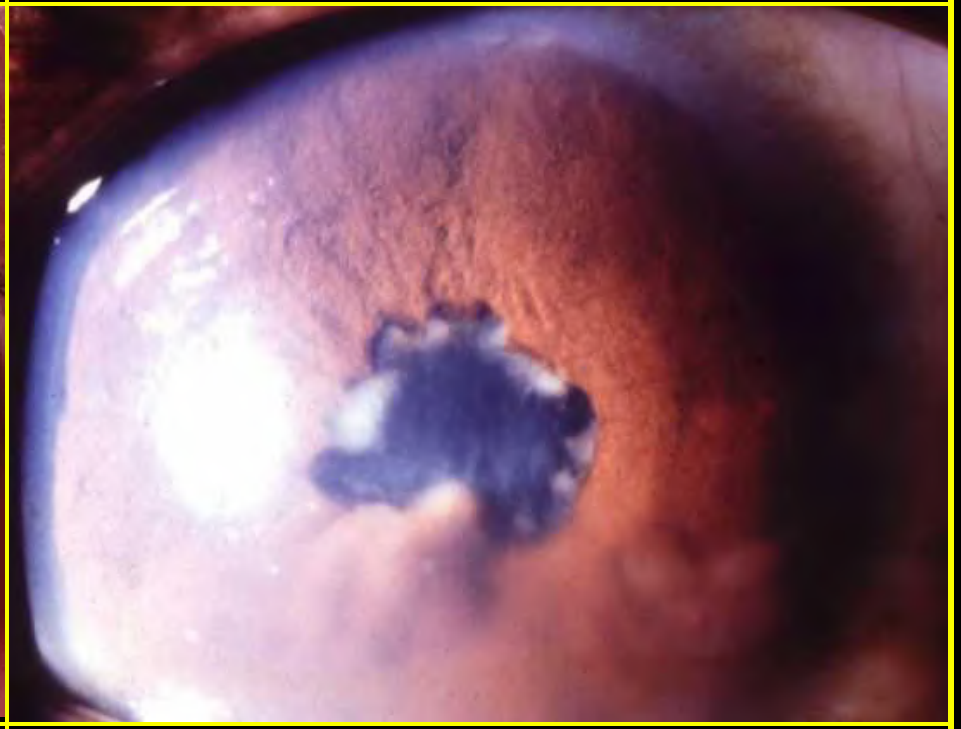


Conjunctival granuloma

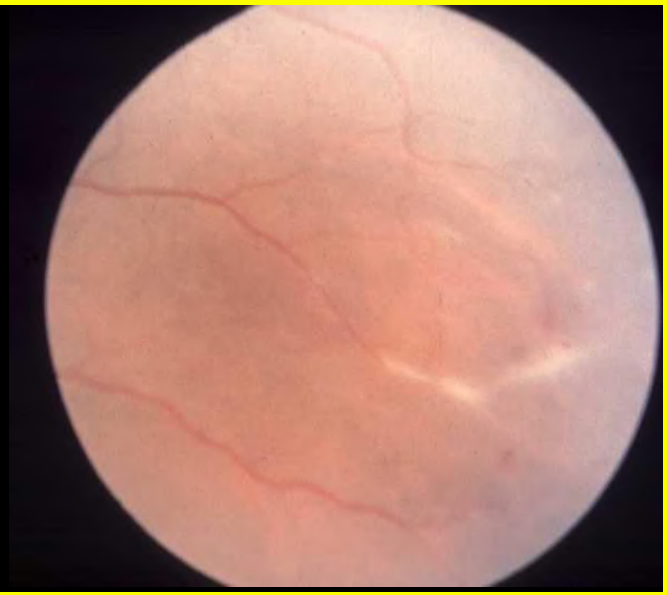


Lacrimal gland involvement and dry eyes

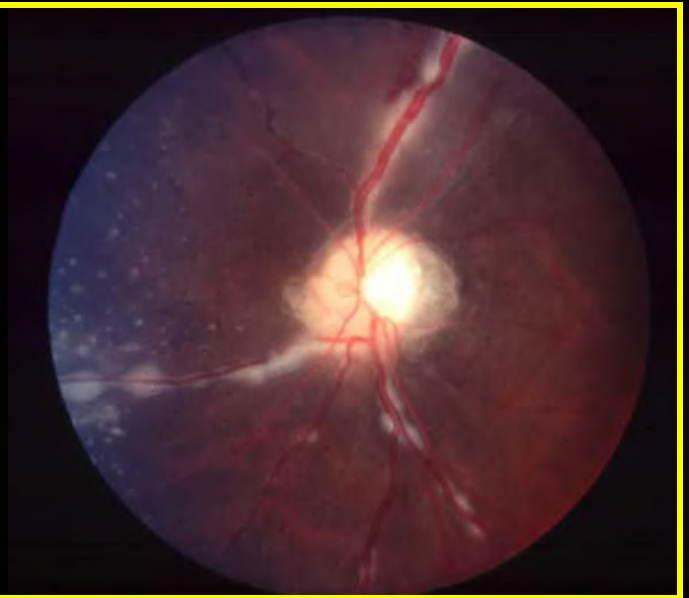
Iridocyclitis in sarcoidosis

Acute non-granulomatous	Chronic granulomatous
	
In young patients with acute sarcoid	In older patients with chronic sarcoid

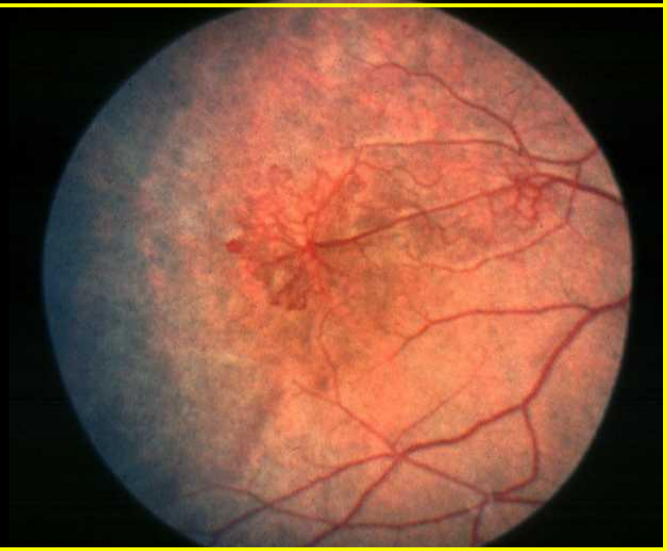
Posterior segment lesions in sarcoidosis



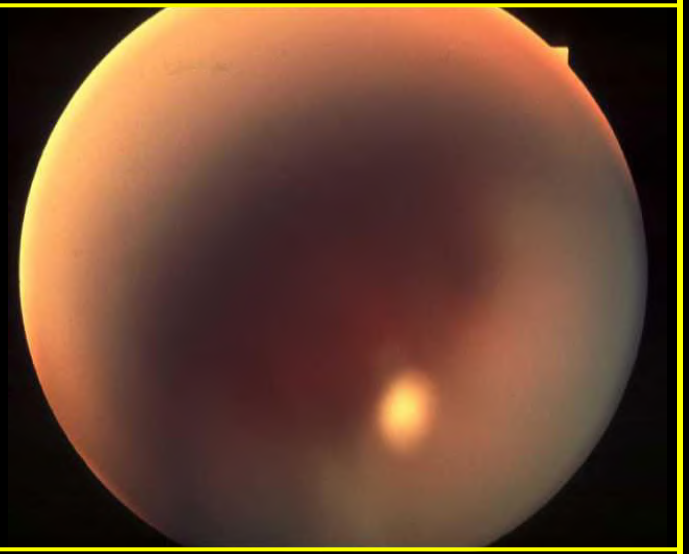
Subtle periphlebitis



Candlewax drippings

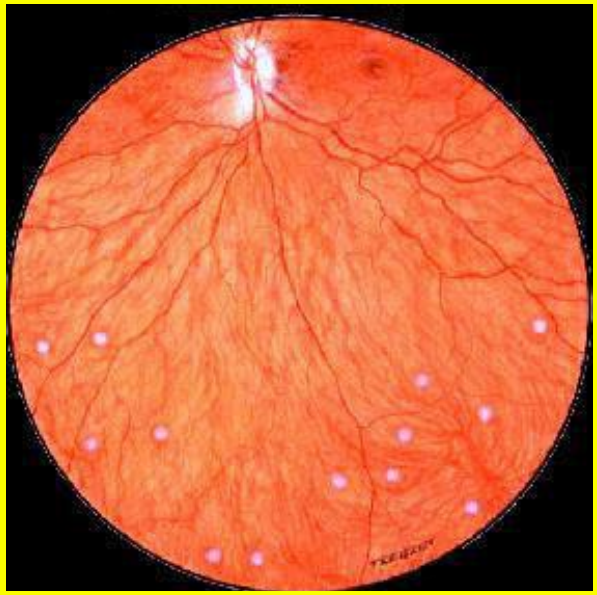
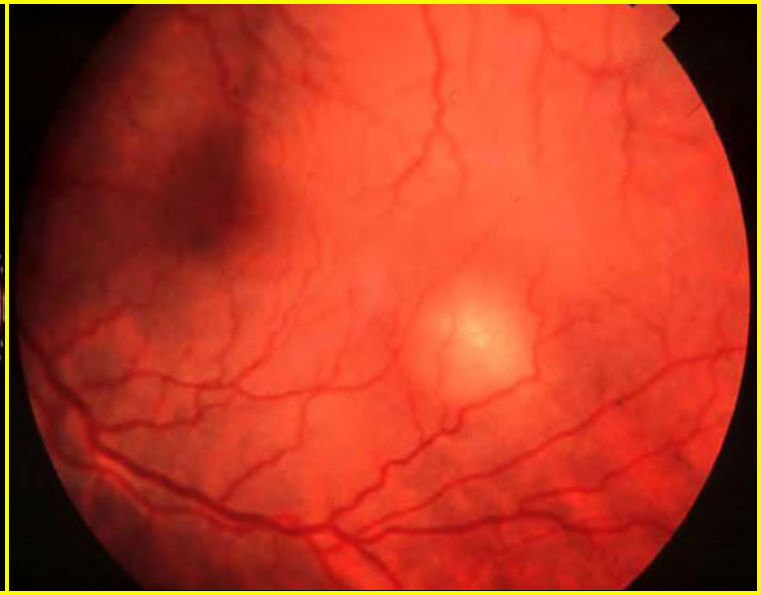
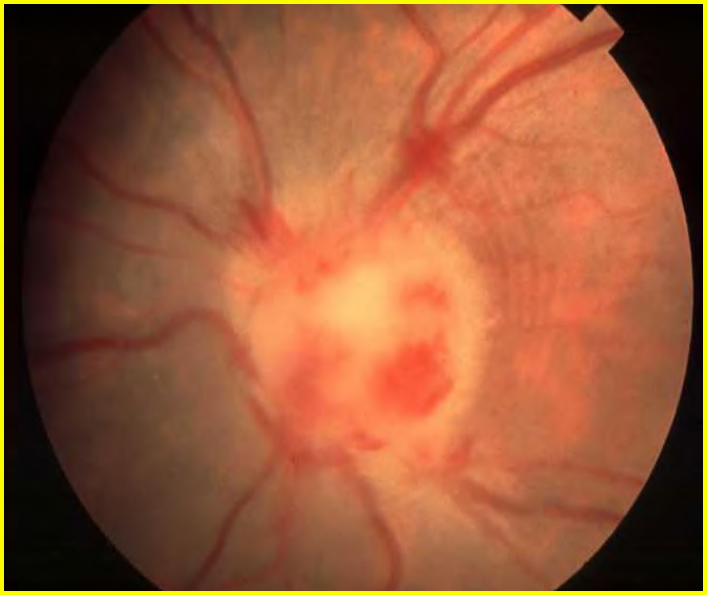


Peripheral neovascularization



Vitritis and snowballs

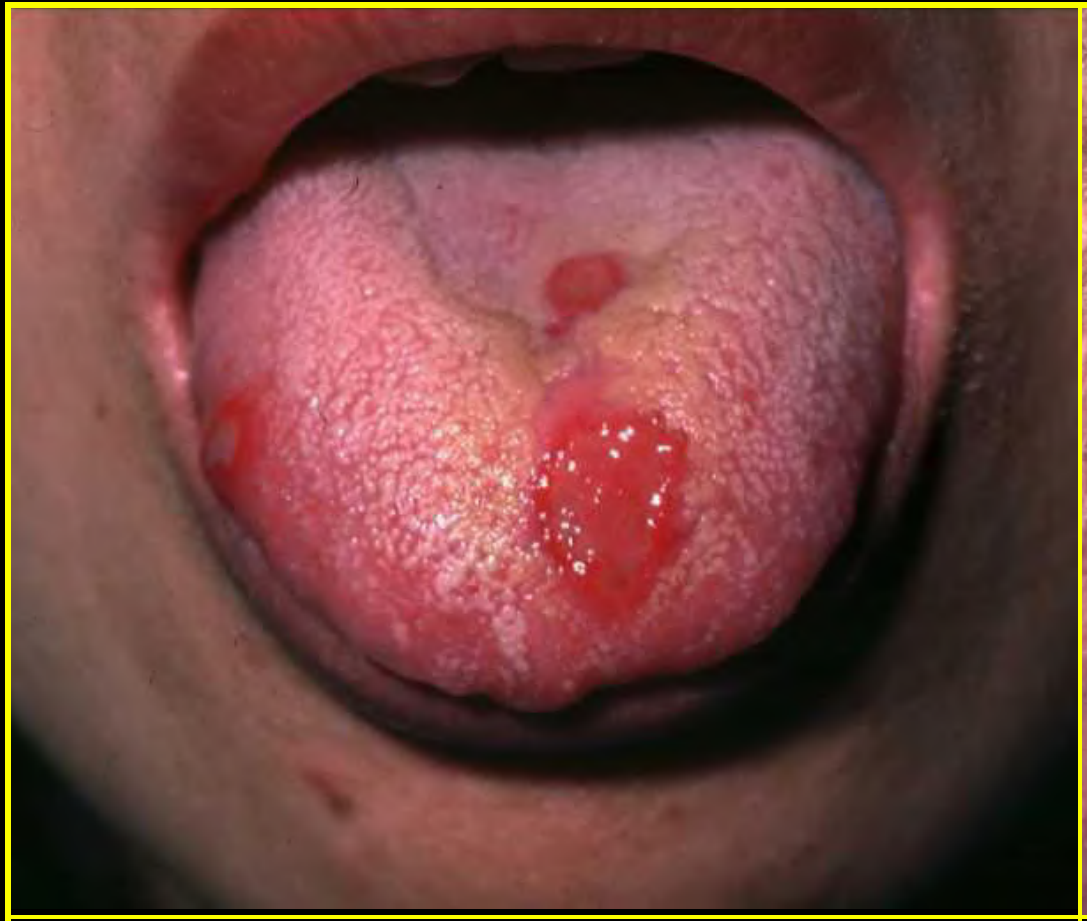
Fundus granulomata in sarcoidosis

		
Retinal and preretinal	Choroidal	Optic nerve head

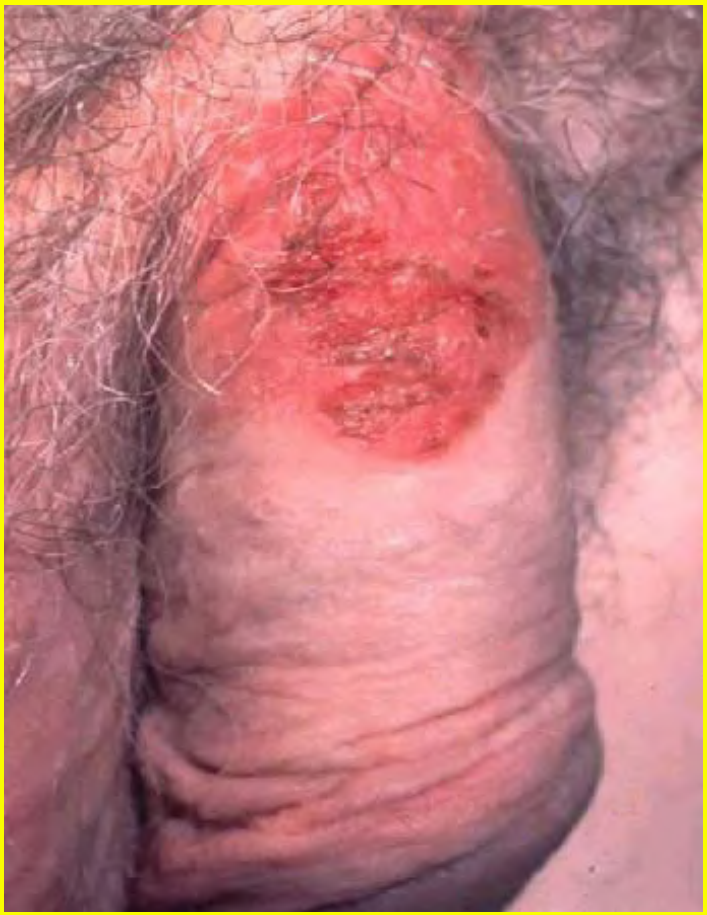
Behçet Disease

1. Idiopathic multisystem disease
2. Presentation - third to fourth decade
3. Most prevalent in Mediterranean region and Japan
4. Associated with HLA-B5 in Turkey and Japan
5. Major diagnostic criteria
   - Oral aphthous ulceration (100%)
   - Genital ulceration (90%)
   - Skin lesions (80%)
   - Uveitis (70%)

Mucocutaneous ulceration in Behçet disease

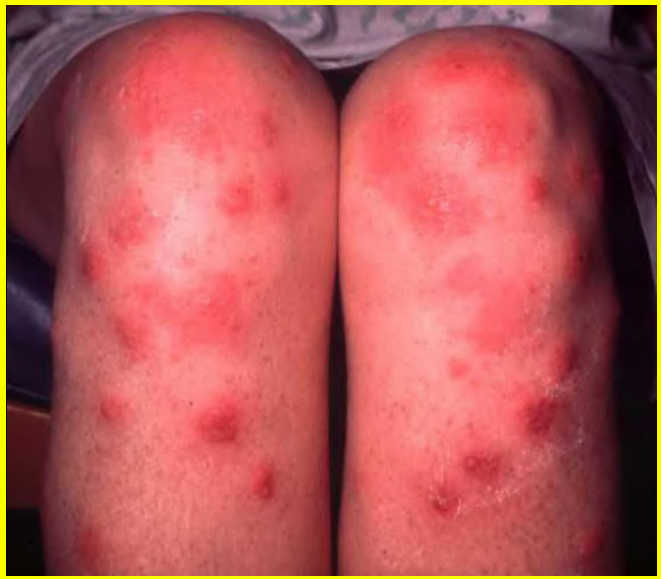


Oral aphthous ulceration - painful, recurrent

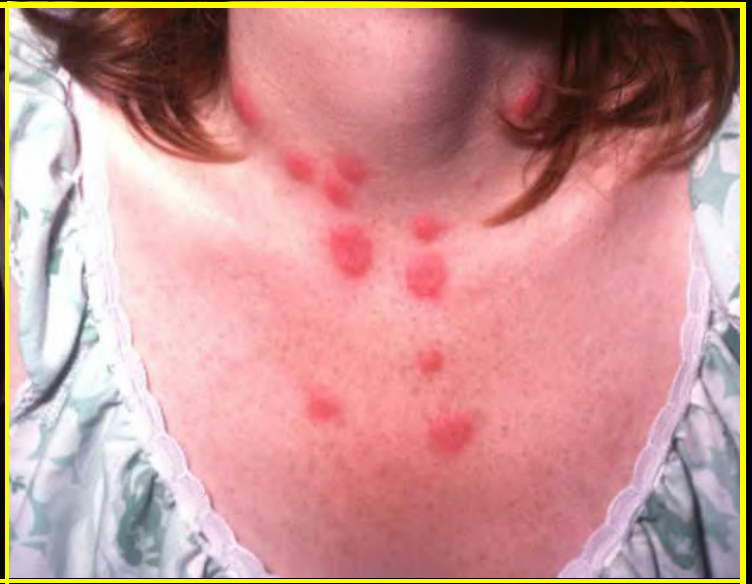


Genital ulceration

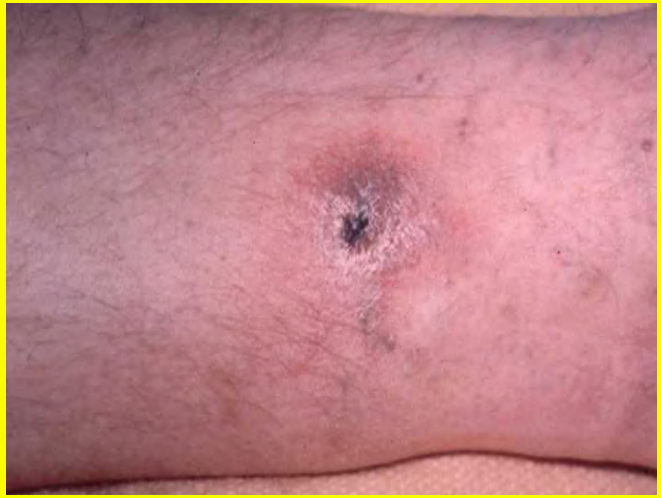
Skin lesions in Behçet disease



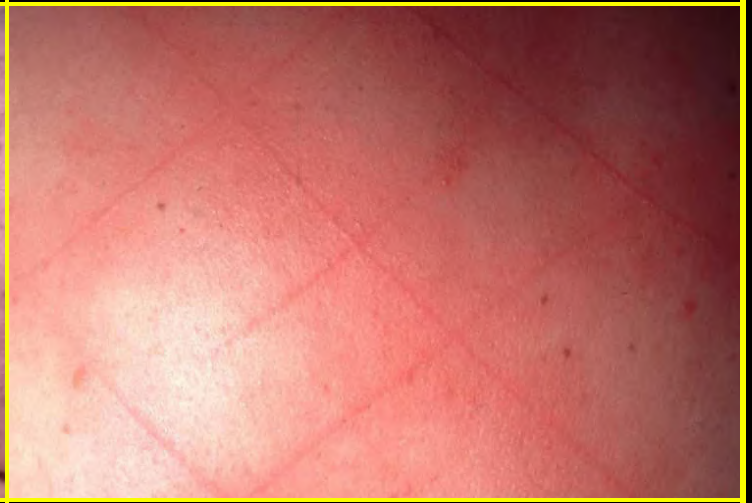
Erythema nodosum



Acneiform

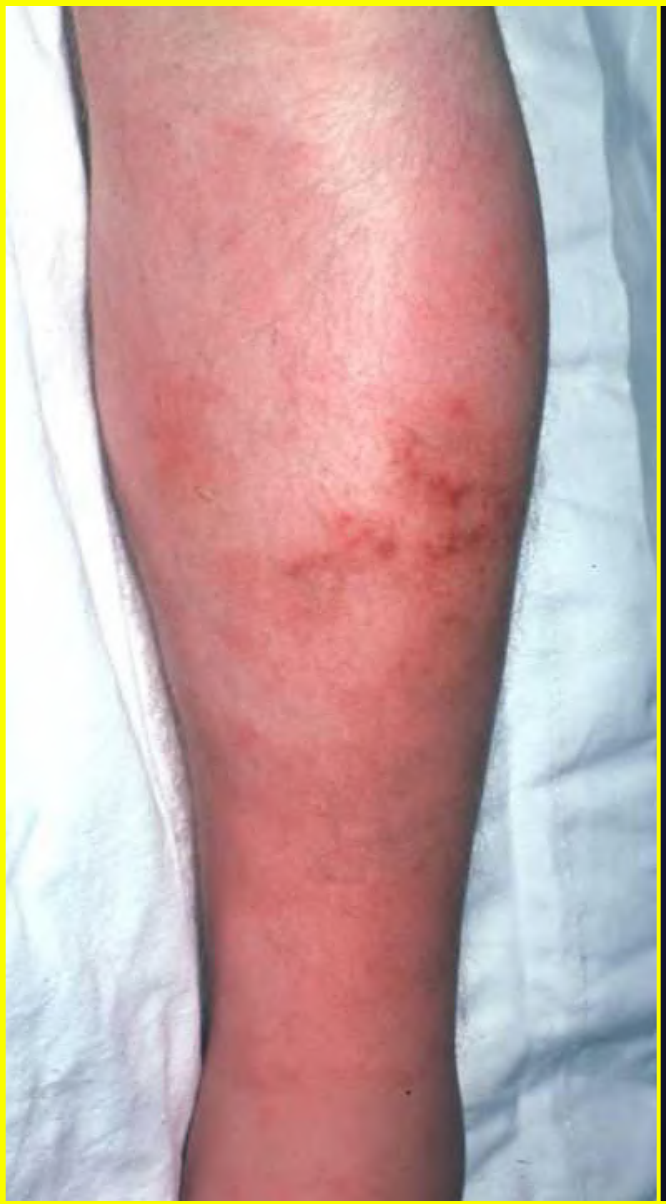


Pustule after scratching skin (pathergy test)

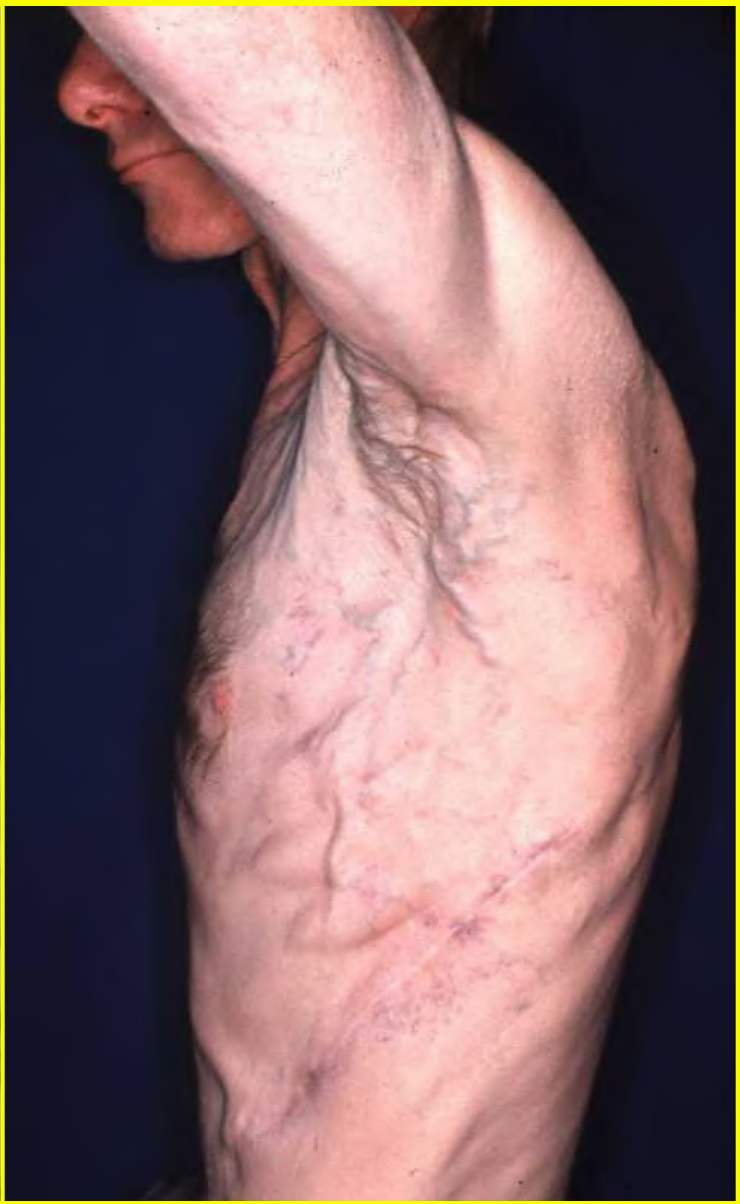


Lines after stroking skin (dermatographism)

Vascular lesions in Behçet disease

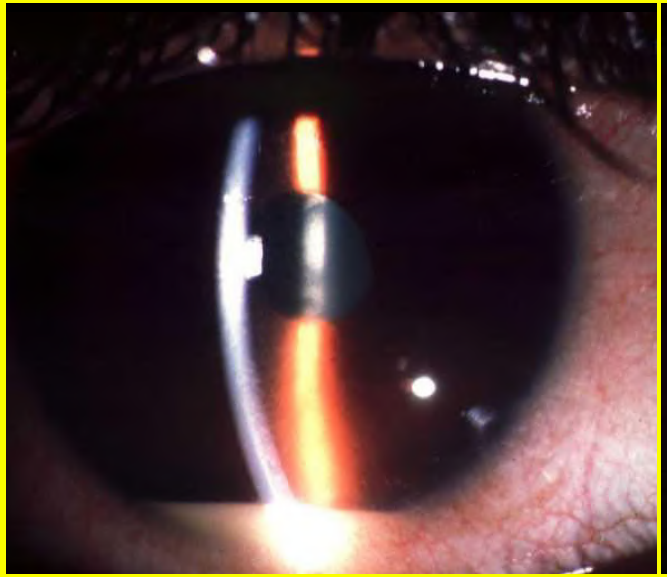


Migratory thrombophlebitis of extremities

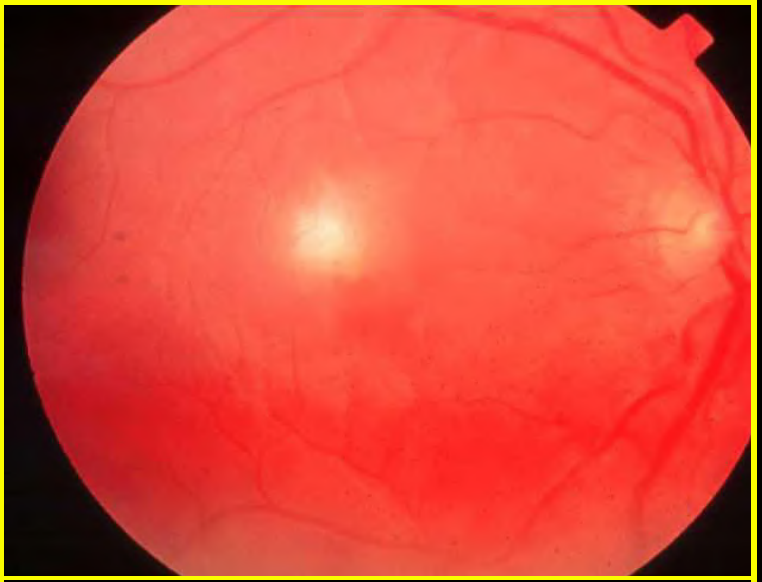


Obliterative thrombophlebitis of major internal veins

Uveitis in Behçet disease

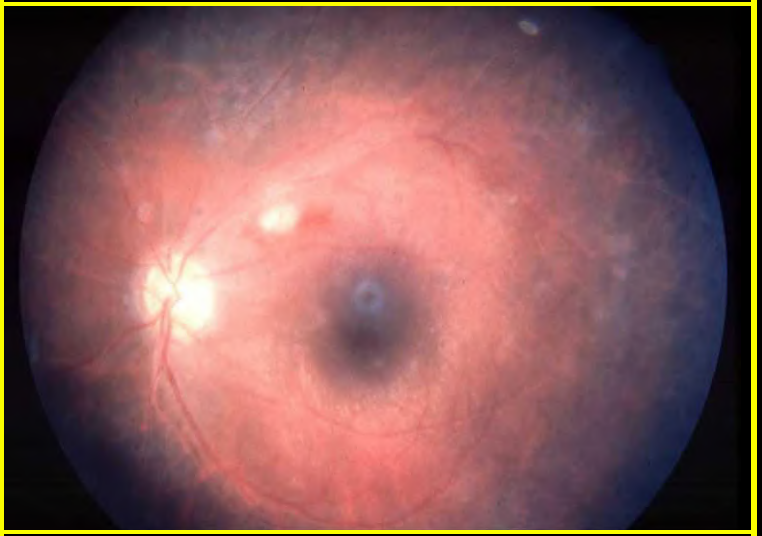


Acute iritis



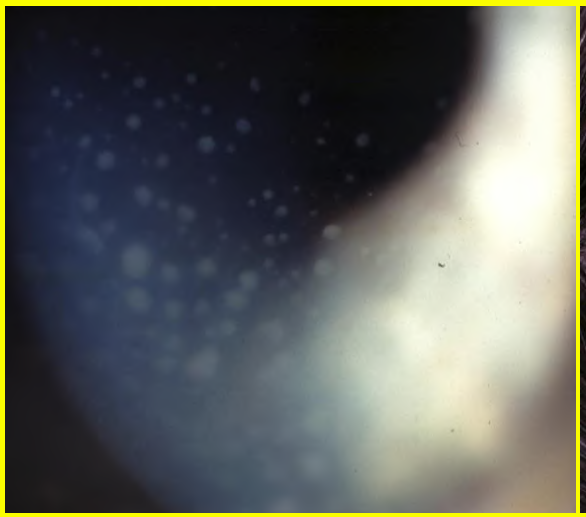
Retinitis

Occlusive periphlebitis

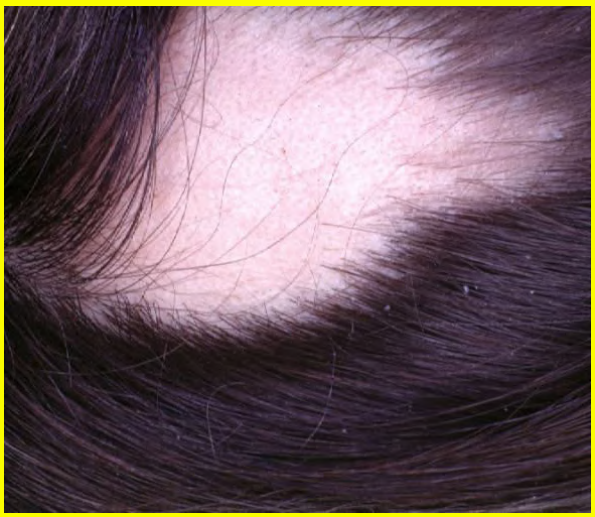


Diffuse leakage

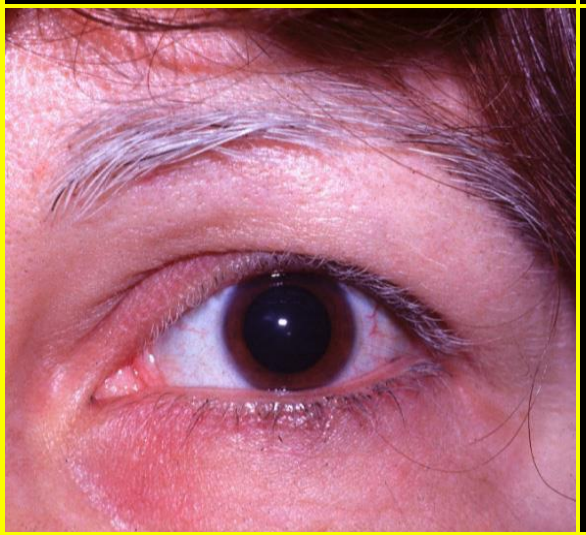
Signs of Vogt-Koyanagi syndrome



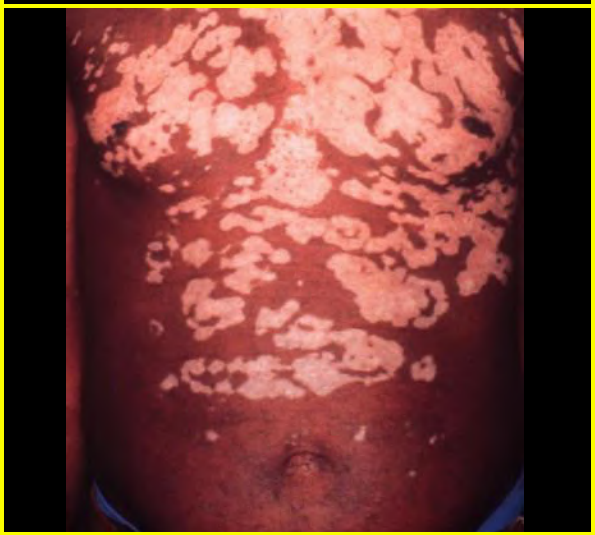
Granulomatous iridocyclitis



Alopecia

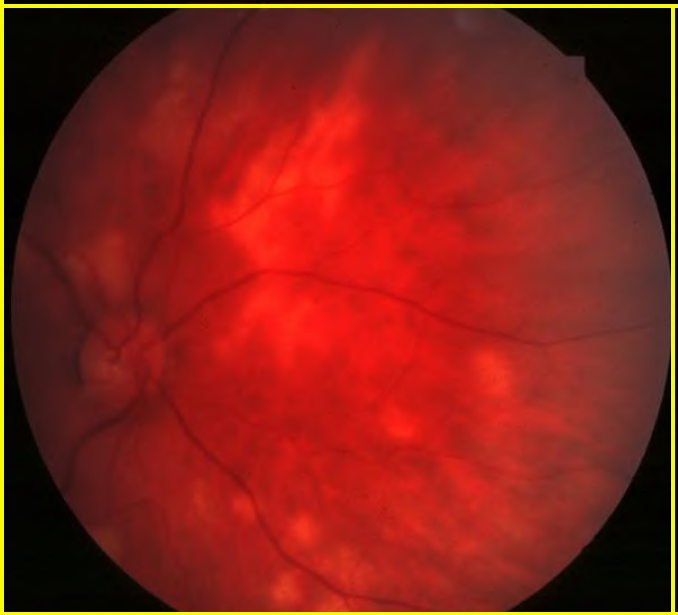
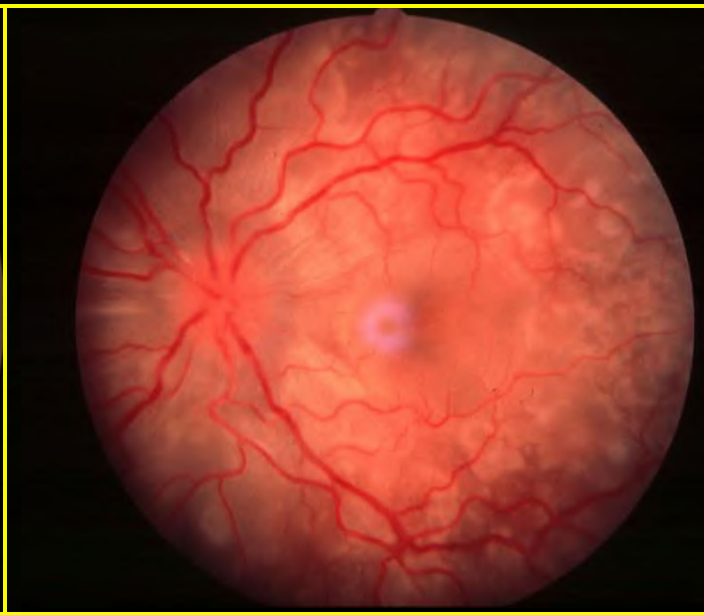
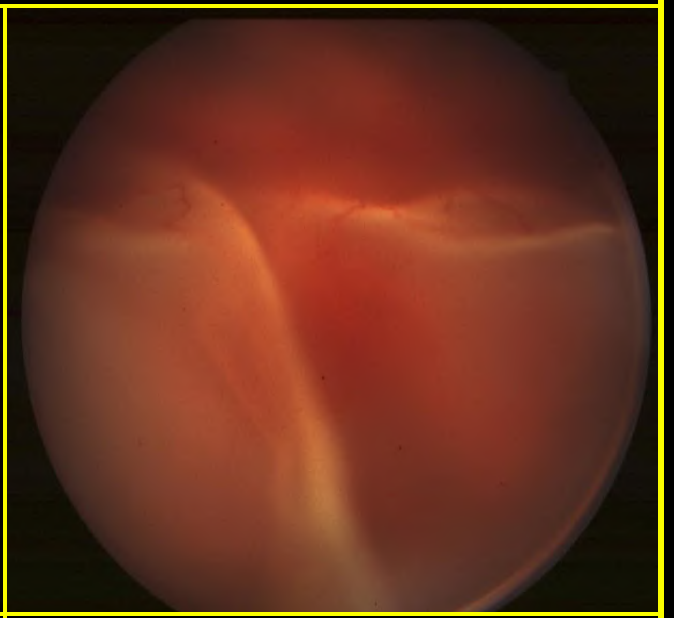


Poliosis

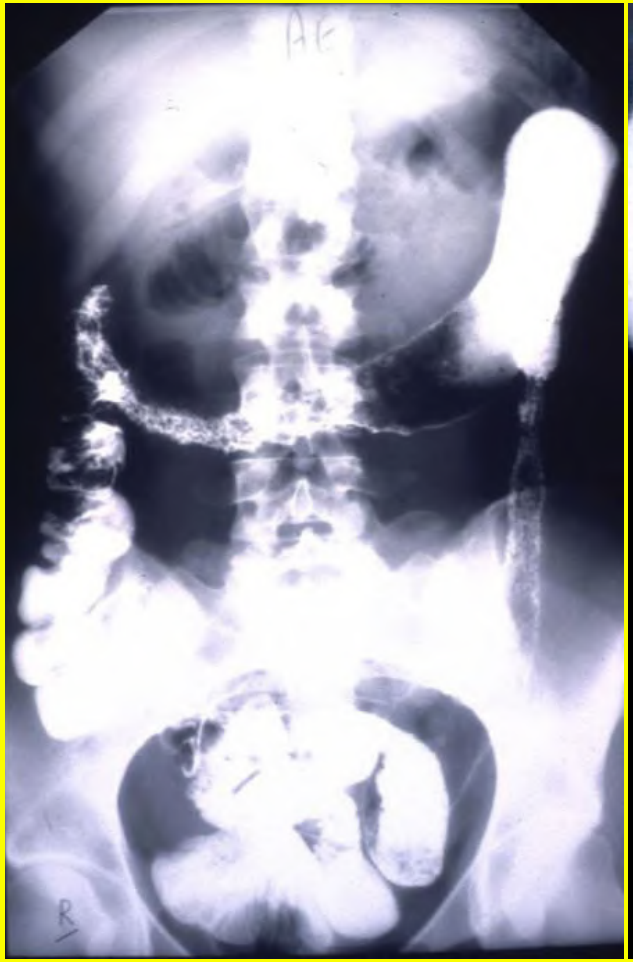
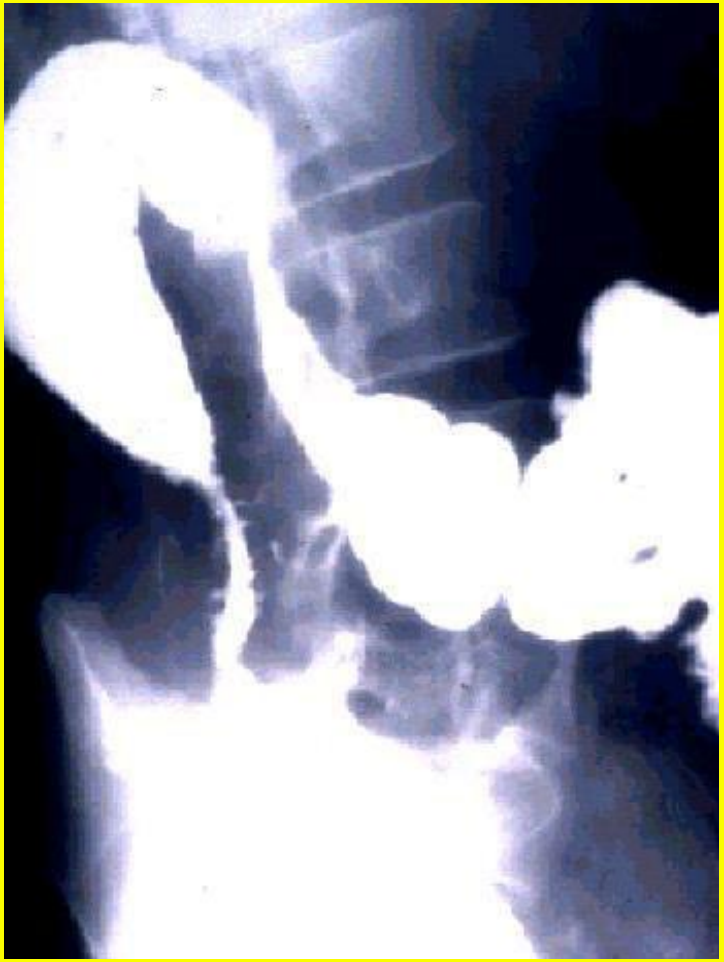


Vitiligo

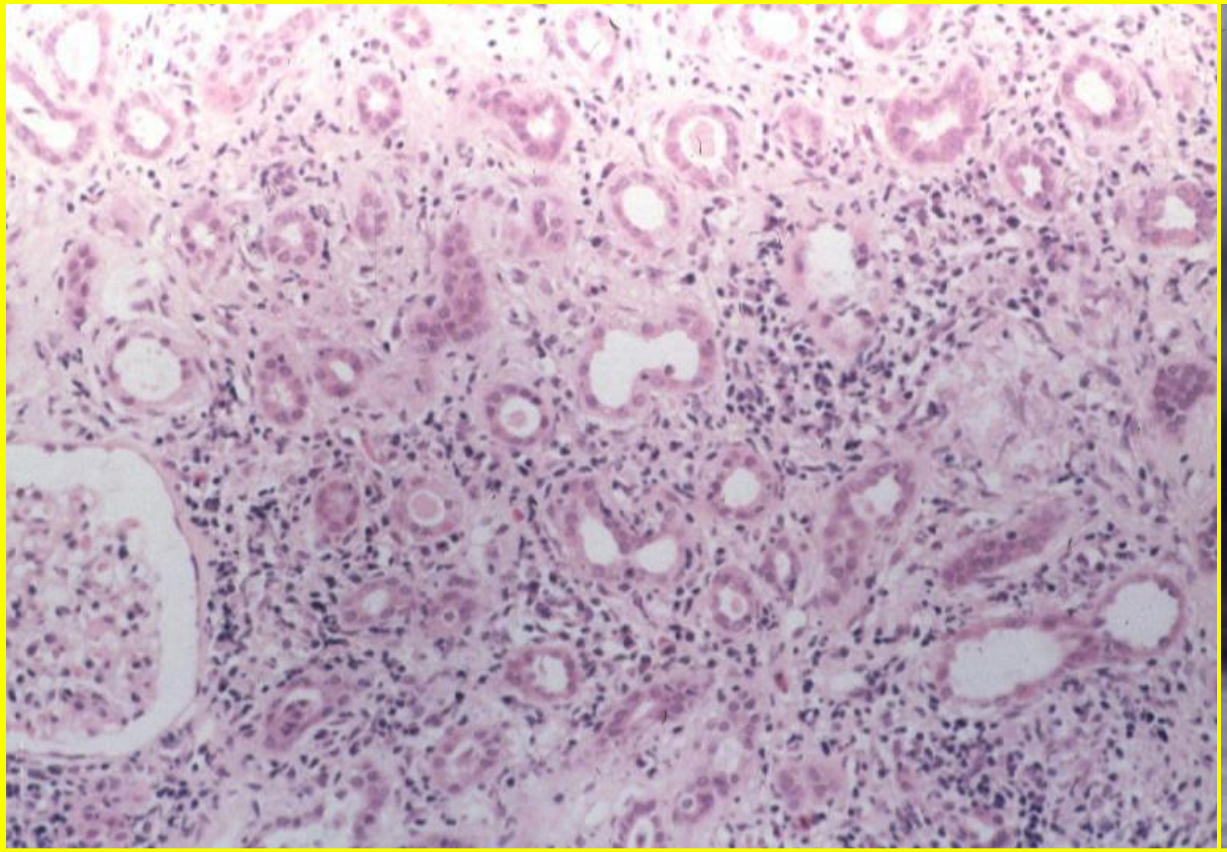
Harada syndrome

Progression of Harada disease		
		
Multifocal choroiditis	Multifocal sensory retinal detachments	Exudative retinal detachment

Inflammatory bowel disease

Ulcerative colitis	Crohn disease
	
· Large bowel ulceration Acute iritis - uncommon	· Stricture and ‘rose thorn’ ulceration · Acute iritis - uncommon

Tubulointerstitial nephritis and uveitis (TINU)

Renal histology	Urine
	
• Most frequently affects women and children • Hypersensitivity reaction to drugs • Bilateral, recurrent anterior uveitis	• Proteinuria and renal failure • Good response to systemic steroids

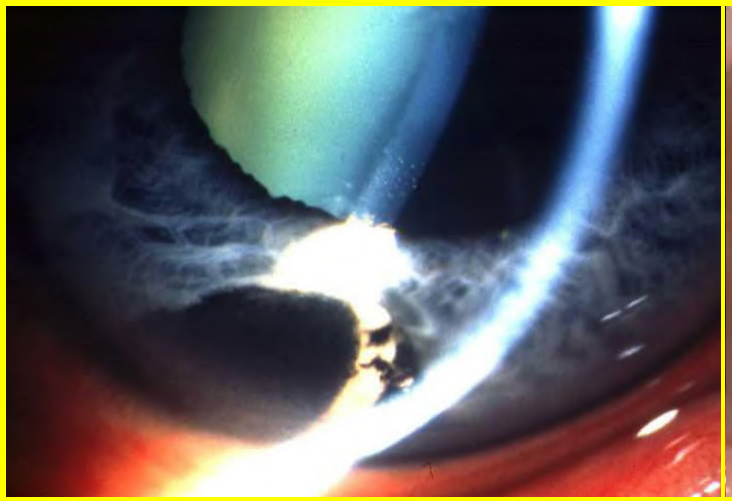
UVEAL TUMOURS

1. Iris melanoma
2. Iris naevus
3. Ciliary boy melanoma
4. Choroidal melanoma
5. Choroidal naevus
6. Choroidal haemangioma
   - Circumscribed
   - Diffuse
7. Choroidal metastatic carcinoma
8. Choroidal osseous choristoma
9. Melanocytoma

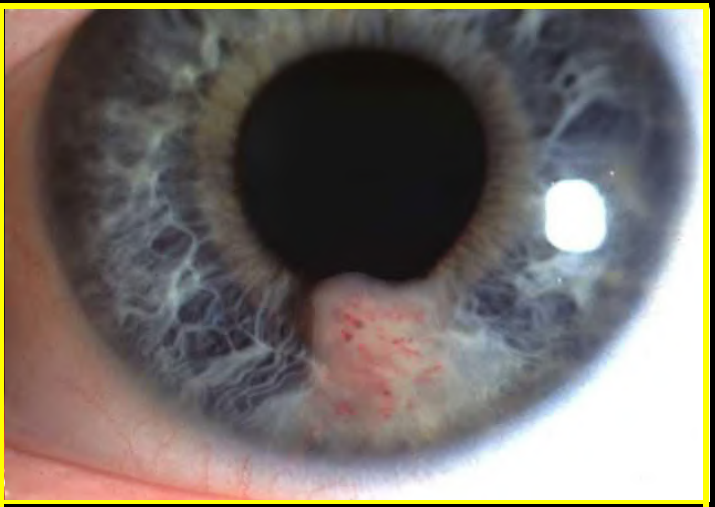
Iris Melanoma

1. Very rare - 8% of uveal melanomas
2. Presentation - fifth to sixth decades
3. Very slow growth
4. Low malignancy
5. Excellent prognosis

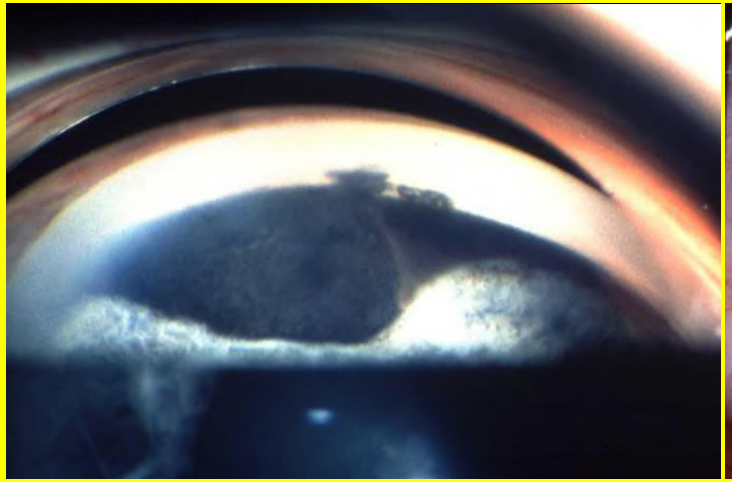
Iris melanoma



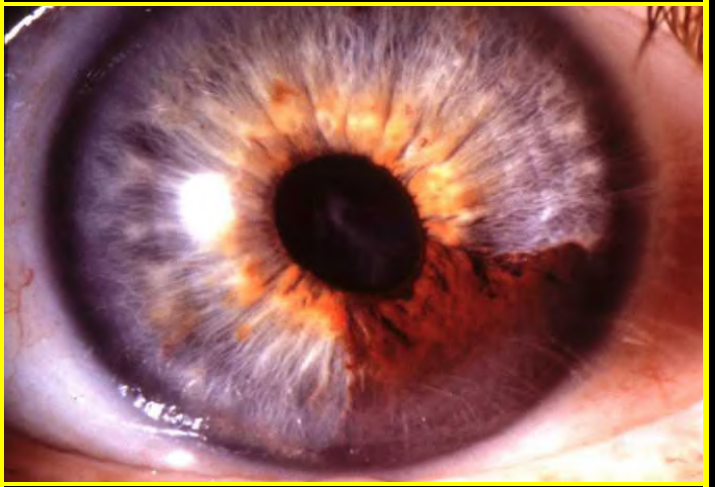
- Usually pigmented nodule at least 3 mm in diameter
- Invariably in inferior half of iris



- Occasionally non-pigmented
- Surface vascularization

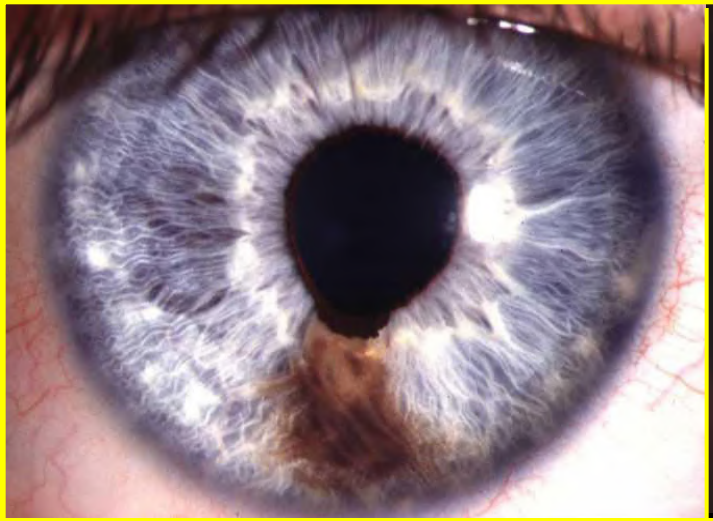


- Angle involvement may cause glaucoma

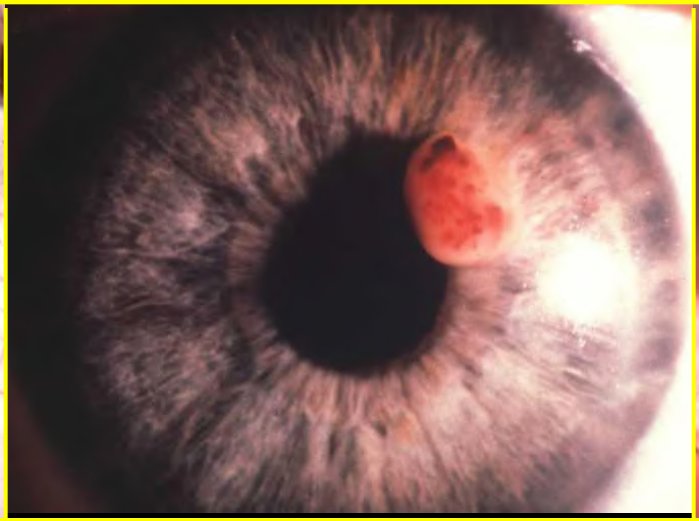


- Pupillary distortion, ectropion uveae and cataract

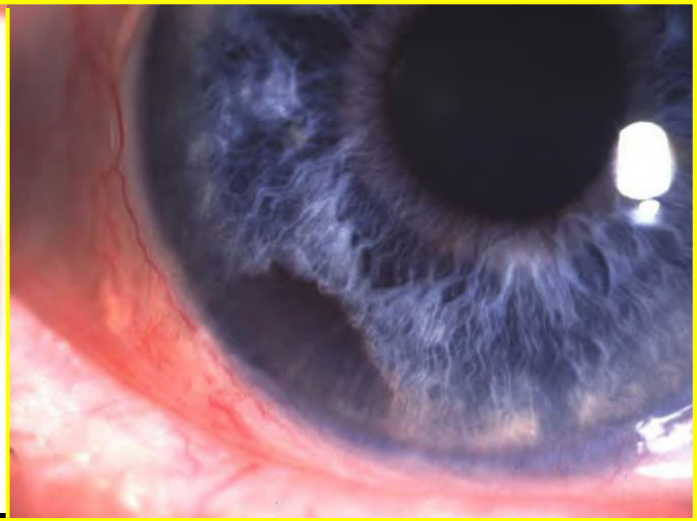
Differential diagnosis of iris melanoma



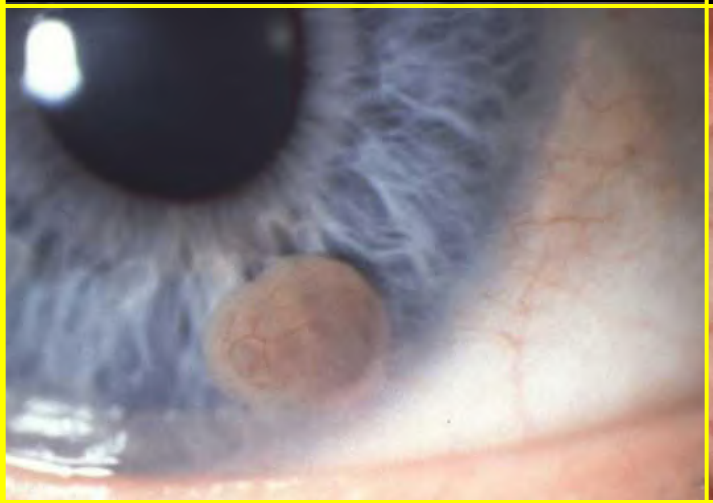
Large iris naevus distorting pupil



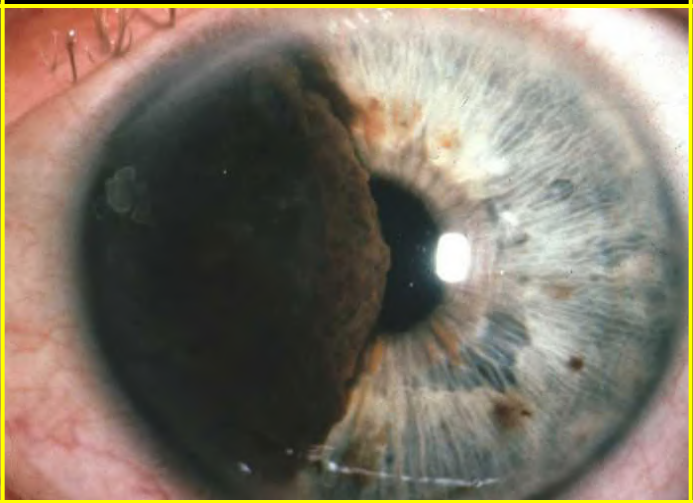
Leiomyoma



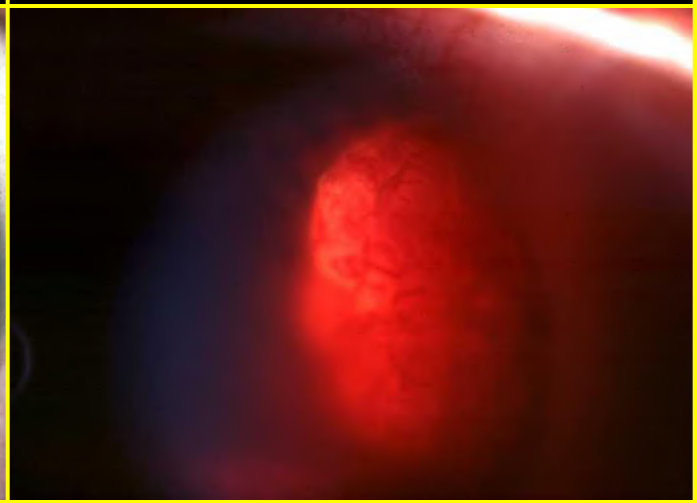
Adenoma of pigment epithelium



Primary iris cyst

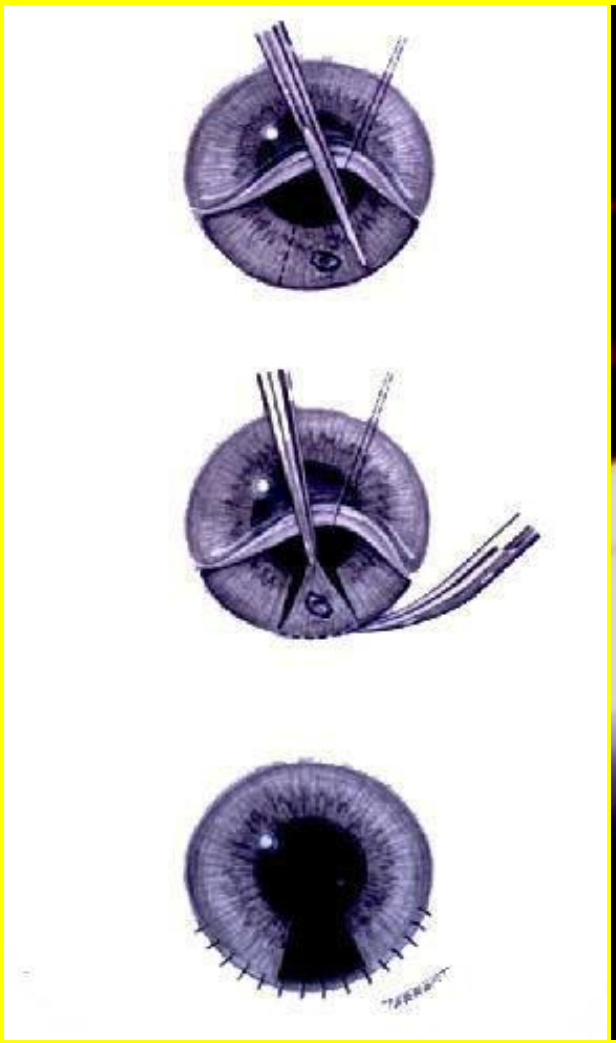


Ciliary body melanoma eroding iris root

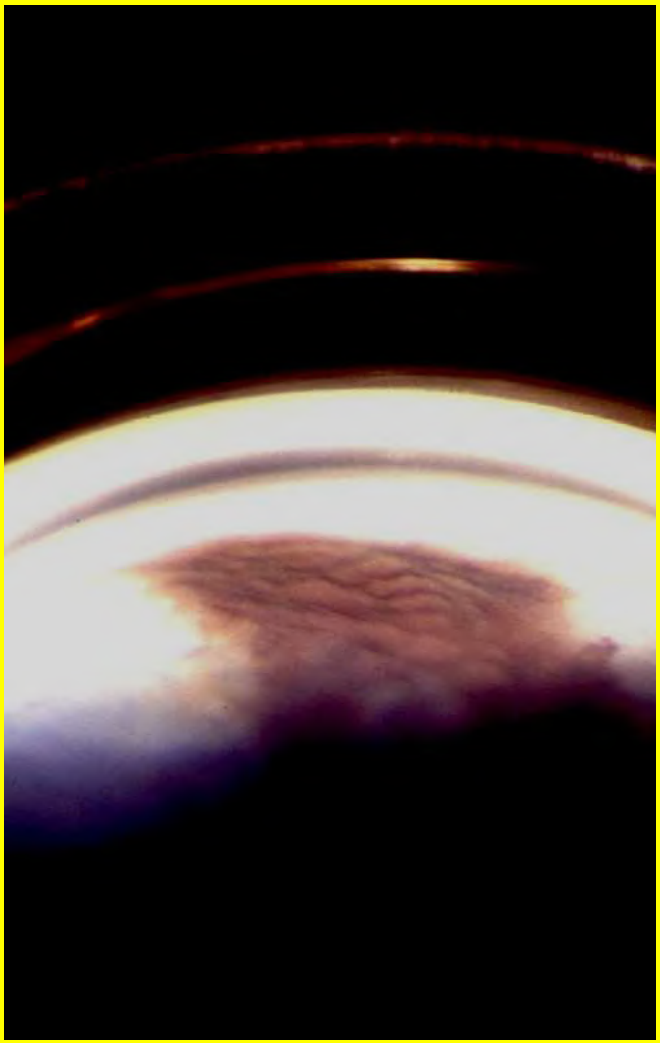


Metastasis to iris

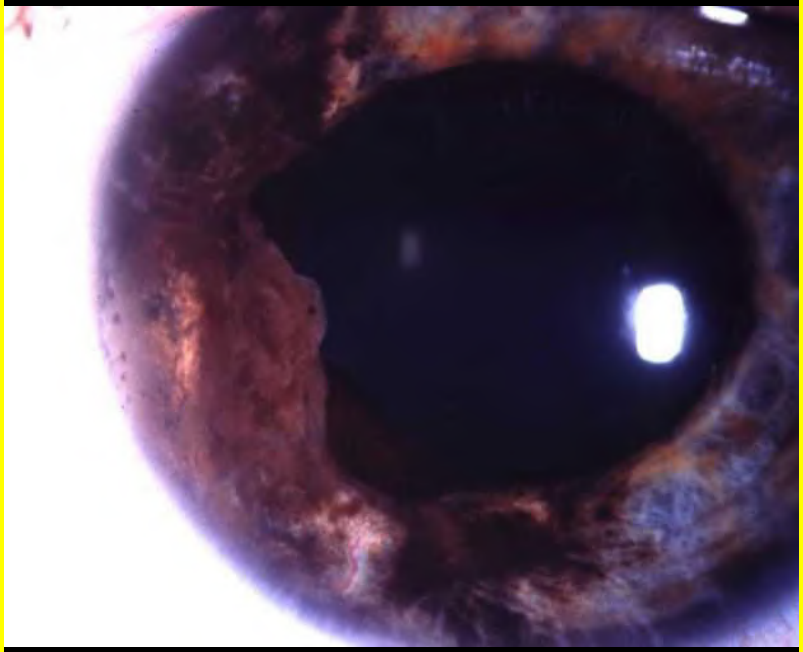
Treatment of iris melanoma



Small tumour
- broad iridectomy

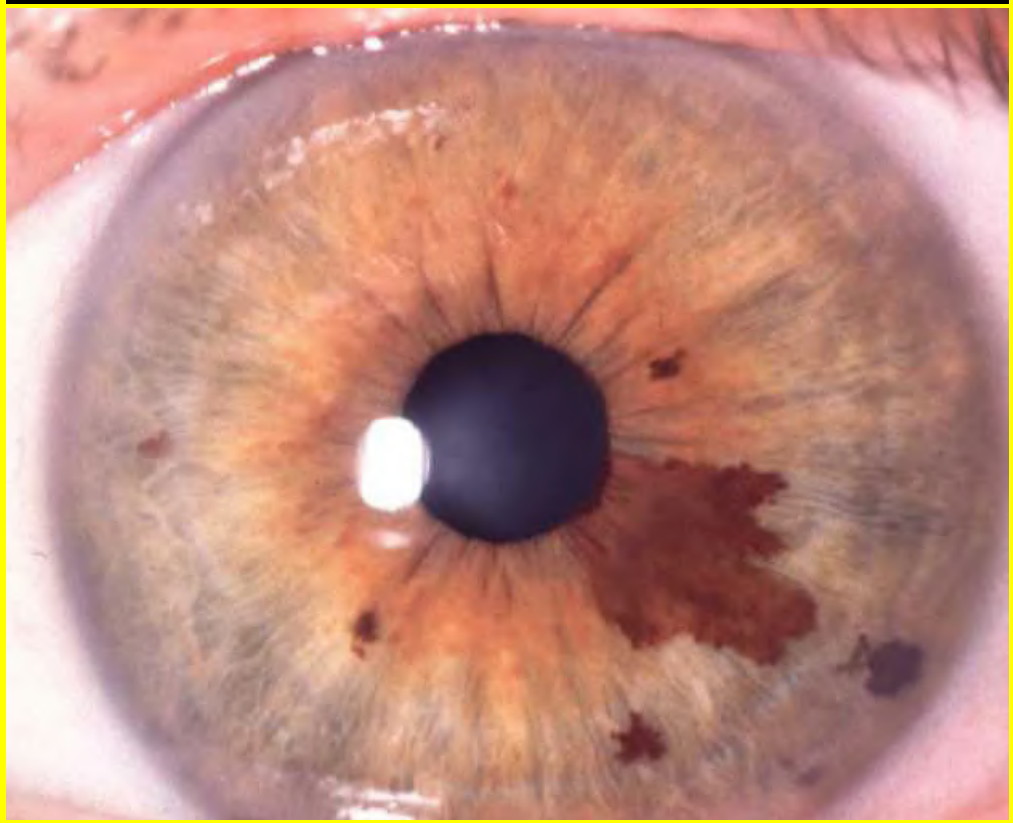


Angle invasion by tumour
- iridocyclectomy

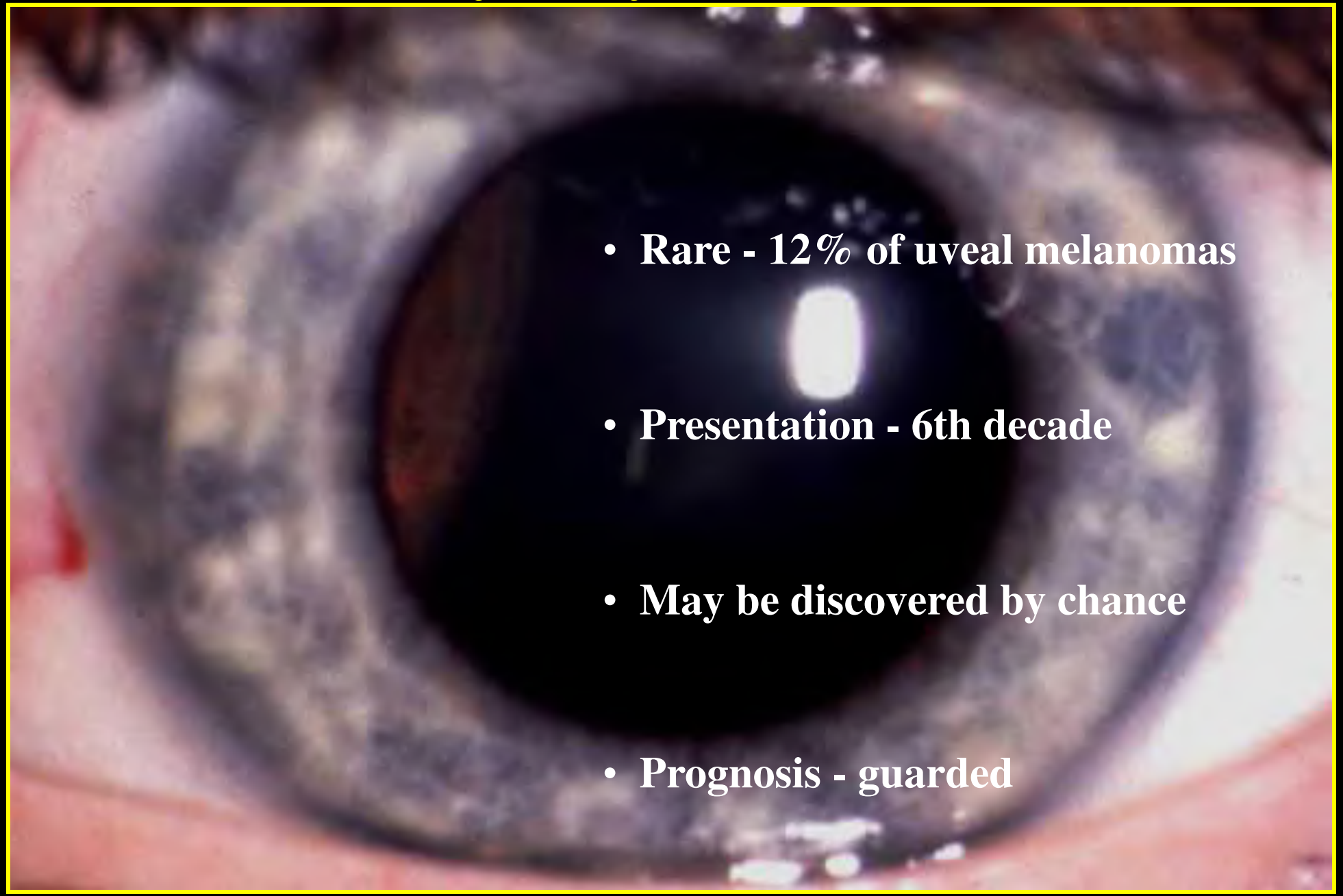


Non-resectable tumour
- radiotherapy or enucleation

Iris naevus

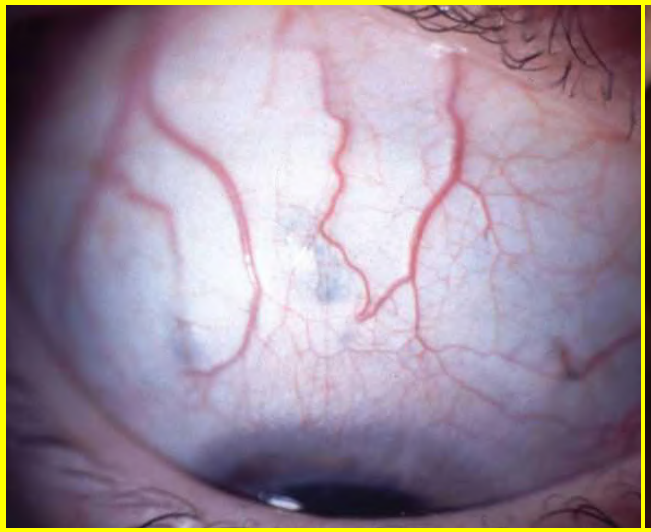
Typical	Diffuse
	
• Pigmented, flat or slightly elevated • Diameter usually less than 3 mm • Occasionally mild distortion of pupil and ectropion uvea	• Obscures iris crypts • May cause ipsilateral hyperchromic heterochromia • May be associated with Cogan-Reese syndrome

Ciliary body melanoma

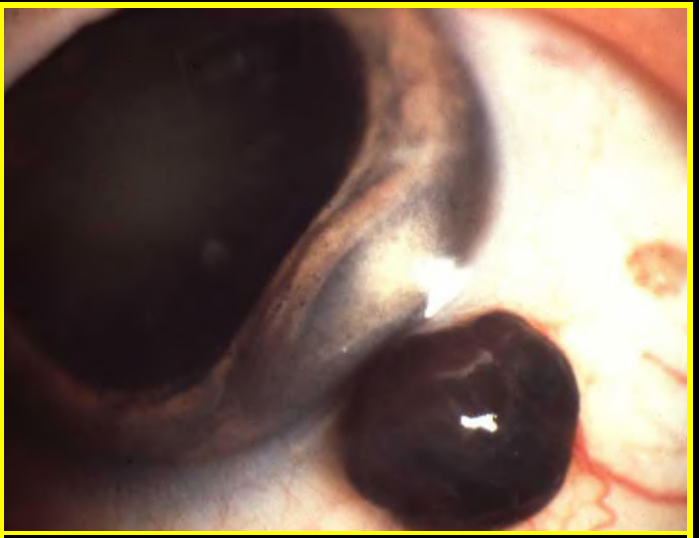


- Rare - 12% of uveal melanomas
- Presentation - 6th decade
- May be discovered by chance
- Prognosis - guarded

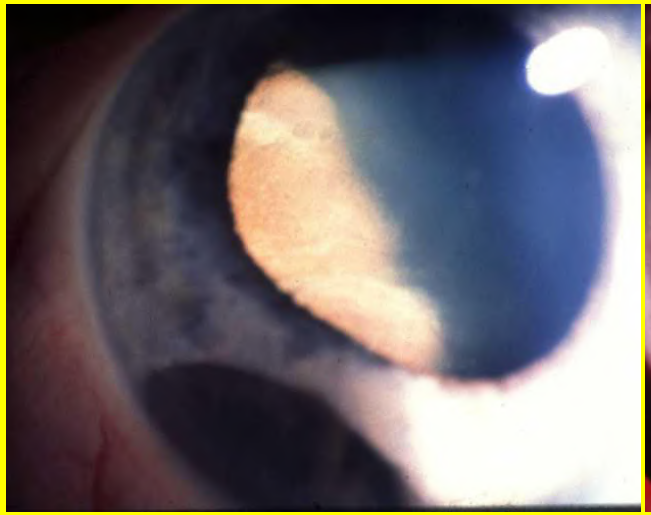
Signs of ciliary body melanoma



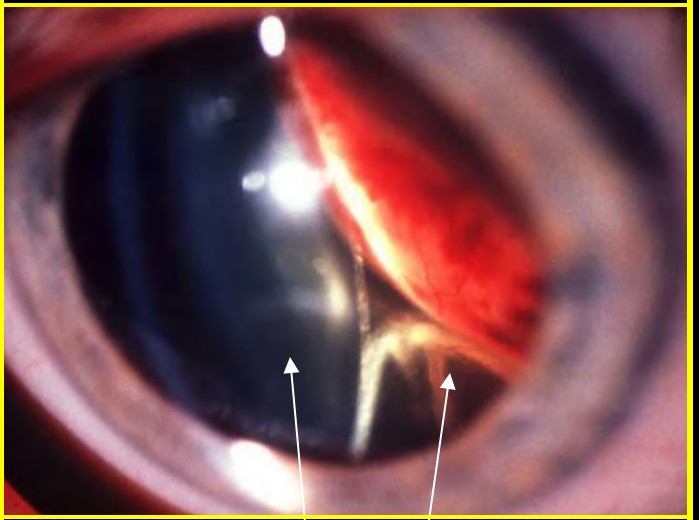
- Sentinel vessels



- Extraocular extension



- Erosion through iris root



- Lens subluxation or cataract
- Retinal detachment

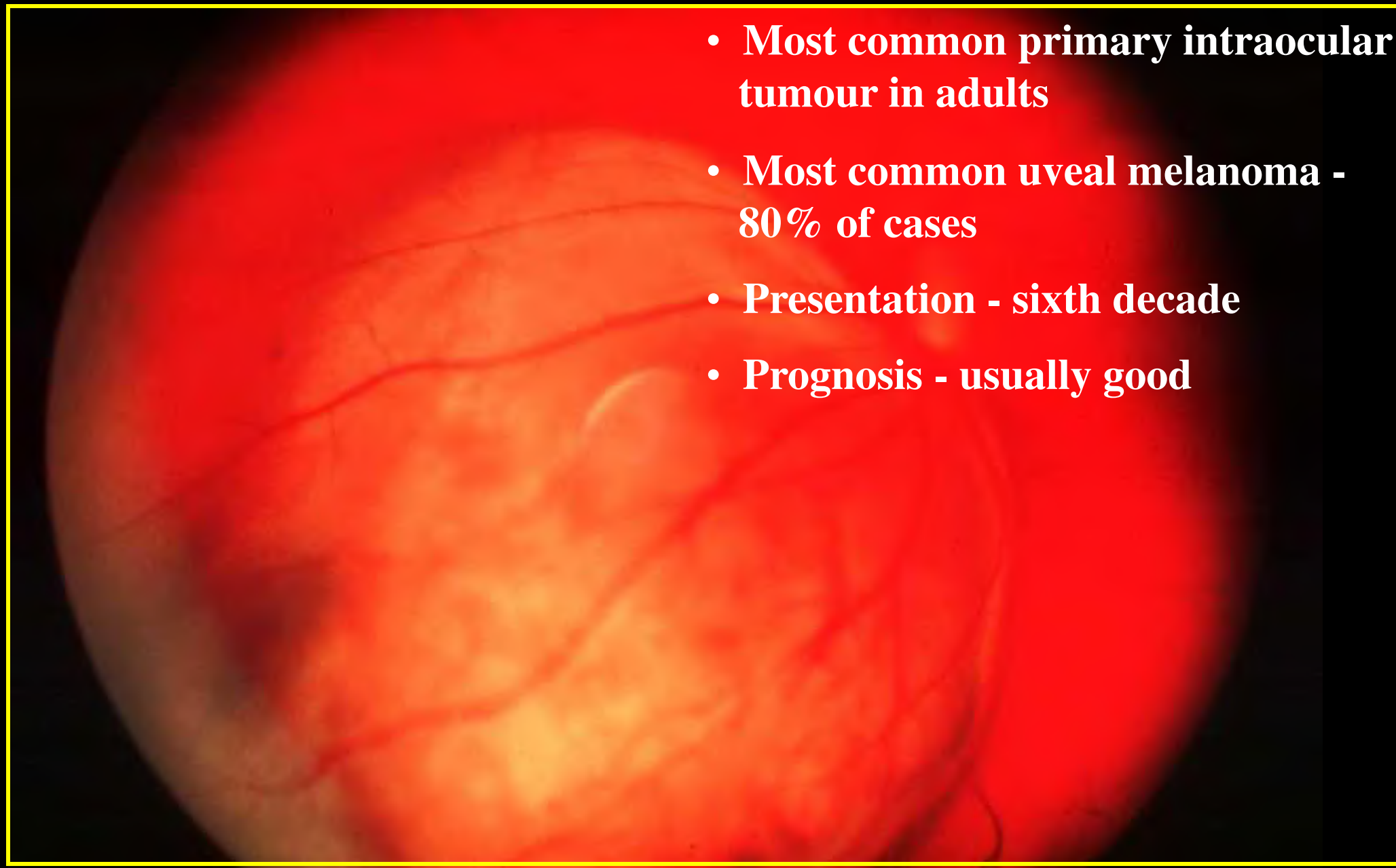
Treatment options of ciliary body melanoma

1. Iridocyclectomy
   - small or medium tumours

2. Enucleation
   - large tumours

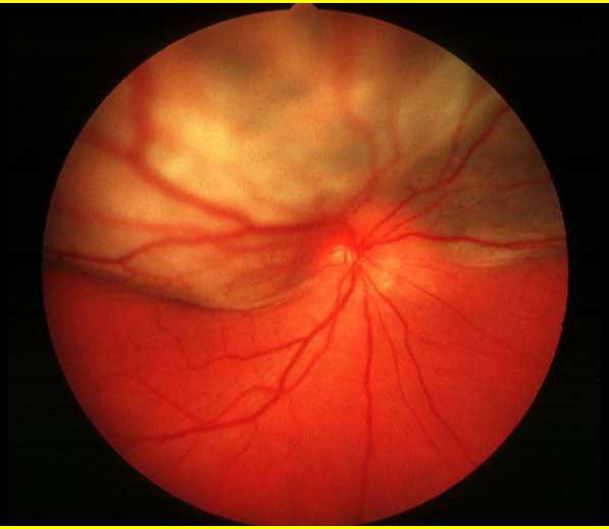
3. Radiotherapy
   - selected cases

Choroidal melanoma

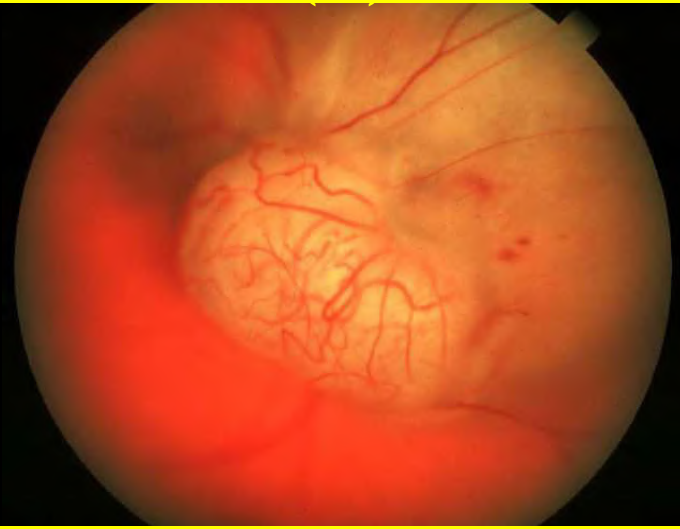


- Most common primary intraocular tumour in adults
- Most common uveal melanoma - 80% of cases
- Presentation - sixth decade
- Prognosis - usually good

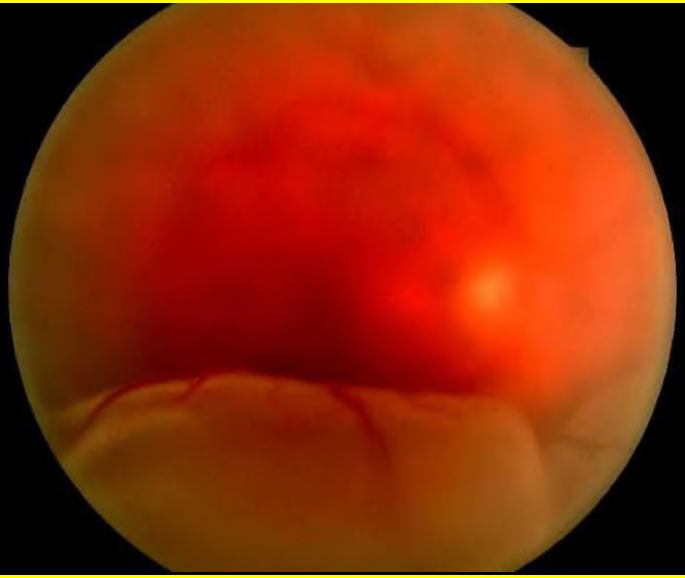
Choroidal melanoma (1)



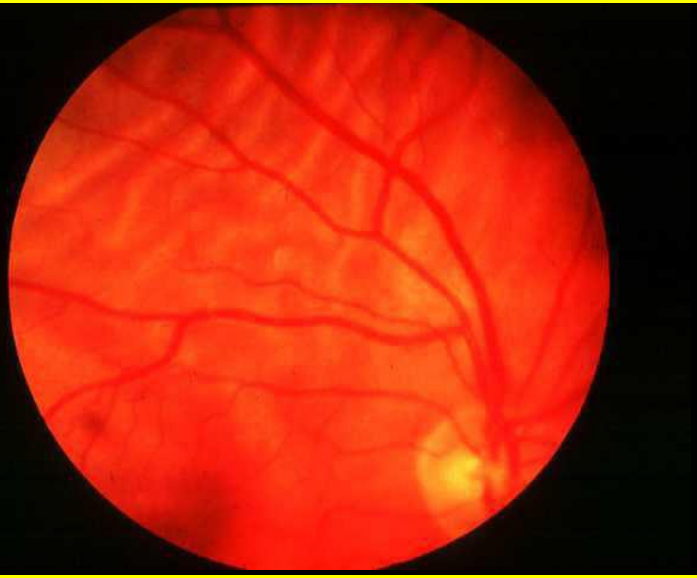
- Brown, elevated, subretinal mass



- Occasionally amelanotic
- Double circulation

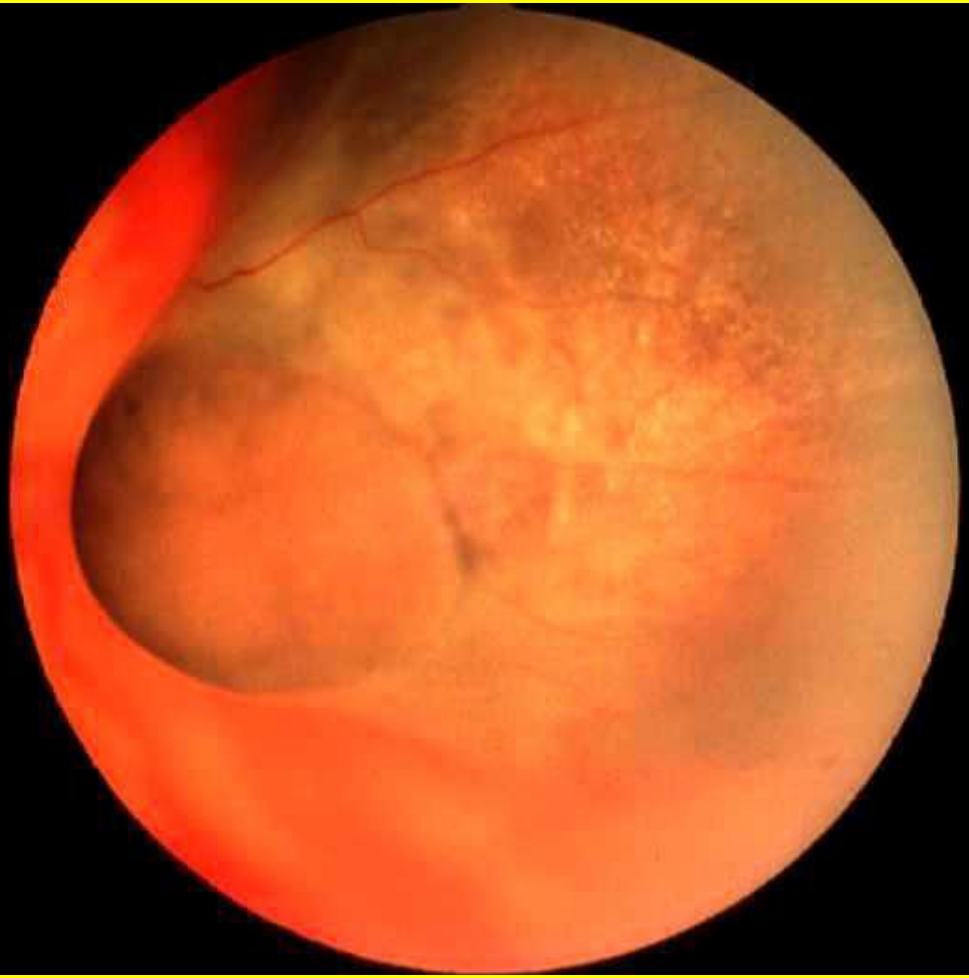


- Secondary retinal detachment

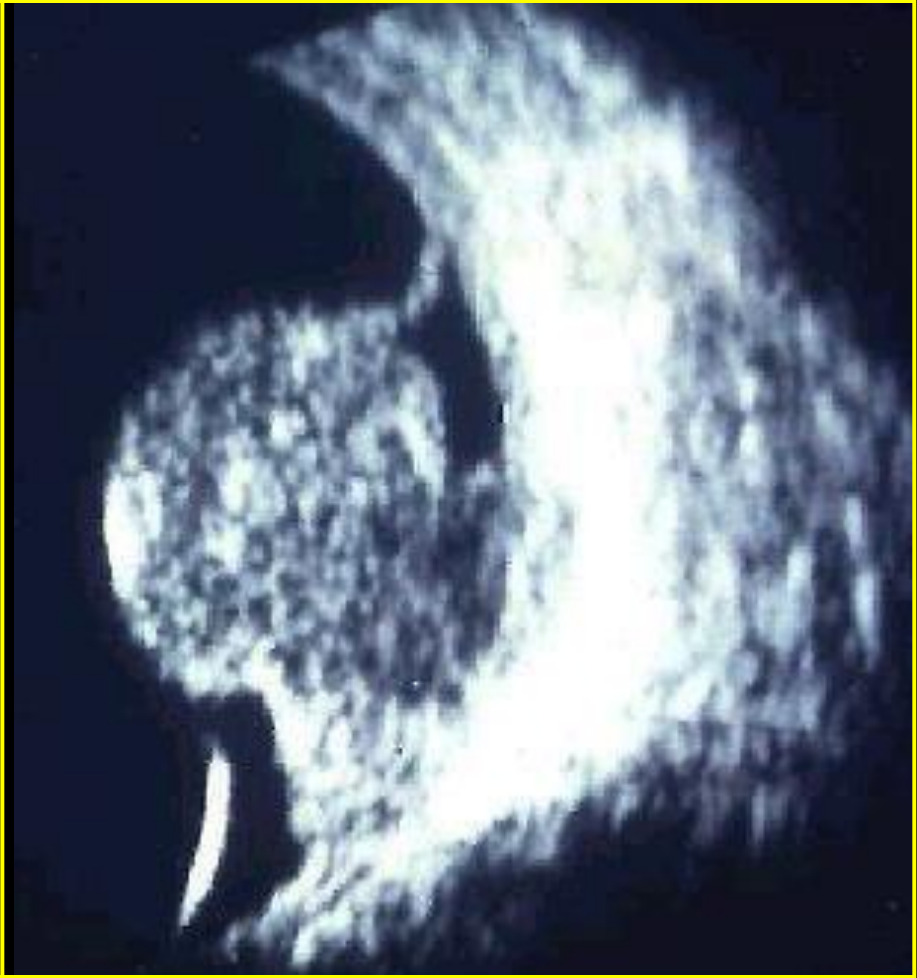


- Choroidal folds

Choroidal melanoma (2)

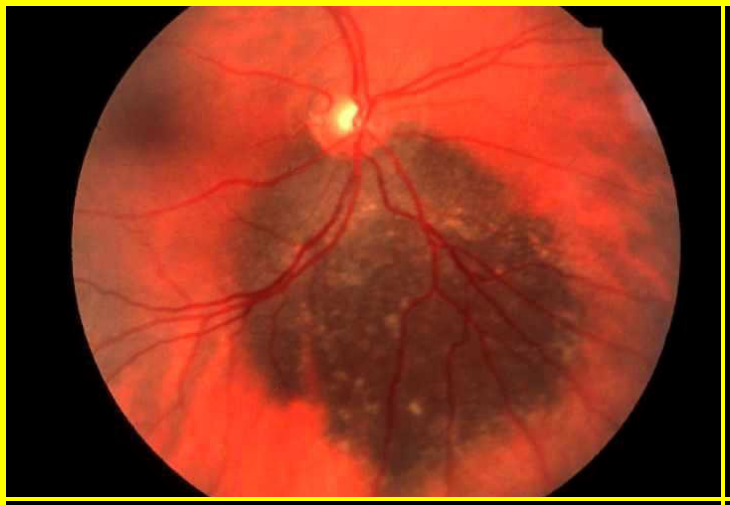


- Surface orange pigment (lipofuscin) is common
- Mushroom-shaped if breaks through Bruch's membrane

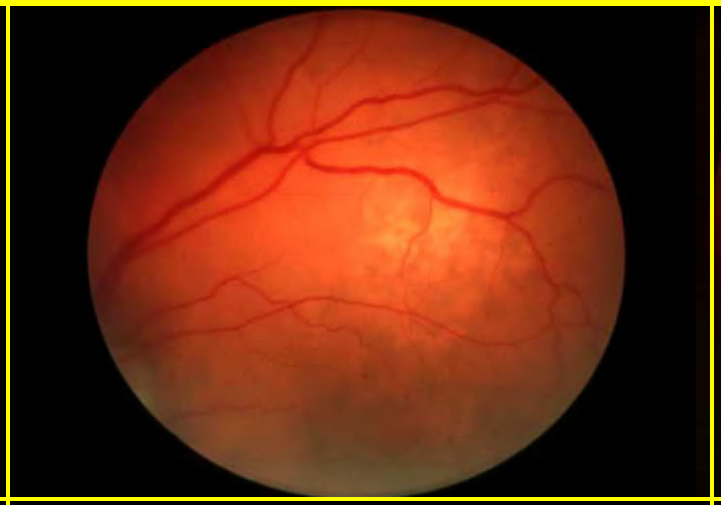


- Ultrasound - acoustic hollowness, choroidal excavation and orbital shadowing

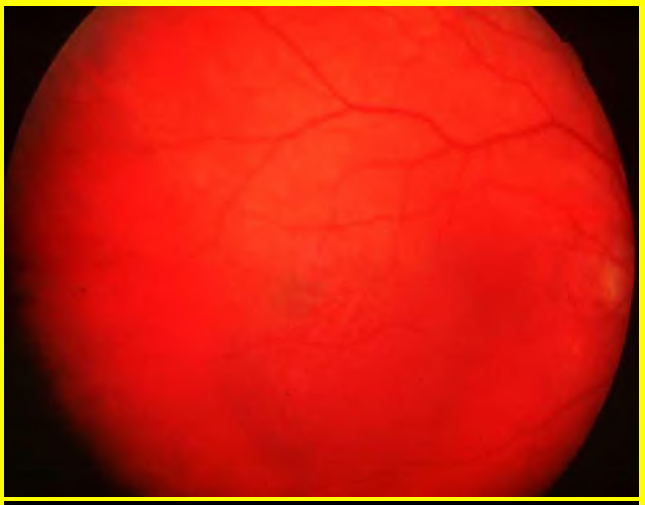
Differential diagnosis of choroidal melanoma



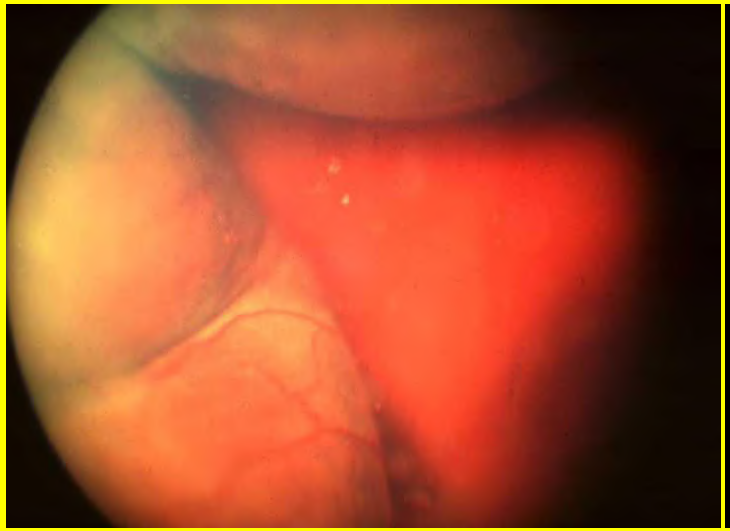
Large choroidal naevus



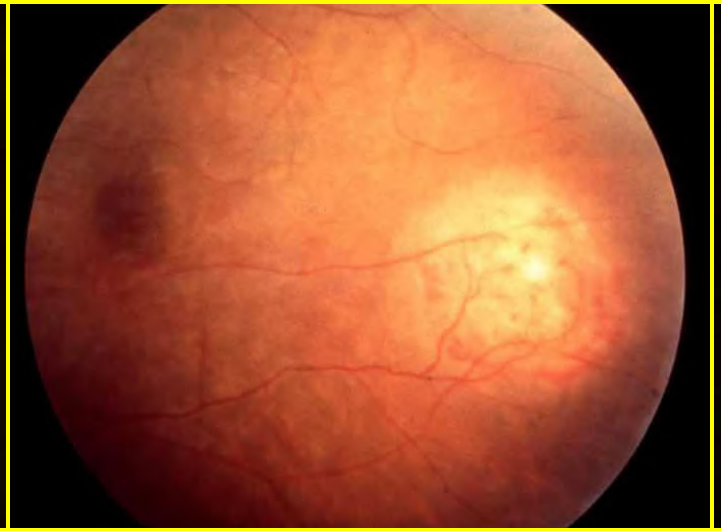
Metastatic tumour



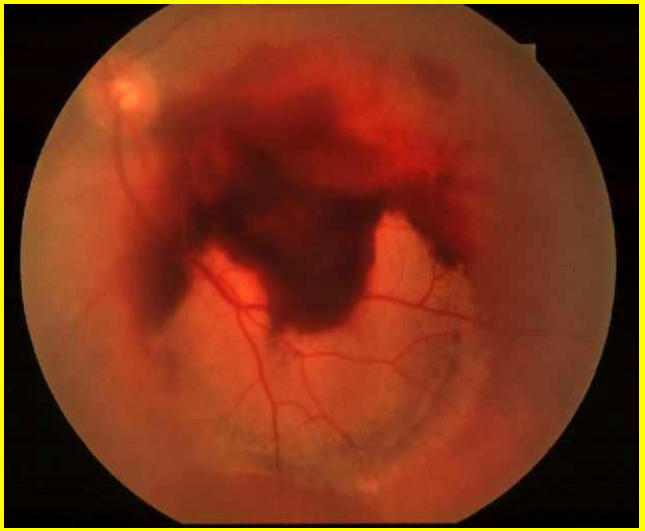
Localized choroidal haemangioma



Choroidal detachment



Choroidal granuloma



Dense sub-retinal or sub-RPE haemorrhage

Treatment of choroidal melanoma

1. **Brachytherapy**
   - less than 10 mm elevation and 20 mm diameter

2. **Charged particle irradiation**
   - if unsuitable for brachytherapy

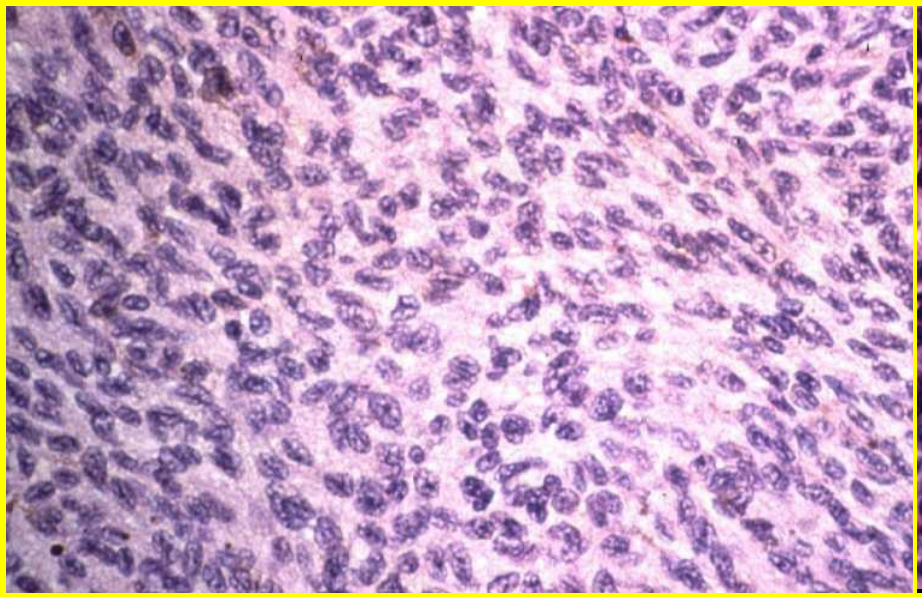
3. **Transpupillary thermotherapy**
   - selected small tumours

4. **Trans-scleral local resection**
   - carefully selected tumours less than 16 mm in diameter

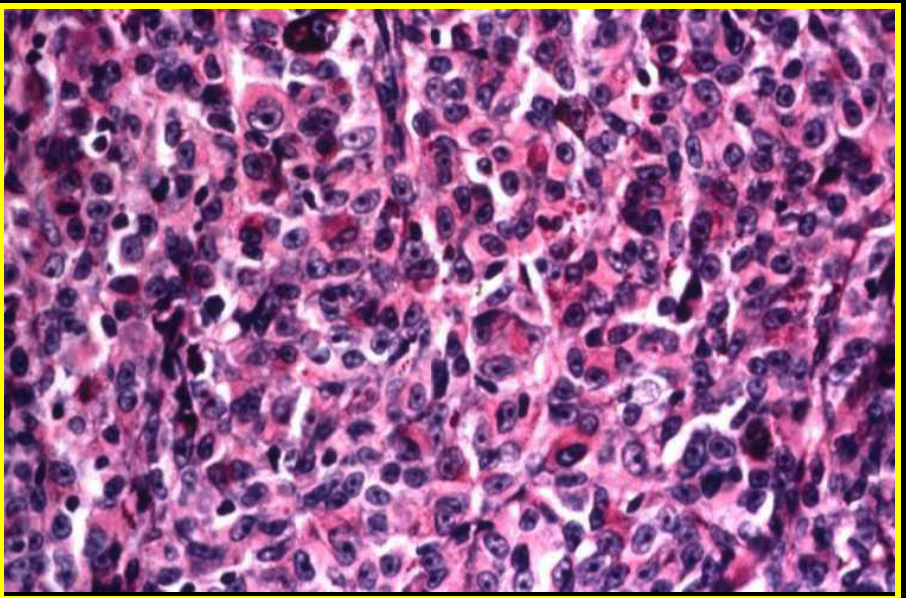
5. **Enucleation**
   - very large tumours, particularly if useful vision lost

6. **Exenteration**
   - extraocular extension

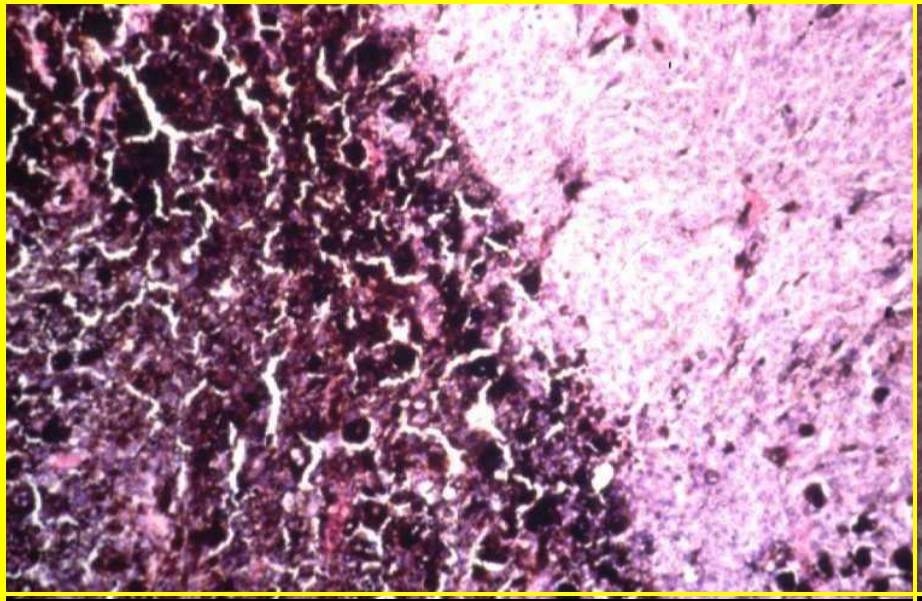
Histological classification of uveal melanomas



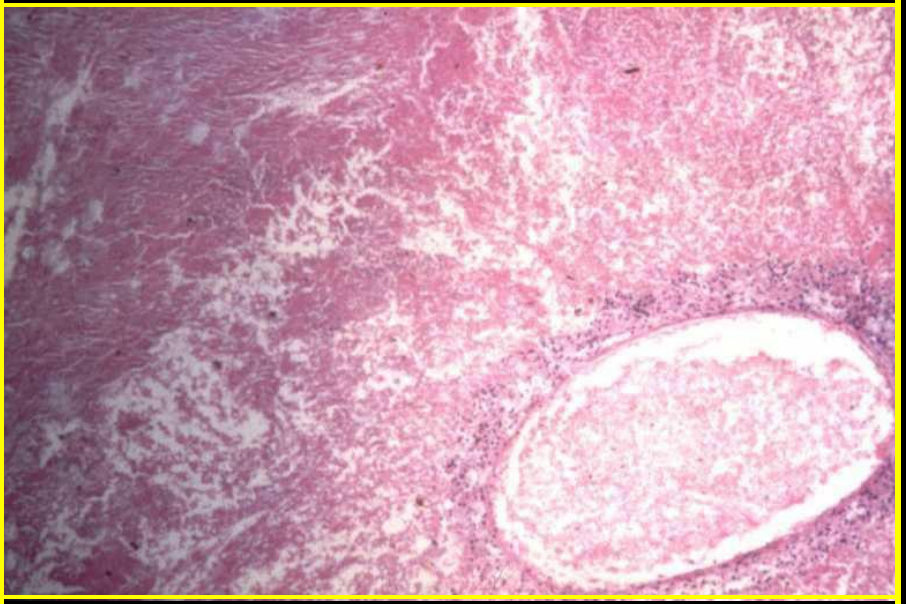
Spindle cell (45%)



Pure epithelioid cell (5%)



Mixed cell (45%)

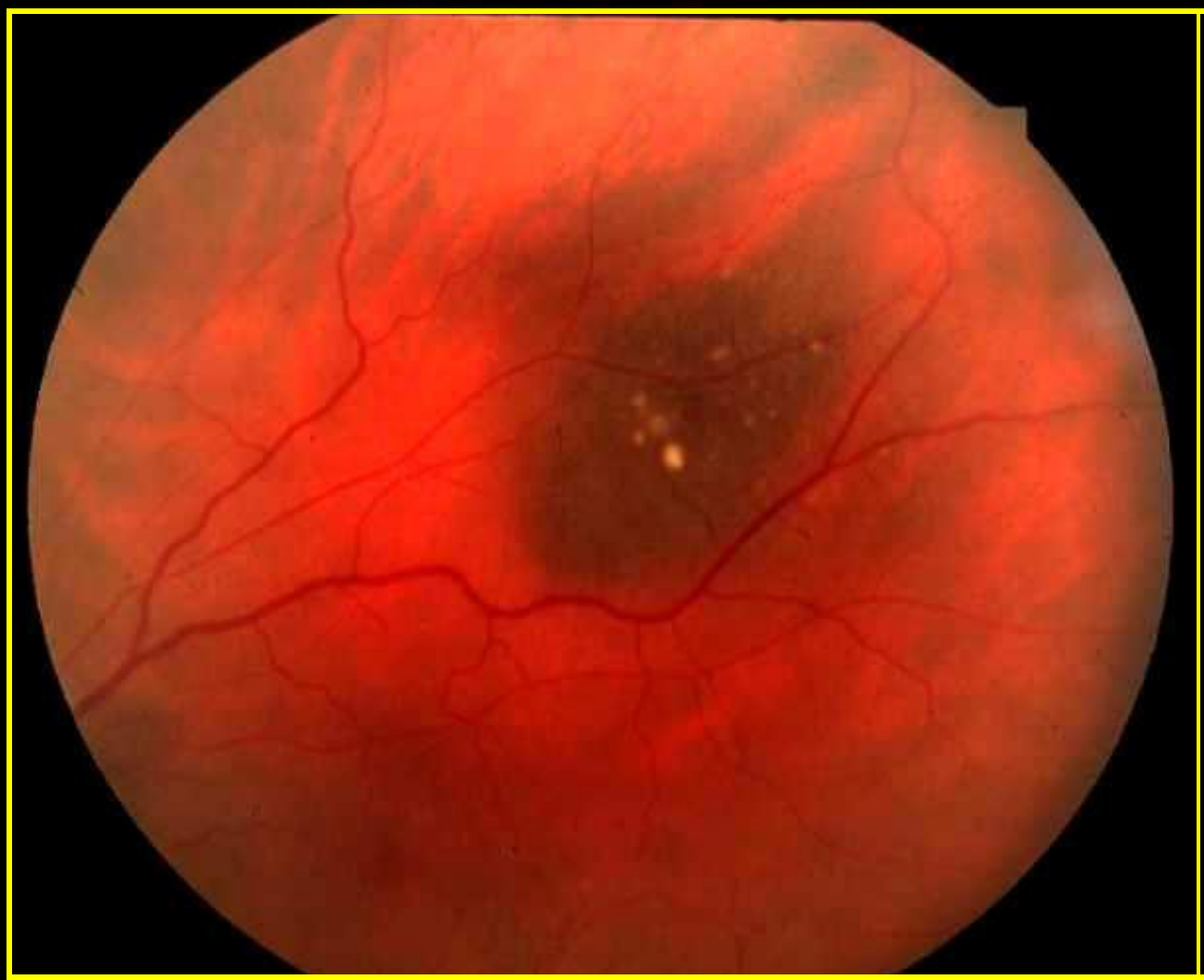


Necrotic (5%)

Poor Prognostic Factors of Uveal Melanomas

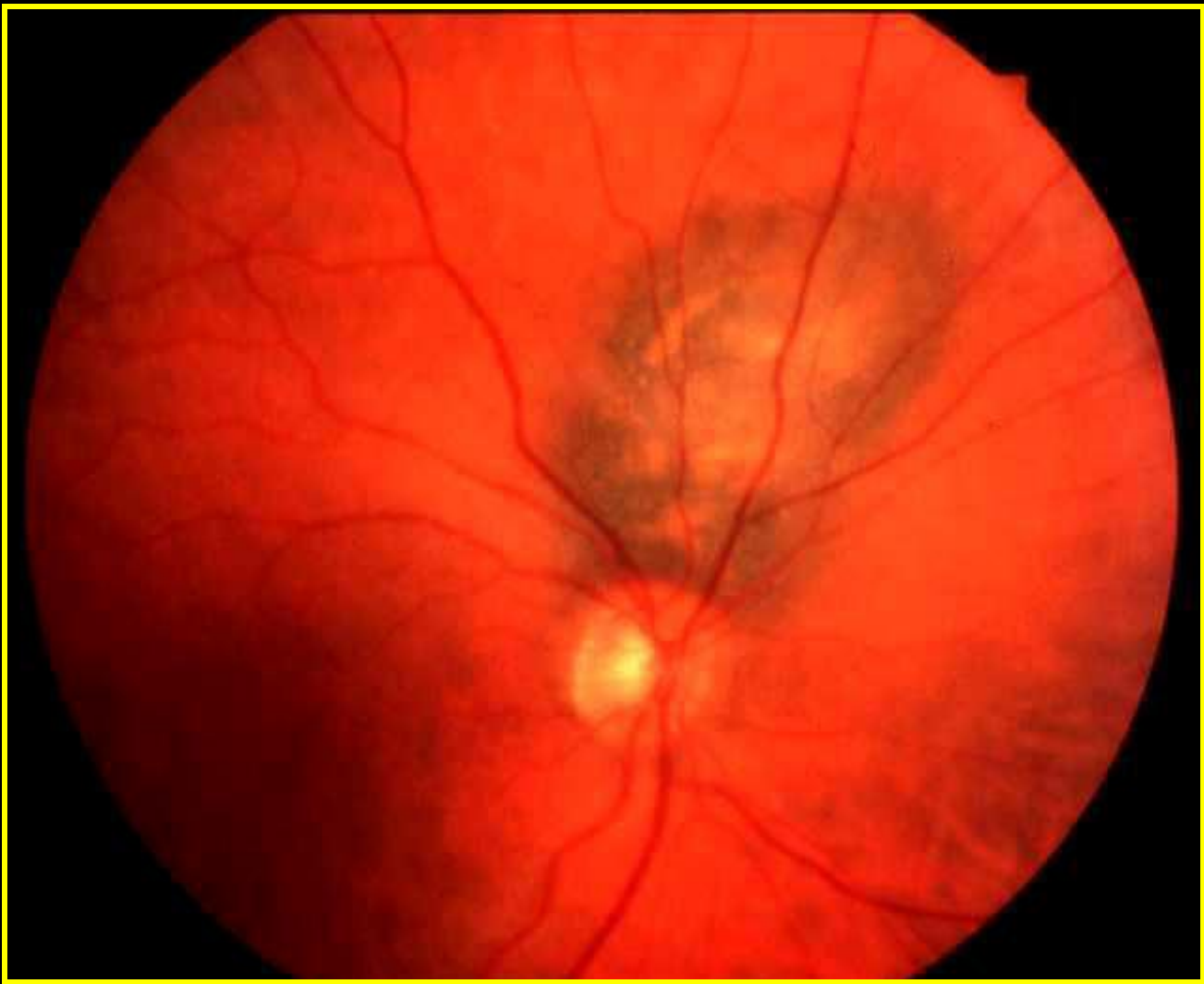
1. **Histological**
   - Epithelioid cells
   - Closed vascular loops
   - Lymphocytic infiltration
2. **Large size**
3. **Extrascleral extension**
4. **Anterior location**
5. **Age over 65 years**

Typical choroidal naevus



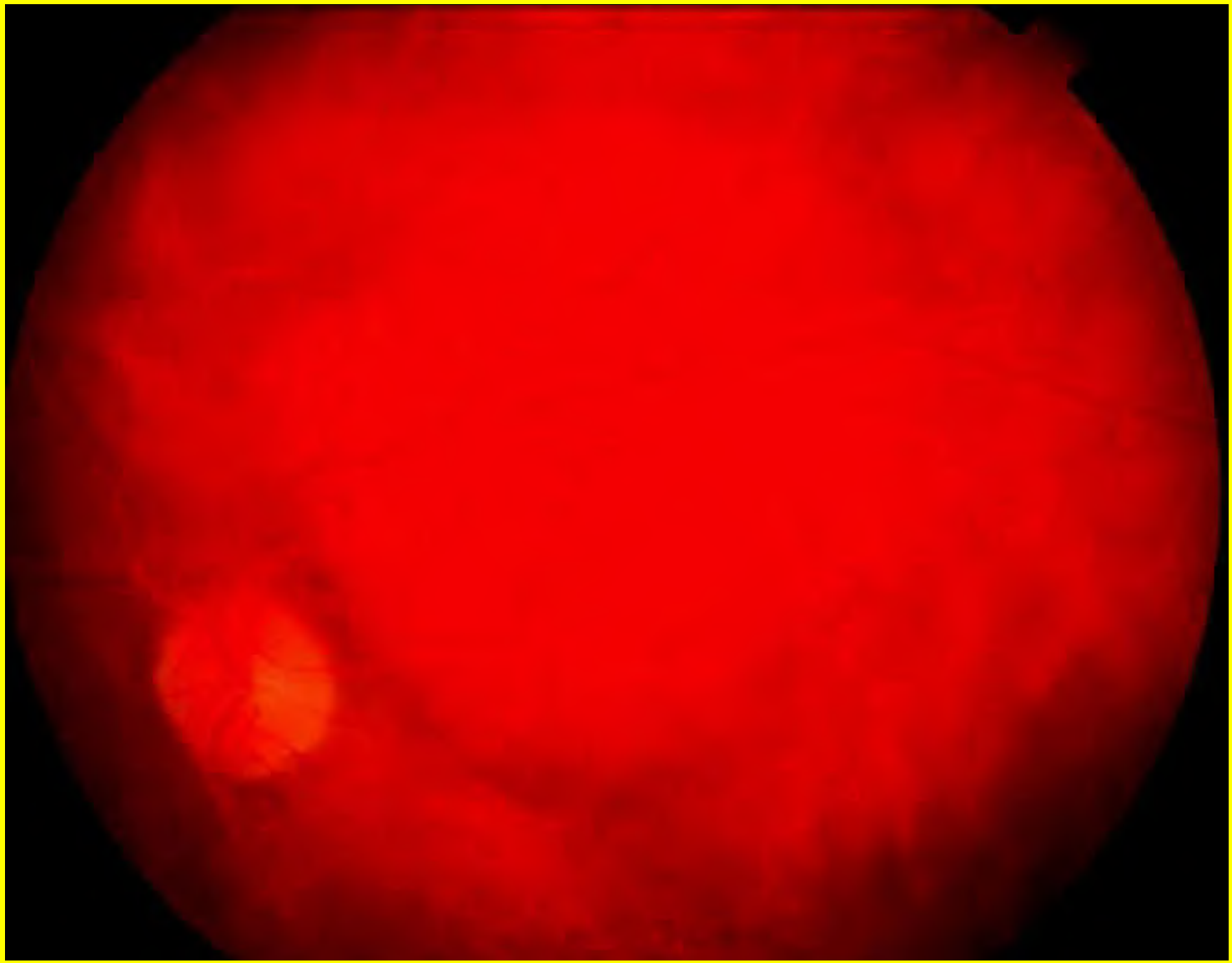
- Common - 2% of population
- Round slate-grey with indistinct margins
- Surface drusen
- Flat or slightly elevated
- Diameter less than 5 mm
- Location - anywhere
- Asymptomatic

Suspicious choroidal naevus



- Diameter more than 5 mm
- Elevation 2 mm or more
- Surface lipofuscin
- Posterior margin within 3 mm of disc
- May have symptoms due to serous fluid

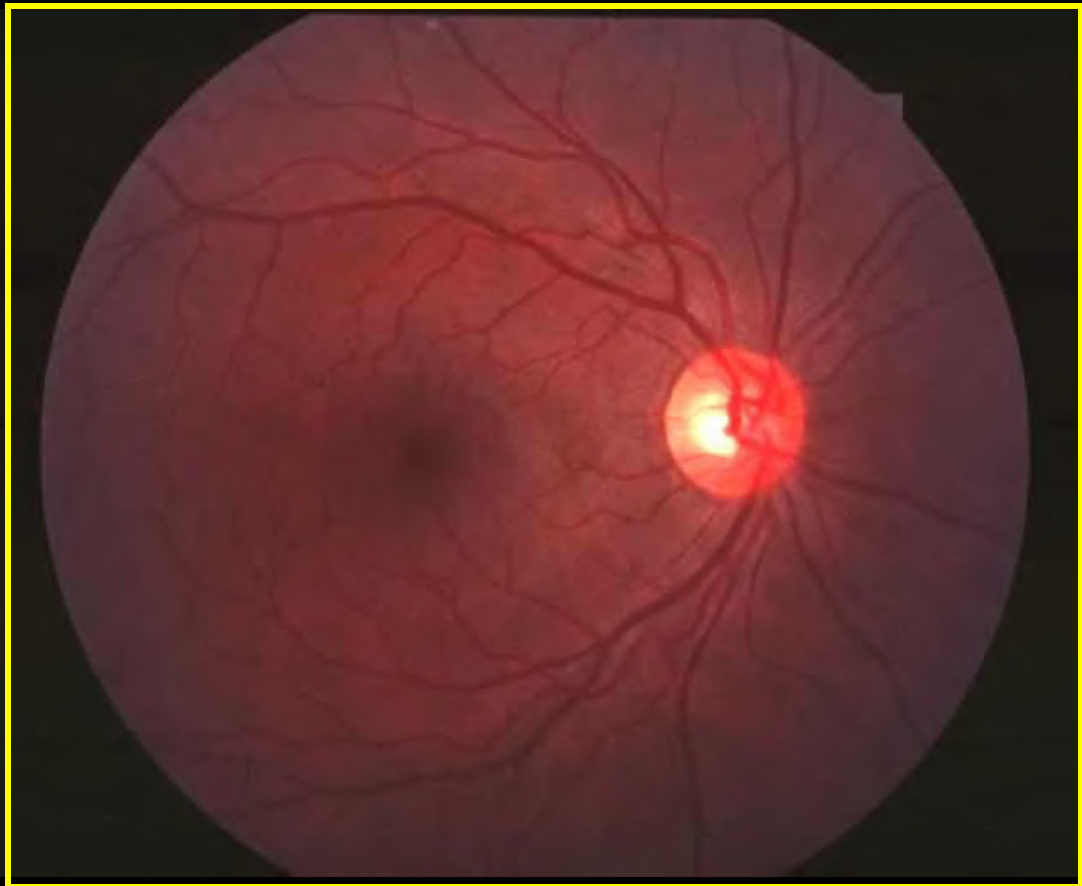
Circumscribed choroidal haemangioma



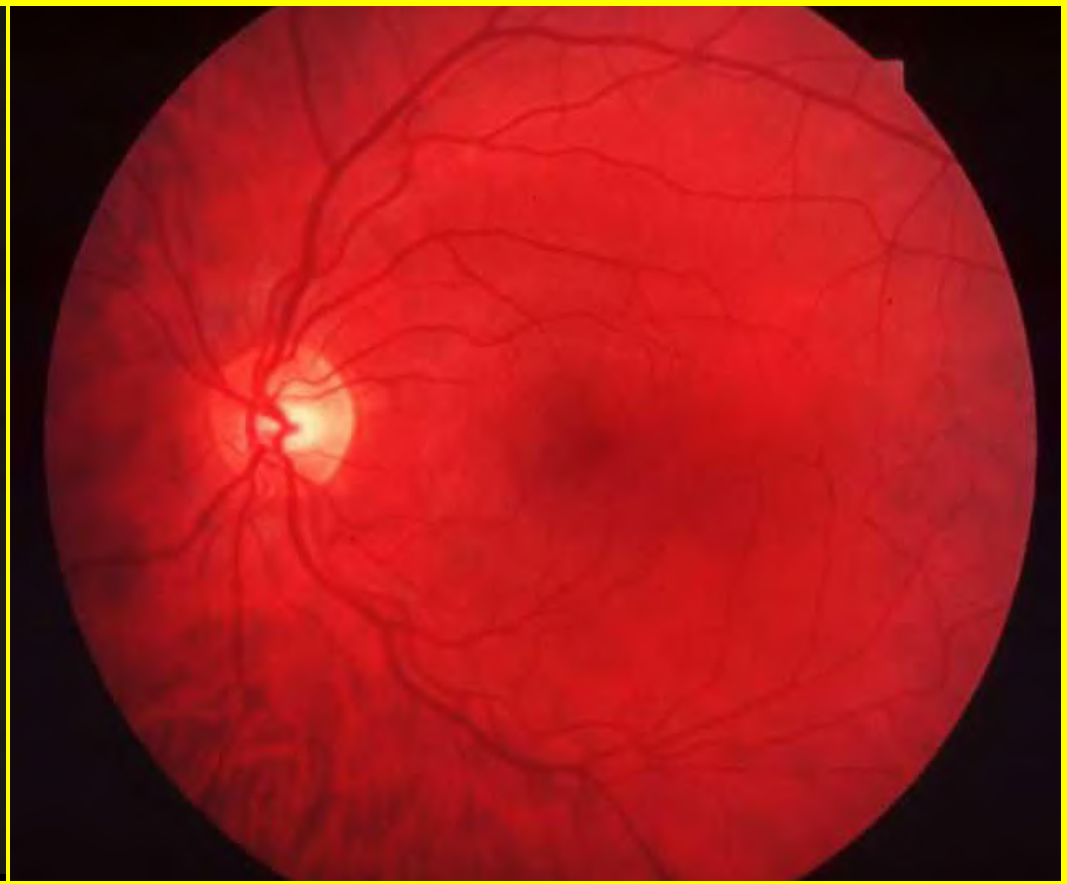
- Presentation - adult life
- Dome-shaped or placoid, red-orange mass
- Commonly at posterior pole
- Between 3 and 9 mm in diameter
- May blanch with external globe pressure
- Surface cystoid retinal degeneration
- Exudative retinal detachment
- Treatment - radiotherapy if vision threatened

Diffuse choroidal haemangioma

Typically affects patients with Sturge-Weber syndrome



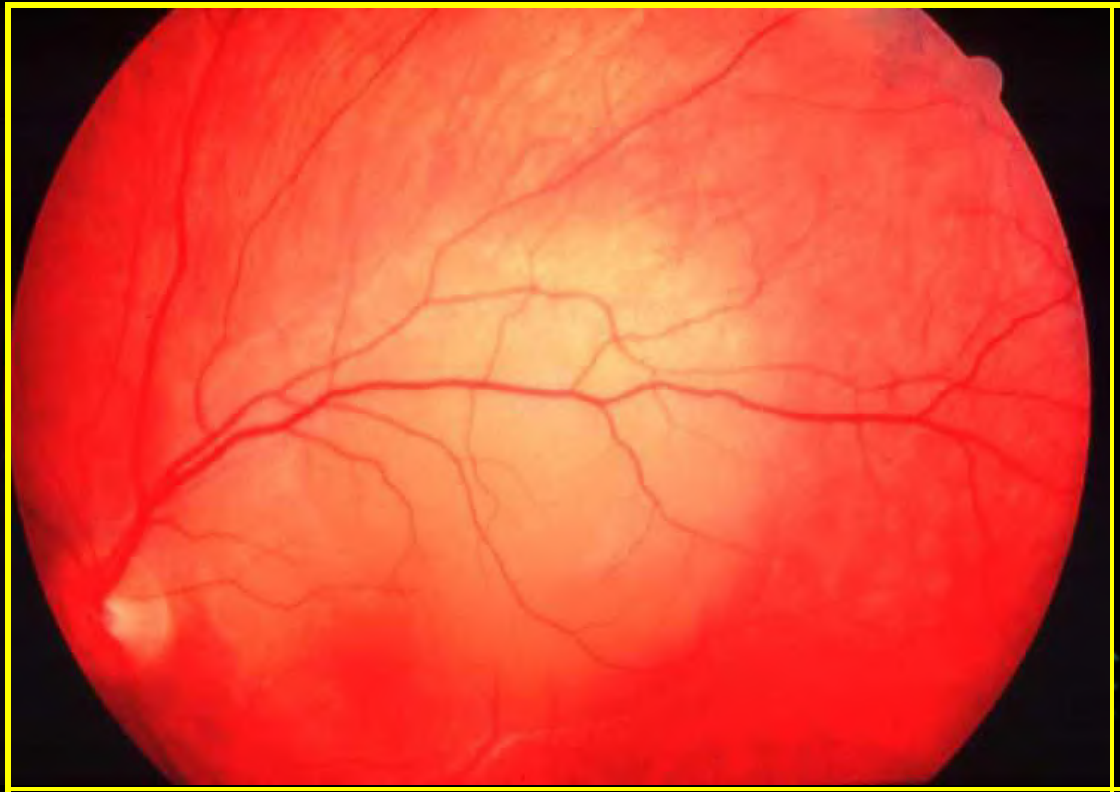
Can be missed unless compared with normal fellow eye as shown here



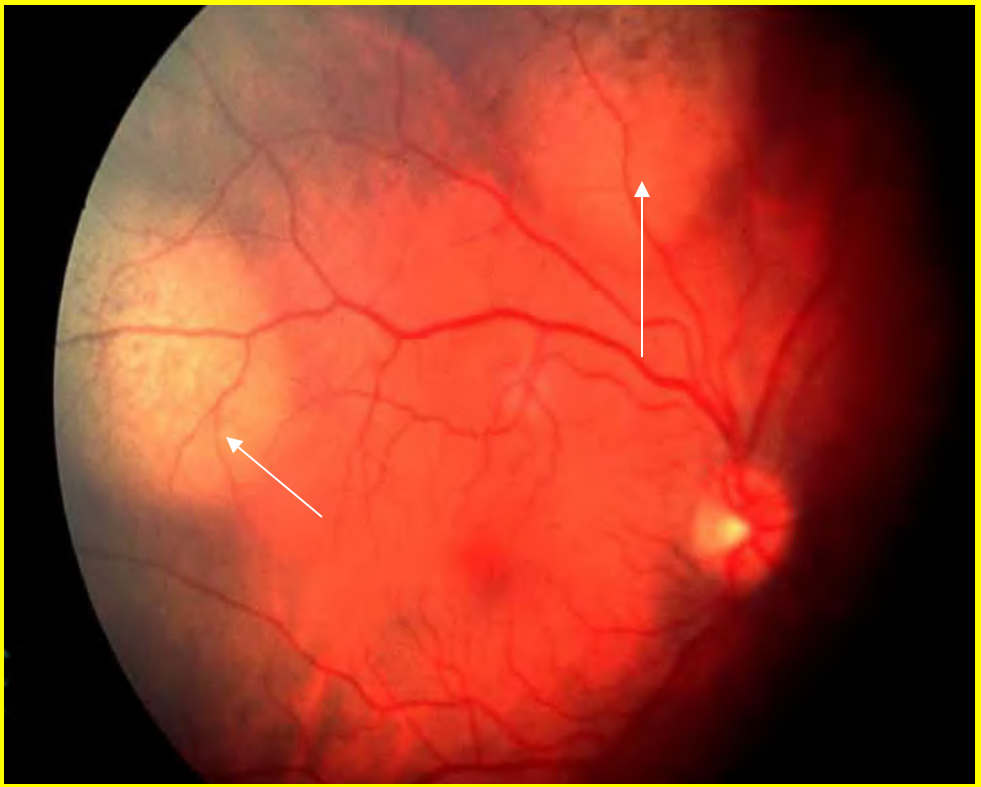
Diffuse thickening, most marked at posterior pole

Choroidal metastatic carcinoma

Most frequent primary site is breast in women and bronchus in both sexes

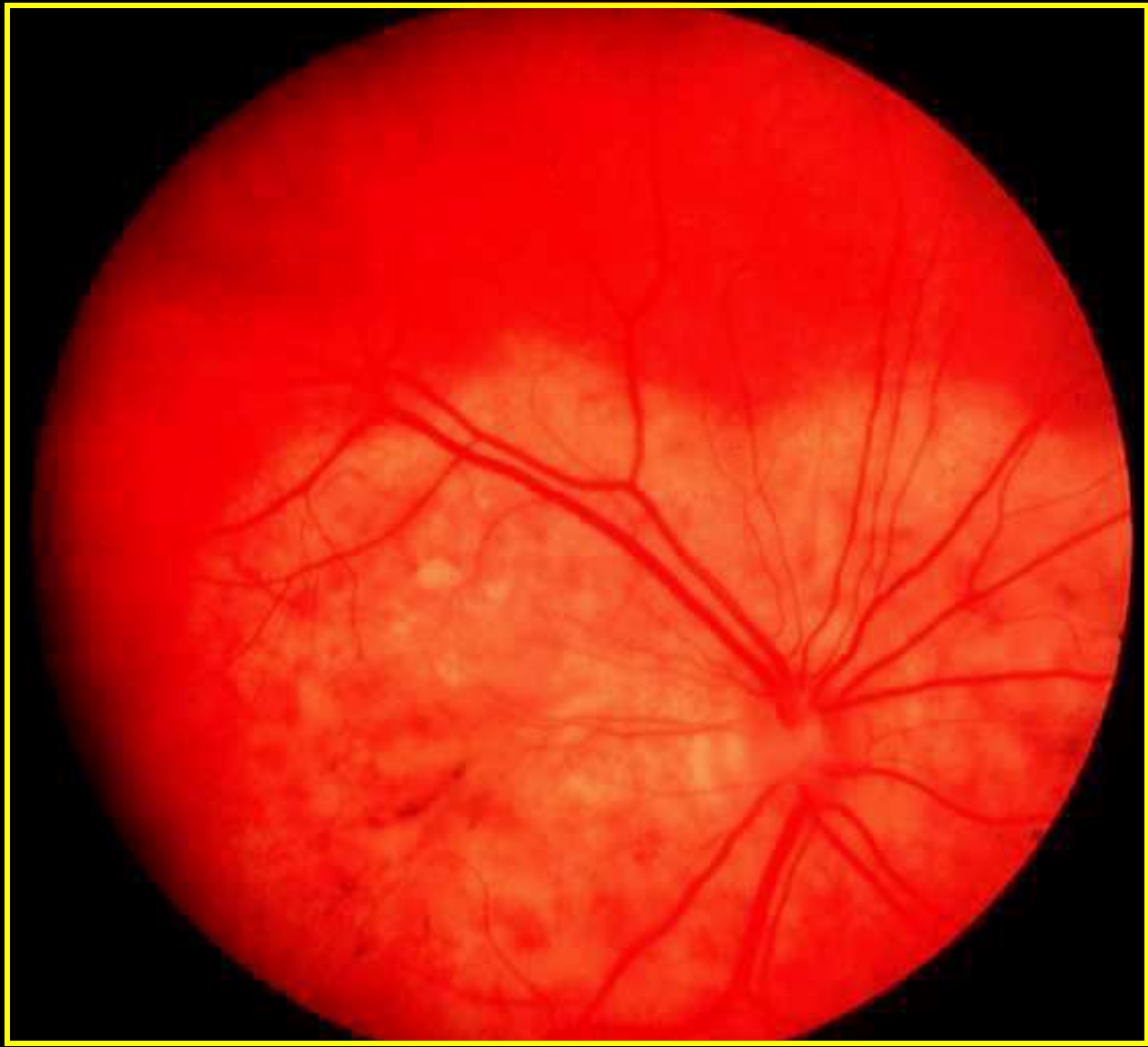


- **Fast-growing, creamy-white, placoid lesion**
- **Most frequently at posterior pole**



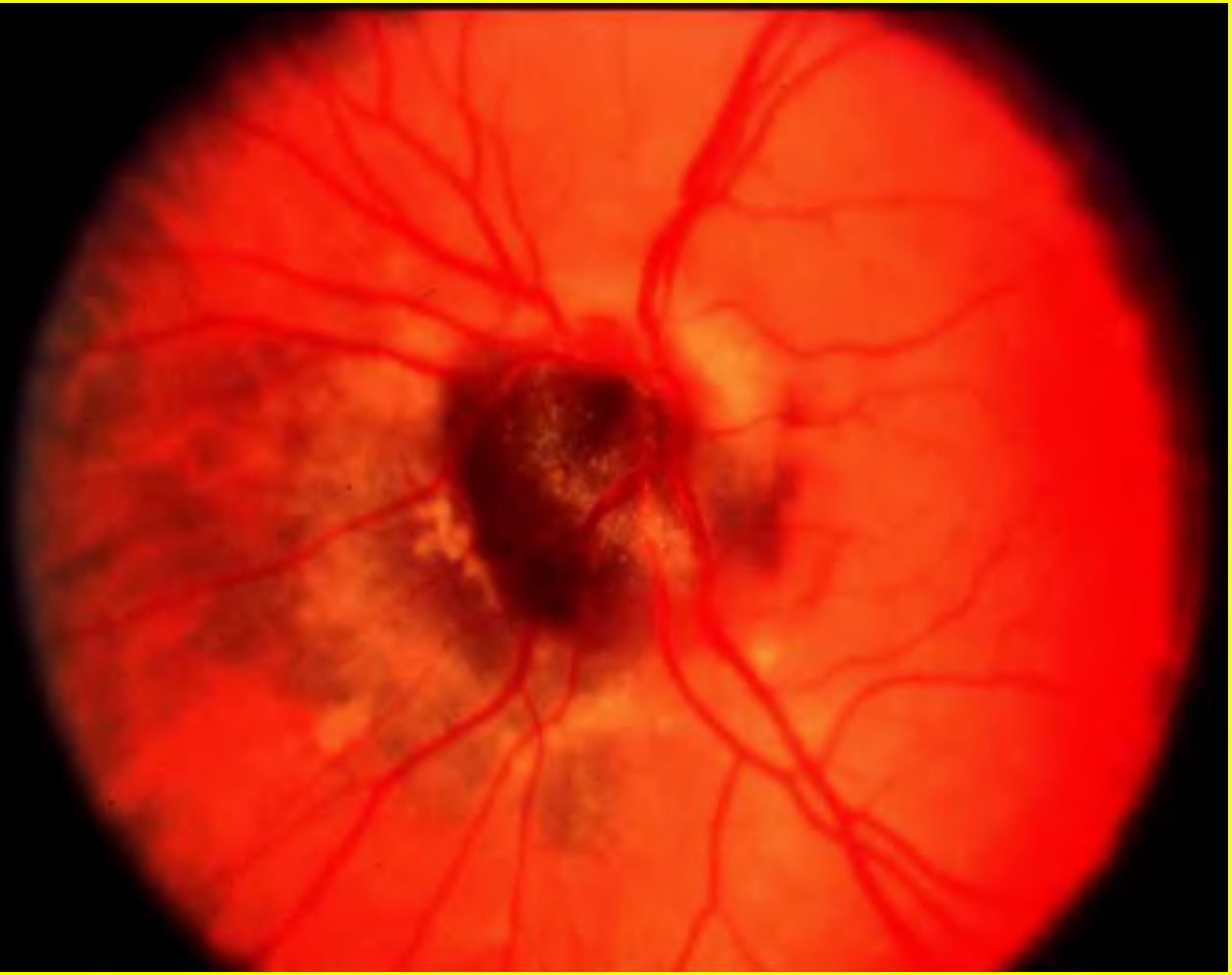
- **Deposits may be multiple**
- **Bilateral in 10-30%**

Choroidal osseous choristoma



- Very rare, benign, slow-growing ossifying tumour
- Typically affects young women
- Orange-yellow, oval lesion
- Well-defined, scalloped, geographical borders
- Most commonly peripapillary or at posterior pole
- Diffuse mottling of RPE
- Bilateral in 25%

Melanocytoma



- Affects dark skinned individuals
- Usually asymptomatic
- Most frequently affects optic nerve head
- Black lesion with feathery edges

Introduction and classification of glaucoma
and
Congenital Glaucoma

Associate Professor Dr Afzal Qadir

Pediatric ophthalmologist

Eye unit HMC

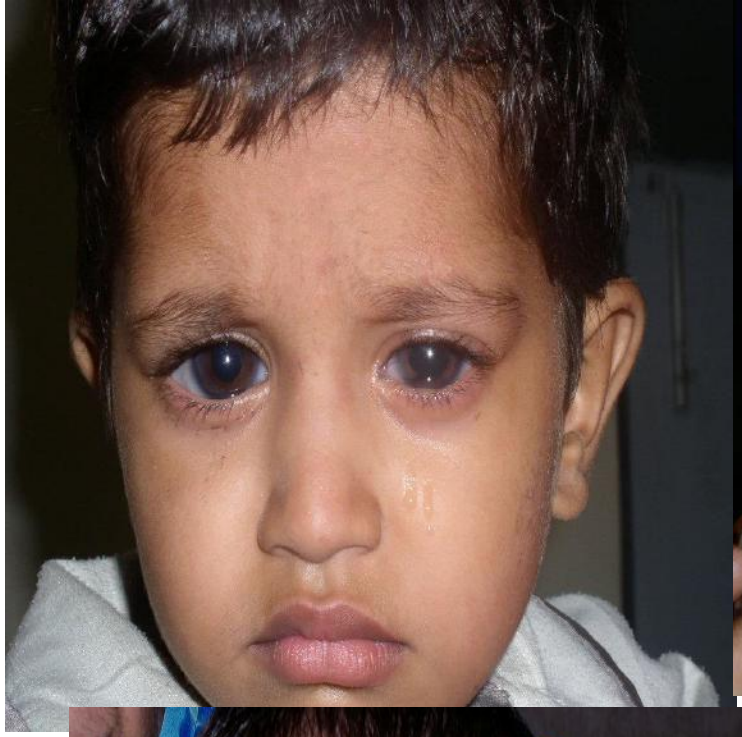
Acknowledgement

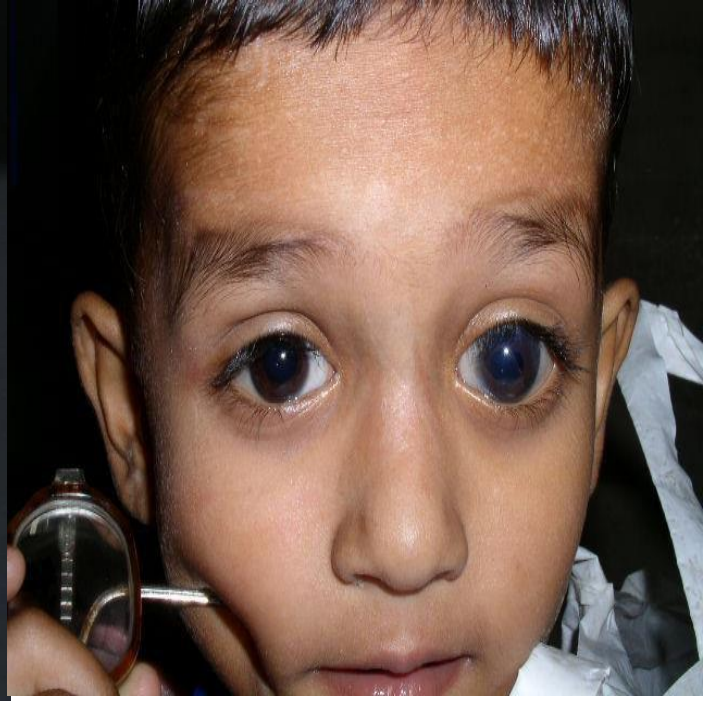
- Becker- Schaffer's Diagnosis and therapy of The Glaucomas (8th Edition).
- Kanski's Clinical Ophthalmology (8th Edition).
- Comprehensive Ophthalmology (A.K.Khurana) (7th Edition).

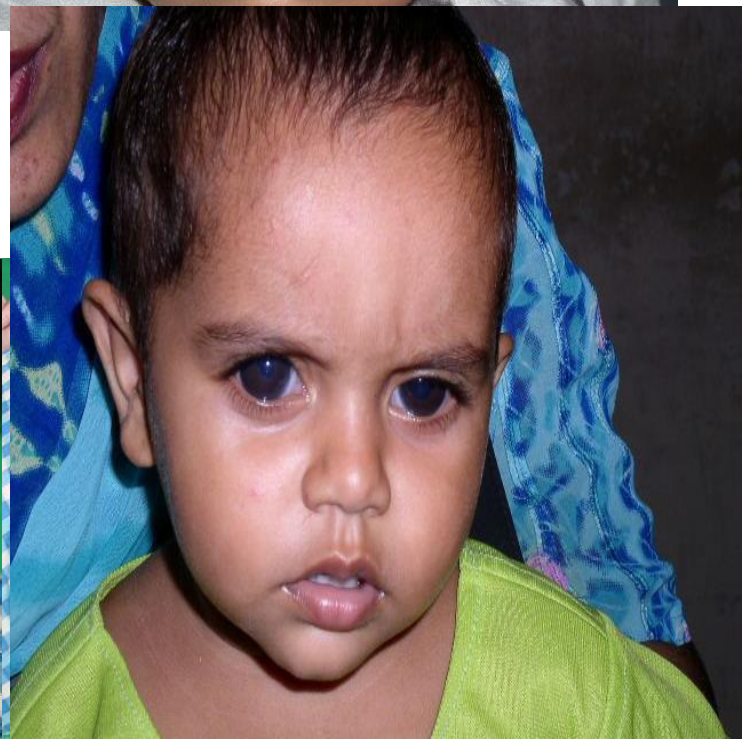
Learning Objectives

- **At the end of this class the students shall be able to :**
- Define and classify glaucoma.
- Define congenital glaucoma.
- Understand the pathogenesis and clinical features of congenital glaucoma.
- Understand the fundamentals of managing congenital glaucoma.

Excessive blinking +/- watering







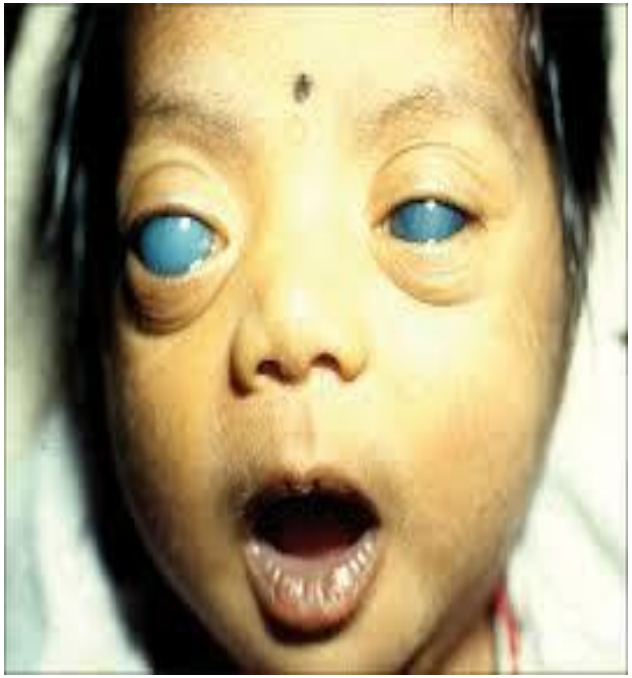
Hazy cornea
Large corneas

Question

- A child presents with watering , photophobia and an enlarged cornea with a diameter of 13mm. Examination of the eye reveals double contoured opacities concentric to the limbus. Which of the following is the most likely diagnosis:
- Superficial keratitis
- Deep keratitis
- Thyroid eye disease
- Congenital glaucoma

What is glaucoma ?

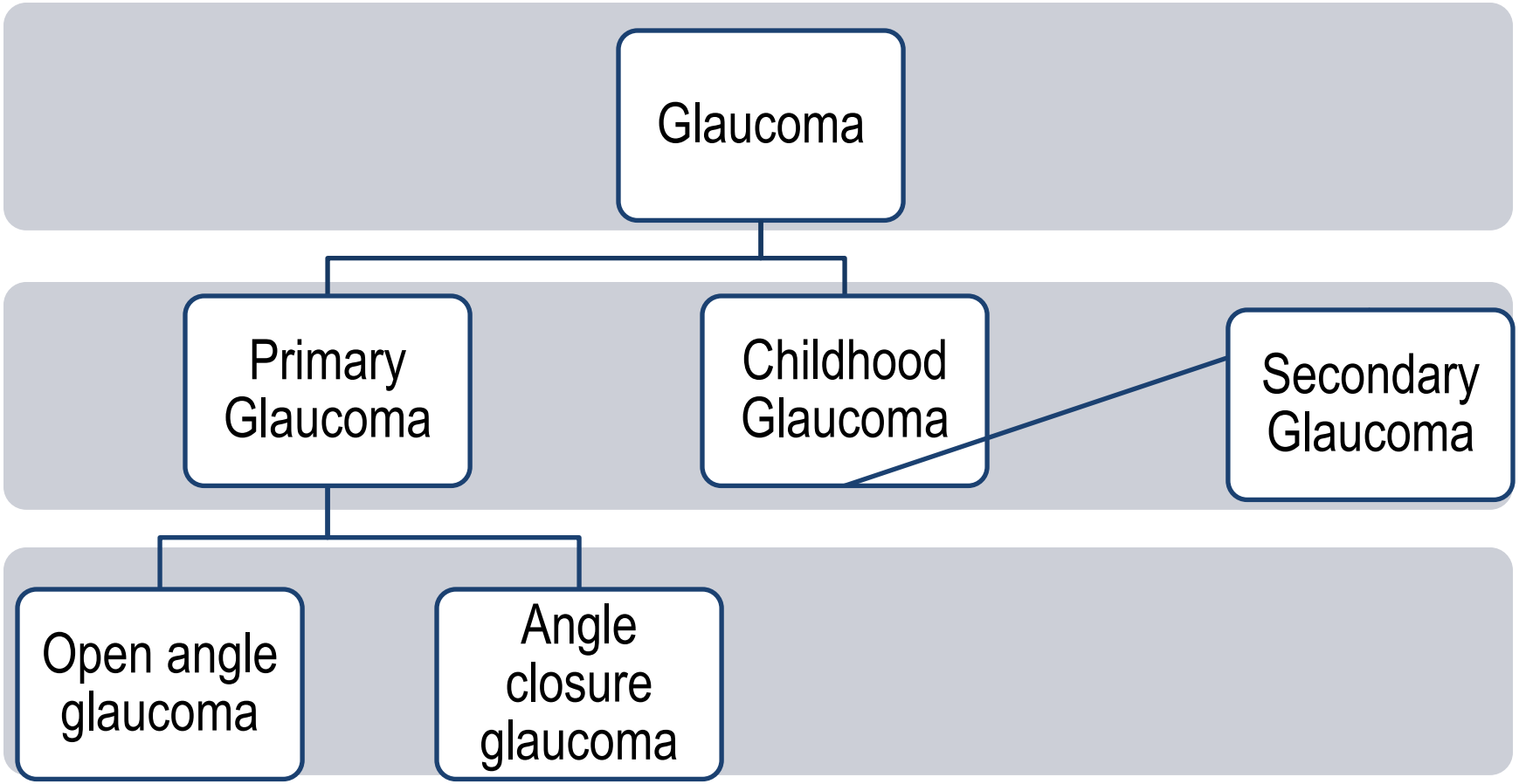
- The term glaucoma is derived from the Greek word “glaukos” meaning “gray blue”
- Second leading cause of blindness worldwide
- Third most common cause of blindness in India
- Not reversible



Definition of glaucoma

- Group of disorders characterized by progressive optic neuropathy resulting in characteristic morphological changes at the optic disc leading to a specific pattern of irreversible visual field defects (with or without a raised IOP).

Classification of glaucoma



Primary glaucoma

- Open angle glaucoma

- Primary Open angle glaucoma
- Normal Tension glaucoma
- Juvenile Open angle glaucoma
- Secondary Open angle glaucoma

- Steroid induced glaucoma
- Pigmentary glaucoma

Primary glaucoma

- Angle closure glaucoma

- Primary angle closure glaucoma
- Secondary angle closure glaucoma

- Swollen lens
- Posterior segment tumours
- Neovascular glaucoma

- Plateau iris syndrome

Childhood glaucoma

- Primary congenital glaucoma
- Glaucoma associated with ocular abnormalities
- Glaucoma associated with systemic abnormalities

Secondary glaucoma

- Glaucomas after ocular surgery
- Steroid induced glaucoma
- Traumatic glaucoma

Childhood glaucoma-Introduction

- Diverse group of disorders
- Primary congenital glaucoma-

Developmental abnormality of angle of anterior chamber leading to high intraocular pressure(IOP).

- Secondary congenital glaucoma

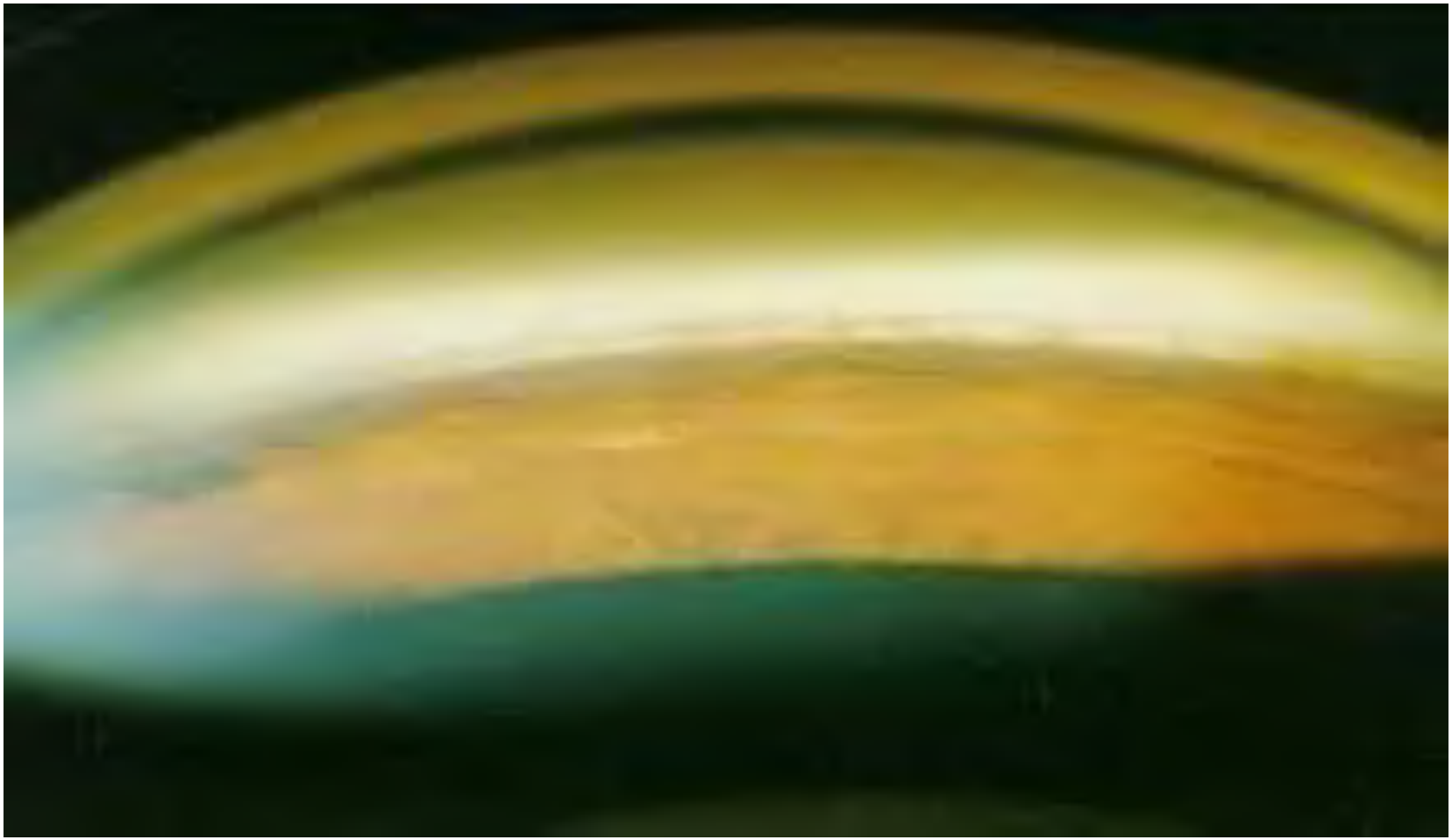
With associated ocular and systemic anomalies

ANGLE OF ANTERIOR CHAMBER

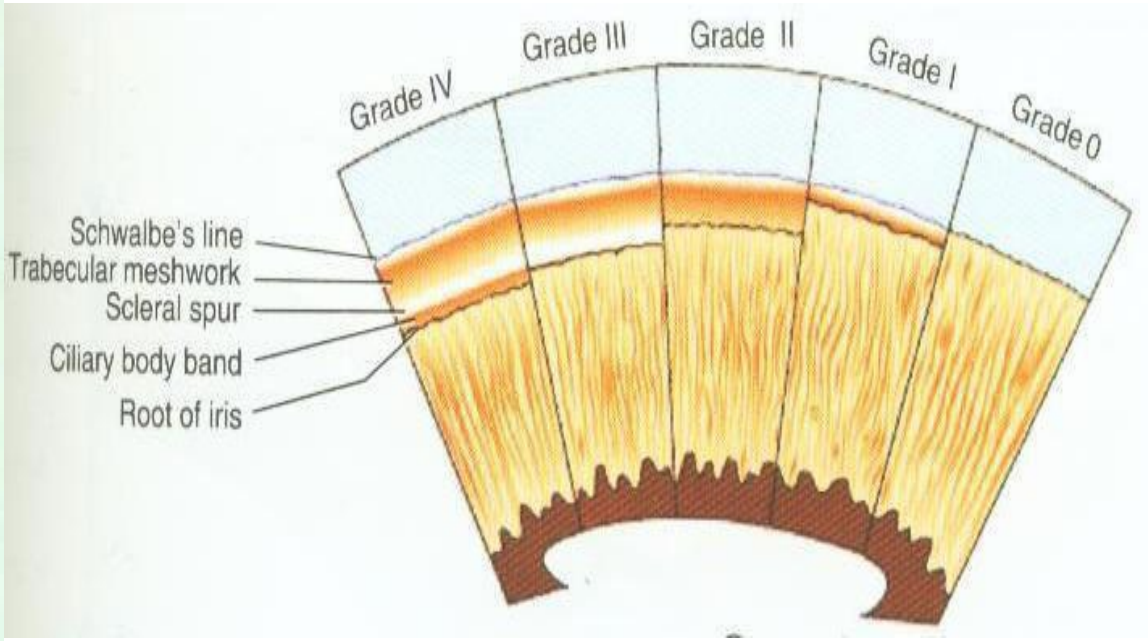
- The peripheral recess of anterior chamber is known as the angle of anterior chamber.
- It is clinically visualized by ***gonioscopy***.
- Starting at the root of iris & progressing anteriorly towards the cornea, the following structures can be identified in a normal angle in an adult :

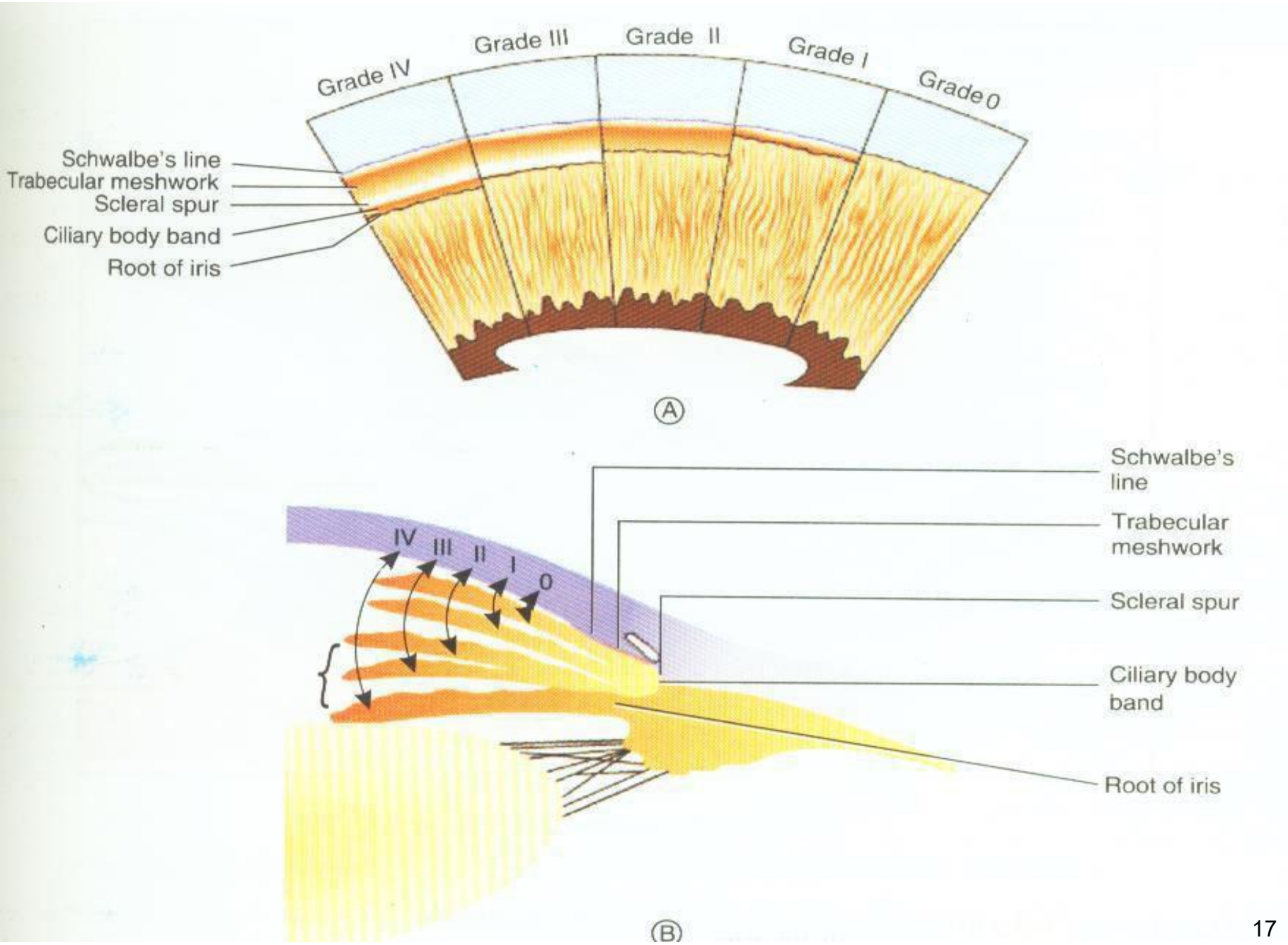
1) Ciliary body band (CBB) & root of iris
2) Scleral spur (SS)
3) Trabecular meshwork (TM)
4) Schwalbe's line (SL)

Angle of anterior chamber as seen on gonioscopy



Grade	Structures visible on gonioscopy
IV	From Schwalbe's line to ciliary body
III	From Schwalbe's line to scleral spur
II	From Schwalbe's line to trabecular meshwork
I	Schwalbe's line only
0	None of the angle structures visible




Grade IV
Grade III
Grade II
Grade I
Grade 0
Schwalbe's line
Trabecular meshwork
Scleral spur
Ciliary body band
Root of iris
A
IV
III
II
I
0
Schwalbe's line
Trabecular meshwork
Scleral spur
Ciliary body band
Root of iris
B

Childhood glaucomas

- True congenital glaucomas- At birth or during intrauterine period.
- Infantile glaucoma- Upto three years of age.
- Juvenile glaucoma- After three years of age and upto 35 years of age.

Prevalence and genetic pattern

- Sporadic occurrence in most cases (90%)
- Autosomal recessive in 10% of cases
- Loci linked with congenital glaucoma are 2p21(GLC3A), 1p36(GLC3B) and 14q24(GLC3C)
- 60% diagnosed by the age of 6 months and 80% diagnosed within the first year of life

Prevalence and genetic pattern

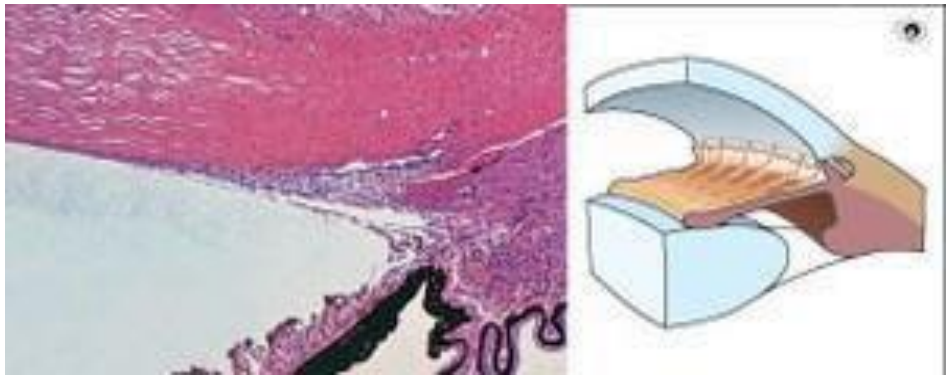
- Bilateral (about 70%) but asymmetrical
- Boys are affected slightly more frequently than girls (65%)
- Prevalence is 1 in 10,000 births
- Chance of second sibling having disease is 3%
- Chance of third sibling (of two affected siblings) having disease is 25%

Pathogenesis

- Faulty development of angle of anterior chamber from neural crest derived cells (trabeculodysgenesis)
- Absence of angle recess with flat/concave iris insertion.

The normal chamber angle: on the left is a histological cross-section; on the right is a drawing of the same

- Impaired aqueous outflow
- Elevated IOP

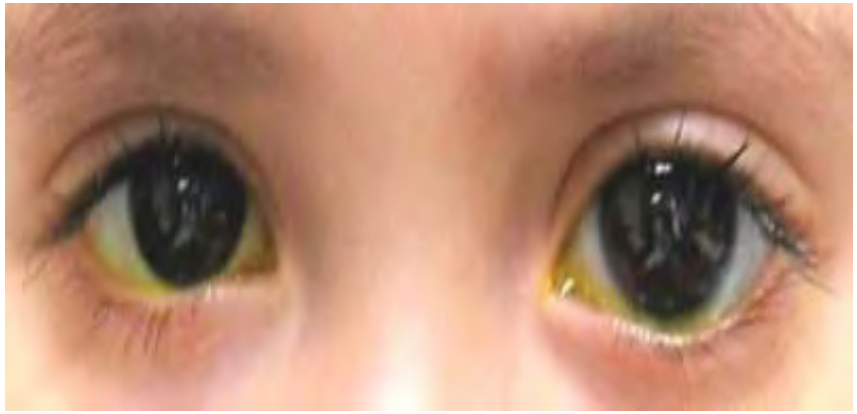


An underdeveloped chamber angle

Clinical presentation

Classic triad of

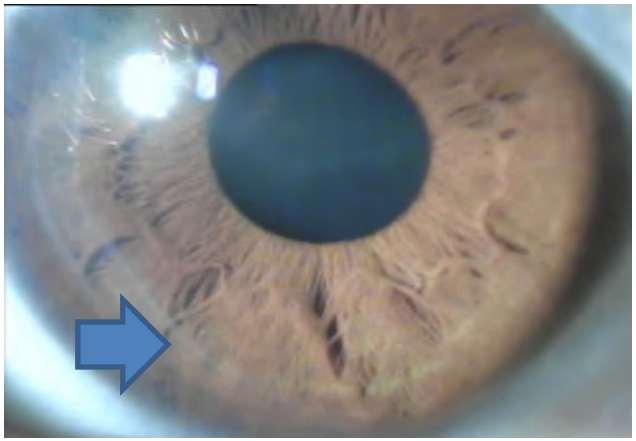
- **Epiphora**
- **Blepharospasm**
- **Photophobia**

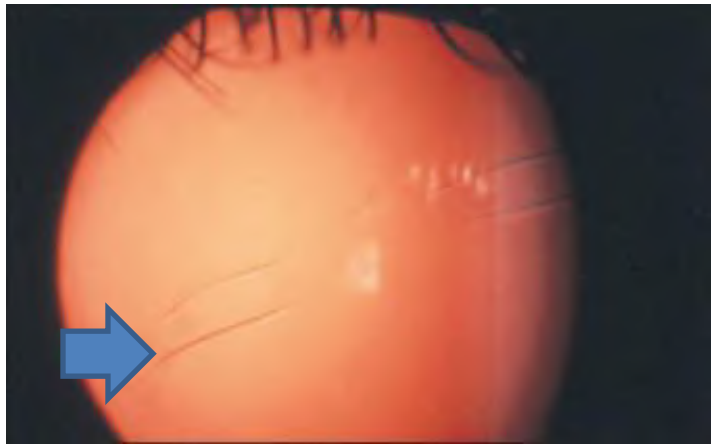


- Babies rub their eyes
- Enlarged eyes
- Vision impaired

Corneal signs

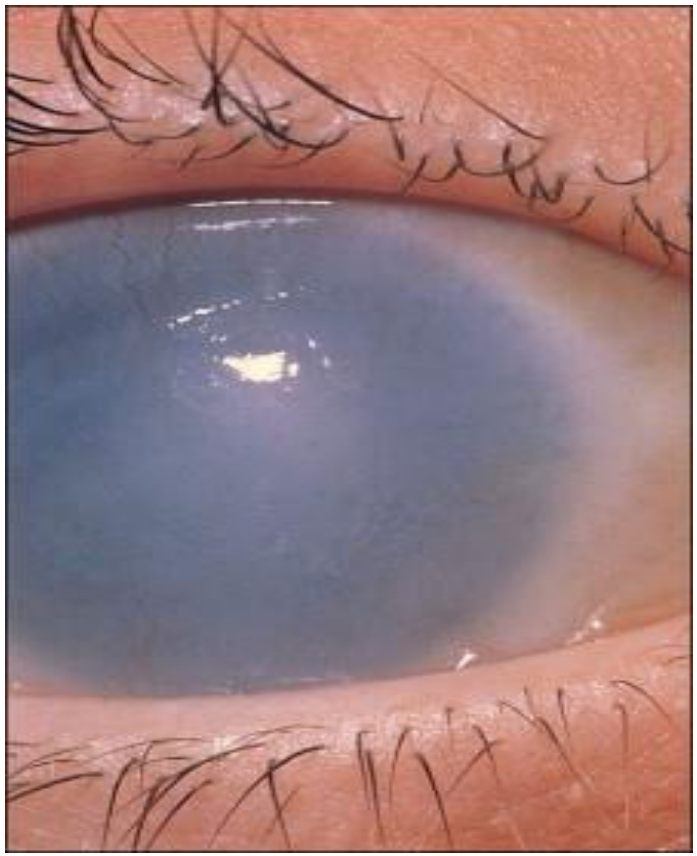
- Corneal oedema
- Corneal enlargement (Corneal diameter>13mm)
- Haab's striae : Descemet's membrane is not very elastic and stretching may result in small linear/circumferential tears that cause a certain degree of corneal opacification.





Clinical presentation

- Buphthalmos: Enlargement of the globe as a result of elevated IOP. All segments of the outer eye especially the cornea and **sclera** expand principally at the corneoscleral junction
- The anatomic landmarks are displaced.
- The anterior chamber is deep



Advanced developmental glaucoma with extensive enlargement and scarring of the cornea.

Clinical presentation

- Sclera becomes thin and appears blue (due to underlying uveal tissue
- Iris- atrophic in later stages
- Optic disc- variable cupping
- Intraocular pressure(IOP)- raised
- Axial myopia- due to increased axial length of eyeball

Examination under anaesthesia

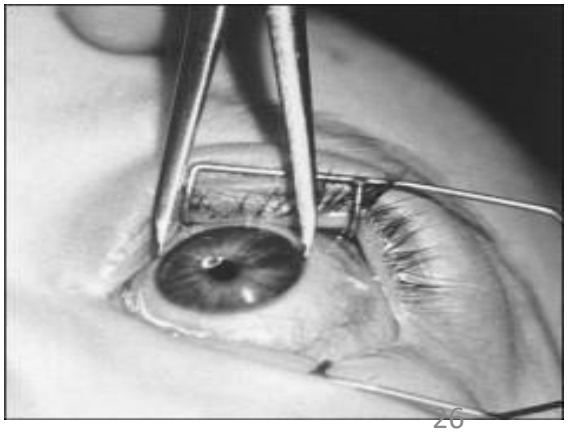
- Mandatory in all cases
- Includes :
- Measurement of IOP –

  Perkins tonometer/Tonopen

  (Normal 10-21 mm Hg)

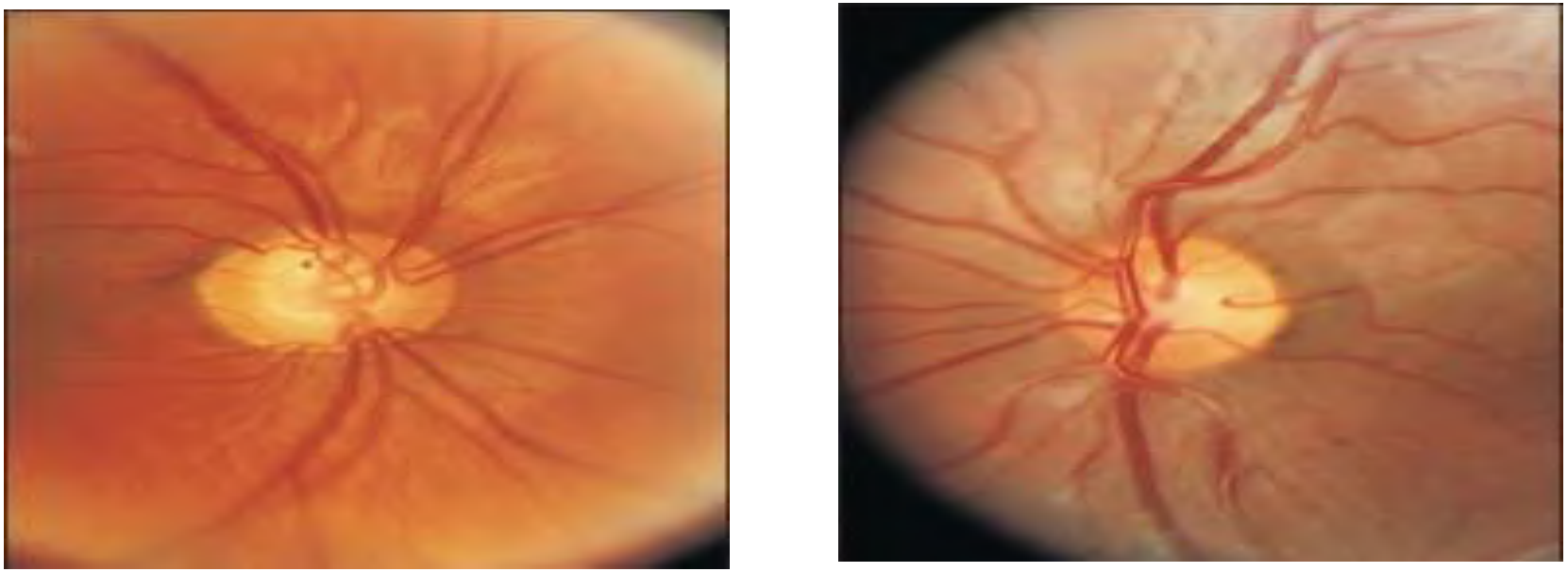
- Measurement of corneal diameter – by callipers

  (Normal 9.5mm-10.5mm)



Examination under anaesthesia

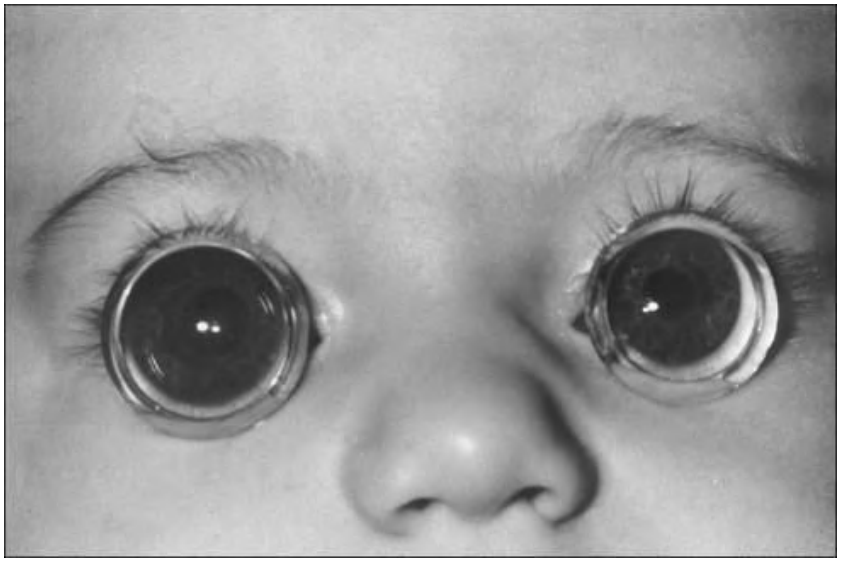
- Slit lamp examination- with portable slit lamp
- Ophthalmoscopy- to evaluate optic disc



Asymmetric disc cupping in a child with developmental glaucoma. (A) Note steep-walled cup. This is typical of glaucomatous cupping in the elastic infant eye. (B) The left eye has no cupping.

Examination under anaesthesia

- Direct Gonioscopy – to examine angle of anterior chamber
- Koeppe's gonioscopy lens is preferable
- Angle is open but immature in congenital glaucoma



Differential Diagnosis

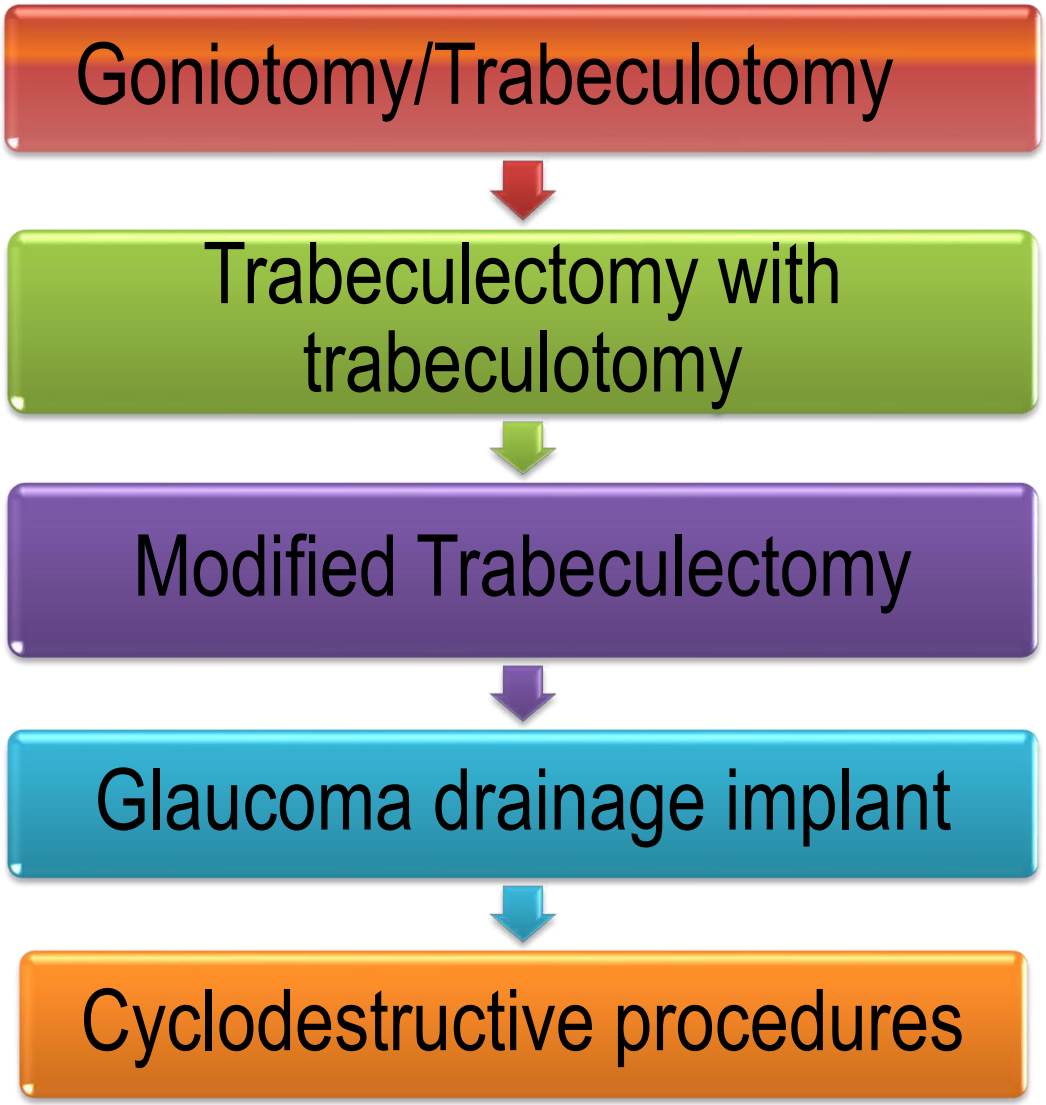
- **Hazy/Cloudy cornea----**
- STUMPED (Sclerocornea, Trauma, Ulcer, Metabolic disorders, Peter's anomaly, Endothelial dystrophy)
- **Watering and intolerance to light-----**

  Congenital Naso Lacrimal Duct obstruction

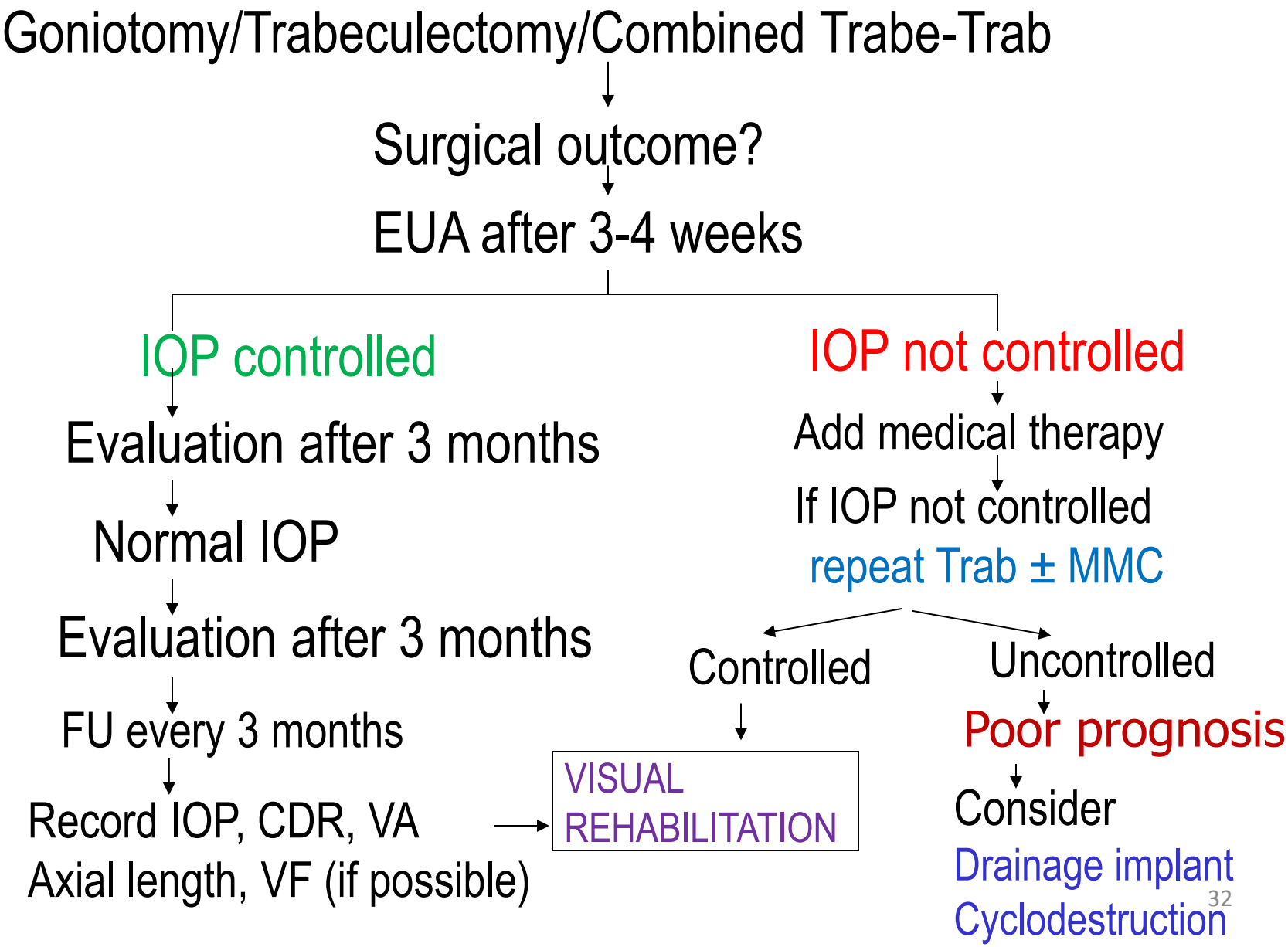
  keratitis, conjunctivitis
- **Optic cupping** ---- disc coloboma, hypoplasia, physiological cupping
- **Corneal enlargement** -- megalocornea, high myopia
- **Descemet's breaks** --- Forceps delivery ,birth trauma

Management

- Glaucoma surgery is the primary option
- Medications are not very effective
- Role of medical management is temporary, till surgery is taken up.
- Beta blockers (Timolol), hyperosmotic agents(Mannitol), carbonic anhydrase inhibitors (acetazolamide/dorzolamide)
- Miotics and Alpha-2 agonists are not used in children.

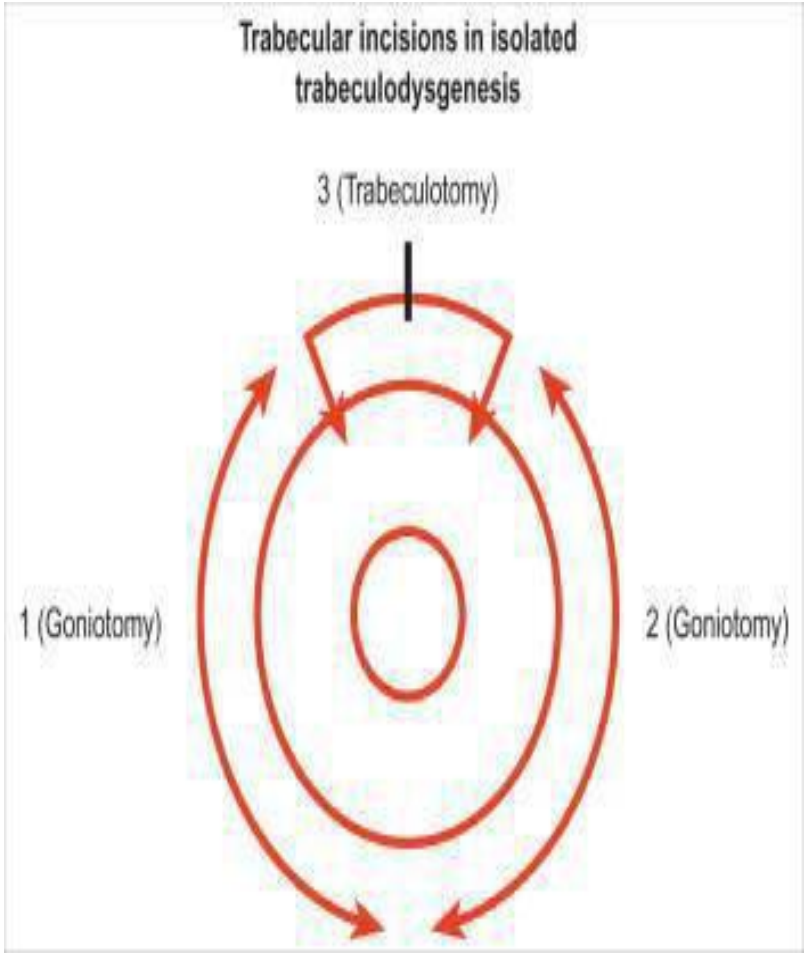

Goniotomy/Trabeculotomy
Trabeculectomy with trabeculotomy
Modified Trabeculectomy
Glaucoma drainage implant
Cyclodestructive procedures

Approach to management



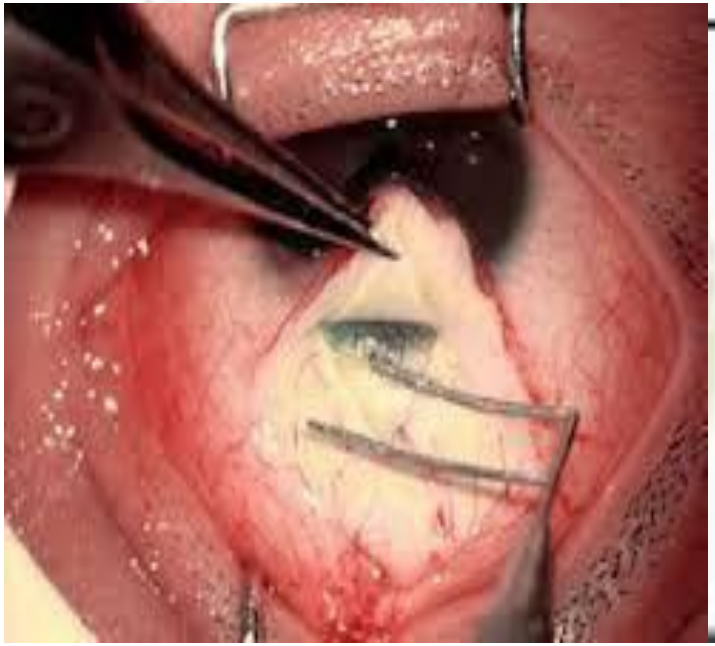
Goniotomy

- Safe procedure when performed skilfully.
- Performed with direct visualization of trabecular meshwork
- Aims to transect Schlemm's canal by ab-interno approach
- Incises only superficial trabecular tissues, necessary to cure this disease



Trabeculotomy

- Ab-externo trabeculotomy has good success rates.



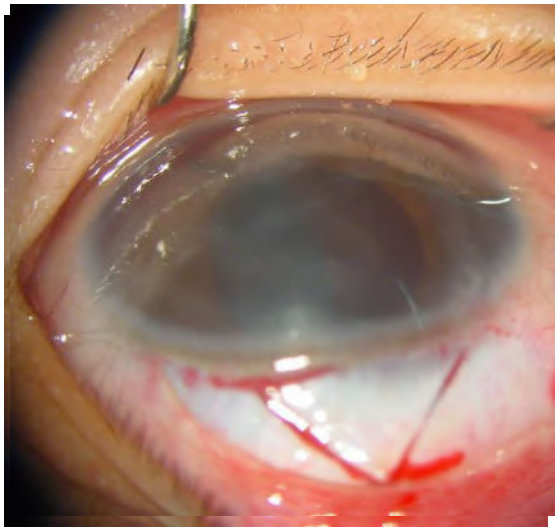
Trabeculotome

Trabeculotomy with trabeculectomy

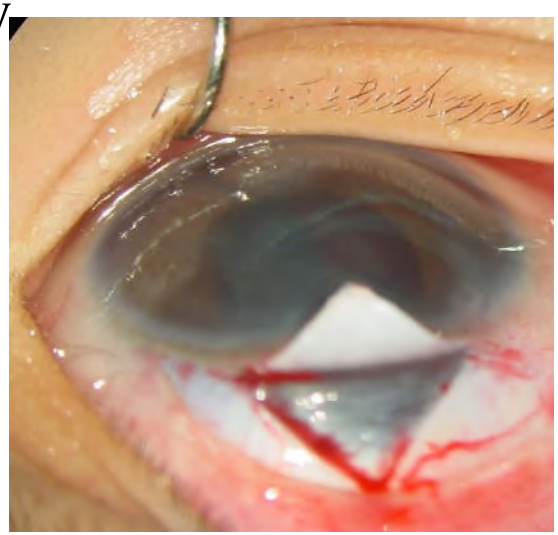
- Most commonly performed surgery in India

- Easy adaptability
- Safe and successful
- Suitable in compromised corneas
- More predictable results

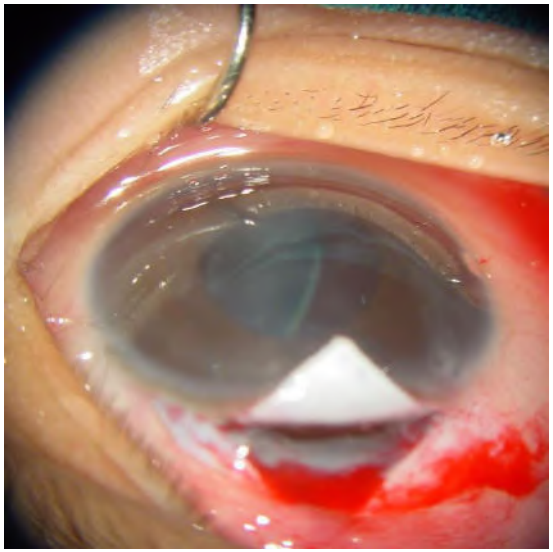
Steps of Trabeculectomy with trabeculotomy



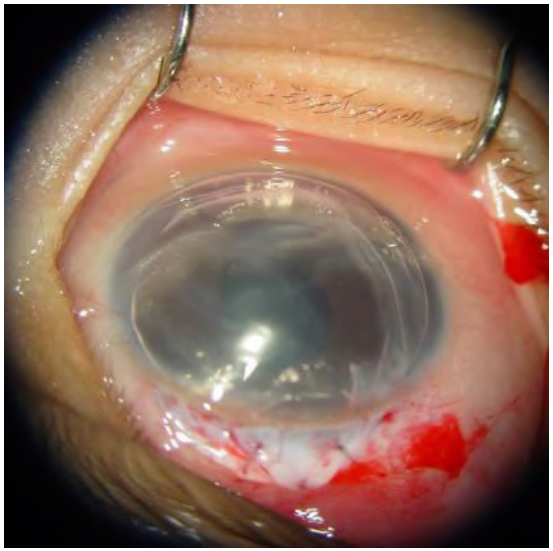
Scleral flap



Dissection upto grey limbus



Trabecular meshwork cut

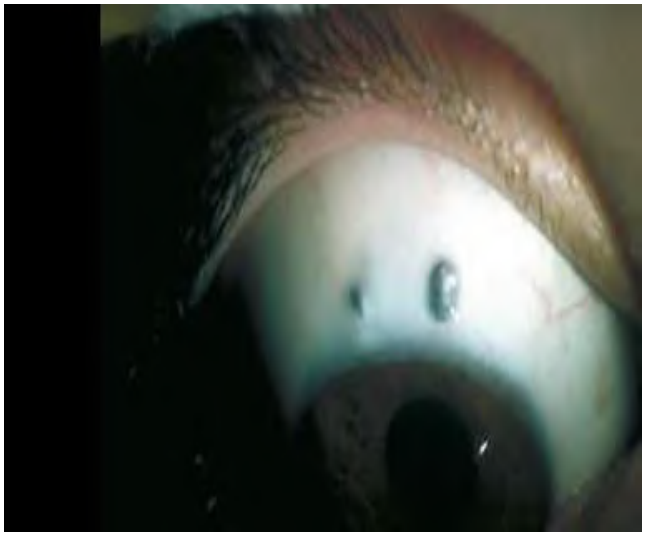


Diffuse subconjunctival bleb

Role of antimetabolites in paediatric glaucoma

- Significantly more complications associated with the use of Mitomycin(MMC) in paediatric glaucomas

- Thin, avascular filtering blebs
- Wound leakage
- Choroidal detachment
- Bleb related endophthalmitis

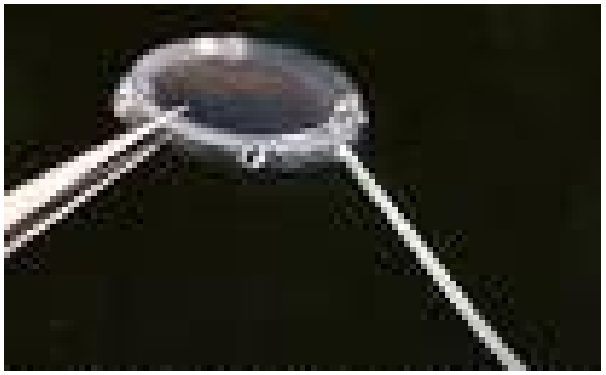


Options for refractory glaucoma ?

- Glaucoma Drainage Devices
- Cyclo-destruction

What is a Glaucoma Drainage Device?

Glaucoma drainage devices (GDDs) create an alternate aqueous pathway from the anterior chamber (AC) by channeling aqueous out of the eye through a tube to a subconjunctival bleb. This tube is usually connected to an equatorial plate under the conjunctiva.



Cyclodestructive procedures

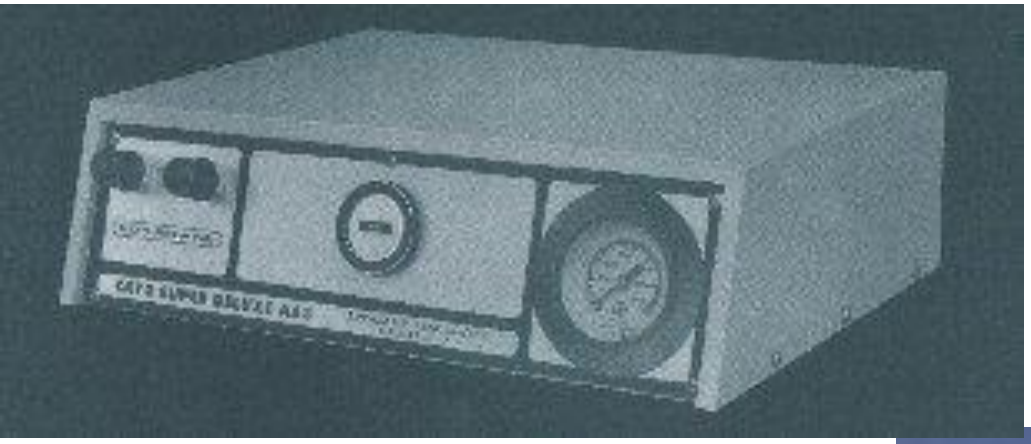
- Cyclocryotherapy

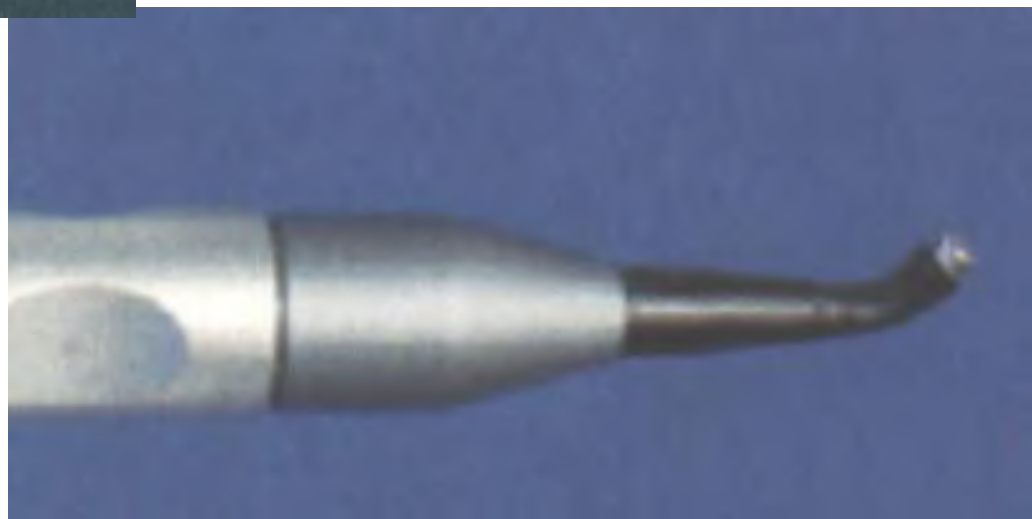
- Cyclophotocoagulation

  - Transscleral
  - Transpupillary
  - Endoscopic

CYCLO CRYOTHERAPY

TREATMENT OF 1950
BIETTI

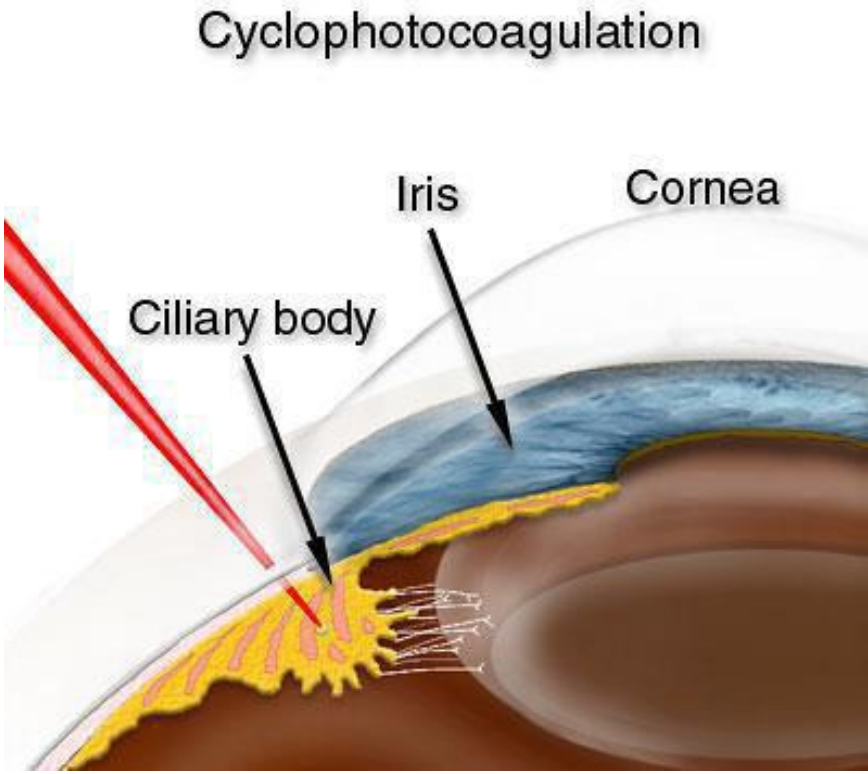




Lasers relatively safer energy

- Trans-scleral route
- Direct application to ciliary epithelium

Trans pupillary



Trans-scleral route diode laser

810 nm wave length

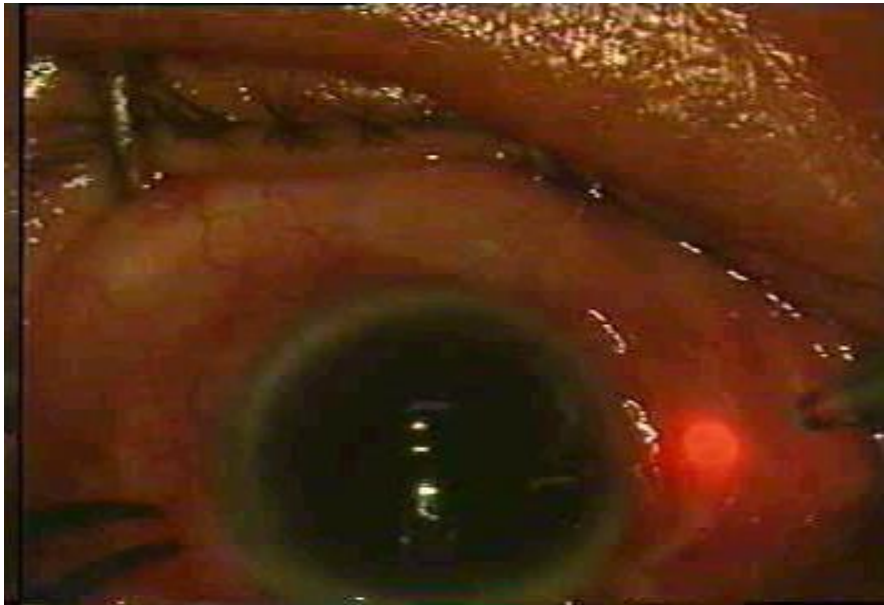
Penetrates through sclera

Contact delivery through fibre optic cable

Diode laser is preferred

Melanin in the ciliary epithelium better absorbs this wavelength

Causes more targeted destruction with less inflammation



VISUAL REHABILITATION

- Correction of refractive error

- Management of media opacities

- Amblyopia therapy to achieve binocular stereoscopic vision

VISUAL REHABILITATION

- Low vision aids

❖ Telescopes (hand-held or spectacle-mounted)

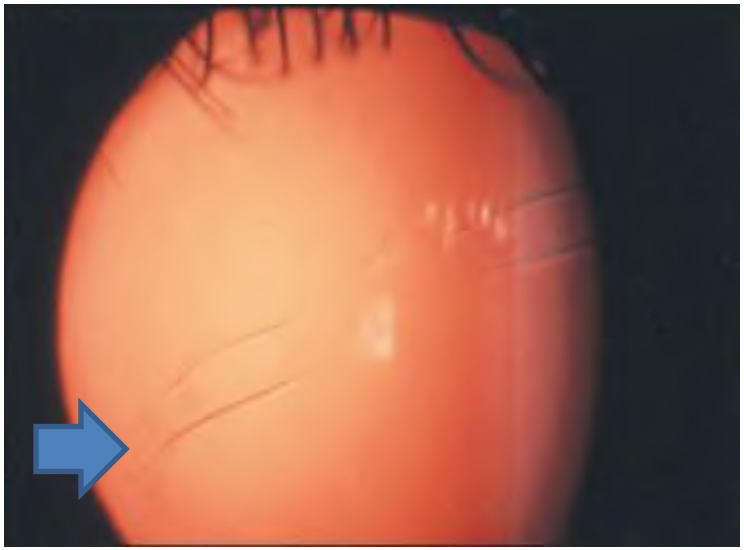
❖ Hand or pocket magnifiers (2× to 3×)

III. Hand held magnifiers

CONCLUSIONS

- Glaucoma is a group of disorders characterized by progressive optic neuropathy.
- Early diagnosis and prompt treatment can preserve vision.
- All children with suspected childhood glaucoma should be examined under anaesthesia.
- Mainstay of management of childhood glaucoma is surgery
- Visual rehabilitation and counseling of the parents of the child is as important as IOP control.

Question



- Identify the abnormality marked by arrow.
- Which structure is involved?
- What type of slit lamp illumination is used in the photograph?
- Mention one differential diagnosis of the condition


Thank you

Dr. Yousaf Jamal Mahsood

MBBS, CHPE, CMEJ, MHR, FICO (UK),
MRCSEd (UK), FRCS (Glasg), FCPS

Fellowship in Glaucoma (Al-Shifa Trust, Pak)
Fellowship in Glaucoma (Univ. of Toronto, Canada)
Advance Fellowship in Glaucoma (BPOS, UK)

Associate Professor Glaucoma
Department of Ophthalmology
Khyber Girls Medical College
Peshawar

Topic

Visual Loss and Intraocular Pressure (IOP)

Learning Objectives

Classify	Visual Loss associated with Anterior segment Visual Loss associated with Posterior segment
Enumerate	Causes of gradual & sudden visual loss
Discuss	Aqueous humor dynamics and its role in IOP
Define and Classify	Glaucoma

Learning Objectives

Classify

Visual Loss associated with Anterior segment

Visual Loss associated with Posterior segment

Visual loss

Anterior segment		
Conjunctiva	Cornea	Lens

Visual loss

Posterior segment			
Uveal tract	Vitreous	Retina	Optic nerve

Learning Objectives

Classify	Visual Loss associated with Anterior segment Visual Loss associated with Posterior segment
Enumerate	Causes of gradual & sudden visual loss
Discuss	Aqueous humor dynamics and its role in IOP
Define and Classify	Glaucoma

Learning Objectives

Enumerate	Causes of gradual & sudden visual loss

Gradual Visual Loss

- Refractive Errors
- Cataracts
- Primary Open Angle Glaucoma
- Age Related Macular Degeneration
- Diabetic Retinopathy
- Cornea (Ectasia, Dystrophy)
- Optic Neuropathies (Compression, Toxic, Drugs, Nutritional deficiency)
- Choroid and Retina (Inflammations, Tumors, Dystrophies)
- Papilledema

Sudden Visual Loss

- Retinal Vascular Diseases (CRVO, BRVO, CRAO, BRVO)
- Retinal Detachment
- Vitreous Hemorrhage
- Acute Angle Closure
- Optic Neuropathy (Optic Neuritis, AION)
- Endophthalmitis
- Trauma

Learning Objectives

Classify	Visual Loss associated with Anterior segment Visual Loss associated with Posterior segment
Enumerate	Causes of gradual & sudden visual loss
Discuss	Aqueous humor dynamics and its role in IOP
Define and Classify	Glaucoma

Learning Objectives

Discuss	Aqueous humor dynamics and its role in IOP

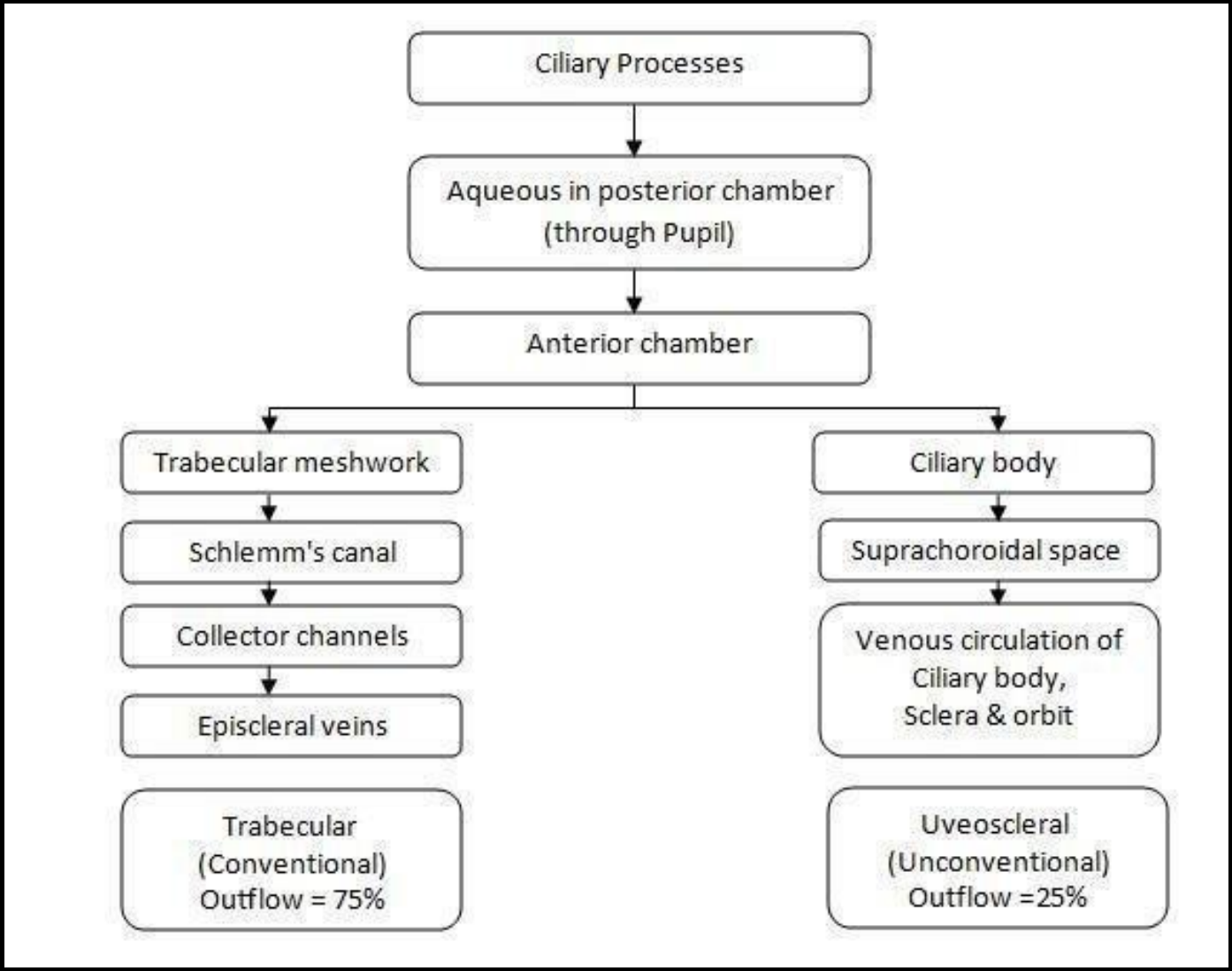
Aqueous Humor (AH)

- Clear fluid that fills and helps form the anterior and posterior chambers of the eye
- Analogous to a blood surrogate for these avascular structures (lens, cornea)
- Equilibrium exists between the production and drainage of AH

AH Formation

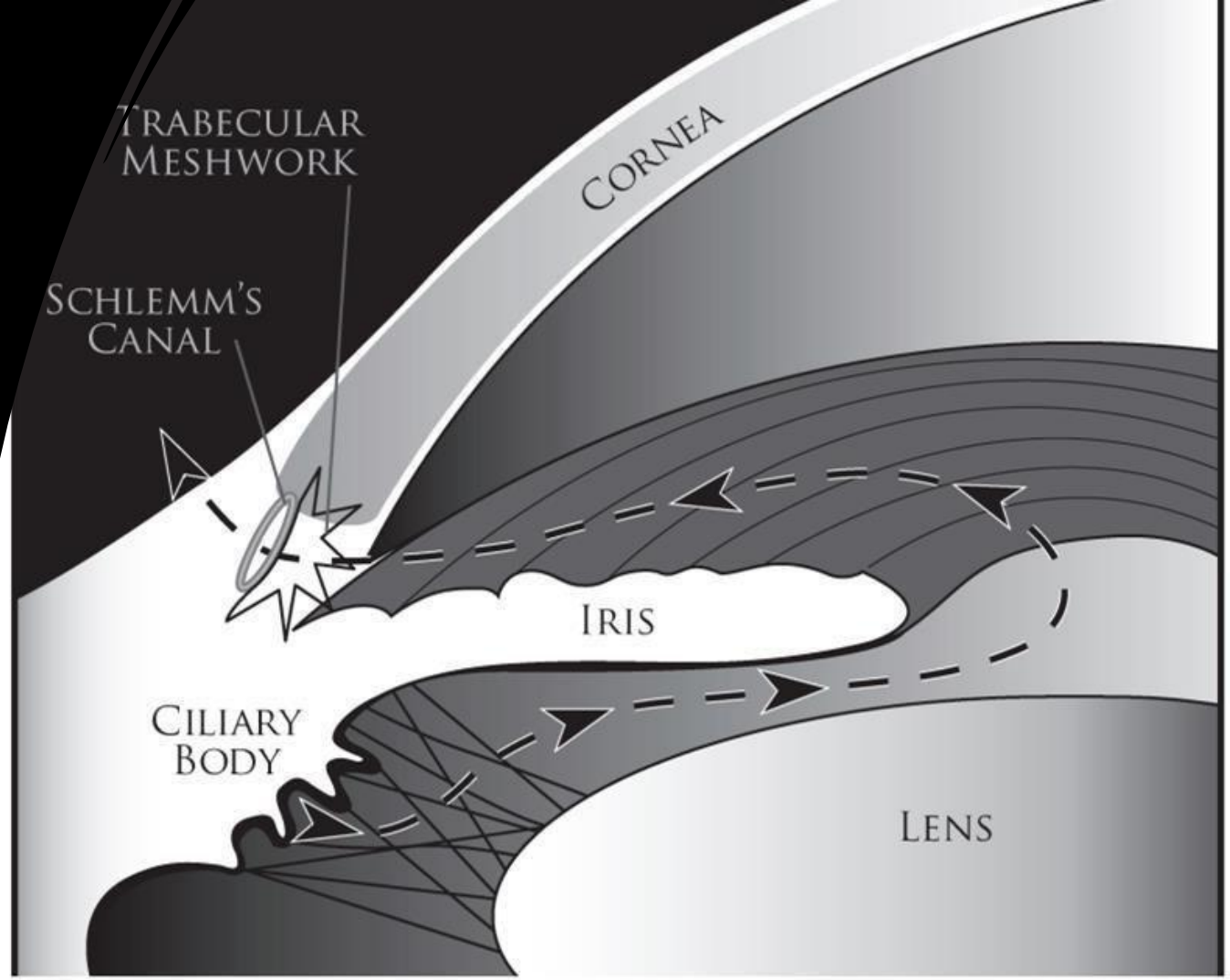
- Formed by non-pigmented epithelium of ciliary body
- Three mechanisms are involved in aqueous humor formation:
    1. Diffusion
    2. Ultrafiltration
    3. Active Secretion (80% of contribution)
- The first two processes are passive

AH Outflow


Ciliary Processes
Aqueous in posterior chamber (through Pupil)
Anterior chamber
Trabecular meshwork
Schlemm's canal
Collector channels
Episcleral veins
Trabecular (Conventional) Outflow = 75%
Ciliary body
Suprachoroidal space
Venous circulation of Ciliary body, Sclera & orbit
Uveoscleral (Unconventional) Outflow =25%

Aqueous Outflow

- Conventional Flow
  - Through TM
  - 75% resistance to aqueous flow by TM



ig. (1). Schematic diagram illustrating the trabecular meshwork onventional outflow pathway. Aqueous humor is produced by the iary body and it flows (dashed line shown with arrowheads) from posterior chamber through the pupil into the anterior chamber. there it flows out through the trabecular meshwork into the

Aqueous Outflow

- Uveoscleral Flow

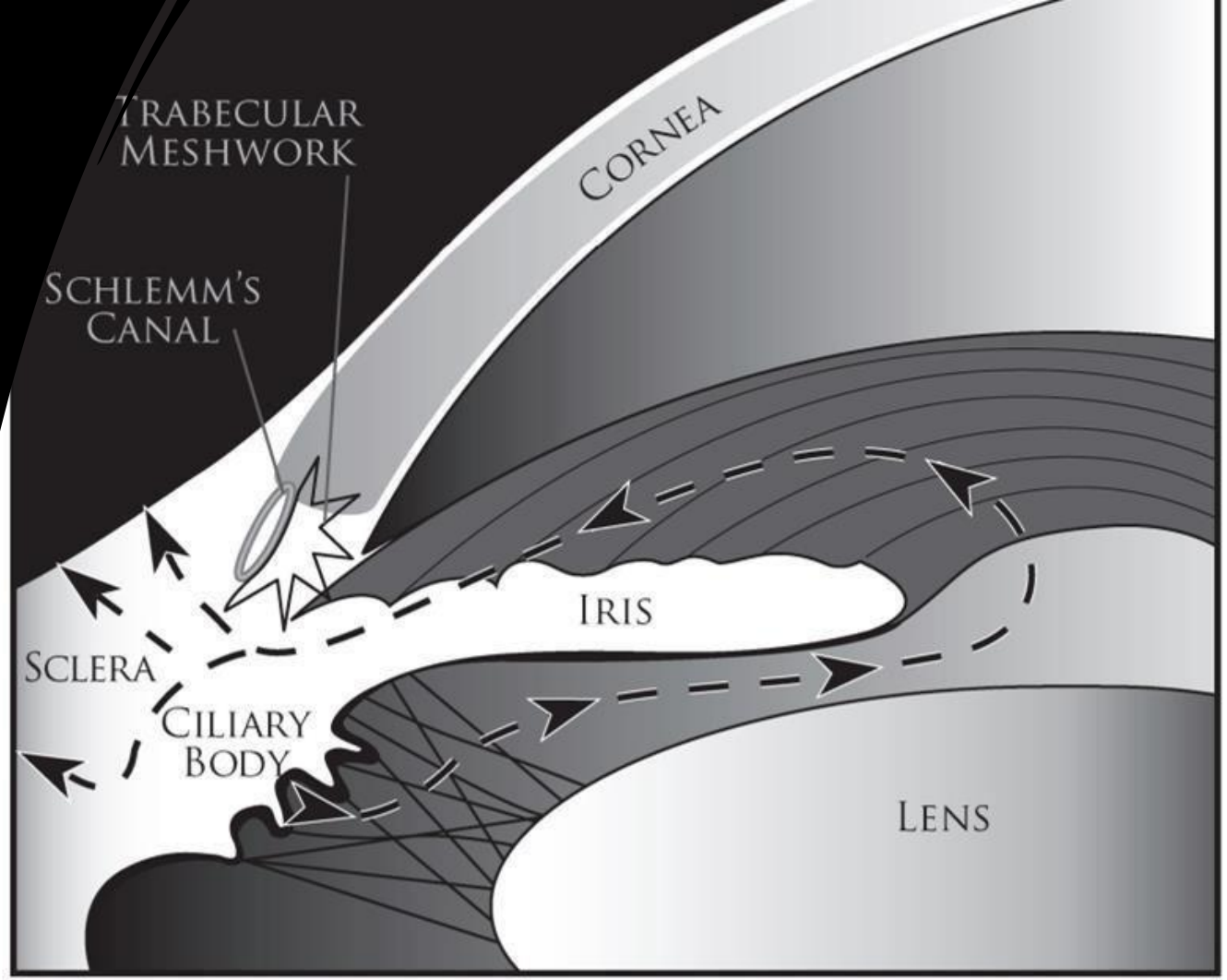


Fig. (2). Schematic diagram illustrating the uveoscleral outflow athway. Aqueous humor is produced by the ciliary body, in veoscleral route, it flows from the posterior chamber through the il into the anterior chamber and then (shown by dashed lines rrowheads) through the face of the ciliary body and iris root to

AH Composition

Amino acids
98% water
Electrolytes (pH = 7.4)
Ascorbic acid
Glutathione
Immunoglobulins

Intraocular Pressure (IOP)

- IOP ranges from 11-21 mmHg (16mmHg average)
- Equilibrium between AH inflow and outflow
- IOP raises with the equilibrium disturbance
- Raised IOP leads to Optic Nerve damage

Learning Objectives

Classify	Visual Loss associated with Anterior segment Visual Loss associated with Posterior segment
Enumerate	Causes of gradual & sudden visual loss
Discuss	Aqueous humor dynamics and its role in IOP
Define and Classify	Glaucoma

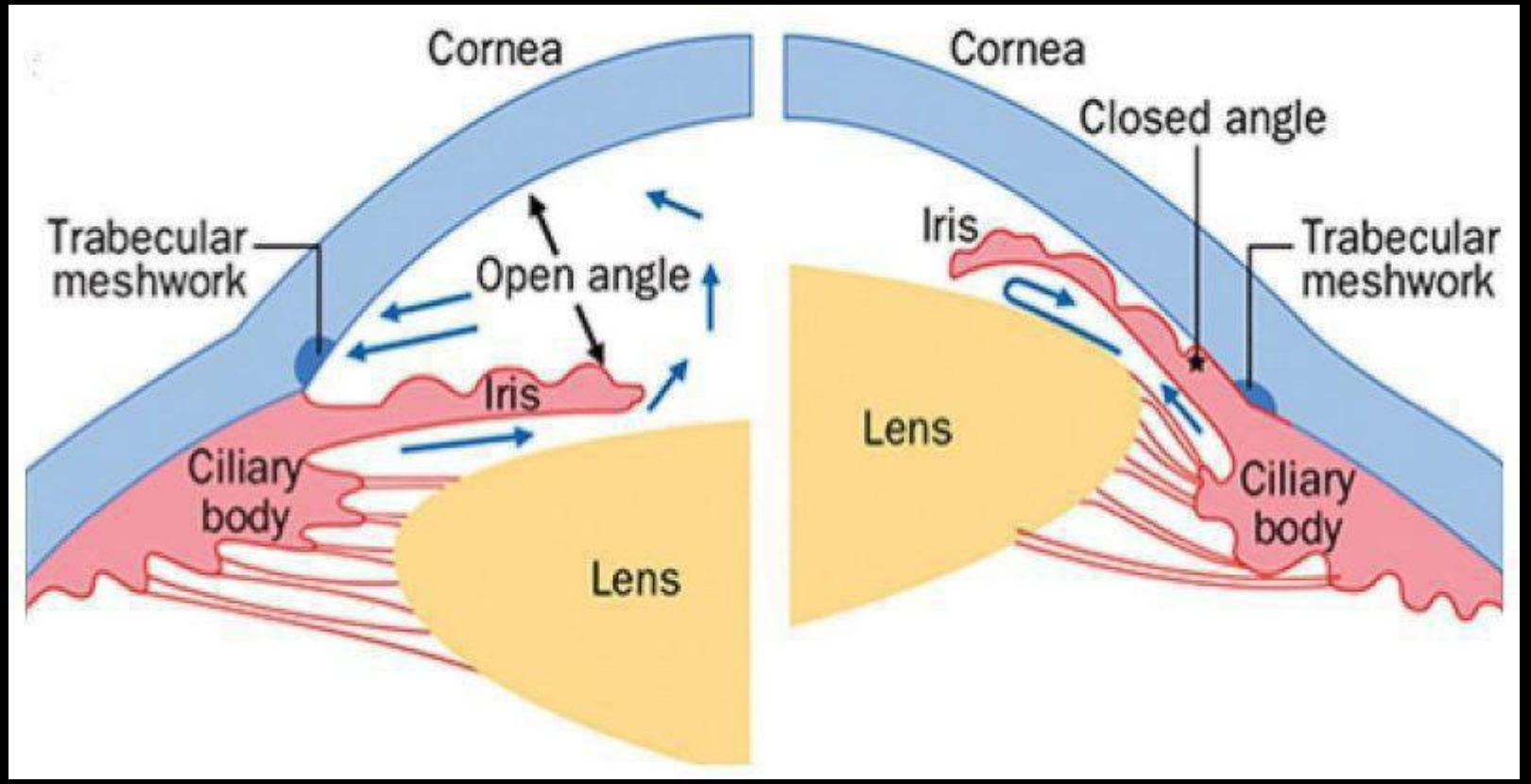
Learning Objectives

Define and Classify Glaucoma

Glaucoma

- Complex entity
- Progressive Optic Neuropathy with characteristics visual field loss and optic nerve (RNFL) damage

Classification



Glaucoma Classification

Open Angle Glaucoma

- Primary OAG (POAG)
- NTG
- JOAG
- Secondary OAG
  - PXF-G
  - Pigmentary
  - Steroids Induced
  - Lens Induced
  - Post surgical
  - ARG

Closed Angle Glaucoma

- Primary ACG (PACG)
- Secondary ACG
  - NVG
  - PXF-G
  - Aniridia
- Trauma

Summary

Learning Objectives

- Classify causes of visual loss in following order:
  - Visual Loss associated with Anterior segment.
  - Visual Loss associated with Posterior segment.
- Enumerate causes of gradual & sudden visual loss.
- Discuss Aqueous humor dynamics and its role in IOP.
- Define and Classify Glaucoma.

Thank You All

Dr. Yousaf Jamal Mahsood

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Topics

- Primary Open Angle Glaucoma (POAG)
- Ocular Hypertension (OHT)
- Normal Tension Glaucoma (NTG)

At the end of this presentation, you must be able to answer these

Learning Objectives

- Discuss
  - Differences between POAG, NTG and OHT
- Discuss
  - Etiology, clinical features, investigation and management of POAG
- Discuss
  - Etiology, clinical features, investigation and management of NTG
- Discuss
  - Etiology, clinical features, investigation and management of OHT

Primary Open Angle Glaucoma

Definition

- Chronic, slowly progressive Optic neuropathy with characteristic patterns of optic nerve damage and visual field loss
- Not caused by another systemic or local disease.

Epidemiology

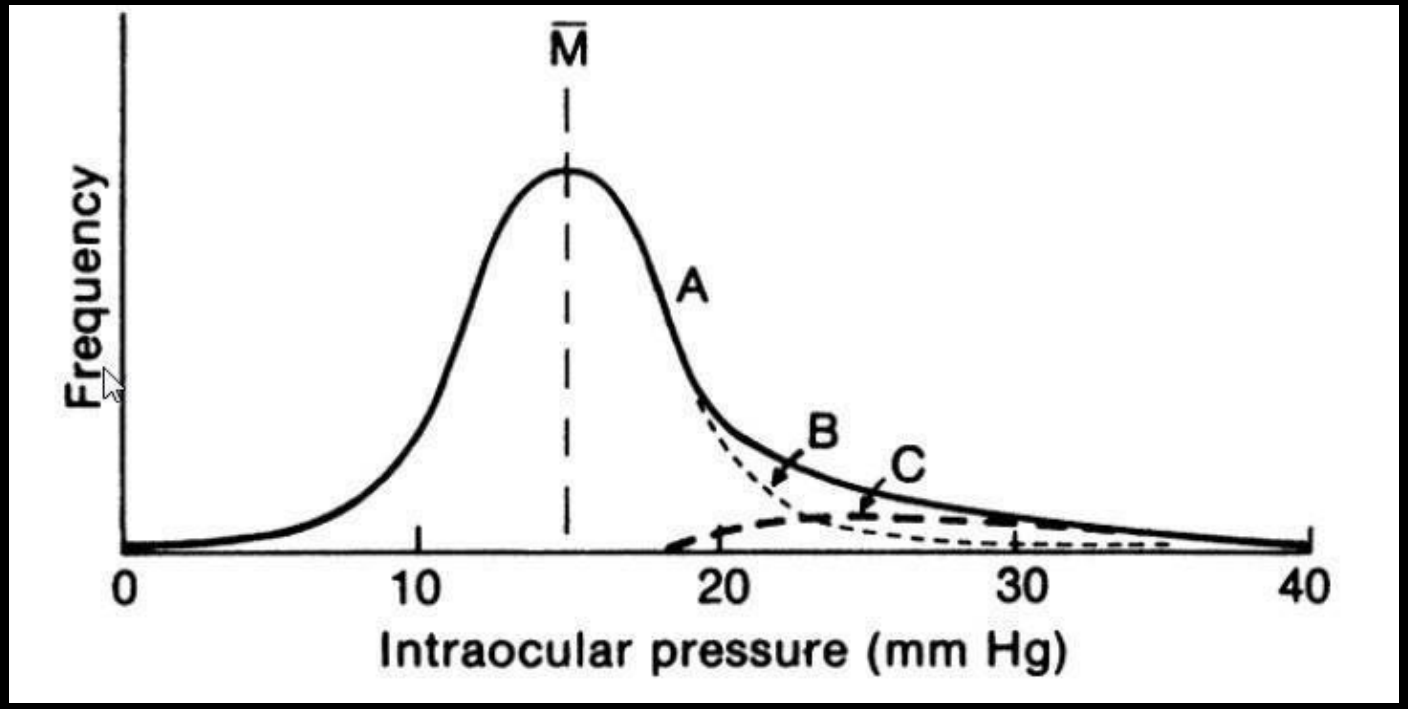
- Glaucoma is the 2nd cause of blindness and leading cause of irreversible blindness worldwide. (AAO 2010)
- POAG … Most common form of glaucoma (60-70%)
- Prevalence of bilateral blindness: 8% in blacks and 4% in whites

Risk factors

- Elevated IOP
- Increased C/D ratio
- CCT (<550 µm)
- Family Hx (x 3.7 in first degree)
- Age (> 60y/o)
- Race (x 6 in African Americans)
- Other (CRVO, Myopia, HTN, DM and Migraine)

Intra Ocular Pressure

- Mean IOP: 15.5 mm Hg ± 2.6 SD



Distribution of intraocular pressure in population.

Line B represents the “normal” population completing a gaussian distribution.
Line C represents the abnormal population, which added to the normal population produces the tail.

Central Corneal Thickness

- Average 525-545 µm
- Increased CCT in OHT
- Decreased CCT in NTG

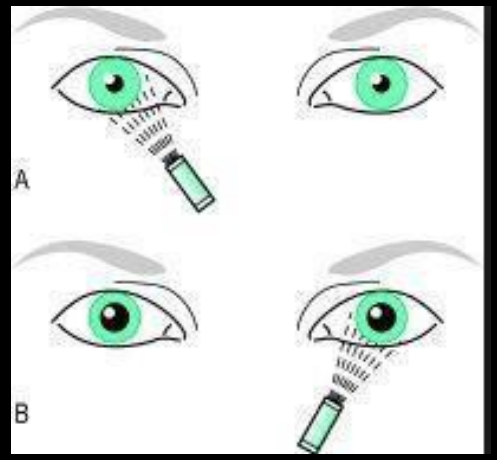
Clinical features

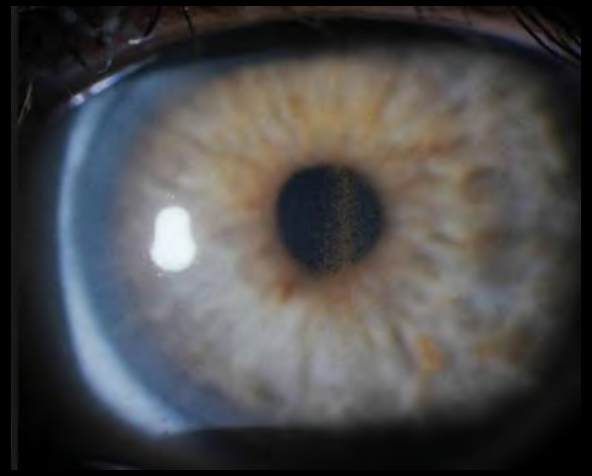
- Insidious
- Slowly progressive
- Painless
- Significant visual loss
- Usually bilateral (asymmetric)

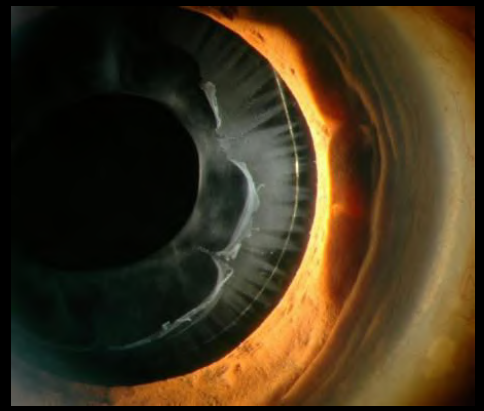
Diagnosis

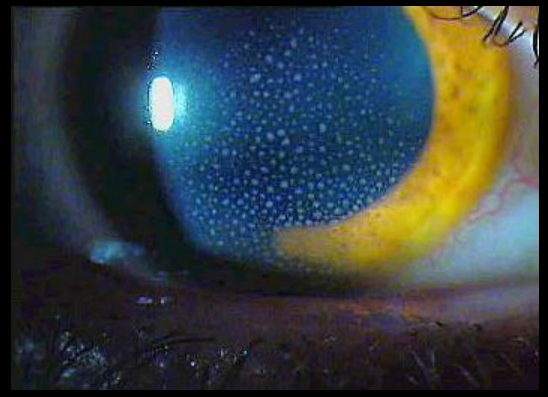
Combination of structural and functional damage of optic nerve

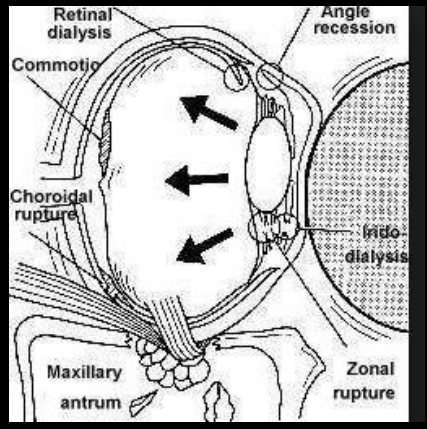
Clinical Examination



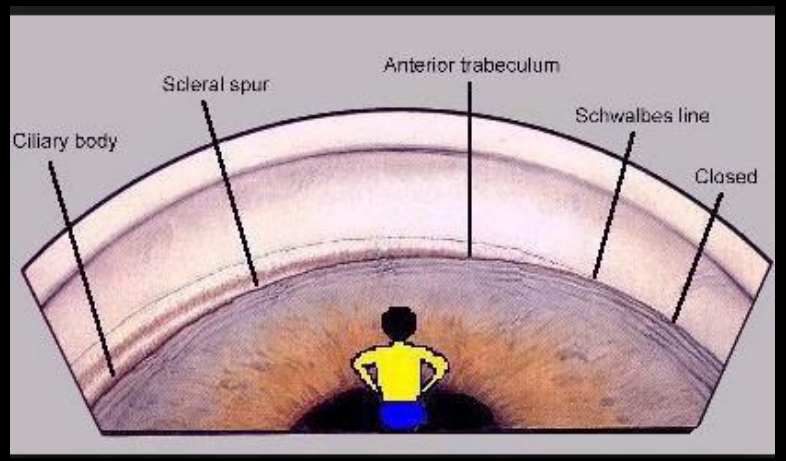


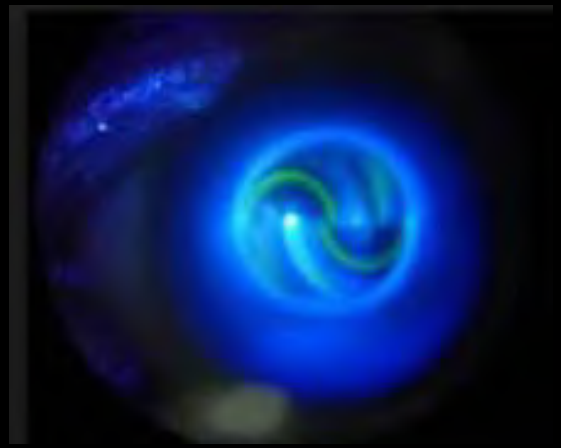


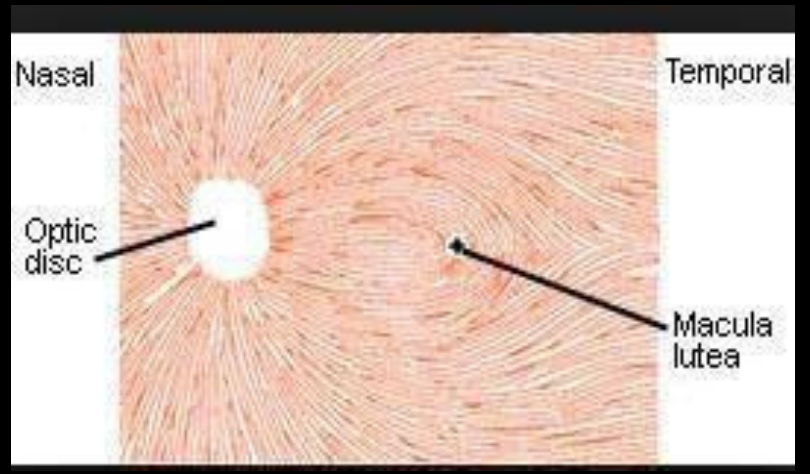




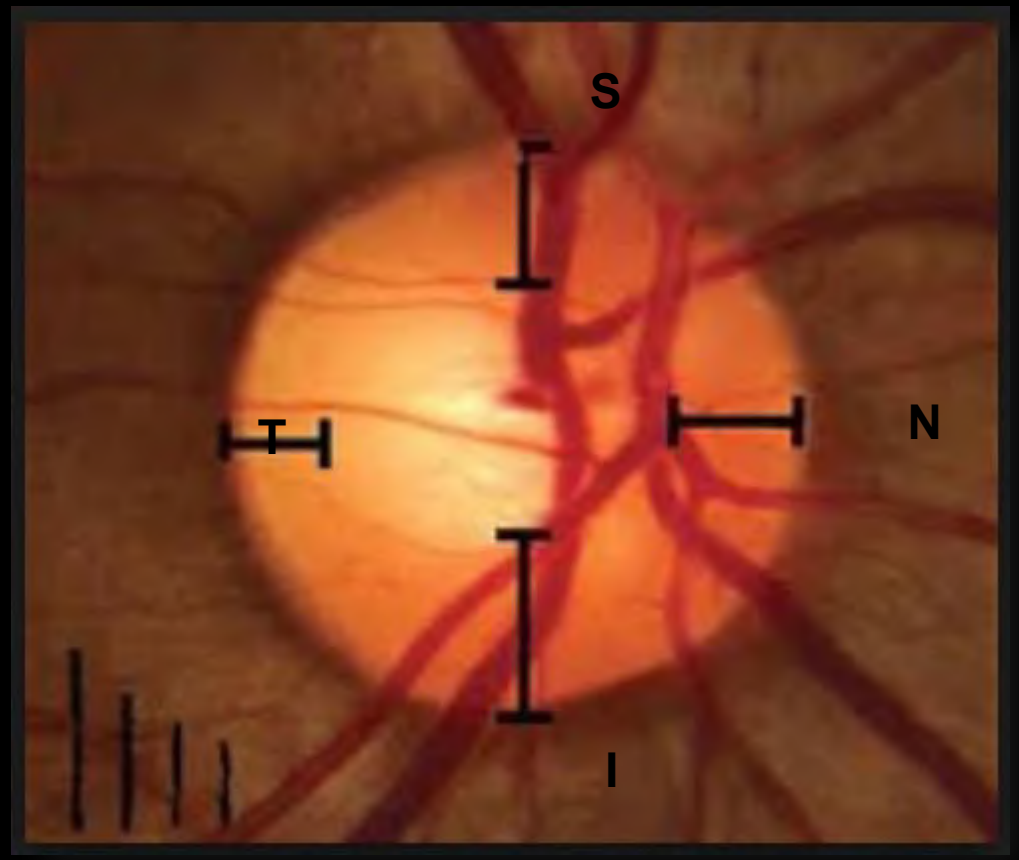
Clinical Examination



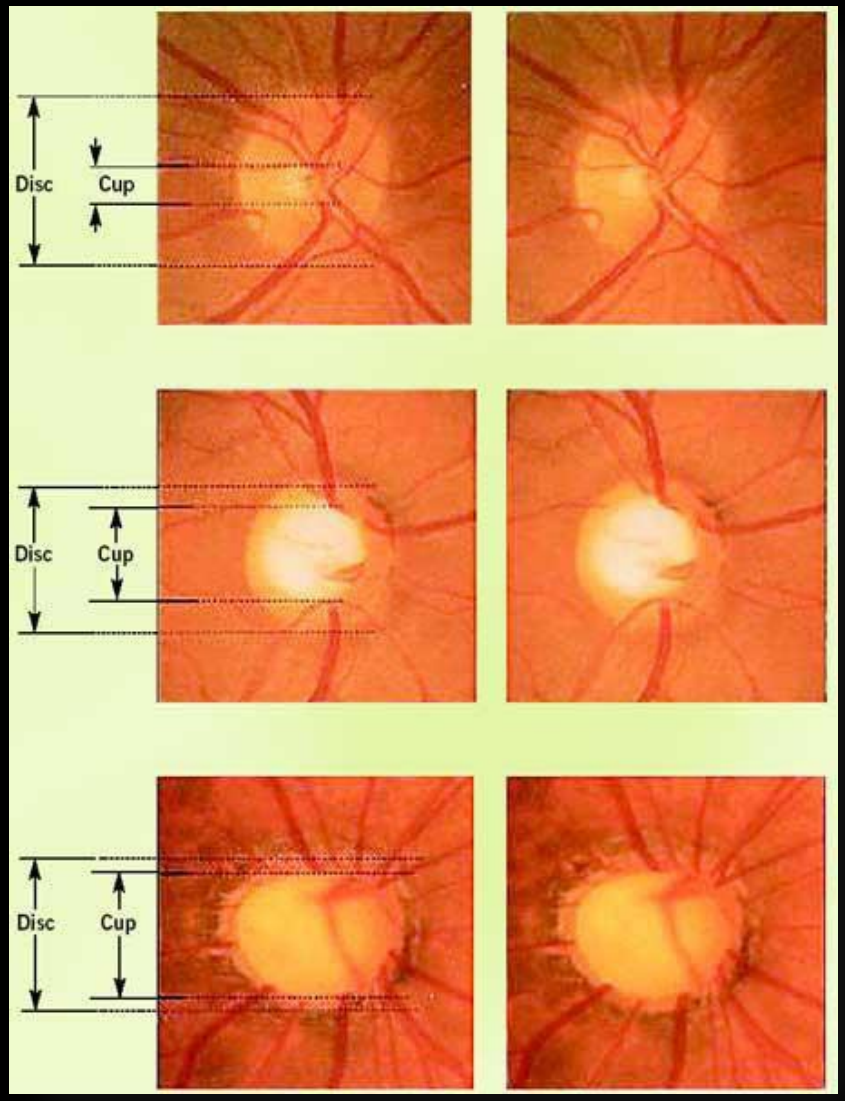




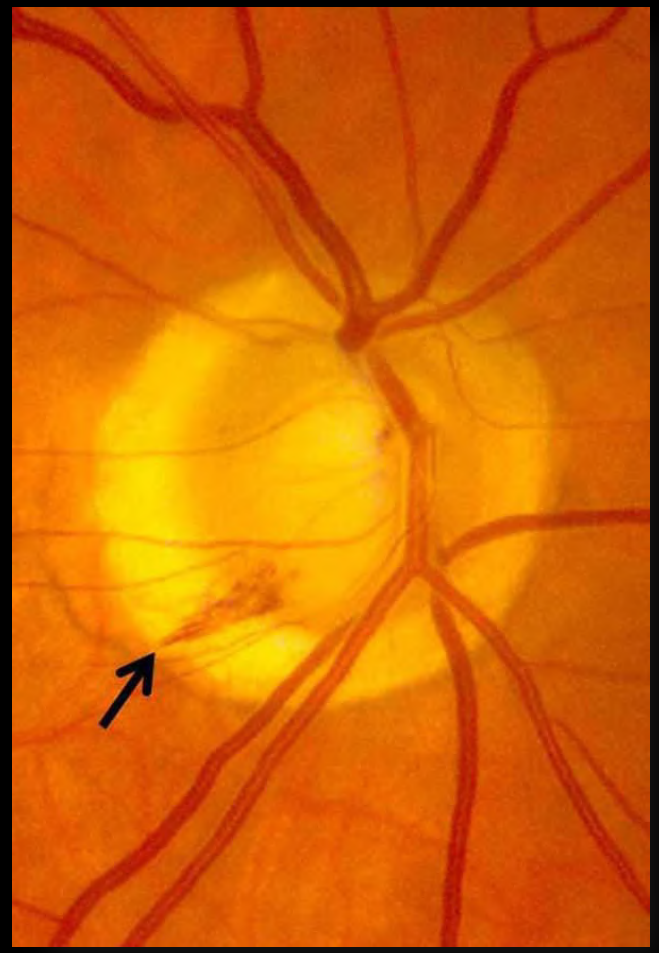
Optic Disc Appearance (ISNT rule)



Asymmetry of the rim or cupping



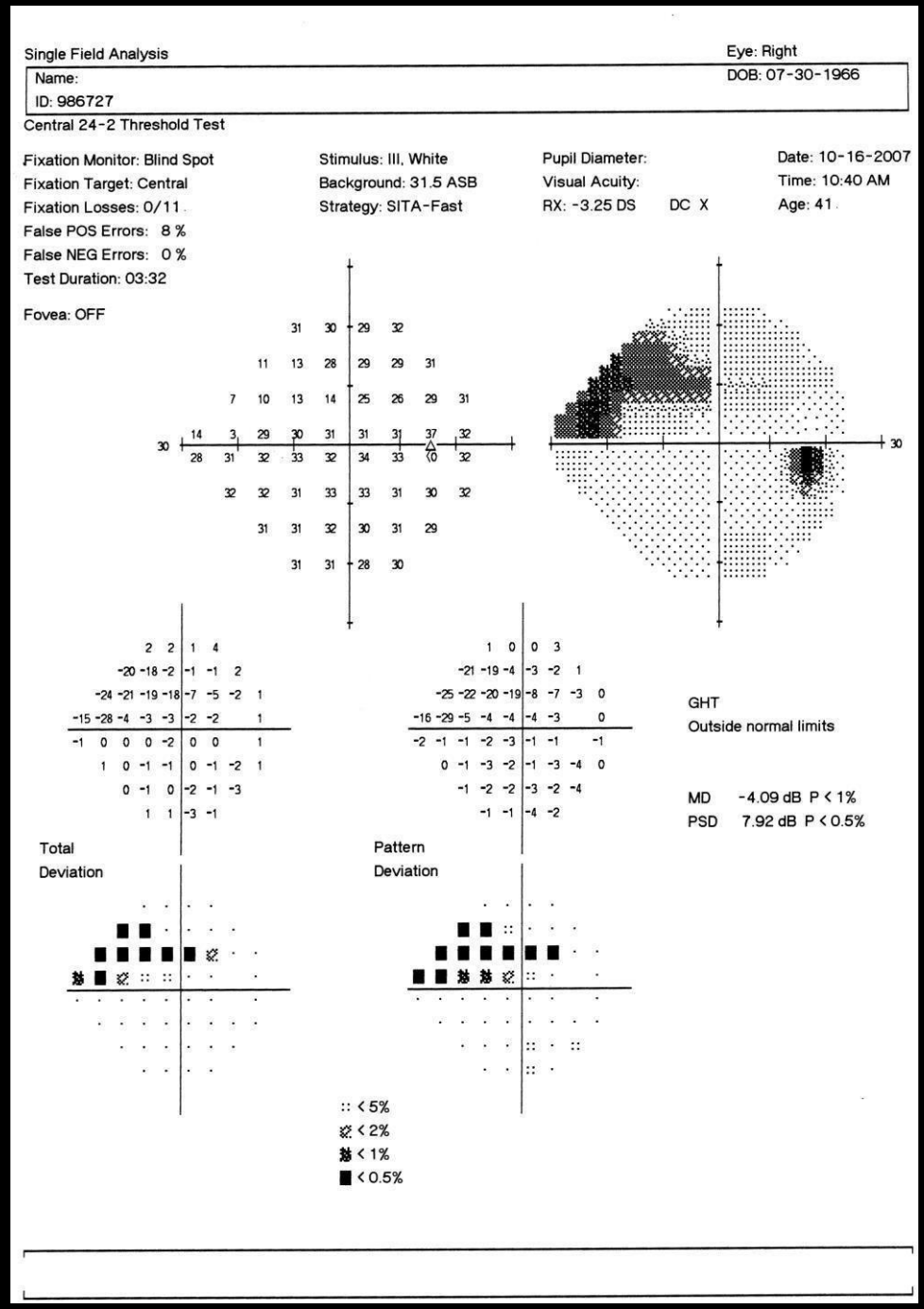
Disc hemorrhage



Visual Field Loss

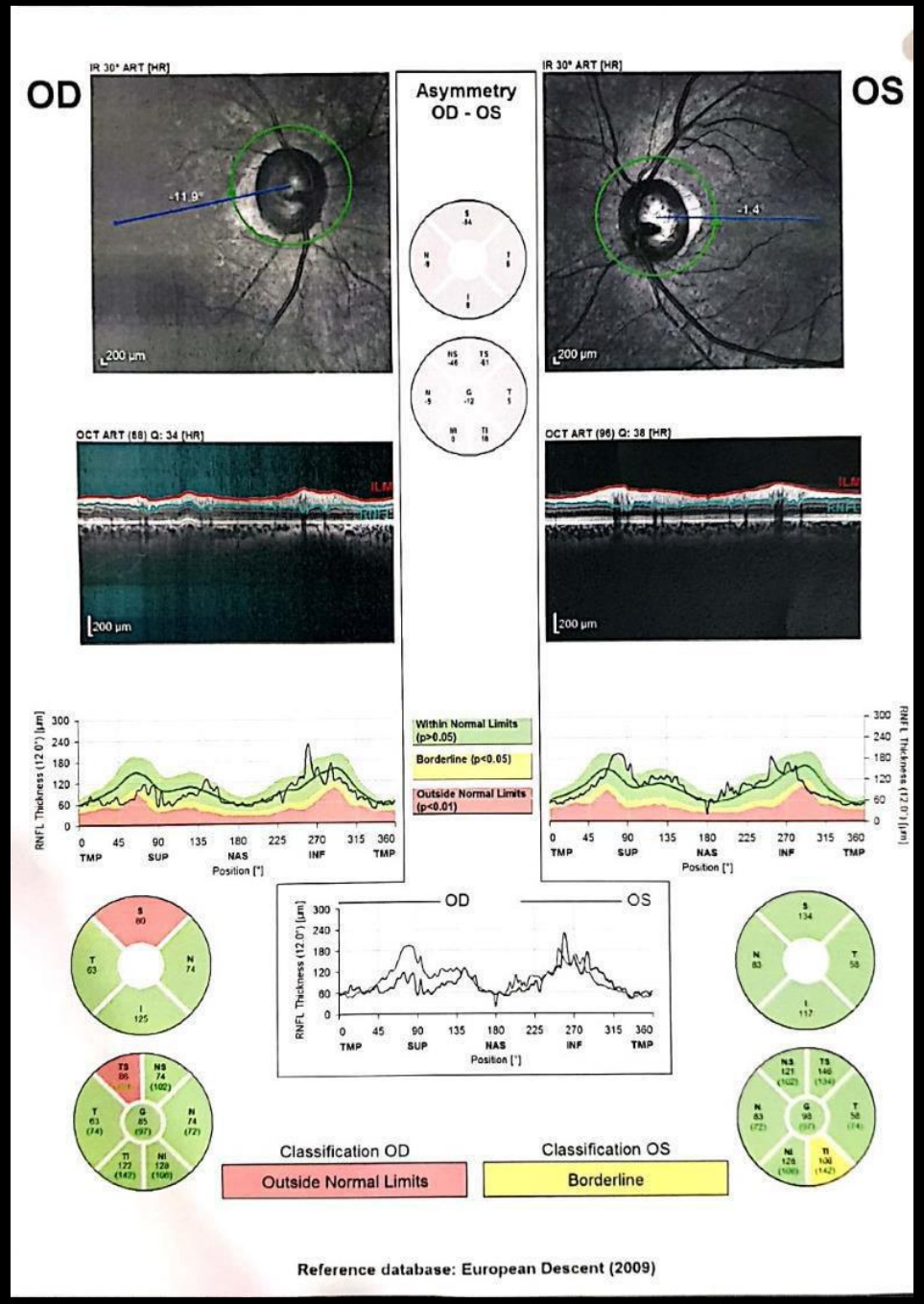
Functional loss

- Humphrey
  - White on White Full Threshold
  - SITA – Swedish Interactive
  - Thresholding Algorithm
  - Blue on Yellow
- Frequency Doubling Test
- Octopus
- High Pass Resolution



Structural Loss

- Optical Coherence Tomography (OCT)
- Heidelberg retinal Tomography (HRT)



Management

- Target is to achieve an IOP which stops progression of the optic neuropathy and development of complications.
    - Medical
    - Laser
    - Surgical

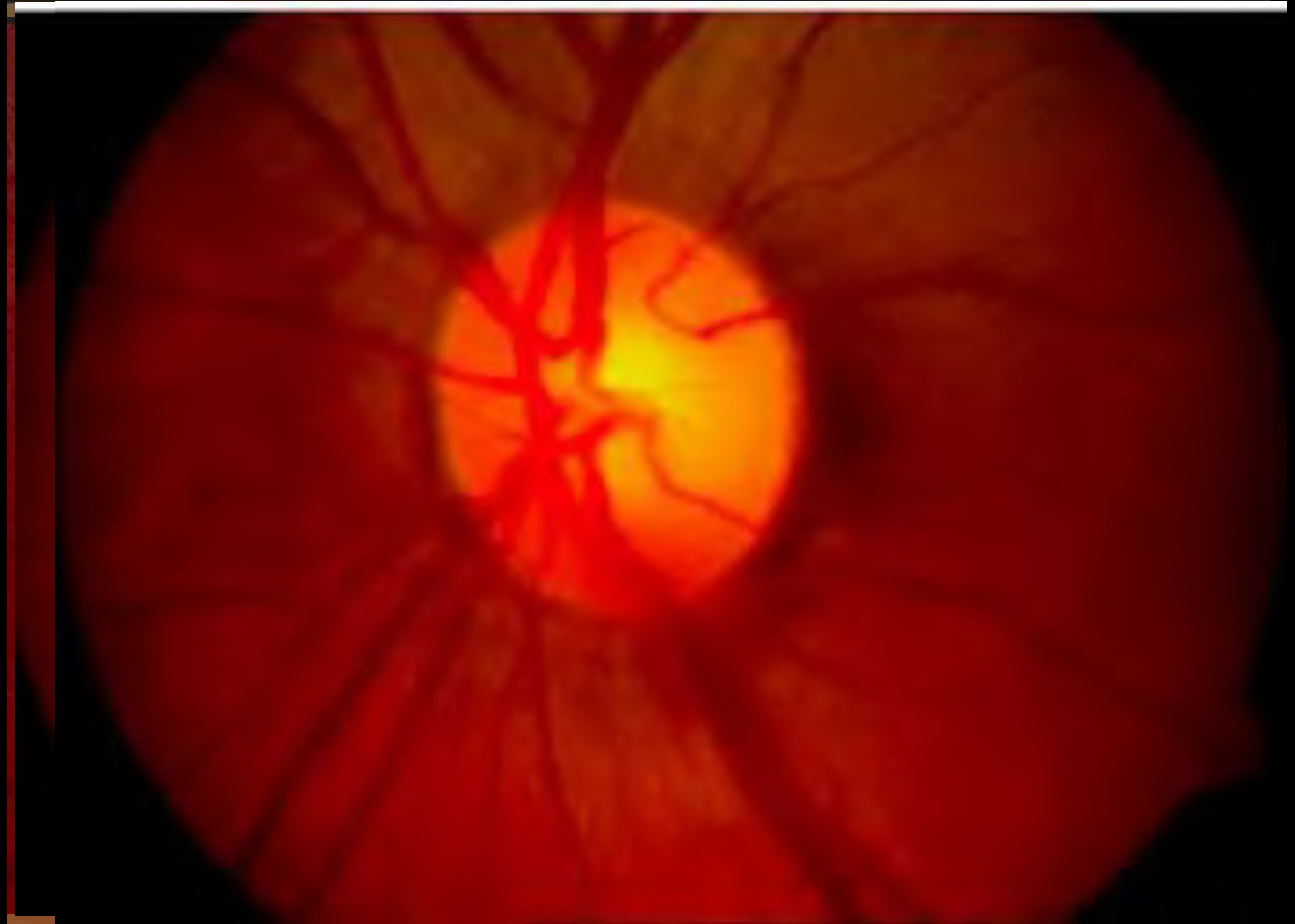
Ocular Hypertension

Definition

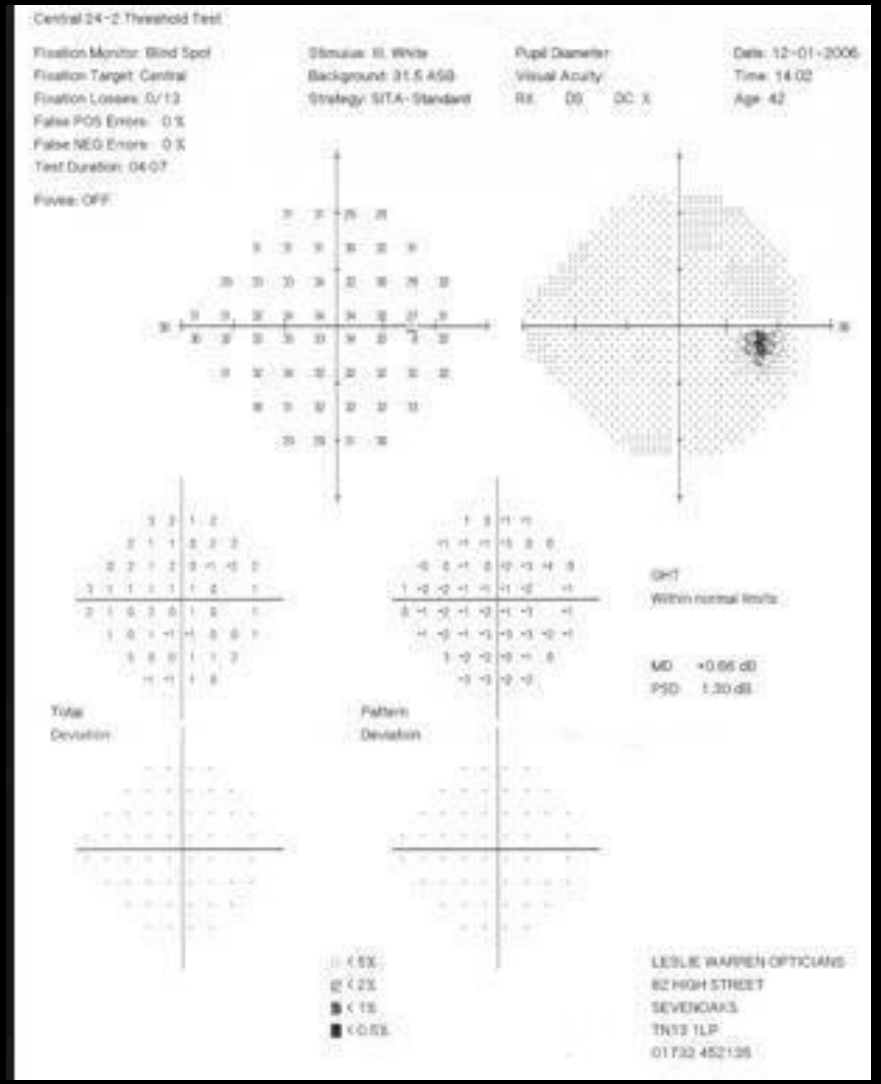
- Elevated intraocular pressure without evidence of structural and functional damage by standard clinical tests.

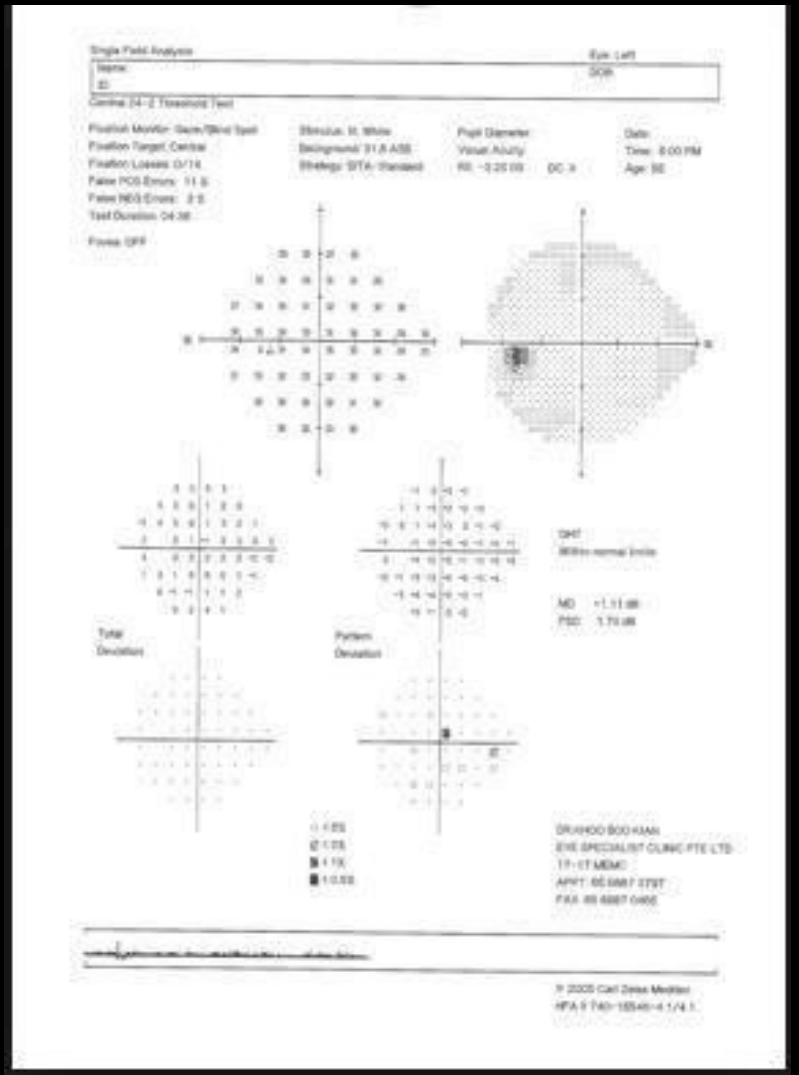
- Must have:
  - Open angles!
  - No ocular or systemic cause of elevated IOP.

>21 mmHg



Next step?


Central 24-2 Threshold Test
Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 0/13
False POS Errors: 0 %
False NEG Errors: 0 %
Test Duration: 04:07
Fovea: OFF
Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA - Standard
Pupil Diameter:
Visual Acuity:
Rx: DS DC: X
Date: 12-01-2006
Time: 14:02
Age: 42
GHT
Within normal limits
MD +0.66 dB
PSD 1.30 dB
Total Deviation
Pattern Deviation
< 5%
< 2%
< 1%
< 0.5%
LESLIE WARREN OPTICIANS
82 HIGH STREET
SEVENOAKS
TN13 1LP
01732 452135


Single Field Analysis
Eye: Left
Name:
ID:
Central 24-2 Threshold Test
Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 0/14
False POS Errors: 11 %
False NEG Errors: 2 %
Test Duration: 04:38
Fovea: OFF
Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA - Standard
Pupil Diameter:
Visual Acuity:
Rx: -0.25 DS DC: X
Date:
Time: 8:00 PM
Age: 50
GHT
Within normal limits
MD +1.11 dB
PSD 1.70 dB
Total Deviation
Pattern Deviation
< 5%
< 2%
< 1%
< 0.5%
DR KHOO BOO KIAN
EYE SPECIALIST CLINIC PTE LTD
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FAX: 65 6887 0400
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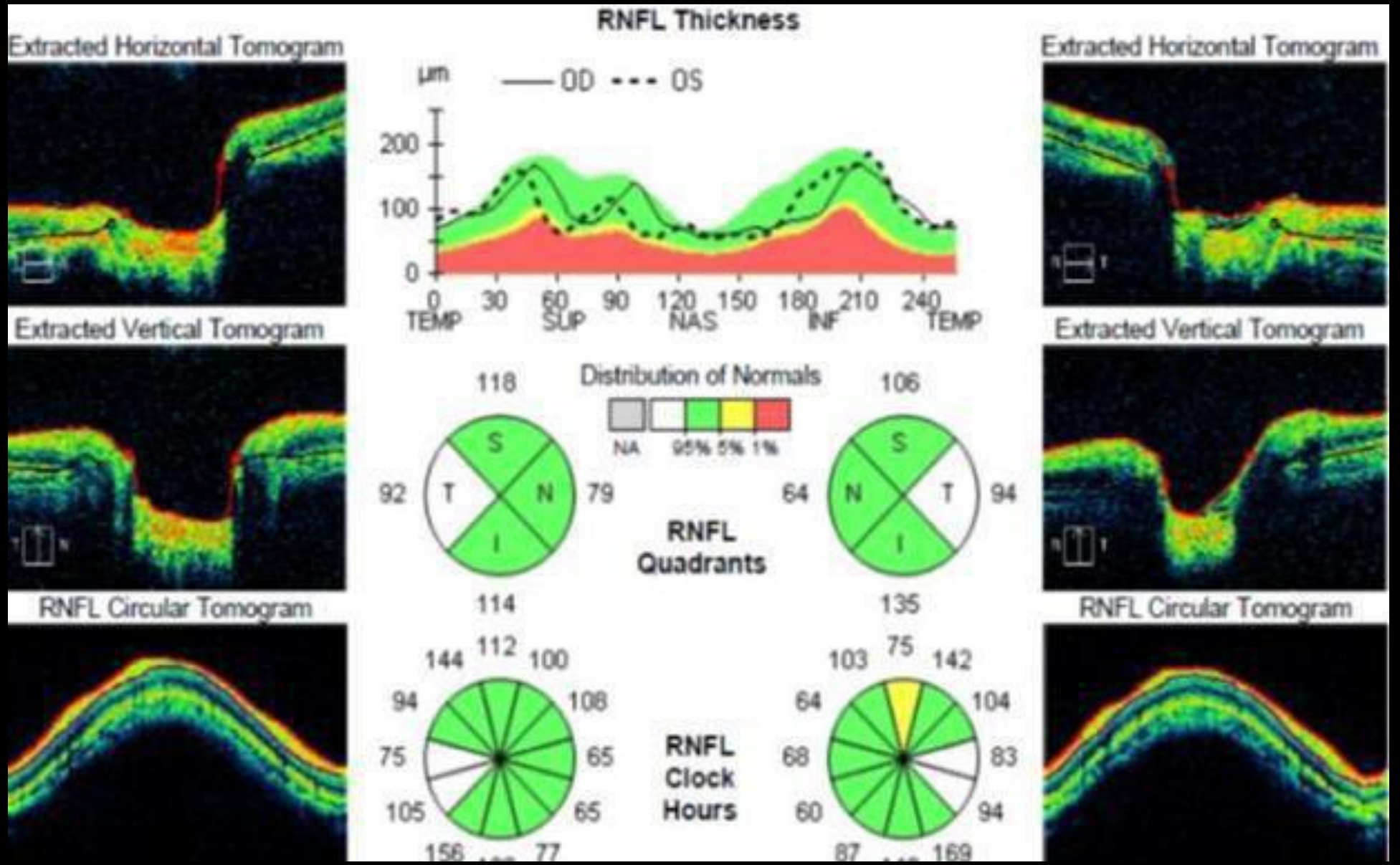
HFA II 740-16540-4.1/4.1


RNFL Thickness
Extracted Horizontal Tomogram
µm
OD
OS
200
100
0
0
30
60
90
120
150
180
210
240
TEMP
SUP
NAS
INF
TEMP
Extracted Horizontal Tomogram
Extracted Vertical Tomogram
Distribution of Normals
NA
95%
5%
1%
118
S
92
T
N
79
I
114
106
S
64
N
T
94
I
135
RNFL Quadrants
Extracted Vertical Tomogram
RNFL Circular Tomogram
144
112
100
94
108
75
65
105
65
156
77
RNFL Clock Hours
103
75
142
64
104
68
83
60
94
87
169
RNFL Circular Tomogram

Conclusion

Topical ocular hypotensive medication is effective in reducing the incidence of glaucomatous VF loss and/or ON deterioration in subjects with IOP between 24-32 mmHg.

Treatment

- Goal:
  - Setting a reasonable target: <21.
- Follow-up at least every 6 months with tests.

Normal Tension Glaucoma

NTG - Definition

- “low-tension glaucoma, normal-pressure glaucoma”
- Chronic optic neuropathy
- Parallels primary open angle glaucoma with:
  - Characteristic optic nerve cupping
  - Visual-field loss

BUT:

- Consistently normal IOP (≤21mmHg)
- Absence of systemic/ocular features contributing to other forms of optic neuropathy

Pathophysiology

- Different schools of thought:
  - Hypersensitivity to IOP
  - Vascular perfusion problem
    - deficiency in short posterior ciliary circulation
  - Autoimmune process

Risk Factors

- Not just IOP!
- Currently not known
- Suspected risk factors:
  - Age → elderly
  - Sex → female preponderance
  - hereditary component (variable)
  - Vasospastic disorders
  - Autoimmune disease

Raynaud's Phenomenon

Diagnostic Evaluation

- Take a good medical history! Ask about:
  - Past steroid use, (prior ocular hypertension)
  - Thin corneas (i.e. past refractive surgery)
  - Past symptoms of anemia
  - Orthostatic symptoms
  - Symptoms of sleep apnea
  - Migraine

Further Diagnostic Evaluation

- Diurnal IOP curve
- Gonioscopy → rule out angle closure, angle recession, previous intraocular inflammation
- Stereoscopic disc evaluation → rule out congenital/acquired disc anomalies
- Visual field → glaucomatous defects
- +/- medical/neurologic evaluation → blood work (anemia, infection etc.), auscultation of carotid arteries, CT/MRI brain/orbits

NTG vs. POAG

- How can you differentiate the two?

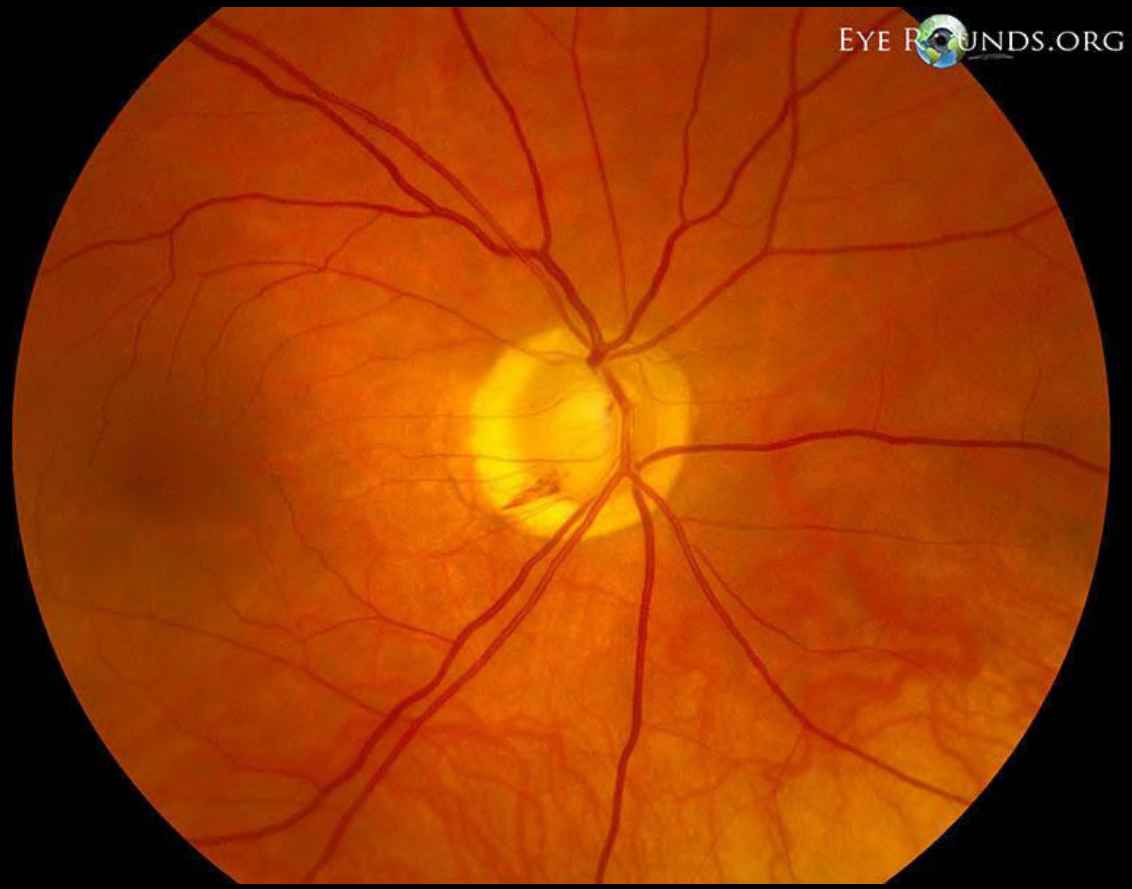
NTG vs. POAG

- Lower IOP
- Visual field defects are deeper, steeper and closer to fixation
- Visual field defects worse than expected based on optic disc appearance
- More focal notching and localized defects on nerve fiber layer
- Higher propensity for disc hemorrhages

NTG vs. POAG Patients

- NTG patients 10 years older on average
- Female
- Familial tendency
- Myopic
- Recurrent disc hemorrhages → linked with progression in visual field

Disc Hemorrhage



Significance of Disc Hemorrhages

- Associated with and a risk factor for disease progression
- Need amplification in glaucoma therapy!

Treatment

- Initiated for NTG, unless optic neuropathy is stable
- CNTGS → results supported reduction of IOP by 30% to reduce progressive visual field loss

In Summary

- NTG → diagnosis of exclusion!
- Need to take a thorough medical history and physical examination over time
- Reducing IOP by 30% is effective in slowing down disease progression

Summary

- Normal Tension Glaucoma
- Ocular Hypertension
- Normal Tension Glaucoma

Any Question ?

MCQ 1

- A 45 years old male is referred for glaucoma evaluation. His IOP is 32 & 24 mm Hg in right & left eye respectively with cupping of optic discs. There also changes on OCT RNFL and Visual field testing with open angles on Gonioscopy on both sides. He denies any systemic illness or trauma history.

What is your most probable diagnosis in this case?

A. Normal tension glaucoma
B. Ocular hypertension
C. Pigmentary glaucoma
D. Primary open angle glaucoma
E. Secondary open angle glaucoma

Ans: D

MCQ 2

- A 65 years old female is referred for glaucoma evaluation. Her IOP is 12 & 11 mm Hg in right & left eye respectively with cupping of optic discs. There are also changes on OCT RNFL and Visual field testing with open angles on Gonioscopy on both sides. She has history of migraine .

What is your most probable diagnosis in this case?

A. Normal tension glaucoma
B. Ocular hypertension
C. Pigmentary glaucoma
D. Primary open angle glaucoma
E. Secondary open angle glaucoma

Ans: A

Thank you

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Fellowship in Glaucoma (Al-Shifa Trust, Pak)
Fellowship in Glaucoma (Univ. of Toronto, Canada)
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PRIMARY ANGLE CLOSURE GLAUCOMA

D. Dx of Visual Loss

Painful	Painless
• Acute Angle Closure Glaucoma	• Cataract (Except LIG)
• Neovascular Glaucoma	• Primary Open Angle Glaucoma / NTG
• Lens Induced Glaucoma	• Central Retinal Vein Occlusion
• Endophthalmitis / Panophthalmitis	• Central Retinal Artery Occlusion
• Optic Neuritis	• Retinal Detachment
• Anterior Uveitis	• Vitreous Hemorrhage
• Temporal Arteritis	• Macular Degeneration
• Trauma	

At the end of this presentation, you must be able to answer these

Learning objectives

- Discuss
  - Stages of PACG
- Discuss
  - Etiology, clinical features, investigation and management of Acute angle closure.

Introduction

- Half of glaucoma related blindness is due to primary angle closure glaucoma (PACG)
- PACG was thought to be acute & symptomatic until majority of cases were found to be chronic & asymptomatic

Quigley HA et al. the number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol. 2006;90:262-7.

- Acute attacks are extremely distressing but transient
- It does not cause permanent loss of vision in many (60-75%) if treated appropriately *

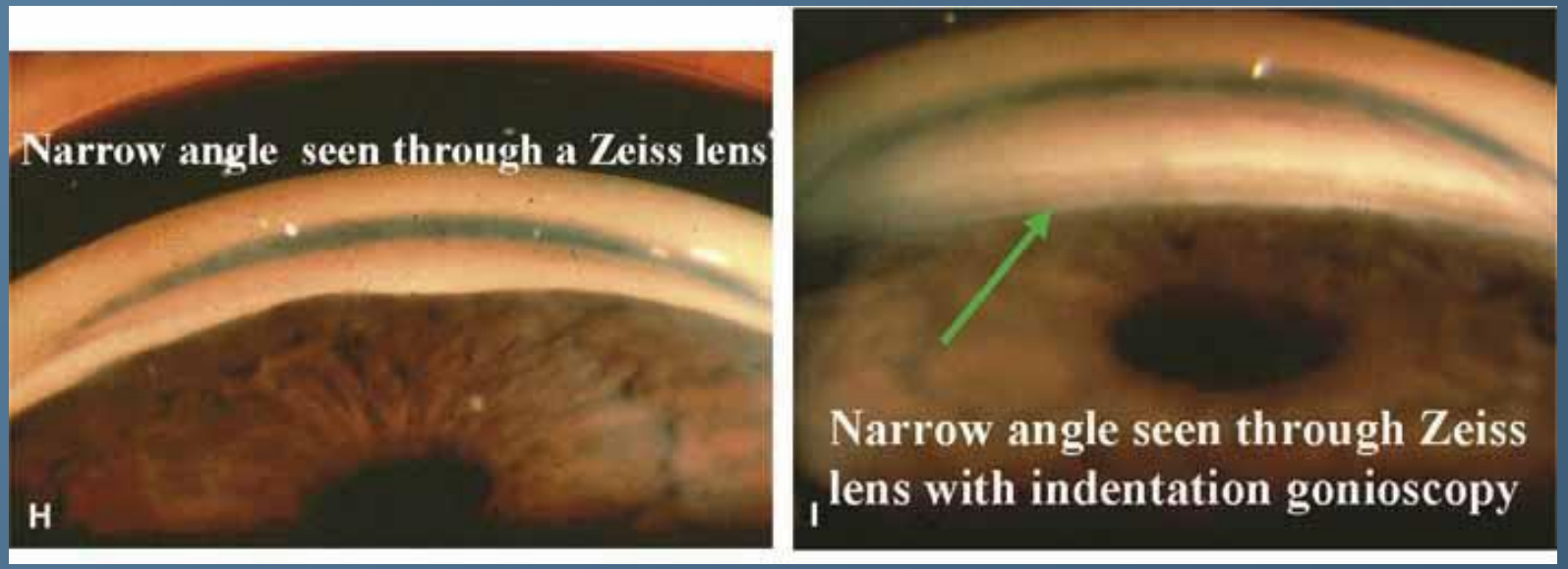
*Aung T et al. the visual field following acute primary angle closure. Acta Ophthalmol Scand 2001;79:298-300.

Classification

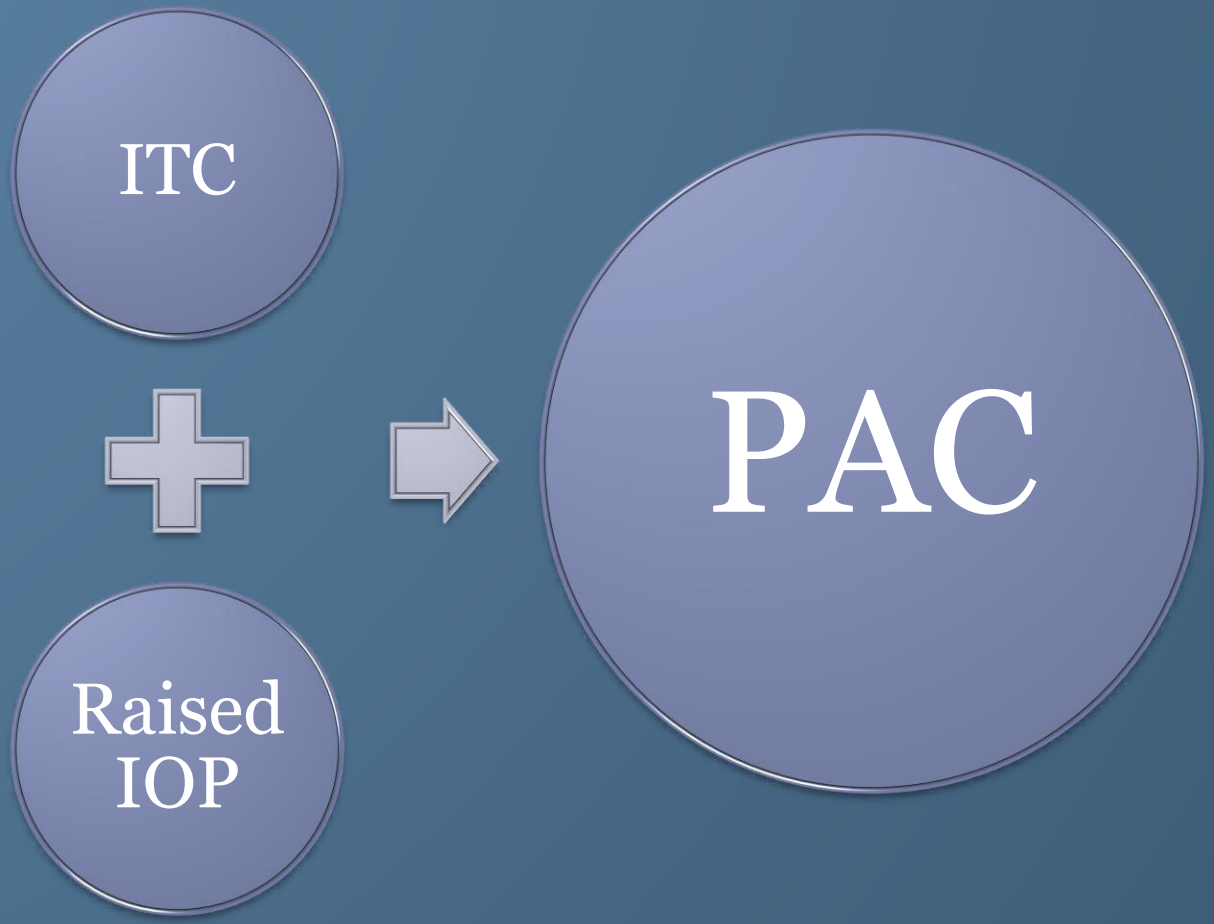
- Based on natural history of angle closure

Stage	Definition
Primary angle closure suspect (PACS)/occludable angles	Iridotrabecular contact (ITC) with normal optic disc, IOP & fields. No PAS. Asymptomatic
Primary angle closure (PAC)	ITC + either raised IOP, PAS or typical symptoms
Primary angle closure glaucoma (PACG)	ITC + structural glaucomatous optic disc changes or glaucomatous visual field loss

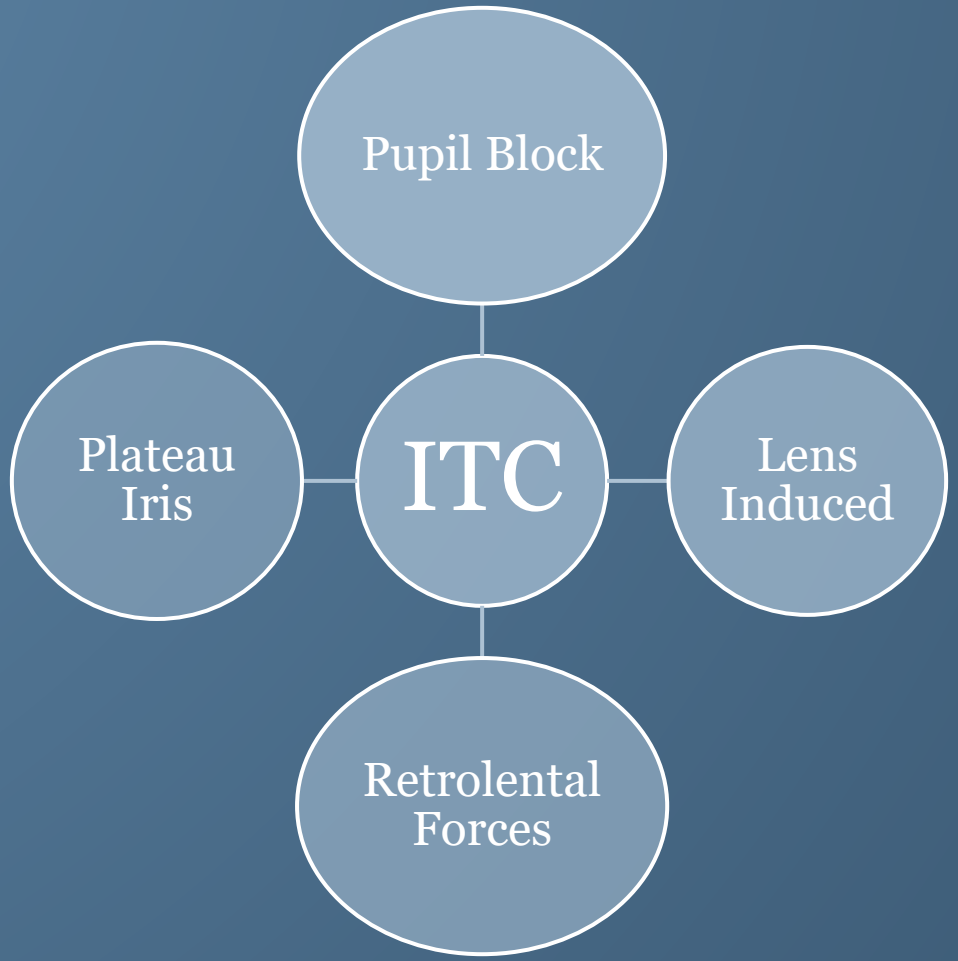
Primary Angle Closure Suspect



Mechanism



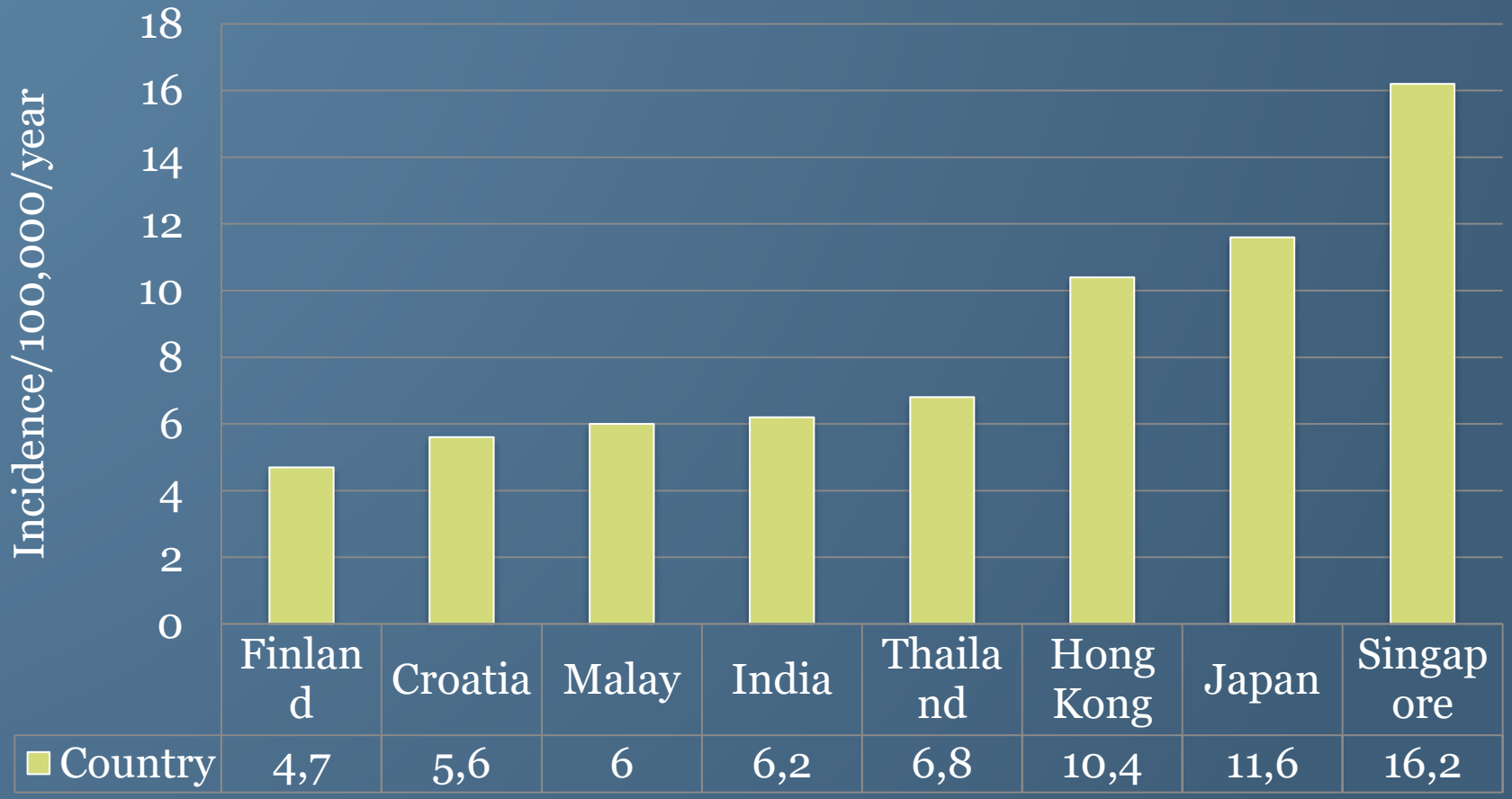
Iridotrabecular contact



Raised IOP

1. Iridotrabecular contact (ITC)…symptomatic angle closure
2. Prolonged appositional ITC leading to PAS
3. Prolonged ITC degrading trabecular meshwork architecture

Incidence (PAC)



Precipitants

- Pharmacological mydriasis…most common recognized event
- Long indoor stay in unpleasant weathers

- Medicines
  - Oral or nebulized ipratropium bromide & salbutamol
  - Tricyclic antidepressants (amitriptyline)
  - SSRI (Paroxetine & Citalopram)
  - Anticholinergic for bladder instability
  - Cold and flu remedies
  - Topiramate

Drug induced angle closure mechanisms

Pupil block	• Anticholinergic (Ipratropium, antihistamine) • Adrenergic • Salbutamol, Naphazoline • TCA, SSRI
Lens-iris diaphragm	• Pilocarpine • Heparin
Choroidal effusion	• Topiramate • Acetazolamide

Risk Factors

- Older age
- Female gender
- Asian parentage
- Singapore Island-wise study*:
  - Female (RR=2.4)
  - Chinese ethnic origin (RR=2.8)
  - Age $\geq$ 60 years (RR=9.1)

*Seath SK et al. Incidence of acute primary angle closure glaucoma in Singapore. An Island-wise survey. Arch Ophthalmol 1997;115:1436-40.

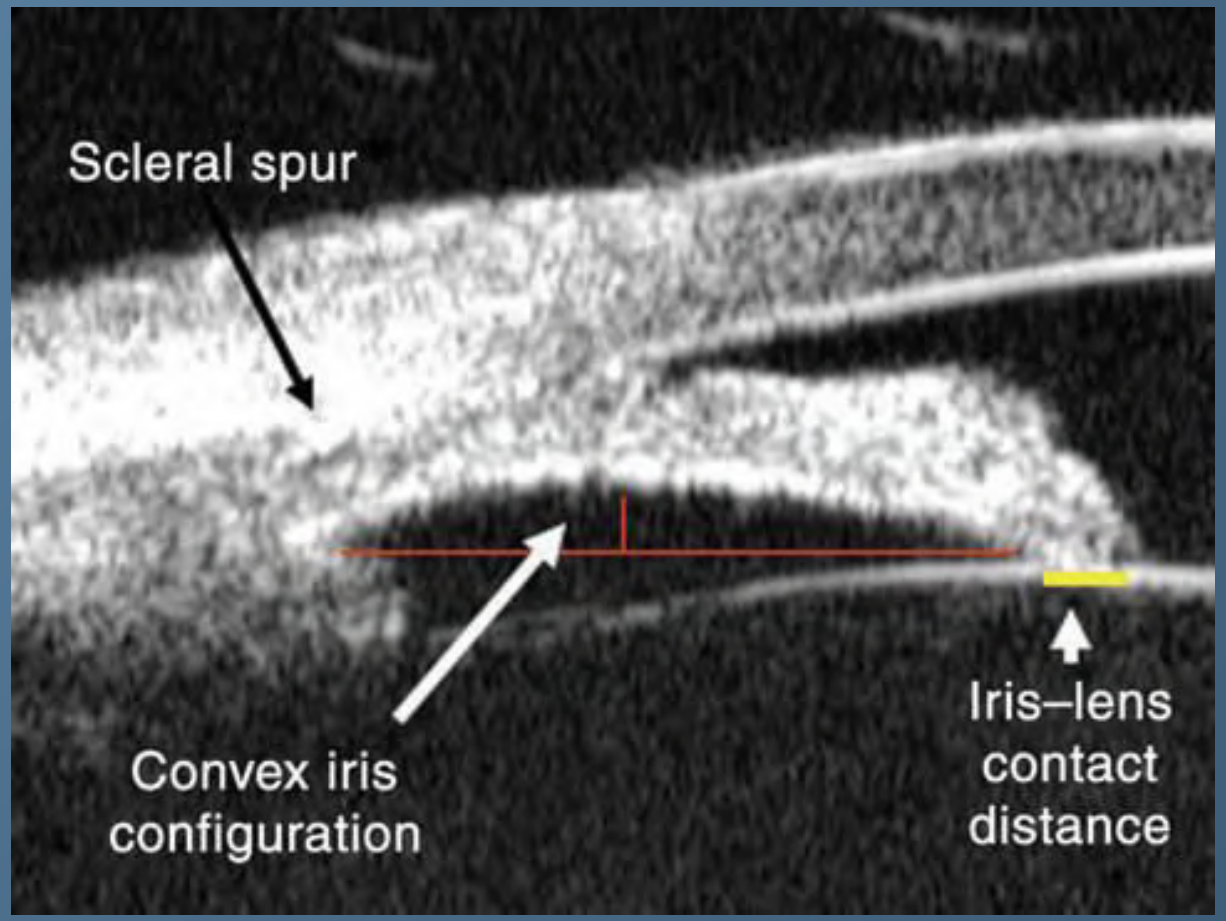
Ocular Risk Factors

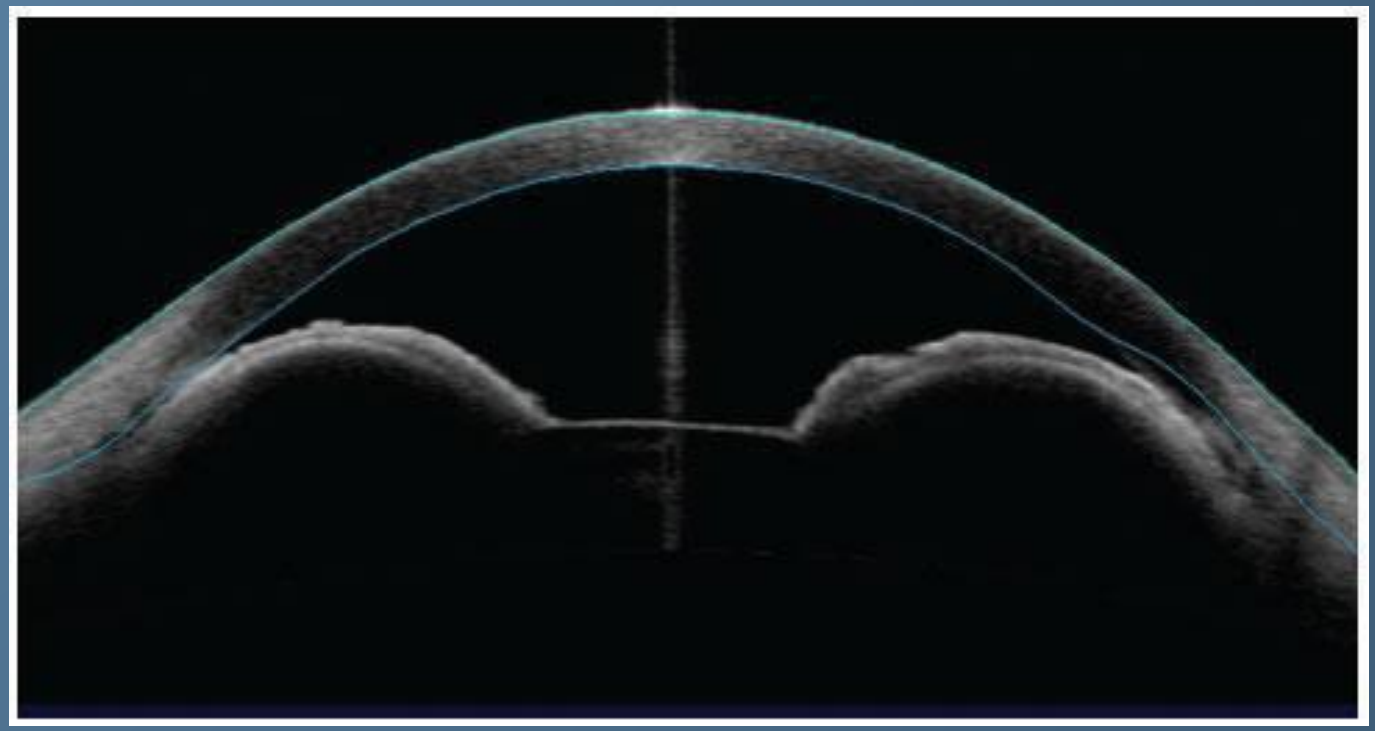
1. Axial length of the globe
   - Shorter axial length
2. Anterior chamber depth
   - Shallow AC
   - Limbal depth of AC is a stronger indicator than axial length
3. Lens position and thickness
   - AC depth is affected by lens

Diagnosing & Testing

- Reference standard… Gonioscopy
- Provocation tests
  - Dark room
  - Pharmacological mydriasis
  - Face-down posture

- Imaging
  - Ultrasound biomicroscopy
  - Anterior segment OCT


Scleral spur
Convex iris configuration
Iris–lens contact distance



Clinical features (Acute attack)

Symptoms

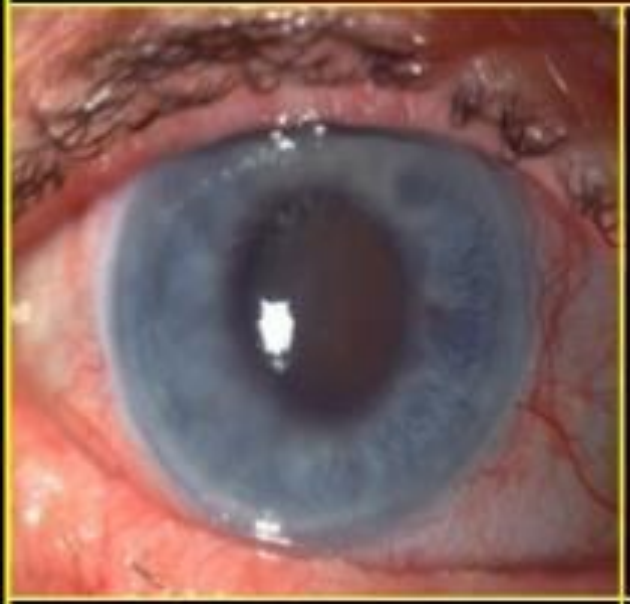
- Ocular pain
- Red eyes
- Headache
- Haloes around light
- Abdominal pain

Signs

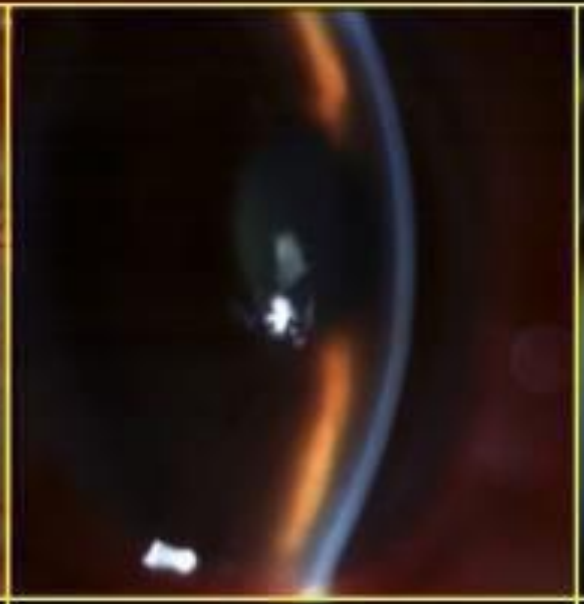
- VA reduced
- Conjunctival hyperemia
- Shallow AC and flare
- Corneal edema
- Mid-dilated pupil
- IOP very high

Acute congestive angle-closure glaucoma

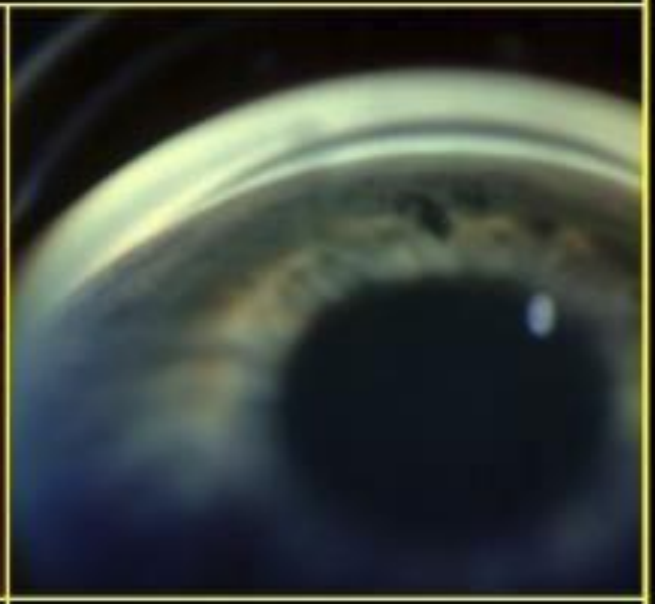
Signs



- Dilated, unreactive, vertically oval pupil



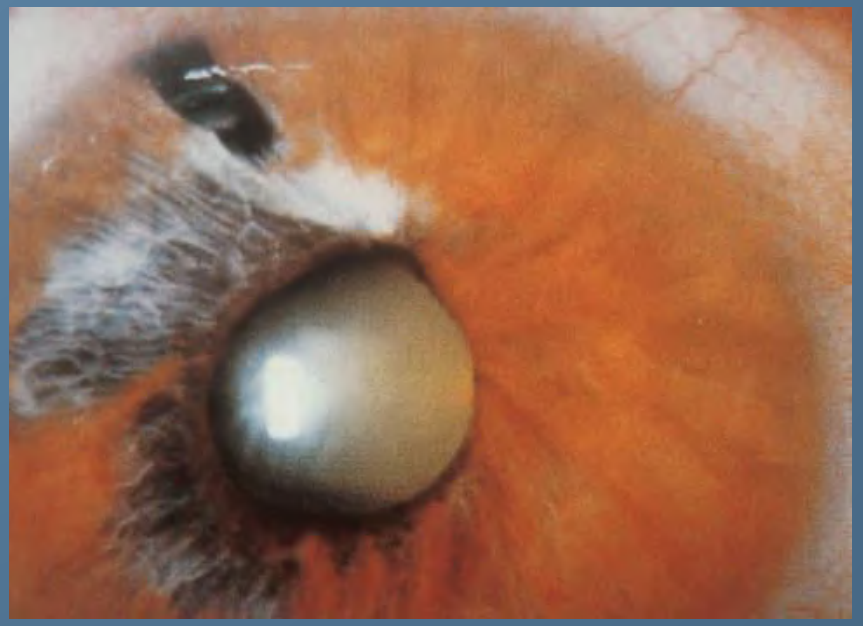
- Shallow anterior chamber

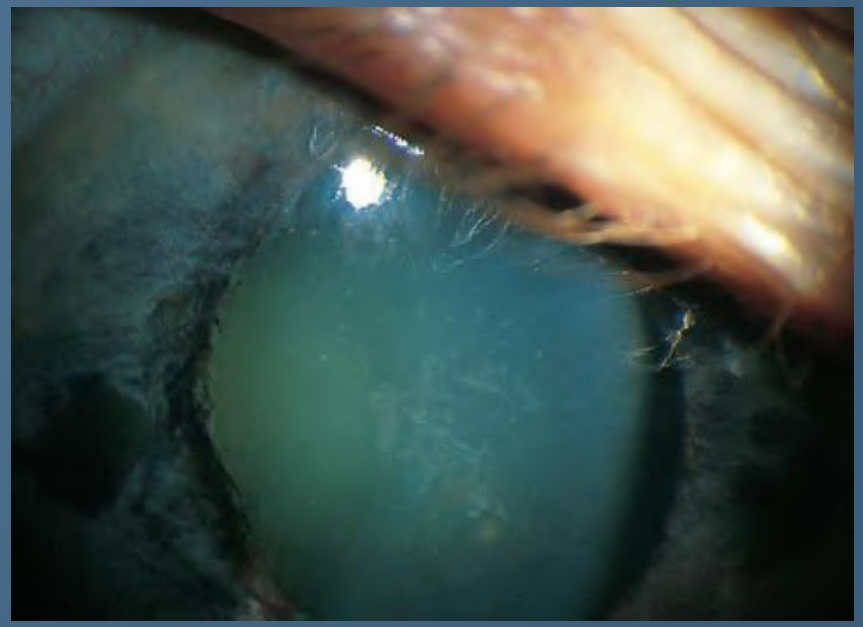


Complete angle closure (Shaffer grade 0)

PACG

- VA normal unless advance damage
- Shallow AC
- Glaukomflecken
- Iris atrophy and PAS
- Peripapillary RNFL loss (4 months)
- Corneal endothelial cell count decreased
  - Raised IOP
  - Toxicity from medicines

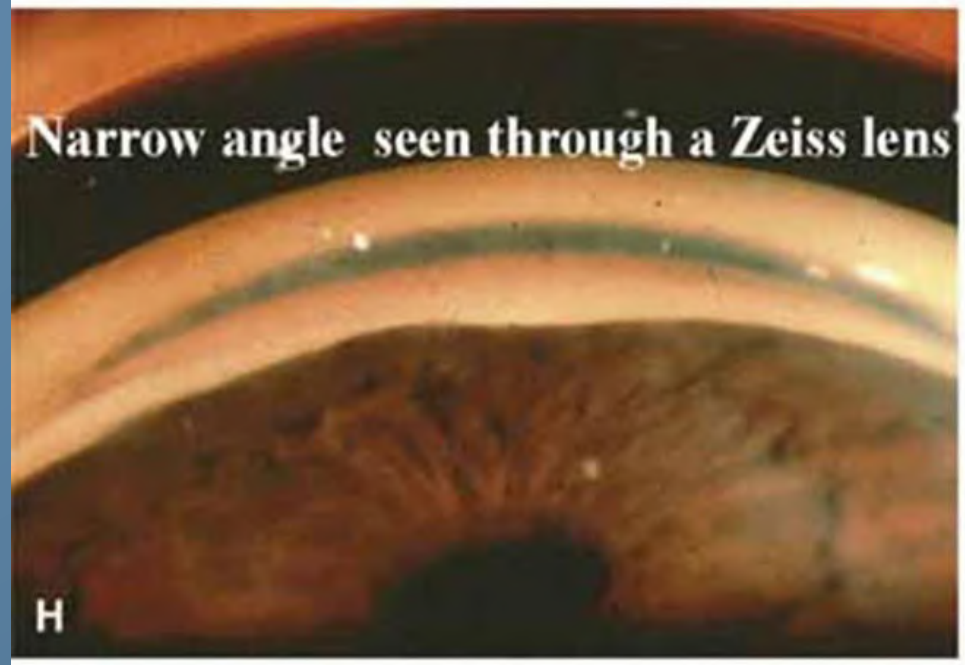


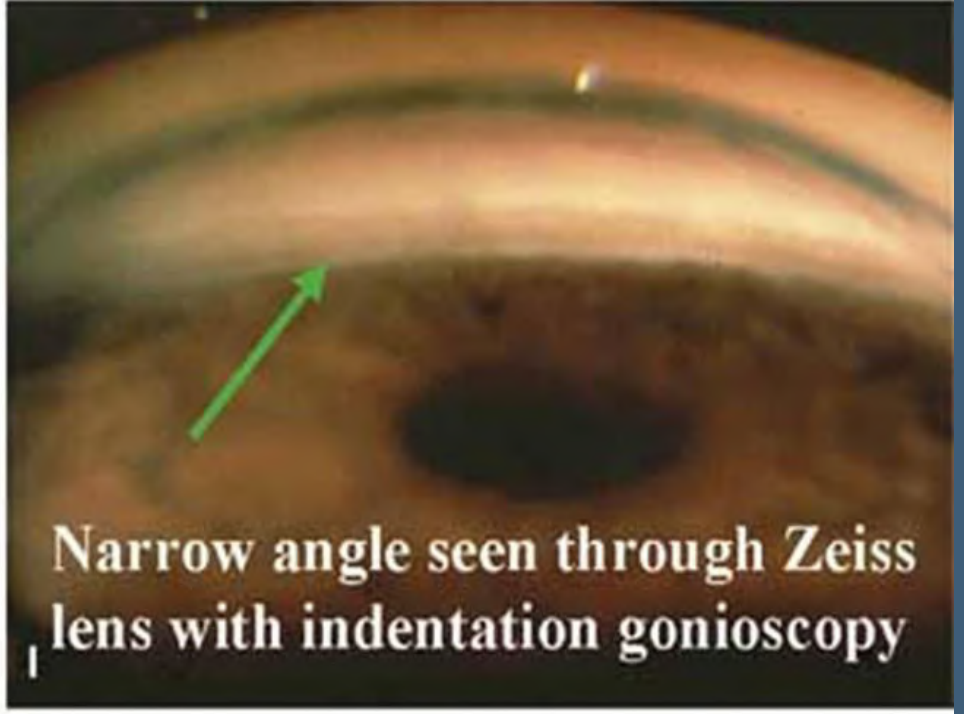


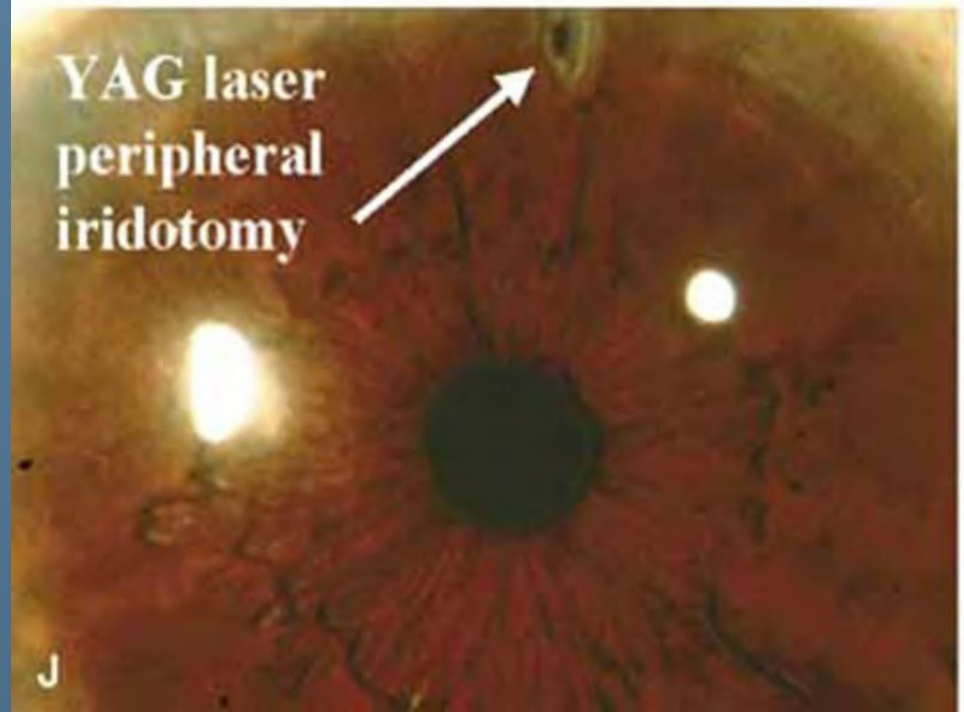
Treatment

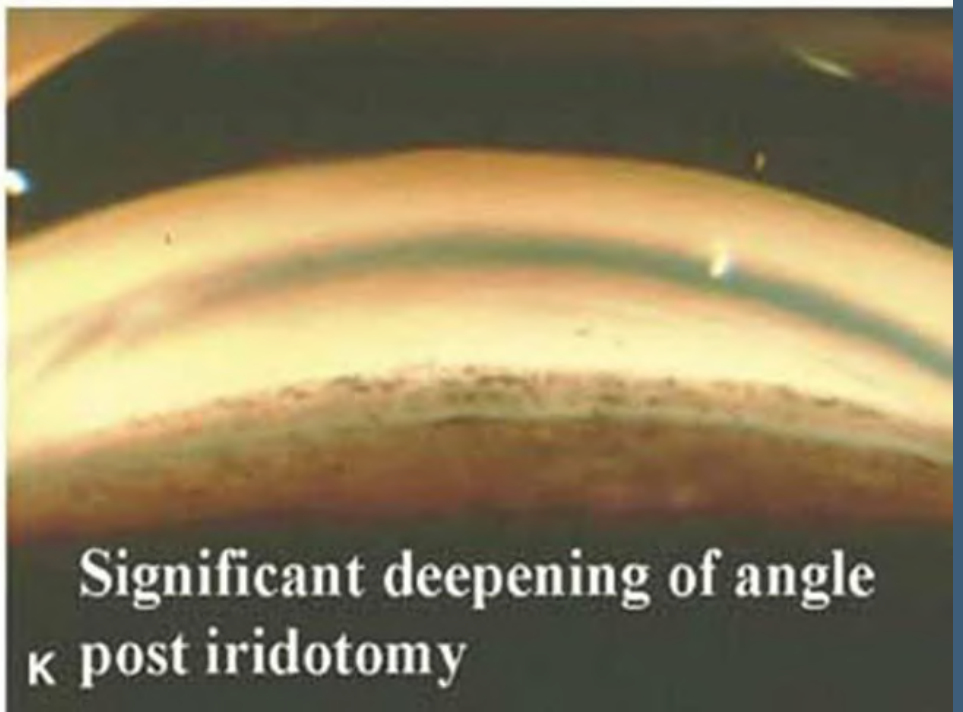
PACS

- Aim
  - To prevent pupillary block
  - Widening of the angles
- Laser iridotomy (the most effective Tx)
- Laser iridoplasty
- Lens extraction if visually significant cataract


Narrow angle seen through a Zeiss lens
H


Narrow angle seen through Zeiss lens with indentation gonioscopy
I


YAG laser peripheral iridotomy
J


Significant deepening of angle post iridotomy
K

Acute Angle Closure

- Emergency
- Intravenous Mannitol / Acetazolamide
- Oral glycerol
- Oral painkillers
- Topical
  - IOP lowering drugs
  - Alpha agonists, beta blockers, CAIs, PGAs, Miotics
  - Glycerol
- Always treat the fellow eye with YLI

Primary angle closure glaucoma

- Low threshold for cataract surgery
- Laser iridotomy for pupillary block
- YLI not effective in synechial angle closure
- Medical agents
  - Same for POAG
  - Prostaglandin analogues outperform b-blockers in PACG*

*Chew PTK et al. Efficacy of latanoprost in reducing intraocular pressure in patients with primary angle closure glaucoma. Surv Ophthalmol 2002;47(Suppl. 1):S125-8

- Surgery:
  - Cataract extraction with synechialysis
  - Trabeculectomy… alone or combined with cataract Sx
  - Glaucoma drainage devices

Prognosis

- Depends on stage of disease
- Untreated:
  - One eye of 33-75% of affected goes blind
  - While 11-27% in Open angle glaucoma
- Earlier stage
  - 75-80% effectively treated with laser iridotomy
  - YLI prevents long-term IOP rise in 90%

Ang LP et al. Acute primary angle closure in an Asian population. Long-term outcome of the fellow eye after prophylactic laser peripheral iridotomy. Ophthalmology 2000;107:2092-6.

Summary

- PACG is rapidly blinding if untreated
- YLI is an effective treatment in early stages

Take home message

- Never forget to treat the fellow eye

Any Questions?

MCQ 1

- A 52 years female presented to emergency with sudden painful visual loss in her right eye. The eye looks congested with hazy cornea and mid dilated pupil. The IOP of that eye is 56 mm of Hg. She denies any systemic illness or trauma.
- What is your most probable diagnosis in this case?

A. Acute angle closure
B. Endophthalmitis
C. Neovascular glaucoma
D. Optic neuritis
E. Panophthalmitis

Ans: A

MCQ 2

- A 52 years female presented to emergency with sudden painful visual loss in her right eye. The eye looks congested with hazy cornea and mid dilated pupil. The IOP of that eye is 56 mm of Hg. She denies any systemic illness or trauma.
- What is best immediate treatment option in this case?

A. Intravenous Mannitol
B. Oral Acetazolamide
C. Topical IOP lowering drugs
D. Topical NSAIDs
E. Topical Steroids

Ans: A

THANK YOU ALL

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MBBS, CHPE, CMEJ, FICO (UK),
MRCSEd (UK), FRCS (Glasg), FCPS
Fellowship in Glaucoma (Al-Shifa Trust, Pak)
Fellowship in Glaucoma (Univ. of Toronto, Canada)
Advance Glaucoma Fellowship (BPOS, UK)

Associate Professor Glaucoma
Department of Ophthalmology
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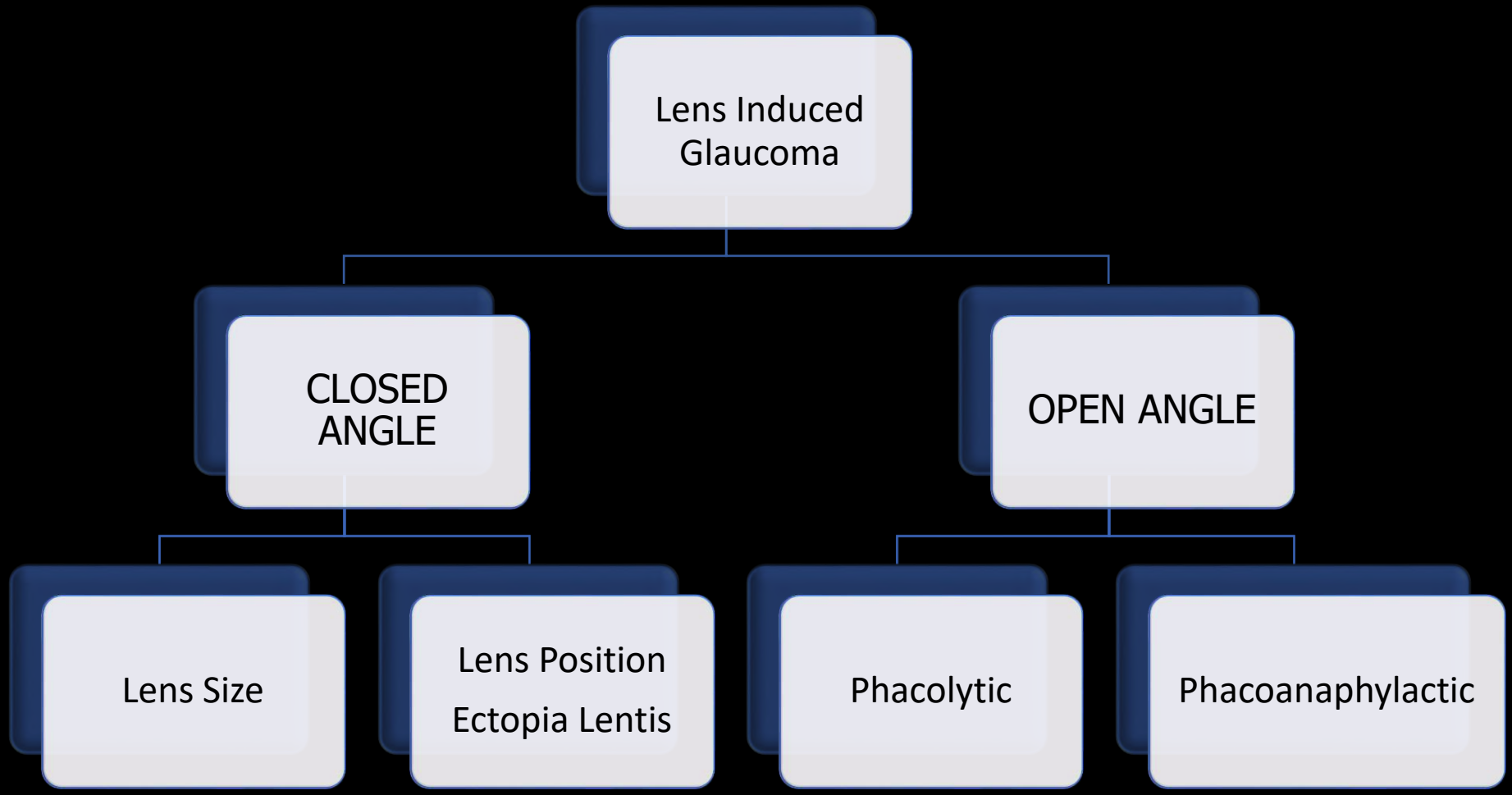
Topics

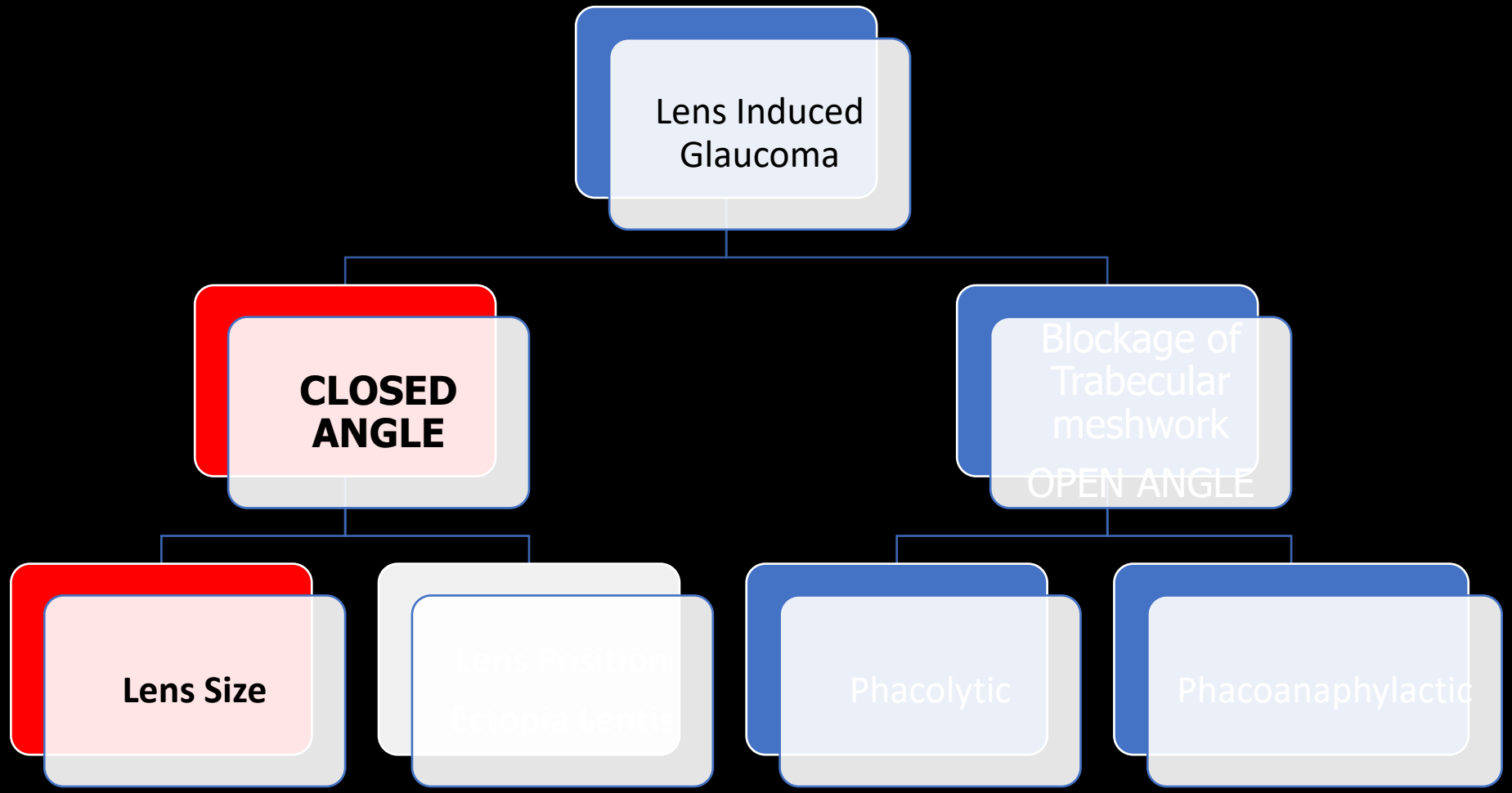
- Lens Induced Glaucoma (LIG)
- Neovascular Glaucoma (NVG)

Learning Objectives

- Discuss
  - Etiology, clinical features, investigation and management of Neovascular glaucoma.
- Discuss
  - Etiology, clinical features, investigation and management of lens induced glaucoma.

Lens Induced Glaucoma


Lens Induced Glaucoma
CLOSED ANGLE
OPEN ANGLE
Lens Size
Lens Position
Ectopia Lentis
Phacolytic
Phacoanaphylactic


Lens Induced Glaucoma
CLOSED ANGLE
Blockage of Trabecular meshwork
OPEN ANGLE
Lens Size
Lens Position
Ectopia Lentis
Phacolytic
Phacoanaphylactic

Closed Angle Lens Induced Glaucoma

1.
   - Predisposing factors
   - Forward movement of lens
2.
   - Relative Pupil Block iris forward bowing
   - Acute angle closure
3.
   - Mechanical angle closure
   - PAS and chronic angle closure

Closed Angle Lens Induced Glaucoma

1

- Predisposing factors
- Forward movement of lens

- Predisposing factors:
  - Shallow AC
  - Short Axial Length
  - Lens dimensions
  - East Asian race

Closed Angle Lens Induced Glaucoma

1

- Predisposing factors
- Forward movement of lens

- Forward movement of lens
  - Aging
    - Lens greater anterior curvature
    - Thicker lens
    - Looser zonues
  - Ectopia Lentis
- Less forward bowing of iris needed to close angle

Closed Angle Lens Induced Glaucoma

1
- Predisposing factors
- Forward movement of lens

2
- Relative Pupil Block iris forward bowing
- Acute angle closure

- What is Pupil Block / Relative Pupil Block?

Closed Angle Lens Induced Glaucoma

1
- Predisposing factors
- Forward movement of lens

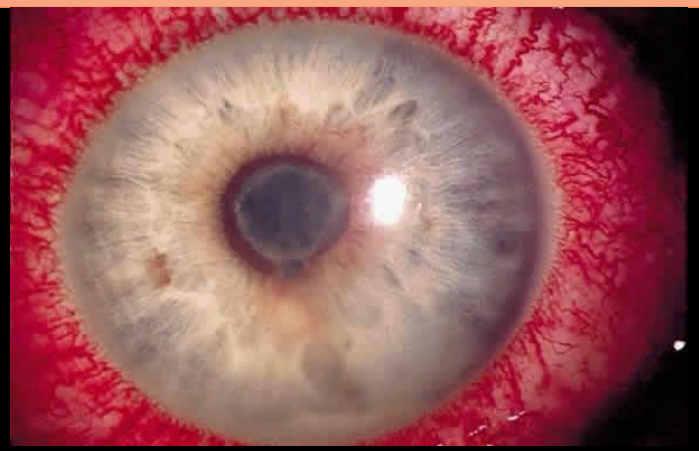
2
- Relative Pupil Block iris forward bowing
- Acute angle closure

- What is Pupil Block?

- “an obstruction to the forward flow of aqueous between the border of the pupil and the anterior capsule of the lens”

Fig. 1. Irido-lenticular pupillary block (iris bombé).

Iridolenticular pupillary block
Iris Bombe
Posterior Synechia binding the pupil margin to the lens capsule. Only “true pupil block”



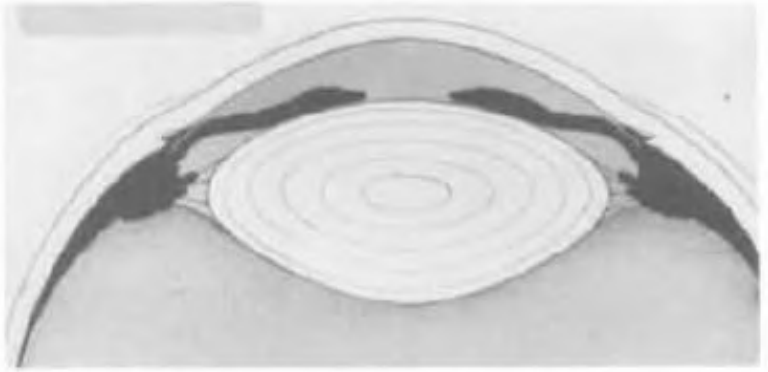
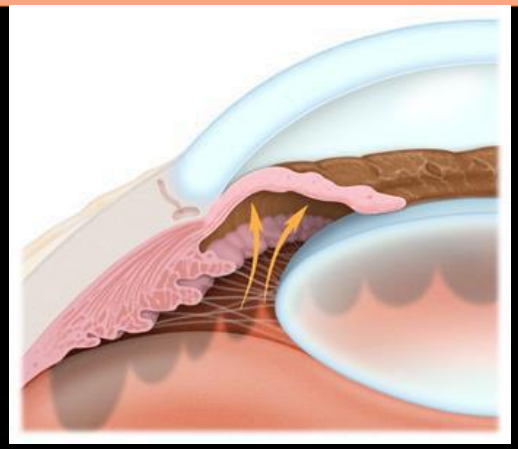


Fig. 2. Irido-lenticular block (relative pupillary block).

Iridolenticular block
Relative Pupil Block
Relative block of aqueous flow in shallow chambered eyes, predisposing to angle closure glaucoma



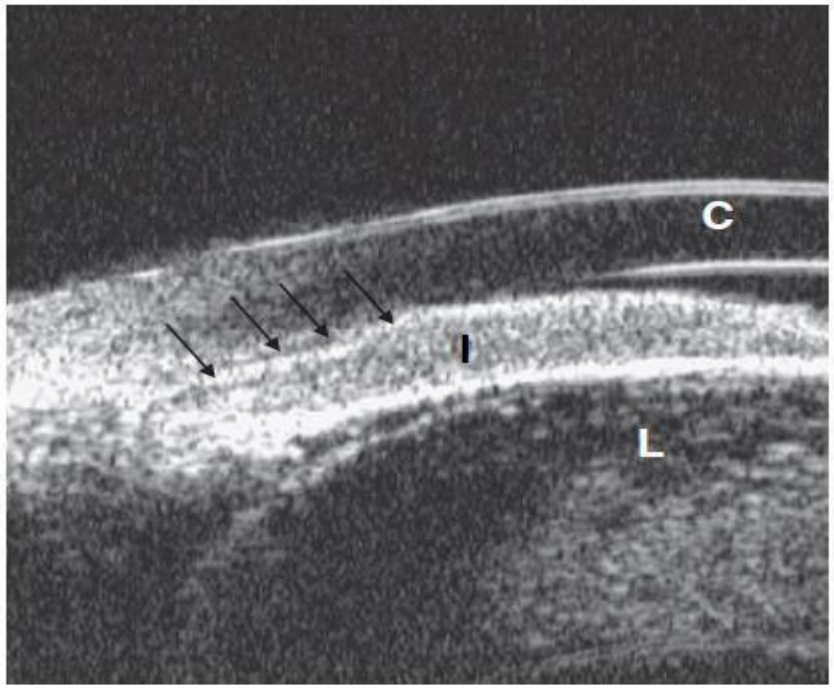
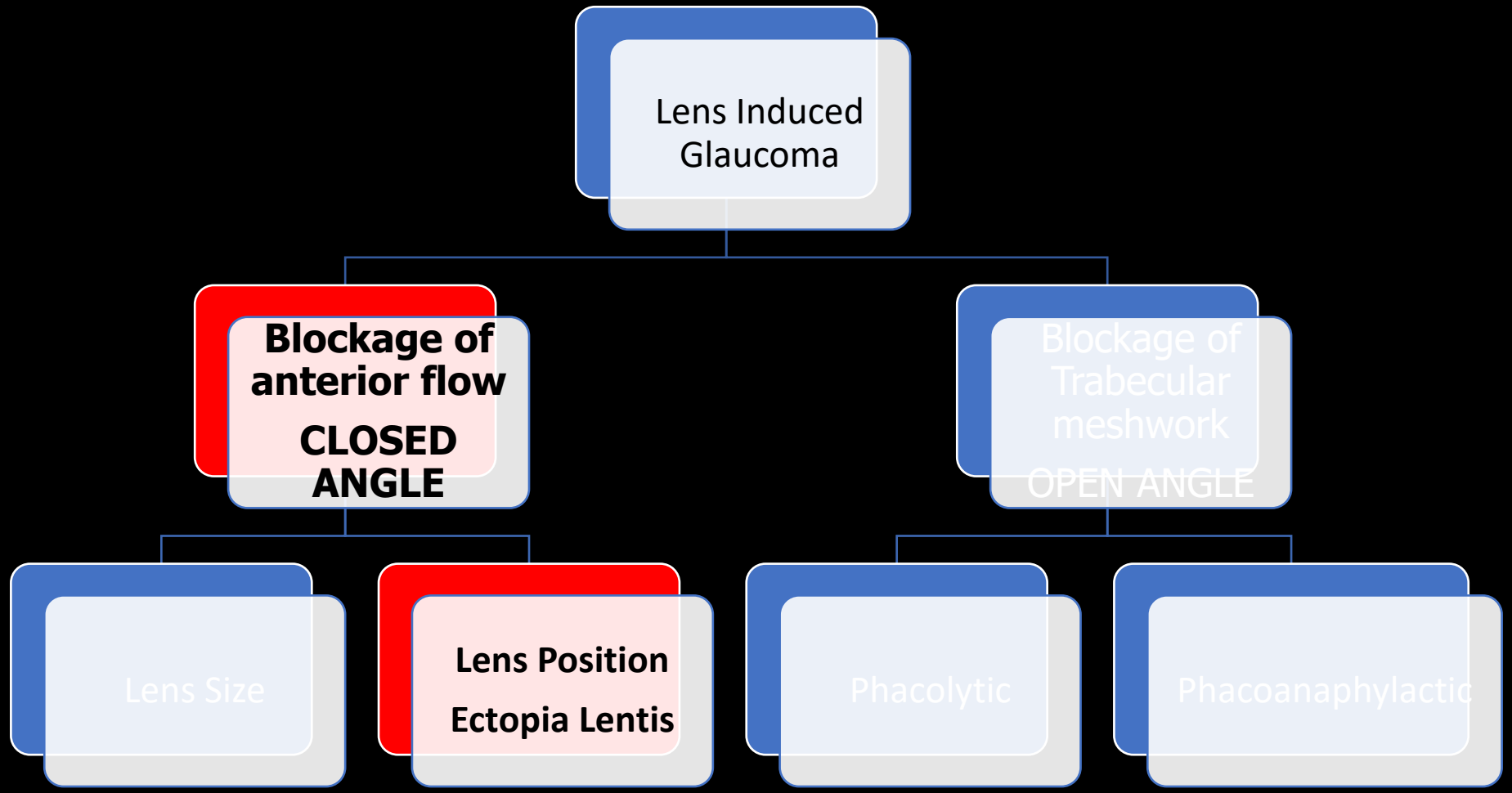


Fig. 5. Phacomorphic angle closure. There is extensive irido-corneal apposition, closed angle, and shallow chamber. Arrows indicate irido-corneal touch. C, cornea; I, iris; L, cataractous lens.

3

- Mechanical angle closure
- PAS and chronic angle closure

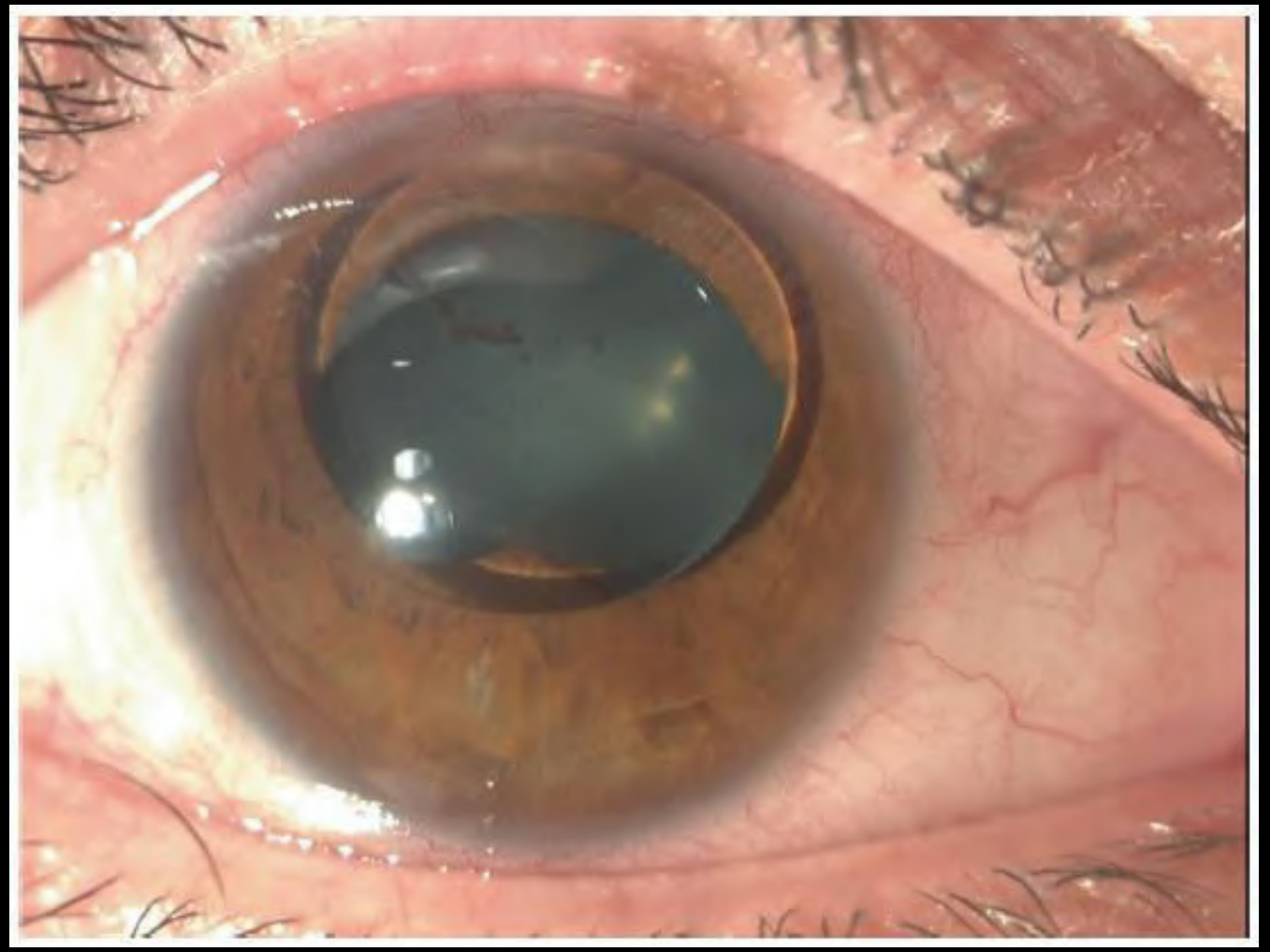
- Lens compresses iris and ciliary body against TM

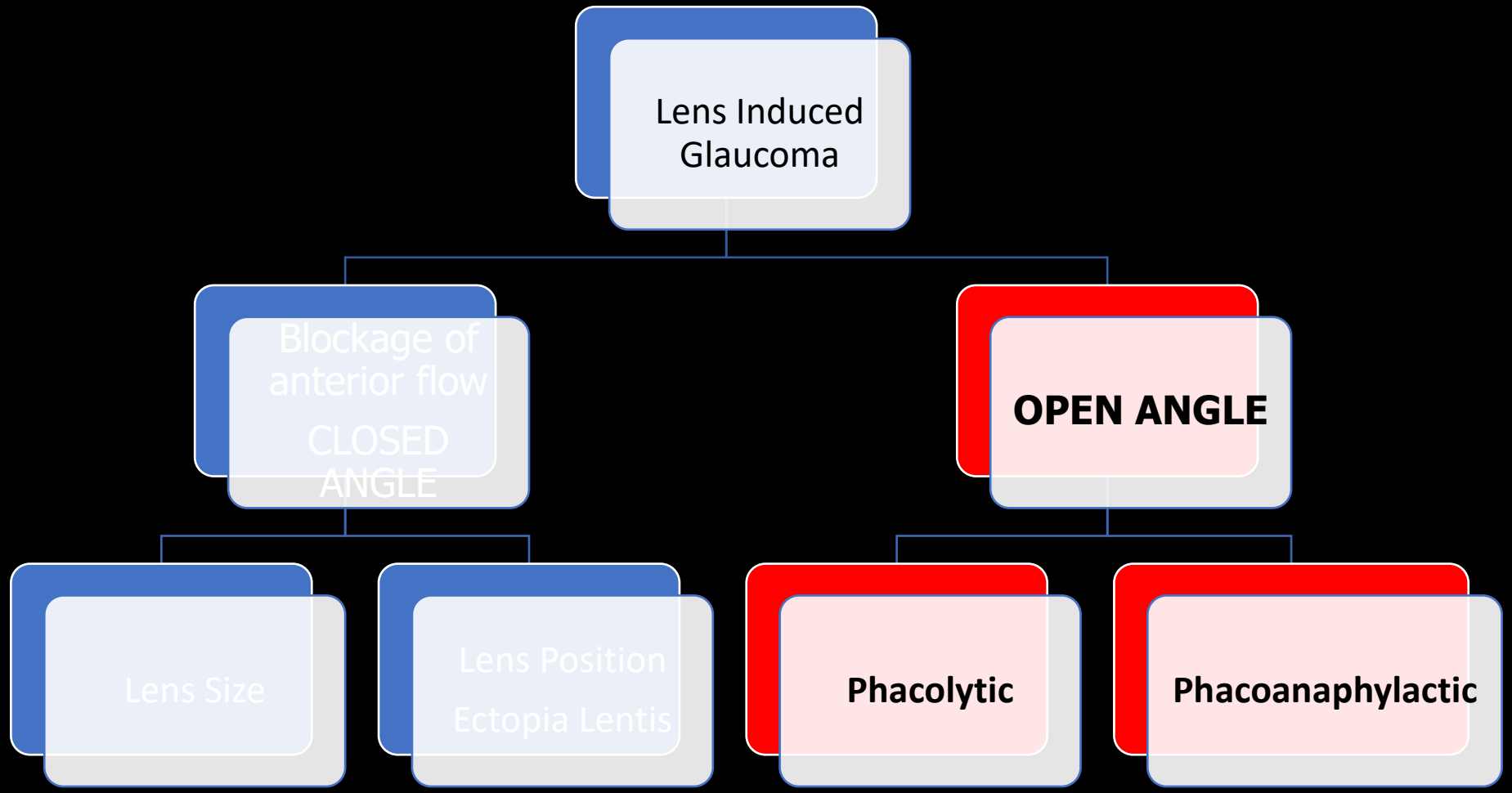

Lens Induced Glaucoma
Blockage of anterior flow
CLOSED ANGLE
Blockage of Trabecular meshwork
OPEN ANGLE
Lens Size
Lens Position
Ectopia Lentis
Phacolytic
Phacoanaphylactic

Ectopia Lentis

- is a displacement or malposition of the Eye's crystalline lens from its normal location
- Forward movement of lens weak zonules

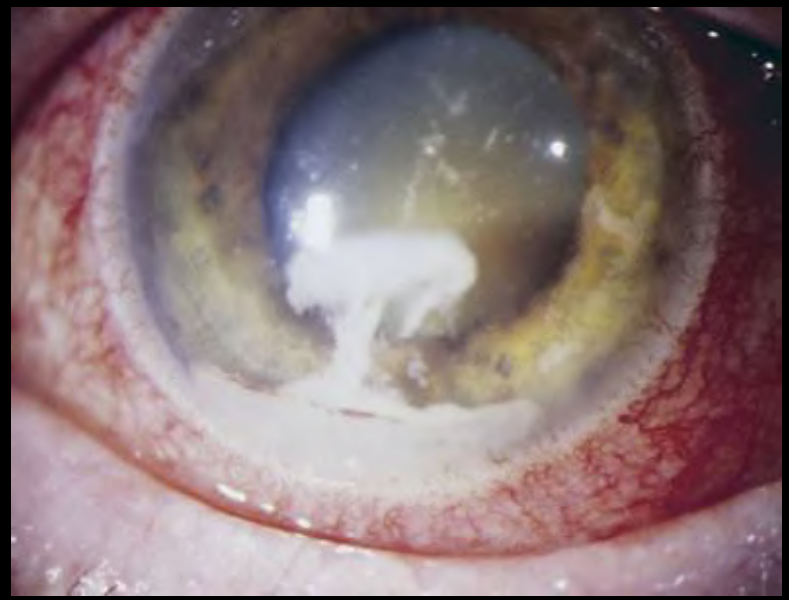
- Present as Acute Angle Closure from sudden forward movement of lens
- Relative Pupil Block
- Chronic Angle Closure




Lens Induced Glaucoma
Blockage of anterior flow
CLOSED ANGLE
OPEN ANGLE
Lens Size
Lens Position
Ectopia Lentis
Phacolytic
Phacoanaphylactic

Phacolytic Glaucoma

- Leakage of lens material through intact capsule
- Senile hypermature cataract
- Red, painful eye, gradual visual loss
- High IOP, corneal oedema, open angles
- Heavy flare, larger cells in aqueous
- Macrophages swollen with eosinophilic lens material they have engulfed
- Heavy Molecular Proteins
- – block TM



Phacoanaphylactic glaucoma
Phacoantigenic

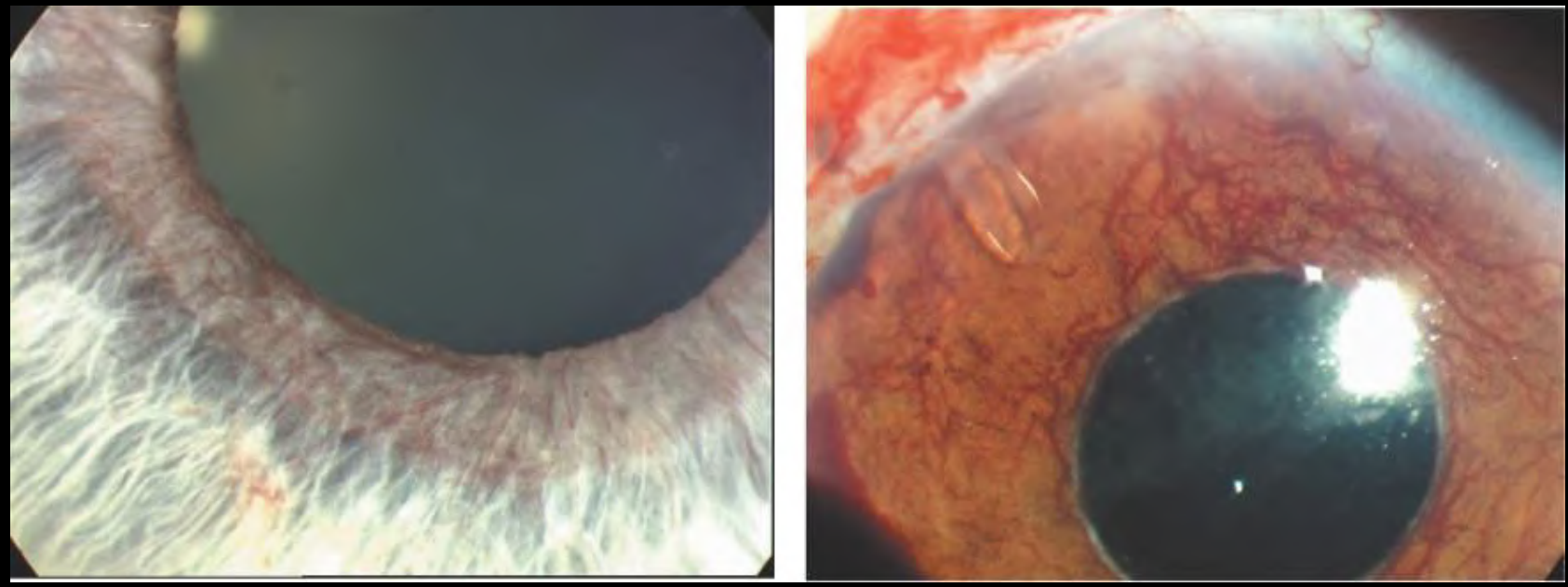
- Inflammation directed against lenticular antigens
- Raised IOP
  - Inflammation of trabecular meshwork
  - Obstruction of TM by inflammatory cells

Treatment

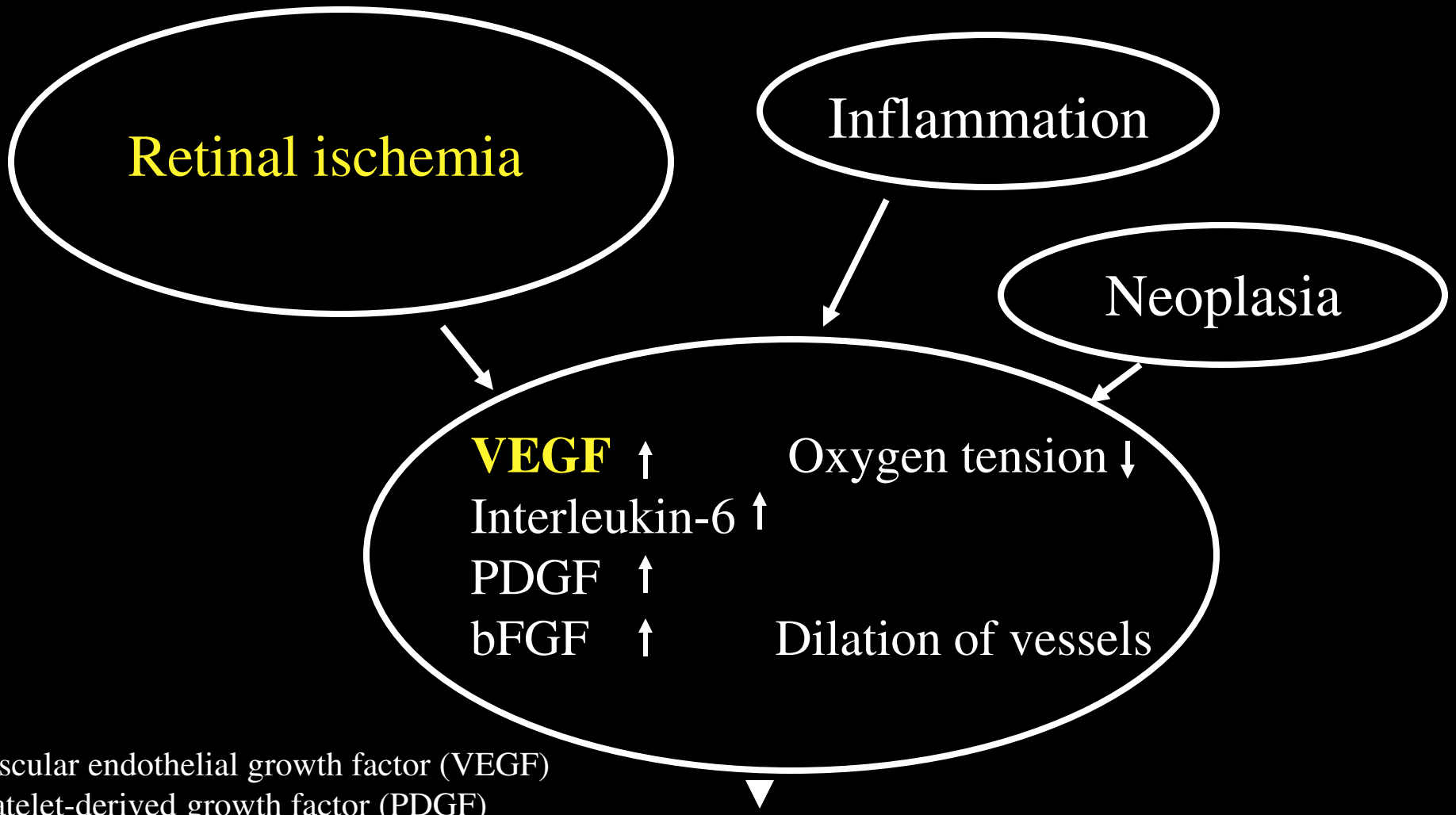
- Control inflammation
- Control IOP
- Remove the cause

Neovascular Glaucoma

Neovascular glaucoma (NVG) occurs when new fibrovascular tissue proliferates onto the chamber angle, obstructs the trabecular meshwork, and produces PAS and progressive angle closure.



Pathogenesis



Vascular endothelial growth factor (VEGF)
Platelet-derived growth factor (PDGF)
Basic fibroblast growth factor (bFGF)

Causes of neovascular glaucoma

- Retinal ischaemic disease
- Inflammatory diseases
- Tumours
- Radiation
- Surgical causes

Retinal Ischaemic disease

1. Diabetic retinopathy

Patients with long-standing diabetes with PDR

Risk ↓ appropriate PRP

↑ cataract extracion

(particularly if the posterior capsule is breached)

2. Central retinal vein occlusion: CRVO

Ischemic CRVO ⟶ 50% of eyes develop NVG

"100 –day glaucoma"

3. Ocular ischemic syndrome

Inflammatory diseases

- Uveitis: chronic iridocyclitis, Behcet disease, Vogt-Koyanagi-Harada syndrome
- Syphlitic retinitis
- Sympathetic ophthalmia
- Endophthalmitis

Ophthalmology *Volume 108, Number 10, October 2001*

Tumors

Iris: melanoma, hemangioma, metastatic lesions

Ciliary Body: ring melanoma

Retina: Retinoblastoma, Large cell lymphoma,

Choroid: melanoma

Conjunctiva: squamous cell carcinoma

Ophthalmology *Volume 108, Number 10, October 2001*

Radiation

- External beam
- Charged particle: proton, helium
- Plaques
- Photoradiation

Ophthalmology *Volume 108, Number 10, October 2001*

Surgical causes

- Carotid endarterectomy
- Cataract Extraction
- Pars plana vitrectomy/lensectomy
- Silicon oil
- Scleral buckle

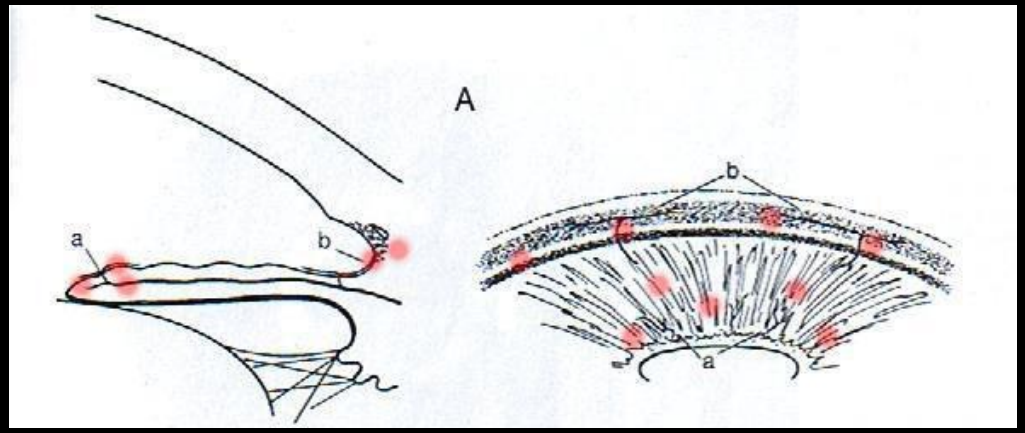
Ophthalmology *Volume 108, Number 10, October 2001*

Classification/Staging

1) Rubeosis iridis (Preglaucoma stage)
2) Secondary open-angle glaucoma
3) Secondary synechial angle-closure glaucoma

1) Rubeosis iridis (Preglaucoma stage)

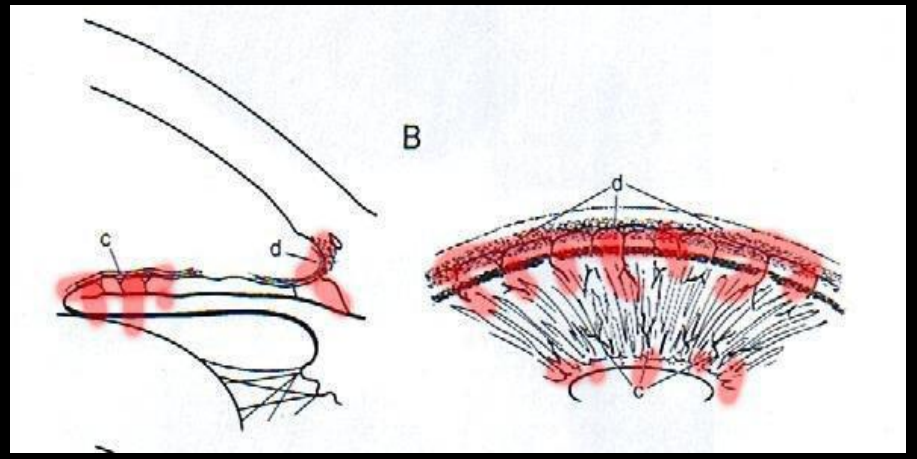
- Tiny dilated capillary tuffs or red spots develop at the pupillary margin.
- The new vessels grow radically over the surface of the iris.
- At this stage the IOP is within normal range.

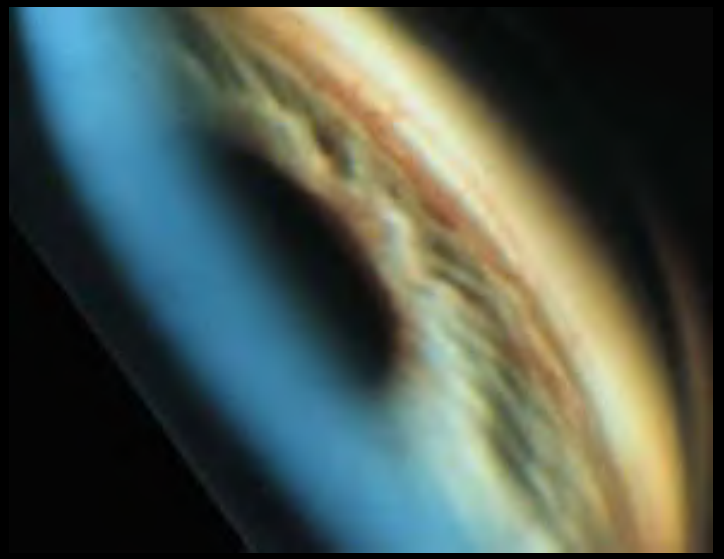


Rubeosis iridis on the pupillary border

2) Secondary open-angle glaucoma

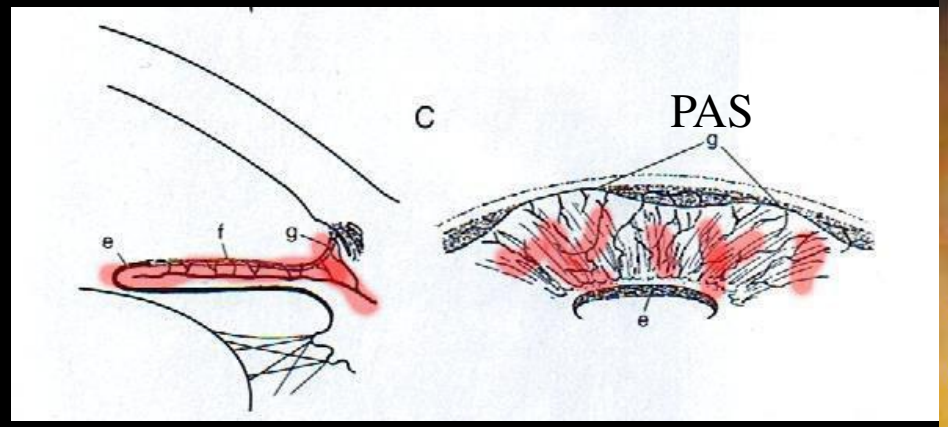
• The new vessels continue to grow across the iris surface towards the iris root.
• The new vessels arborize and form a fibrovasucular membrane, which blocks the trabecular meshwork and gives rise to a secondary open-angle glaucoma.

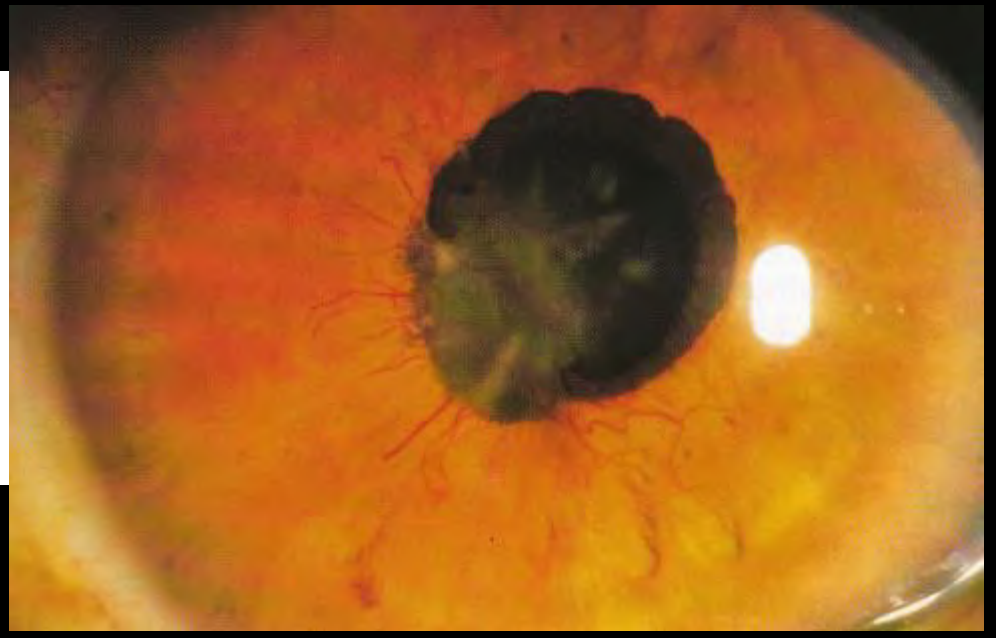




3) Secondary angle-closure glaucoma

• This is caused by contraction of fibrovascular tissue in the angle with pulling of the peripheral iris over the trabecular meshwork.
• The iris become flattened and ectropion uvea is present.



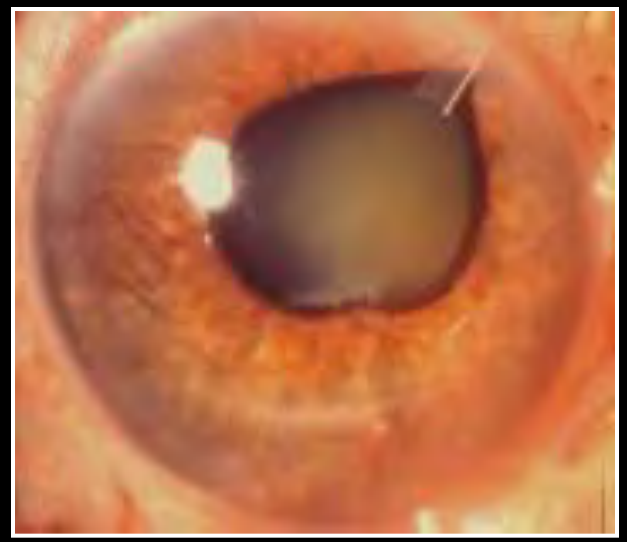


Distortion of the pupil and ectropion uvea.

Neovascular Glaucoma: Signs

In addition to new vessels:

- Mild anterior chamber reaction
- Conjunctival injection
- Corneal oedema
- Ectropion uveae
- Glaucomatous optic nerve and field defects



Diagnosis

Clinicians should maintain a high level of suspicious about neovascularization of iris or angle

1) Medical History
2) Visual acuity, IOP
3) Pupil
4) GONIOSCOPY

    10% of non ischemic CRVO have NVA without NVI

5) Dilated fundus examination

Differential diagnosis

- Primary congestive angle-close glaucoma
- Uveitic glaucoma
- Postvitrectomy inflammation
- Haemolitic glaucoma / Ghost cell glaucoma

Treatment

1) Treatment of the underlying disease process responsible for rubeosis
   - A) Panretinal photocoaguration (PRP)
   - B) Anti-inflammatory agents

2) Treatment of the high IOP
   - A) Medical management
   - B) Surgical treatment
   - C) Intravitreal Bevacizumab (Avastin)

Summary

- Painful visual loss
  - NVG
  - LIG

Thank you

Dr. Yousaf Jamal Mahsood

MBBS, CHPE, CMEJ, FICO (UK),
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Fellowship in Glaucoma (Al-Shifa Trust, Pak)
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Advance Fellowship in Glaucoma (BPOS, UK)

Assistant Professor Glaucoma
Department of Ophthalmology
Khyber Girls Medical College
Peshawar

Treatment Options in Glaucoma

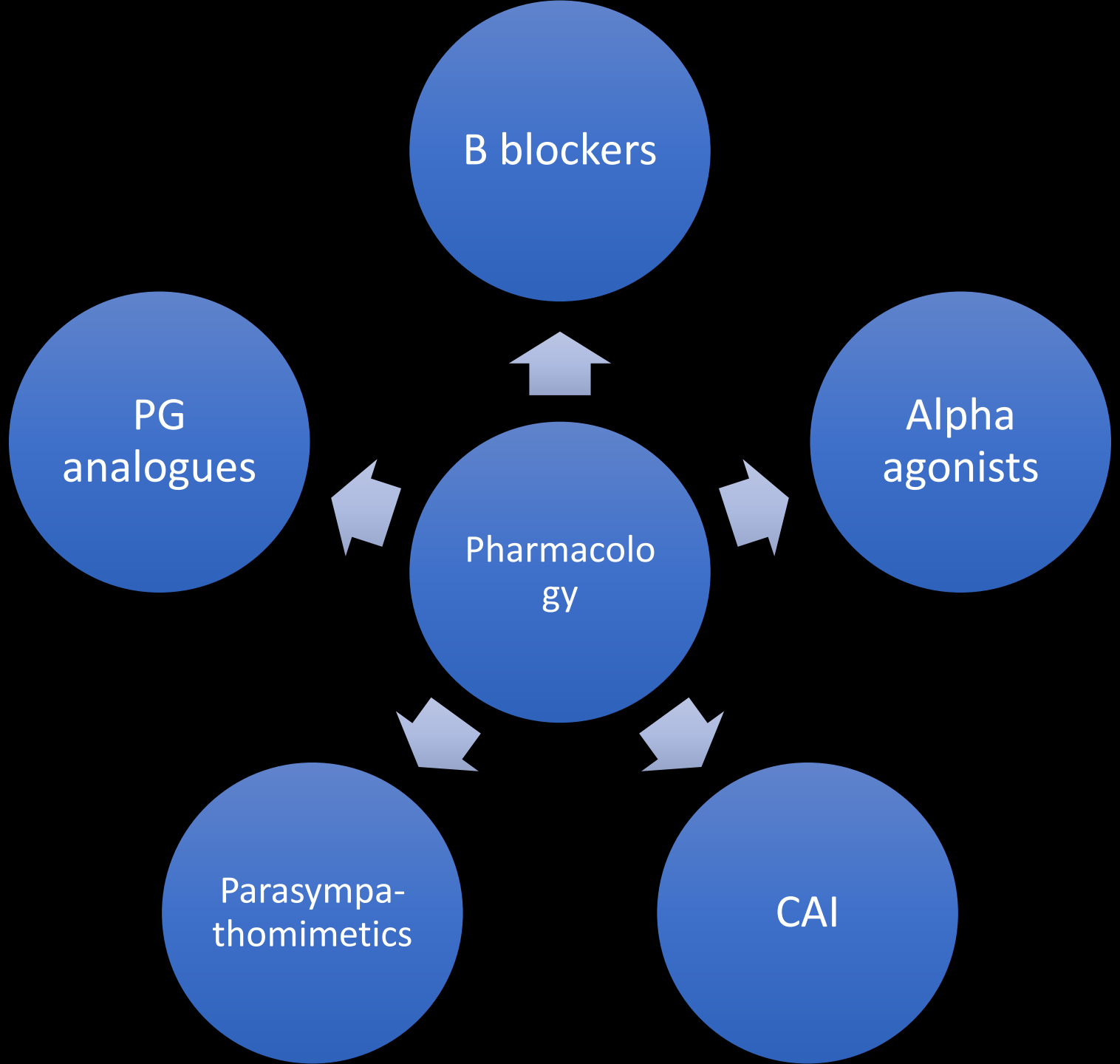
Learning Objectives

- Enumerate
  - Different treatment options in glaucoma
- Discuss
  - Indications of each treatment option

Treatment Options

- Medical
- Lasers
- Surgery

Medical Tx


B blockers
PG analogues
Alpha agonists
Pharmacology
Parasympa-thomimetics
CAI

Drug Classes

Drug class	Medication	Mean ↓ IOP	Percent ↓ IOP
PGF2 analogue	Latanoprost Bimatoprost Travoprost	6-8 mm Hg 7-8 mm Hg 7-8 mm Hg	25-30%
Beta-blocker (non selective)	Timolol	~ 6mm Hg	20-30%
Beta-blocker (selective)	Betaxolol	4-5 mm Hg	15-20%
Alpha-2 agonist	Brimonidine	2-6 mm Hg	15-20%
CAI	Dorzolamide	3-5 mm Hg	15-20%

Lasers

- Laser Peripheral Iridotomy (LPI)
- Argon Laser Trabeculoplasty (ALT)
- Selective Laser Trabeculoplasty (SLT)
- Diode cyclodestruction
  - Trans-Scleral Diode (TSD)
  - Endoscopic Cyclo-Photoablation (ECP)

Thermal Effects

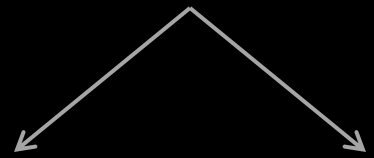
Target tissue absorbs laser E

↓

↑ temp

↓

Induced chemical changes



Local inflammation + scarring (photocoagulation)

Vaporizes intra+extra- cellular fluids →incision in tissue (photocoagulation)

Q Which laser is the photo**coagulator** prototype?

Q Which laser is the photo**disruptor** prototype?

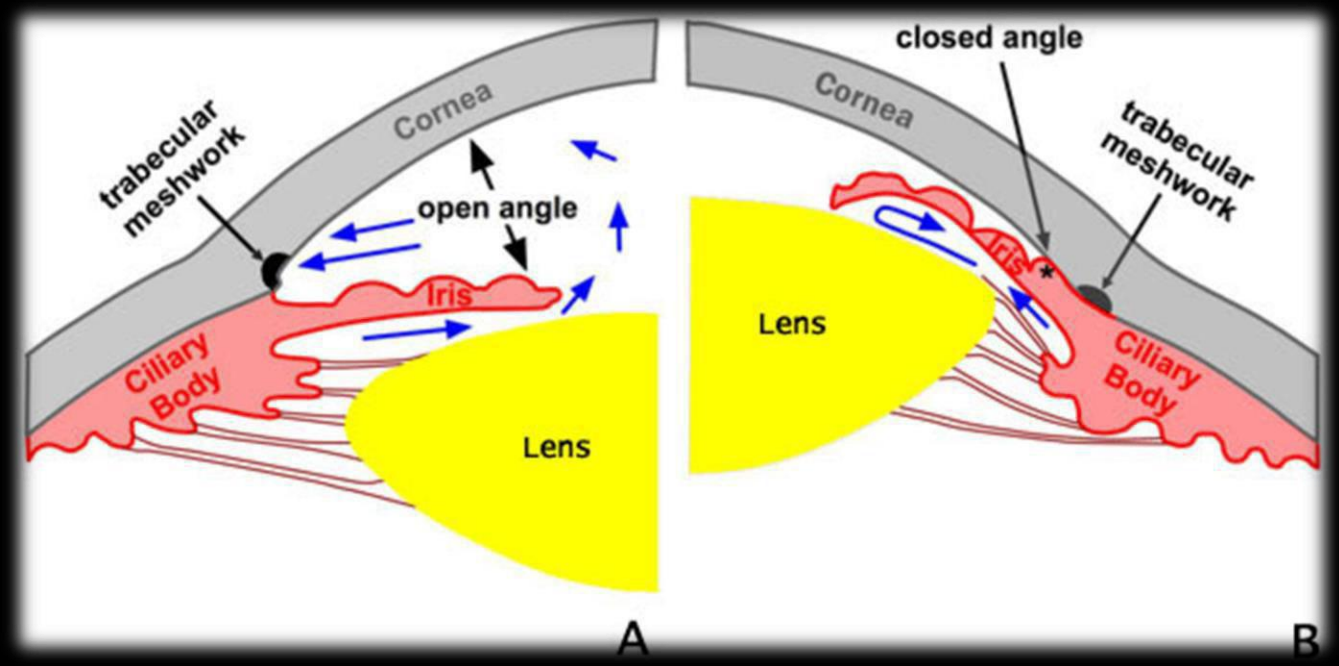
Q Which laser is the photo**coagulator** prototype?

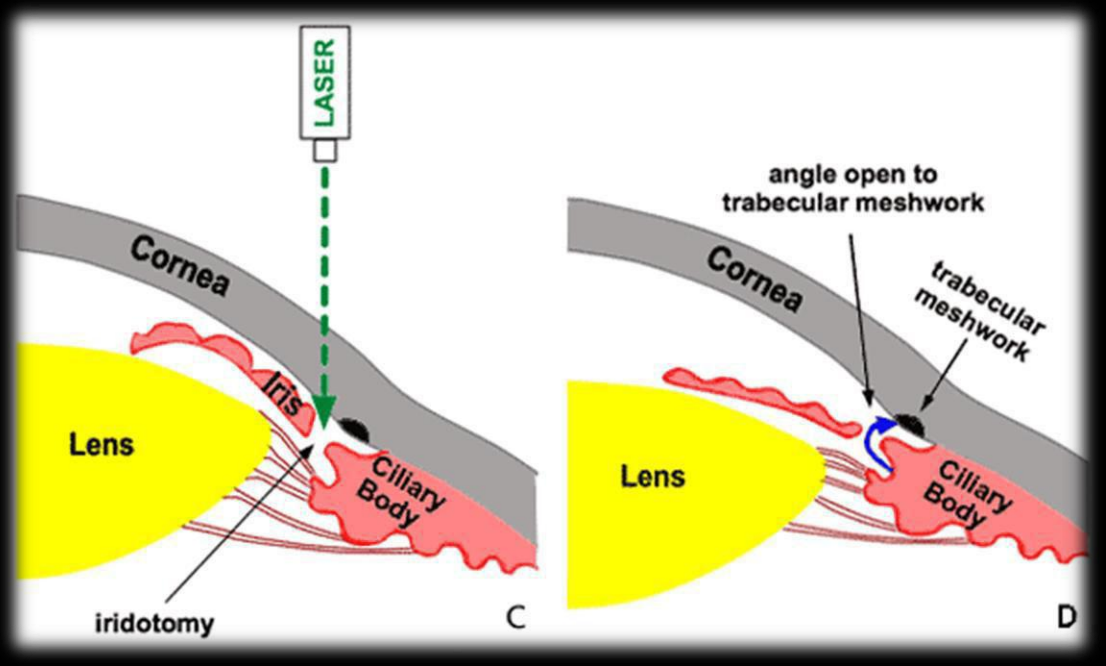
A Argon Laser

Q Which laser is the photo**disruptor** prototype?

A Neodymium:YAG laser

Laser Peripheral Iridotomy





Glaucoma Laser Trial

(Ophthalmology 97:1403,1990)

Demonstrated that ALT was an alternative to topical medical therapy in patients with newly diagnosed POAG.

Glaucoma Laser Trial

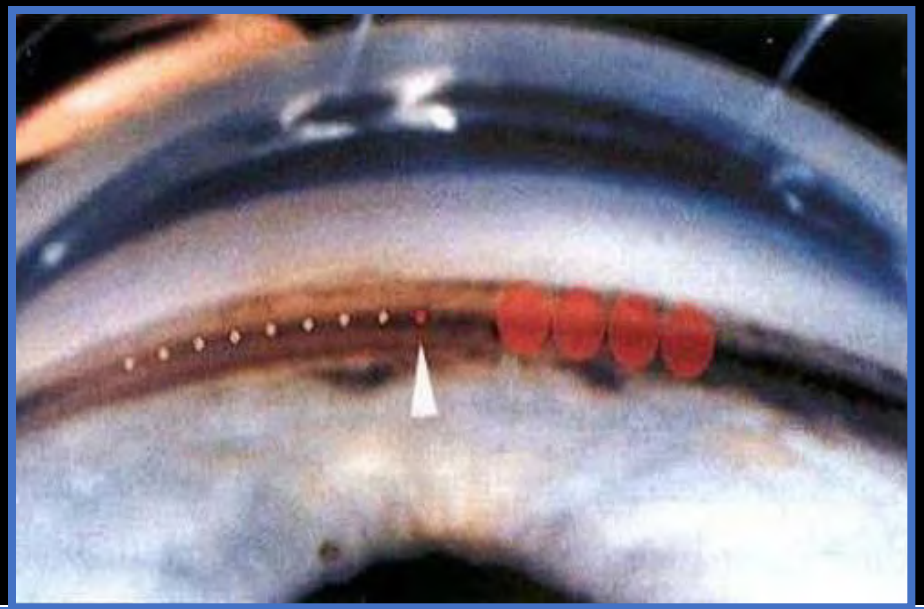
(Ophthalmology 97:1403,1990)

Demonstrated that ALT was an alternative to topical medical therapy in patients with newly diagnosed POAG:

- IOP controlled at 2 years:
  - Laser: 44% ALT alone
    70% ALT + timolol
    89% ALT + meds
  - Medication: 30% timolol alone
    66% multiple meds

- **BUT** ALT is not used as a primary treatment option for glaucoma

- Why?
  - ALT requires operator expertise
    - MUST identify the TM
    - hitting other structures significant consequences such as PAS and K endothelial cell loss
  - Waning effect with longer follow-up (50% at 5years, 75% at 10 years)
  - Structural changes are permanent ∴ repeatability is a problem



ALT

- Argon laser (514nm)
- Spot size = 50um
- 50 spots
- Energy = 500 mW
- Fluence = 40,000 mj/mm²
- Exposure Time = 0.1 s
- **Thermal Damage**

SLT

- Frequency doubled YAG (532nm)
- Spot size = 400um
- 50-60 spots
- Energy < 1% of ALT
- Fluence < .00015% of ALT
- Exp T = 0.0000000003 s
- **No Thermal Damage**

ALT & SLT

- Application of laser energy to the trabecular meshwork
- Result in an improvement in aqueous outflow facility (mechanism still not well understood)
- Has no influence on aqueous production

1. Mechanical theory: thermal burn contracts tissue and stretches open adjacent, untreated regions of the meshwork to increase outflow (ALT only)

2. Cellular model suggests that laser stimulates the replication of trabecular endothelial cells that normally do not divide

3. Biochemical mediators (cytokines) are released and macrophages are recruited into the laser treatment zone

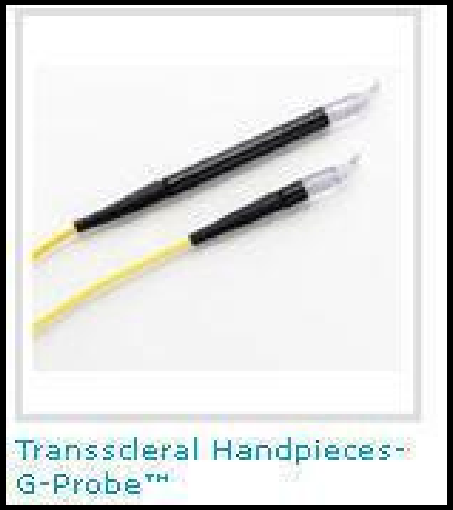
Mean IOP	ALT	SLT
baseline	22.5	22.8
1 mo.	19.5	20.1
6 mos.	17.7	17.8

- SLT has an equal IOP lowering effect to ALT
- SLT is easy to administer
- SLT does not cause structural damage
- SLT is theoretically repeatable

Trans-Scleral Diode Indications

- Refractory glaucomas:
  - NVG, trauma, aphakia, congenital, uveitis, PKP, silicon oil, conjunctival scarring, multiple tubes
- Glaucoma and low vision (worse than 6/60)
- Blind painful eye with high IOP

Diode laser



Transscleral Handpieces-
G-Probe™

TSD - Mechanism of Action

- Destruction of ciliary epithelium
- Vascular supply destruction
- Increased outflow through pars plicata

TSD - Complications

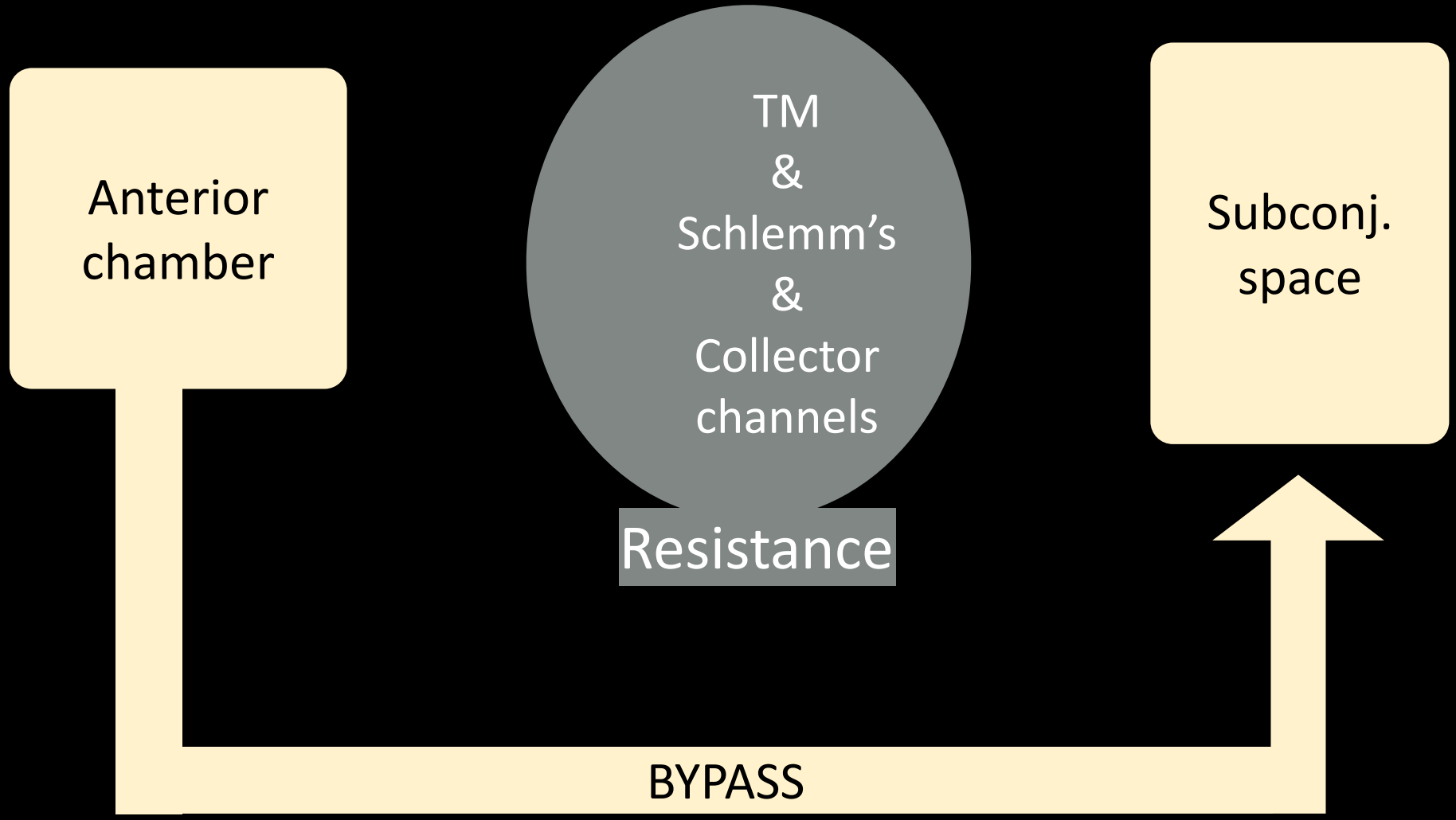
- Phthisis
- Hypotony
- Uveitis
- Pain
- Transient IOP rise
- Vision loss
- Hyphema
- Choroidal detachment
- Choroidal neovascularization
- Malignant glaucoma
- Ant segment ischemia
- Sub retinal fibrosis
- Lens subluxation
- Cataracts
- Sympathetic ophthalmia

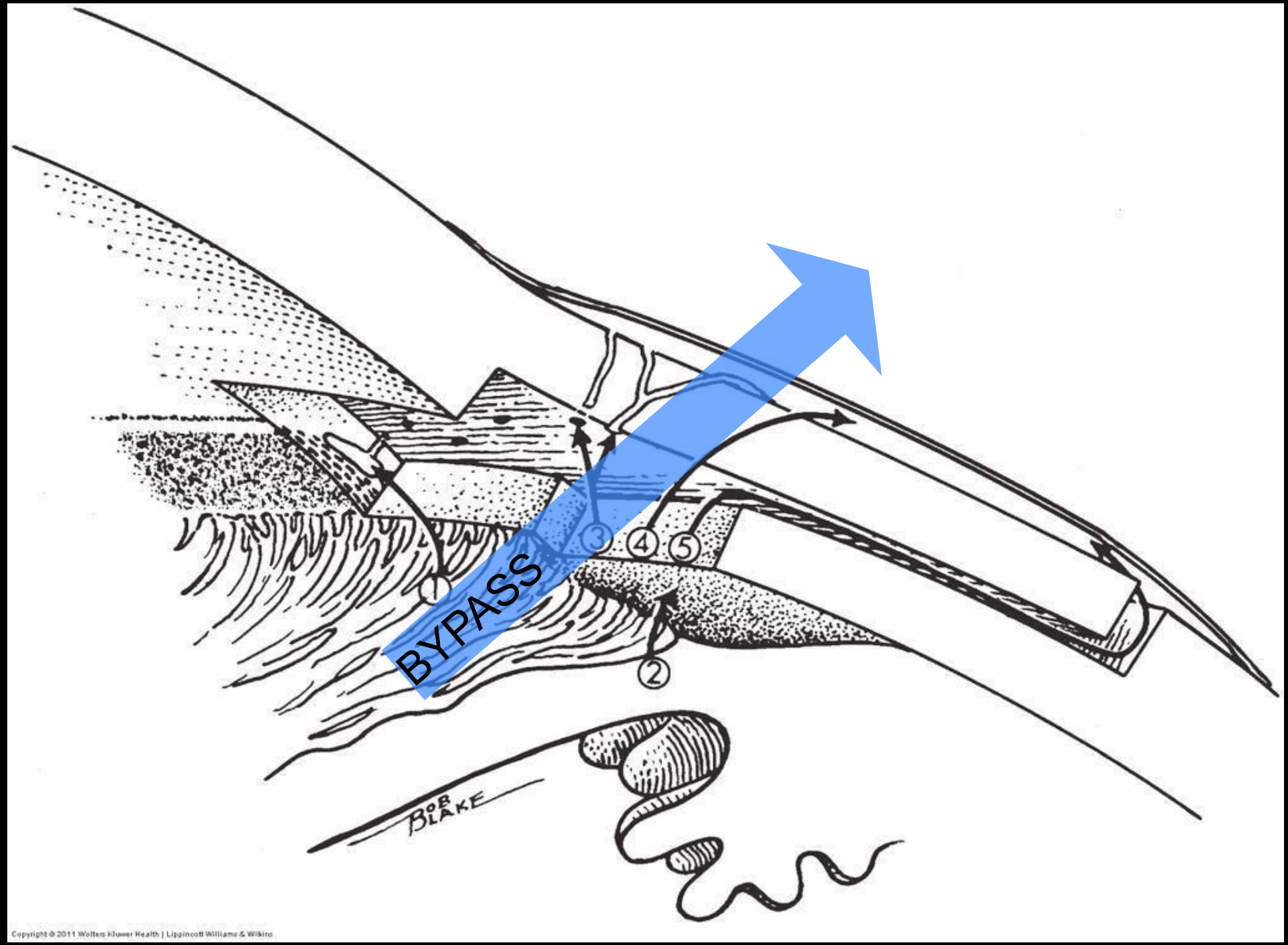
Surgery

Glaucoma Surgeries

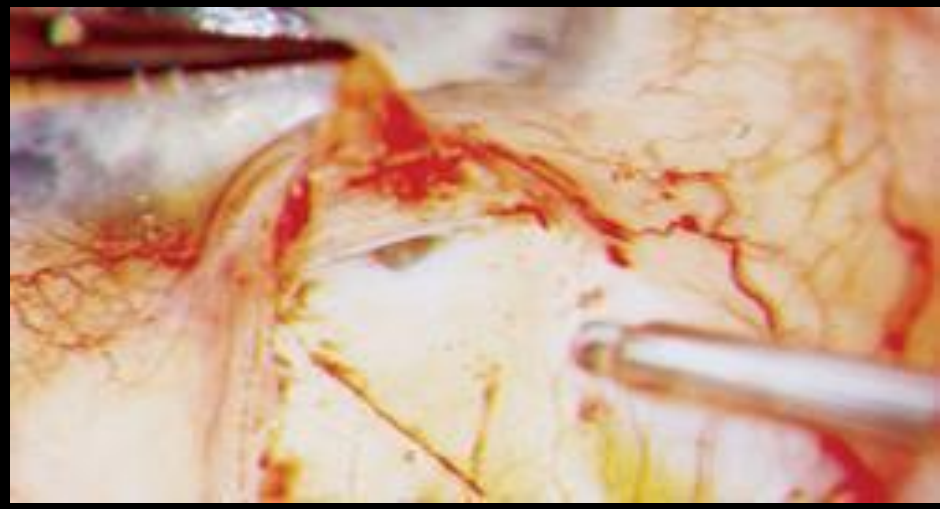
- Trabeculectomy
- Combined procedure= Trab+ Phaco
- Glaucoma valve implant
- Non penetrating filteration surgeries
- Goniotomy
- Trabeculotomy
- Surgical Iridectomy

PRINCIPLE


Anterior chamber
TM & Schlemm's & Collector channels
Resistance
Subconj. space
BYPASS



Trabeculectomy (Indications)

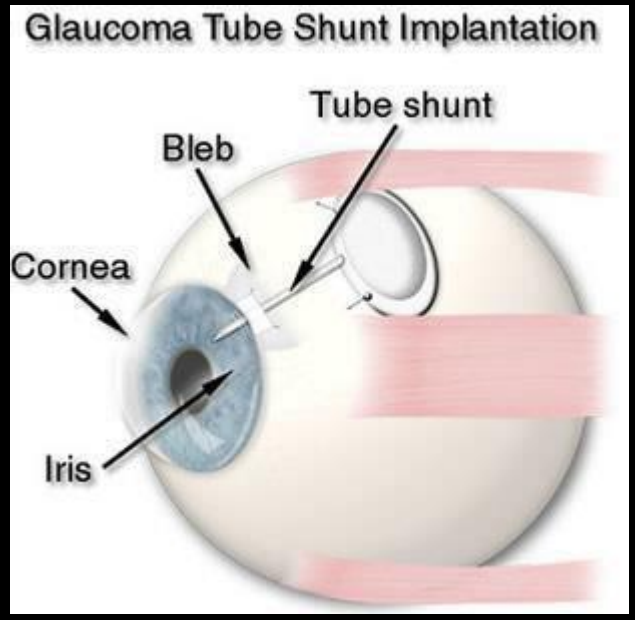


- Uncontrolled high IOP
- IOP above target in spite of maximum tolerated medical therapy (with progression)
- Documented glaucomatous progression with IOP below target
- Diurnal variation of >5 mm of Hg in spite of maximum Rx

  (Mederios FA et al, J Ocul Pharmacol Ther 2002; 18:489-98)
- Poor compliance with medical therapy: relative indication

Glaucoma Drainage Implant

- Previous failed trabeculectomy
- Superior conjunctival scarring due to Cataract sx
- Neovascular glaucoma
- Congenital glaucoma
- Uveitic glaucoma



Glaucoma Drainage Devices

Summary

- Medical Tx
- Lasers Tx
- Surgical Tx

Thank you All

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Complications of Cataract Surgery

Learning objectives

- Discuss the etiology, clinical features, investigation, and management of Endophthalmitis.
- Discuss the etiology, clinical features, investigation, and management of Panophthalmitis.

Introduction

- Frequently asked question
- Needs to be remembered on finger-tips
- No surgery is without complications
- Can be answered in many ways

Usual classification

Complications of anesthesia

Intraoperative

Early postoperative

Late postoperative

Complications of anesthesia

General Anesthesia

- Respiratory complications
  - Laryngoscope & intubation
  - Respiratory obstruction & spasm (isoflurane)
  - Hypoxemia (N_2O)
  - Hypercapnia / Hypocapnia
  - Hypoventilation
  - Aspiration pneumonia
  - Chest infections

Cardiovascular complications

Hypertension
Hypotension
Cardiac arrhythmias
Death (halothane)

Neurological complications

Headache
Delayed recovery
Perioperative neuropathy
Hallucinations & unpleasant dreams (ketamine)

Postoperative nausea & vomiting (propofol, etomidate)

Temperature changes

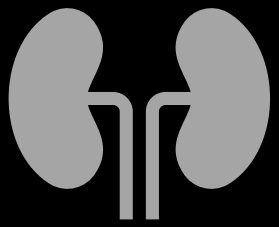
- Hypothermia
- Hyperthermia (atropine, halothane)

Allergic drug reactions

- Itching
- Anaphylaxis
  - Hypotension, arrhythmia, tachycardia
  - Bronchospasm, cough, dyspnea

Complications of positioning

Air embolism
Nerve palsies (Brachial plexus, Radial nerve, Ulnar nerve)
Skin necrosis



Miscellaneous

Renal dysfunction (enflurane)
Muscle pain (succinylcholine)

Regional Anesthesia

Peribulbar / Retrobulbar Anesthesia

- Lids trauma (ecchymosis)
- Ptosis
- s/conj hemorrhage
- Muscles damage
- Globe penetration & its related complications
- Retrobulbar hemorrhage
- Optic nerve damage
- Putscher-type retinopathy *
- Brainstem anesthesia

* Lemagne, J.S., Michiels, X., Van Causenbroeck et al (1990). Putscher-type retinopathy after retrobulbar anesthesia. Ophthalmology, 97,859-61.

Retrobulbar Hemorrhage

Common complication

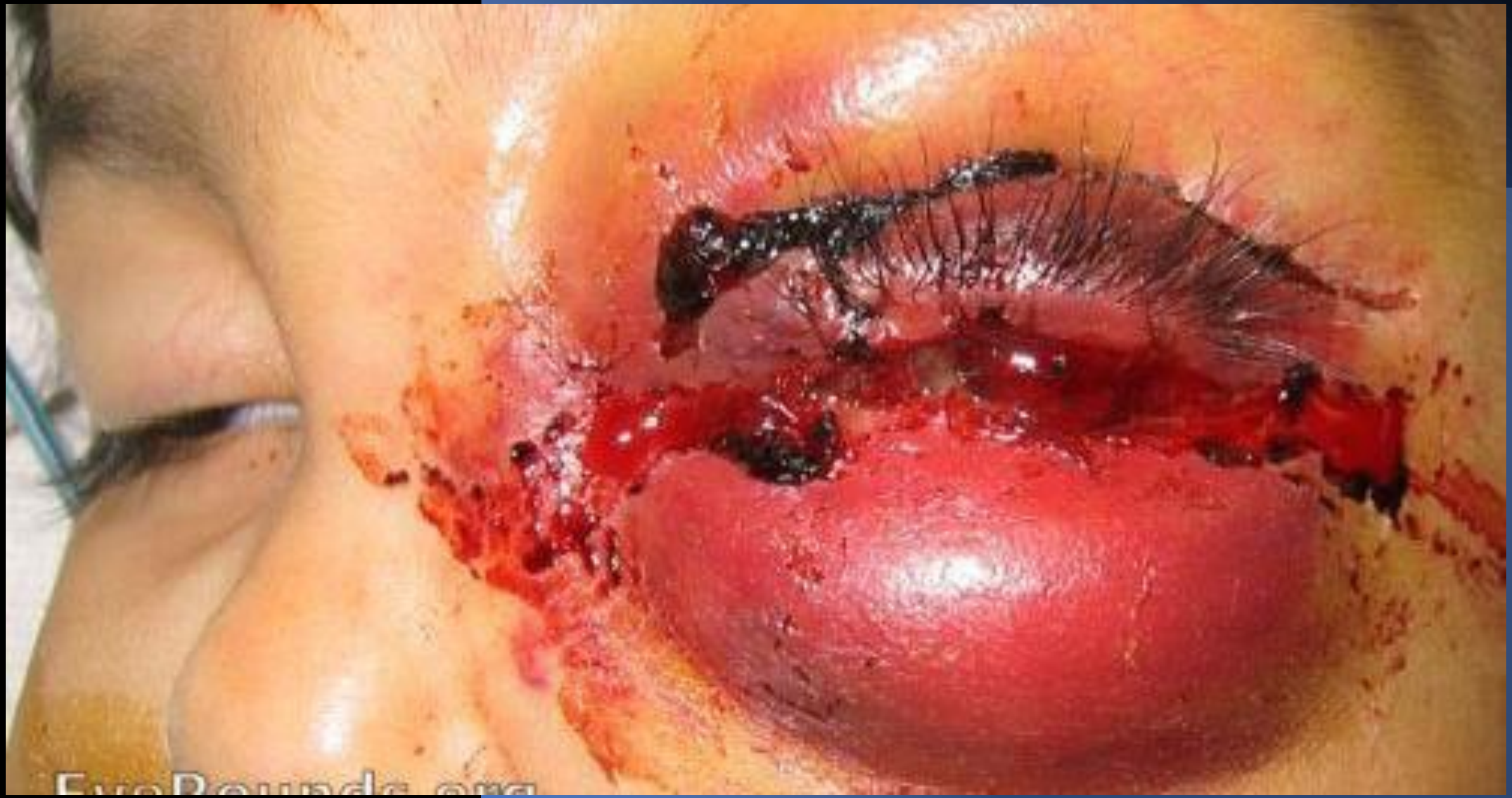
Causes proptosis & raised IOP

Incidence of severe RBH = 0-3%

A reported case of CRAO *

Management

- Continue with surgery if minimal
- Lateral canthotomy for severe cases


EyeRounds.org

Brainstem Anesthesia

- Life-threatening
- Mechanism
  - Entry of agent in ON sheath & then subarachnoid space
- Onset = 2 min, duration = 10-20 min
- Manifestations
  - Confusion, cranial nerve palsies
  - Convulsions, hemiplegia, quadriplegia
  - CVS instability, respiratory arrest

S/Conj. & S/Tenon Anesthesia

- Common
  - Pain on injection (15-33%)
  - Chemosis (6-100%)
  - s/conj hemorrhage (7-100%)
- Rare
  - Globe perforation
  - Retrobulbar hemorrhage
  - Hyphema
  - Muscle trauma
  - Diplopia
  - Spread to CNS
  - Retinal dysfunction
  - Orbital cellulitis
  - Chronic dilated pupils

Indian j ophthalmol 2006;54:77-84

Intraoperative Complications

- Wound related
  - Iris prolapse
- Corneal
  - Descemet's membrane detachment
  - Corneal burns
- Anterior chamber
  - Iridodialysis
  - flattening of anterior chamber
  - Hyphema
  - Intraoperative floppy iris syndrome
- Lens related
  - Dropped nucleus
  - Retained lens mater
  - Posterior loss of lens fragments
- IOL related
  - IOL dislocation
- Posterior segment
  - Posterior capsule rupture
  - Cyclodialysis
  - Suprachoroidal effusion & hemorrhage

Early Postoperative Complications

- Wound related
  - Wound leak
  - Iris prolapse
  - induced astigmatism
- Corneal
  - Corneal edema
  - Striate keratopathy
- Anterior chamber
  - AC reaction
  - Hyphema
  - TASS
  - Vitreous in AC
- IOP related
  - Raised
  - Low
- IOL related
  - Decentered
  - Dislocated
  - Tilted
  - Pupillary capture
  - Capsular block syndrome
- Acute Endophthalmitis

Late Postoperative Complications

- Wound related
  - Astigmatism
- Corneal
  - Bullous keratopathy
  - Corneal decompensation
  - Corneal melting
  - Brown-McLean syndrome
  - Epithelial down growth
- IOP related
  - Glaucoma
- Anterior chamber
  - Chronic uveitis
  - UGH syndrome
  - Iris atrophy / cysts
- IOL related
  - Malposition, glare
  - PCO & Phimosis
- Posterior segment
  - Retinal light toxicity
  - Macular infarction
  - CME
  - R/D
- Chronic Endophthalmitis

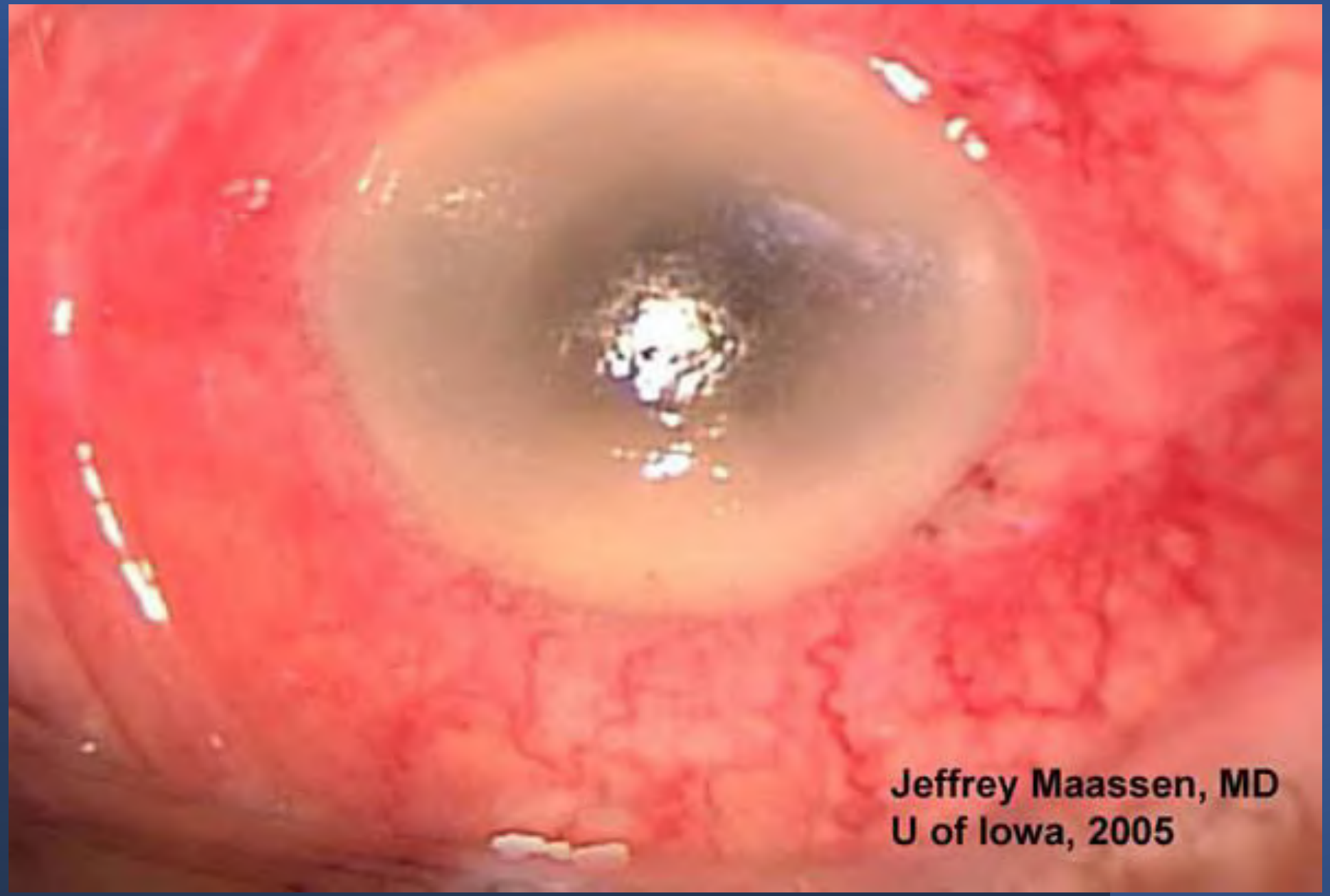
Endophthalmitis

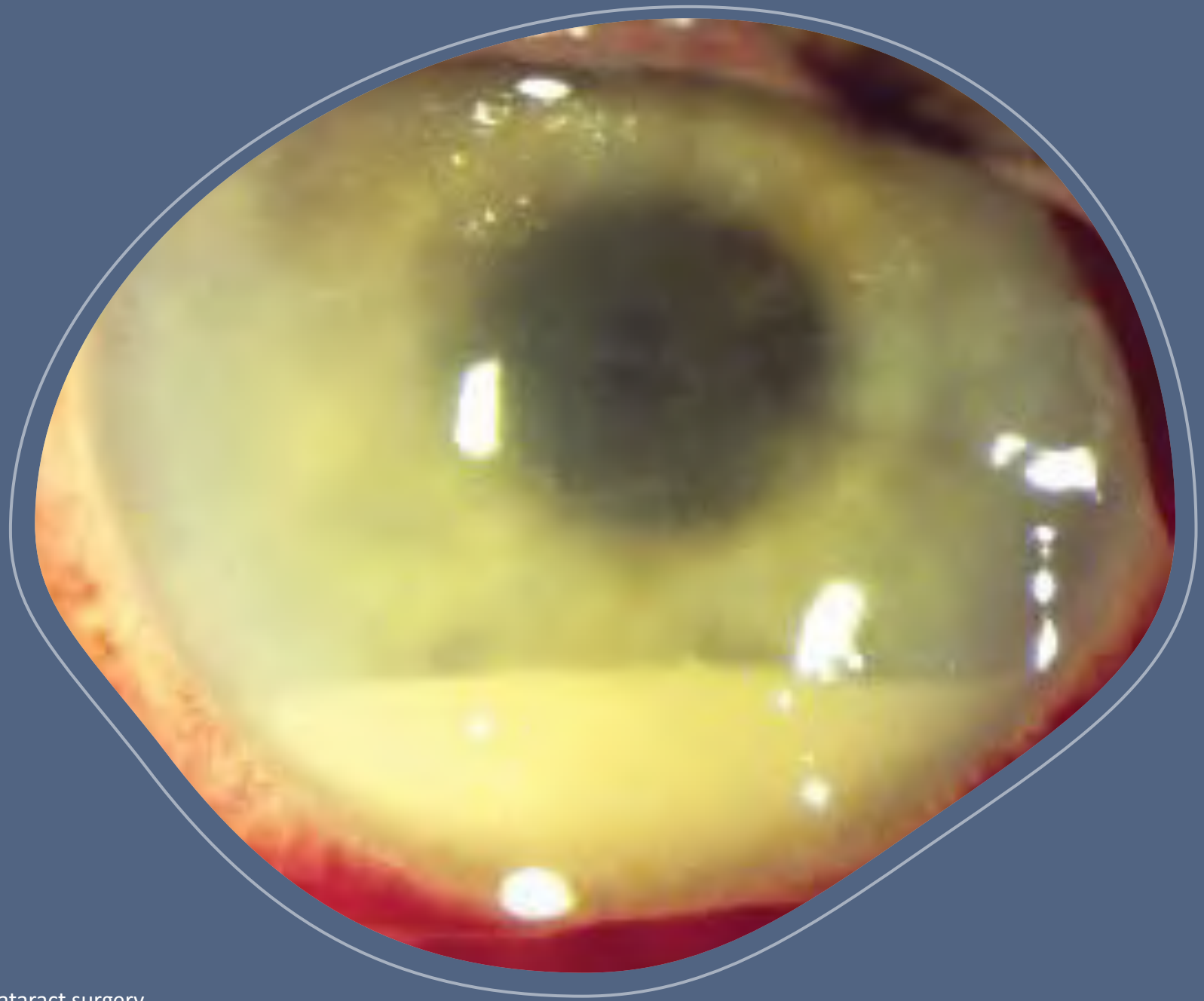
- An inflammatory condition of the intraocular cavities (aqueous or vitreous) usually caused by infection
- Estimated incidence…0.15%
- Risk factors
  - Age > 80 yrs
  - DM, secondary IOL implantation
  - PCR, combined surgery

- Pathogenesis
  - Lids & conjunctival flora…most common source
  - Contaminated instruments & solutions
  - Theater environment, surgeon & other staff
- Organisms
  - Gram +ve…85-90%
    - S. epidermidis…70%
    - S. aureus, streptococcus spp & other gram +ve..15-20%
  - Gram -ve…5-6%
    - Pseudomonas, proteus, P.acnes

Acute Endophthalmitis

- Presents…2-5 days post surgery up to 6 weeks
- Fulminant course, Gram +ve organisms mainly
- Features
  - Severe pain & visual loss
  - Chemosis, conjunctival injection & discharge
  - RAPD
  - Corneal haze
  - Fibrinous exudates, Hypopyon
  - Vitritis, poor fundus view


Jeffrey Maassen, MD
U of Iowa, 2005

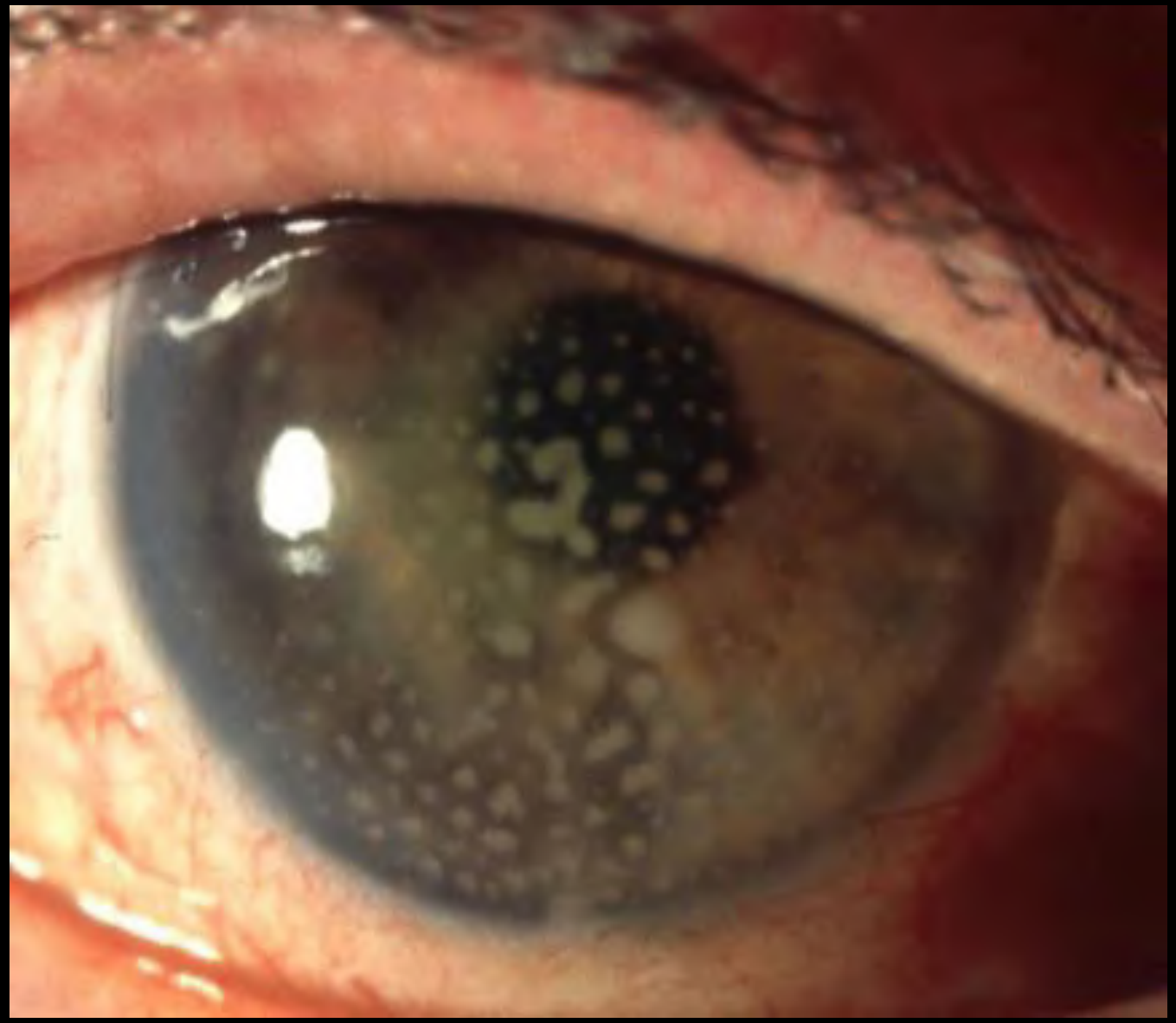


Management

- Vitreous ± aqueous samples
- Antibiotics
  - I/vit, periocular, topical, oral
- Steroids
  - Oral…when fungal infection is excluded
  - Topical…for ant uveitis
  - Periocular…if systemic steroids contraindicated
- PPV

Chronic Endophthalmitis

- Presents…weeks to months to years (9 months)
- Low virulence organisms
  - P. acnes…mainly
  - S. epidermidis, corynebacterium, Candida
- Features
  - Mild or no pain
  - Visual loss
  - Low grade ant uveitis, mutton fat KPs
  - Vitritis common but not Hypopyon



Management

- Vitreous ± aqueous samples
- Antibiotics
  - I/vit
- Topical steroids
- Removal of capsular bag, IOL, residual cortex
- PPV

Prevention of Endophthalmitis

- Preoperative
  - Treatment of pre-existing infections
    - Blepharitis, conjunctivitis, CDC, fellow eye
  - Topical quinolone 3 days preoperative…controversial
- Perioperative
  - Proper eye, adnexa & nearby organs swab
  - Povidone-iodine 5% drops in the conjunctival sac
  - Proper draping
  - Aseptic technique & instruments
  - Intracameral cefuroxime at the end, water-tight closure

Panophthalmitis

- Intense purulent inflammation of the whole eyeball
- Symptoms
  - Severe ocular pain n headache
  - Complete loss of vision
  - Profuse watering
  - Purulent discharge
  - Marked redness and swelling

Signs

- Proptosis And Painful Movement Of Eyeball
- Lid Edema
- Chemosis
- Corneal Edema N Clouding
- Anterior Chamber Full Of Pus
- IOP
- Globe Perforation

Complications

- Orbital cellulitis
- Cavernous sinus thrombosis
- Meningitis

Treatment

- Anti-inflammatory and analgesic
- Broad spectrum antibiotic
- Evisceration

TAKE HOME MESSAGE

Complication-free surgery is impossible all the time…**BUT**

Surgeons must be aware of complications & their proper management

Proper anesthesia techniques, sterilization & patient preparation…mandatory

Learning objectives

- Discuss the etiology, clinical features, investigation, and management of Endophthalmitis.
- Discuss the etiology, clinical features, investigation, and management of Panophthalmitis.

Thank you,
Any questions?

MCQ

3. Which of following preoperative measures has proven most effective in reducing risk of Endophthalmitis

a. Administering oral amoxicillin 3 days before surgery
b. Topical antibiotics for 2 weeks following surgery
c. Decreasing duration of surgery
d. Administering topical 5% Povidone-iodine solution at time of surgery
e. Injecting Vancomycin into the I/A solution

Ans. d

COMPLICATIONS OF CATARACT SURGERY

Asst. Professor
DR.AFZAL QADIR

COMPLICATIONS OF CATARACT SURGERY

1. Operative complications

- Vitreous loss
- Posterior loss of lens fragments
- Suprachoroidal (expulsive) haemorrhage

2. Early postoperative complications

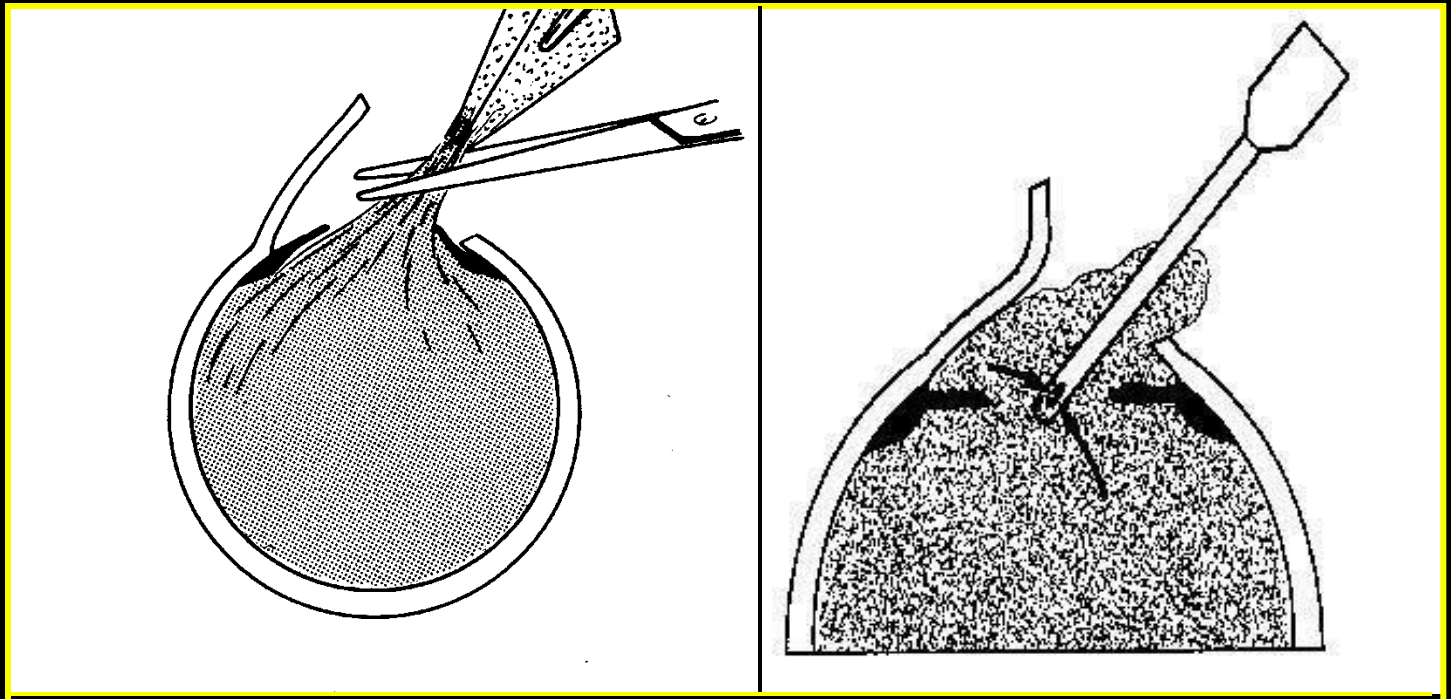
- Iris prolapse
- Striate keratopathy
- Acute bacterial endophthalmitis

3. Late postoperative complications

- Capsular opacification
- Implant displacement
- Corneal decompensation
- Retinal detachment
- Chronic bacterial endophthalmitis

Operative complications of vitreous loss

Management

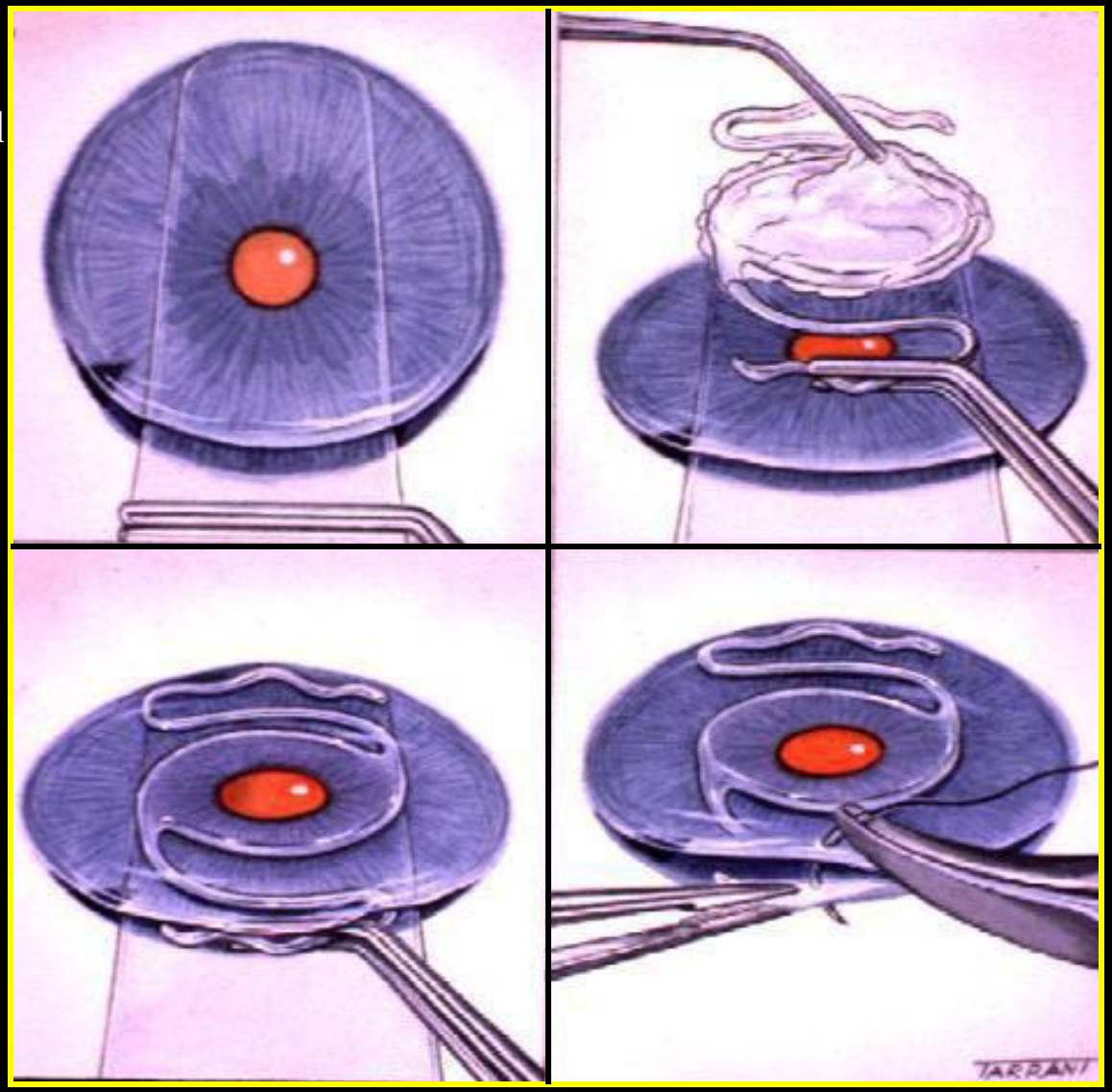


Sponge or automated anterior vitrectomy
Insertion of PC-IOL if adequate casular support present

Insertion of AC-IOL

If adequate capsular support absent

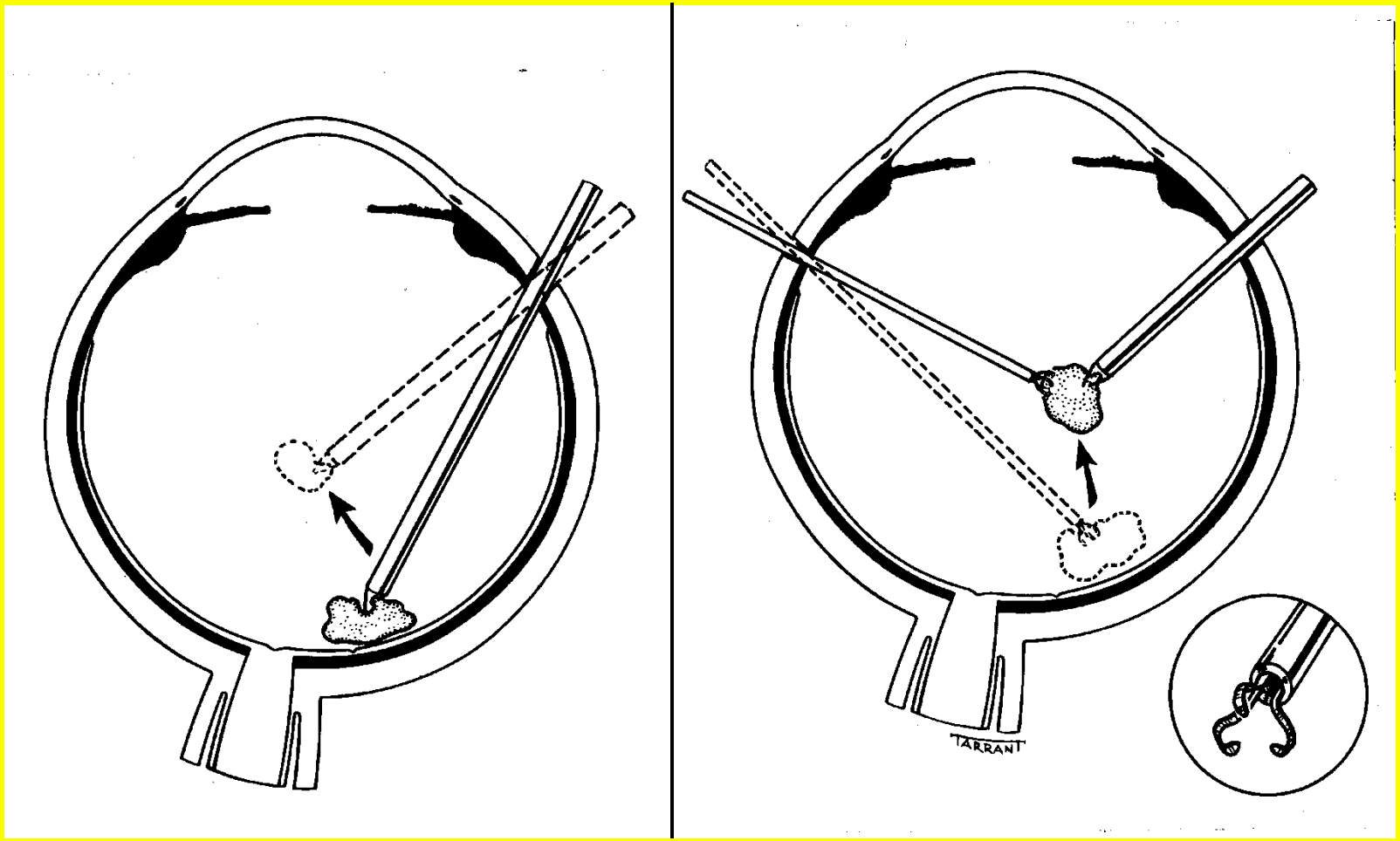
1. Constriction of pupil
2. Peripheral iridectomy
3. Glide insertion



4. Coating of IOL with viscoelastic substance
5. Insertion of IOL
6. Suturing of incision

Management of posterior loss of lens fragments

Fragments consisting of 25% or more of lens should be removed

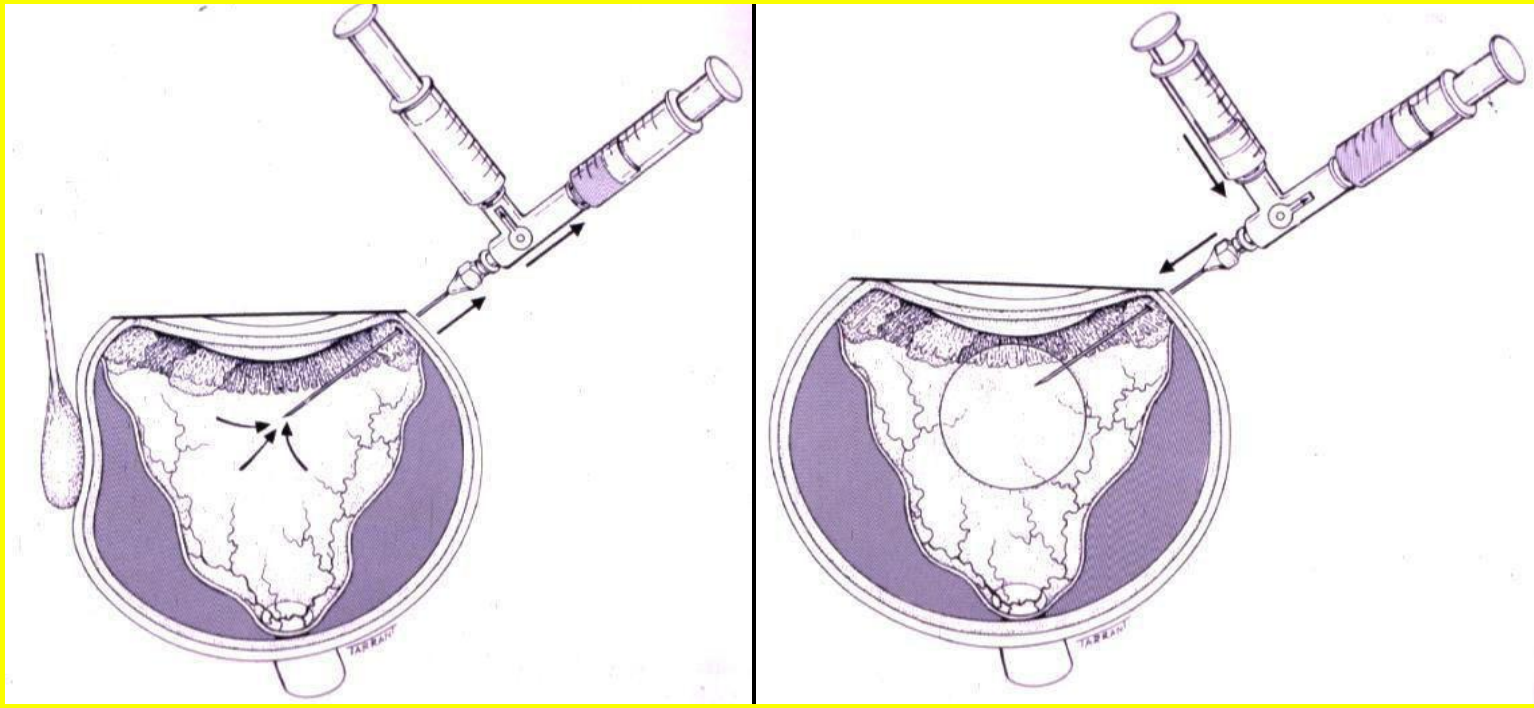


Pars plana vitrectomy and removal of fragment

Management of suprachoroidal (expulsive) haemorrhage

Close incision and administer hyperosmotic agent

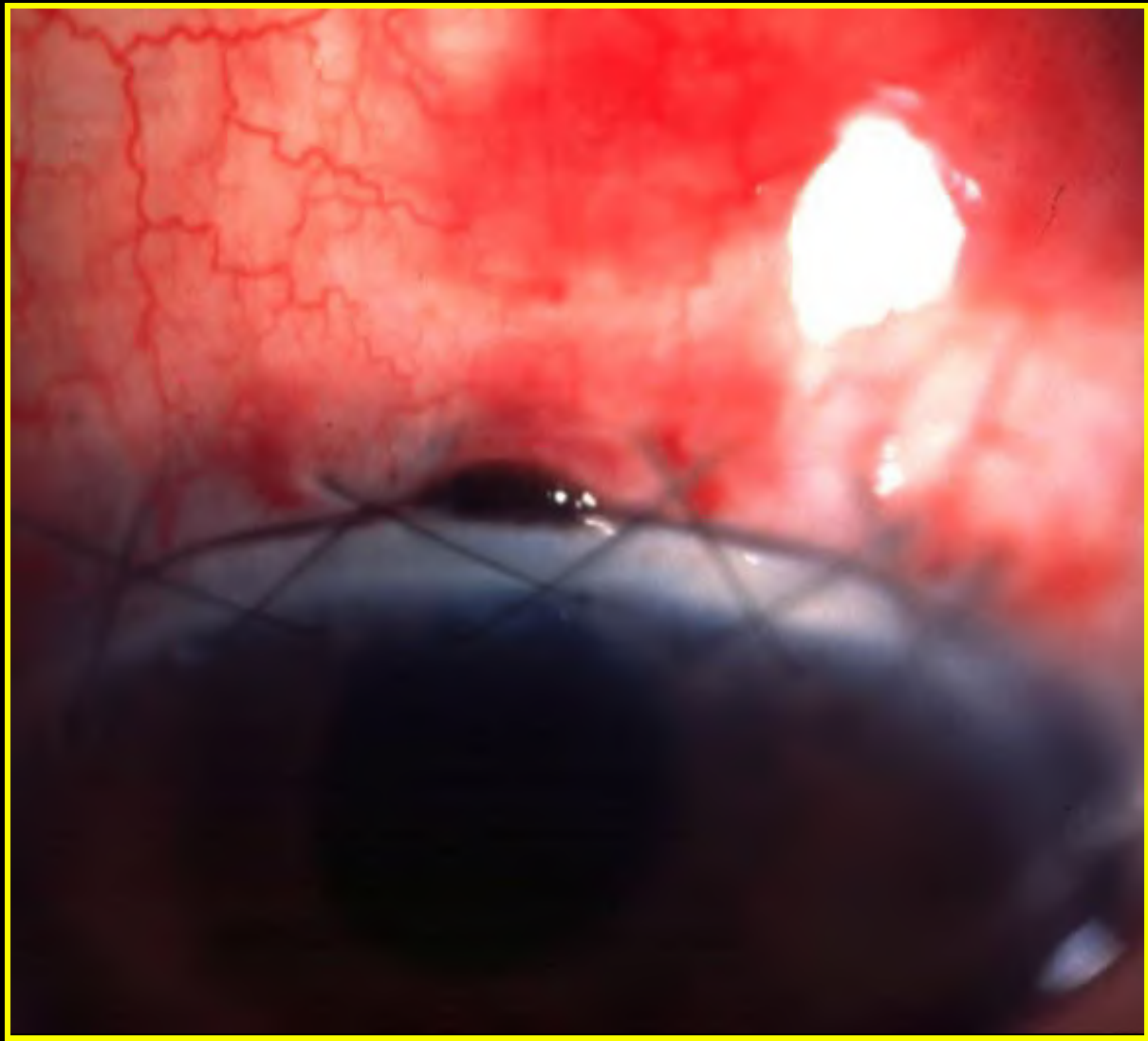
Subsequent treatment after 7-14 days



- Drain blood
- Pars plana vitrectomy
- Air-fluid exchange

Early postoperative complications

Iris prolapse



Cause

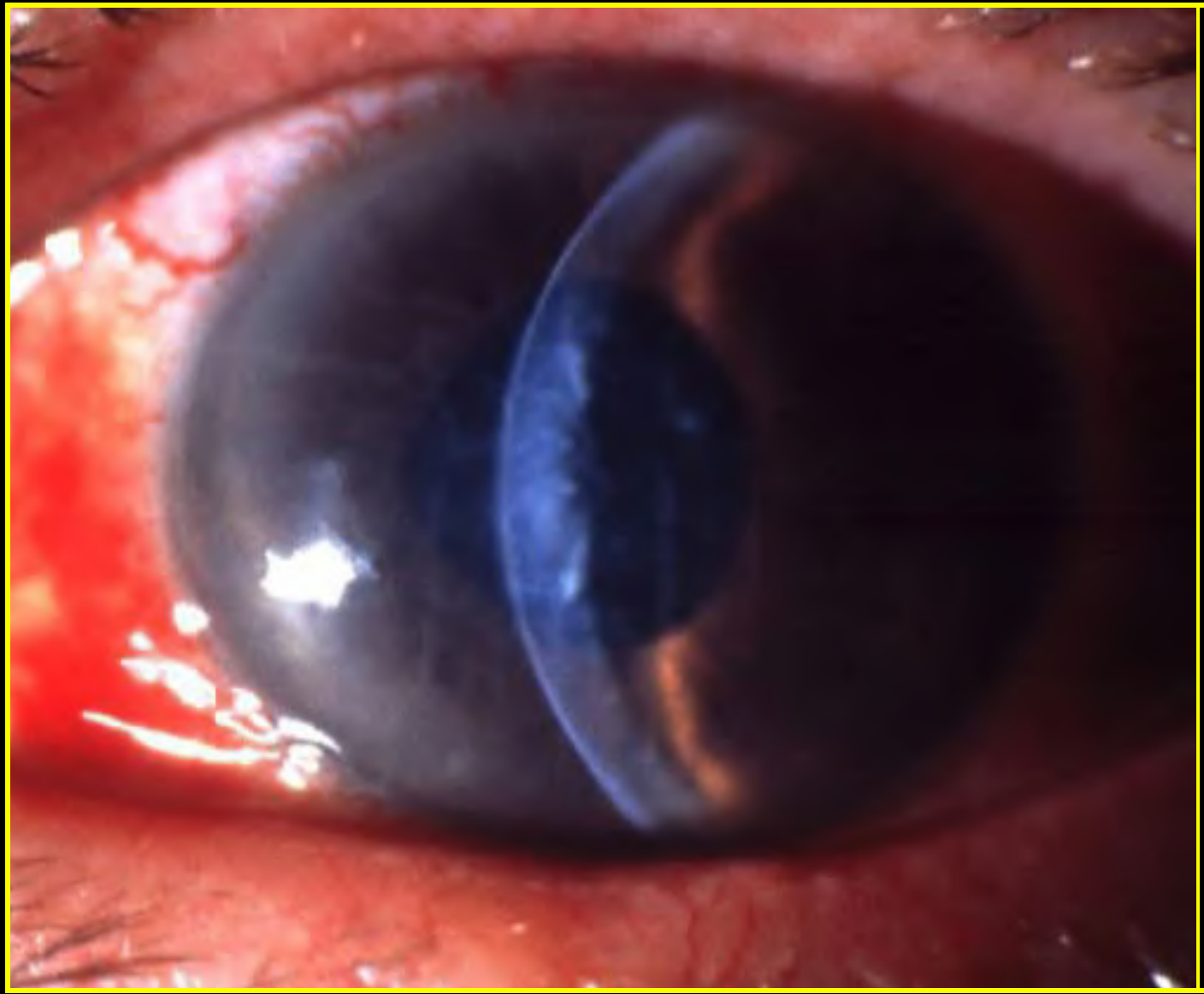
- Usually inadequate suturing of incision
- Most frequently follows inappropriate management of vitreous loss

Treatment

- Excise prolapsed iris tissue
- Resuture incision

Striate keratopathy

Corneal oedema and folds in Descemet membrane



Cause

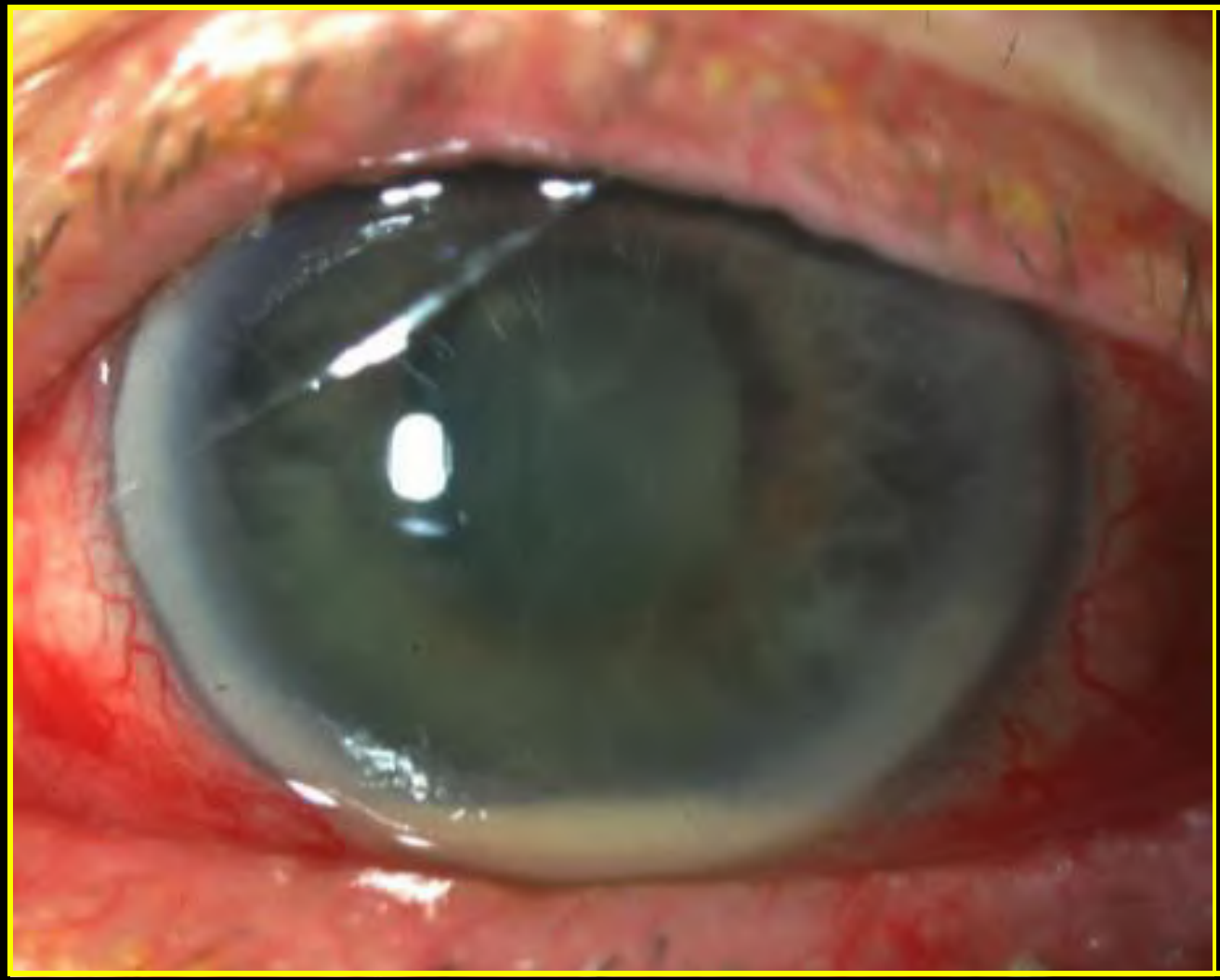
- Damage to endothelium during surgery

Treatment

- Most cases resolve within a few days
- Occasionally persistent cases may require penetrating keratoplasty

Acute bacterial endophthalmitis

Incidence - about 1:1,000



Common causative organisms

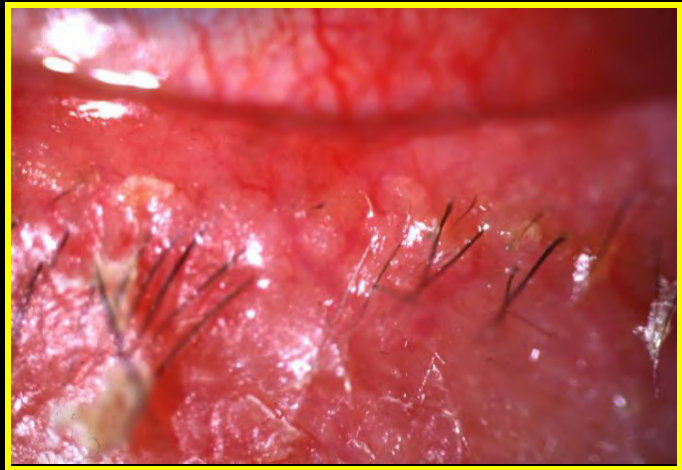
- *Staph. epidermidis*
- *Staph. aureus*
- *Pseudomonas* sp.

Source of infection

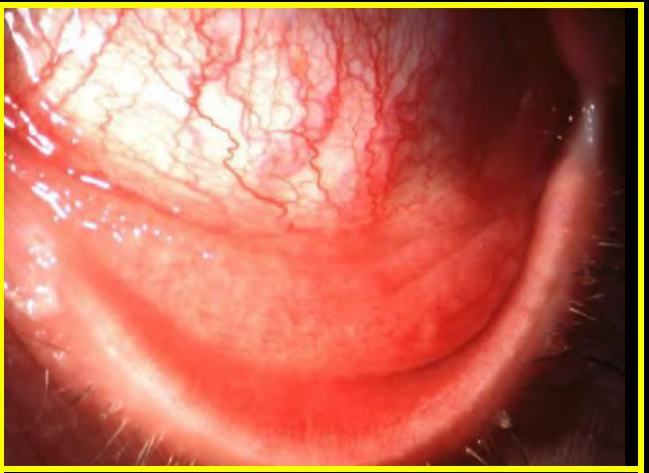
- Patient's own external bacterial flora is most frequent culprit
- Contaminated solutions and instruments
- Environmental flora including that of surgeon and operating room personnel

Preoperative prophylaxis

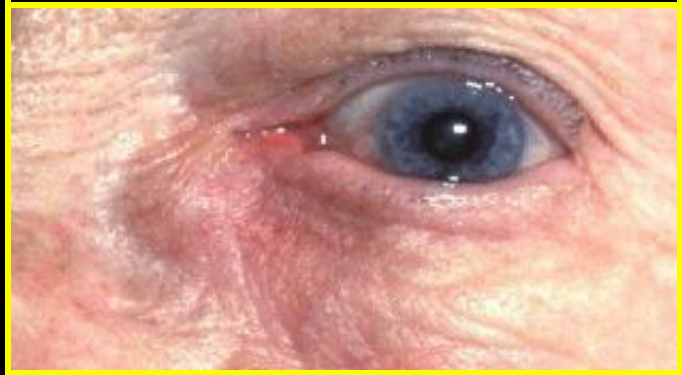
Treatment of pre-existing infections



Staphylococcal blepharitis



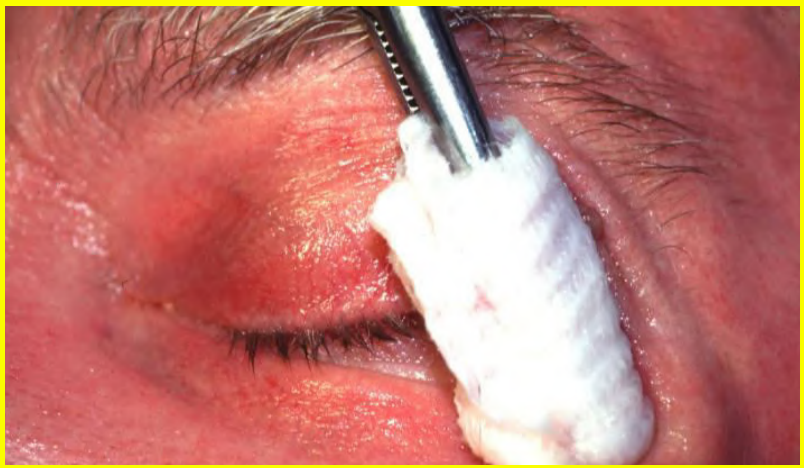
Chronic conjunctivitis

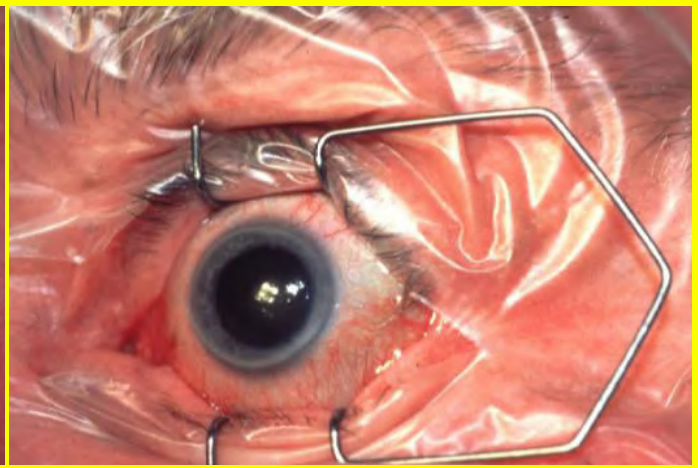


Chronic dacryocystitis

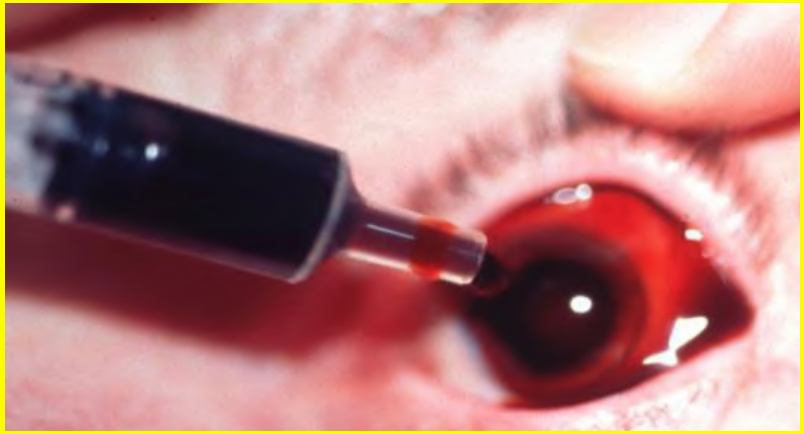
Infected socket

Peroperative prophylaxis

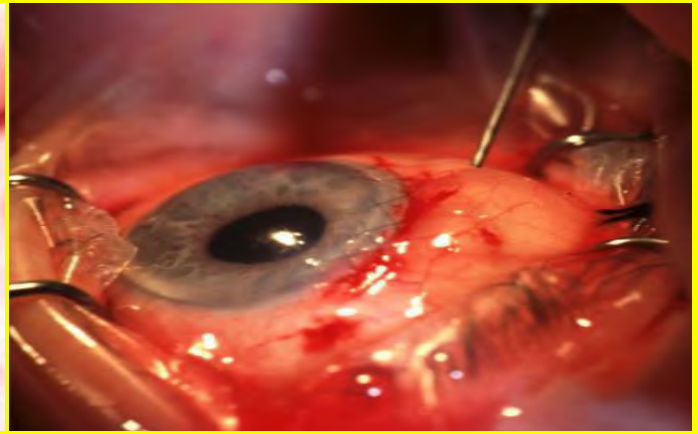




Meticulous prepping and draping



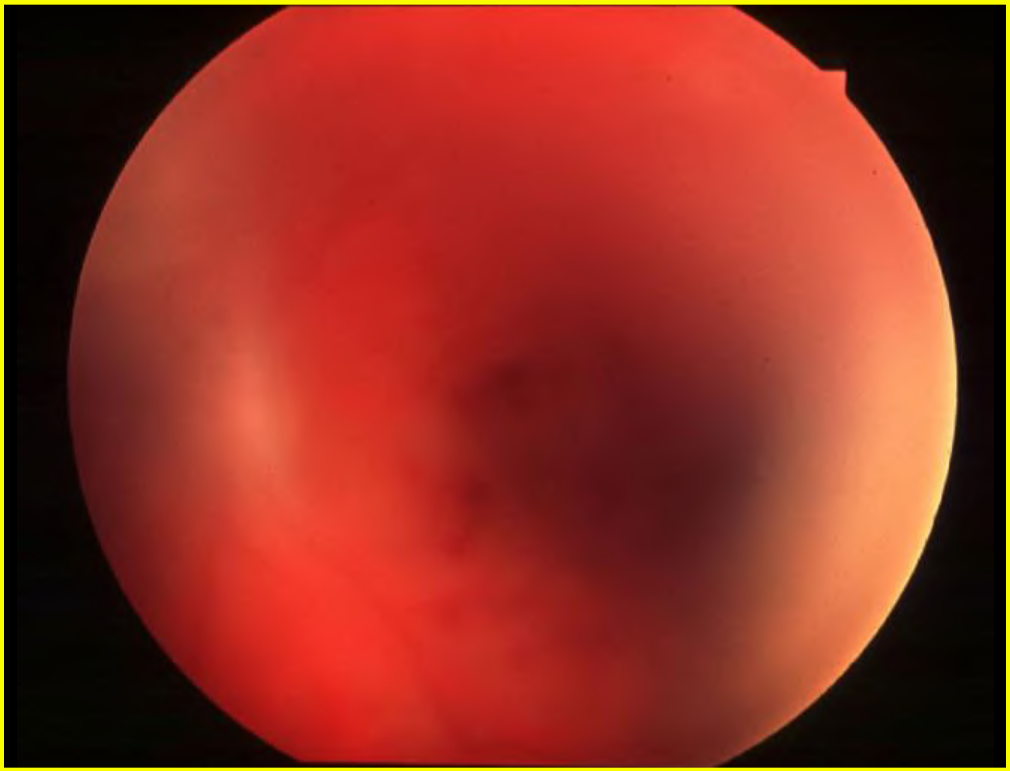
Instillation of povidone-iodine



Postoperative injection of antibiotics

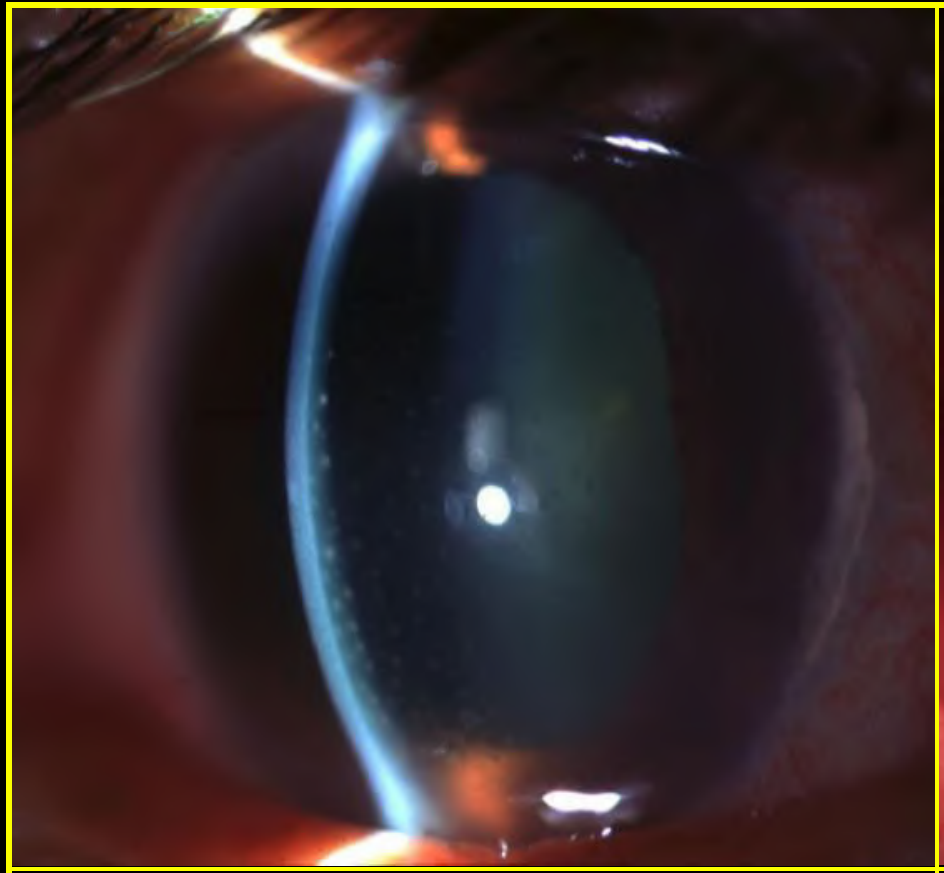
Signs of severe endophthalmitis

- Pain and marked visual loss
- Corneal haze, fibrinous exudate and hypopyon

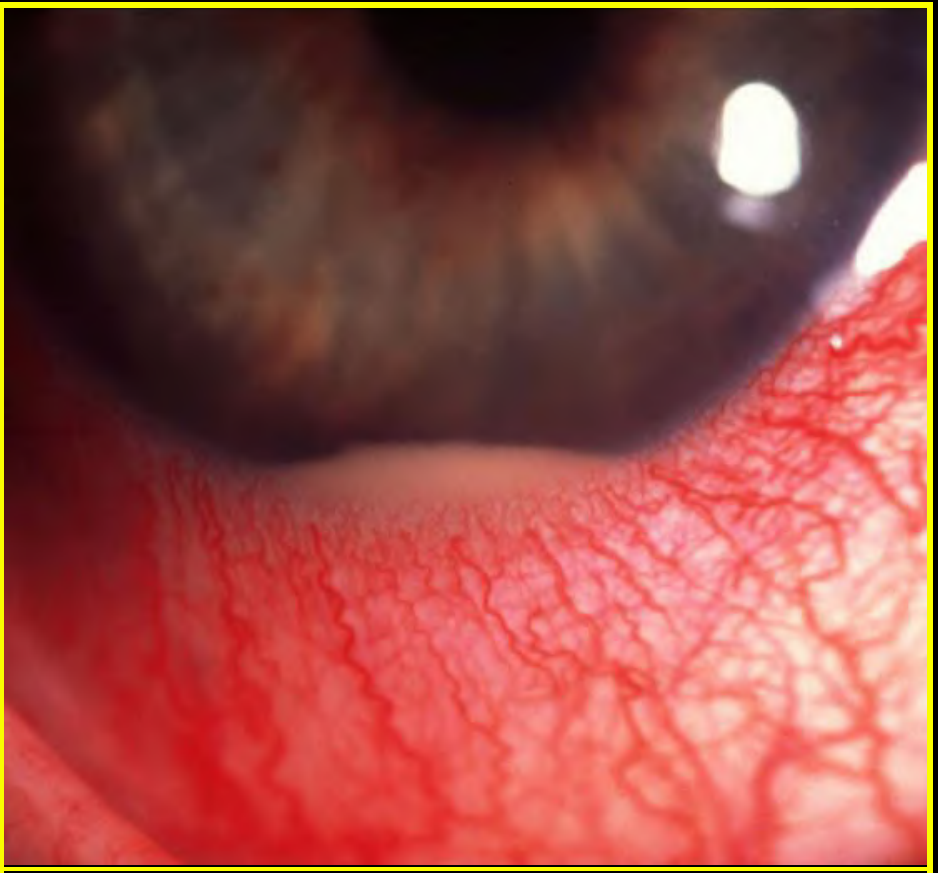


- Absent or poor red reflex
- Inability to visualize fundus with indirect ophthalmoscope

Signs of mild endophthalmitis

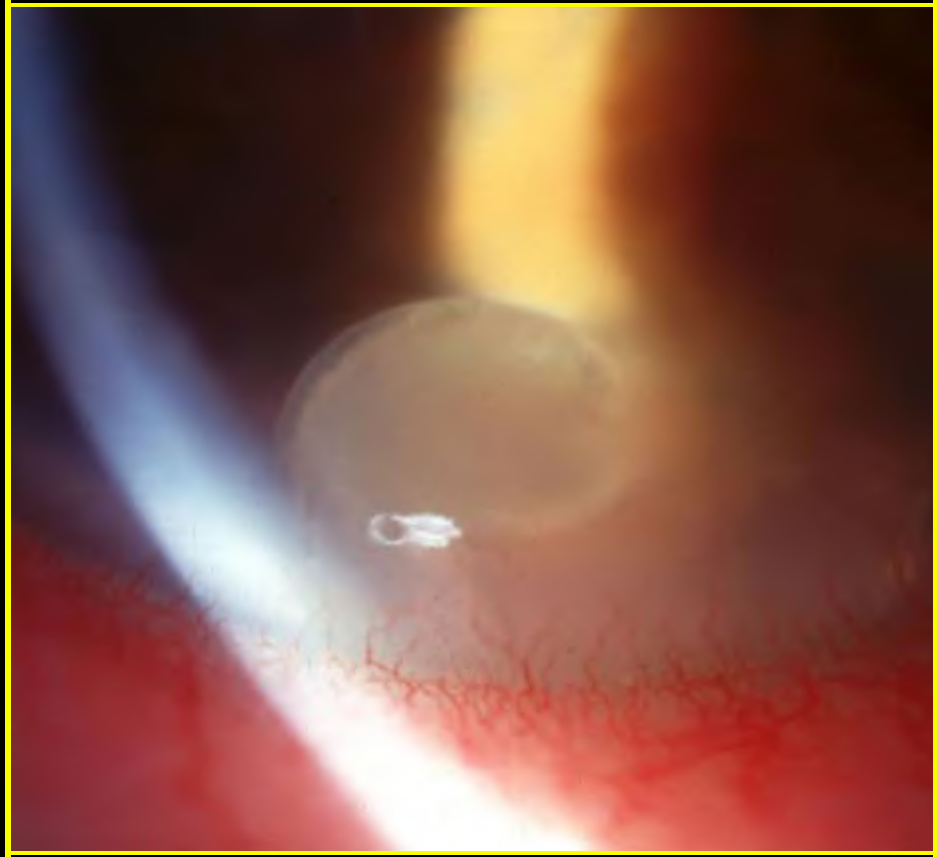
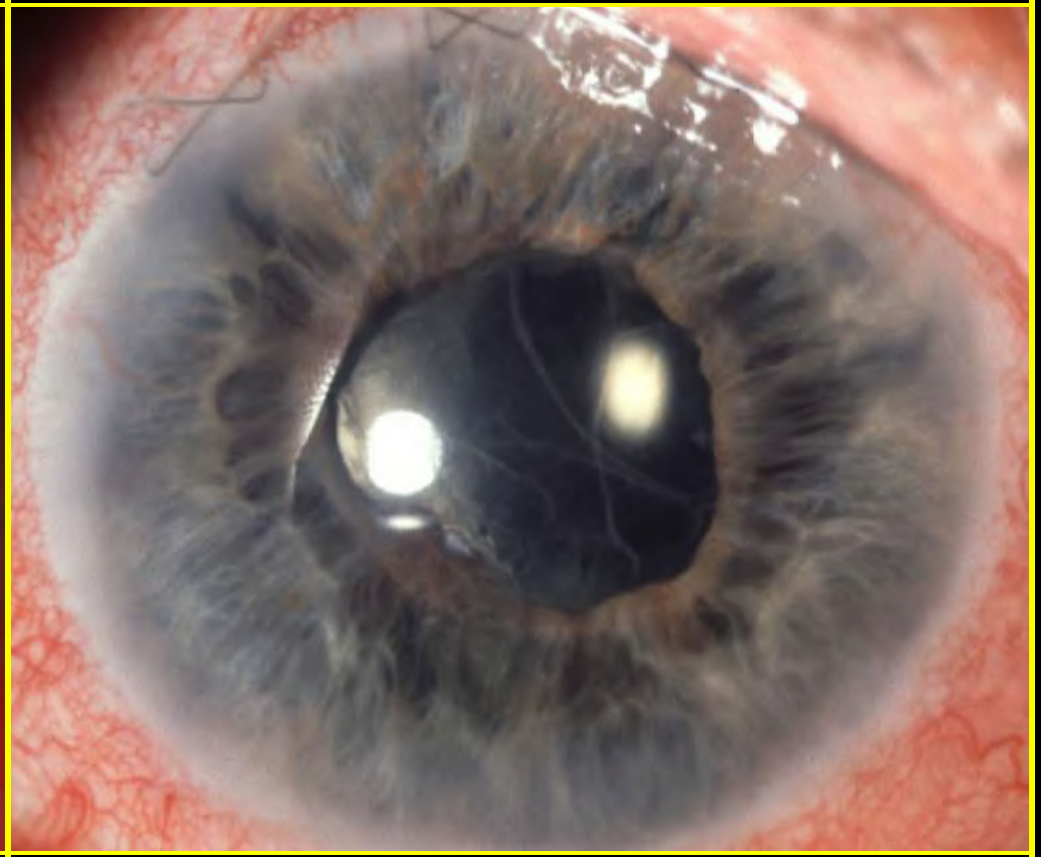


- Mild pain and visual loss
- Anterior chamber cells



- Small hypopyon
- Fundus visible with indirect ophthalmoscope

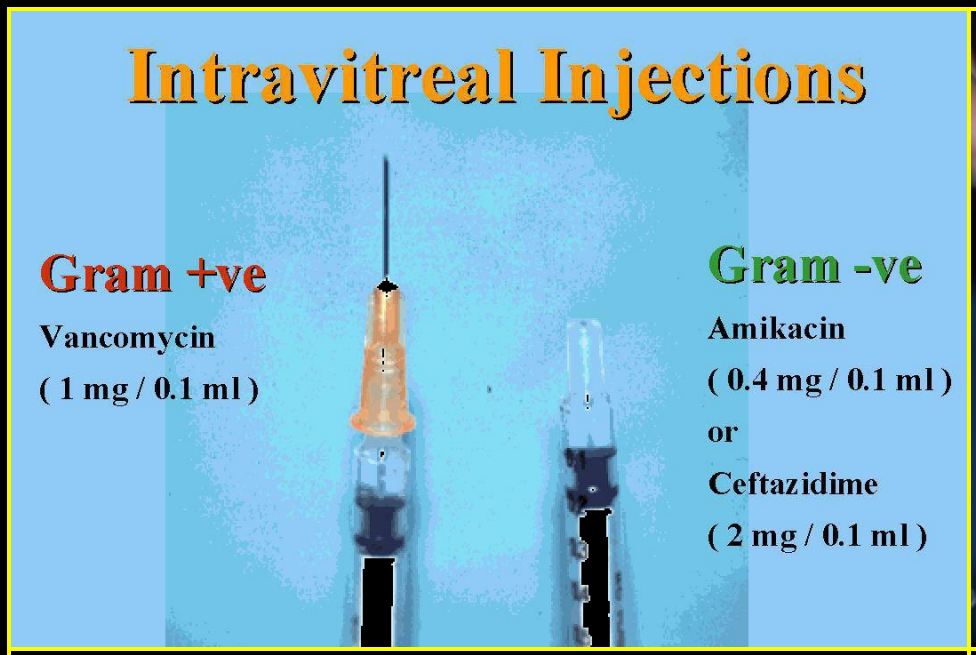
Differential diagnosis of endophthalmitis

Uveitis associated with retained lens material	Sterile fibrinous reaction
	
• No pain or hypopyon	• No pain and few if any anterior cells • Posterior synechiae may develop

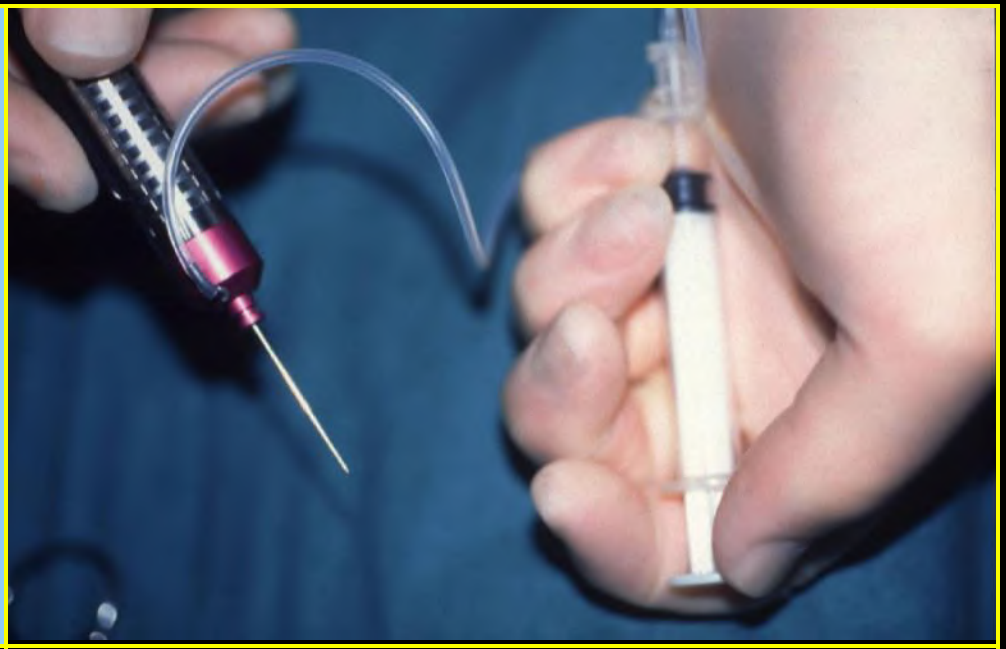
Management of Acute Endophthalmitis

1. Preparation of intravitreal injections
2. Identification of causative organisms
   - Aqueous samples
   - Vitreous samples
3. Intravitreal injections of antibiotics
4. Vitrectomy - only if VA is PL
5. Subsequent treatment

Preparation for sampling and injections

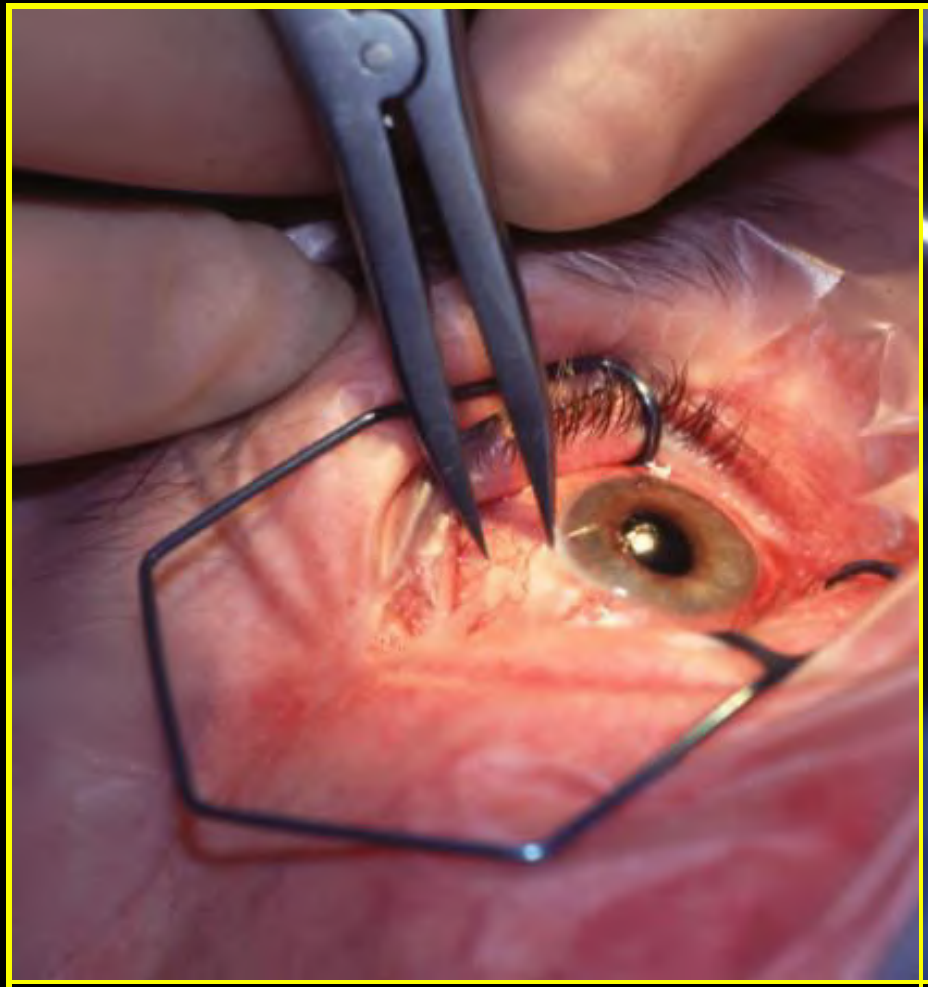


Antibiotics

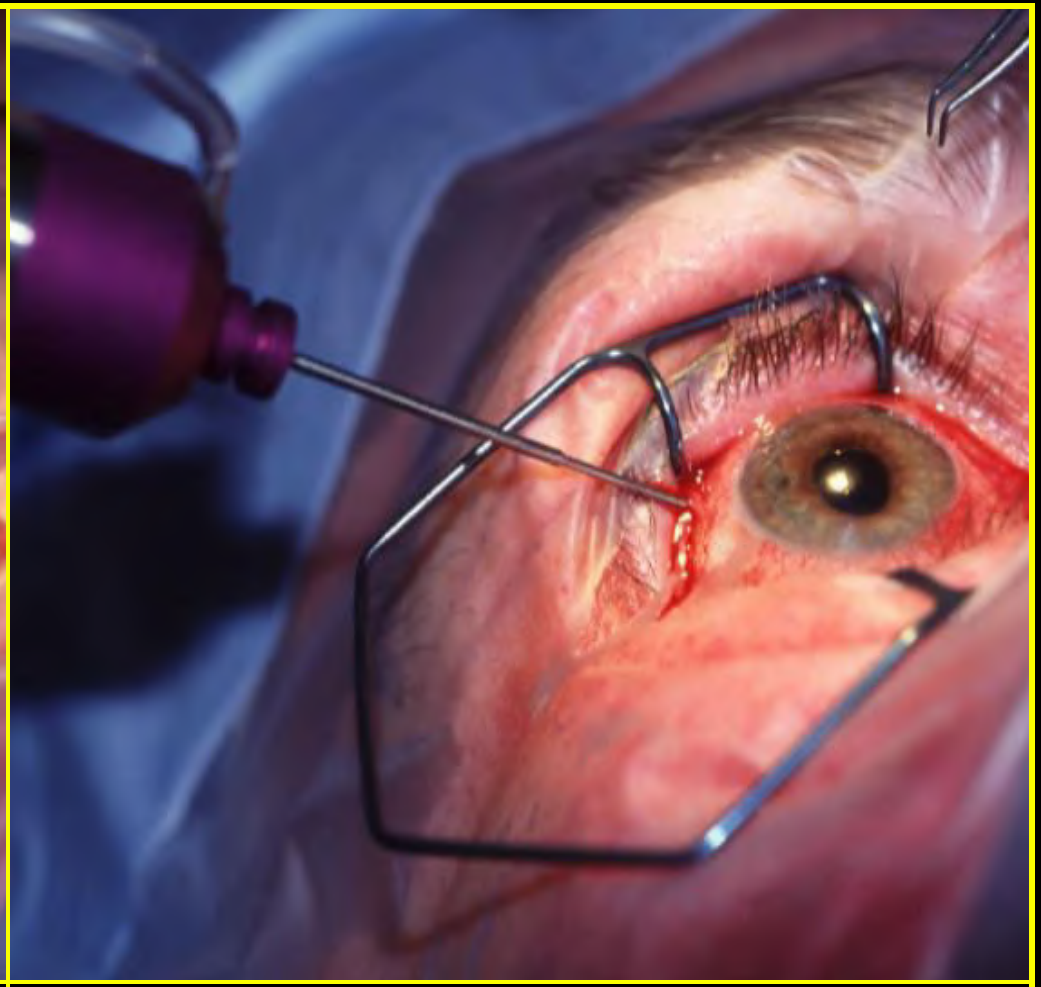


Mini vitrector

Sampling and injections (1)

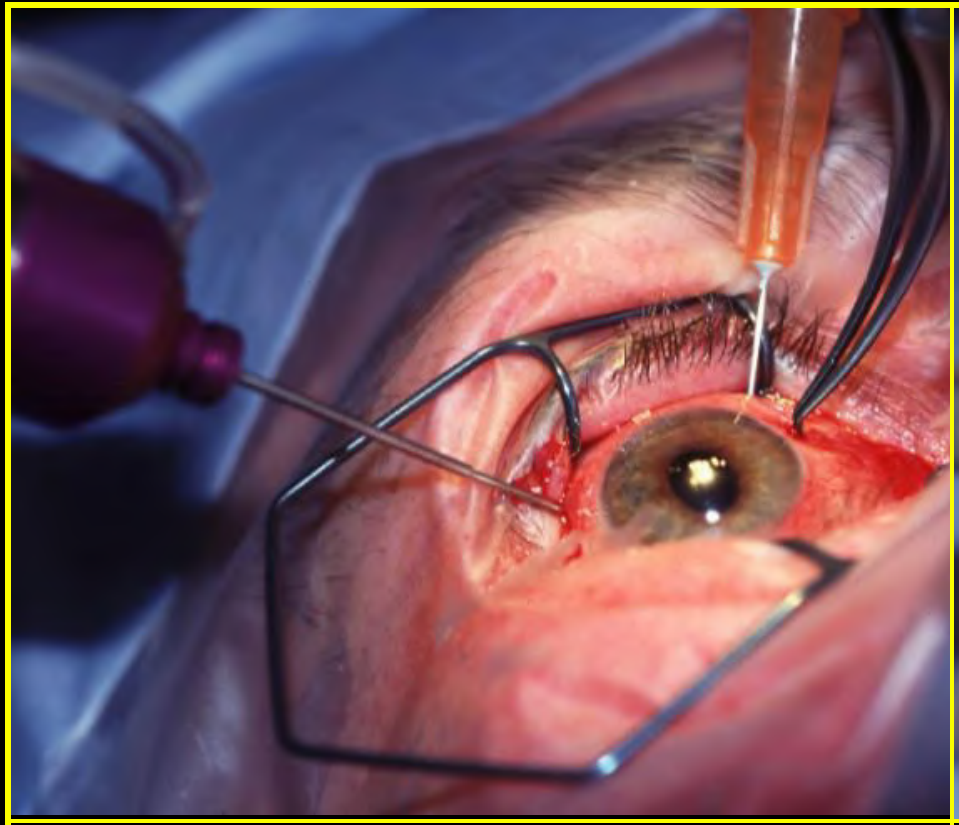


Make partial-thickness sclerotomy
3 mm behind limbus

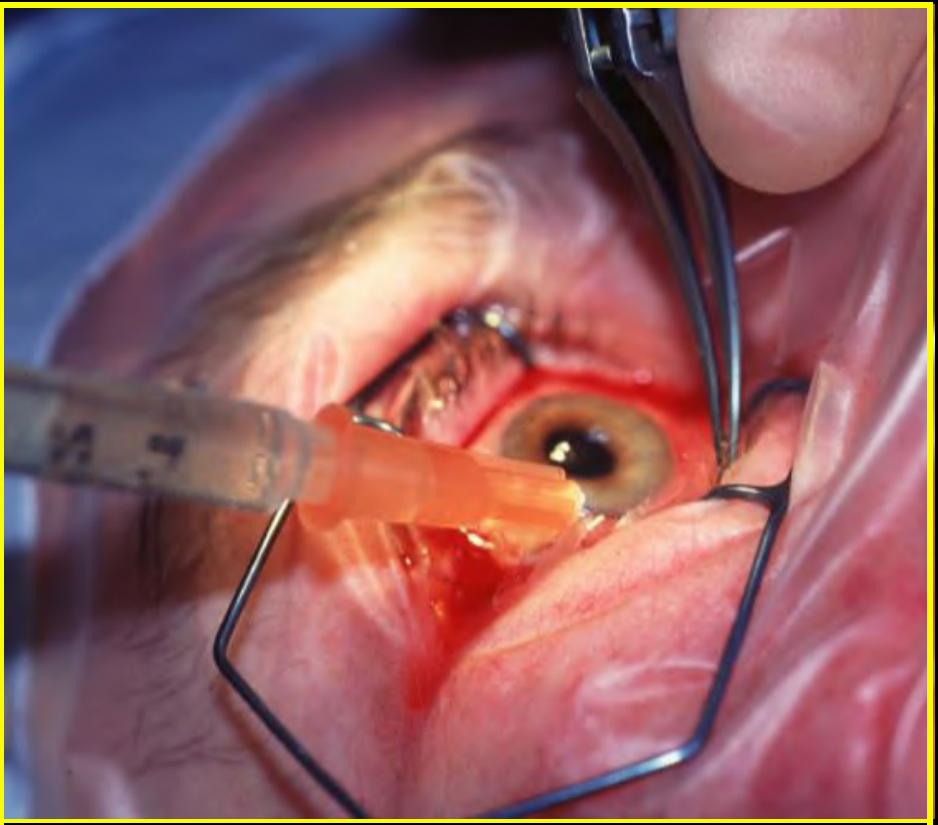


Insert mini vitrector

Sampling and injections (2)



- Insert needle attached to syringe containing antibiotics
- Aspirate 0.3 ml with vitrector
- Give first injection of antibiotics
- Disconnect syringe from needle
- Give second injection



- Remove vitrector and needle
- Inject subconjunctival antibiotics

Subsequent Treatment

1. Periocular injections

- Vancomycin 25 mg with ceftazidime 100 mg or gentamicin 20 mg with cefuroxime 125 mg
- Betamethasone 4 mg (1 ml)

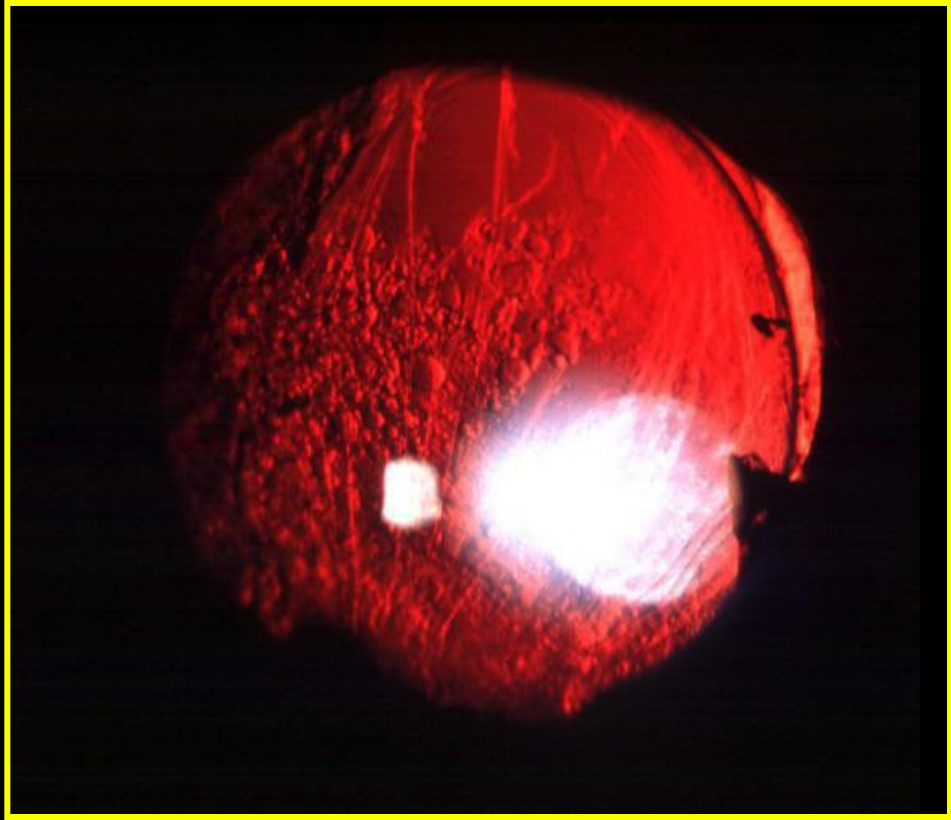
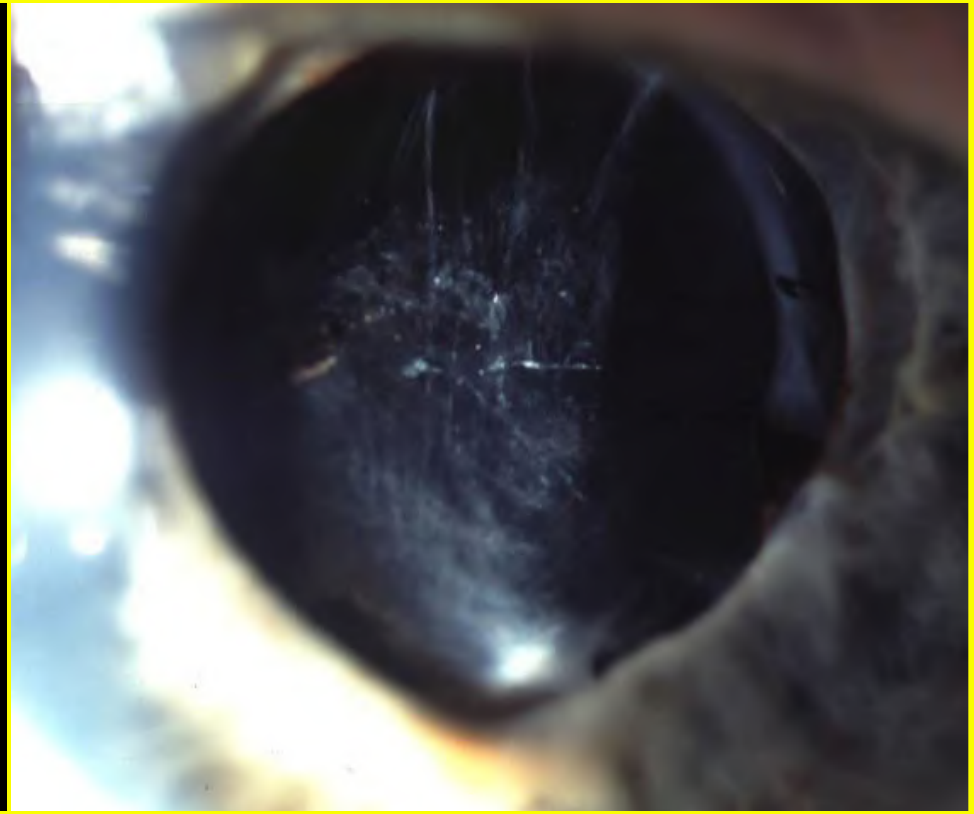
2. Topical therapy

- Fortified gentamicin 15 mg/ml and vancomycin 50 mg/ml drops
- Dexamethasone 0.1%

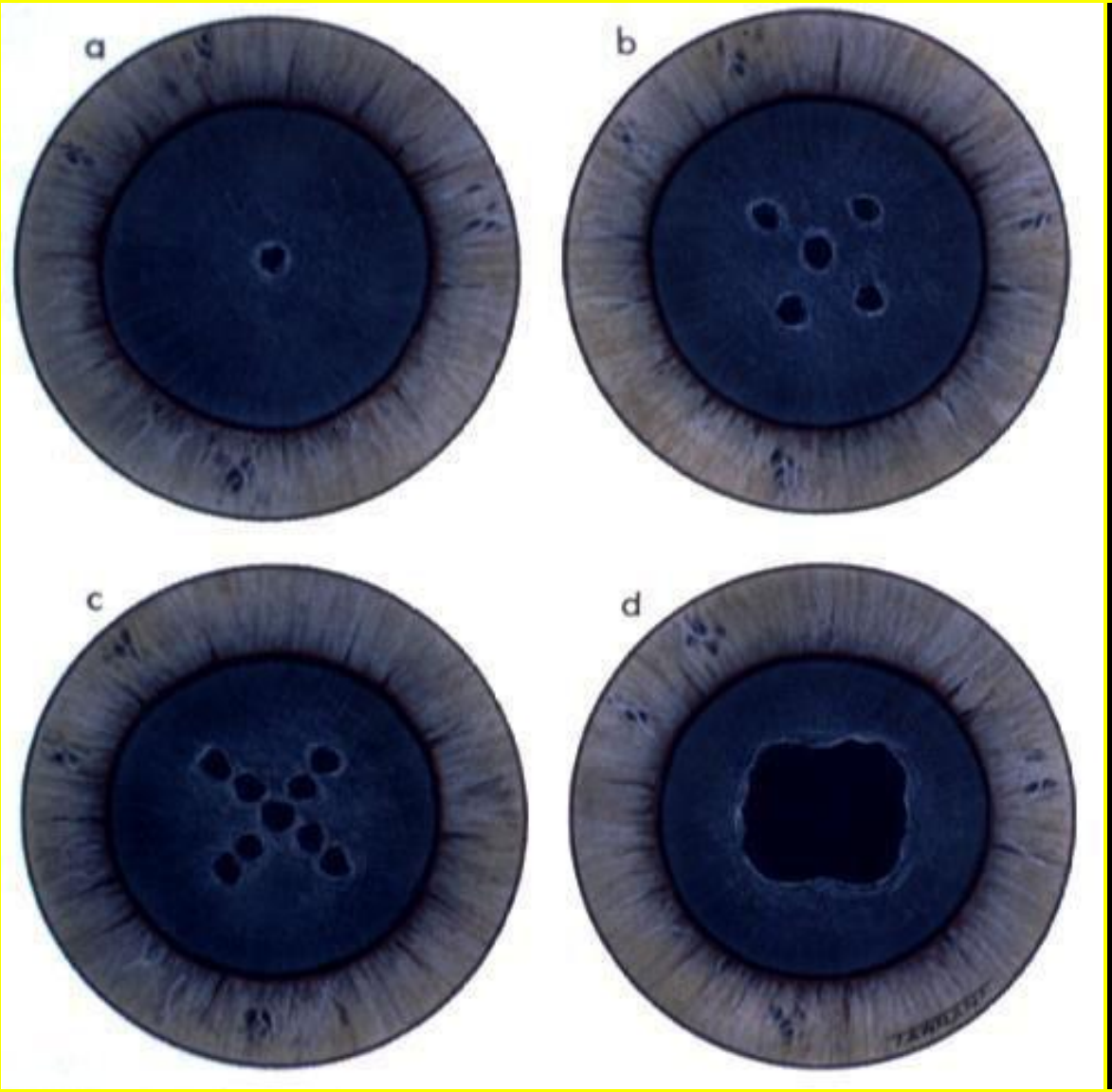
3. Systemic therapy

- Antibiotics are not beneficial
- Steroids only in very severe cases

Types of capsular opacification

Elschnig pearls	Fibrosis
	
• Proliferation of lens epithelium • Occurs after 3-5 years	• Usually occurs within 2-6 months • May involve remnants of anterior capsule and cause phimosis

Treatment of capsular opacification



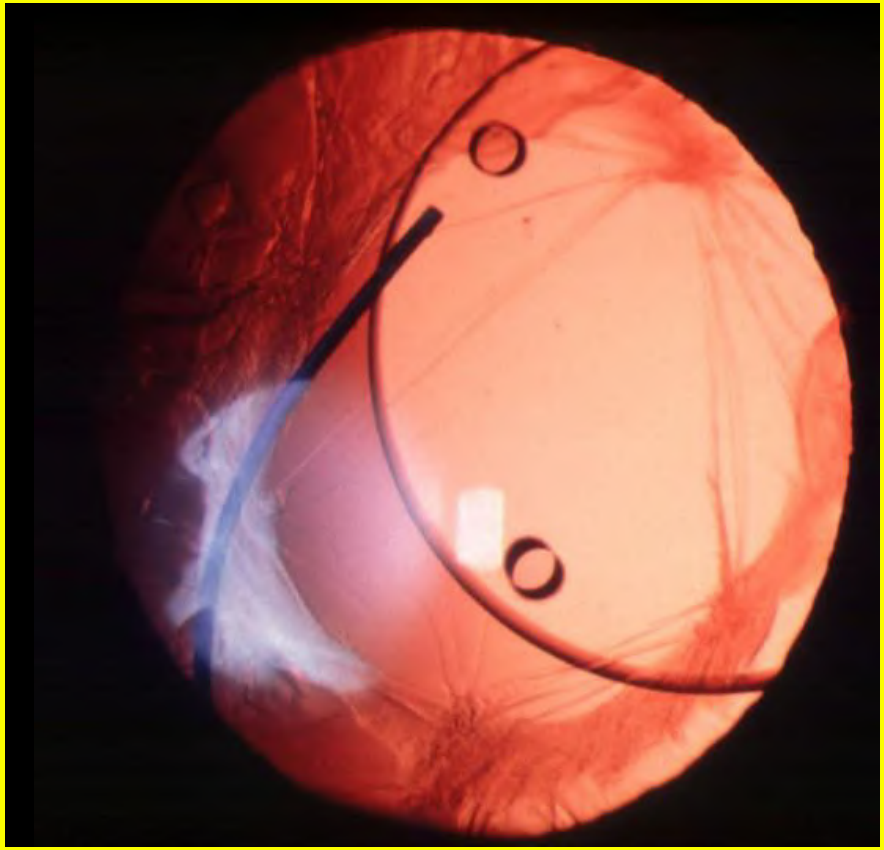
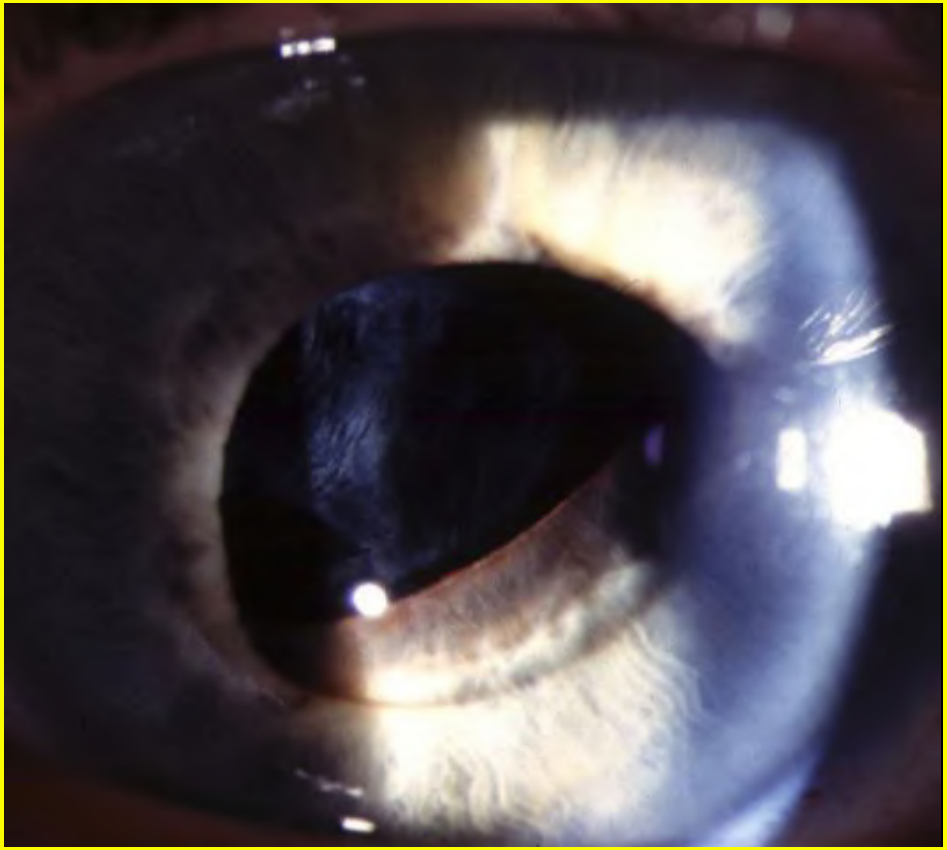
Nd:YAG laser capsulotomy

- Accurate focusing is vital
- Apply series of punctures in cruciate pattern (a-c)
- 3 mm opening is adequate (d)

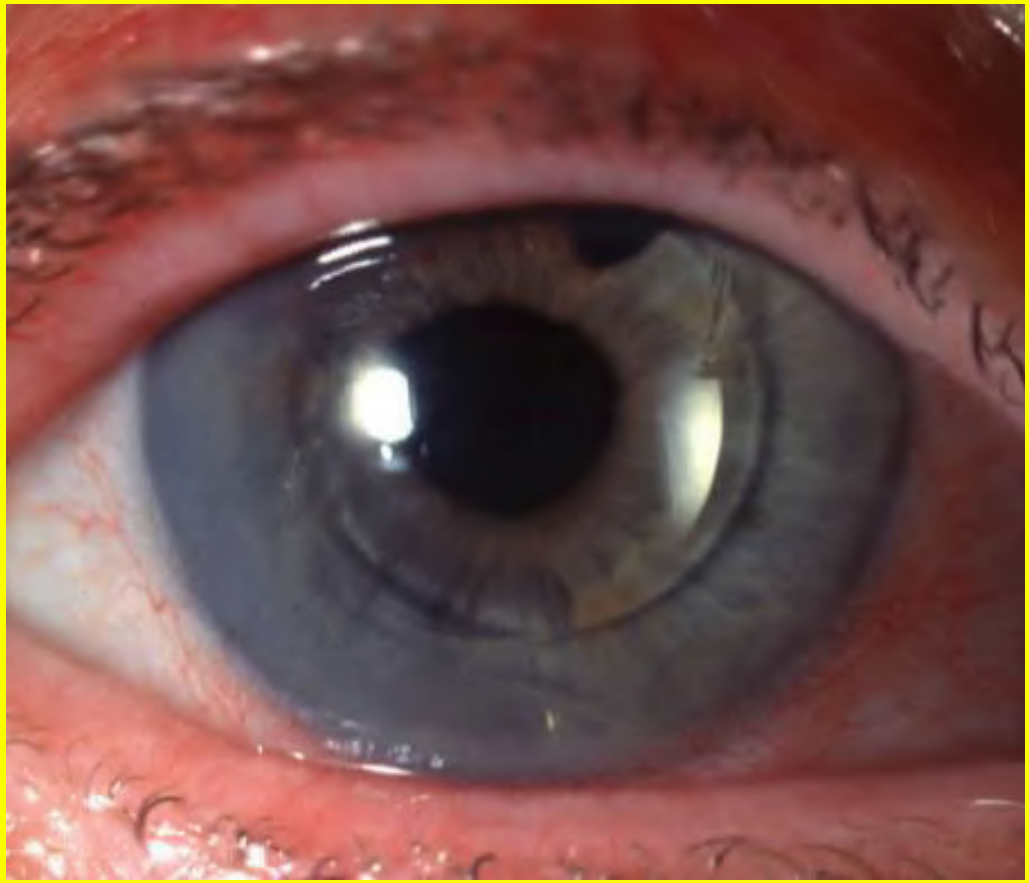
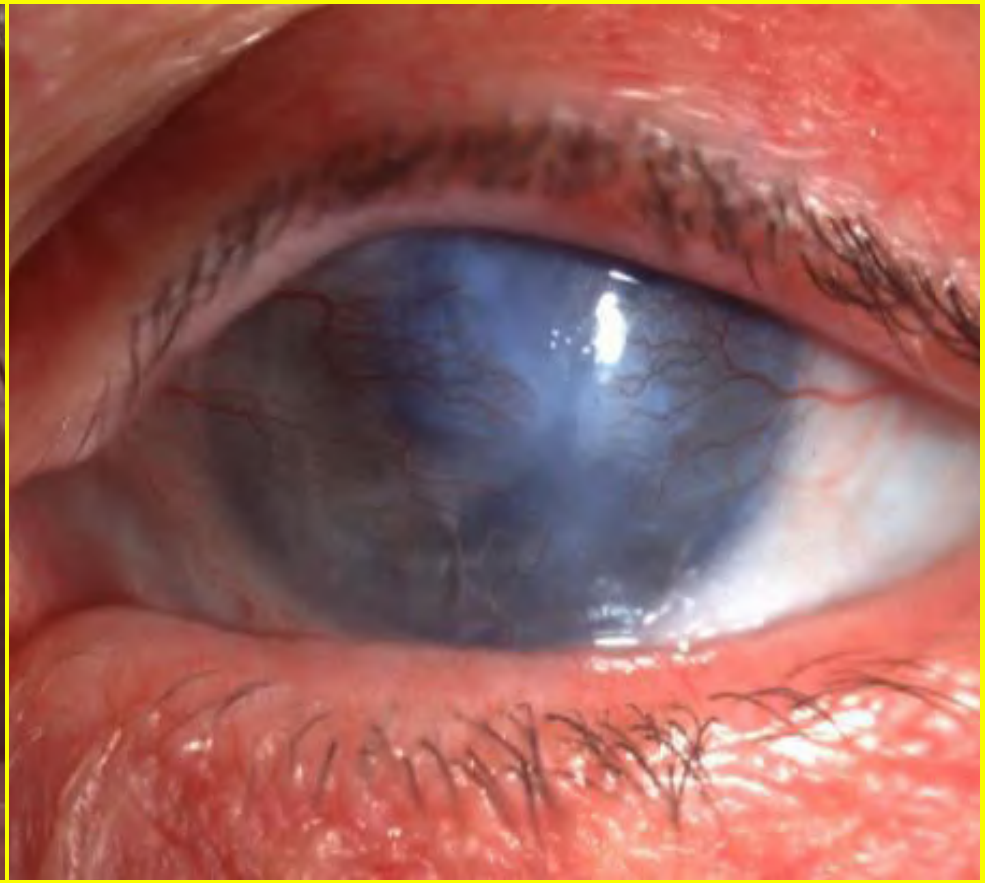
Potential complications

- Damage to implant
- Cystoid macular oedema - uncommon
- Retinal detachment - rare except in high myopes

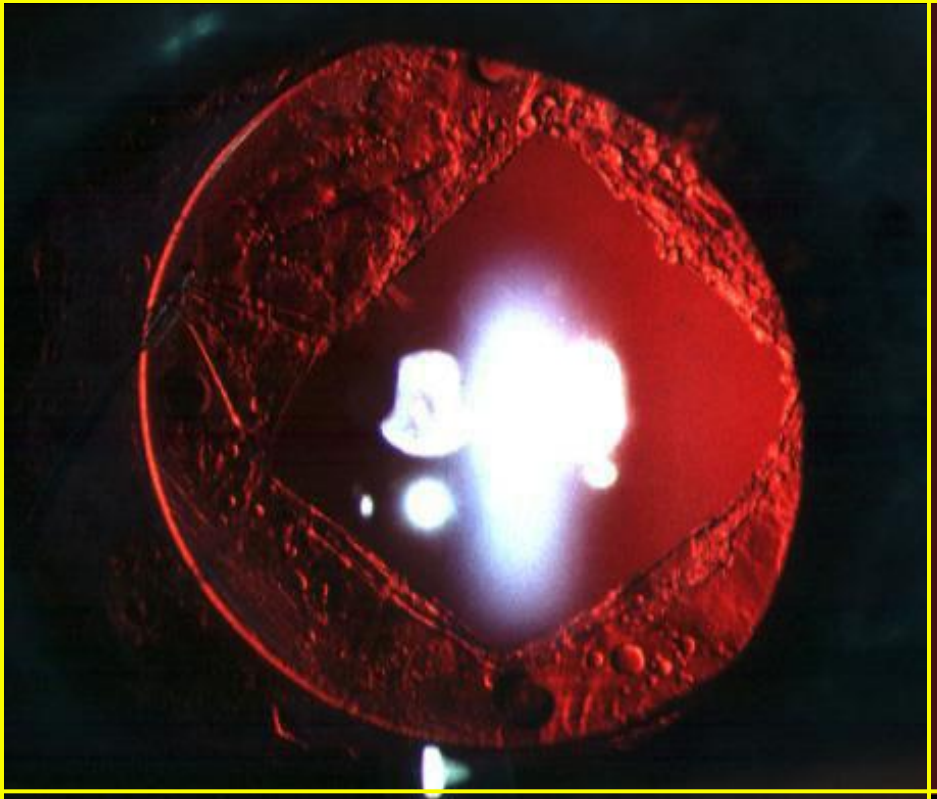
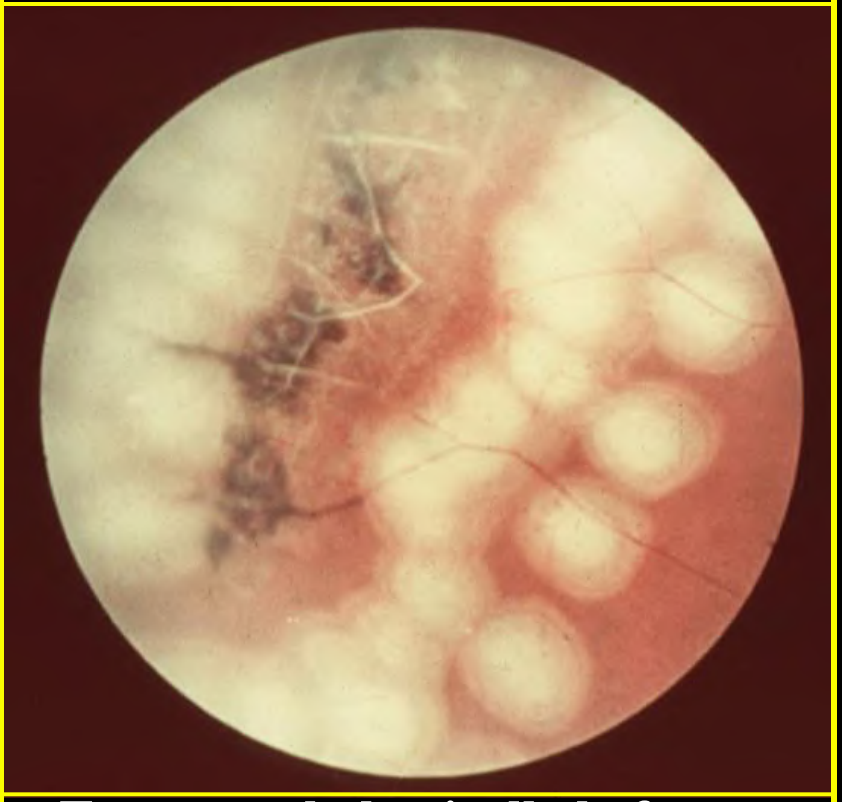
Implant displacement

Decentration	Optic capture
	
• May occur if one haptic is inserted into sulcus and other into bag • Remove and replace if severe	• Reposition may be necessary

Corneal decompensation

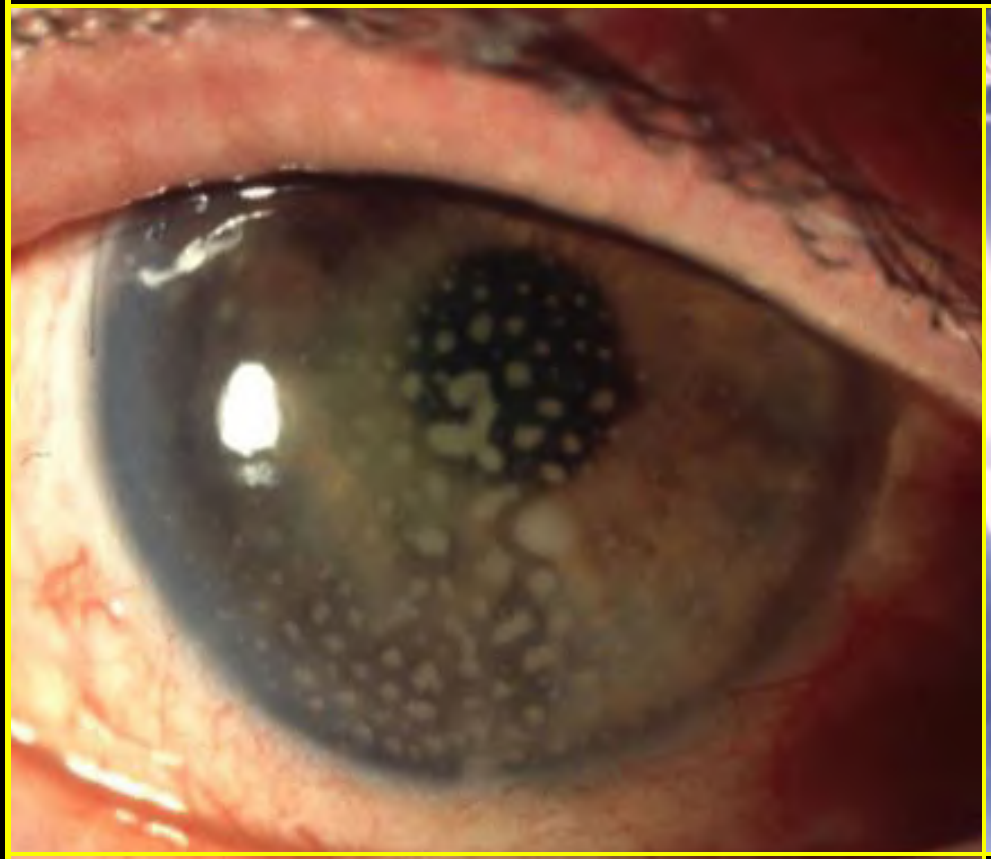
Predispositions	Treatment
	
• Anterior chamber implant • Fuchs endothelial dystrophy	• Penetrating keratoplasty in severe cases • Guarded visual prognosis because of frequently associated CMO

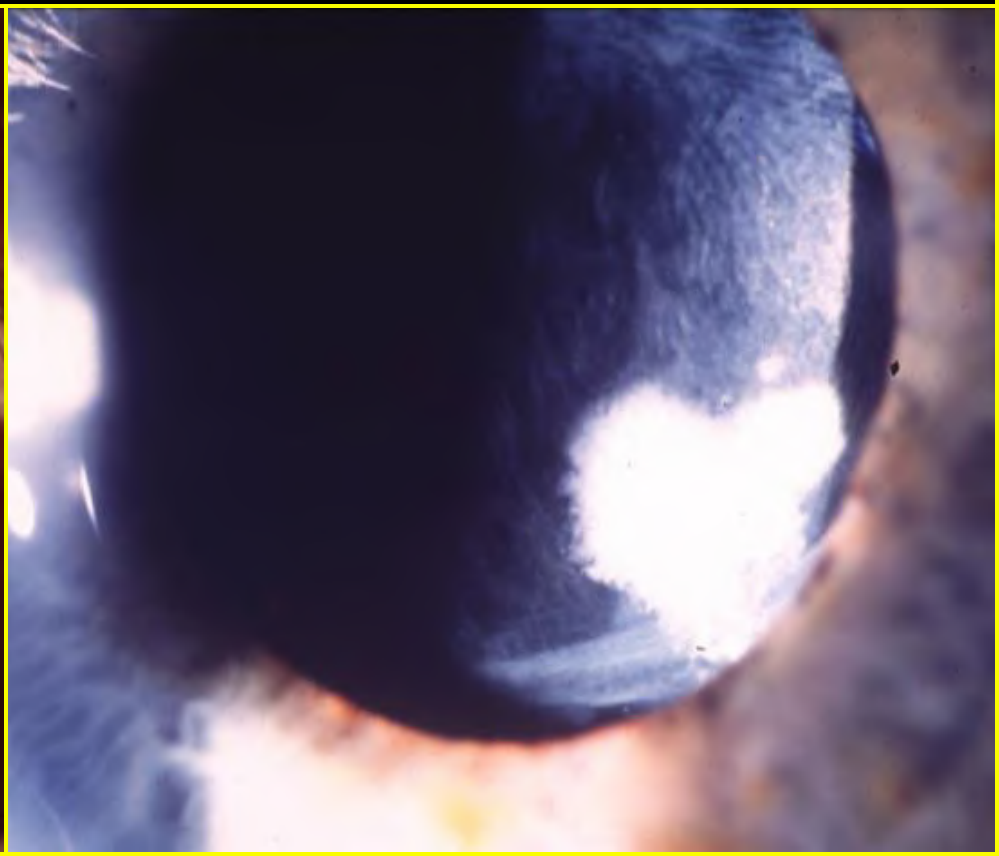
Retinal detachment risk factors

Disruption of posterior capsule	Lattice degeneration
	
• Intraoperative vitreous loss • Laser capsulotomy, particularly in high myopia	• Treat prophylactically before or soon after surgery

Chronic bacterial endophthalmitis

Signs

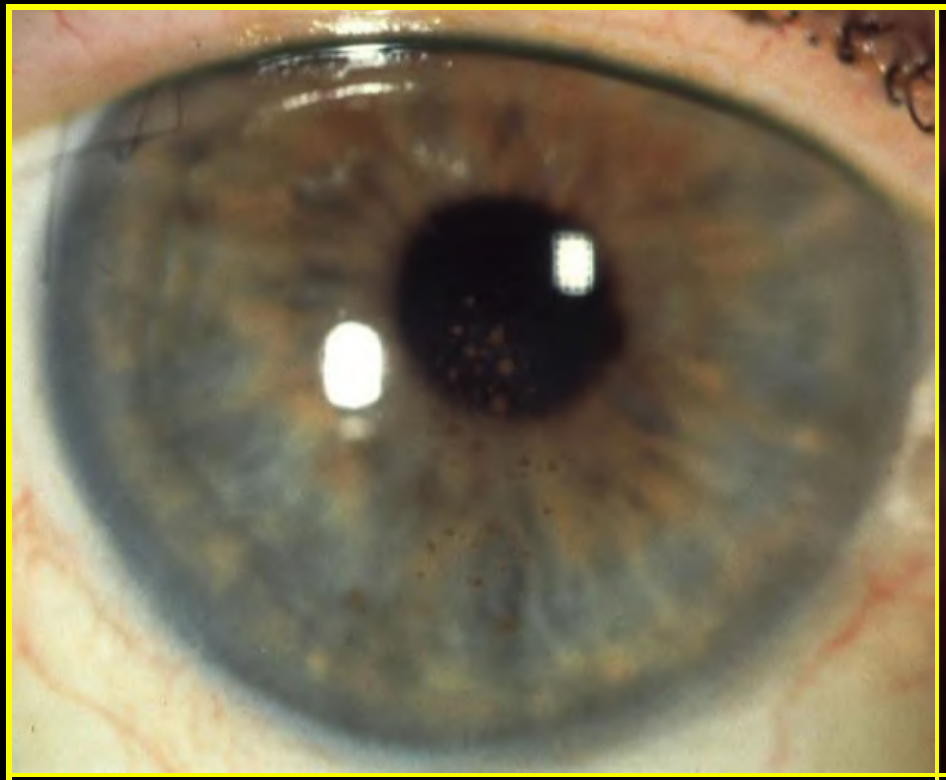




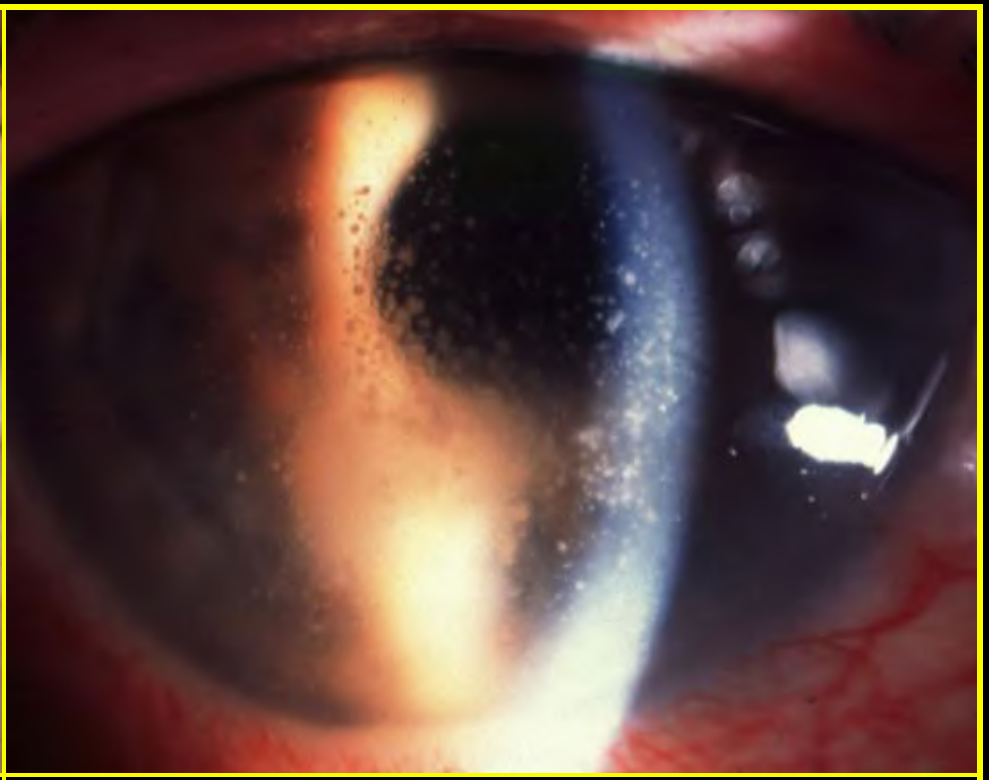
- Late onset, persistent, low-grade uveitis - may be granulomatous
- Commonly caused by *P. acnes* or *Staph. epidermidis*

- Low virulence organisms trapped in capsular bag
- White plaque on posterior capsule

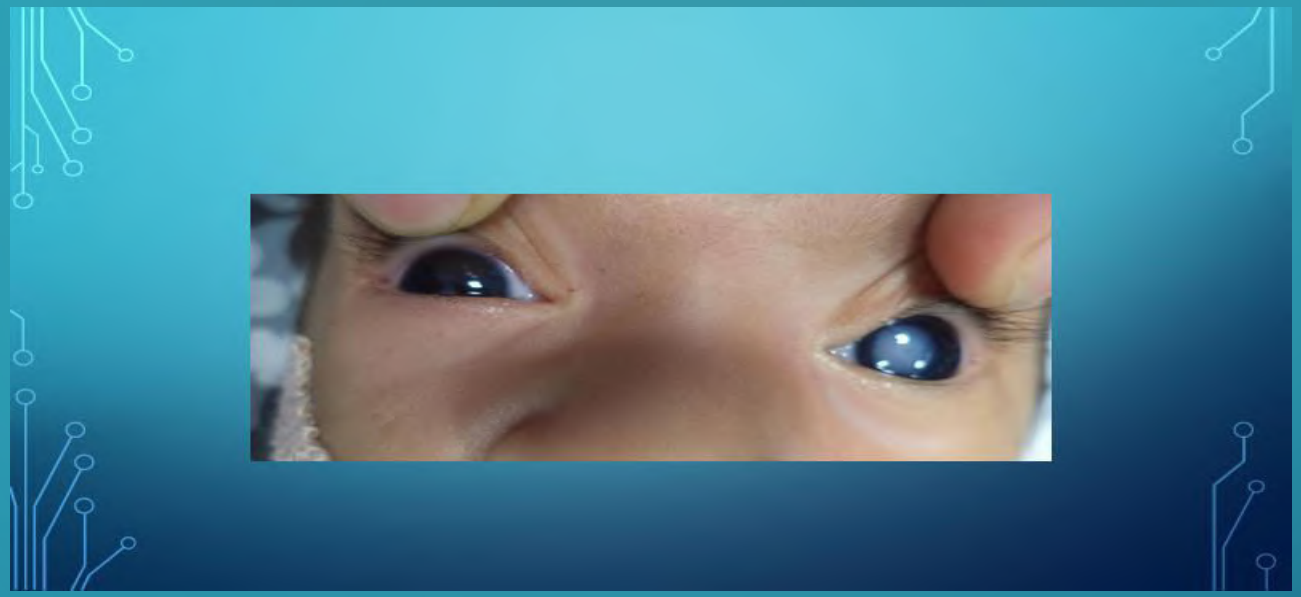
Treatment of chronic endophthalmitis



- Initially good response to topical steroids



- Recurrence after cessation of treatment
- Inject intravitreal vancomycin
- Remove IOL and capsular bag if unresponsive



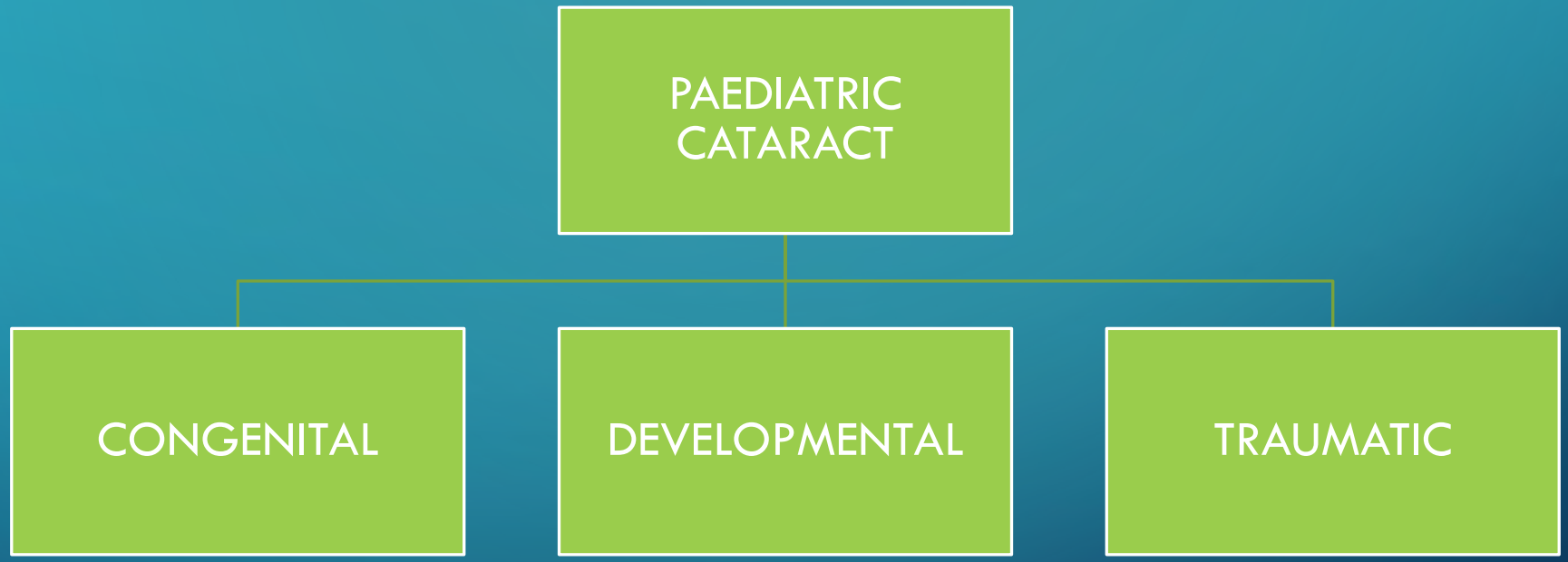
PEADIATRIC CATARACT

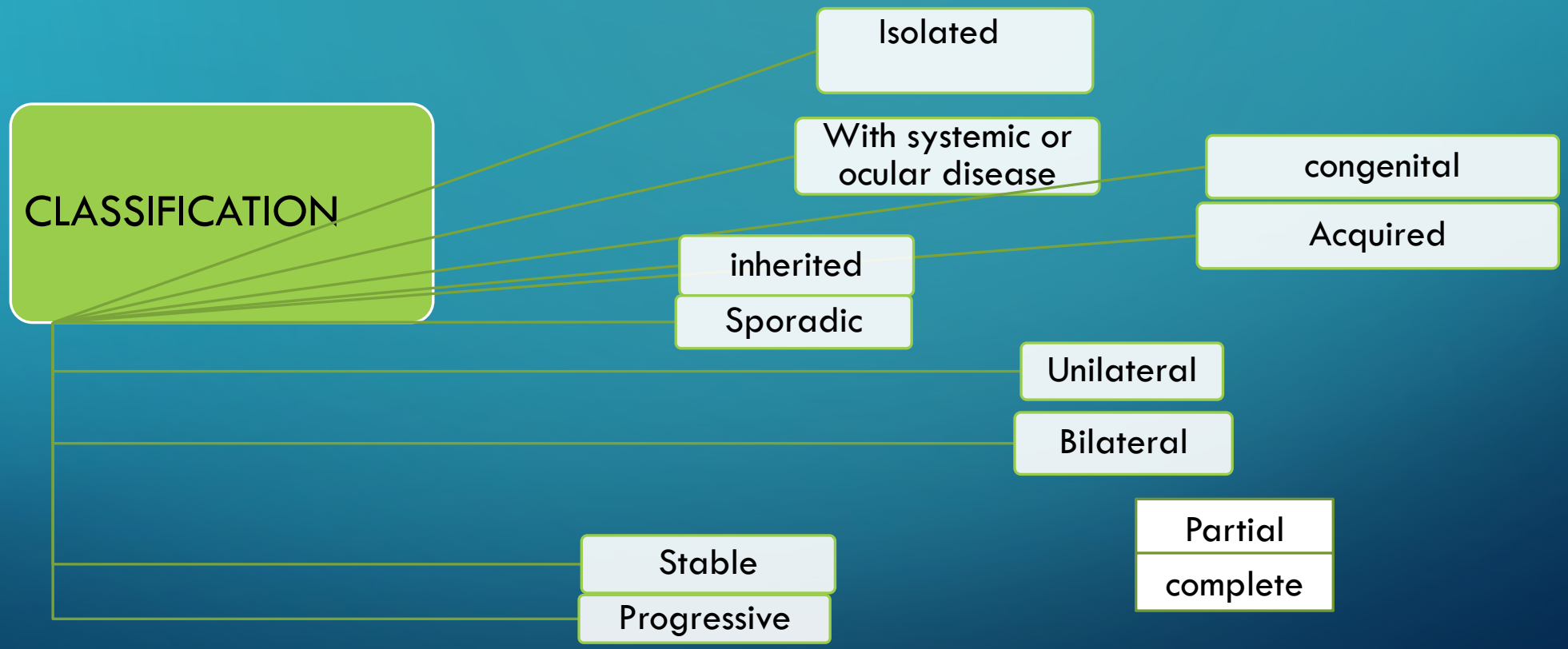
ETIOLOGY AND PRESENTATION

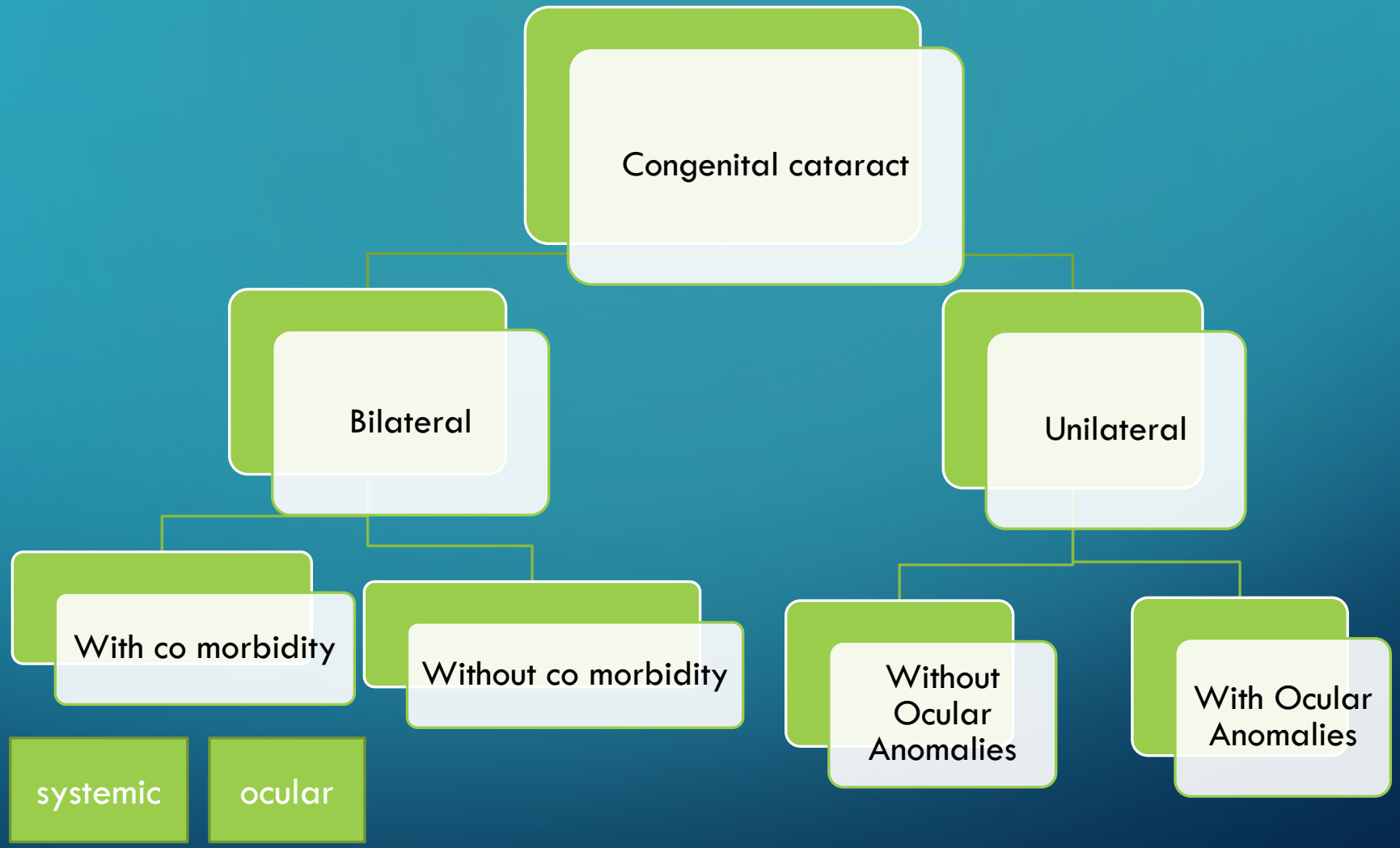
DR AFZAL QADIR

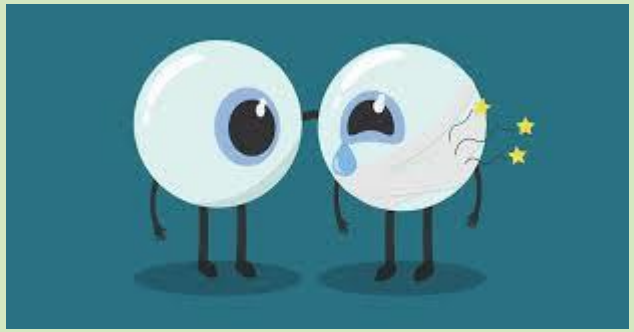
ALL MAJOR SURGERIES SINCE START OF 2022 =1503

PAEDIATRIC CAT = 158


PAEDIATRIC CATARACT
CONGENITAL
DEVELOPMENTAL
TRAUMATIC

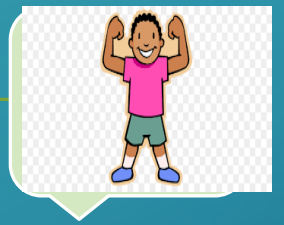

CLASSIFICATION
Isolated
With systemic or ocular disease
inherited
Sporadic
congenital
Acquired
Unilateral
Bilateral
Partial
complete
Stable
Progressive


Congenital cataract
Bilateral
Unilateral
With co morbidity
Without co morbidity
Without Ocular Anomalies
With Ocular Anomalies
systemic
ocular



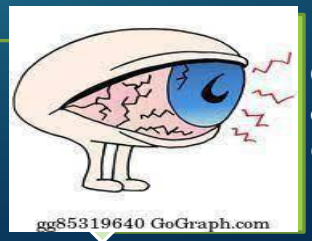
Unilateral Cataract

No Family history



Healthy

cause



Other ocular anomalies

Hereditary(usually dominant)

Idiopathic

With ocular anomalies

(27%)

. PFV
Aniridia
Coloboma
Microphthalmos
Buphthalmos

systemic
22%

Metabolic

Genetic
mutation
(Autosomal
Dominant

Galactosemic
Lowe
Hypoparathyroidism
Fabry
Hypoglycemia

Skeletal
Hallerman
Streiff Francois
syndrome

Chromosomal
Trisomy –21
Trisomy-18
Trisomy -13

Ocular Syndrome
Anterior segment
Dysgenesis

Infective
TORCH

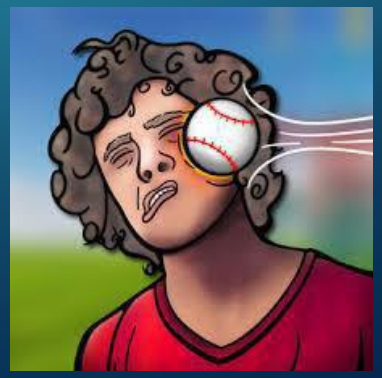
Idiopathic

Bilateral cataracts	Unilateral cataracts
Idiopathic	Idiopathic
Hereditary cataracts (autosomal dominant)	Ocular anomalies
Genetic and metabolic diseases	PFV
Down syndrome	Anterior segment dysgenesis
Lowe syndrome	Posterior lenticonus
Galactosemia	Posterior pole tumors
Marfan's syndrome	Traumatic (rule out child abuse)
Trisomy 13-15	Rubella (rarely)
Hypoglycemia	Asymmetric bilateral cataract
Alport syndrome	
Myotonic dystrophy	
Fabry disease	
Hypoparathyroidism	
Maternal infection	
Rubella	
Cytomegalovirus	
Varicella	
Syphilis	
Toxoplasmosis	
Ocular anomalies	
Aniridia	
Anterior segment dysgenesis syndrome	
Corticosteroids	

PFV: Persistent fetal vasculature

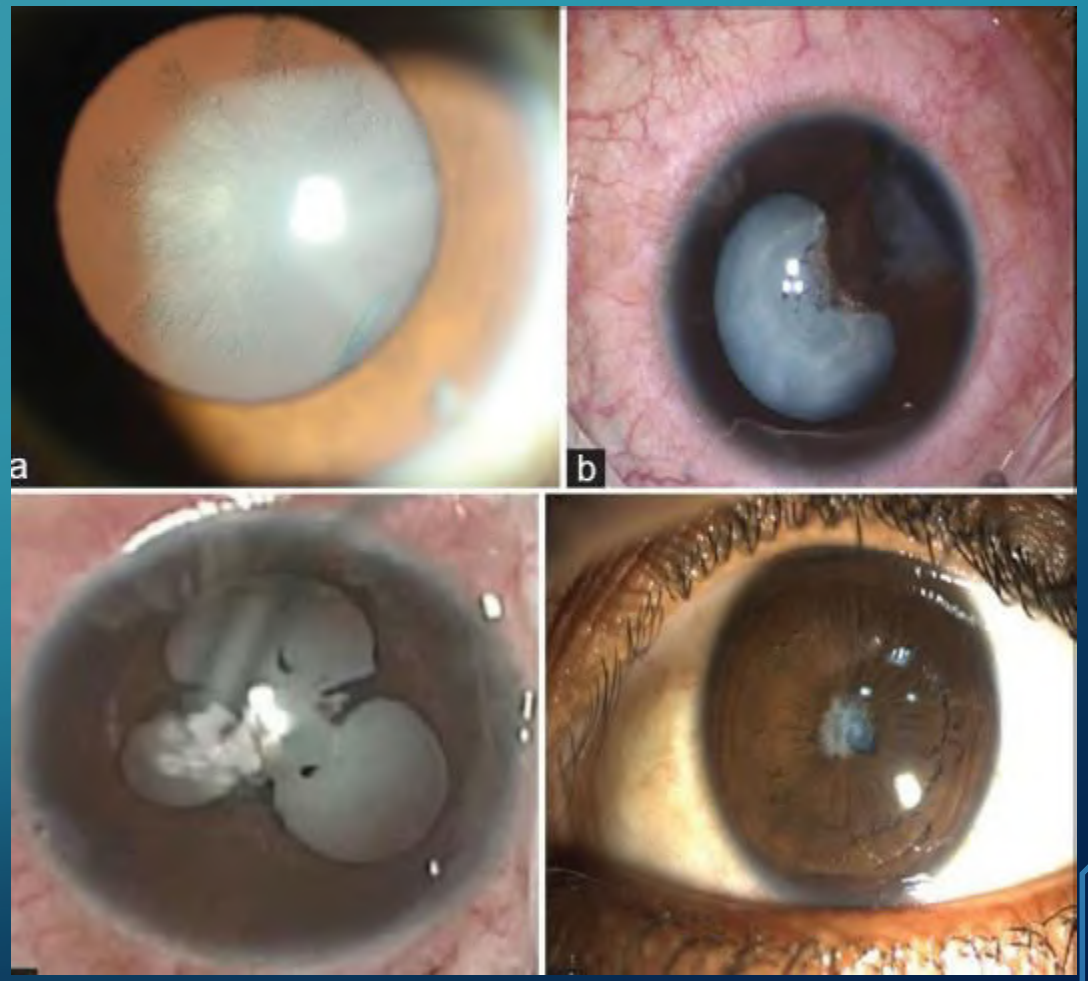
SURPRISINGLY….

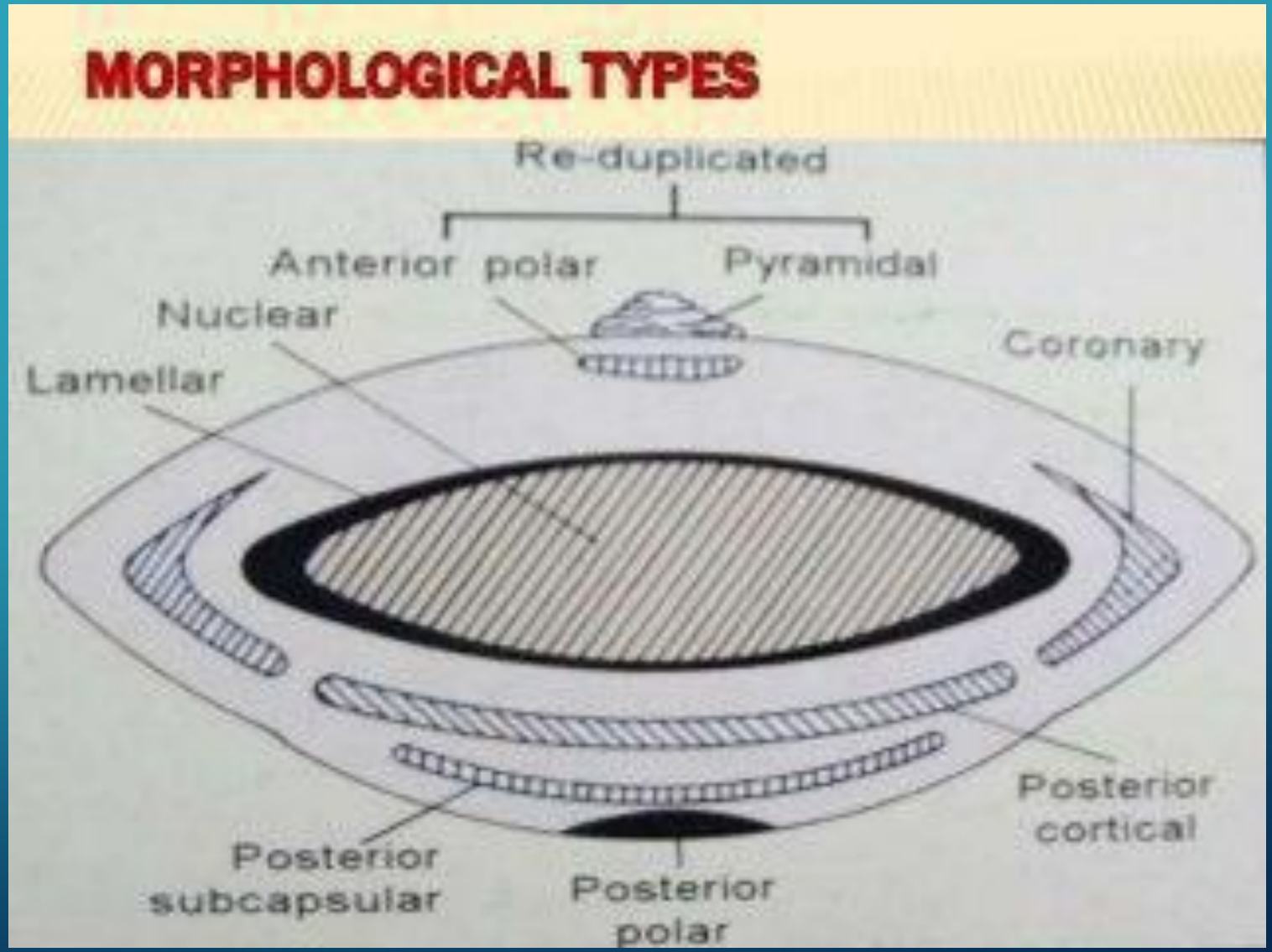
TRAUMATIC PAEDIATRIC CATARACT



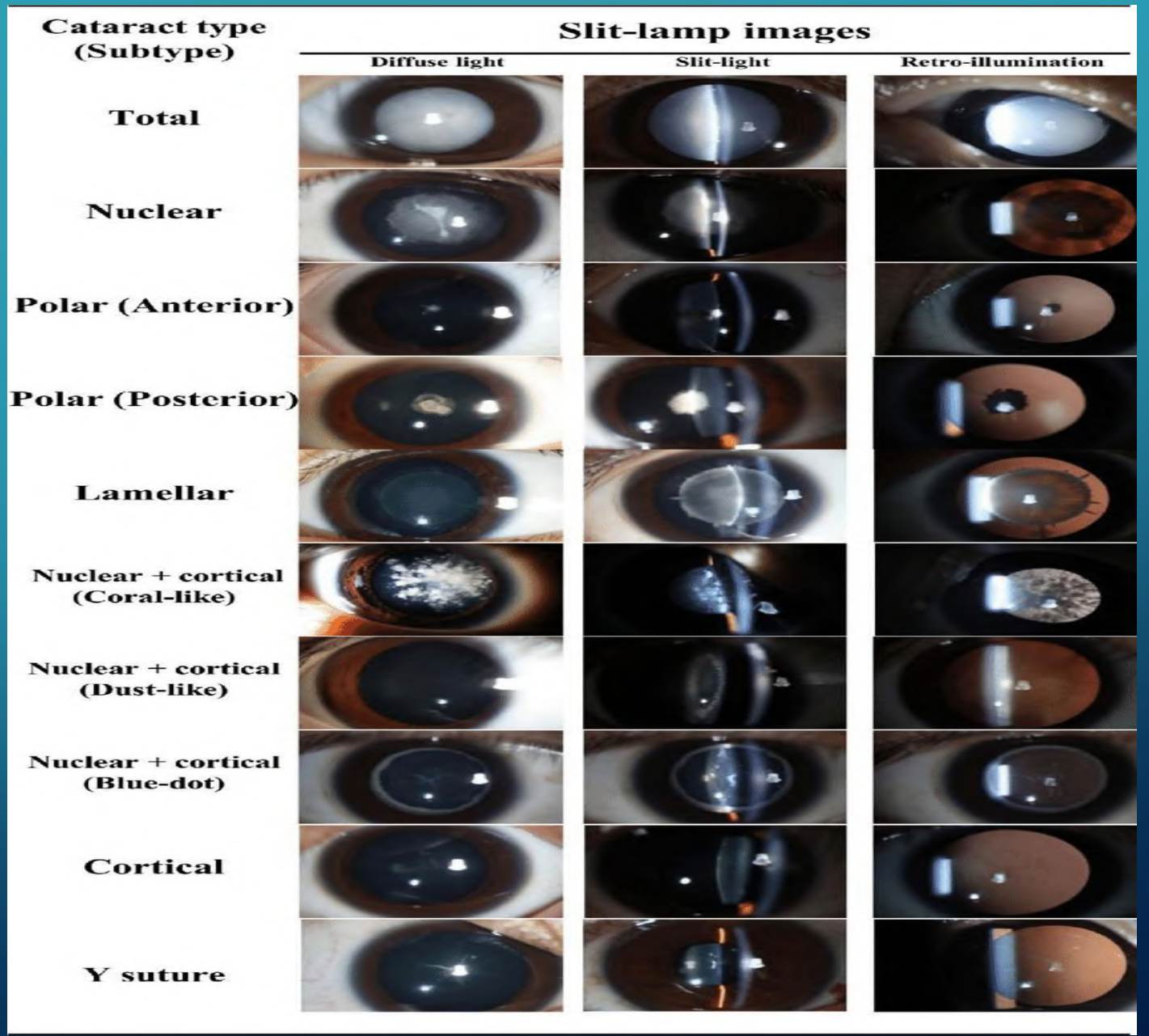
TRAUMATIC CATARACT

- Open or closed
- Blunt or sharp
- Early or late

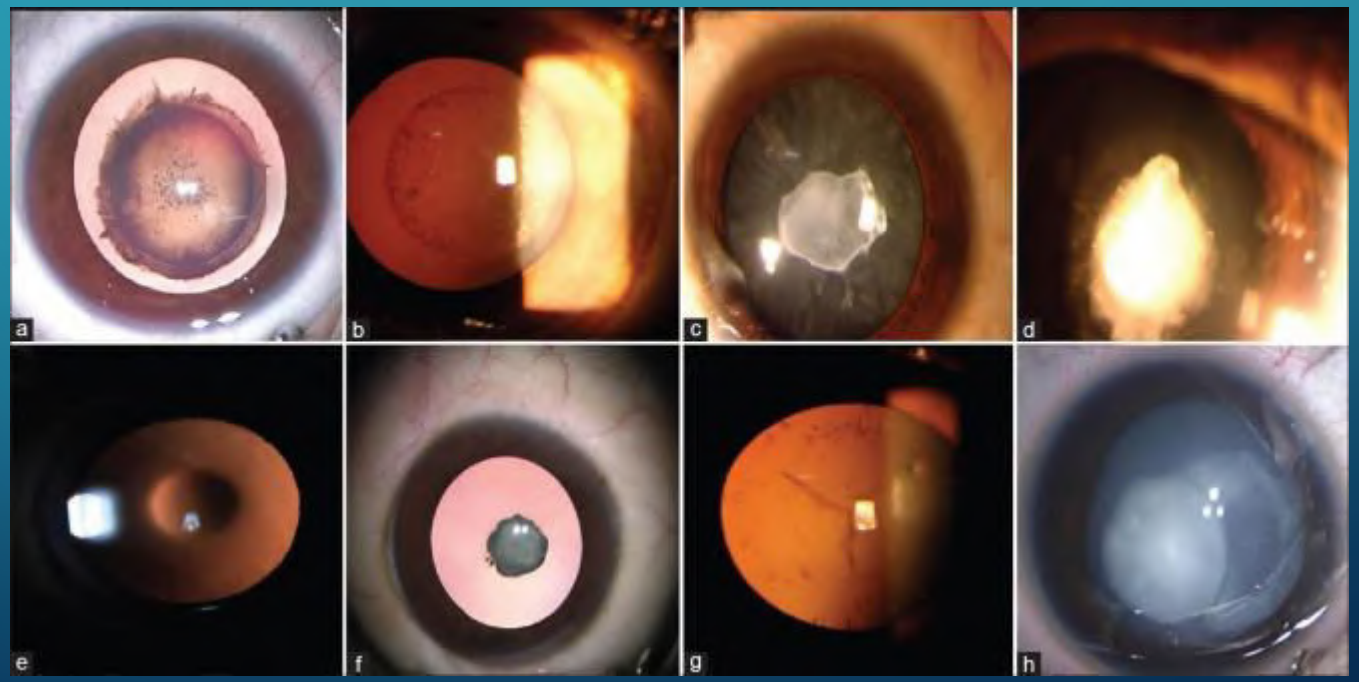



MORPHOLOGICAL TYPES
Re-duplicated
Anterior polar
Pyramidal
Nuclear
Lamellar
Coronary
Posterior cortical
Posterior subcapsular
Posterior polar

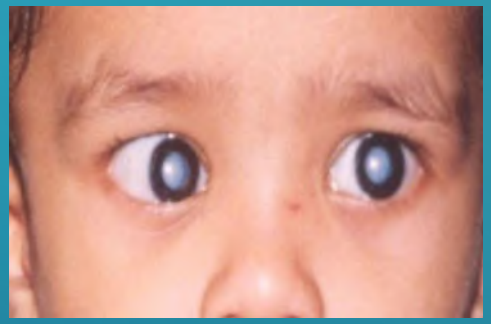
Morphology


Cataract type (Subtype)
Slit-lamp images
Diffuse light
Slit-light
Retro-illumination
Total
Nuclear
Polar (Anterior)
Polar (Posterior)
Lamellar
Nuclear + cortical (Coral-like)
Nuclear + cortical (Dust-like)
Nuclear + cortical (Blue-dot)
Cortical
Y suture

Whole lens	Central	Anterior	Posterior	Miscellaneous
Total	Lamellar	Anterior polar	Mittendorf dots	Punctate lens opacities
Congenital Morgagnian	Central pulverulent	Dot like	Posterior cortical	Sutural
Membranous	Ant egg	Plaque like	Posterior subcapsular	Coralliform
	Nuclear	Anterior pyramidal	Posterior lenticonus	Wedge shaped
	Oil droplet	Anterior subcapsular		Persistent hyperplastic primary vitreous
	Cortical	Anterior lenticonus		
	Coronary			

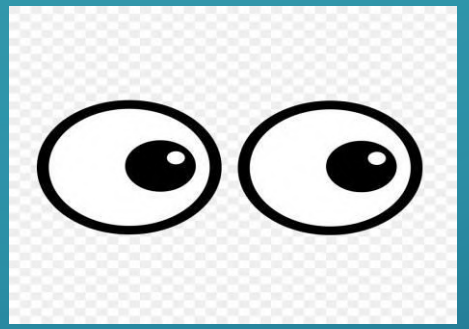


SYMPTOMS



White pupil

(24%)



Squinting eyes

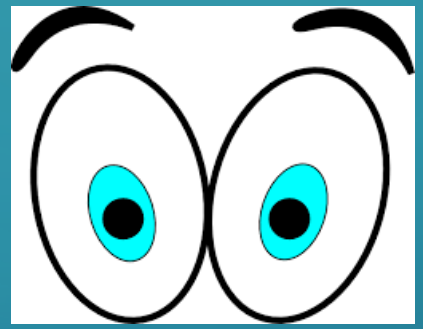
(19%)

Accidental Finding

(41%)

SYMPTOMS

Does not follow objects / localize light



Nystagmus

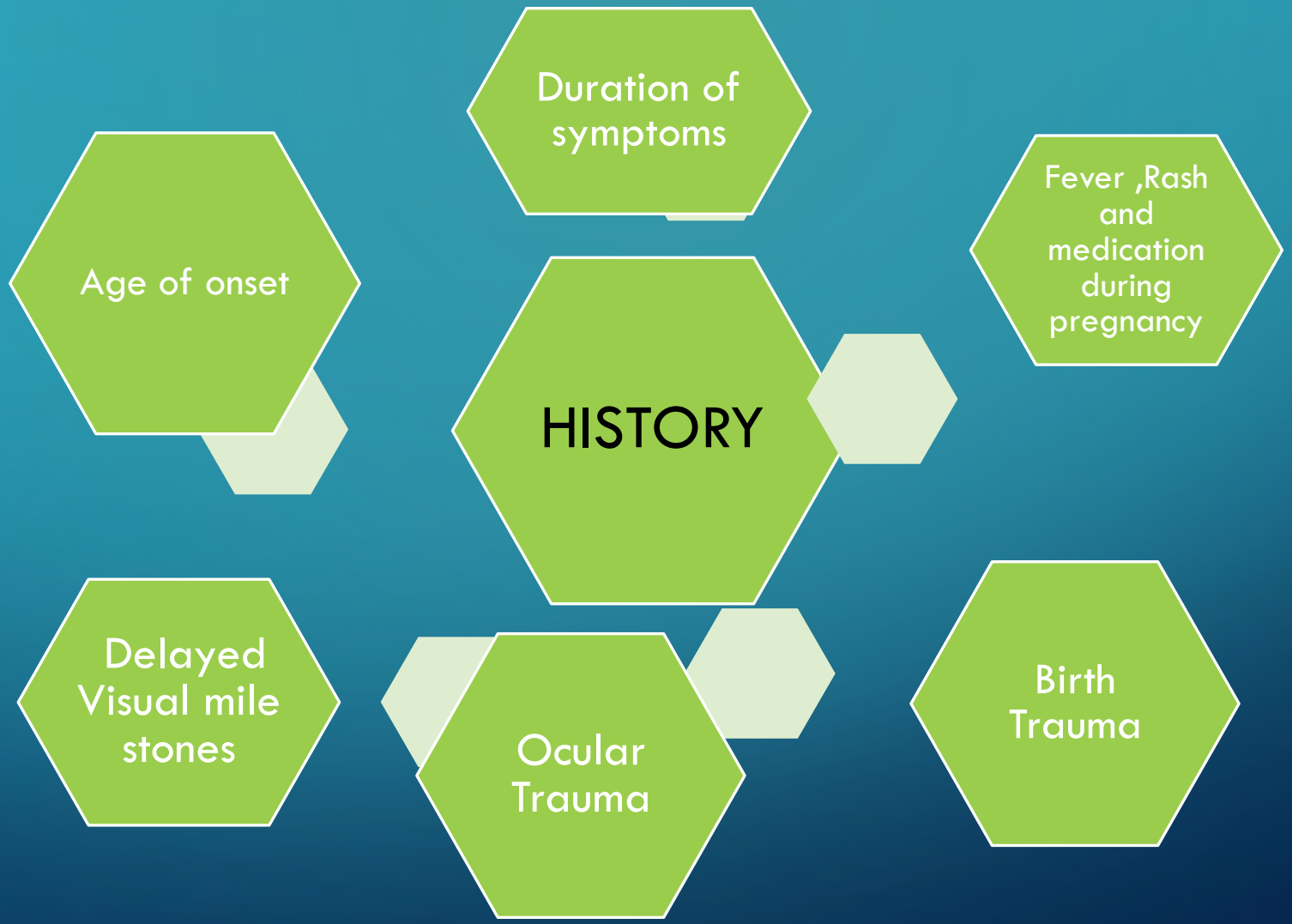
Associated with systemic disease

SYMPTOMS

Photophobia

Microphthalmia

Buphthalmos


Duration of symptoms
Age of onset
HISTORY
Fever ,Rash and medication during pregnancy
Delayed Visual mile stones
Ocular Trauma
Birth Trauma

DEVELOPMENTAL CATARACT

- Older Children
- Difficulty in viewing distant objects
- Inability to read black board
- Bringing objects close to face
- Viewing TV at close distance

THANK YOU!

“ TEE TIME ”

Dr. Yousaf Jamal Mahsood

MBBS, CHPE, CMEJ, MHR, FICO (UK),
MRCSEd (UK), FRCS (Glasg), FCPS

Fellowship in Glaucoma (Al-Shifa Trust, Pak)
Fellowship in Glaucoma (Univ. of Toronto, Canada)
Advance Fellowship in Glaucoma (BPOS, UK)

Assistant Professor Glaucoma
Department of Ophthalmology
Khyber Girls Medical College
Peshawar

Cataract

Learning Objectives

- Define cataract
- Describe the types of Age-related cataract
- Describe the pathogenesis and complications of cataract
- Describe the management of cataract

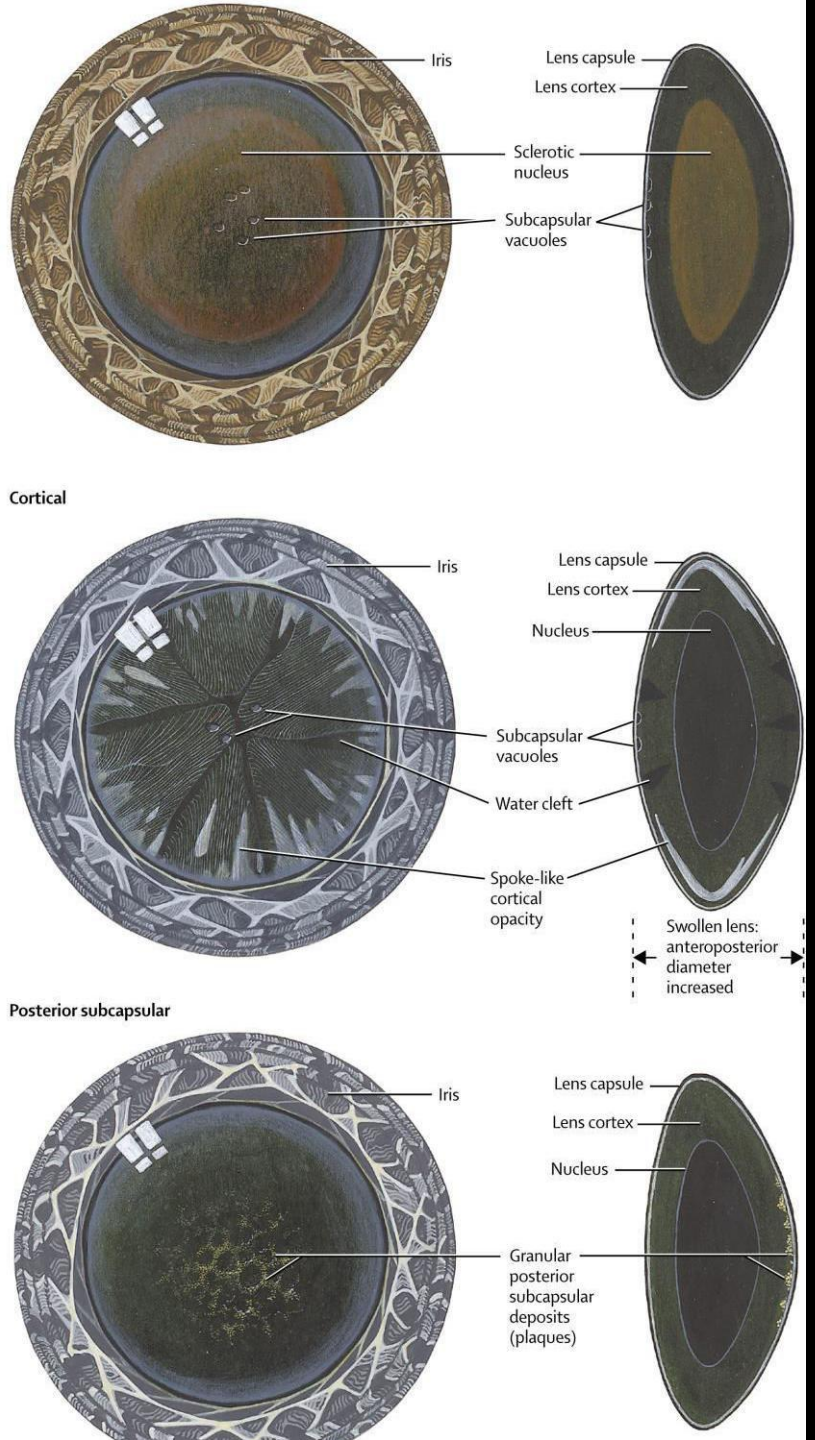
Learning Objectives

Define cataract

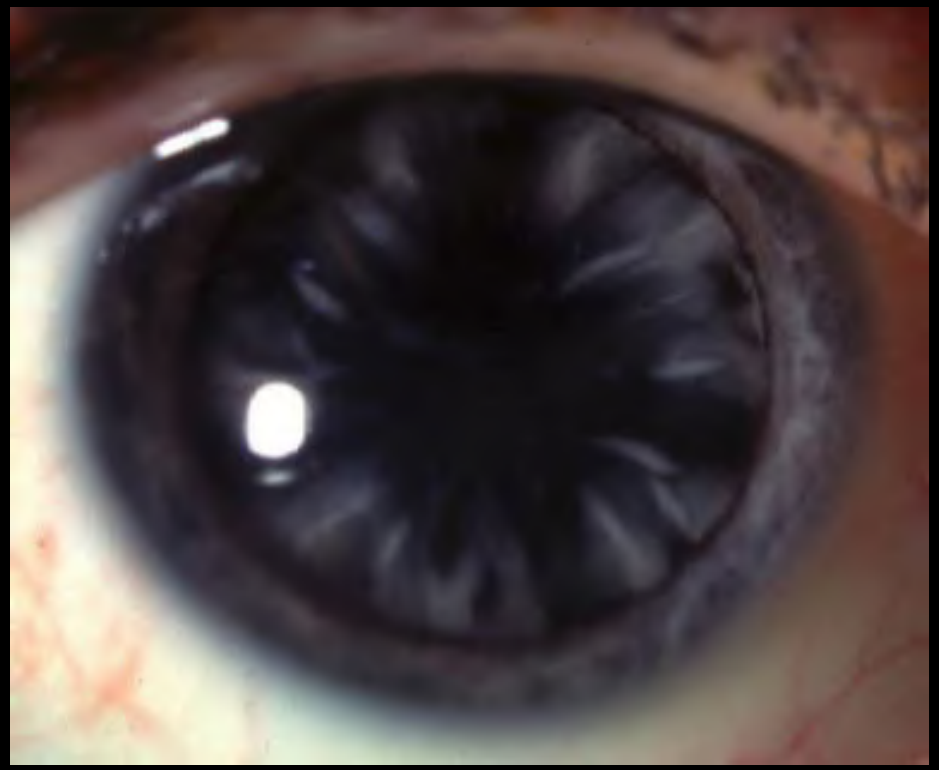
Opacification of the crystalline lens or capsule

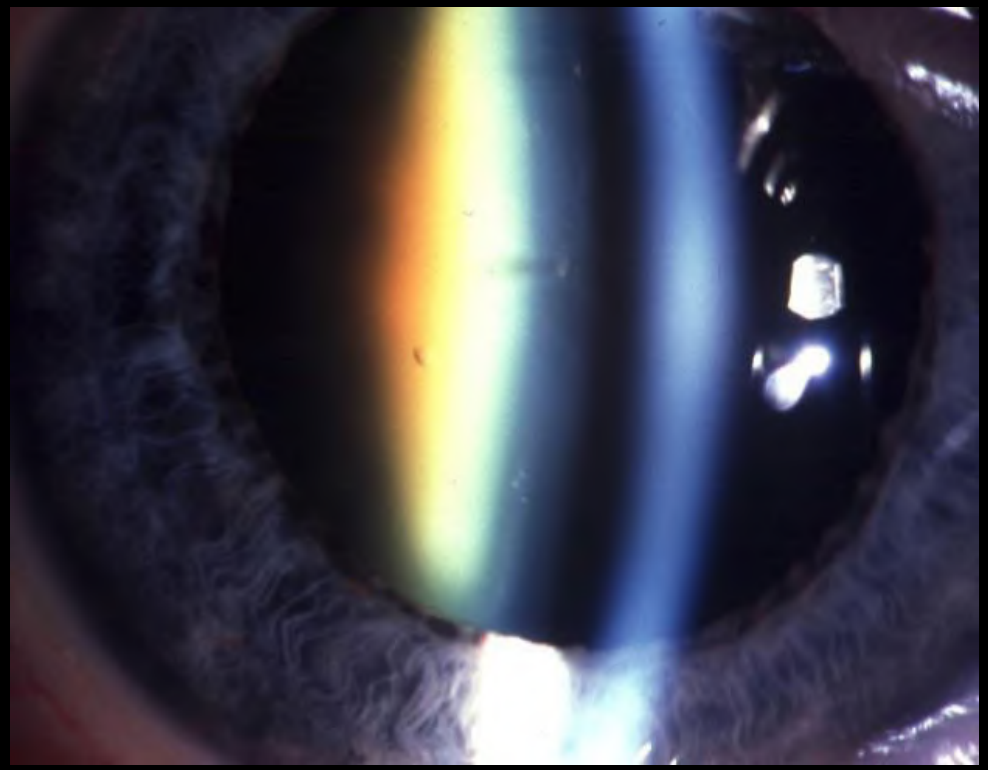
Types of Age-related cataract

- Nuclear
- Cortical
- Subcapsular



Quiz





Pathogenesis

- Multifactorial
  - Genetic
  - Environmental
  - Metabolic

Complications

- If left untreated can lead to:
  - Visual impairment
  - Reduced contrast sensitivity
  - Color vision
  - Glare
  - Double vision
  - Secondary glaucoma

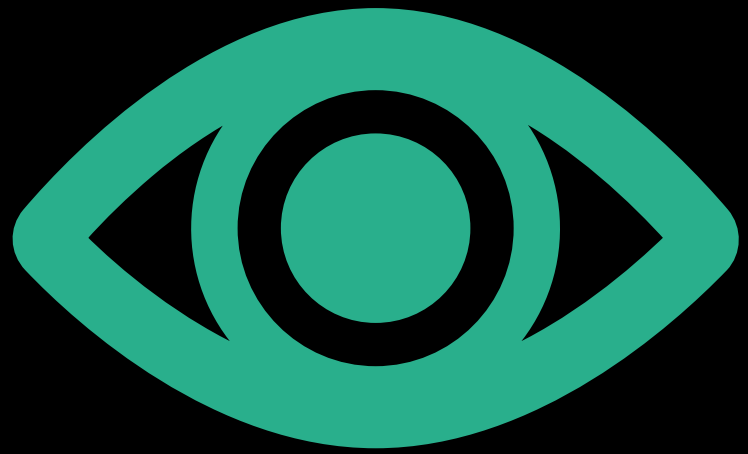
Pre-op assessment

Aims of pre-op assessment

- Confirm diagnosis
- Determine prognosis
- Plan surgery

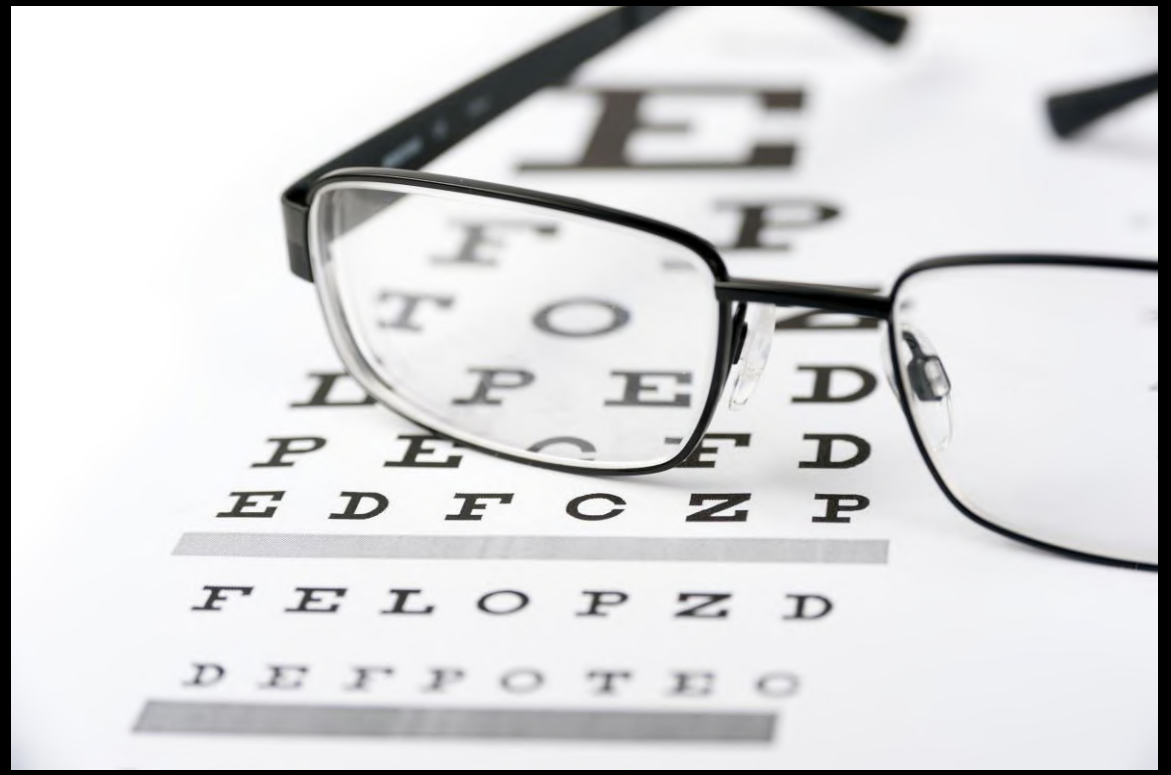
Confirm diagnosis

- Ensure visual loss is due to cataract
- Check:
  - VA
  - Pupil reaction
  - Red reflex
  - IOP
- Remember: Glaucoma is also a common cause of gradual loss of vision in older people



Determine prognosis

- Identify co-morbidity
  - Diabetes
  - Glaucoma
  - ARMD
  - Previous trauma
  - Corneal opacity
  - Amblyopia



Determine risk

- Identify risk factors for complications
  - Corneal opacity
  - Shallow AC
  - Small pupil
  - Stuck pupil
  - Pseudo-exfoliation
  - Uncooperative patient

Indications for surgery

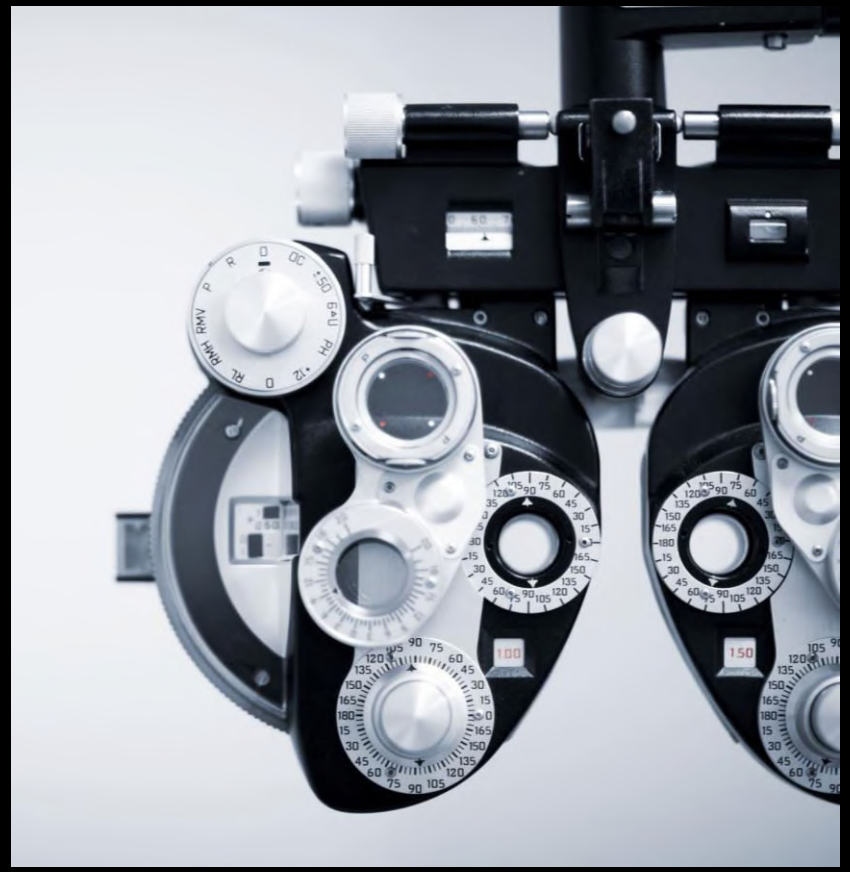
- VA is sufficiently poor that surgery is likely to improve it
- Patient has symptoms of poor vision, which will be relieved by cataract surgery
- Patient experiences limitations in normal activities because of poor vision
- Complications of cataract – uveitis, glaucoma

Plan surgery

- Choose the appropriate operation, based on:
  - Eye examination
  - Surgeon skills
  - Available equipment
- Prepare patient accordingly

Biometry

- Determining correct power of IOL for individual patient.
- Keratometry measures power of cornea
- A-scan / Laser interferometer measures length of eye
- Calculate correct power of IOL to focus light on retina, using regression formula



Cataract surgery

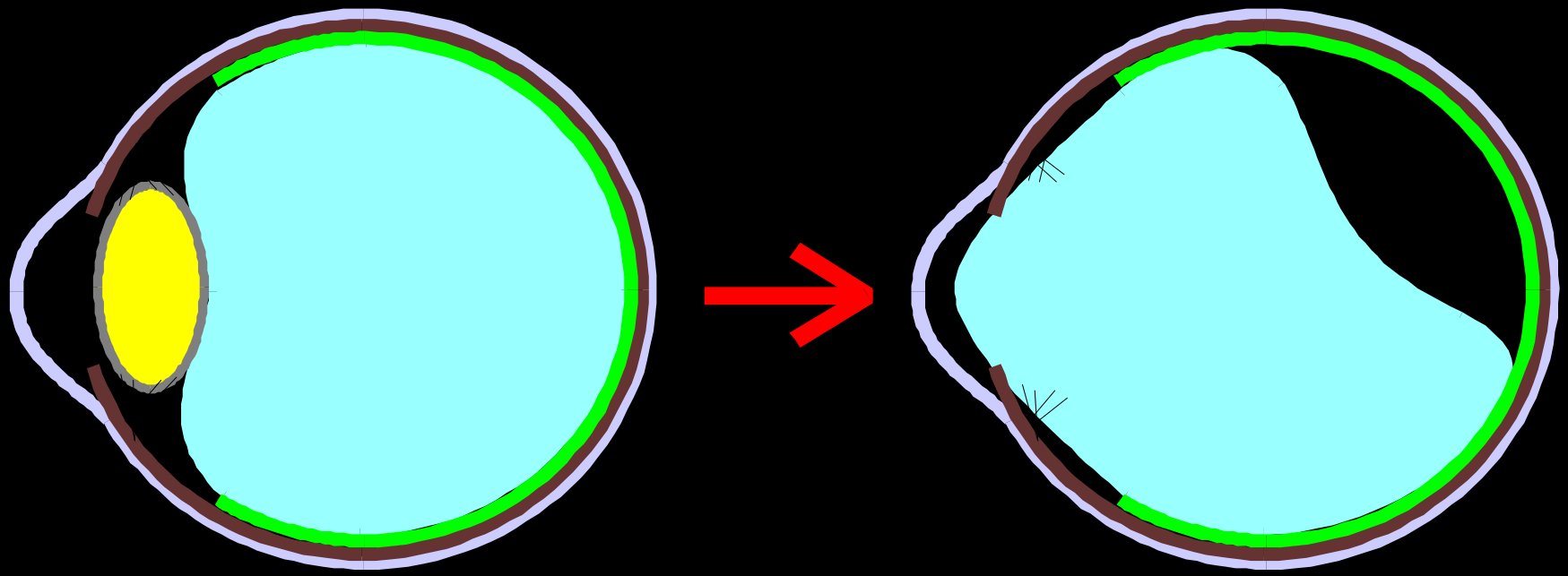
ANESTHESIA

- Retrobulbar
- Peribulbar
- Sub-tenon’s
- Topical
- General

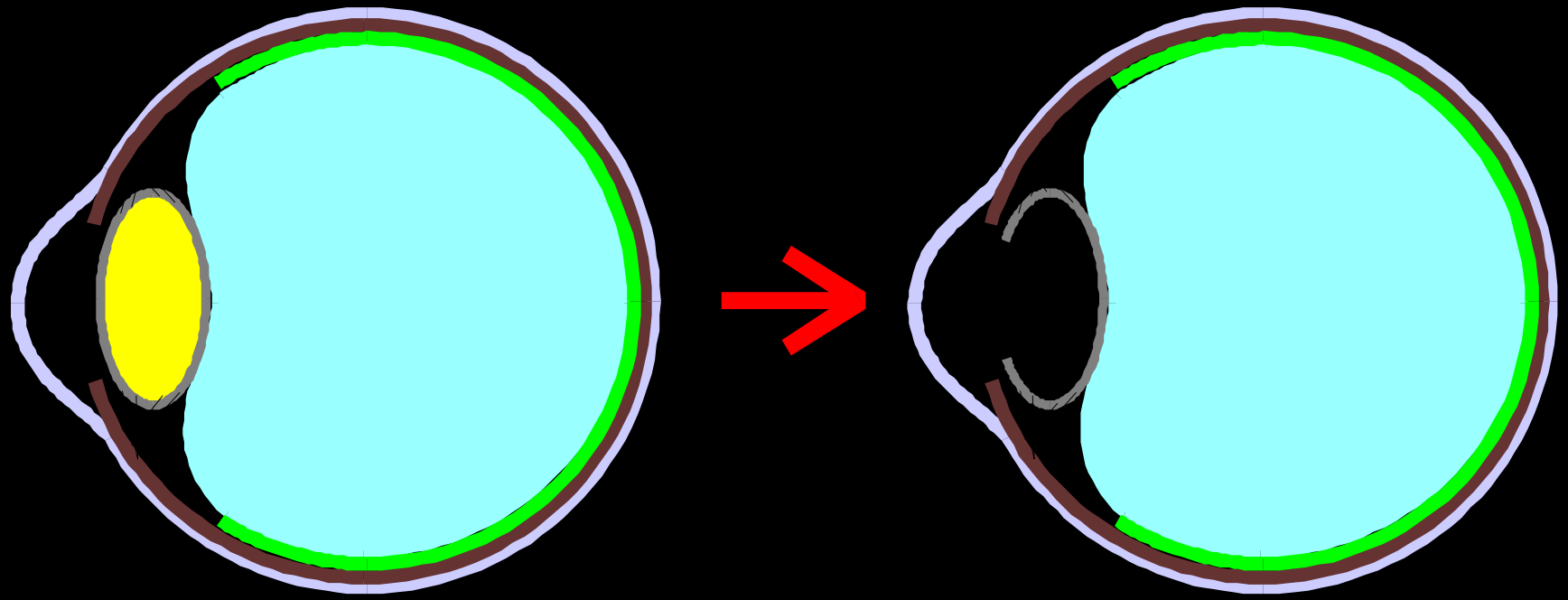
Introduction

- Surgical techniques to remove a cataract:
  - Intracapsular (ICCE)
  - Extracapsular (ECCE)
  - Suture less / MSICS (ECCE)
  - Phacoemulsification (Phaco)
  - FLACS

ICCE



ECCE



Phacoemulsification

Summary

Learning Objectives

- Define cataract
- Describe the types of Age-related cataract
- Describe the pathogenesis and complications of cataract
- Describe the management of cataract

Any questions

Thank you everyone

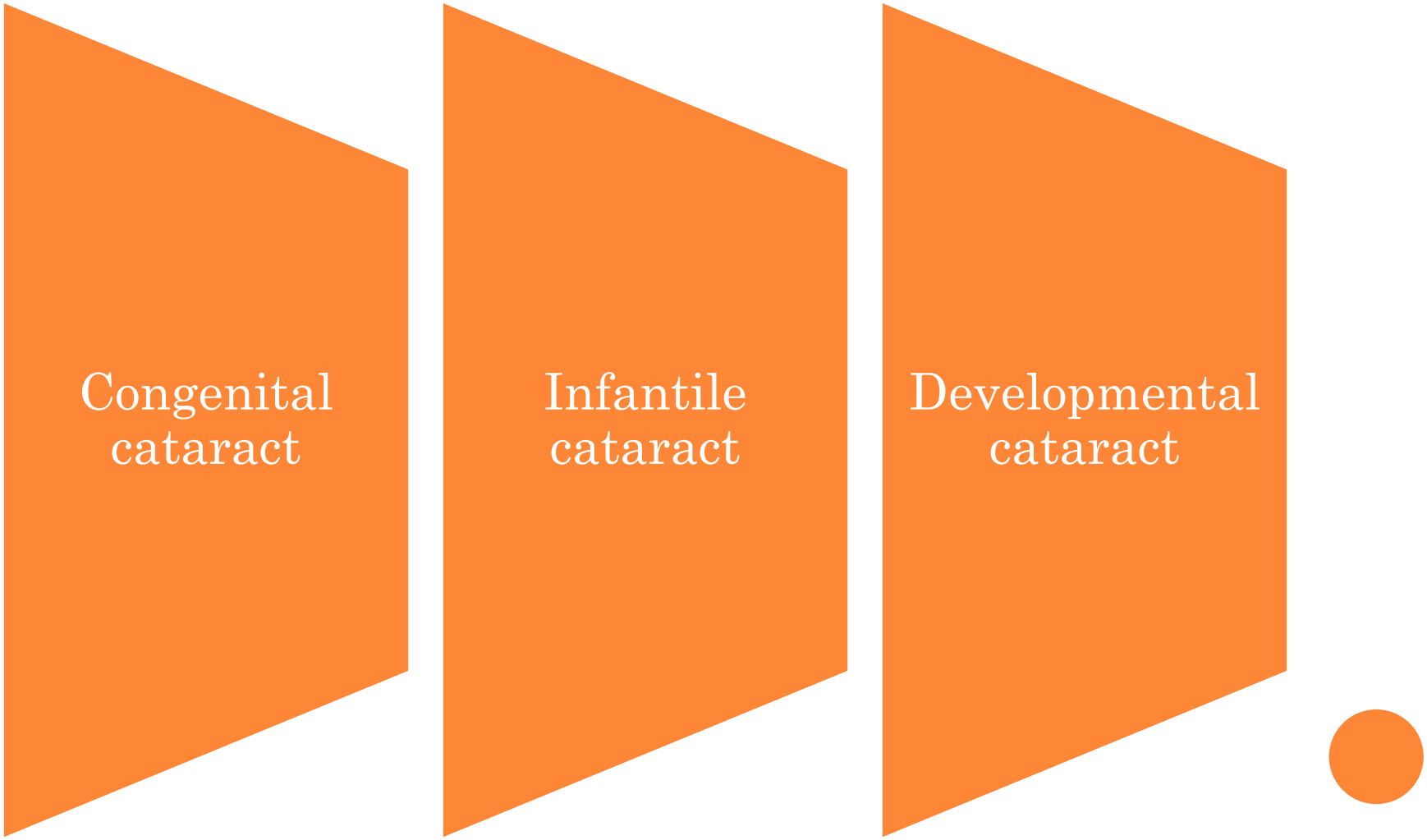
Next

Cataract Surgery Complications

CONGENITAL CATARACT

Dr. Irfan Ullah Khattak
Pediatric Ophthalmologist
HMC

Pediatric Cataracts



Definition

- Opacity of crystalline lens matter / capsule present at birth or shortly after birth
- Congenital cataract:- at birth
- Infantile cataract:- develop during 1st year of Life..
- Both term used synonymous

CONGENITAL CATARACT

A SERIOUS CONCERN

WHY

1. One of the leading cause of blindness worldwide.

2. Increased blind person years.

3. Socioeconomic Effects.

DEMOGRAPHICS

- One of the leading causes of childhood blindness.
- The incidence ranges from **1.8 to 3.6/10,000** per year
- Median prevalence is about **1.03/10,000** children **(0.32–22.9/10,000).** [Eye (London), 2016]
- The prevalence is higher in low-income economies compared to that of high income economies.

CONTD…….

- There is no difference in the prevalence based on gender or laterality.
- Approximately 1 to 15/10,000 children worldwide, accounting for 5%-20% of childhood blindness
- Globally, about 200,000 children are blind from bilateral cataracts

(Foster et al ; Medsinge et al, 2015)

CONTD…….

- About 20,000 to 40,000 new cases of bilateral congenital cataract are diagnosed each year

  (Apple et al, 2000)

- In India, approximately 10% of childhood blindness is caused by cataract.

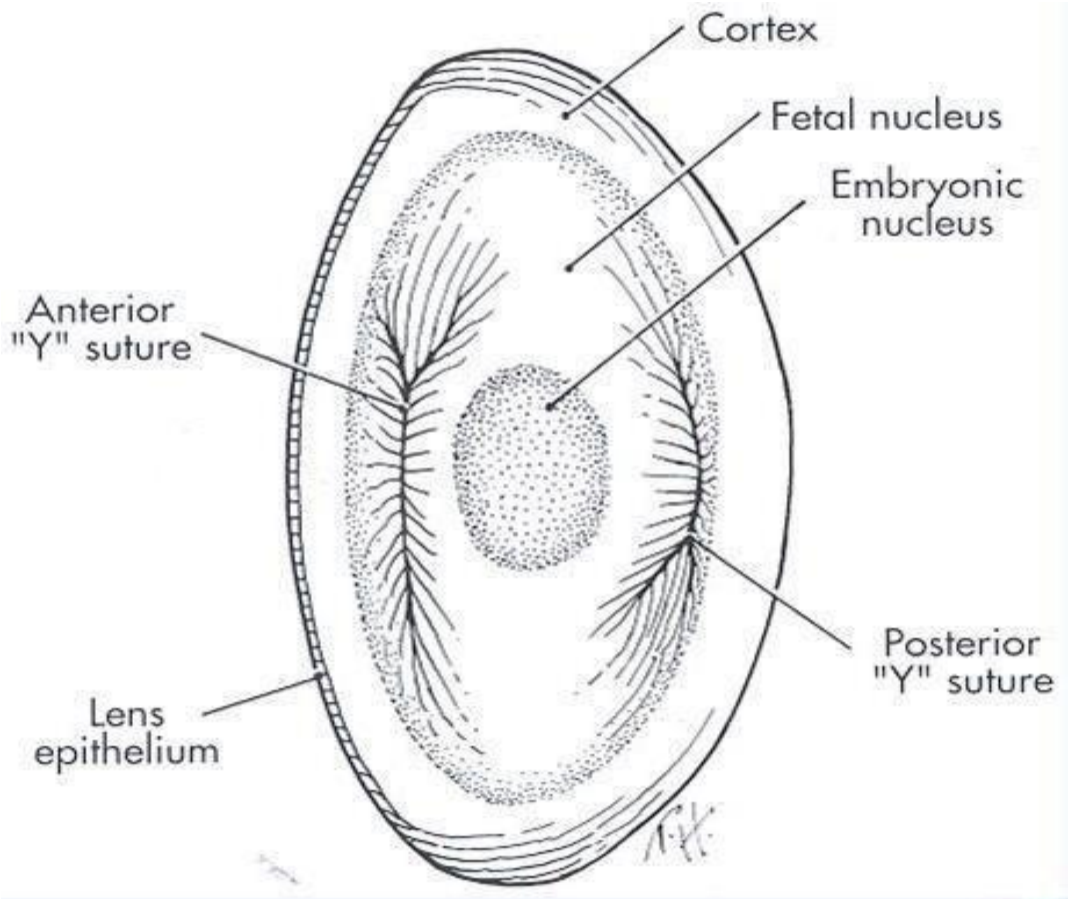
- Non traumatic cataracts(88.4%) and 11.6% were traumatic cataracts.
- (aao.org)

GLOBAL BLINDNESS

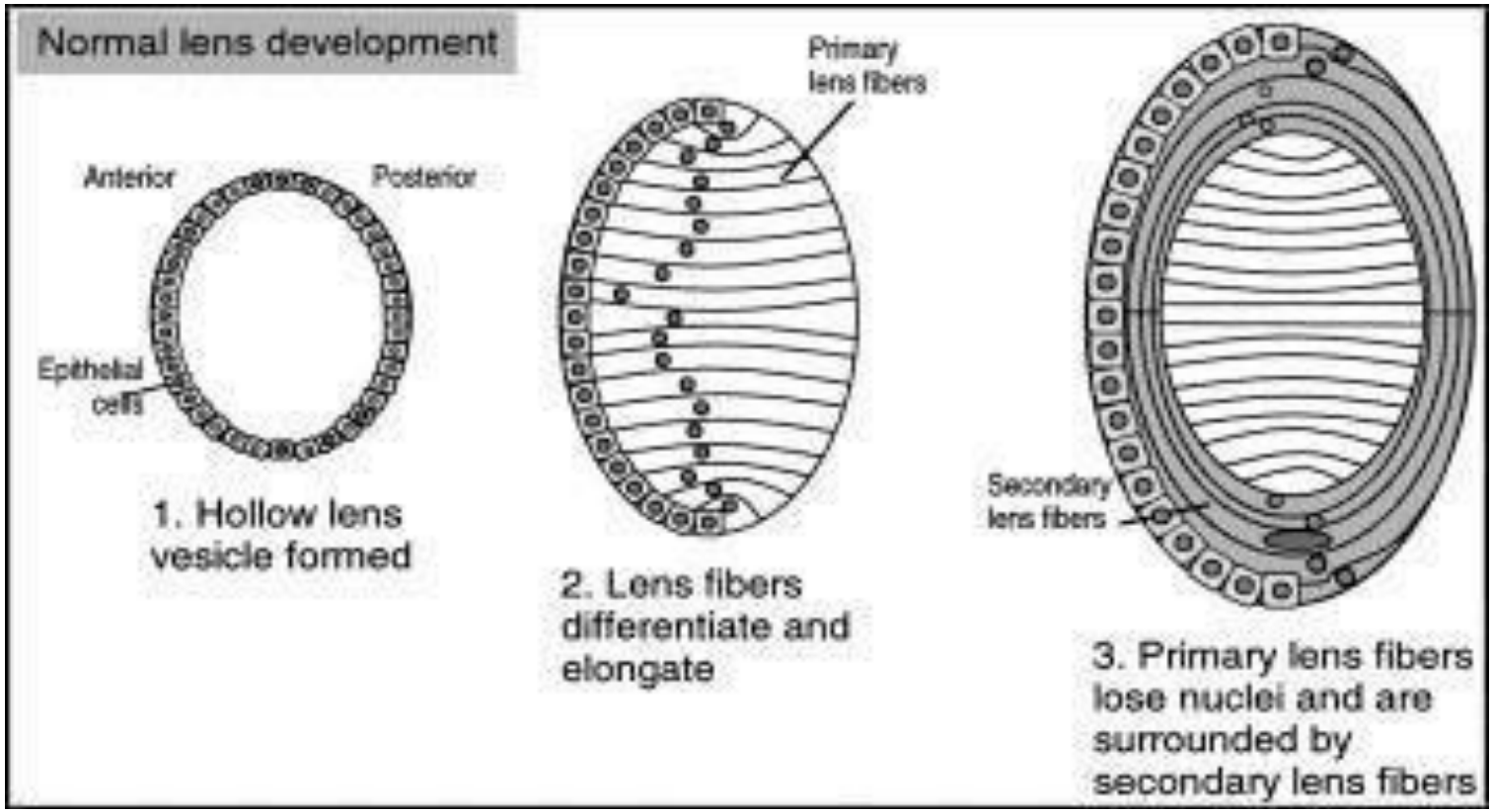
- Childhood blindness is only second to adult cataract as a cause of blind-person-years.

- Approximately 70 million blind-person years are caused by childhood blindness of which about ***10 million blind-person years (14%) is due to childhood cataract.*** (Community Eye Health Volume 17)

Cross section of juvenile lens



EMBRYOLOGY OF LENS



EMBRYOLOGY OF LENS

- **Embryonic nucleus** develop – 6week of gestation
- Arises from primitive post lens epithelium
- **Fetal nucleus-next** to develop from lens fibers from equatorial epithelial cells
- They stretch ant & post to around embryonic nucleus
- At birth both form most of lens fibers..
- Cortical Lens fiber Mostly develop postnatal

FETAL NUCLEUS

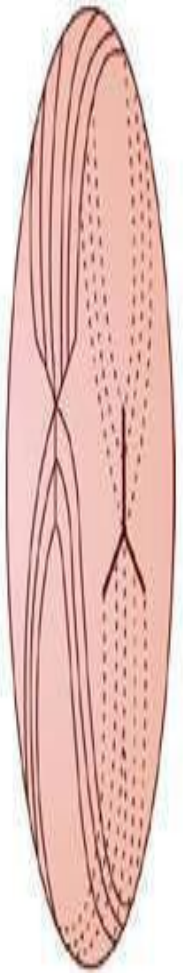
Y SUTURE is important Landmark

Fetal nucleus

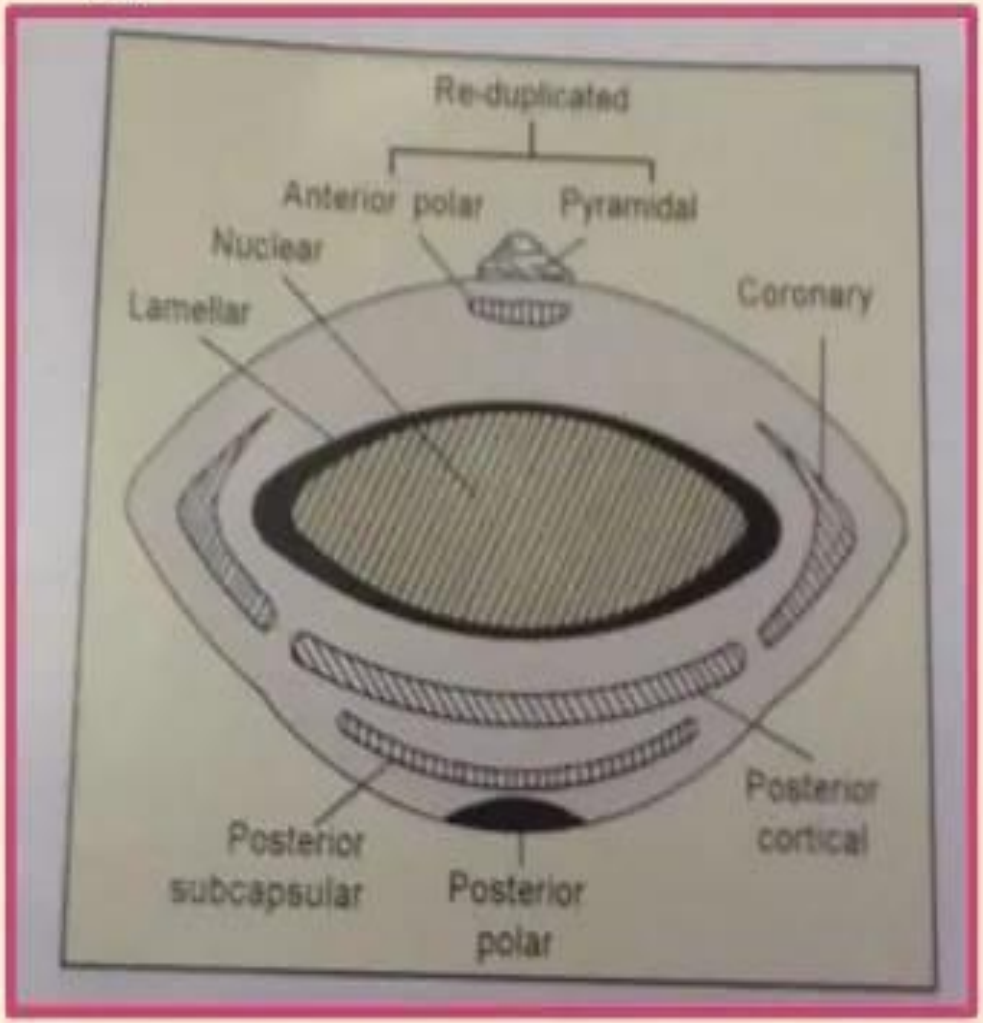
Peripheral to Y suture cortex

Within and Y SUTURE include NUCLEUS

Anterior upright Y WHILE Posterior it Inverted Y SUTURE Fibers



Morphology:



(Basak, SK., 2007)

Morphological classification

- **Anterior**:-
- Anterior polar
- Anterior pyramidal
- Anterior sub capsular cataract
- **POSTERIOR**:-
- Posterior polar cataract
- Posterior lenticonus
- Persistent fetal vasculature
- Posterior subcapsular cataract

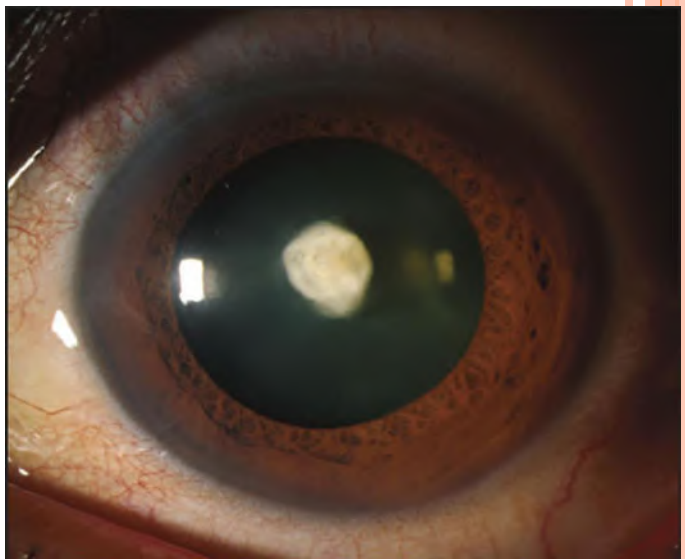
Morphological classification

- **CENTRAL** :-
- Lamellar cataract
- Sutural cataract
- Nuclear

- **DIFFUSE**:-
- Blue dot cataract(CERULEAN)
- Membranous cataract

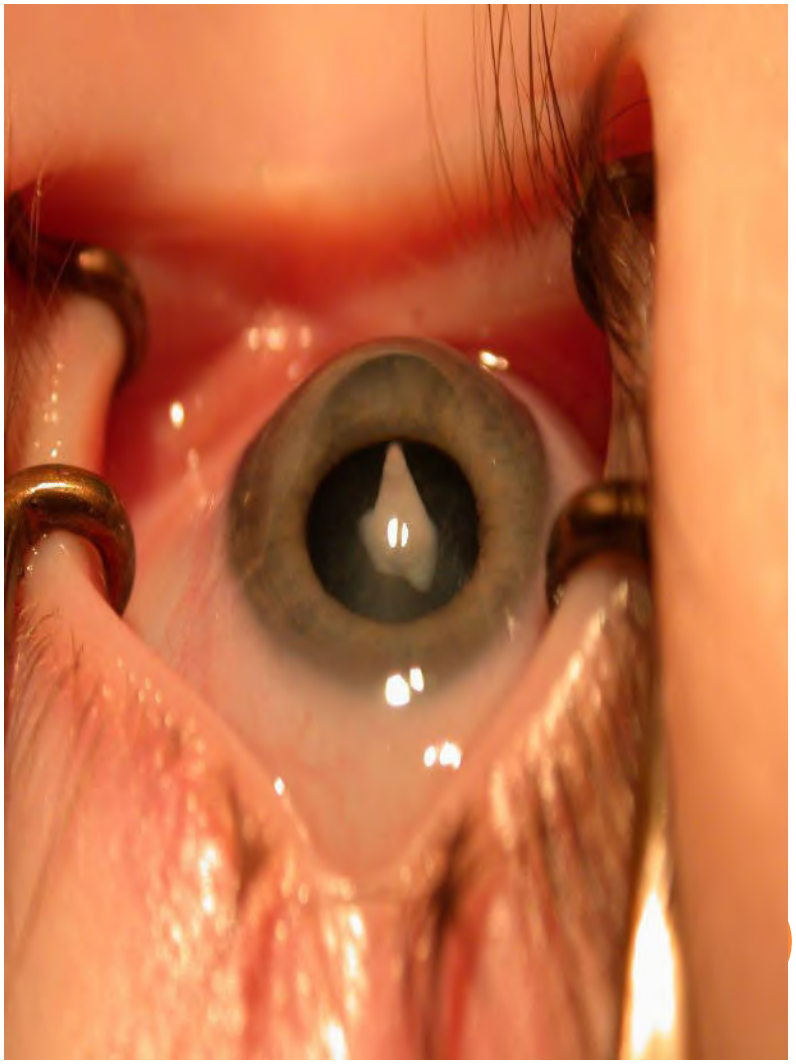
Anterior Polar Cataract

- White opacity Locate at center anterior Capsule
- Small 1-2mm.
- Derive from abnormal separation of lens vesicle from surface ectoderm.
- 1/3 bilateral
- 90% sporadic 10% AD



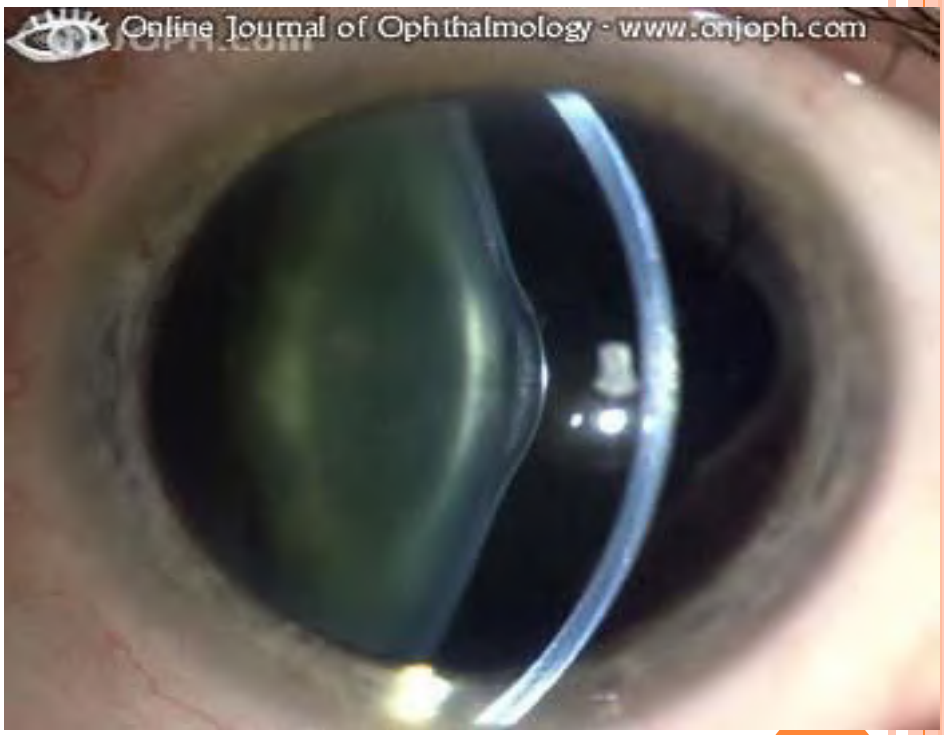
Anterior Pyramidal Cataract

- Bilateral . Mostly Sporadic
- Type of anterior polar cataract..
- Anterior capsular fibrosis
- Conical in shape Apex projected in A/C.
- 1-2mm cone opacity.



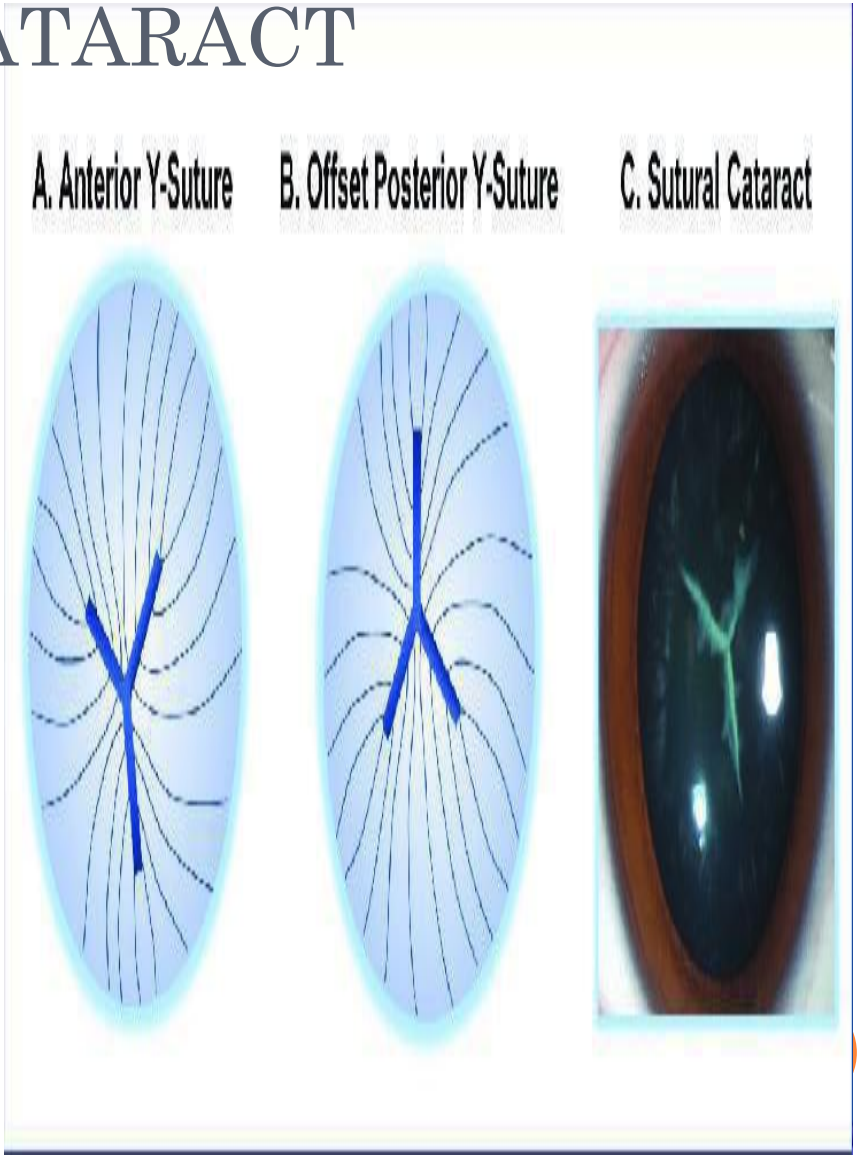
Anterior lenticonus

- This refers to a thinned-out central anterior capsule with or without anterior cortical opacities.
- Anterior lenticonus is said to be characteristic of Alports syndrome. Spontaneous rupture of the lens can occur, resulting in a hydrated Total cataract



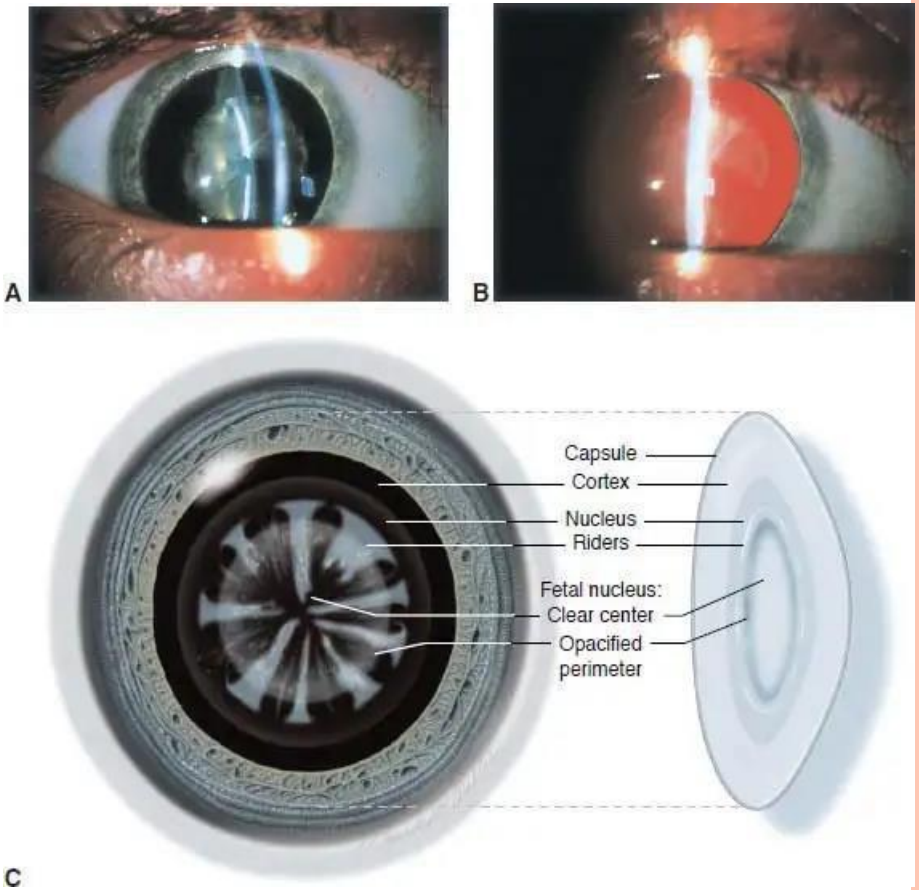
SUTURAL CATARACT

- AD
- Type of cong nuclear cataract with opacity Along Y suture in fetal nucleus.
- Progressive
- Expand into cortex and embryonic nucleus



LAMELLAR CATARACT (ZONULAR)

- Most common type
- Mostly Bilateral
- Opacification of specific zone/layer
- Layer of Opacification involving fetal nucleus surrounding clear center and surrounde in turn by layer of clear cortex

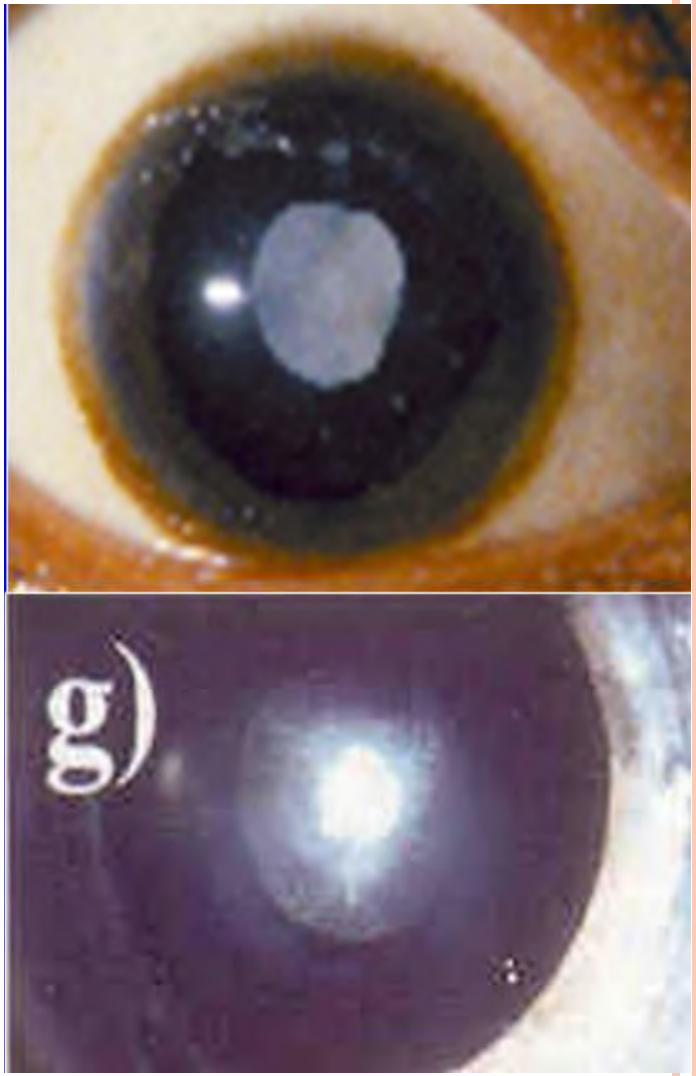


LAMELLAR CATARACT

- RIDER OPACITIES:-
  Arcuate opacity
  straddle the equator…
- Spoke of wheel

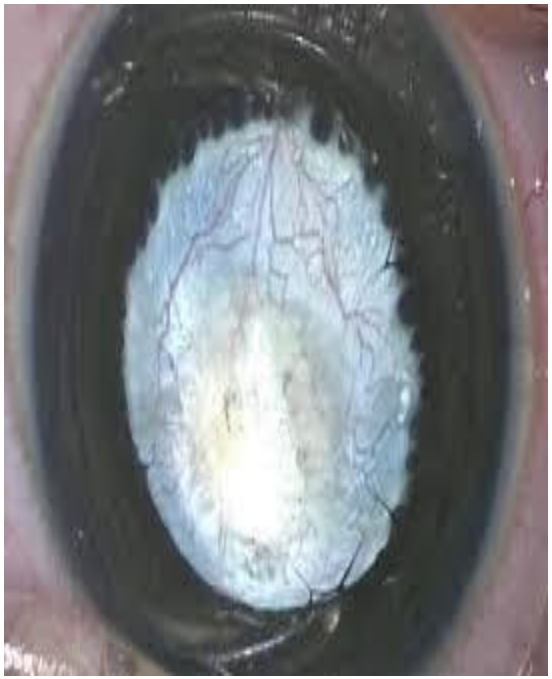
Nuclear cataract

- Opacity within embryonic nucleus or fetal nucleus..
- Mostly bilateral with AD
- Non progressive
- Congenital onset
- Common presentation intrauterine infections specially RUBELLA cataract



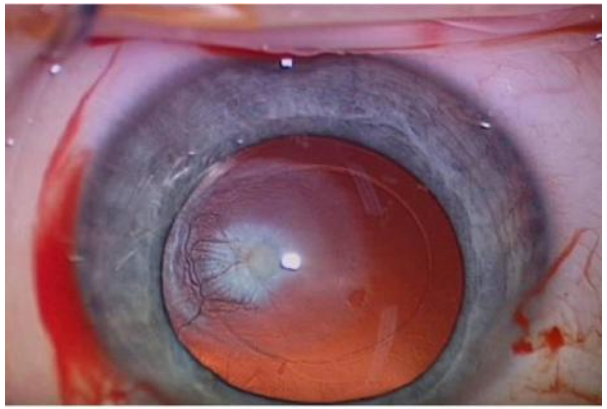
PFV
PERSISTENT FETAL VASCULATURE

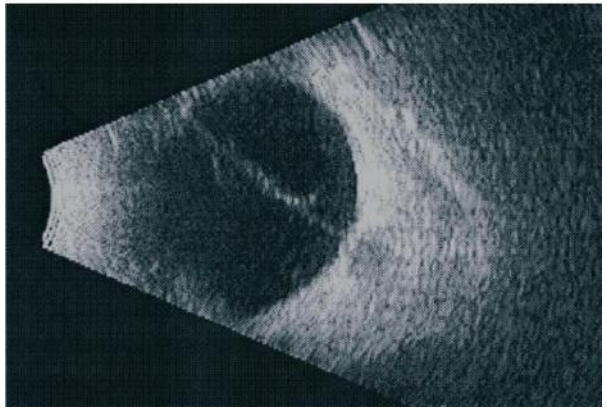
- Previously (Persistent hyperplastic primary vitreous)
- MOST COMMON CAUSE OF UNILATERL CATARACT
- Isolated, sporadic
- Progressive. Anterior chamber shallowing causing Secondary glaucoma



PFV

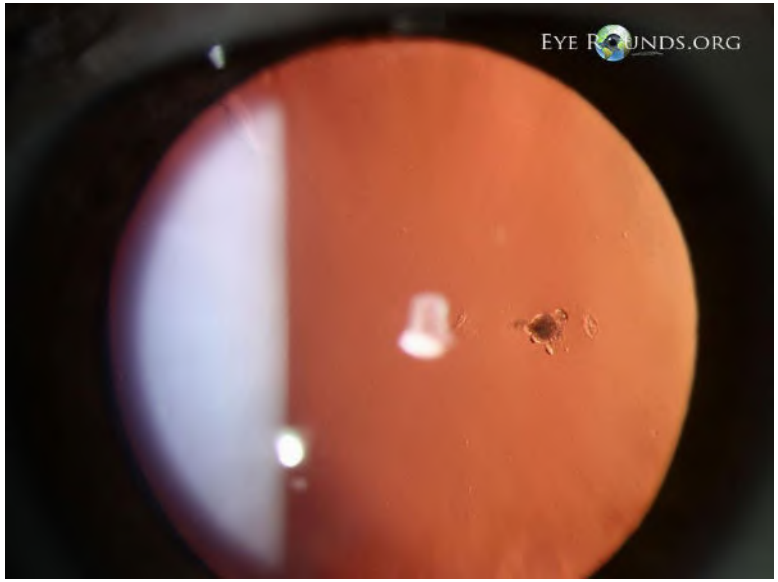
- The lens opacities in patients with PFV are generally capsular and can be associated with *shrinkage, thickening, and vascularization of the capsule*.
- There may be a posterior plaque outside or involving the lens capsule with a clear lens that must be Treated as a cataract





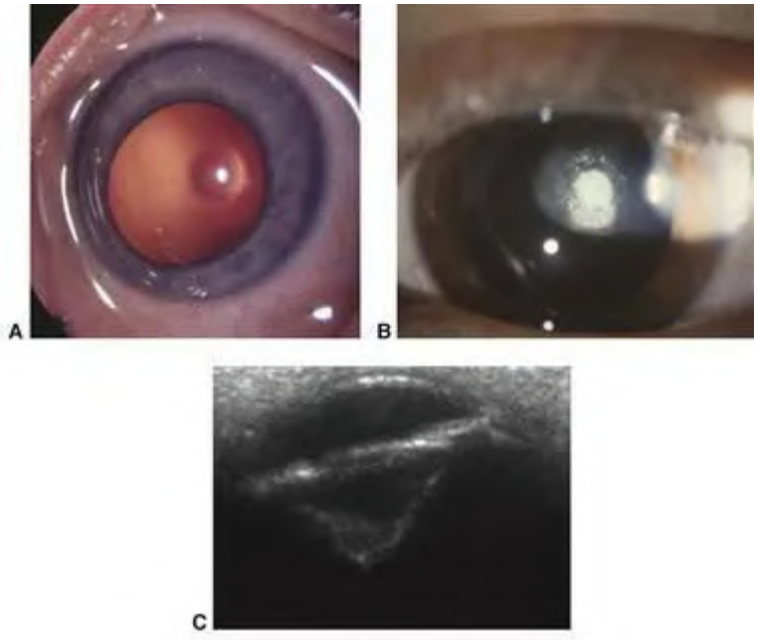
Mittendorf Dot

- Small, circular opacity on the posterior lens capsule, classically nasal in location
- Represents the anterior attachment of the hyaloid artery.
- Mildest form of PFV.
- NON Progressive, do not interfere vision



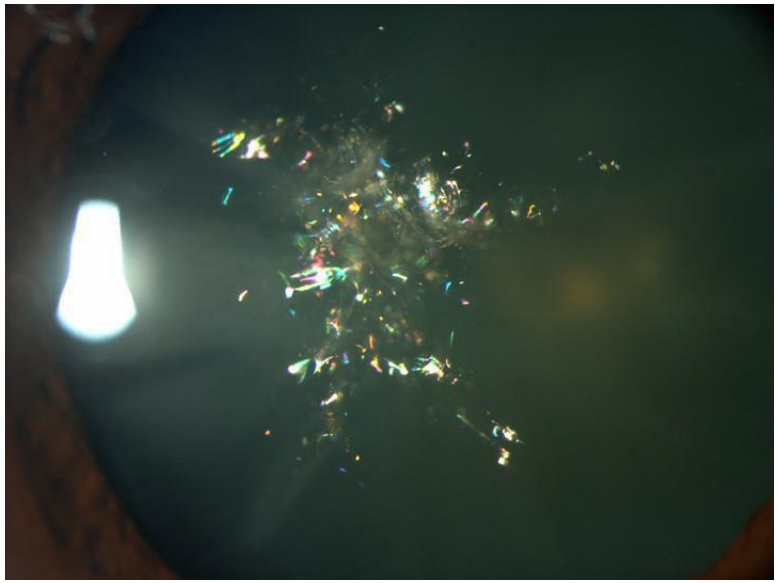
Posterior Lenticonus

- Mostly uniLateral
- Posterior capsule is thin and bulges posteriorly..
- This usually occurs at the location where the hyaloid system attaches to the eye.
- The distortion can cause a localized area of myopic refraction
- May or may not be subcapsular cortical opacification



Christmas tree cataract

- Multiple.. small flecks in cortex
- Cataract with polychromatic luster..
- Appearance of various colors
- Associated with myotonic dystropy, hypoparathyriodism

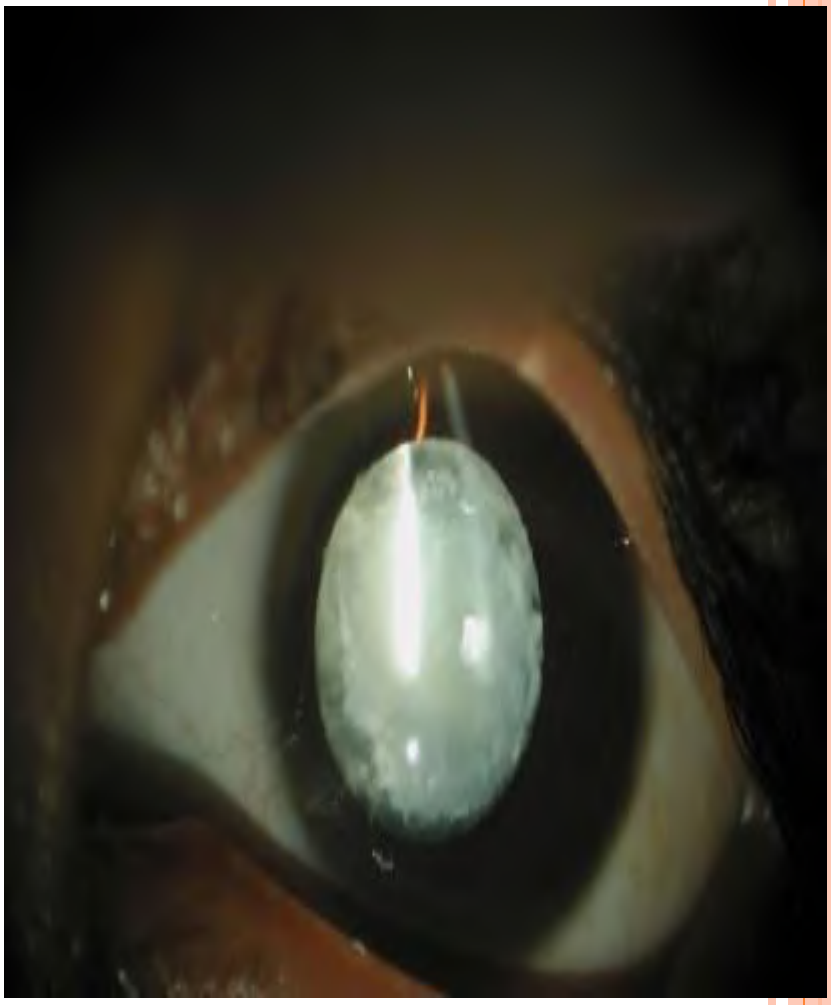


OIL DROP CATARACT

- Commonly seen in patients with galactosemia
- Bilateral
- Central aspect of Posterior lens cortex opacity with "oil droplet" appearance on retro illumination
- Restrict galactose from the diet will reverse cataract

Membranous cataract

- End stage cataract
- Lens matter absorb with ant & post capsule apposition
- TORCHS infection
- PHPV Cataract
- Long standing congenital cataract



Cerulean cataract

- Bilateral ..slowly progressing
- Scattered bluish-white opacities in cortex
- AD ..also in down syndrome
- Do not require cataract surgery

Etiology:

BILATERAL

- Idiopathic (60%)
- Hereditary (30%)
- Intrauterine infection
- Associated with ocular disorders
- Tumor
- Metabolic
- Maternal drug ingestion/ malnutrition
- Trauma

UNILATERAL

- Idiopathic (80%)
- Intrauterine infection
- Ocular abnormalities (10%)
- Trauma (9%)

(Friedman, NJ & Kaiser, PK, 2009)

Hereditary Cataract(30%)

- AD inheritance pattern is most common cause of bilateral congenital cataract
- 25% of these case represent a new AD mutation.
- AR is uncommon often associated with Consanguinity
- X-Linked cataracts are rare

Maternal Infections (3%) (TORCHS)

- Mostly Bilateral
- Dense nuclear / membranous cataracts.
- Among TORCHS RUBELLA is commonly associated with congenital cataract

TORCHS Infection

CASE STUDY
INTRO
TOXOPLASMO.
SYPHILIS
PARVO. B19
VZV
LISTERIA
RUBELLA
CMV
HSV-2
REVIEW
SUMMARY

TORCH

HIGH YIELD

* TOXOPLASMA GONDII
* OTHER AGENTS:
  - ~ SYPHILIS
  - ~ PARVOVIRUS B19
  - ~ VARICELLA ZOSTER VIRUS
  - ~ LISTERIA
* RUBELLA
* CYTOMEGALOVIRUS
* HERPES SIMPLEX VIRUS-2 (HSV-2)

HIGH YIELD

* VERTICALLY TRANSMITTED

A. via PLACENTA

B. via BLOOD, BODY FLUIDS or BREAST MILK

Rubella Cataract

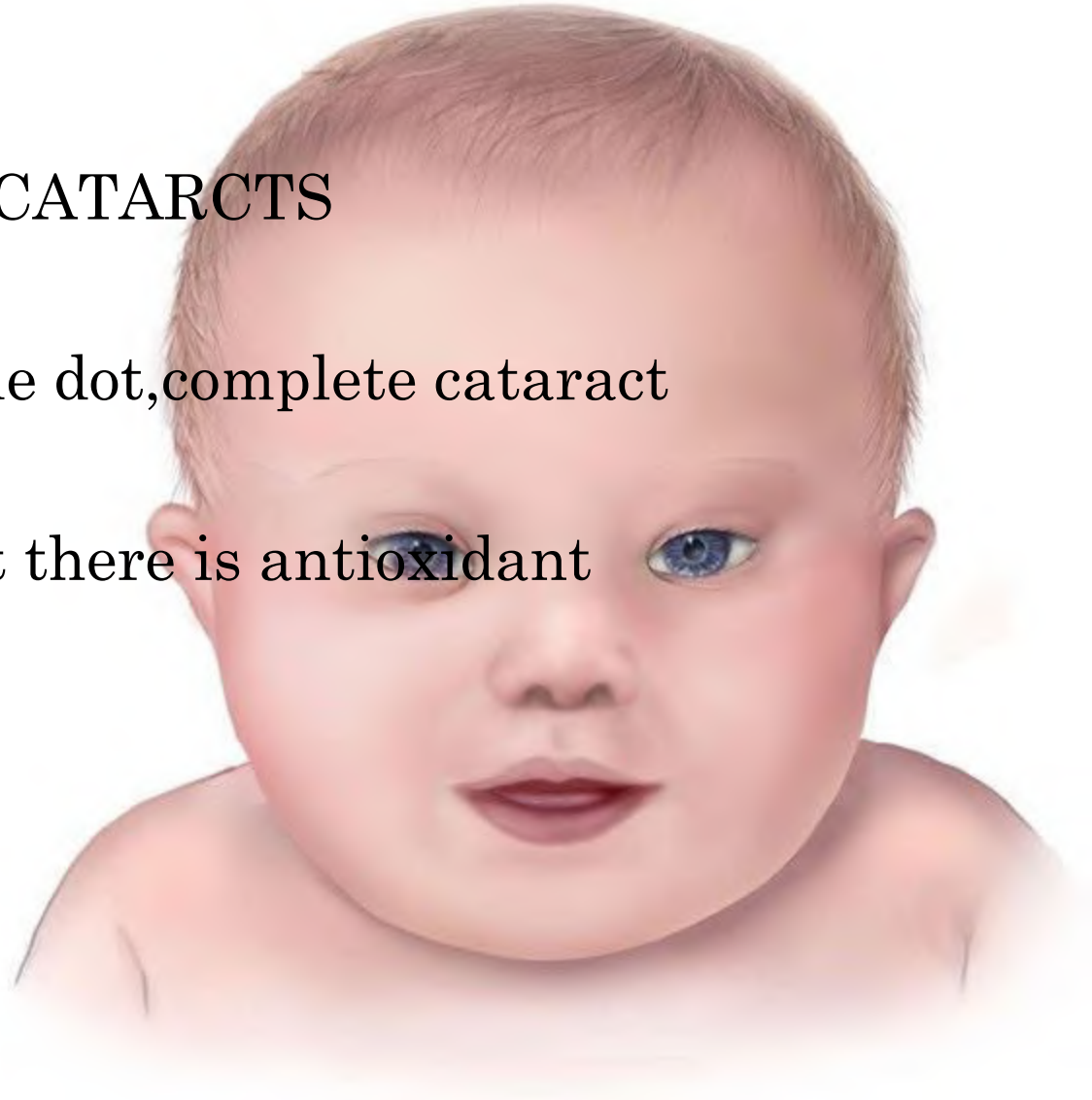
- Caused by invasion of lens by rubella virus
- 1st trimester of pregnancy
- **Ocular**:-Bil cataract, rarely unilateral
- Dense nuclear / membranous cataracts
- microphthamia, retinopathy, corneal Haze, glaucoma
- **CRS**:-Cong heart defect, hearing loss, mental retardation

Chromosomal Abnormality

- Downs syndrome (21)
- Edward syndrome (28)
- Patau syndrome (13)

DOWN'S SYNDROME

- CONG/INFANTILE CATARCTS
- Sutural ,Nuclear, blue dot,complete cataract
- In Trisomy21 patient there is antioxidant enzyme defect



METABOLIC DISEASES

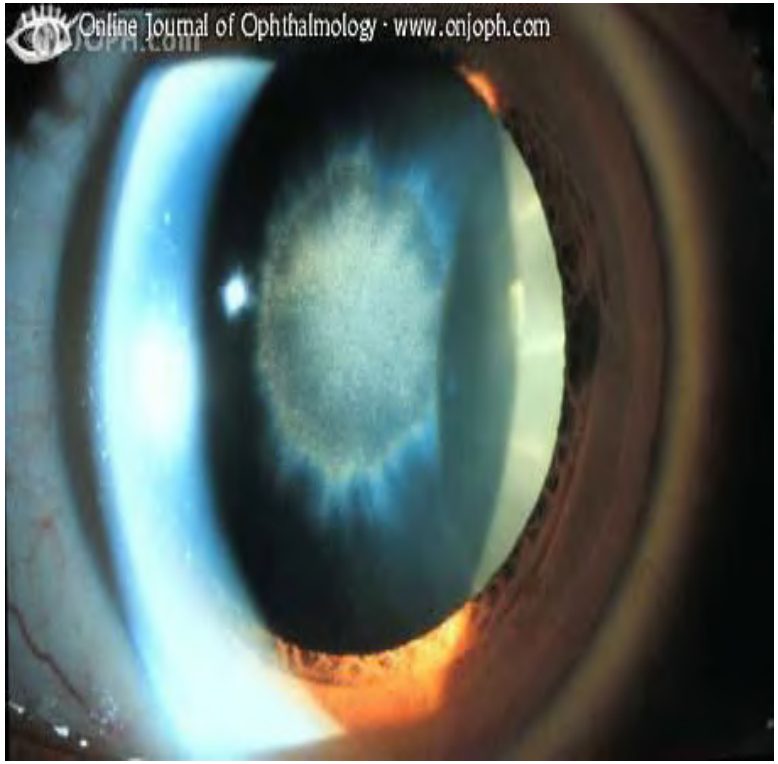
- **GALACTOSEMIA**:-
- AR.
- Oil drop cataract ,mental retardation,GI distrabance
- Galactose free milk
- **FABRY SYNDROME**:-
- X-LR
- Error of glycosphingolipid
- Posterior sub capsular/ anterior subcapsular

Metabolic diseases

- Alpha **Mannosidosis**:-
- AR
- Defective degradation of lipoproteins
- Mental redardation,coarse facies
- Cataracts
- **Refsum disease:-**
- AR
- Accumulation of phytanic acid
- RP with cataract

Wilson Disease

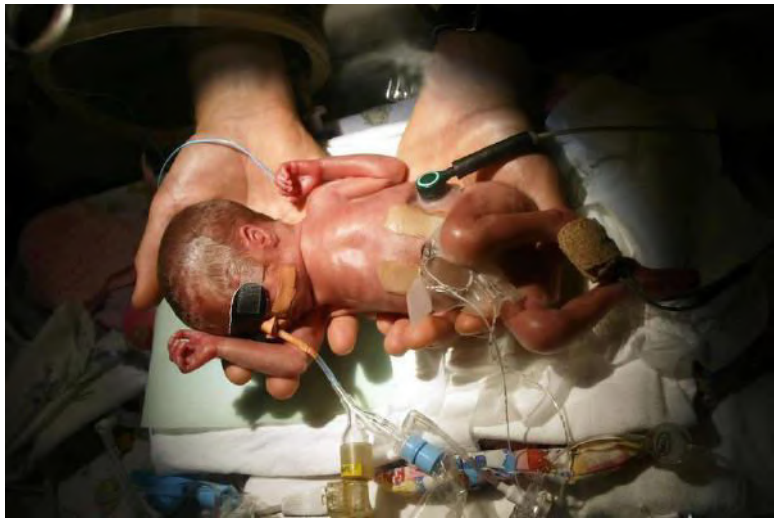
- AR.
- DEFECCTIVE Inborn error of Cu metabolism ..into ceruloplasim
- Cu accumulation at anterior capsule..
- Sunflower cataract resolve with Treatment (penicillamine)



Sunflower cataract of Anterior capsule

Cataract with prematurity

- Transient lens opacities noted in premature infants
- Bilateral
- Some cataracts appear after argon laser for ROP
- RESOLVE spontaneously after2-4 weeks



Renal diseases

Low syndrome

- X-LR
- Oculocerebrorenal
- Congenital catarcts with glaucoma

Alports syndrome

Unilateral cataracts

- Idiopathic (80%)
- Ocular abnormalities (10%)
- A.Post Lenticonus
- B.PHPV
- C.ANT.segment dysgensis
- Masked bilateral cataract(6%)
- Traumatic (4%)(must rule out child abuse)

ASSESSMENT

- History
- Observation of fixation and following reflexes
- Forced Choice Preferential Looking
- OKN
- VEP

NON SURGICAL

- Dilating Drops
  - Small cataract
  - Partial cataract
- Optical Correction
  - Lamellar cataract

Visual assessment

- Anterior capsule opacities are not visually significant
- Posterior capsule opacities are usually visually significant
- Small axial cataract often maintain good vision if pupil is dilated continuously

INDICATIONS FOR SURGERY

- When the visual defect in the child with congenital cataract is sever enough to interfere significantly with visual development
- Dense and total cataract
- Partial cataract

Preparation

- GA fitness
- Biometry
- B. Scan
- Blood Tests

- Informed consent
- Post op care
- Follow up

Timing of surgery

- Unilateral
  - 4-6 weeks
- Bilateral
  - 6-8 weeks

Surgical management

- LMA+ PC+AV± IOL

Complications of surgery

- Operative and immediate postoperative complications
  - Hyphaema
  - Posterior capsule rapture
  - Vitreous loss
  - Endothelial cell loss
  - Endophthalmitis

Complications of surgery

- Late postoperative complications
  - Posterior capsule opacification
  - Retinal detachment (1-10%)
  - Glaucoma (30%)
  - CMO
  - Vitreo-retinal haemorrhage
  - Corneal oedema

Optical correction

- The changing environment
- Spectacles
  - Can be worn at any age
  - Not unduly expensive
  - Can be readily changed
  - Safe
  - Make microphthalmic eyes appear normal
  - May be the form of optical correction available in a community

Optical correction (cont'd)

- Contact lenses
  - Well established method for unilateral aphakia
  - Silicon hydrogel lenses for extended wear
- Primary IOLs
- Secondary IOLs
- Epikeratophakia

Optical correction (cont'd)

- Primary IOLs
  - Calculation is difficult, may be done UGA
  - Full correction /under correction
  - Choose IOL acrylic hydrophobic, heparin coated
- Secondary IOLs

Amblyopia Therapy

- Occlusion
- Penalization

Thank you

Age related cataract

Dr Muhammad zia ud din Khalil

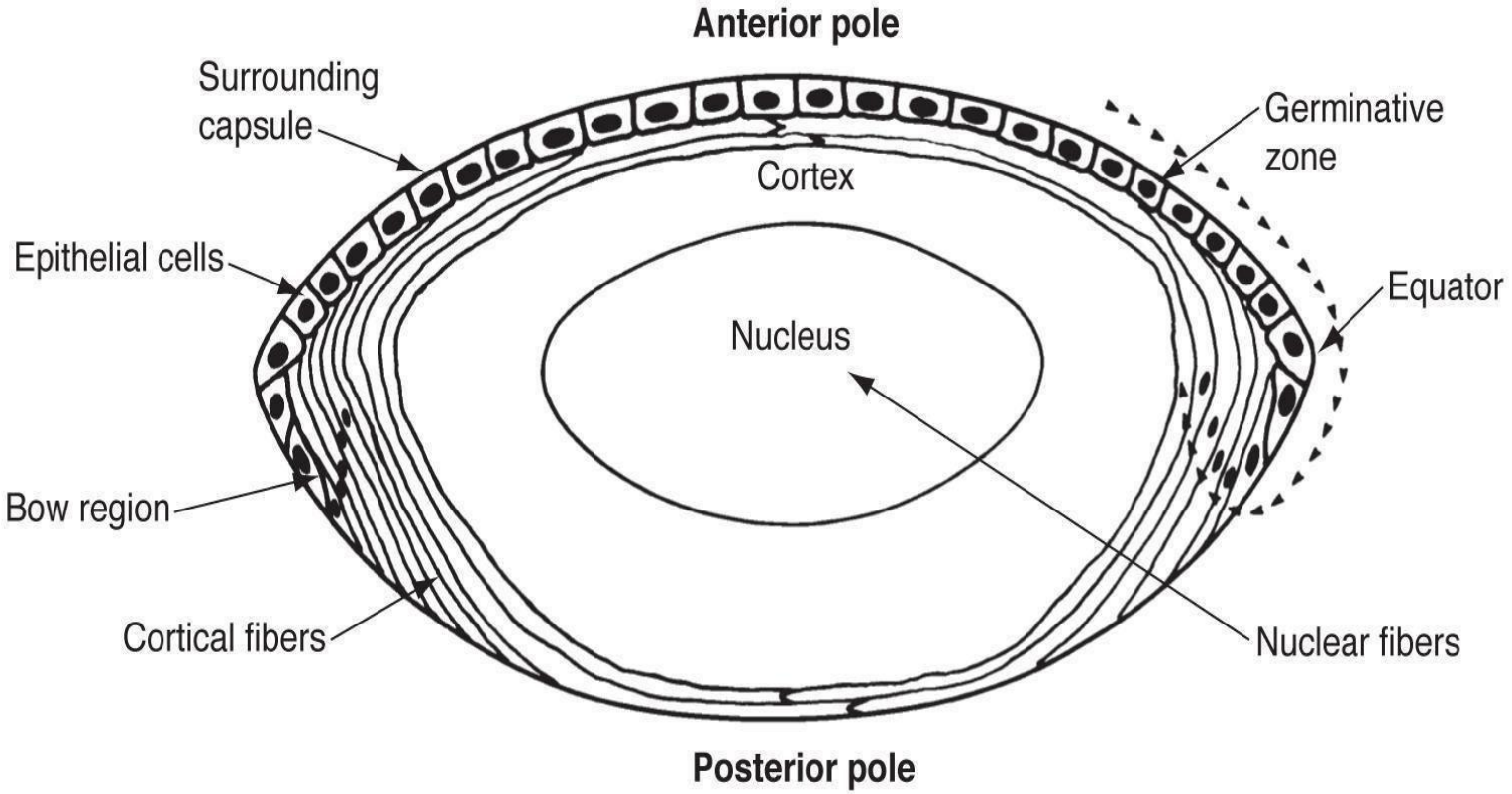
Assistant professor KGMC

FCPS(OPHTHALMLOGY),FCPS(VITREORETINA),FRCS(GLASGOW),
FICO(UK),FELLOWSHIP IN VITREO-RETINA(GERMANY),MRCS(EDINGBURGH)

Age related cataract

- Cataract is defined as opacification of the lens
- Age related cataract occurs in old age.

Anatomy of lens



Types of catract

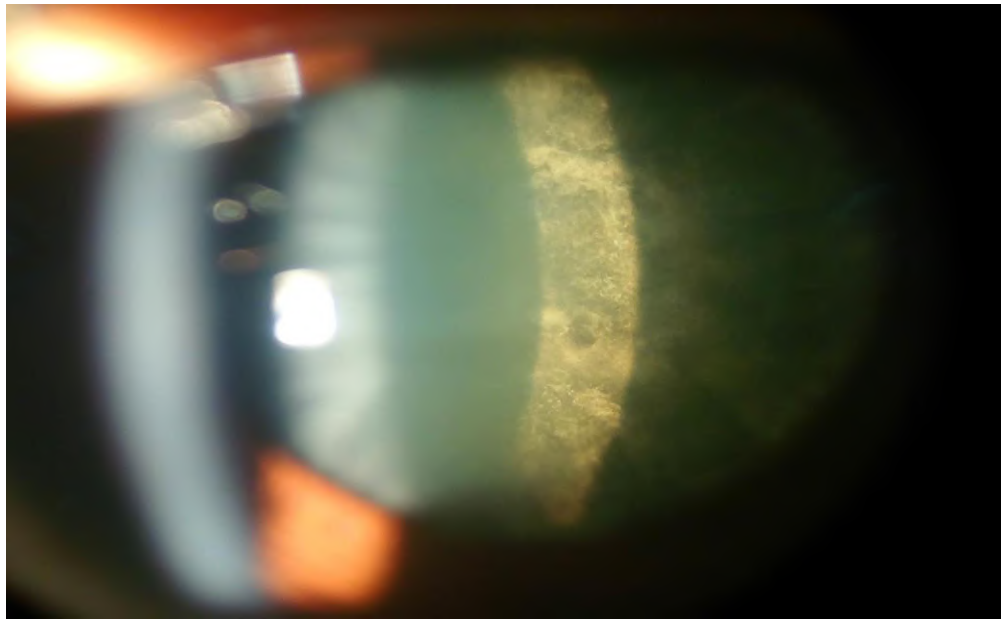
- morphological classification
- Classification according to maturity

Morphological classification

Morphological classification

- *Subcapsular cataract*
  - *Anterior subcapsular cataract*
  - *Posterior subcapsular cataract*
- *Nuclear cataract* involves the nucleus of lens.
  - Yellow to brown voloration
- *Cortical cataract*
  - wedge shaped or radial spoke-like opacities.
- *Polar cataract*

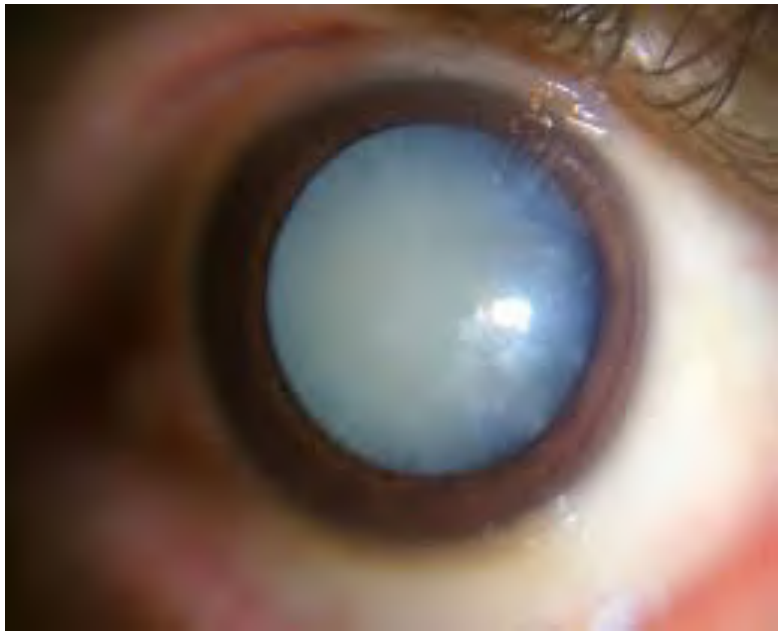
Types of cataract

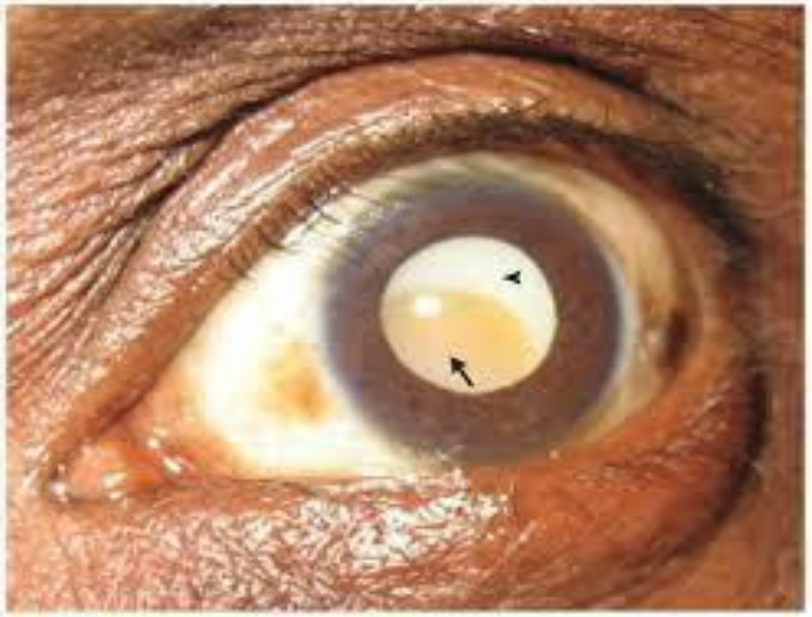


Classification according to maturity

- Early cataract
- Mature cataract
- Hypermature cataract
- Morgagnian cataract

Types of cataract





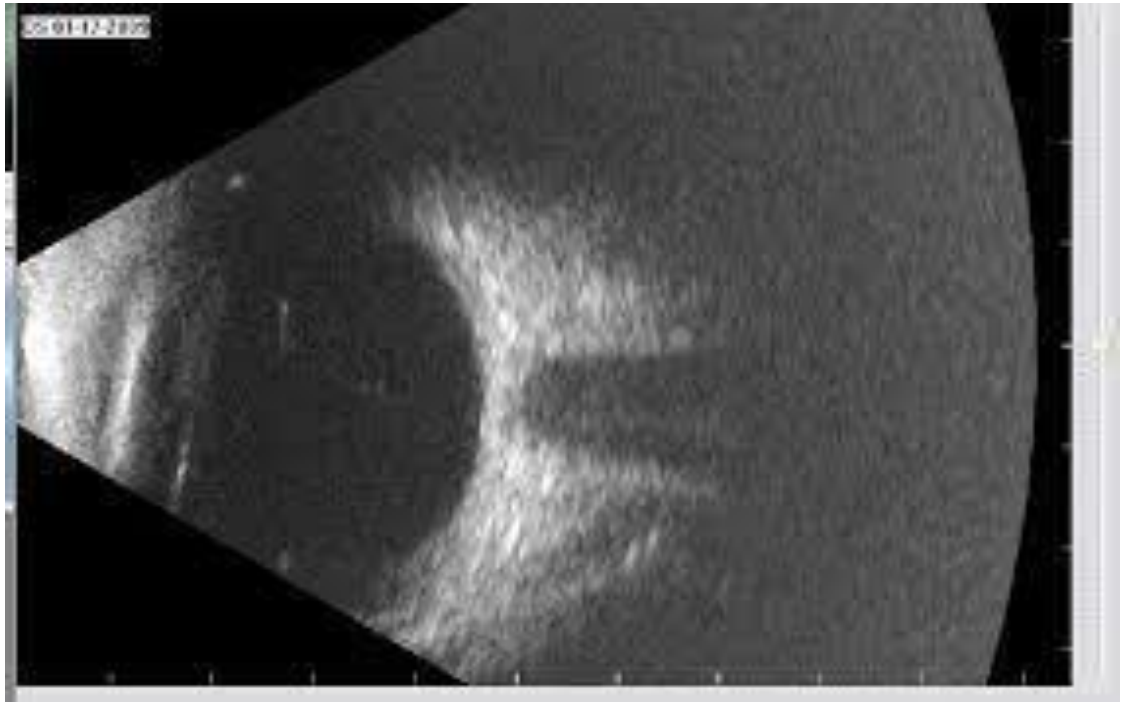
Symptoms of cataract

- Decreases vision
- Near vision changes
- Changes in vision according to light intensity

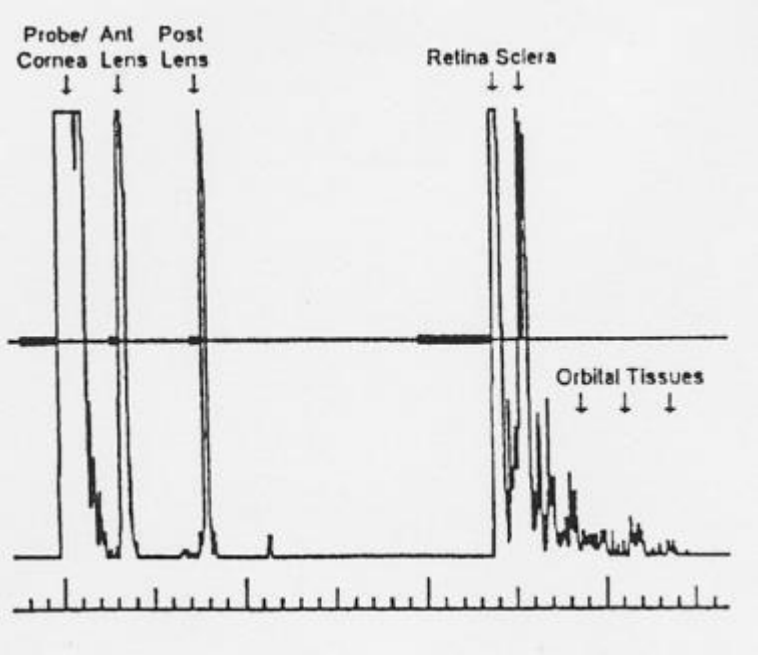
Management of cataract

- Investigations , viral profile, blood chemistry,
- Ocular investigations
- Biometry
- B-scan
- Cataract surgery

B-scan



Biometery



OD right

AL: 23.63 mm (SNR = 220.1)
K1: 42.45 D / 7.95 mm @ 12°
K2: 45.79 D / 7.37 mm @ 102°
R / SE: 7.66 mm / 44.12 D
Cyl.: 3.34 D @ 102°
ACD: 4.32 mm

Status: Phakic

SA60D3		Alcon ACIOL	
SF:	1.28	SF:	-0.31
IOL (D)	REF (D)	IOL (D)	REF (D)
20.5	-0.90	18.0	-1.06
20.0	-0.55	17.5	-0.65
19.5	-0.20	17.0	-0.26
19.0	**0.15**	**16.5**	**0.13**
18.5	0.49	16.0	0.52
18.0	0.83	15.5	0.90
17.5	1.17	15.0	1.28
Emme. IOL: 19.22		Emme. IOL: 16.67	

Alcon MA50 & MA60		Alcon SN60WF	
SF:	1.73	SF:	1.85
IOL (D)	REF (D)	IOL (D)	REF (D)
21.5	-0.99	22.0	-1.17
21.0	-0.65	21.5	-0.83
20.5	-0.31	21.0	-0.49
20.0	**0.03**	**20.5**	**-0.15**
19.5	0.36	20.0	0.18
19.0	0.69	19.5	0.51
18.5	1.01	19.0	0.83
Emme. IOL: 20.04		Emme. IOL: 20.27	

OS left

AL: 23.67 mm (SNR = 59.1)
K1: 42.94 D / 7.86 mm @ 172°
K2: 46.04 D / 7.33 mm @ 82°
R / SE: 7.60 mm / 44.49 D
Cyl.: 3.10 D @ 82°
ACD: 4.43 mm

Status: Phakic

SA60D3		Alcon ACIOL	
SF:	1.28	SF:	-0.31
IOL (D)	REF (D)	IOL (D)	REF (D)
20.0	-0.95	17.5	-1.06
19.5	-0.60	17.0	-0.66
19.0	-0.25	16.5	-0.27
18.5	**0.10**	**16.0**	**0.12**
18.0	0.44	15.5	0.51
17.5	0.77	15.0	0.89
17.0	1.11	14.5	1.26
Emme. IOL: 18.64		Emme. IOL: 16.16	

Alcon MA50 & MA60		Alcon SN60WF	
SF:	1.73	SF:	1.85
IOL (D)	REF (D)	IOL (D)	REF (D)
21.0	-1.05	21.0	-0.89
20.5	-0.71	20.5	-0.55
20.0	-0.37	20.0	-0.22
19.5	**-0.04**	**19.5**	**0.11**
19.0	0.29	19.0	0.44
18.5	0.62	18.5	0.76
18.0	0.94	18.0	1.08
Emme. IOL: 19.45		Emme. IOL: 19.67	

Complications of cataract

- Glaucoma

I. Phacomorphic glaucoma
II. Phacolytic glaucoma
III. Phacoantigenic

- Subluxation
- Luxation

DIABETIC RETINOPATHY

SANAULLAH JAN

FRCS (Glasgow), FRCS (Edinburgh), FCPS (Pak), Fellowship in Vitreo-retina (Germany, India)

Introduction

- Diabetes mellitus ---------- group of metabolic diseases.

- Affect various organs of the body including eye.

National / Local data

Community Based Survey: HbA1c (screening test)

18856 Participants (Aged 20 yr or more), Pakistan (16 Districts)

- **Diabetics Type II Diabetes "n = 3201"** **16.98%** (95% CI 16.44-17.51)
- **Pre-diabetics** **"n = 2057"** **10.91%** (95% CI 10.46 - 11.36)

Diabetic Population **19 million** **Pakistan** **(IDF)**

Aamir AH et al. Diabetes prevalence survey of Pakistan (DPS-PAK).BMJ Open 2019

Diabetes in Pakistan

- 537 million (53.7 crore) diabetics (world) **IDF 2021**
  By 2045 this will rise to 135.7 million

- Total adult population Pakistan (123,526,400/12.356 crore)

- Prevalence in adults 26.7 %
- Total cases in adults 32,964,500 / 3.296 crore

DIABETIC RETINOPATHY

- DIABETIS MELLITUS
- COMPLICATIONS

  RETINOPATHY

  NEPHROPATHY

  VASCULOPATHY

  PERIPHERAL NEUROPATHY

  DIABETIC FOOT

DIABETIC OCULAR MANIFESTATIONS

- LIDS & ADNEXA
- OCULAR SURFACE & CORNEA
- UVEA (Iris, CB, Choroid)
- Pupil
- LENS & GLAUCOMA
- VITREOUS, RETINA
- ORBIT & OPTIC NERVE
- CRANIAL NERVES & OCULAR MUSCLES

National / Local data:

Hospital based studies

- **DR in diabetics (NIDDM) Eye OPD (LRH) 38.4 % Yr 2003**

- **Retinal Digital Imaging Type II DM 38.34% Yr 2012 (DR) 2123 patients Endocrinology Unit/HMC**

- **DR in diabetics Retina Clinic 68.61% Yr 2018**

- **Amer AH & Sanaullah jan**. Frequency of DR in a tertiary care hospital using Digital Retinal Imaging Technology. **JPGMI 2012**; 26(1): 29-33.
- Khan MN, Naseem A, **Sanaullah jan et al.** Presentation of Diabetic Retinopathy. **JPGMI, 2003** Vol 17 (1): 26—31.
- **Sanaullah Jan et al.** Status of DR & its presentation patterns in diabetics at ophthalmology clinics. **JPGMI 2018**; 32(1):24-26.

Diabetic Retinopathy: Challenge

Remember

One third

Diabetic Population -- Diabetic Retinopathy -- Vision-threatening DR

Recent studies suggests DR progression and vision loss is lower in the modern era due to improvements in systemic control and treatment advances

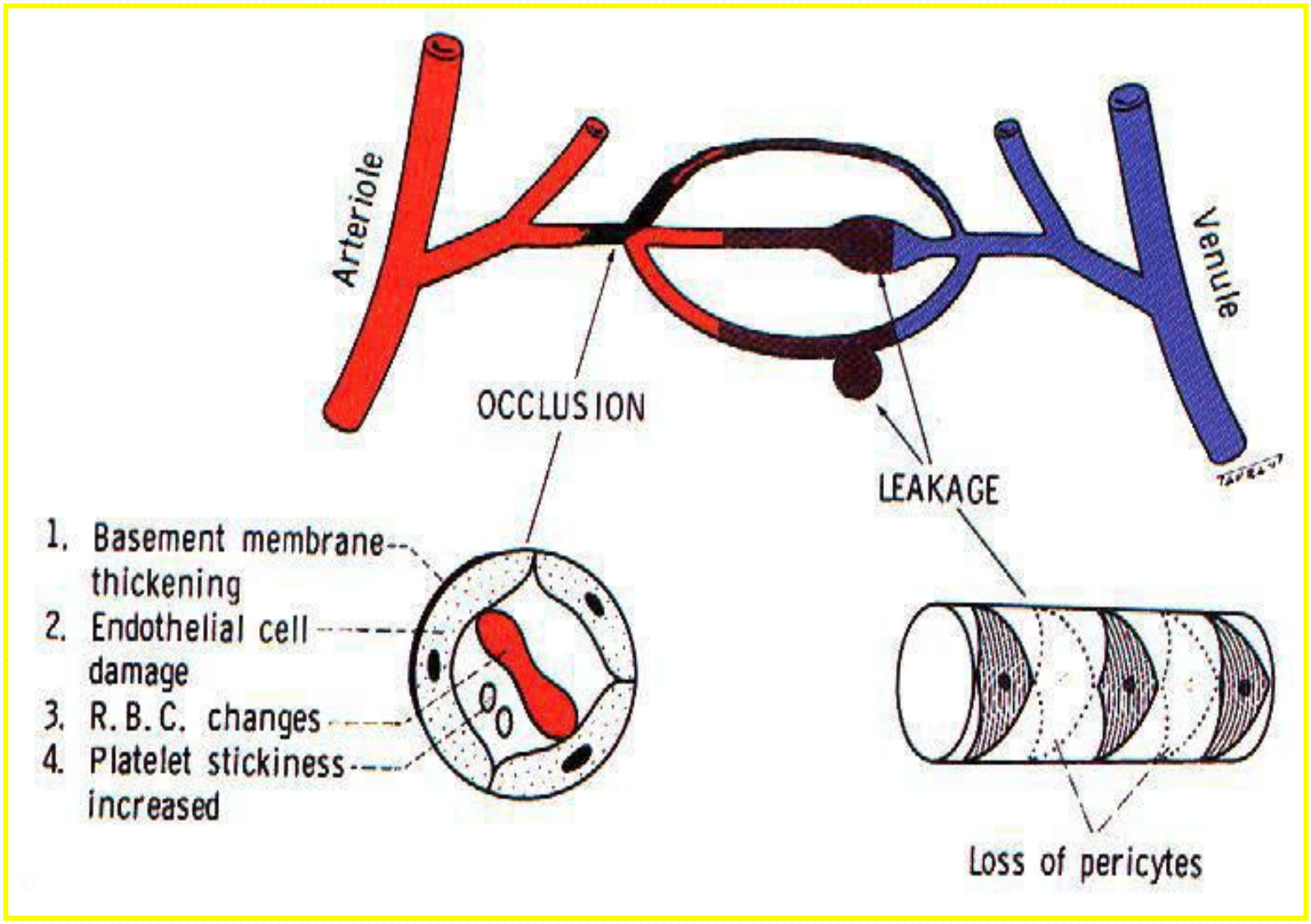
Introduction

- Diabetic retinopathy------ leading cause of preventable blindness
- Early detection and treatment---- prevent visual loss and blindness.
- One third of diabetic people :
  - Never had any ophthalmoscopic examination and that more than half of these individuals have eye disease.

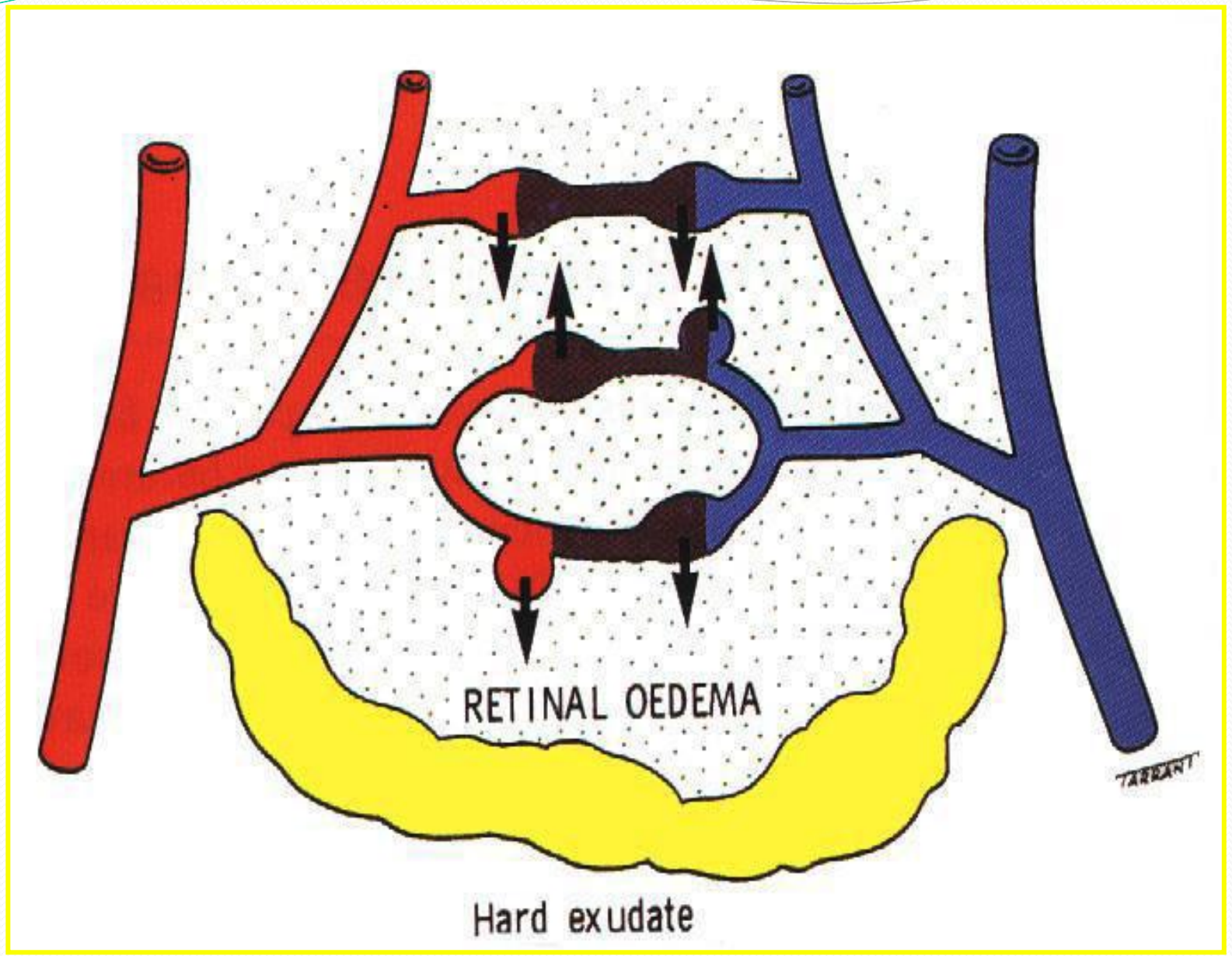
IDENTIFIED RISK FACTORS FOR DR

- Hyperglycemia
- Type of DM
- Duration of DM
- High blood pressure
- Hypercholesterolemia
- Nephropathy
- Anemia
- Pregnancy
- Puberty
- Cataract surgery
- Smoking

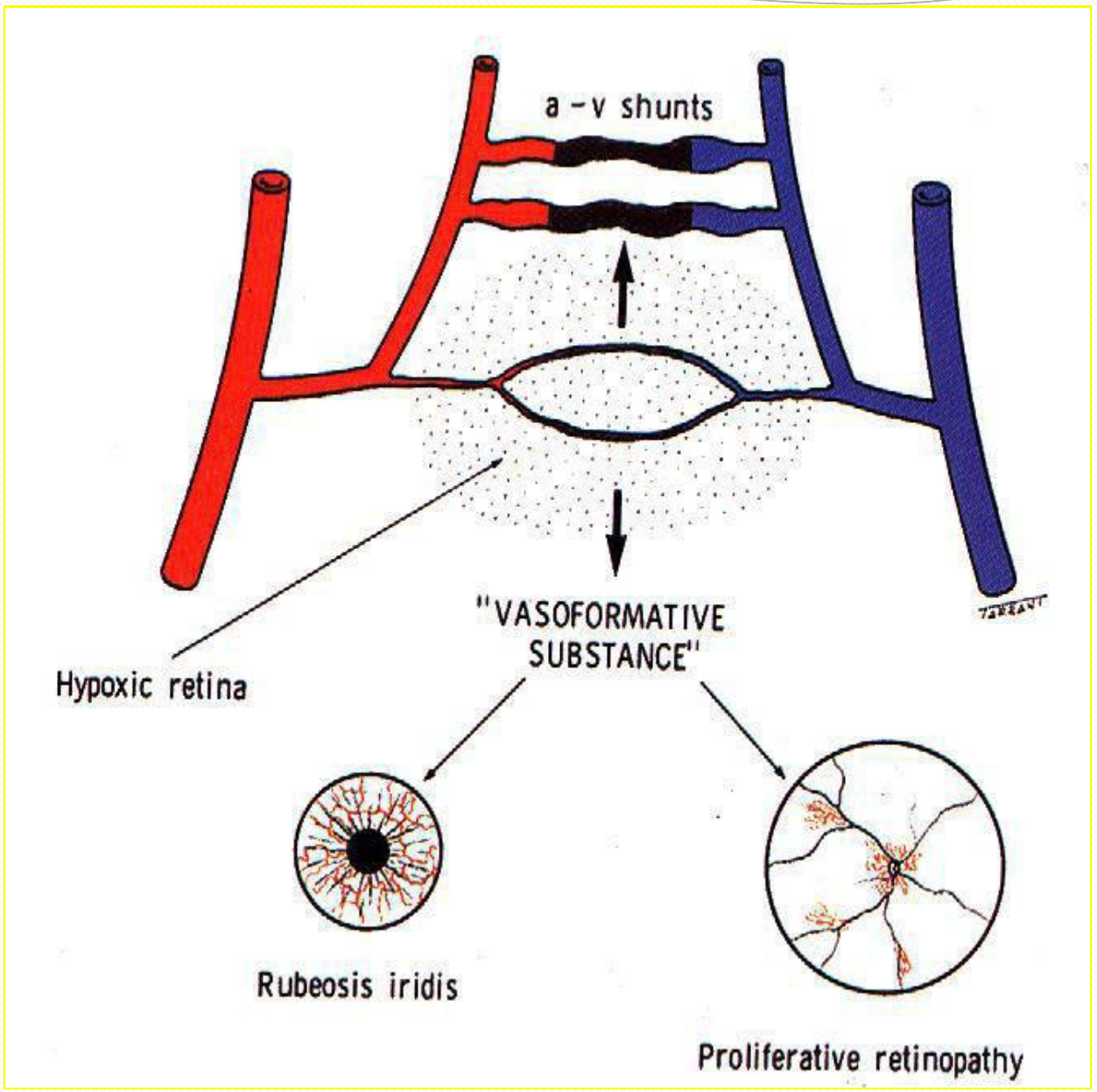
DIABETIC RETINOPATHY: Pathogenesis



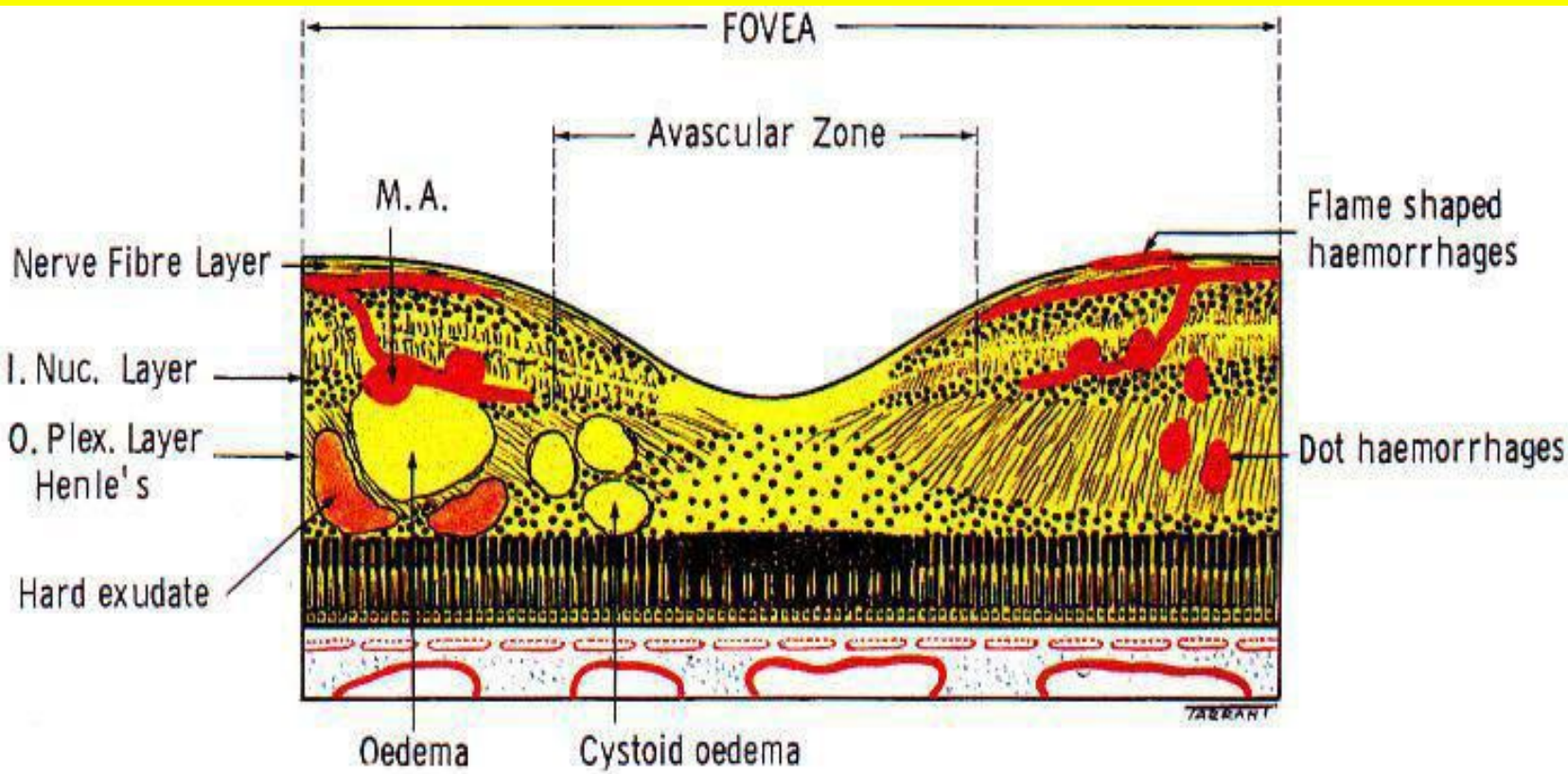
Consequences of chronic leakage



RETINAL ISCHEMIA



Location of lesions in Non-Proliferative diabetic retinopathy



Non-proliferative diabetic retinopathy (NPDR)

No DR

Very mild NPDR

Microaneurysms only

Mild NPDR

Any or all of: microaneurysms, retinal haemorrhages, exudates, cotton wool spots, up to the level of moderate NPDR. No intraretinal microvascular anomalies (IRMA) or significant beading

Moderate NPDR

- Severe retinal haemorrhages (more than ETDRS standard photograph 2A: about 20 medium–large per quadrant) in 1–3 quadrants or mild IRMA
- Significant venous beading can be present in no more than 1 quadrant
- Cotton wool spots commonly present

Severe NPDR

The 4–2–1 rule; one or more of:

- Severe haemorrhages in all 4 quadrants
- Significant venous beading in 2 or more quadrants
- Moderate IRMA in 1 or more quadrants

Very severe NPDR

Two or more of the criteria for severe NPDR

Proliferative diabetic retinopathy (PDR)

Mild–moderate PDR

New vessels on the disc (NVD) or new vessels elsewhere (NVE), but extent insufficient to meet the high-risk criteria

High-risk PDR

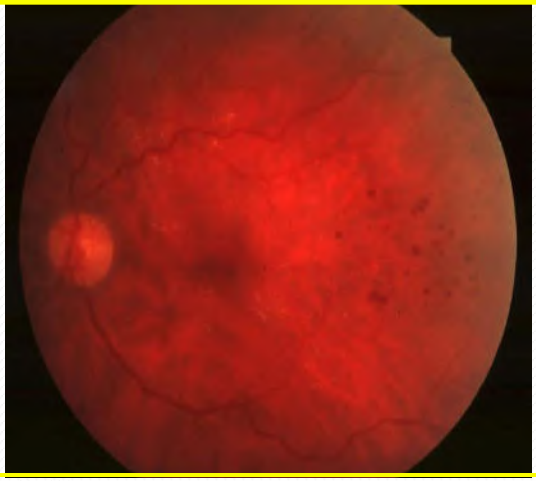
- New vessels on the disc (NVD) greater than ETDRS standard photograph 10A (about ⅓ disc area)
- Any NVD with vitreous haemorrhage
- NVE greater than ½ disc area with vitreous haemorrhage

Advanced diabetic eye disease

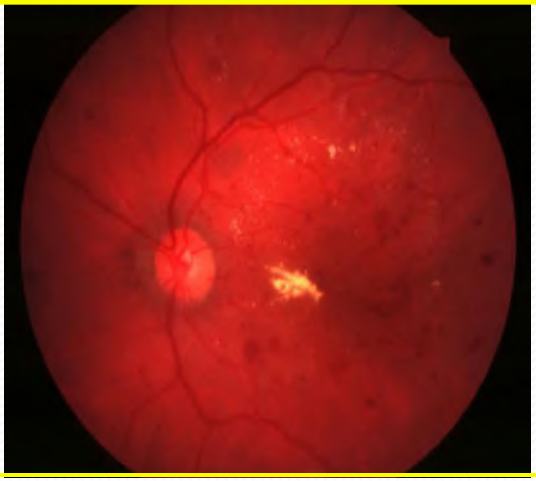
1. Persistent vitreous hemorrhage
2. Tractional retinal detachement
3. NVG

ETDRS Classification of Diabetic retinopathy

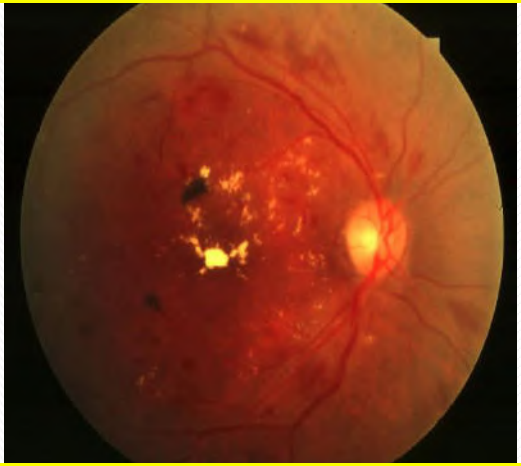
Diabetic retinopathy: NPDR



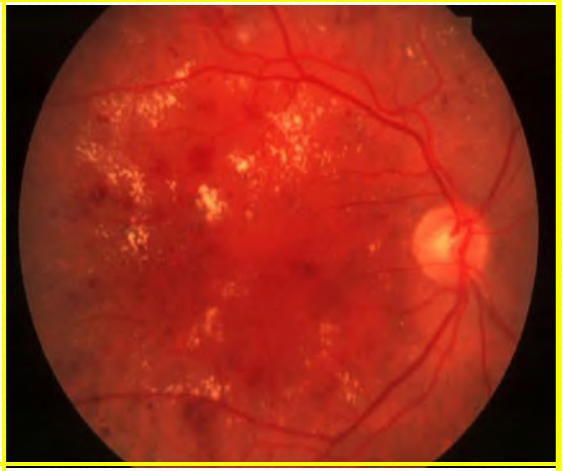
Microaneurysms



Intraretinal dot and blot haemorrhages



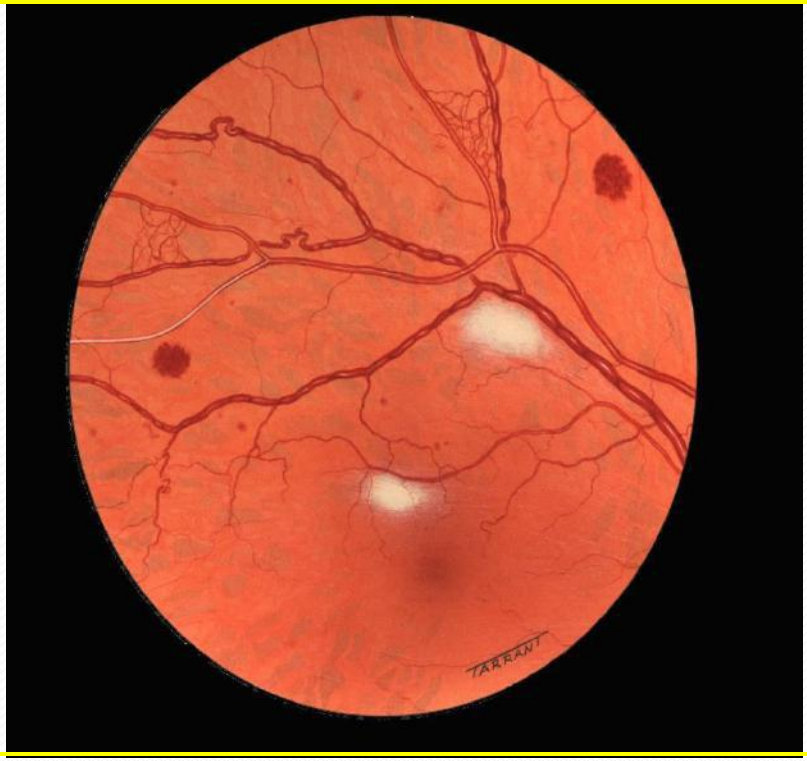
Hard exudates



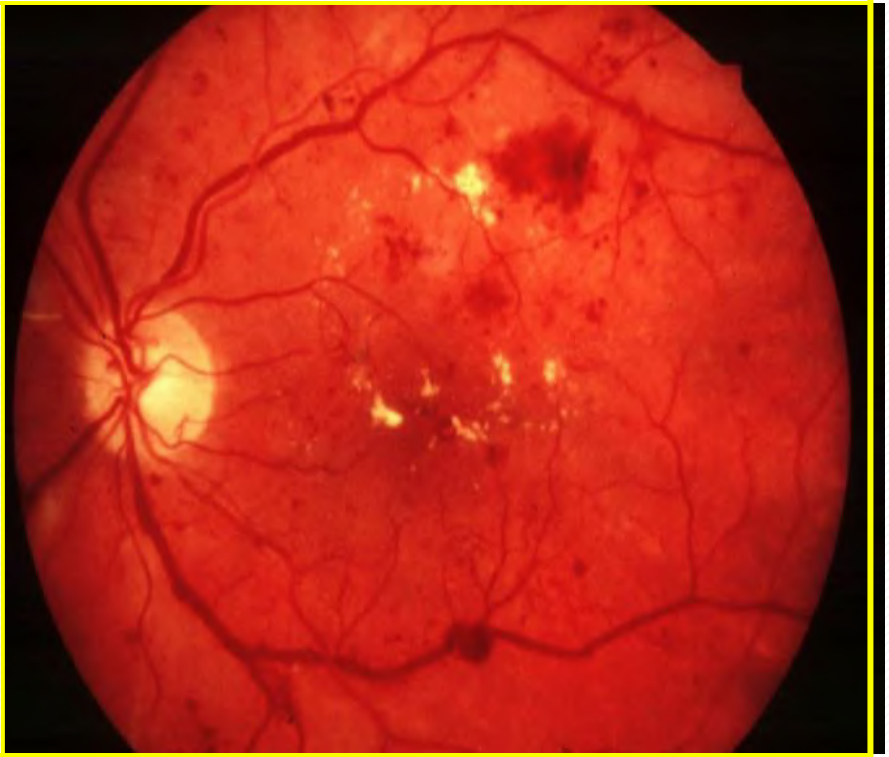
Retinal edema

Non-Proliferative diabetic retinopathy

Signs



- Cotton-wool spots
- Venous irregularities



- Dark blot haemorrhages
- Intraretinal microvascular abnormalities (IRMA)

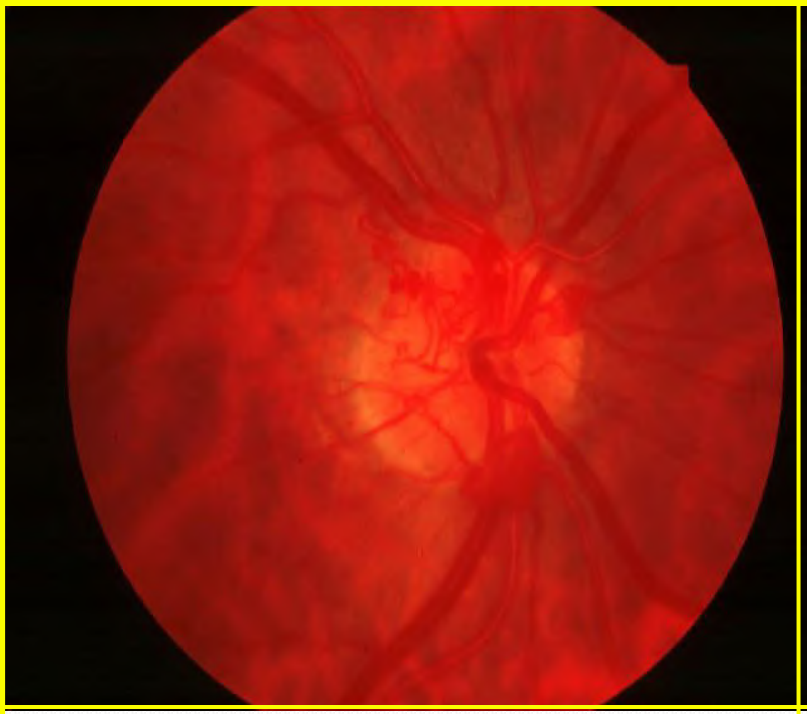
Treatment - watch for proliferative disease

Proliferative diabetic retinopathy

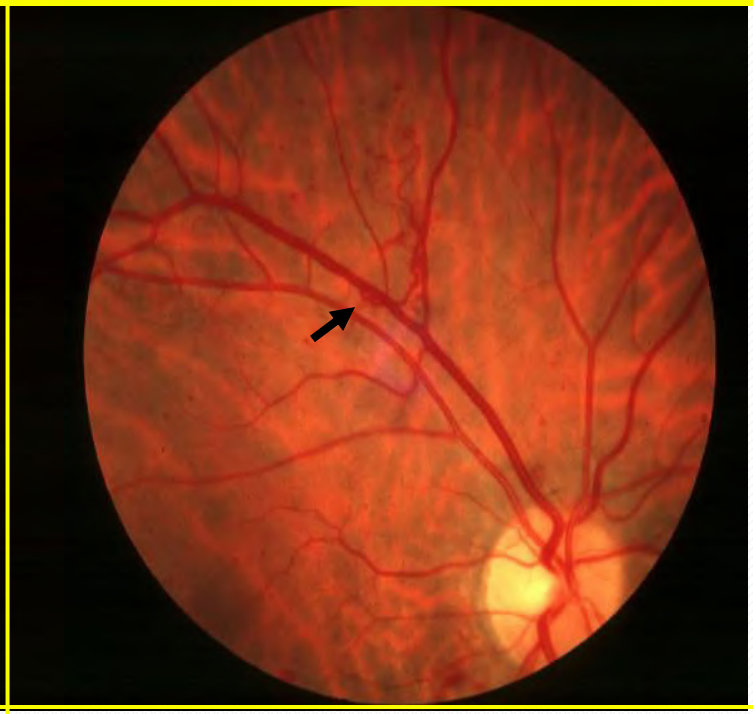
- Affects 5-10% of diabetics
- IDD at increased risk (60% after 30 years)

Neovascularization

- Flat or elevated
- Severity determined by comparing with area of disc

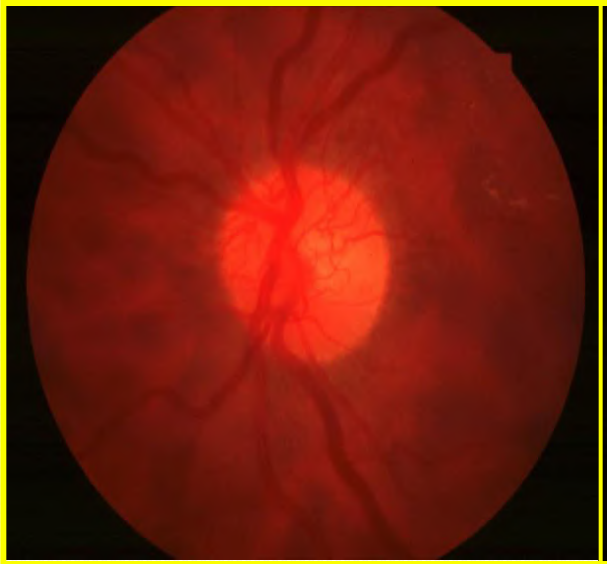
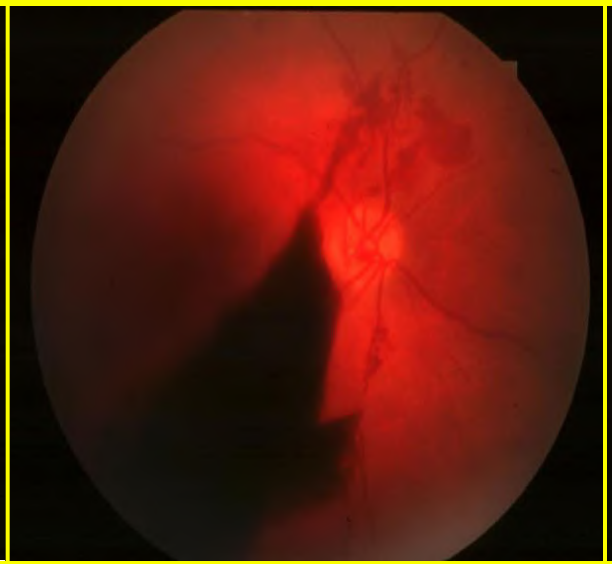
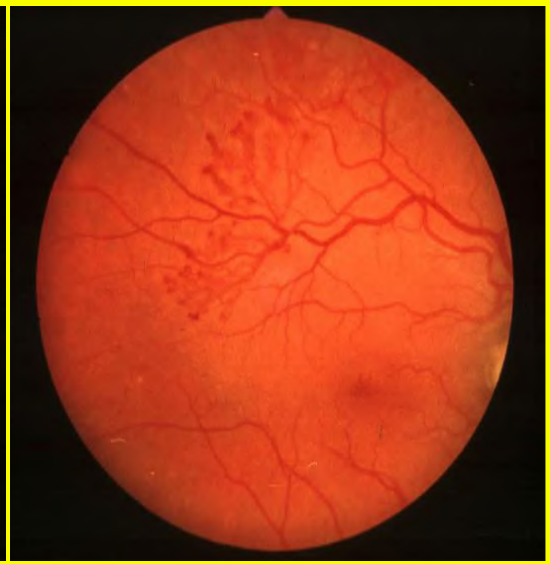


Neovascularization of disc



Neovascularization elsewhere

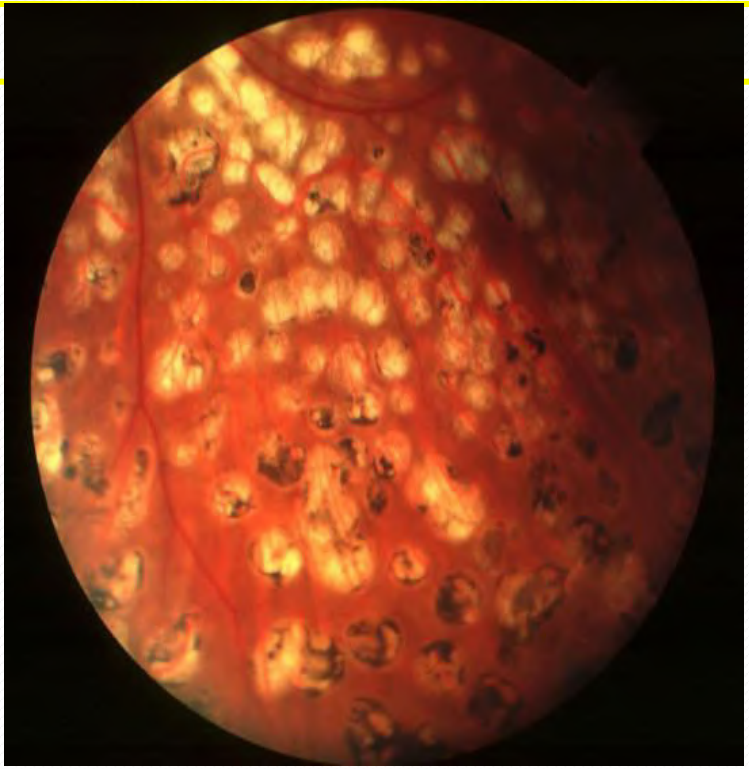
Indications for treatment of proliferative diabetic retinopathy

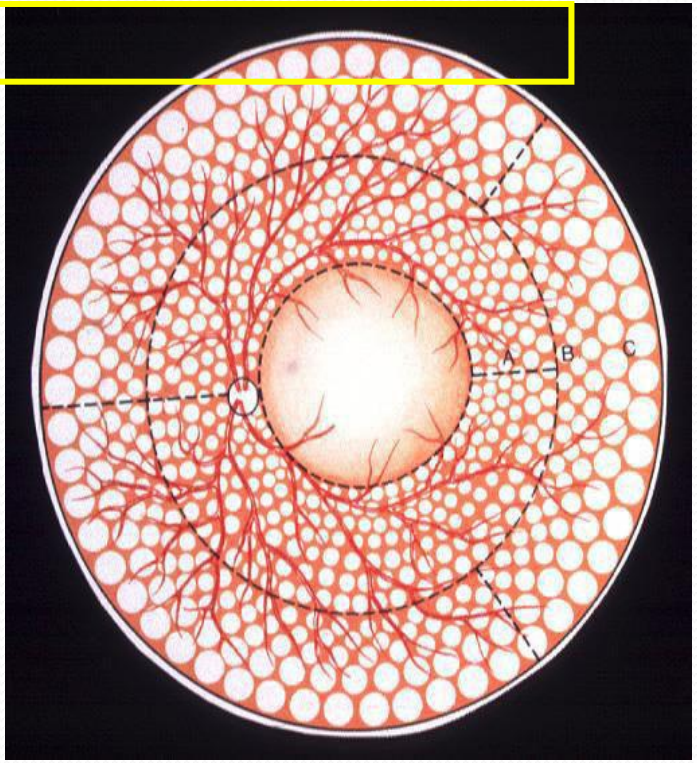
		
NVD > 1/3 disc in area	Less extensive NVD + haemorrhage	NVE > 1/2 disc in area + haemorrhage

Proliferative diabetic retinopathy

50% of untreated patients with proliferative retinopathy------- legally blind -----five years

Laser Pan-Retinal Photocoagulation

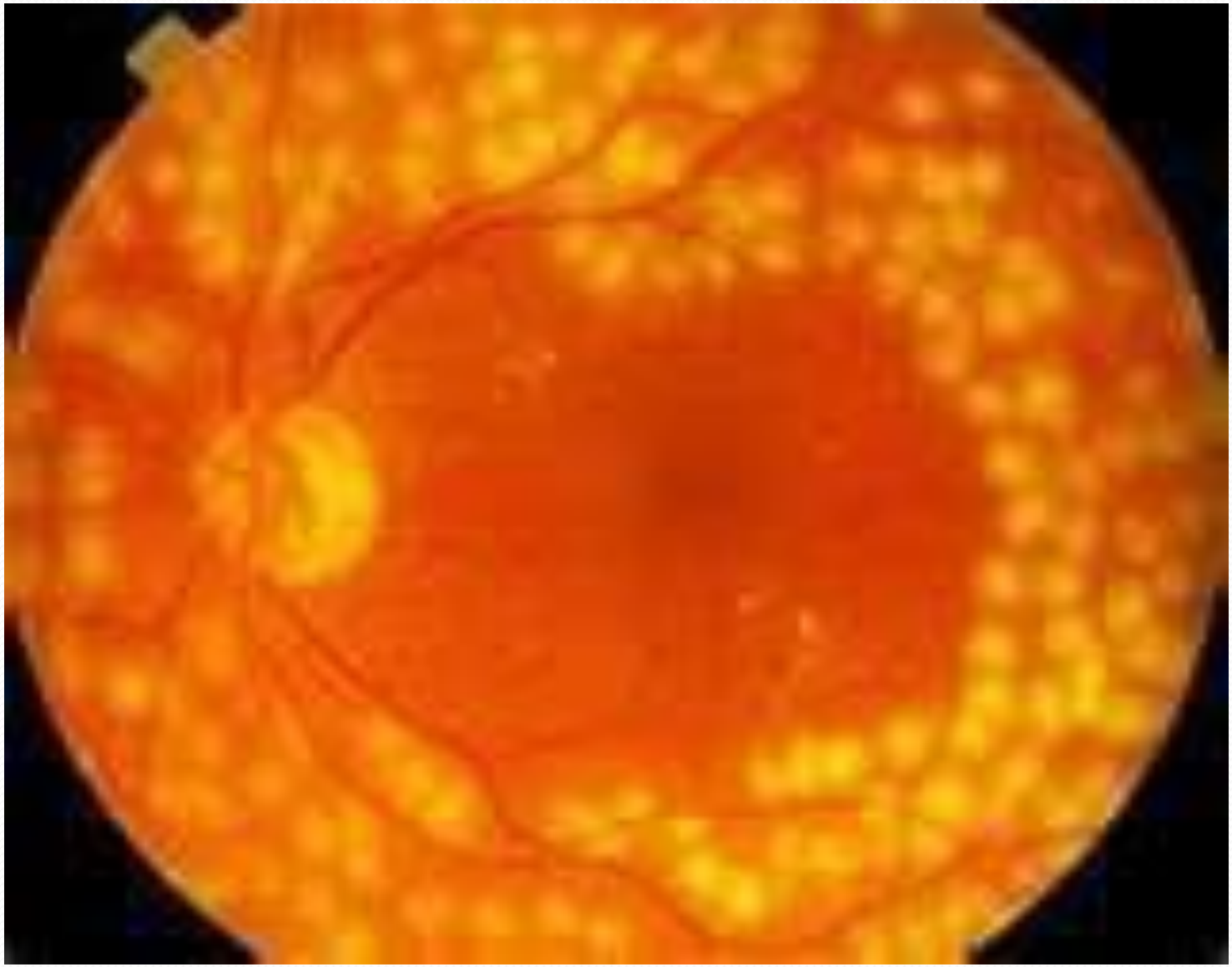




- Initial treatment is 2000-3000 burns
- Spot size (200-500 µm) depends on contact lens magnification
- Gentle intensity burn

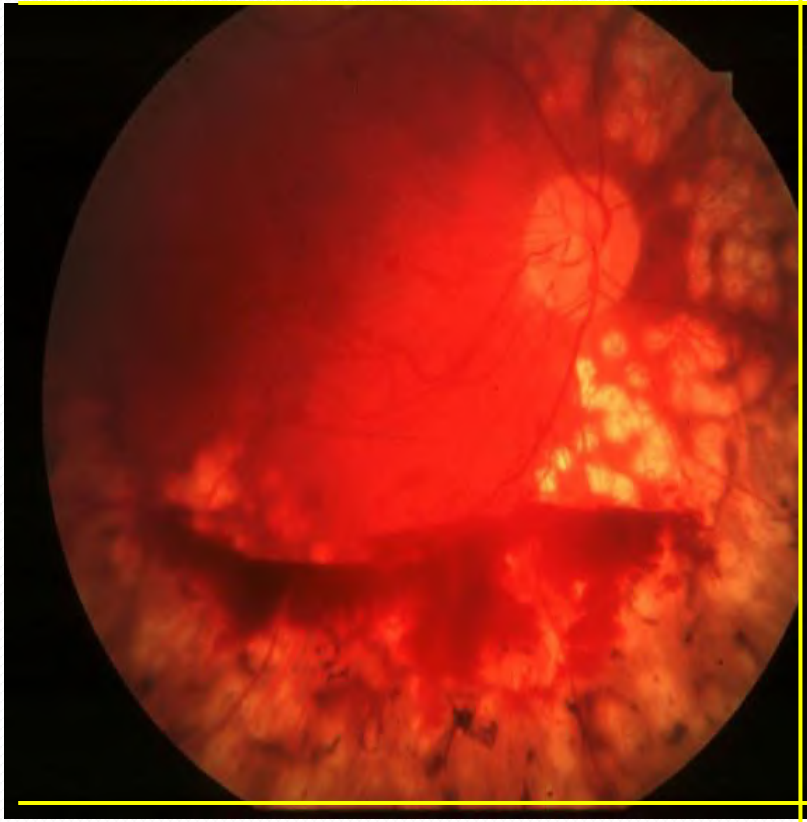
Area covered by complete PRP

Follow-up 4 to 8 weeks



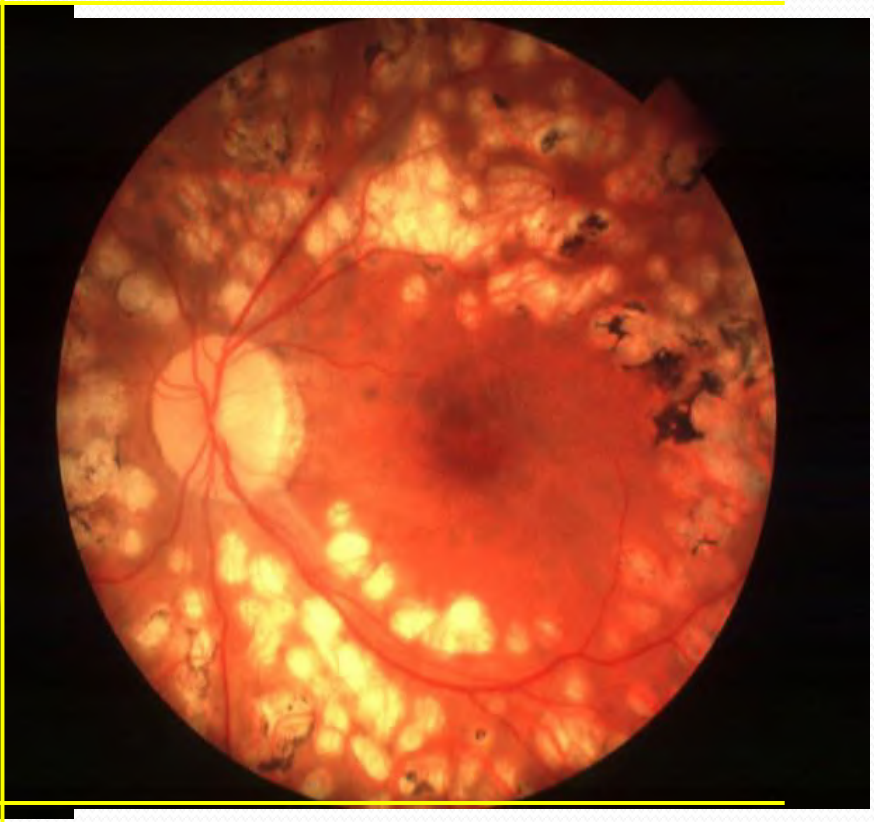
Assessment after photocoagulation

Poor involution



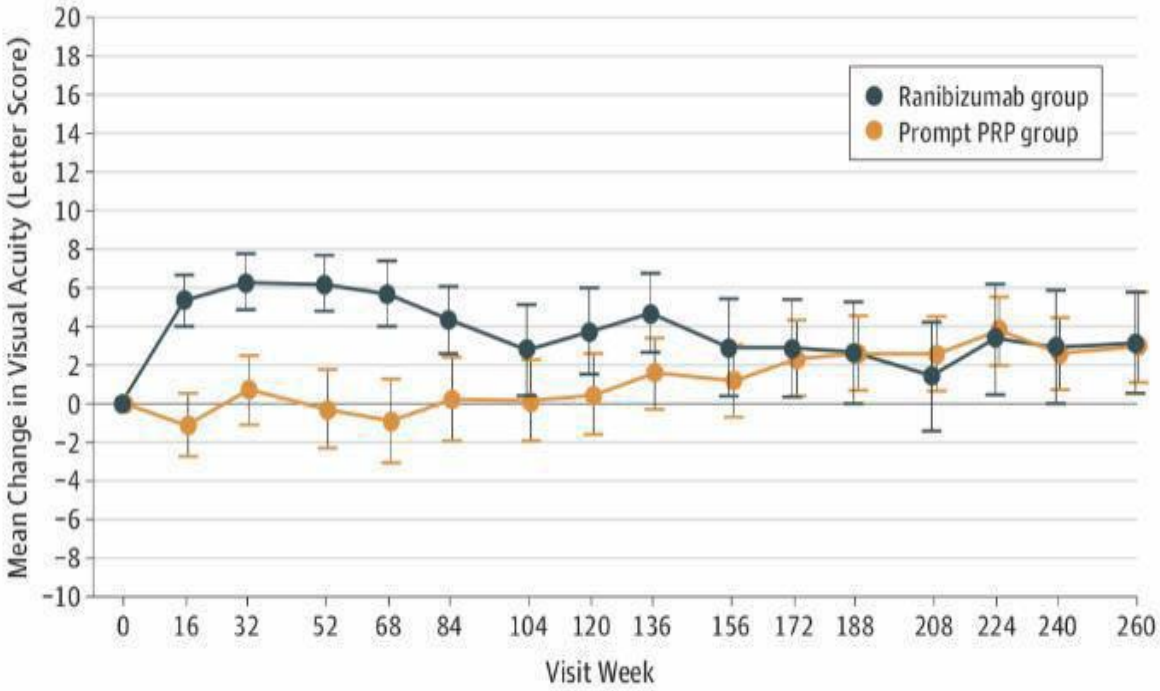
- Persistent neovascularization
- Haemorrhage
- Re-treatment required

Good involution



- Regression of neovascularization
- Residual ‘ghost’ vessels or fibrous tissue
- Disc pallor

Protocol S: 5 yr Results Visual Acuity Change



No. of eyes					
Ranibizumab group	191	160	139	124	117
Prompt PRP group	203	168	146	134	123
Change in visual acuity, mean (SD)					
Ranibizumab group		2.8 (15.2)	2.9 (14.9)	1.4 (15.7)	3.1 (14.3)
Prompt PRP group		0.2 (13.7)	1.2 (11.4)	2.6 (11.0)	3.0 (10.5)
Adjusted difference (95% CI)		2.2 (-0.5 to 5.0)	1.6 (-1.1 to 4.3)	-0.7 (-4.0 to 2.5)	0.6 (-2.3 to 3.5)
P value		.11	.24	.66	.68

Ocular & Systemic Adverse Effects

Variable	No. (%) Participants With 2 Study Eyes (1 in Each Group)	Ranibizumab Group	PRP Group	*P* Value
Systemic Adverse Events[a,b]				
No. of participants	89	102	114[c]	NA
Vascular events defined by APTC criteria occurring at least once through 5 y[d]				
Nonfatal myocardial infarction[e]	3 (3)	6 (6)	4 (4)	.64
Nonfatal stroke[e]	3 (3)	6 (6)	7 (6)	.65
Death due to potential vascular cause or unknown cause[f]	6 (7)	7 (7)	2 (2)	.13
Any event	12 (13)	18 (18)	12 (11)	.31
Prespecified events occurring at least once through 5 y				
Death from any cause	8 (9)	13 (13)	7 (6)	.24
Hospitalization	54 (61)	66 (65)	61 (54)	.24
Serious adverse event	56 (63)	68 (67)	63 (55)	.21
Hypertension	28 (31)	38 (37)	28 (25)	.13
Ocular Adverse Events[a,g]				
No. of eyes	NA	191	203	NA
No. of injections	NA	3132	981	NA
Ocular adverse events occurring at least once through 5 y				
Endophthalmitis	NA	1 (<1)	0	NA
Inflammation[h]	NA	3 (2)	10 (5)	.05
Retinal tear	NA	1 (<1)	0	NA
Cataract surgery	NA	31 (16)	38 (19)	.62
Elevation in IOP (met any of the criteria)[i]	NA	30 (16)	36 (18)	.58
Increase of IOP ≥10 mm Hg from baseline at any visit	NA	17 (9)	29 (14)	.10
IOP ≥30 mm Hg at any visit	NA	6 (3)	11 (5)	.39
Initiation of glaucoma medications at any visit	NA	18 (9)	21 (10)	.67
Received glaucoma procedure at any visit	NA	6 (3)	4 (2)	.37

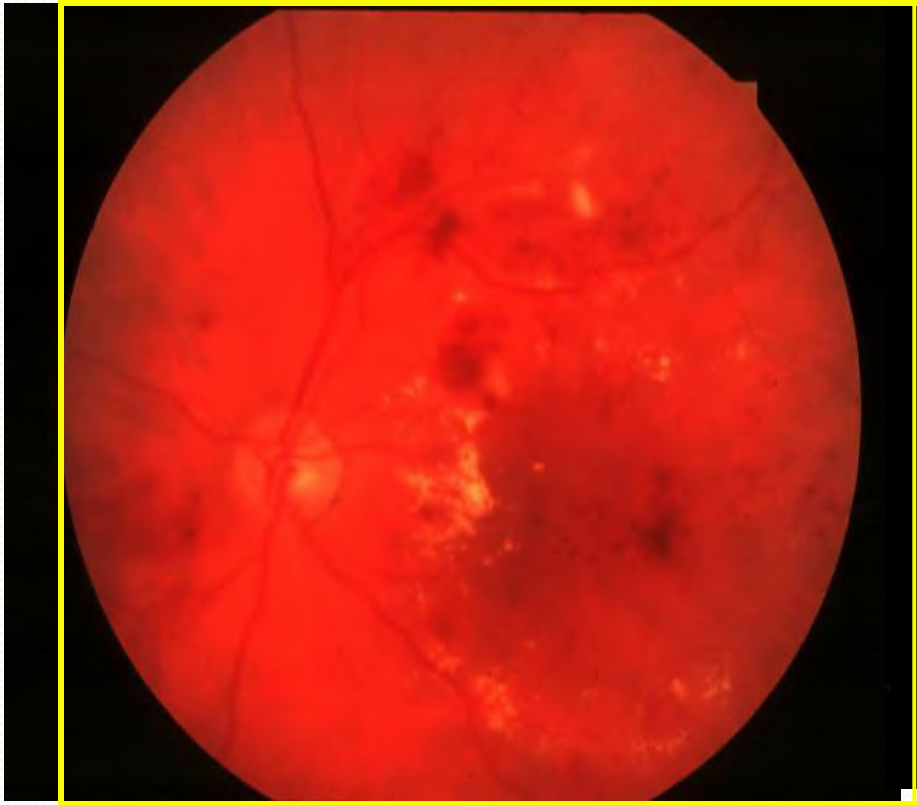
PDR: How to treat?

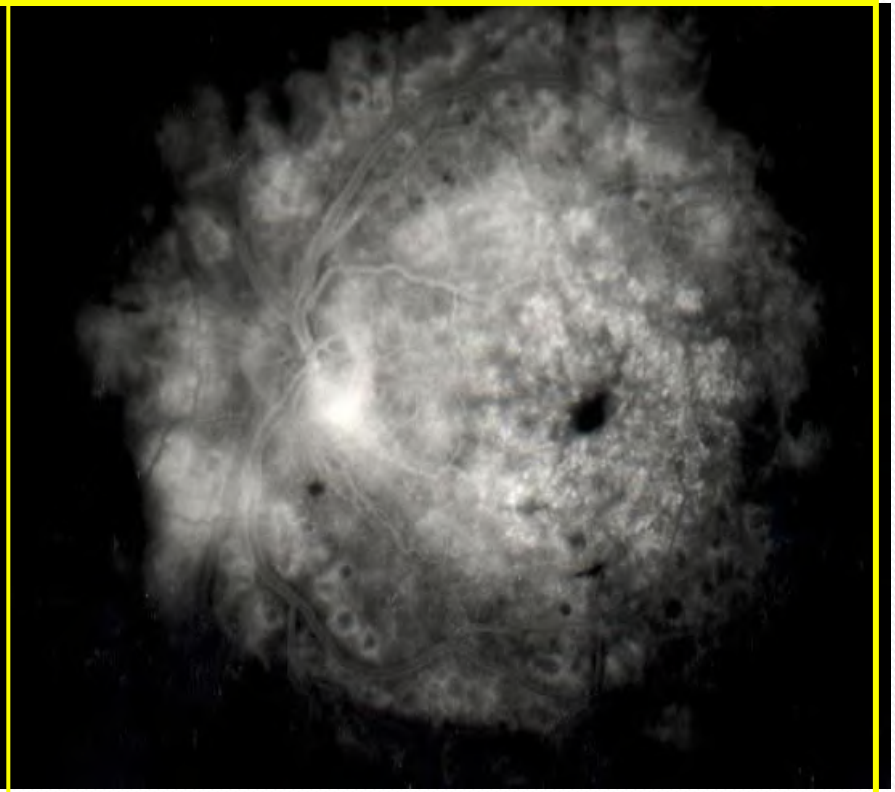
Protocol *S* : Individualized your patients

This should be based on

- **Systemic risk factors control**
- **Cost**
- **Compliance**
- **Presence of DME**

Diffuse diabetic maculopathy

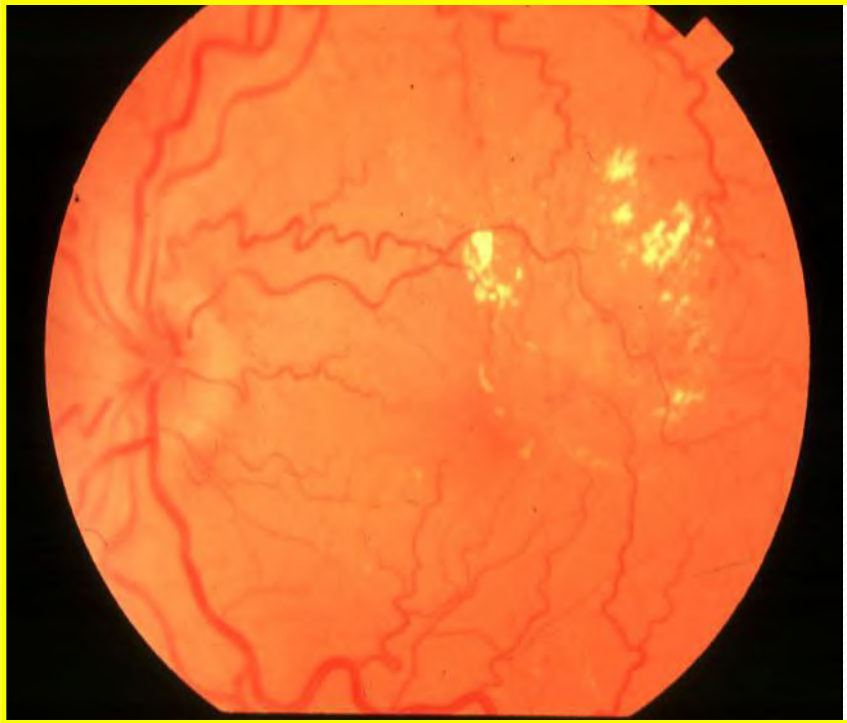




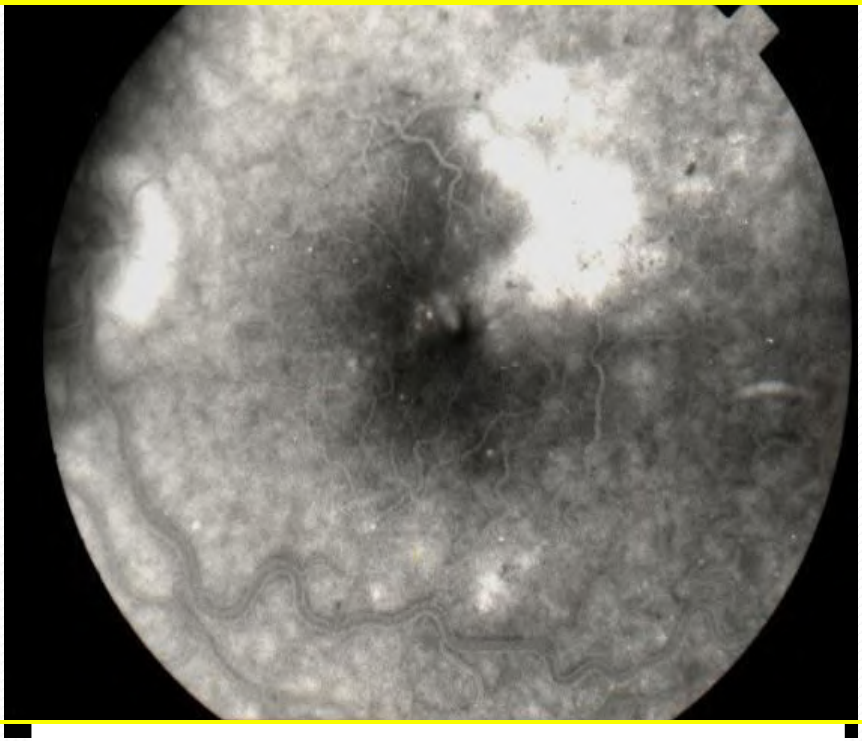
- Diffuse retinal thickening
- Cystoid macular oedema
- Impairment of visual acuity

- Generalized leakage on FA
- Grid photocoagulation ??
- Anti-VEGF

Focal diabetic maculopathy

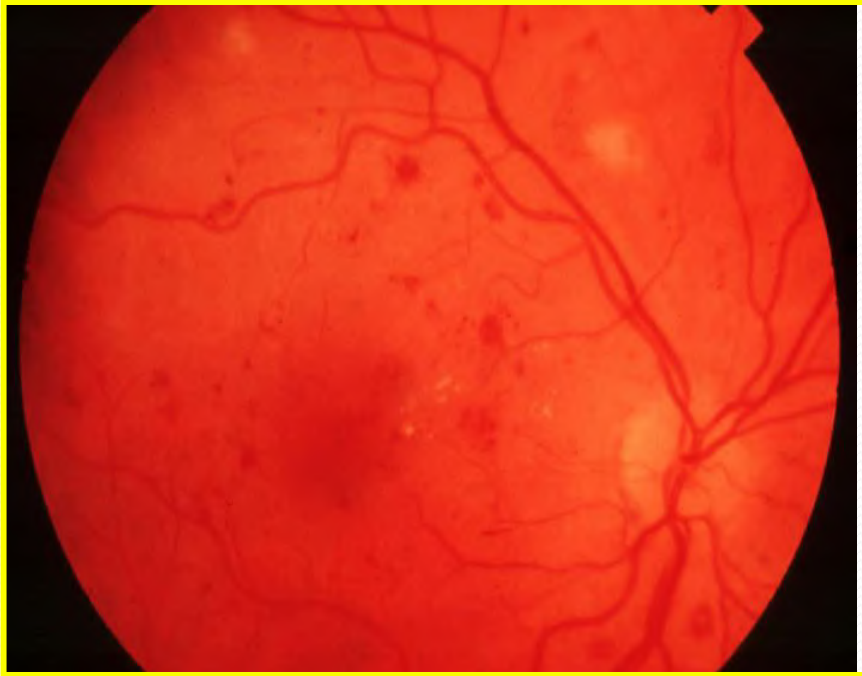


- Circumscribed retinal thickening
- Complete or incomplete circinate hard exudates

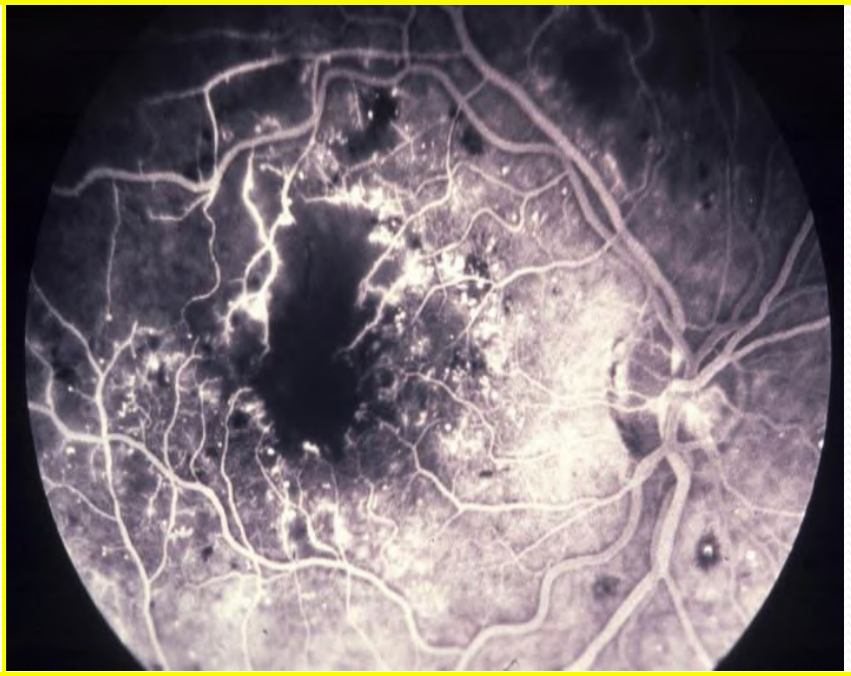


- Focal leakage on FA
- Focal photocoagulation
- Good prognosis

Ischaemic diabetic maculopathy

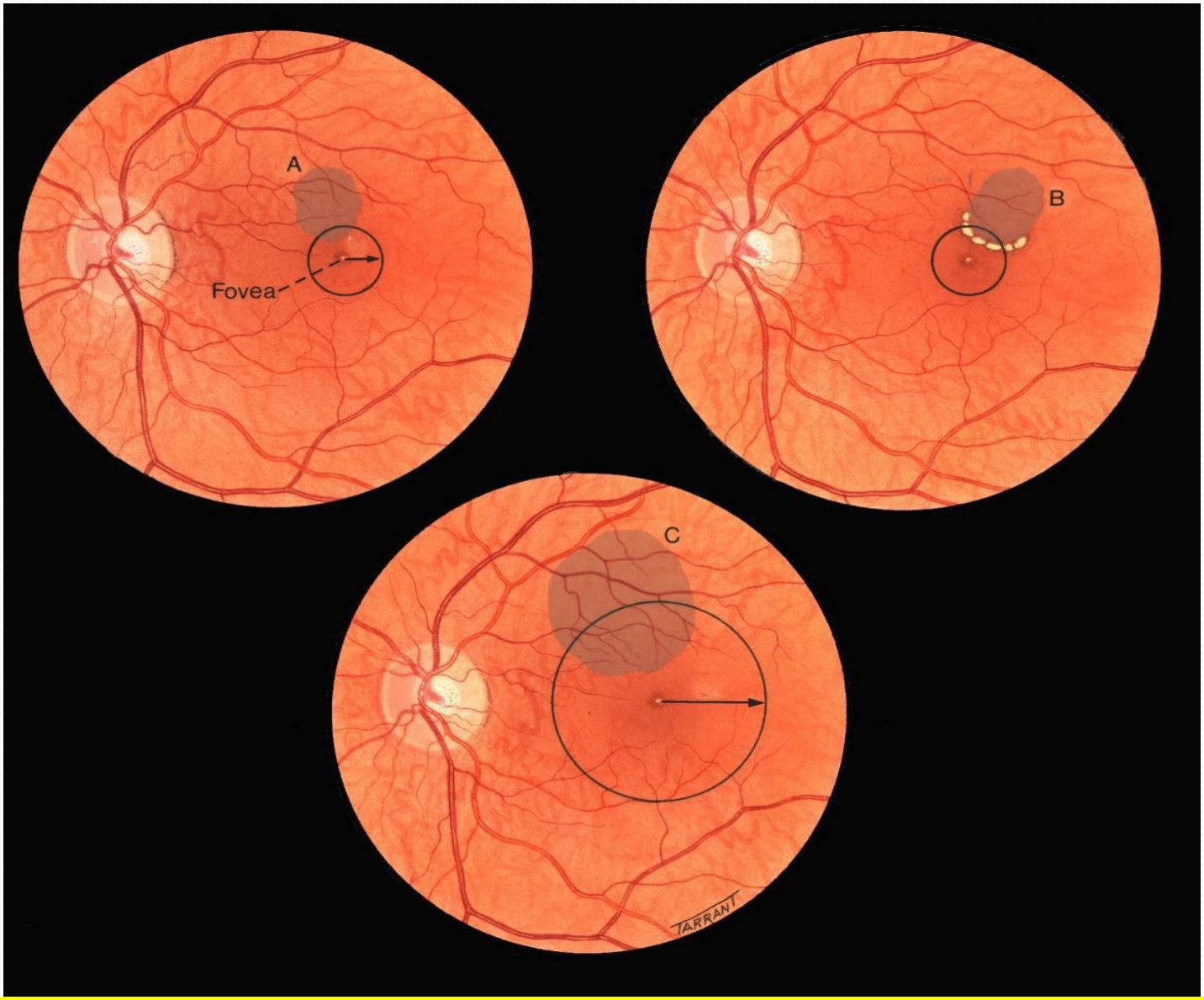


- Macula appears relatively normal
- Poor visual acuity

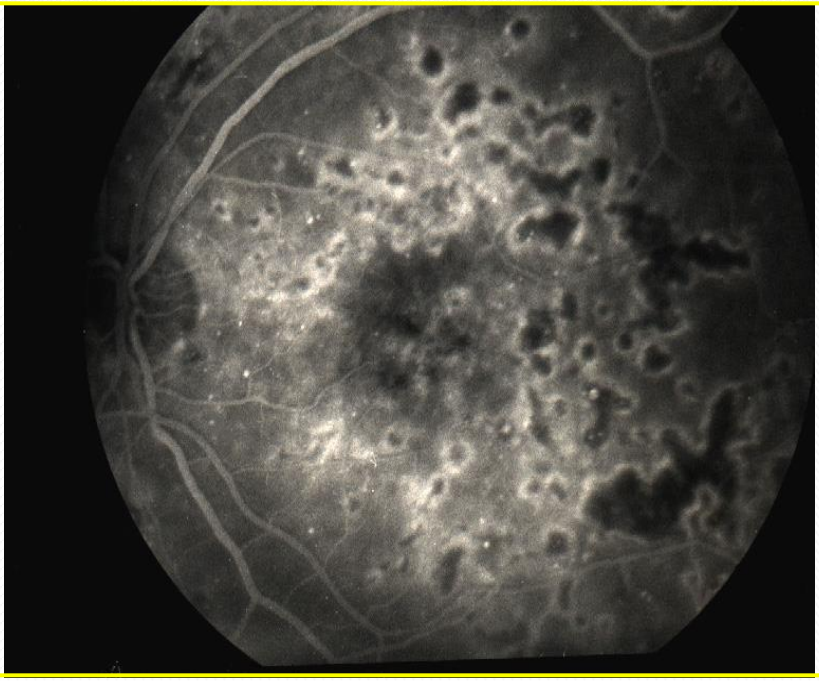
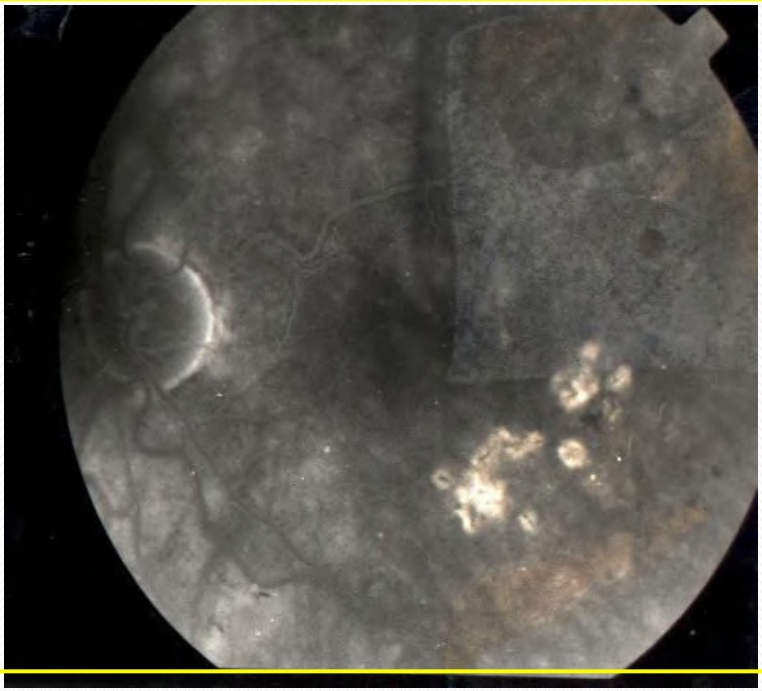


- Capillary non-perfusion on FA

Clinically Significant Macular Edema



Treatment of clinically significant macular edema

Grid treatment	Focal treatment
	
·Grid Pattern 500 µm from temporal margin of disc 500 µm away from centre of fovea · Gentle burns (100-200 µm, 0.10 sec), one burn width apart	· For microaneurysms in centre of hard exudate rings

Diabetic Macular Edema

- Macular Edema ….. No traction
- Center involving DME … CI DME
- Non-Center involving DME … NCI DME
- Anti VEGF

  Avastin

  Lucentis

  Eyelea

Intra vitreal Anti VEGF

Intra vitreal Steriods

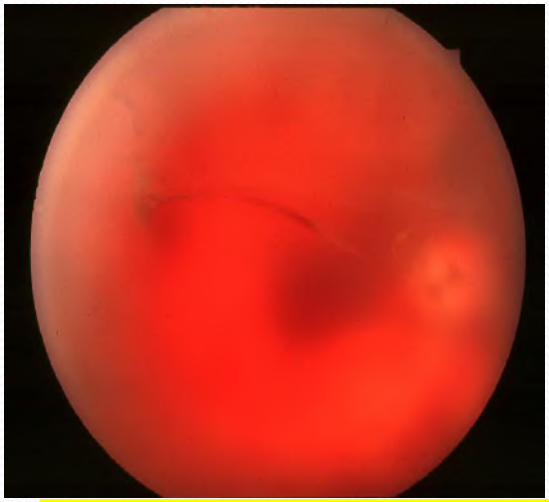
(TA, Ozerdex,Retisert)

Laser/Micropulse Laser

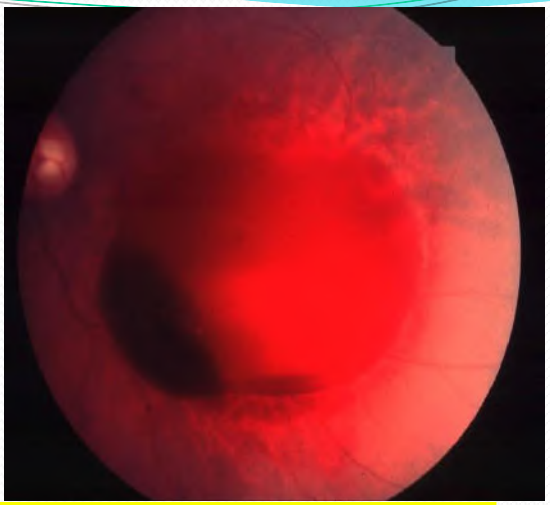
Risk factors control

Follow up

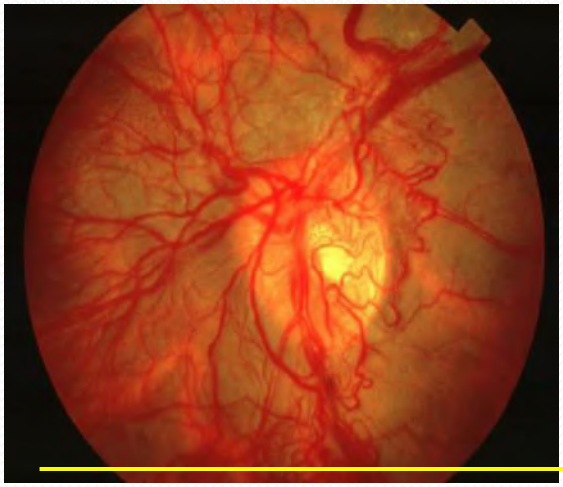
Indications for vitreoretinal surgery



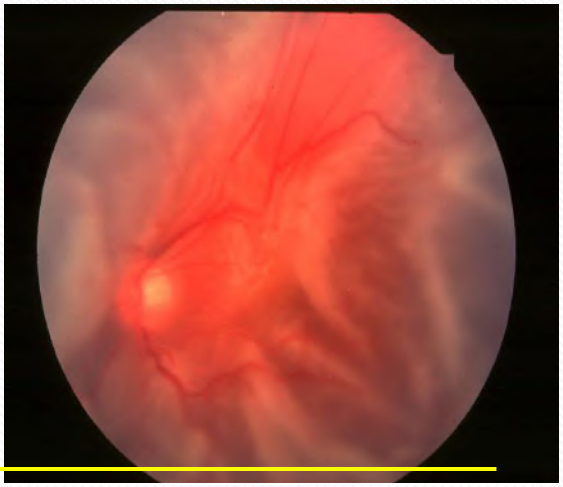
Severe persistent vitreous haemorrhage



Dense, persistent premacular haemorrhage



Progressive proliferation despite laser therapy



Retinal detachment involving macula

National / Local data:

Hospital based studies

Diabetic Control

80% DM not controlled HBA1C

Poor controlled 62.62% , Controlled 19.62%, Borderline 17.76%

Presentation to Ophthalmologist

Referrals by physicians/endocrinologist/GP/Knew about DR **Only 29 %**

71% presented with DR /complications/with irrelevant symptoms

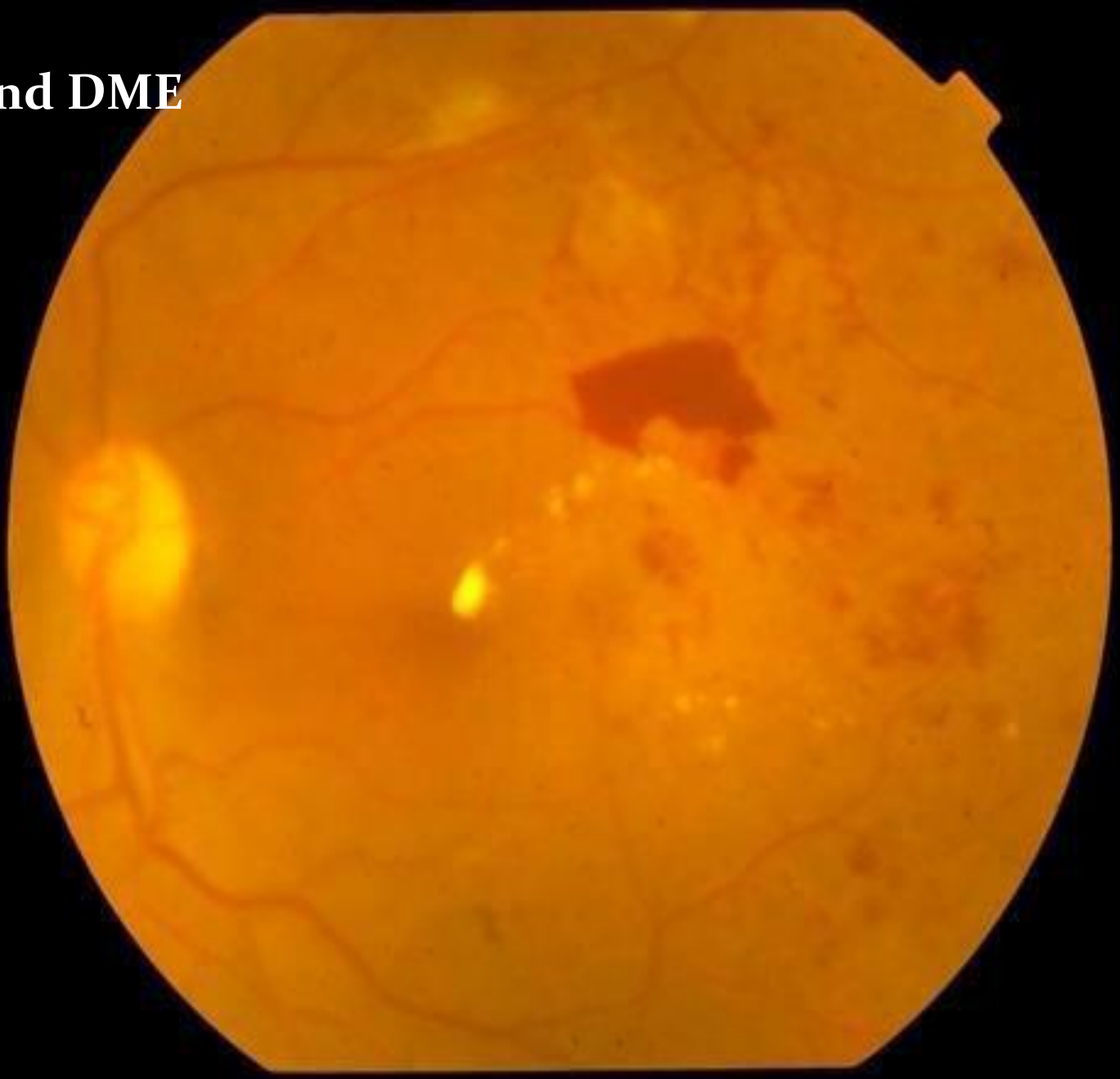
Risk factors Awareness

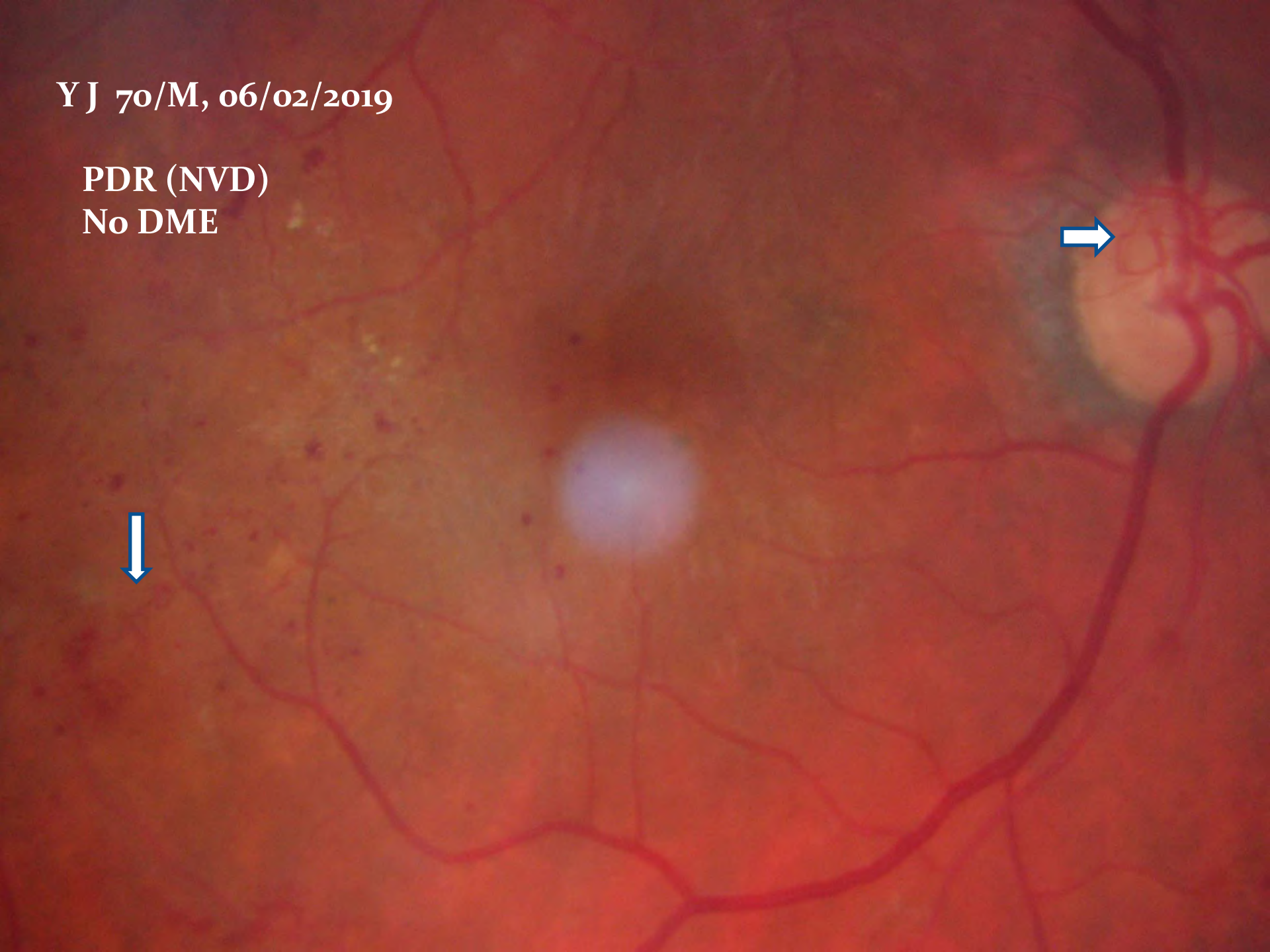
80% or more had no clue regarding any DR risk factors

Sanaullah Jan et al. Status of Diabetic Retinopathy and its presentation patterns in diabetics at ophthalmology clinics. J Postgrad Med Inst 2018; 32(1):24-26.

Sanaullah Jan et al. Diabetic retinopathy: Risk Factors Awareness and Presentation. OAJ Ophthalmol 2017; 2(2):000122

PDR and DME

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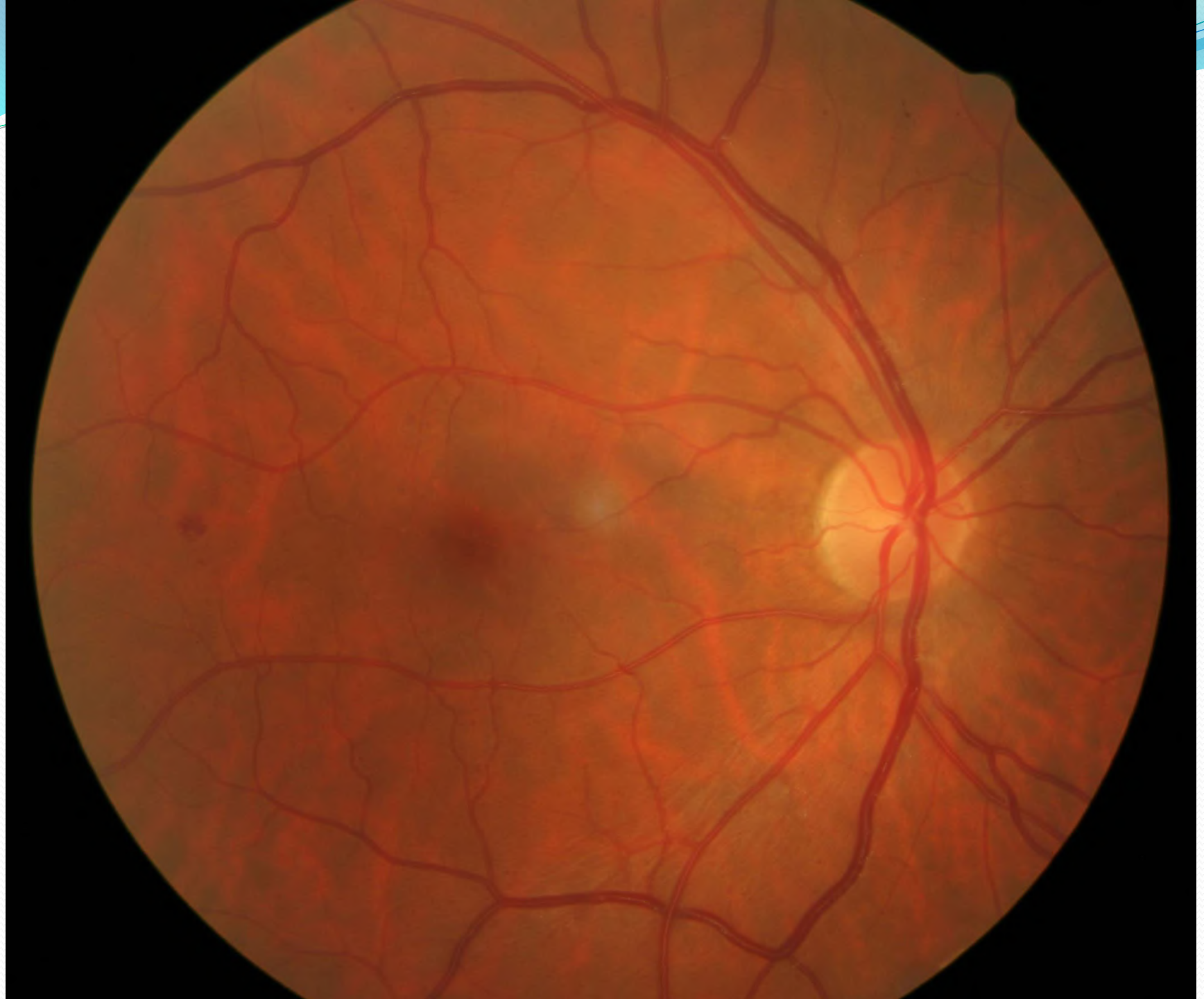

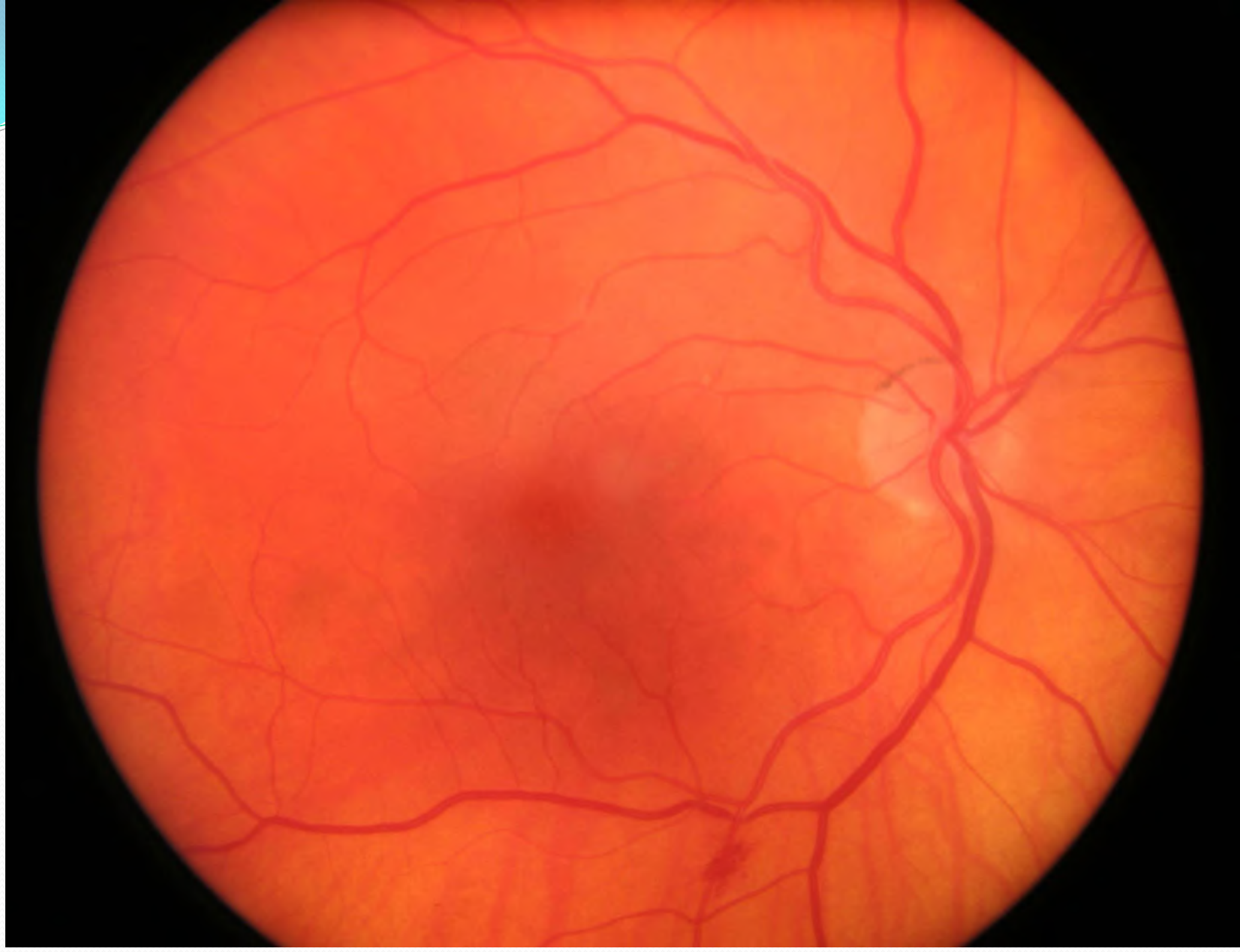

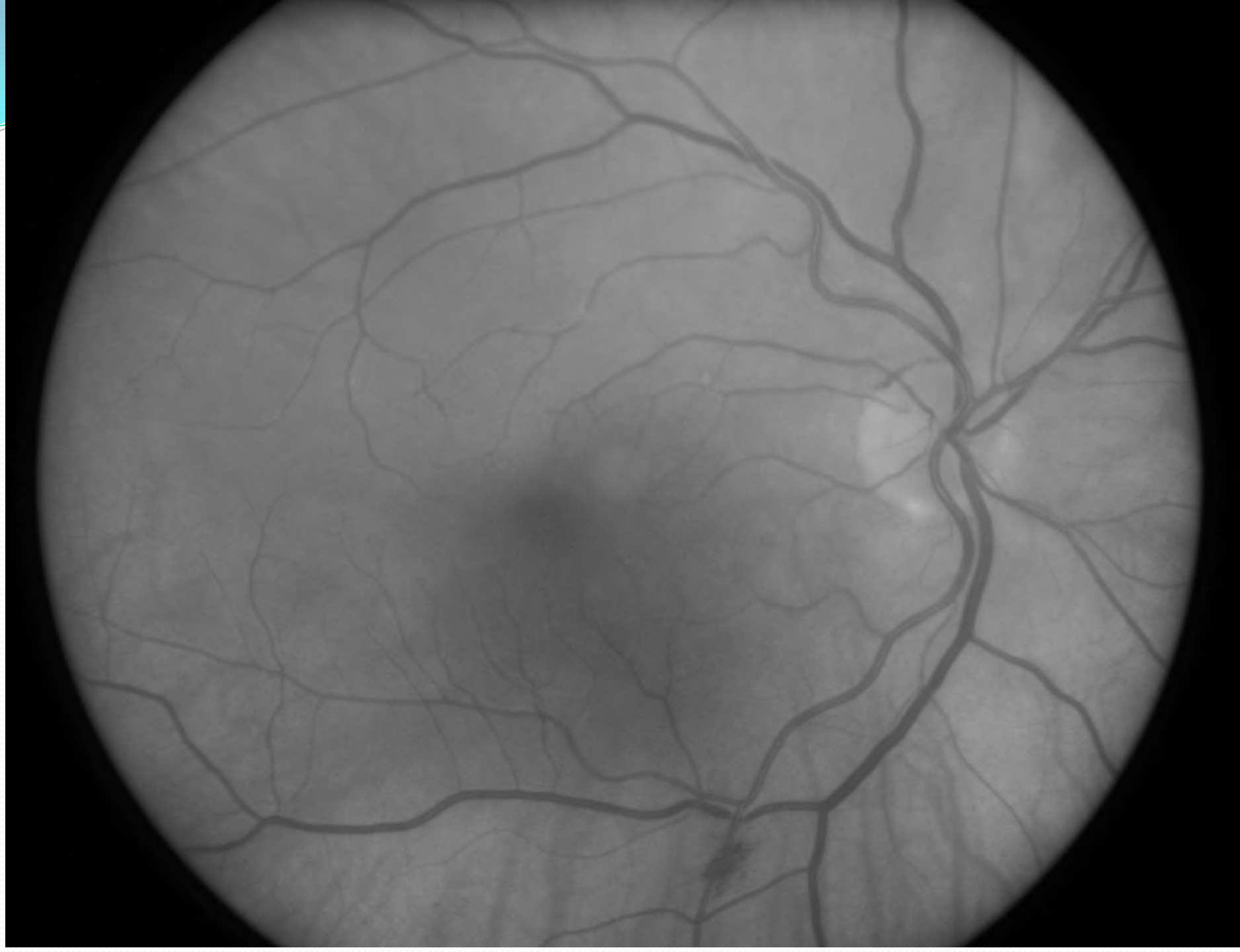

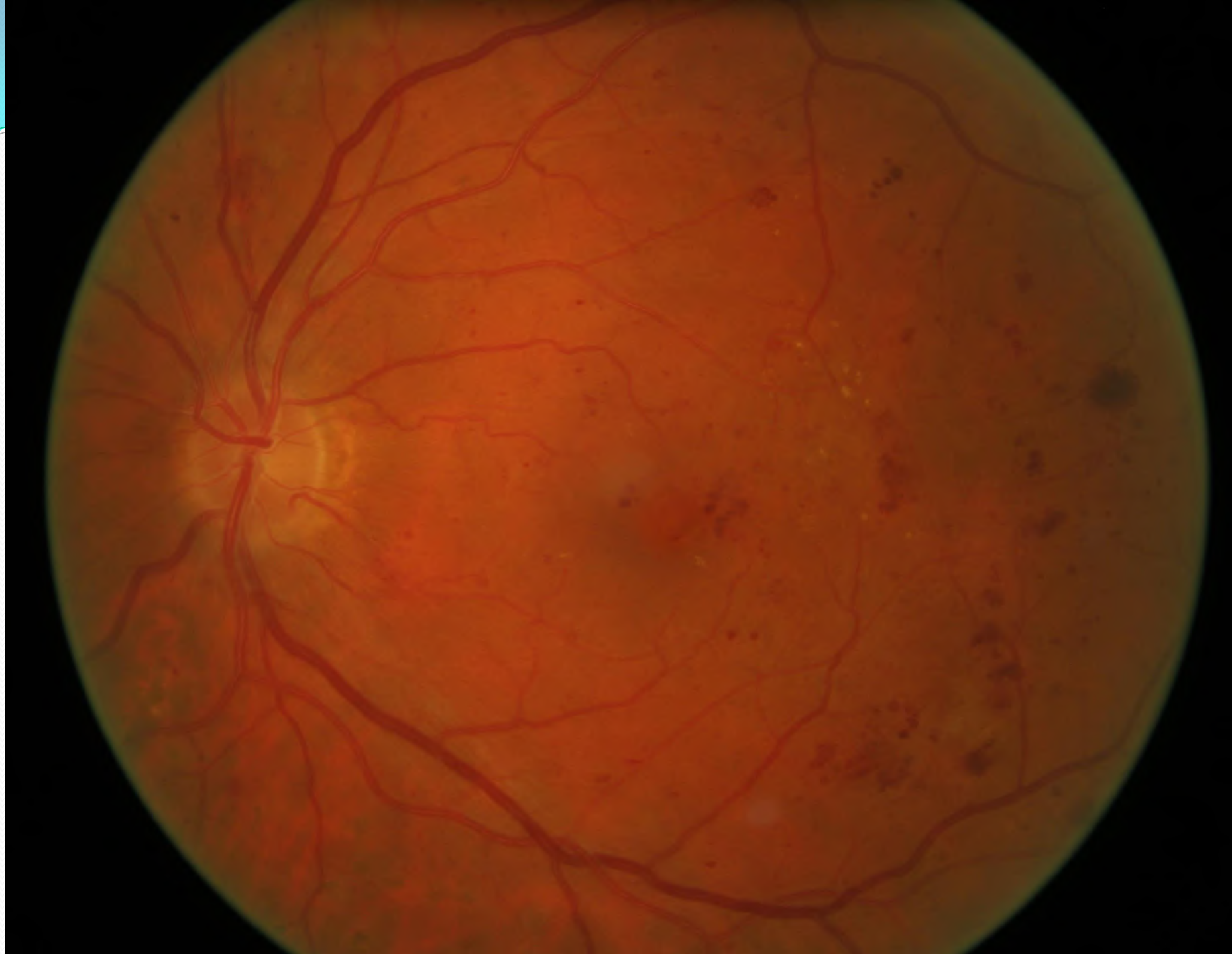

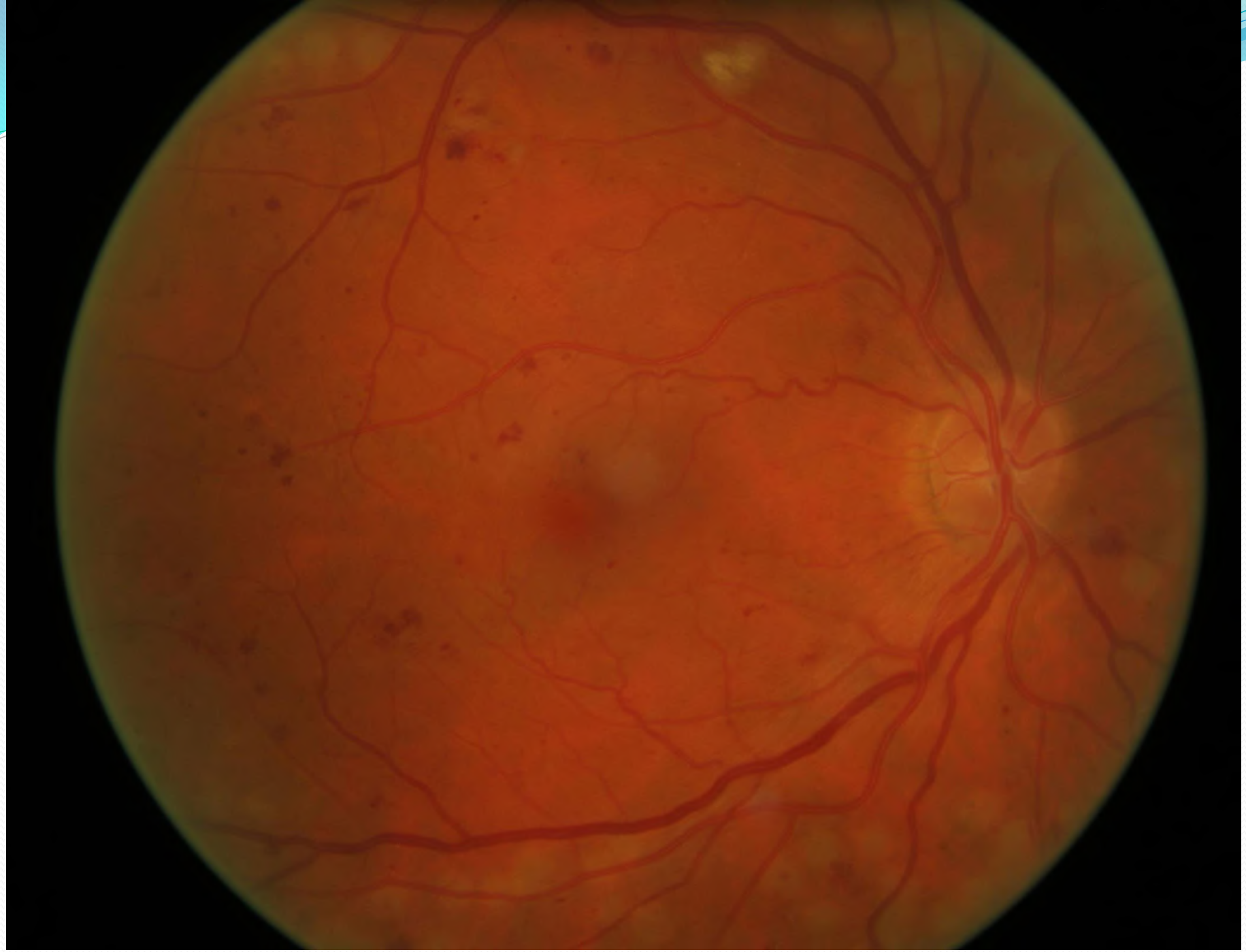

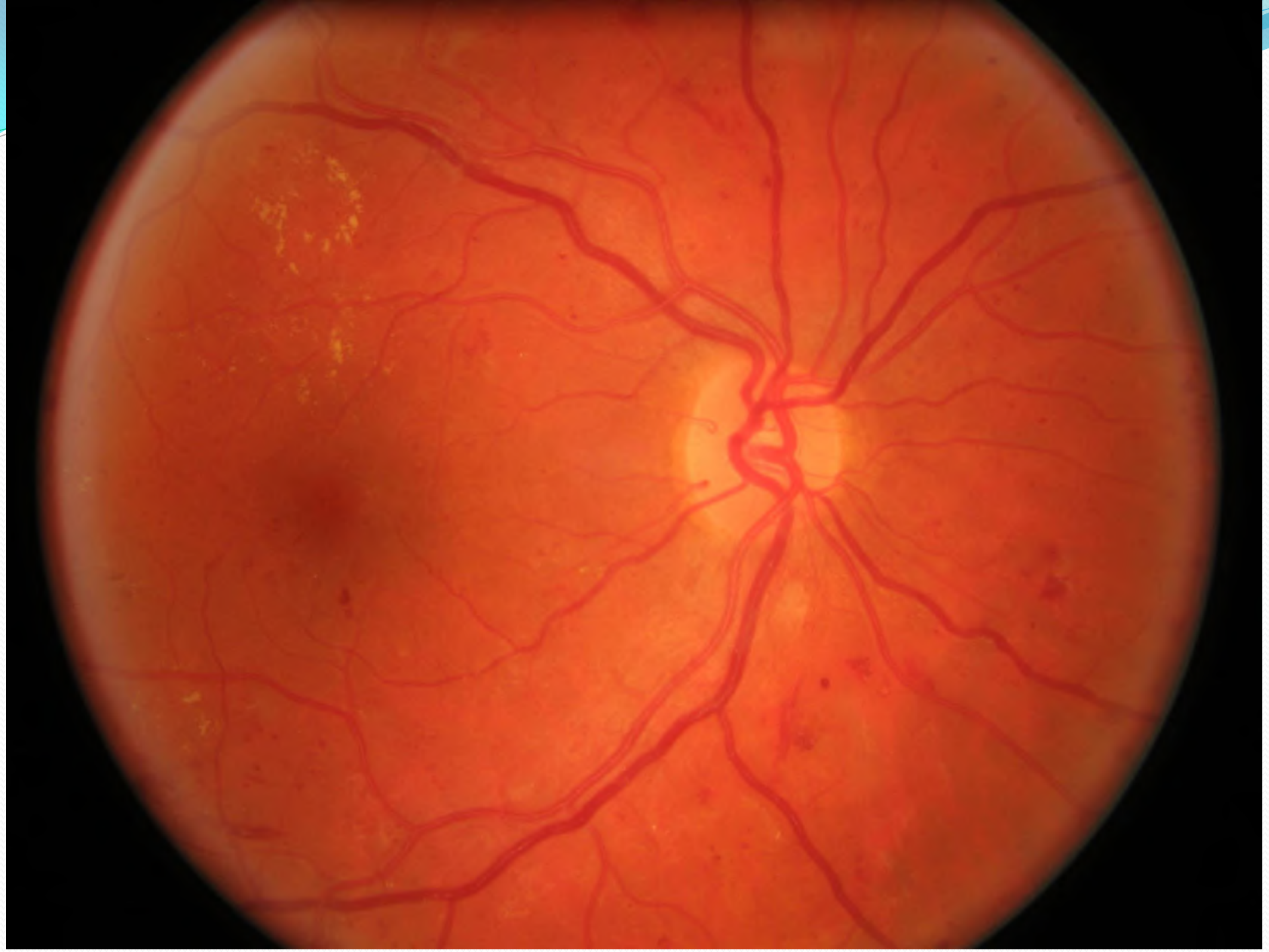

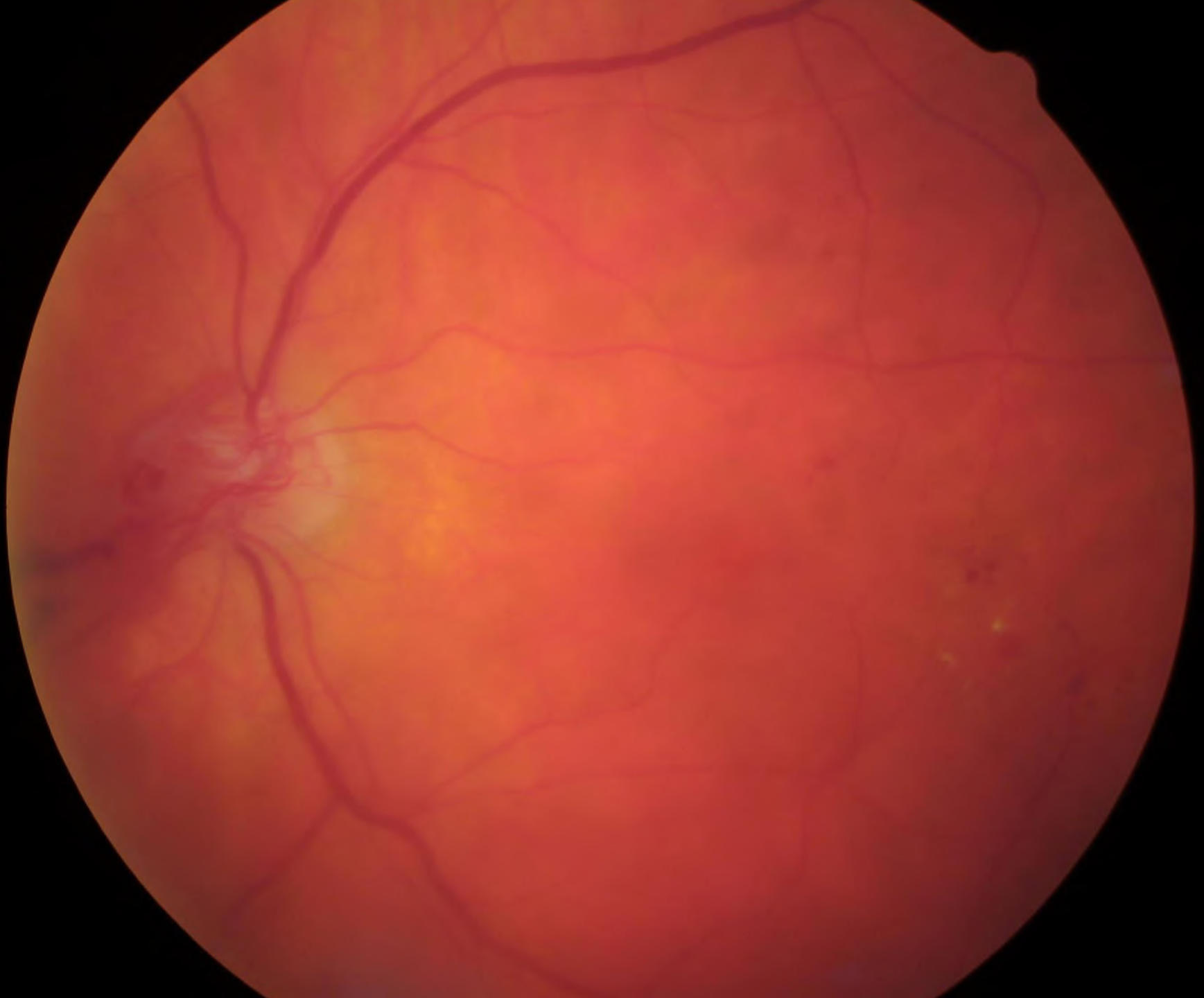

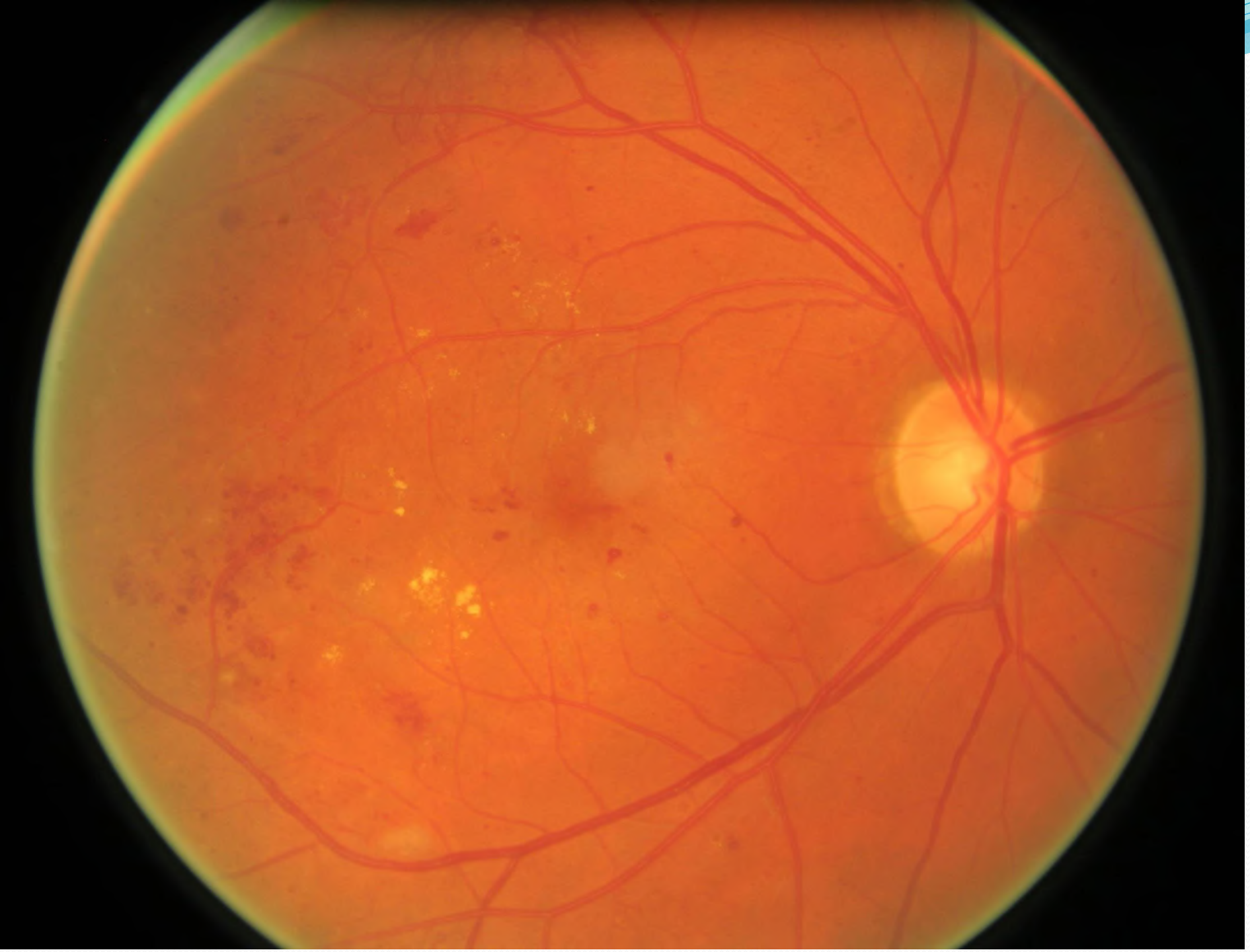

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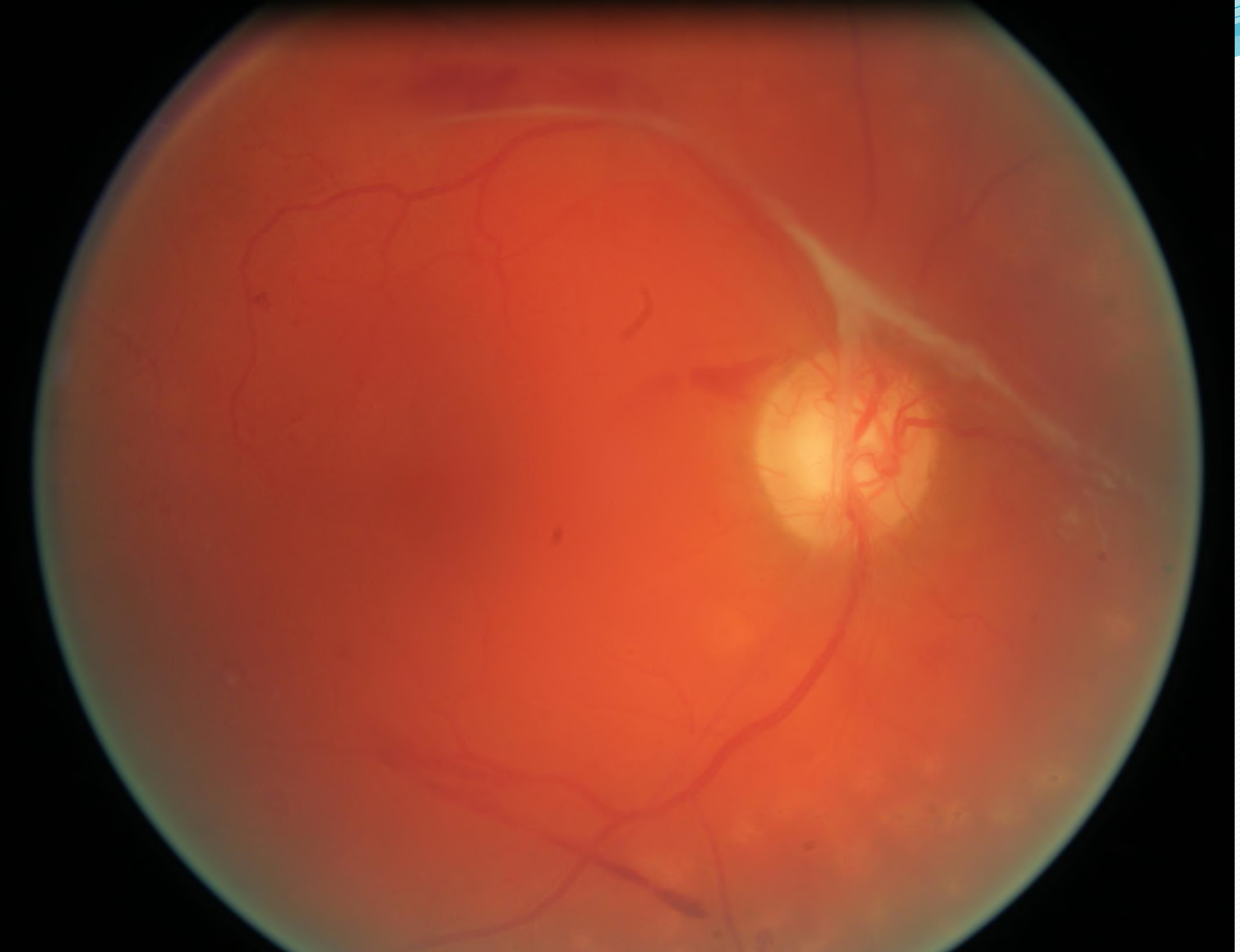

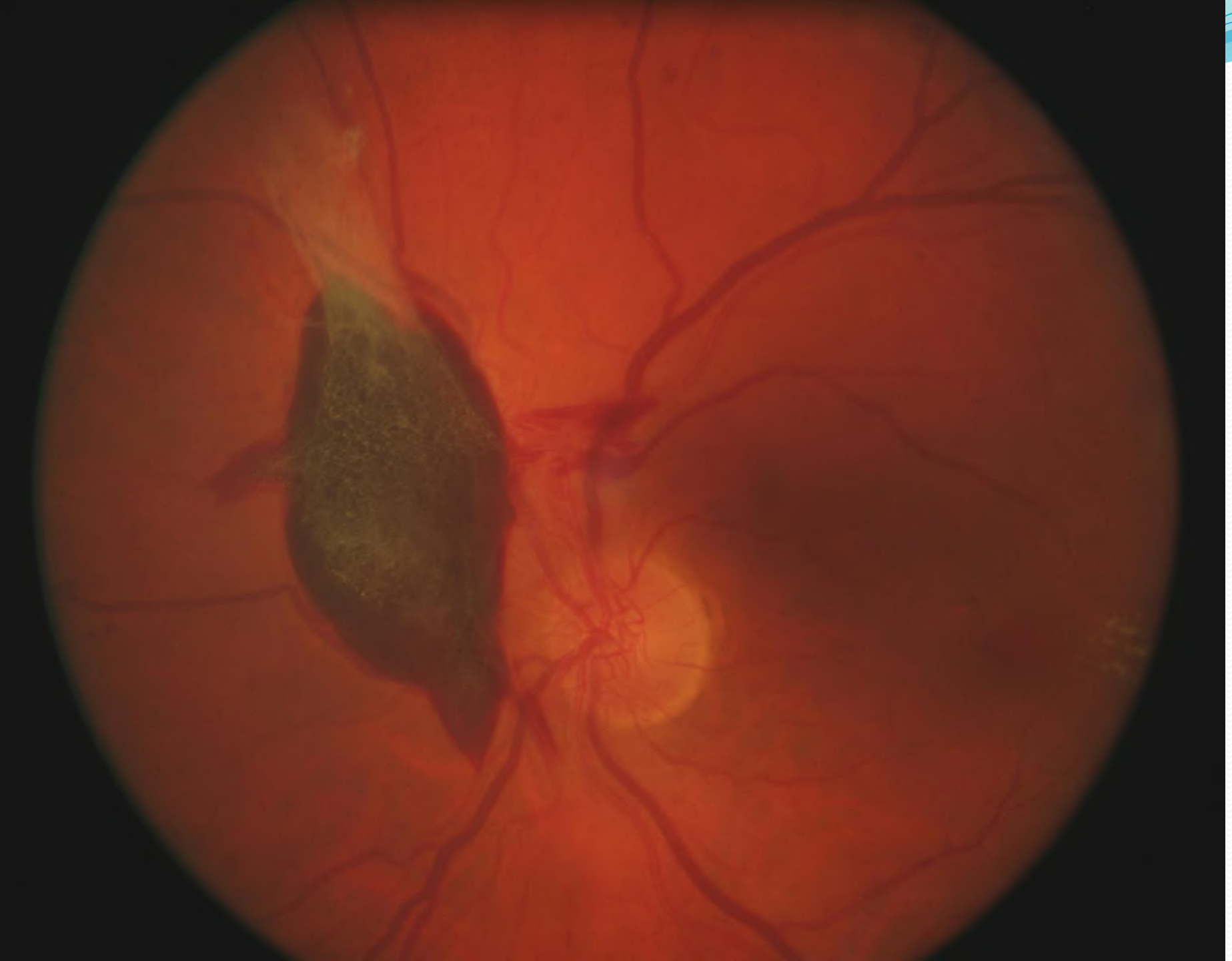

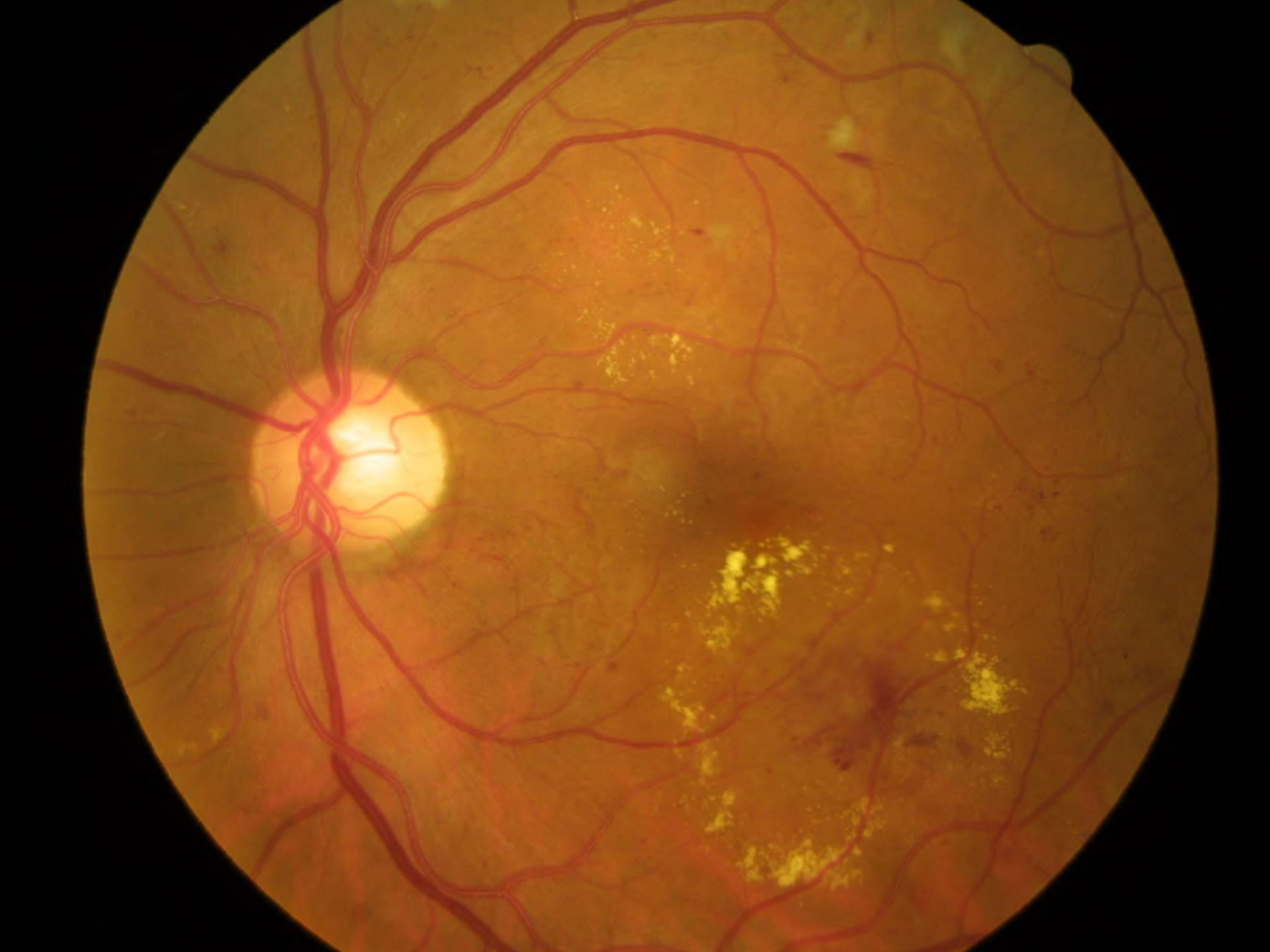

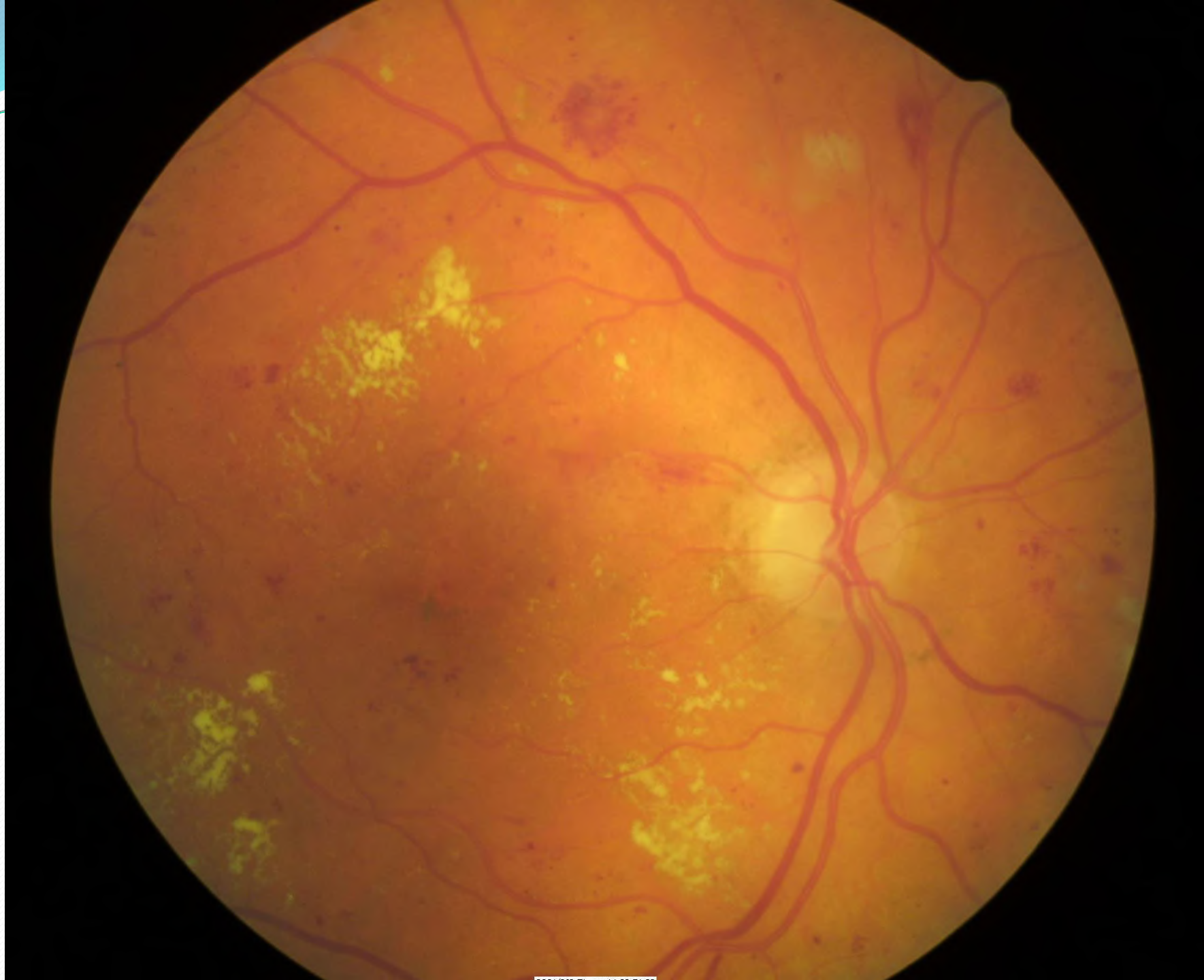

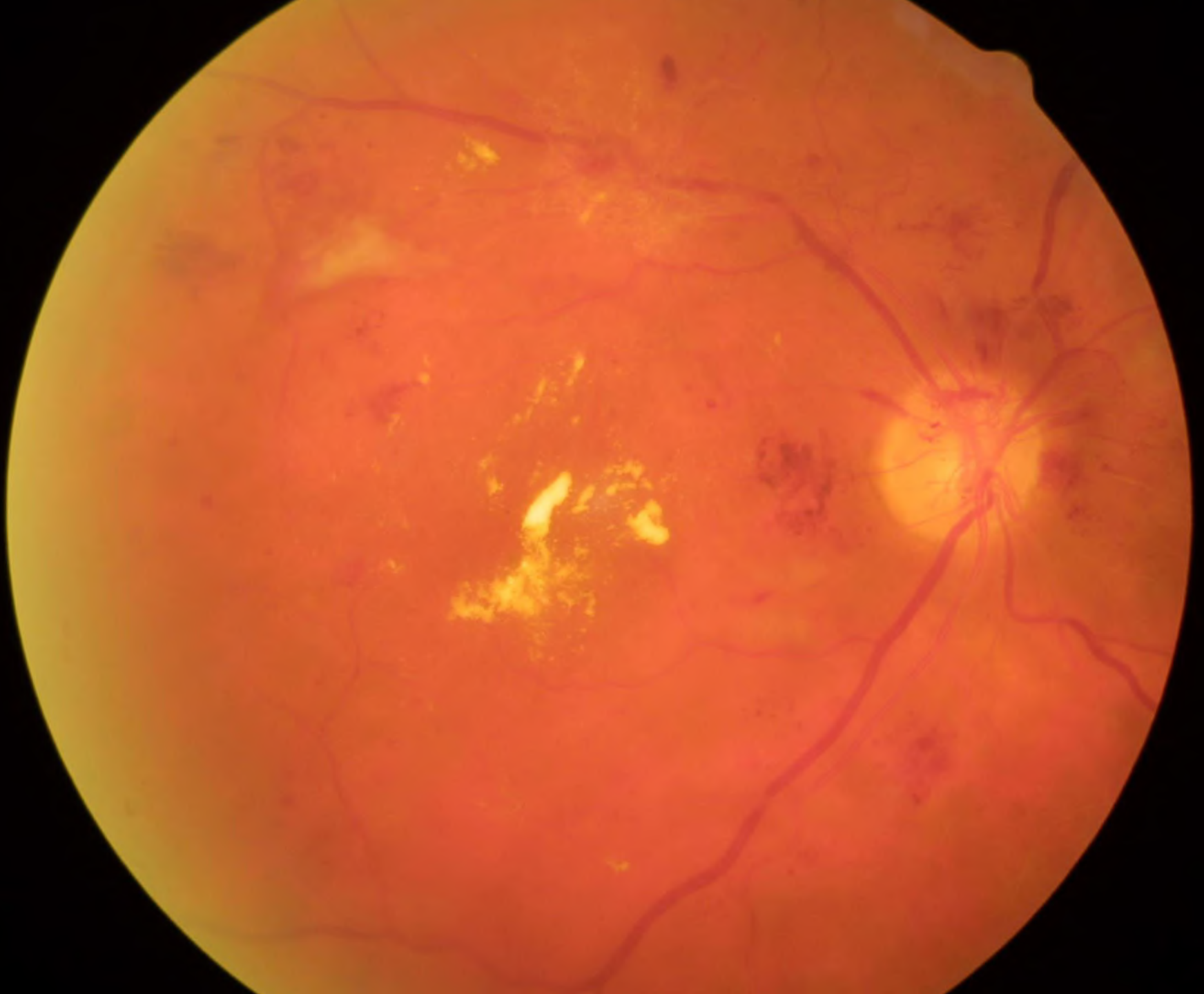

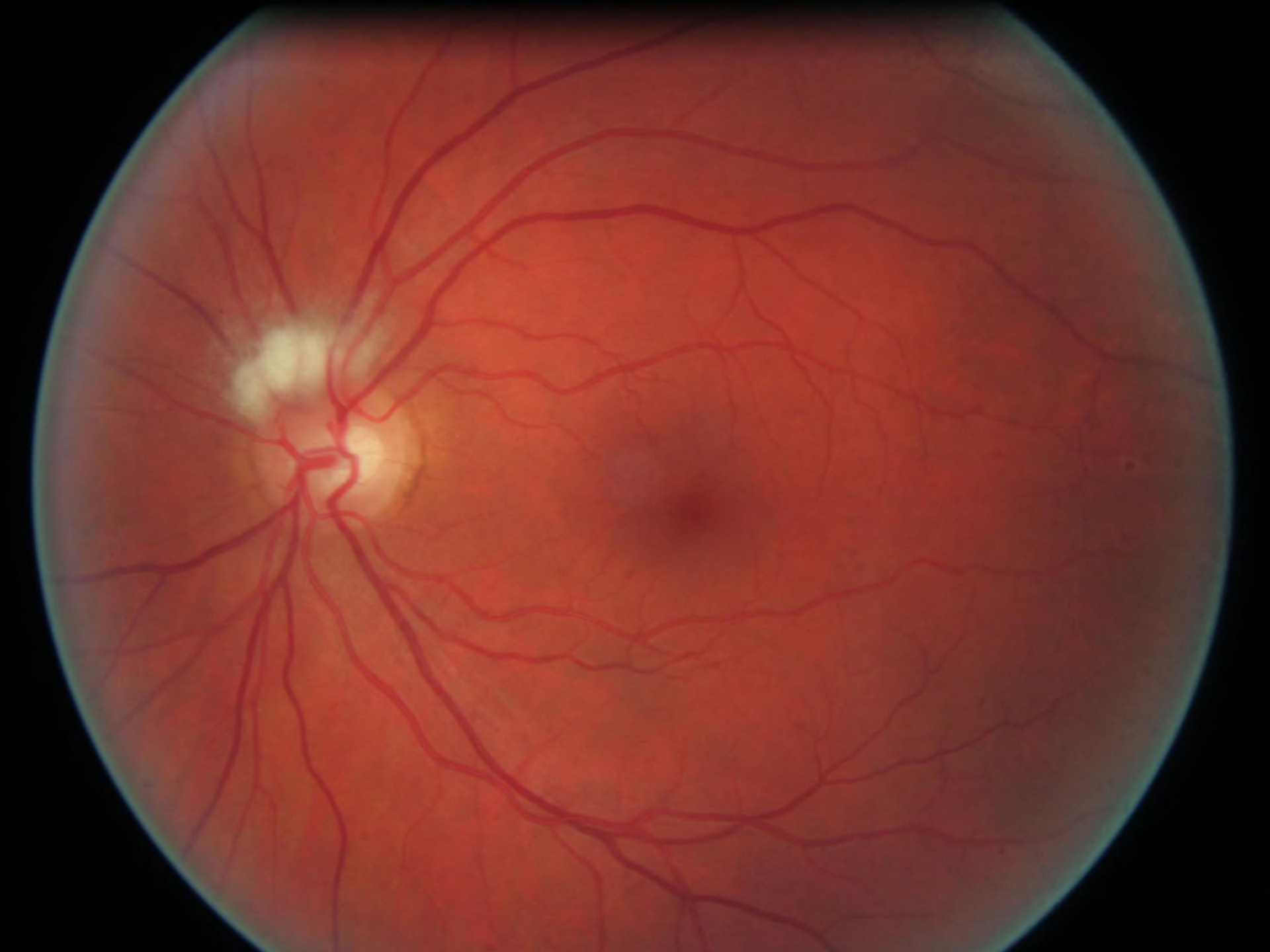

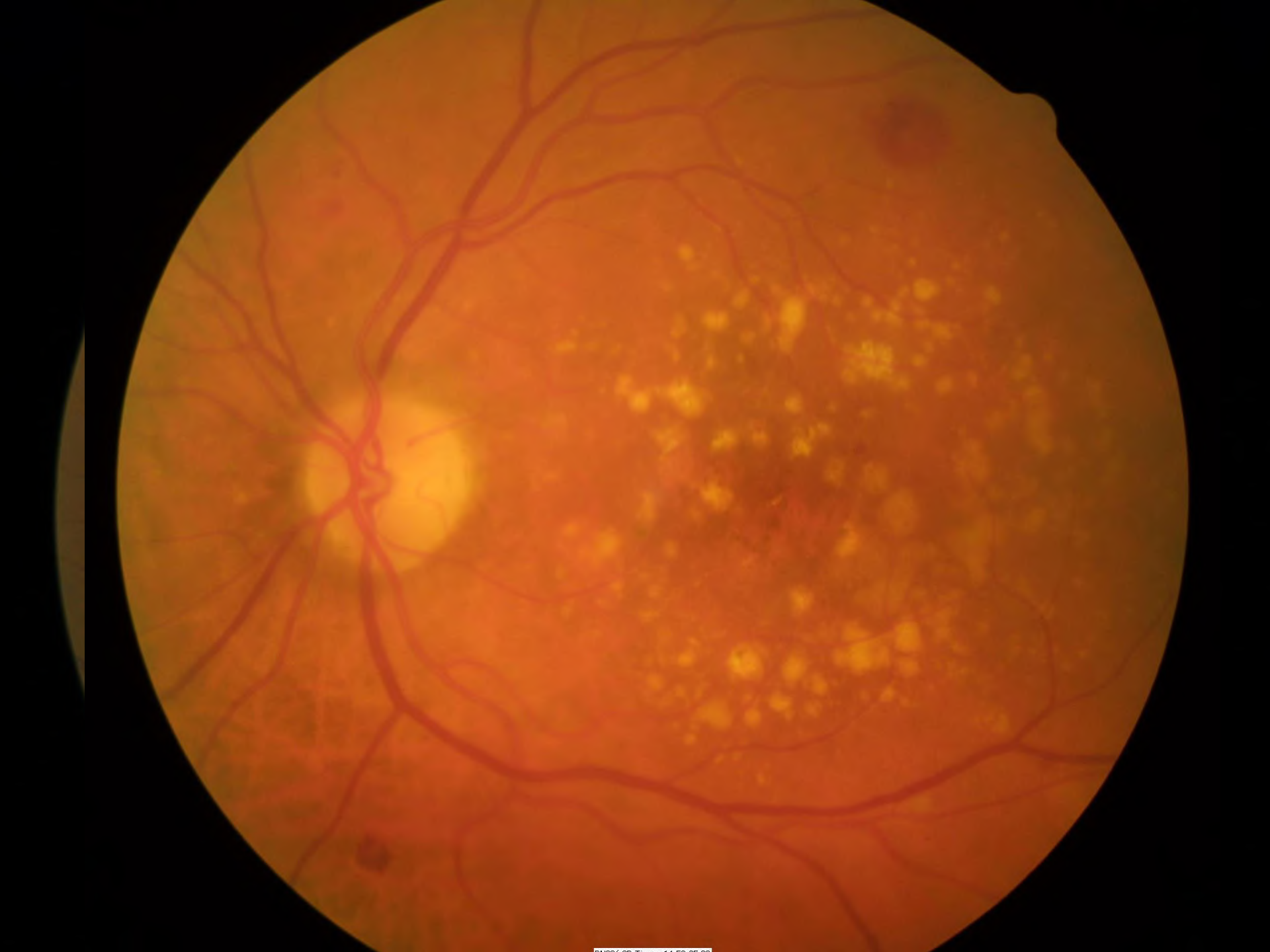

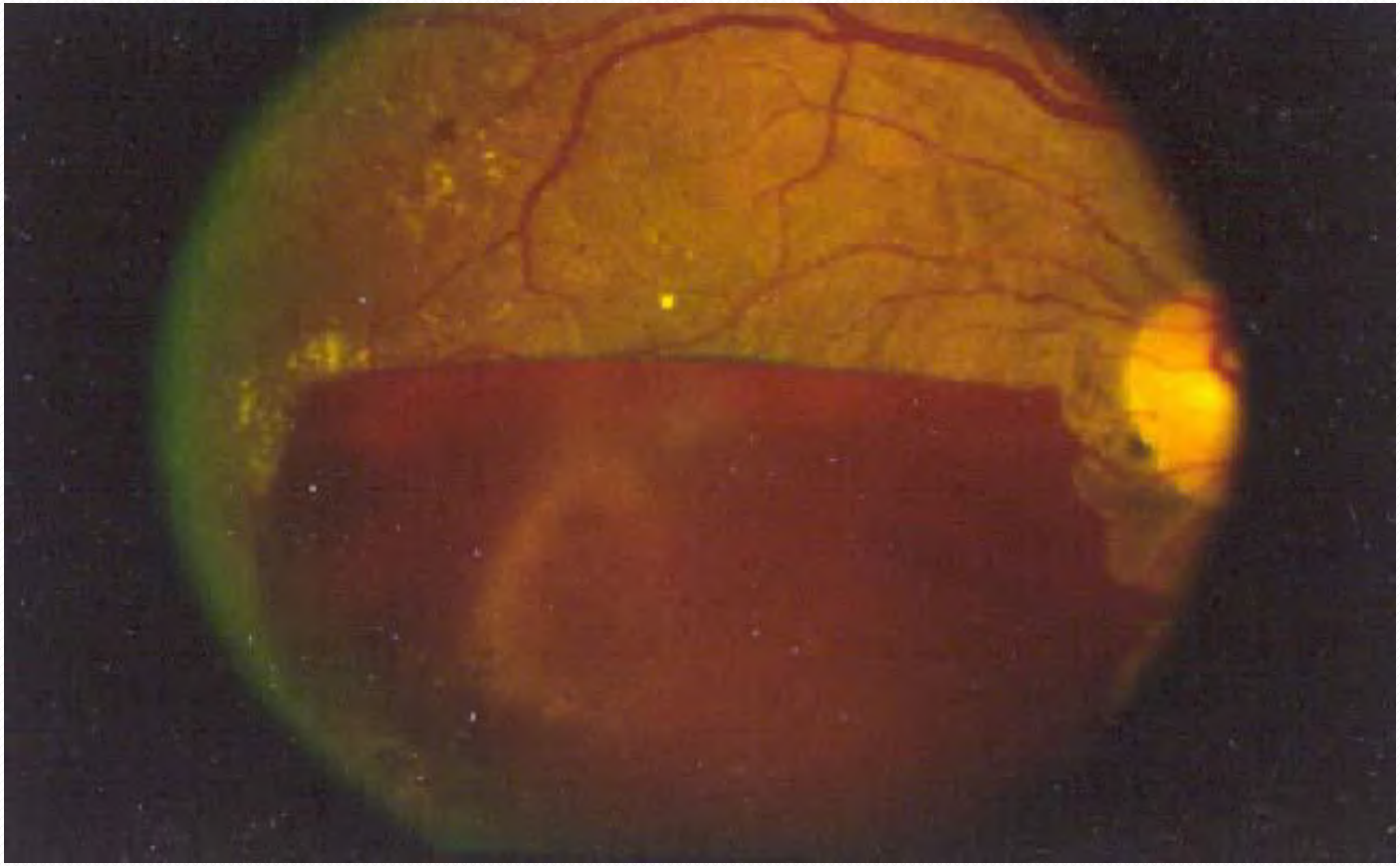

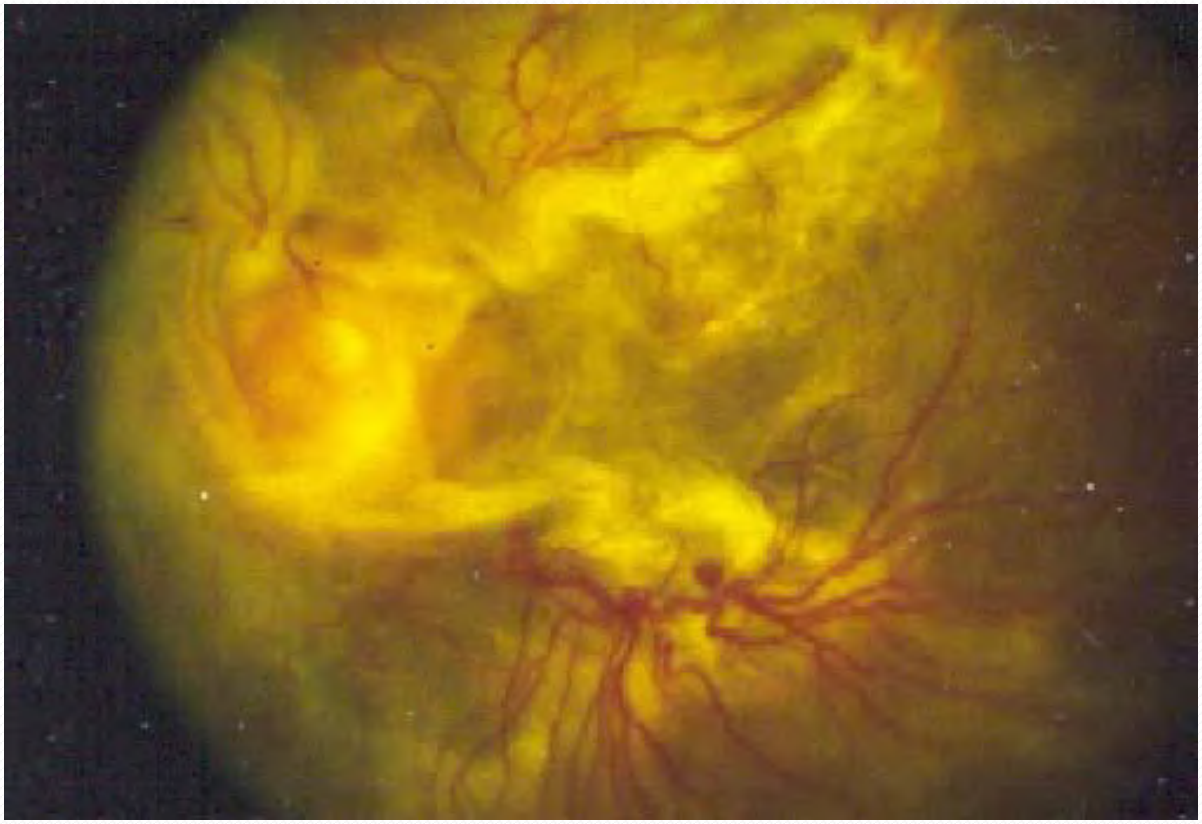

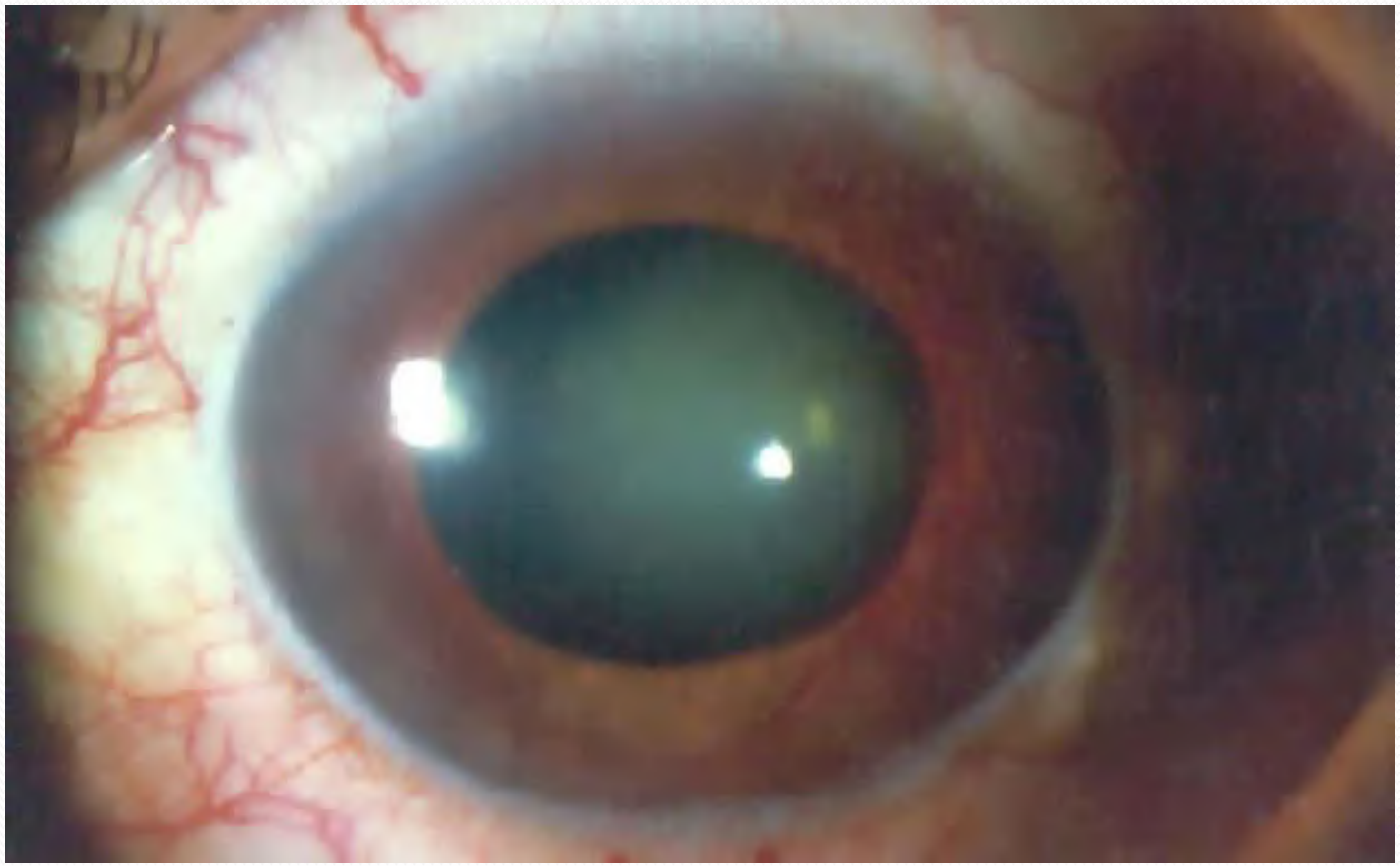

THANKS

Central Retinal Arterial Occlusions

PROF. DR. SANAULLAH JAN
FRCS (Edinburgh, UK), FRCS (Glasgow, UK), FCPS (Pak)
Fellowship in Vitreo-Retina (Germany & India)

Director
Pakistan Institute of Community Ophthalmology
&
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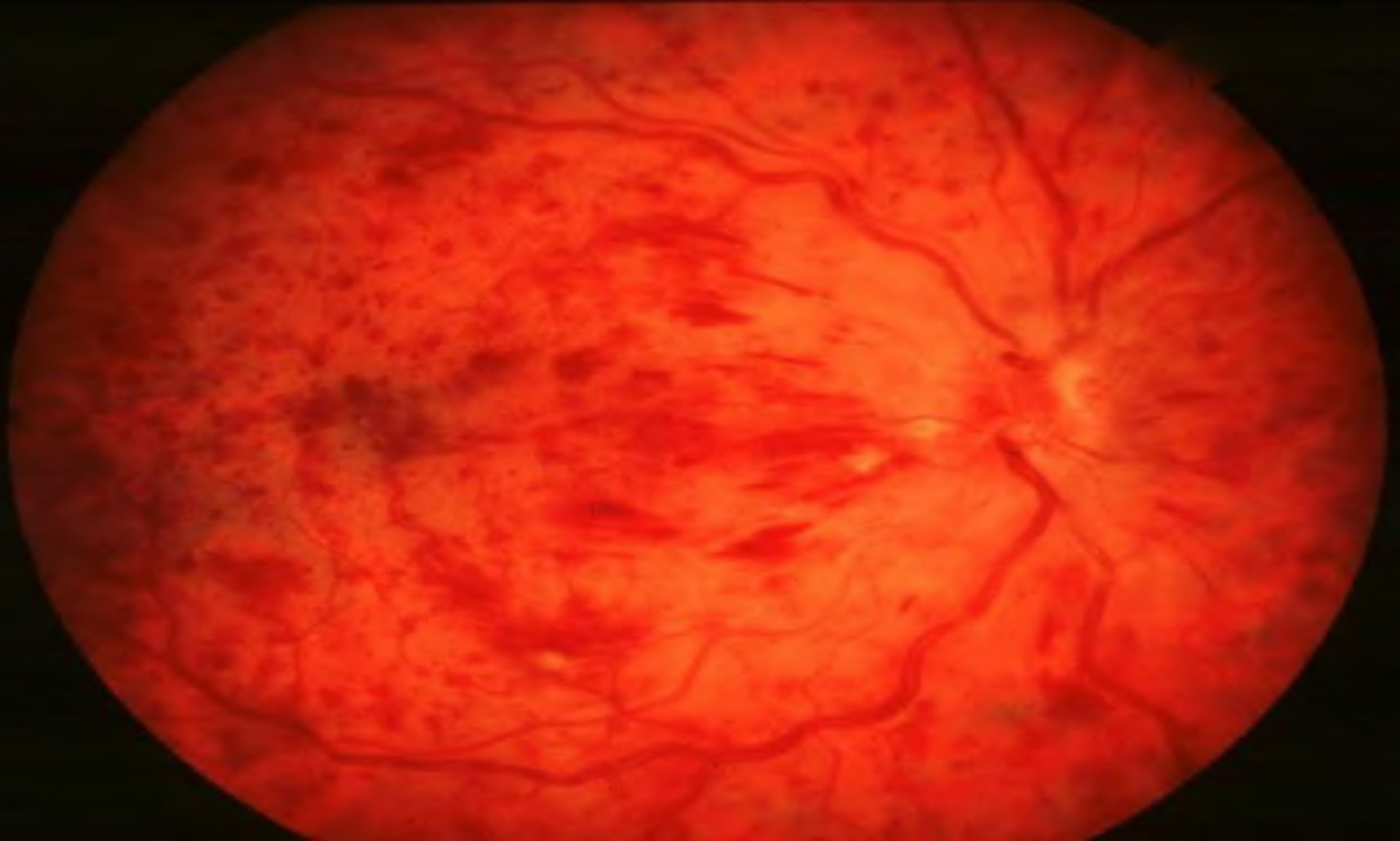
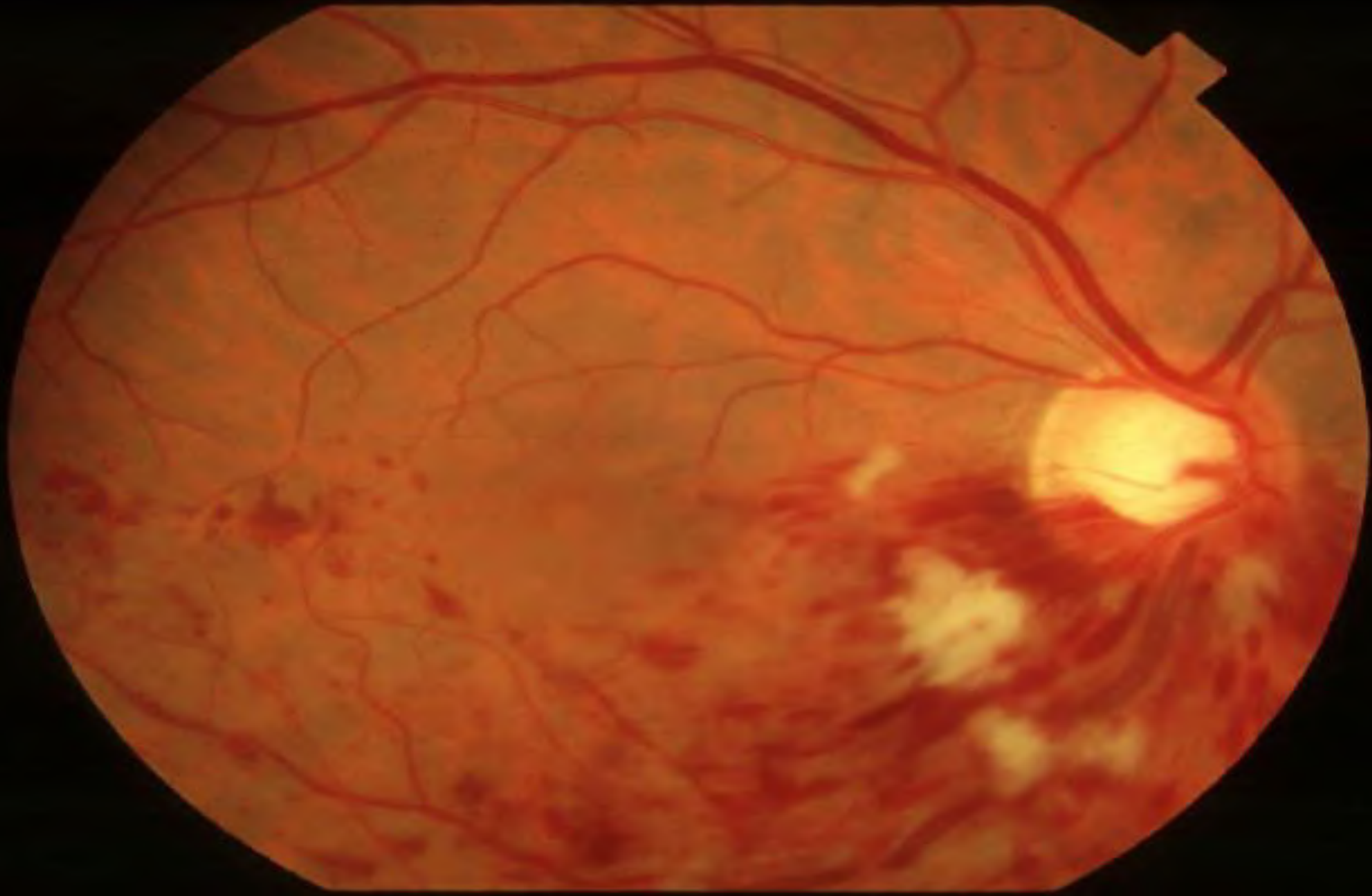
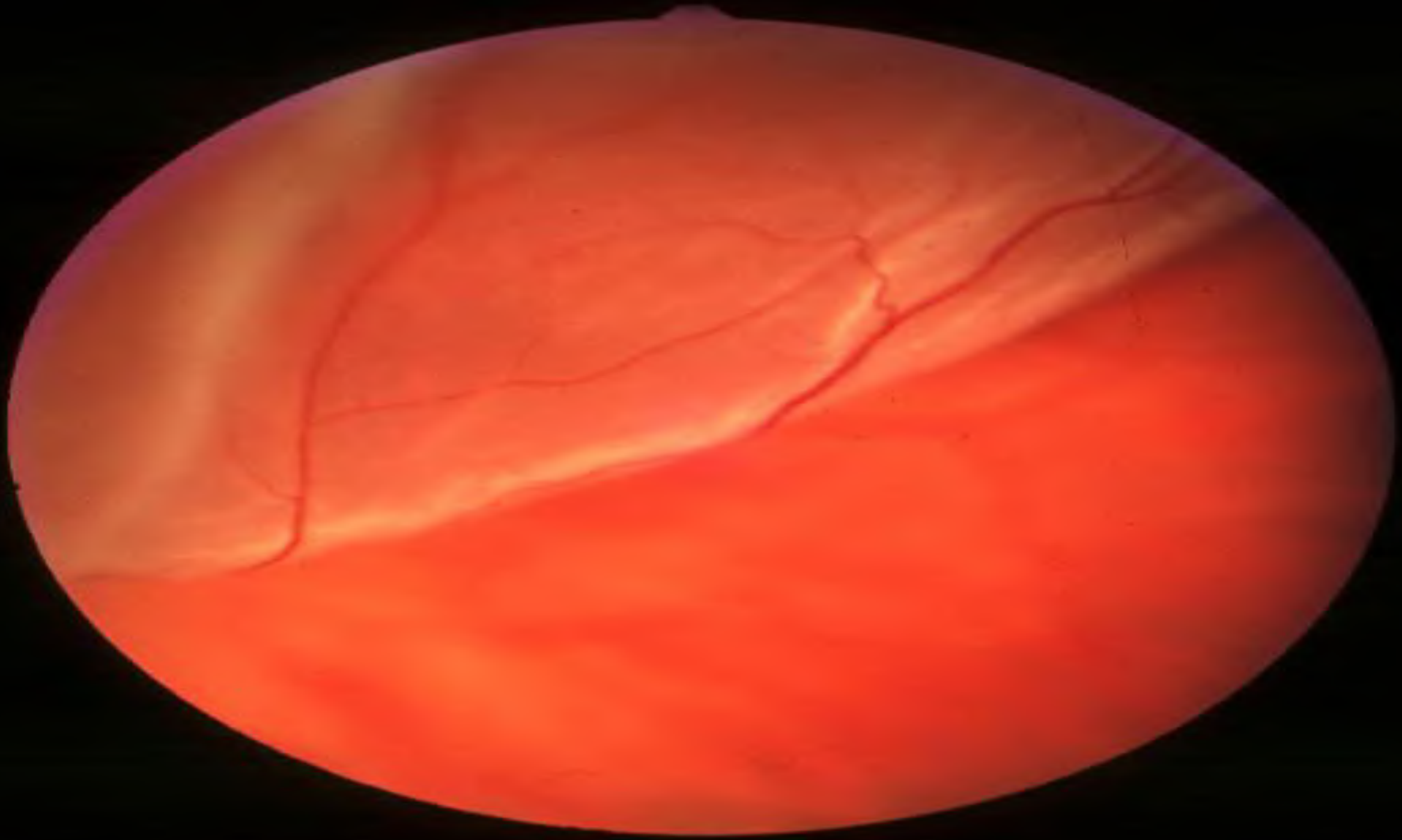
Hayatabad Medical Complex
Peshawar, Pakistan

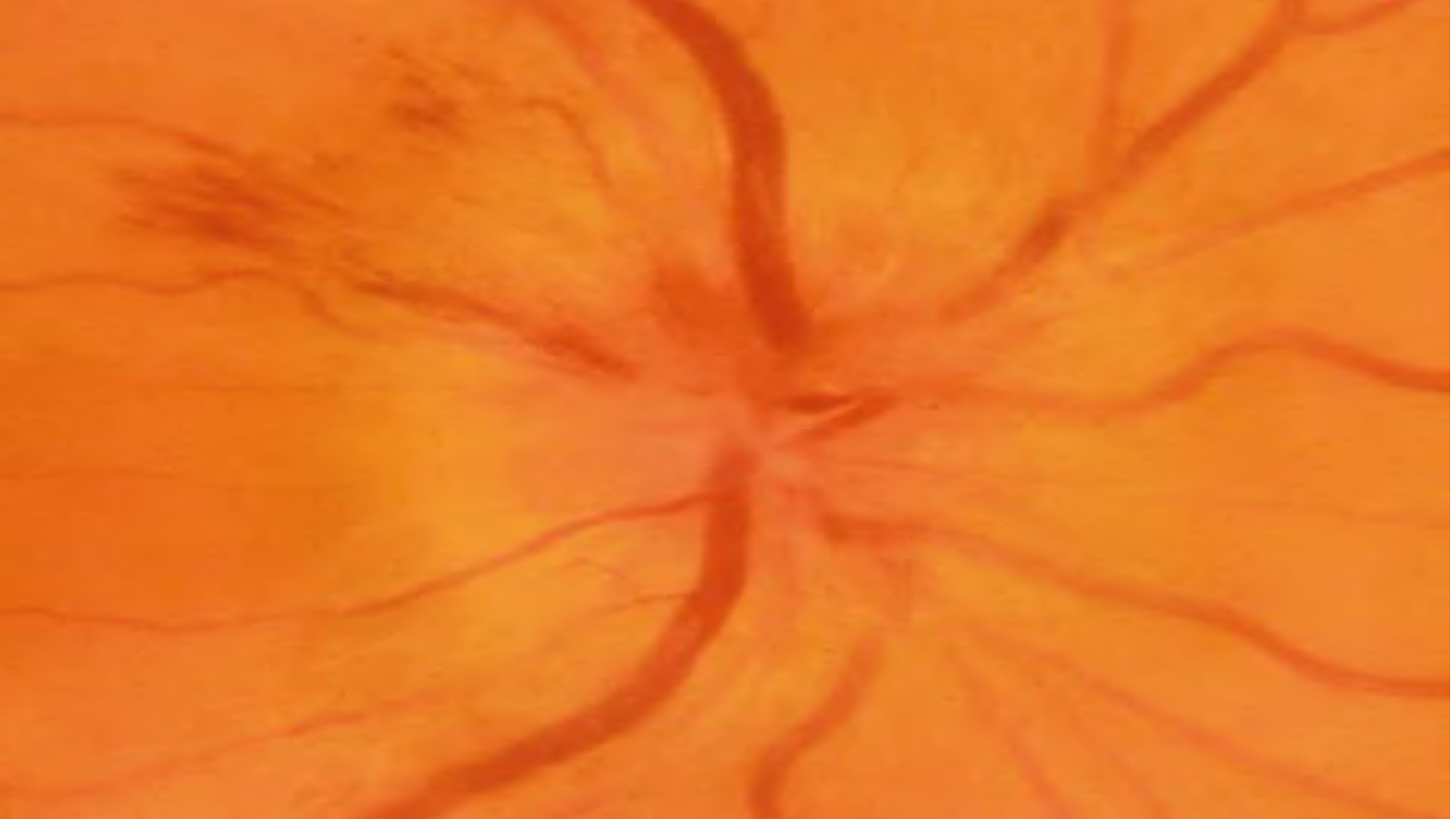
No financial disclosures

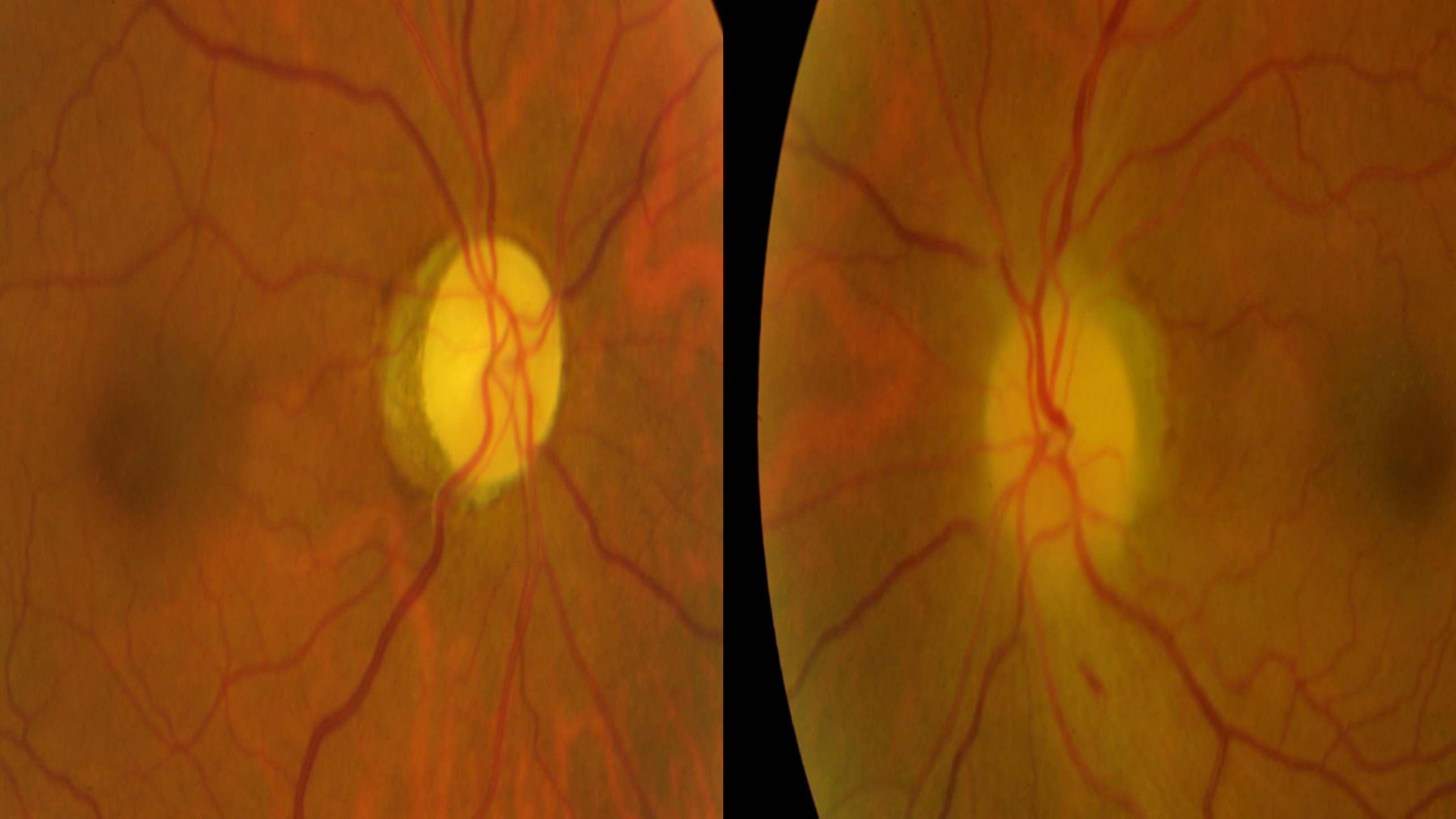
Most important people in Life

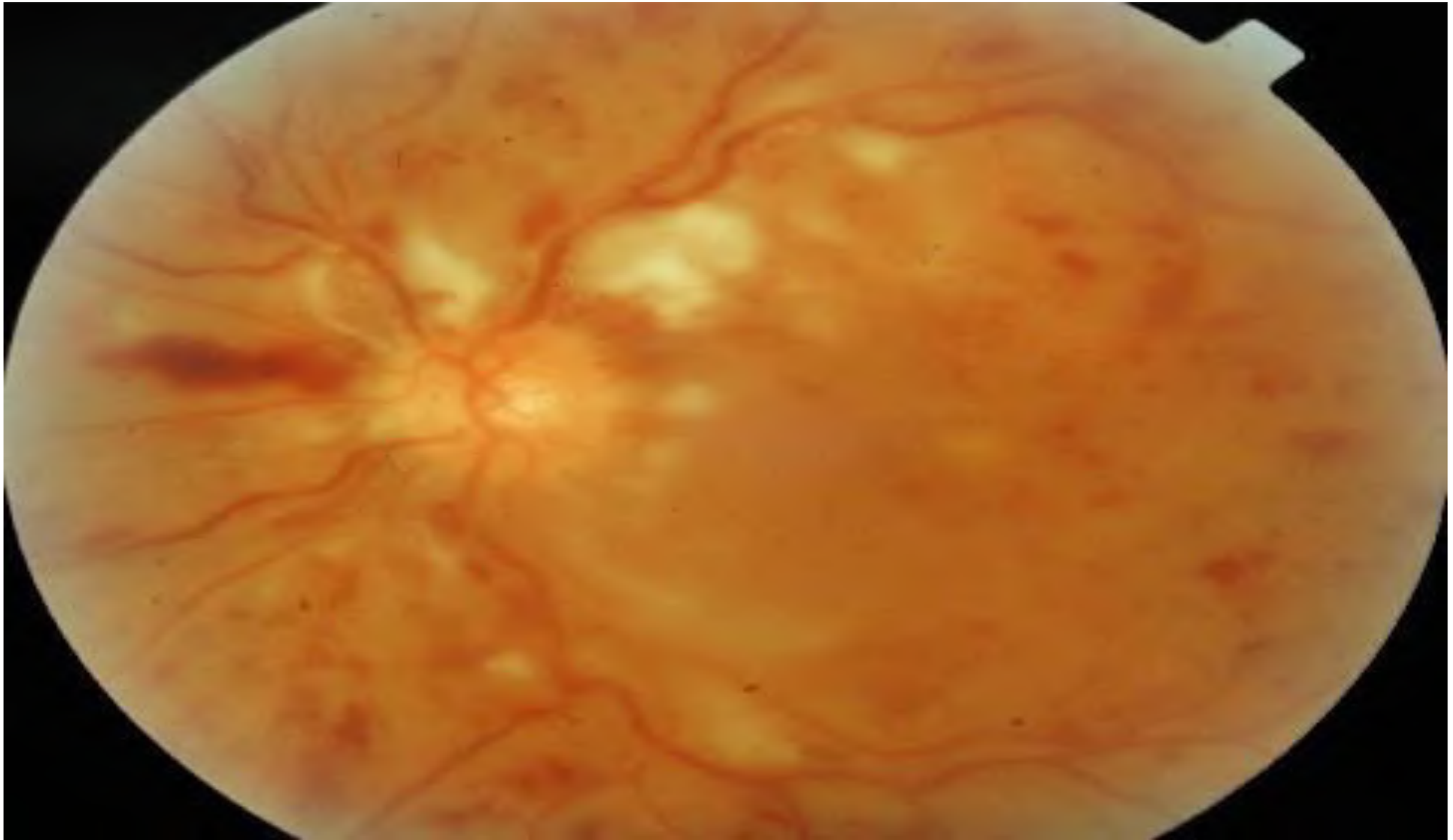


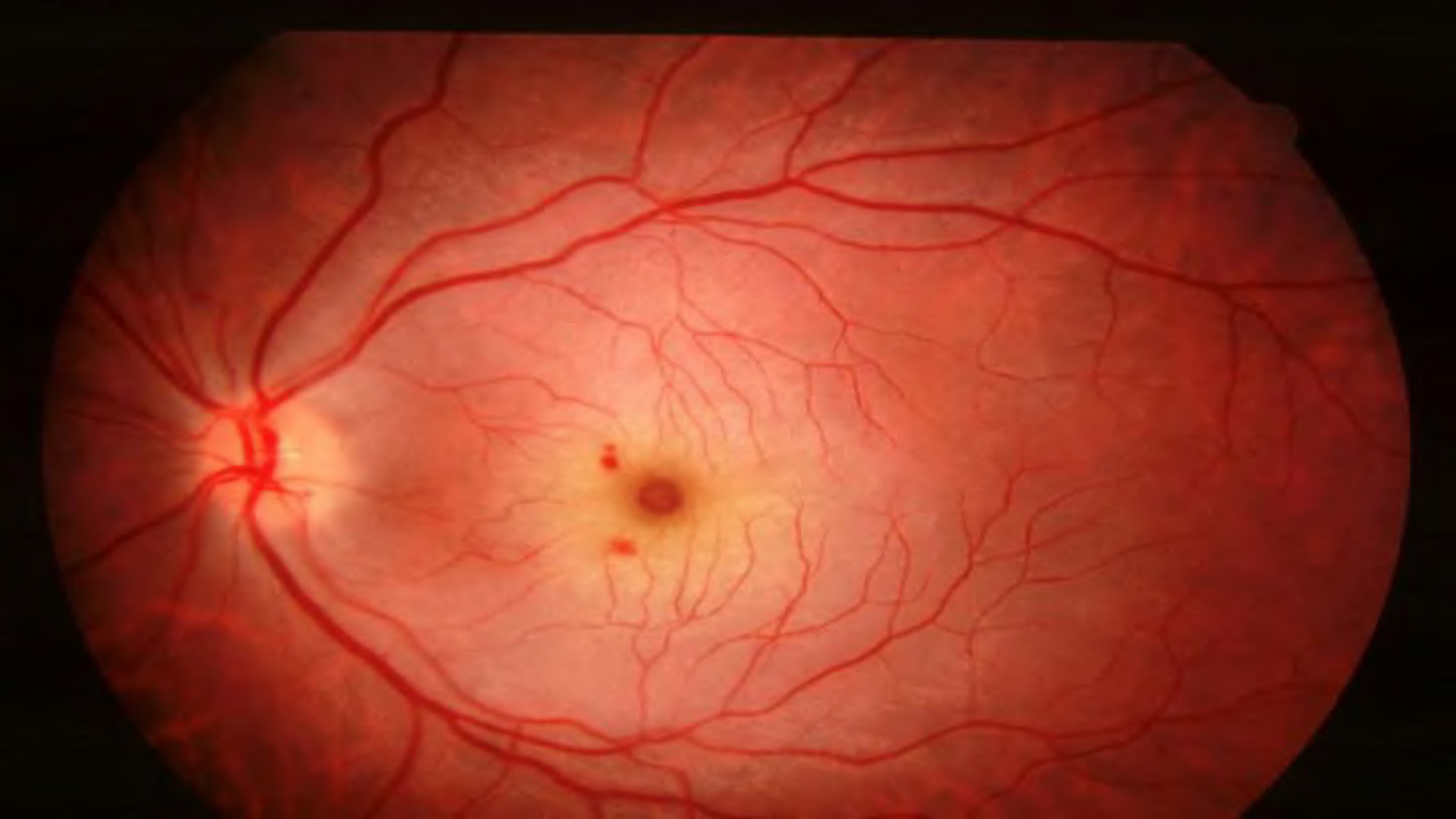


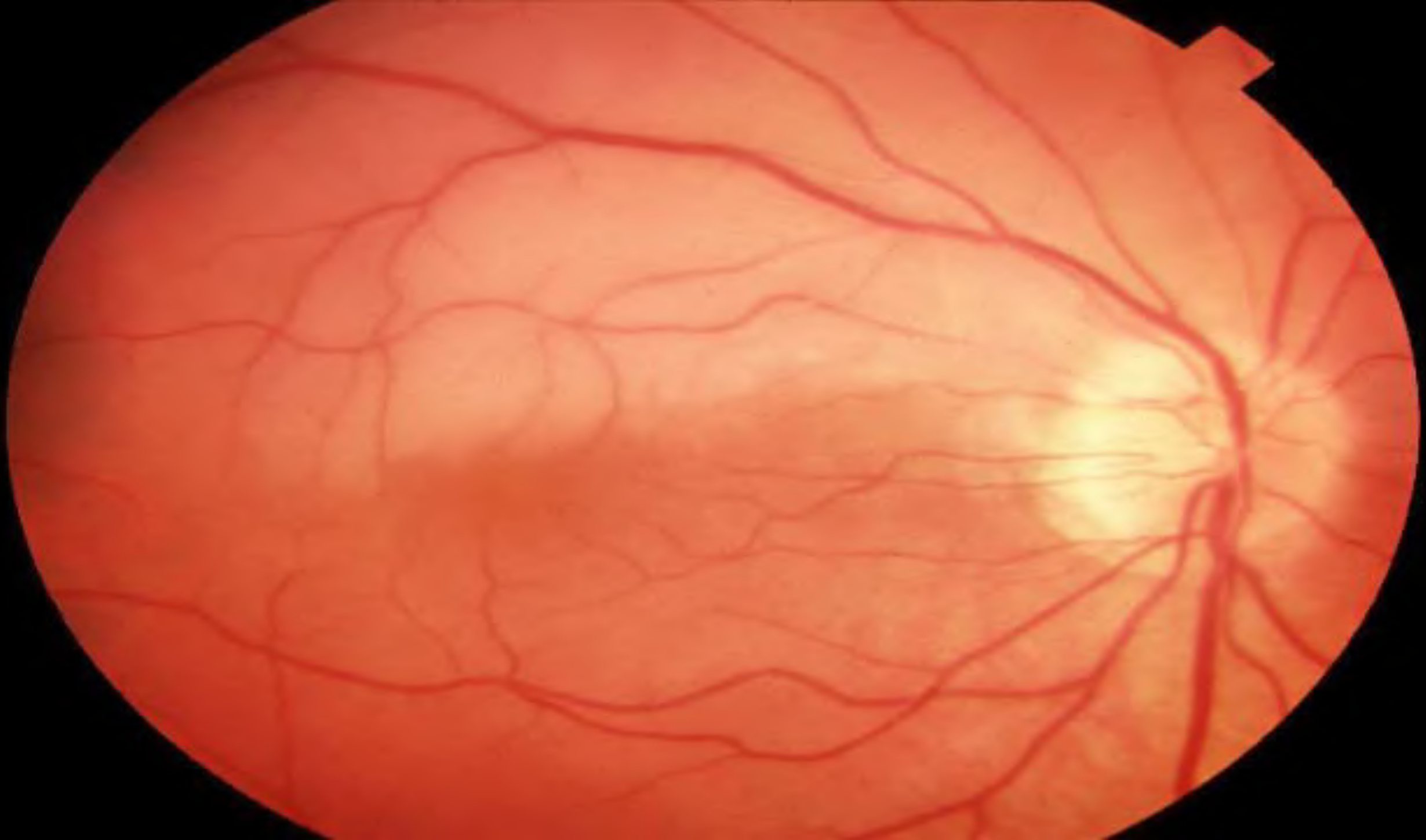




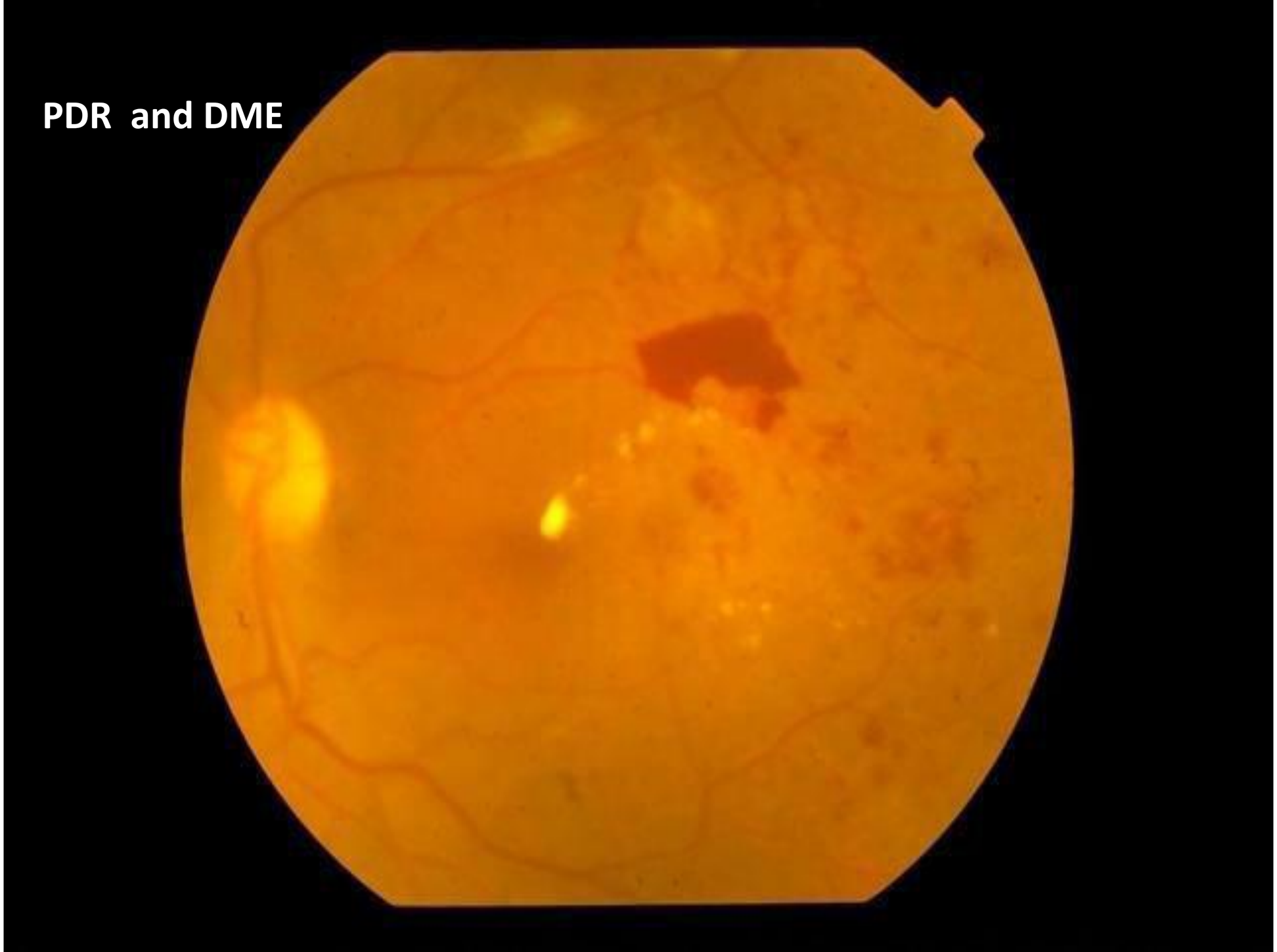








PDR and DME

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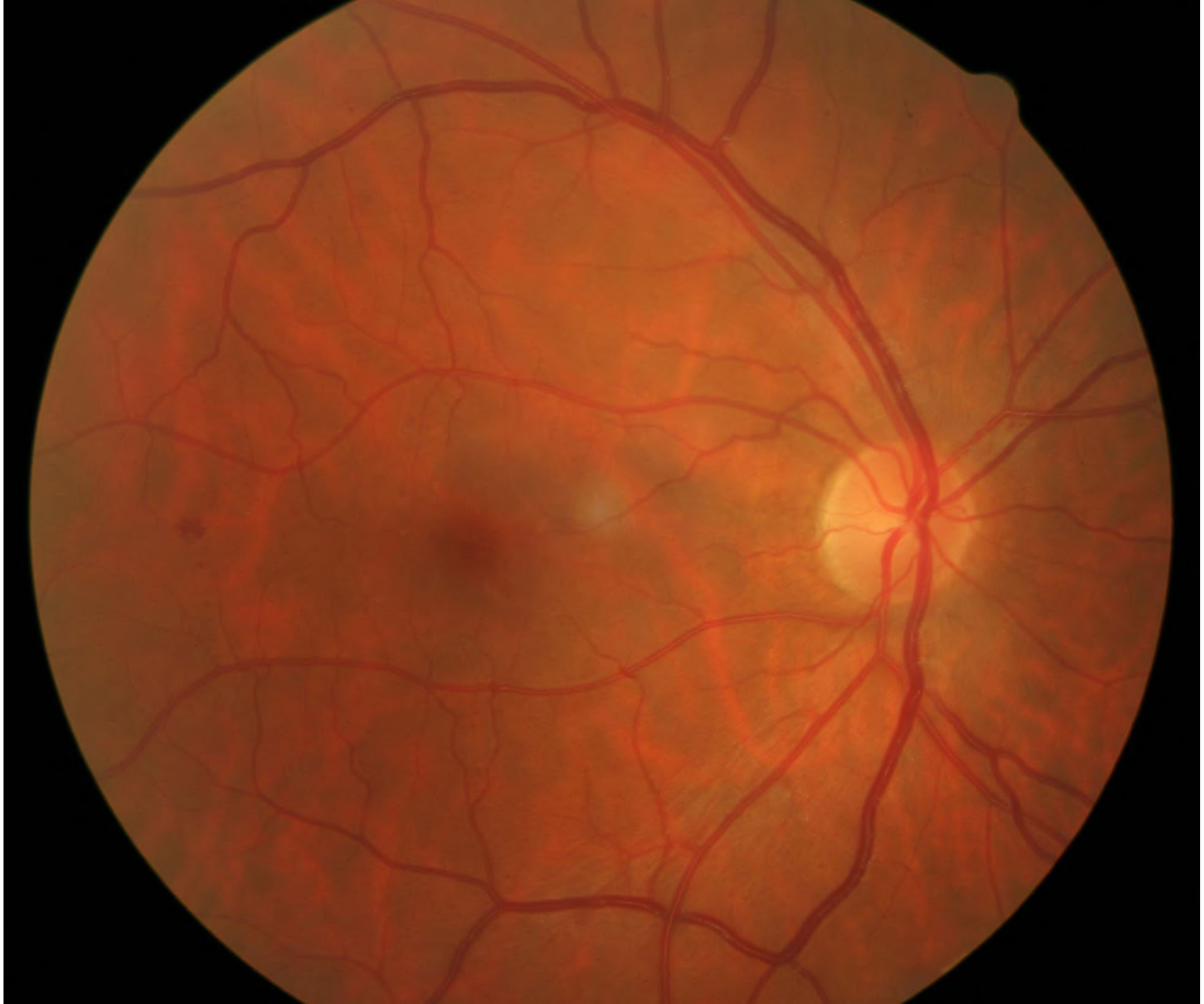

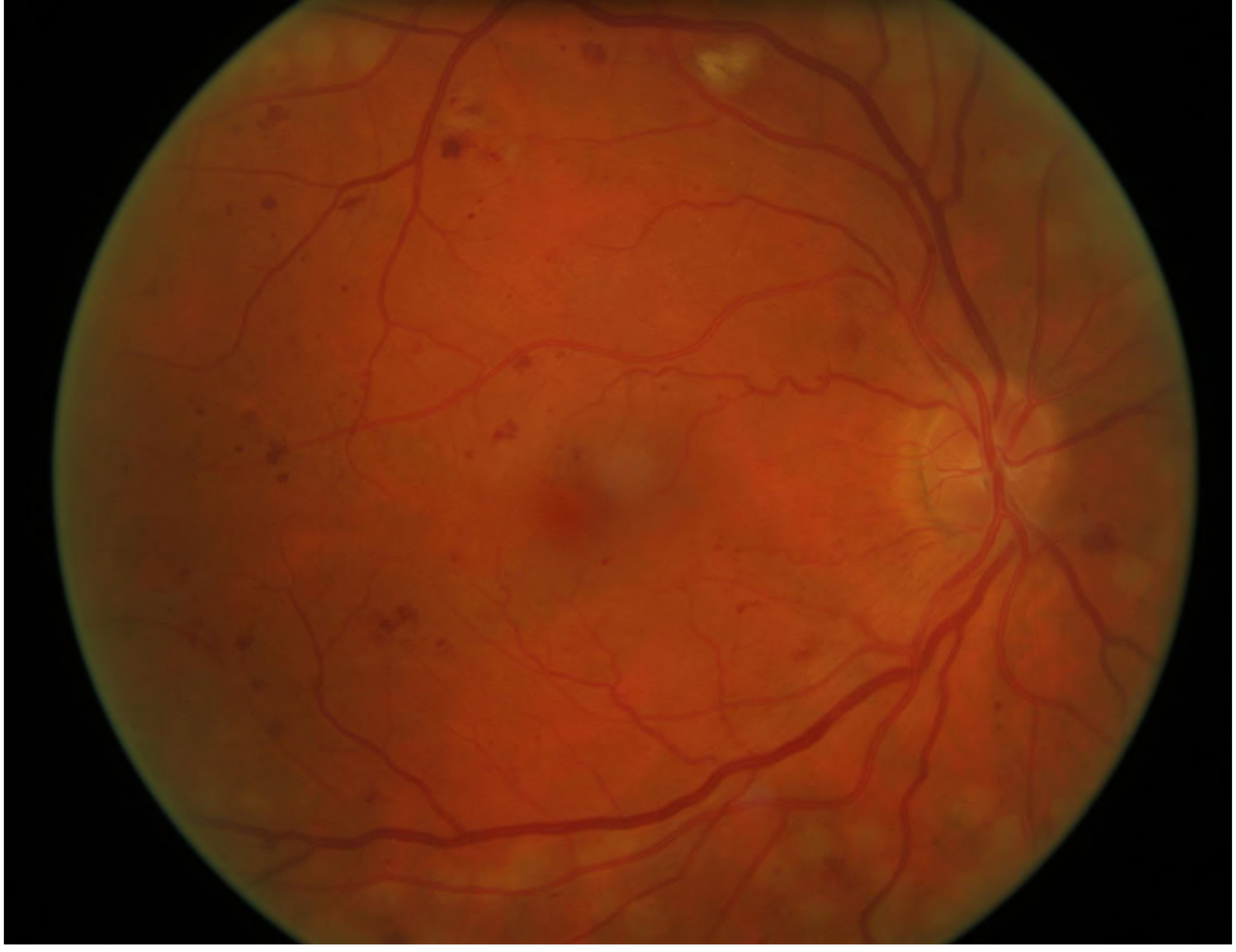

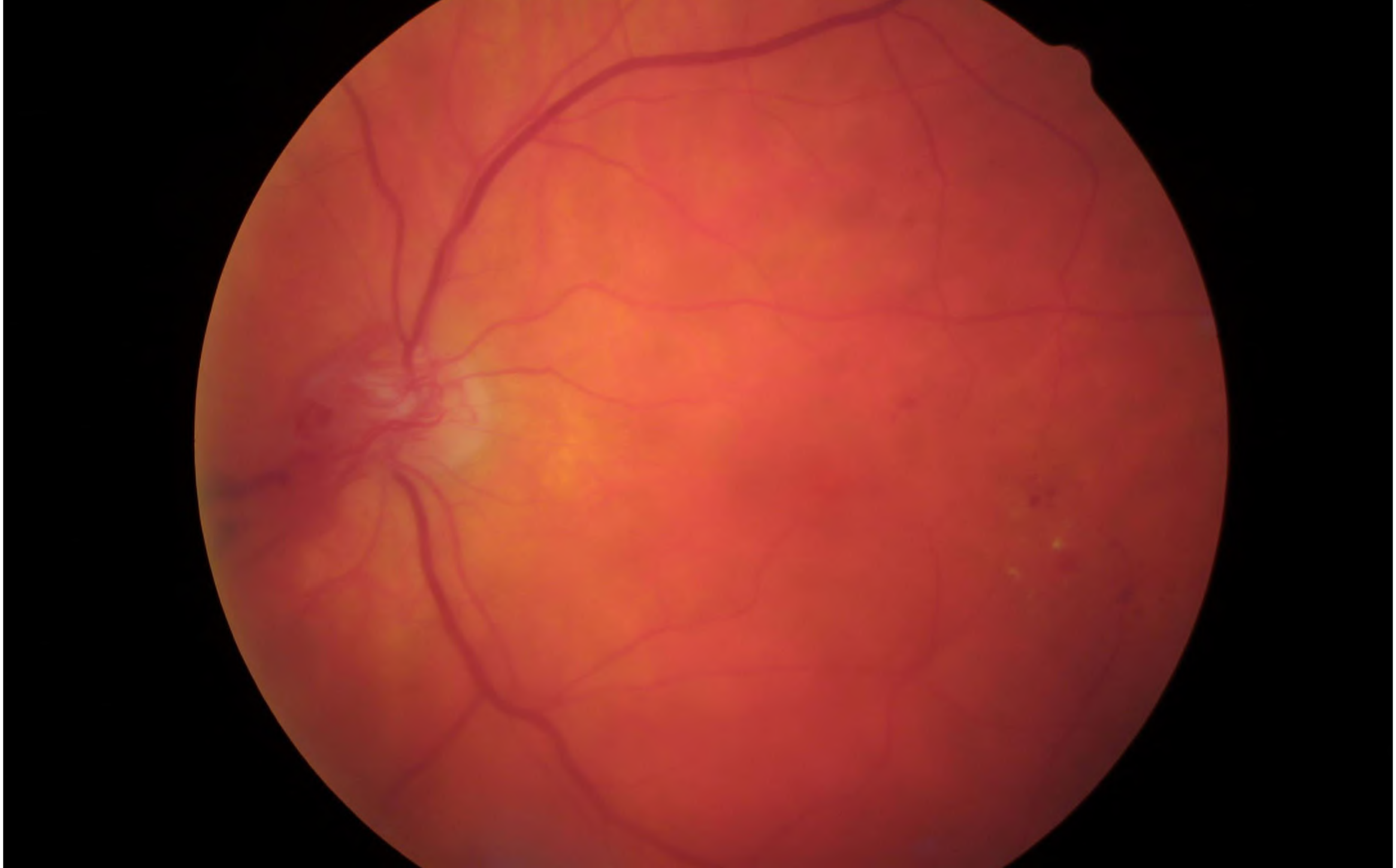

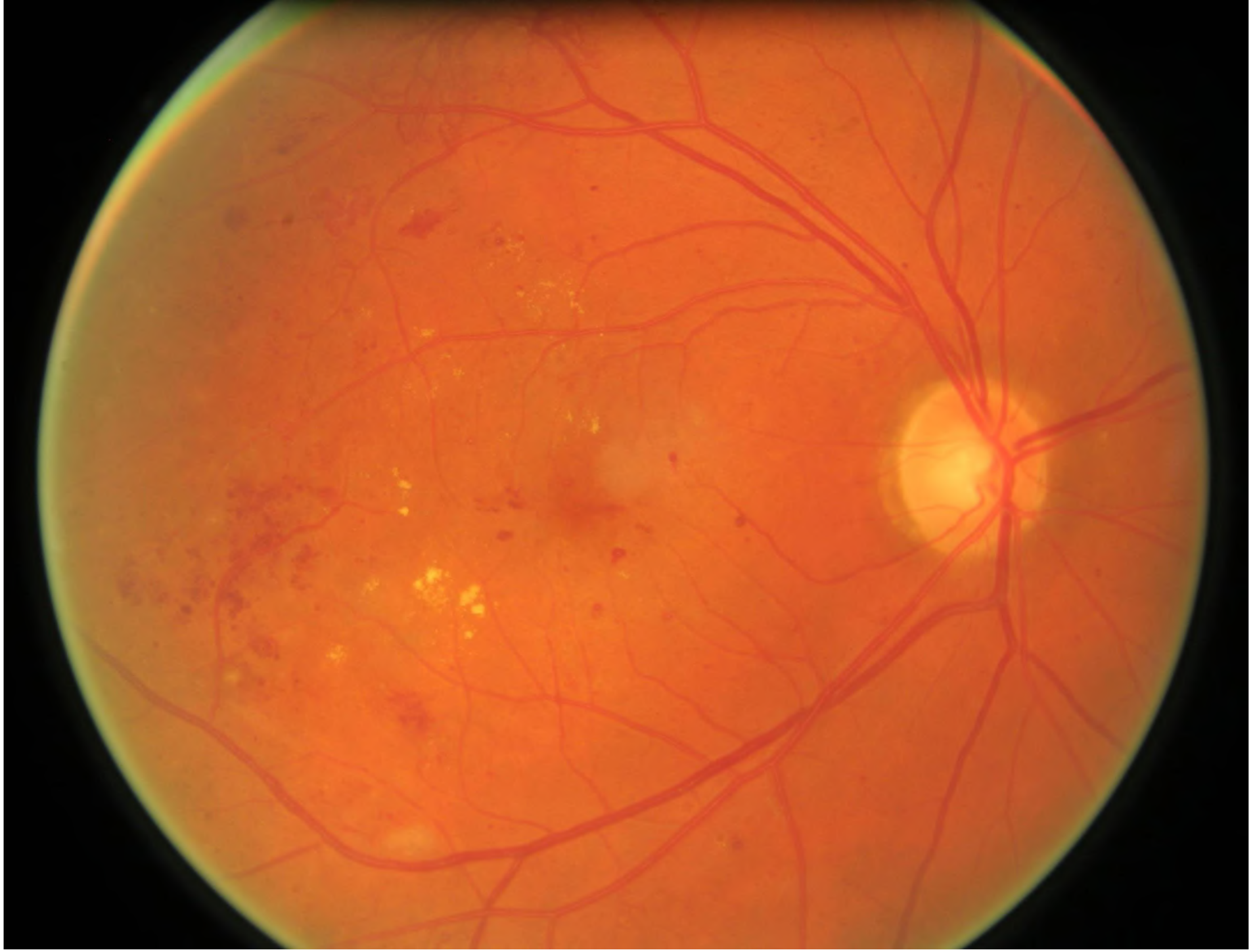

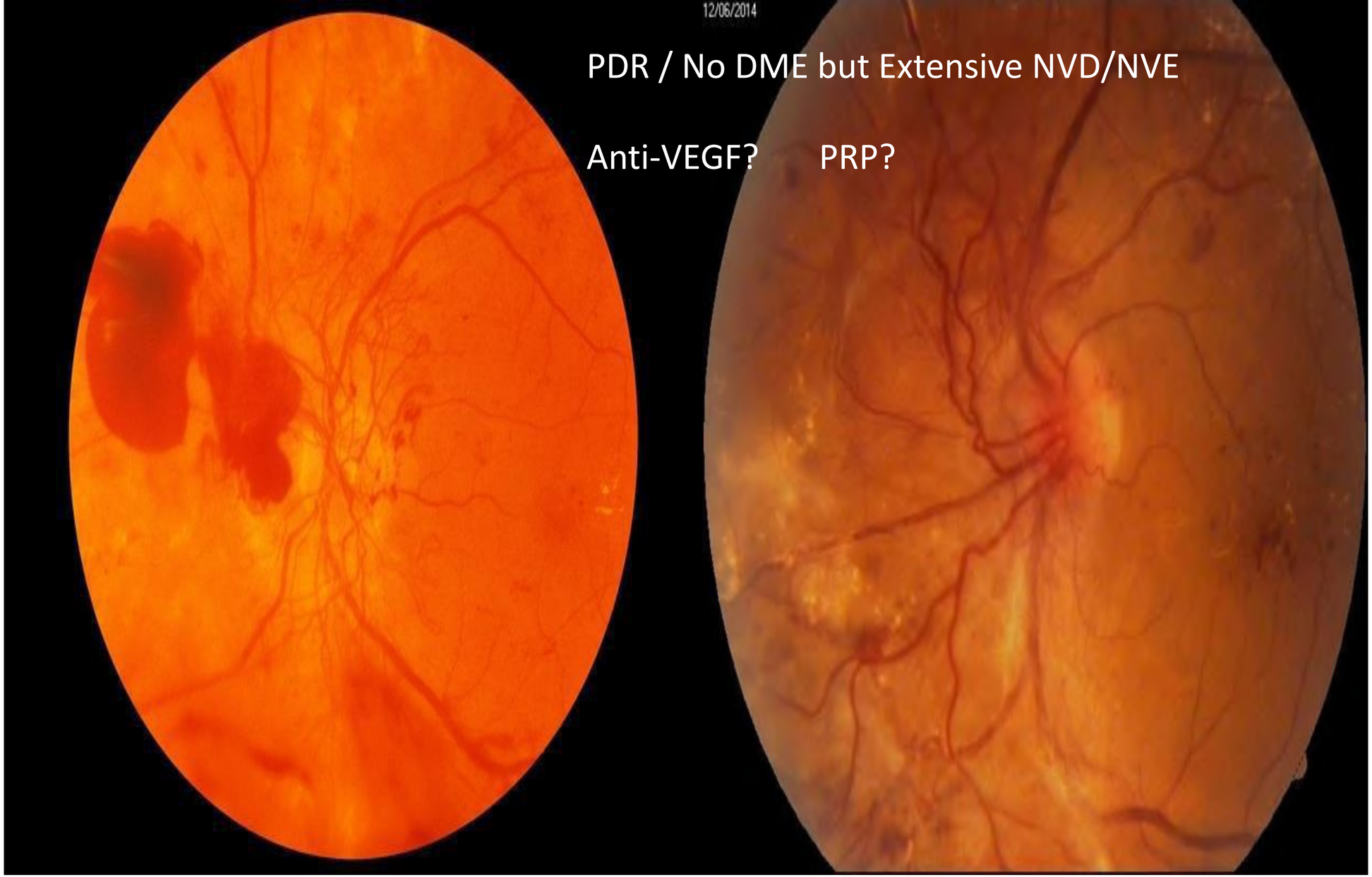

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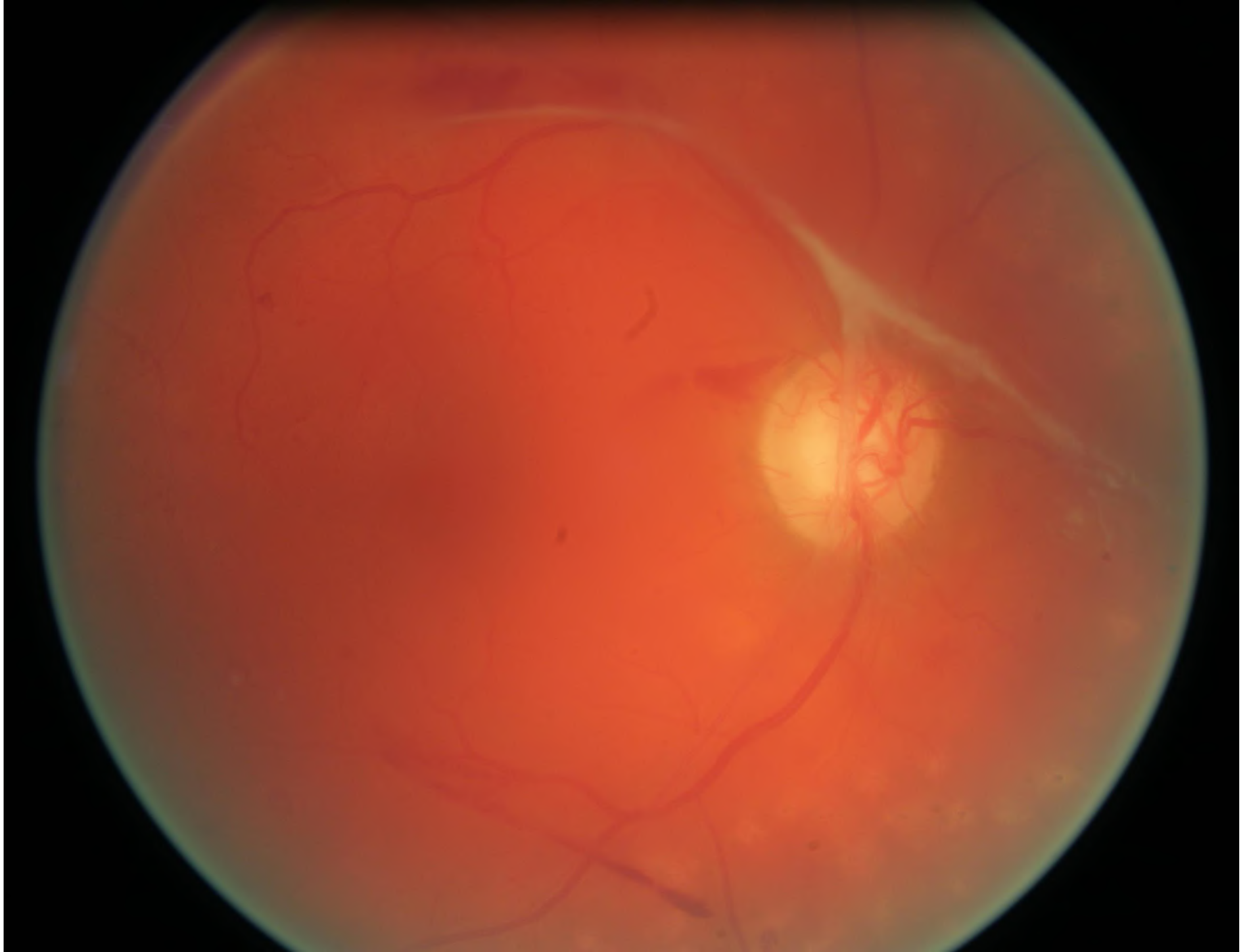
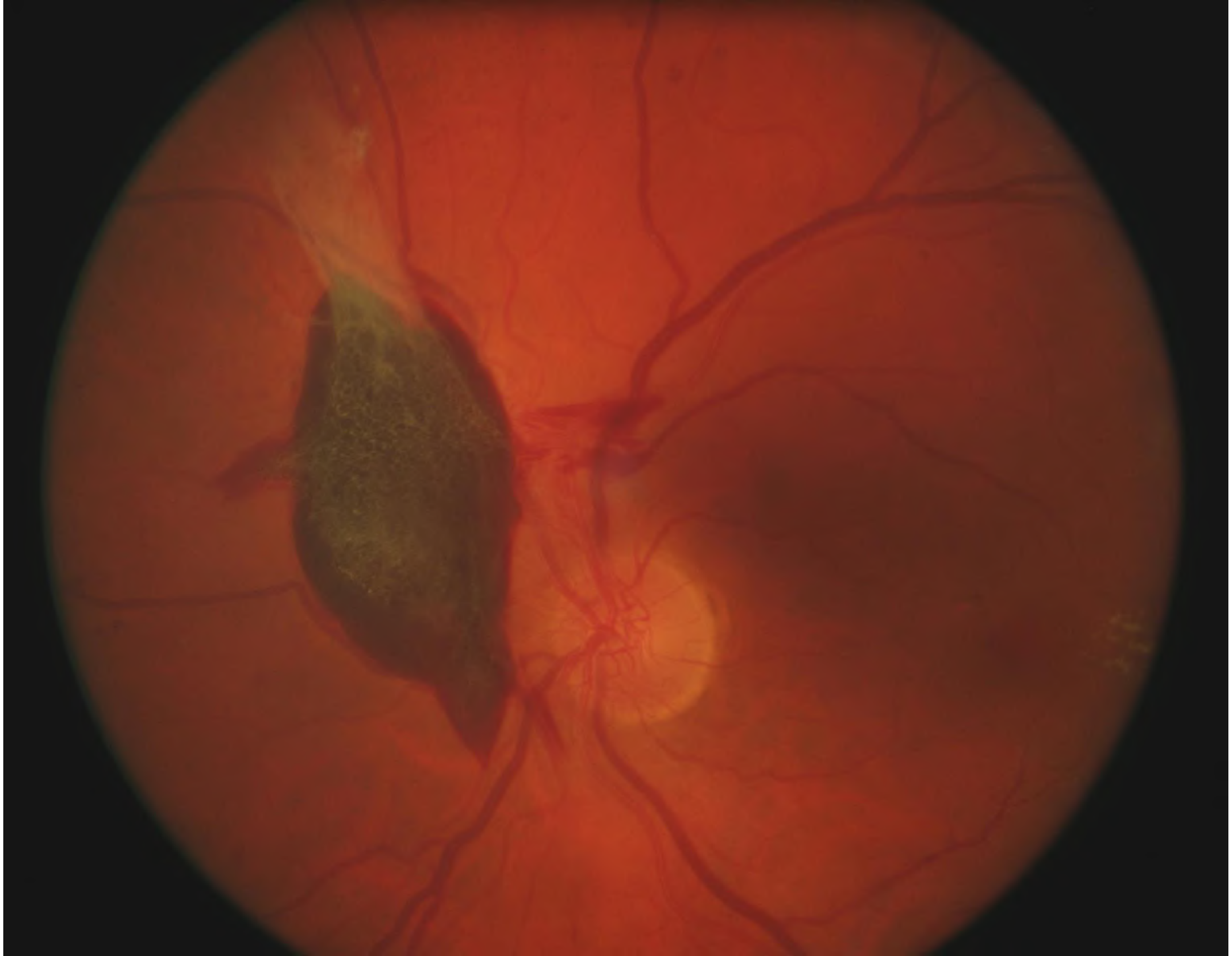
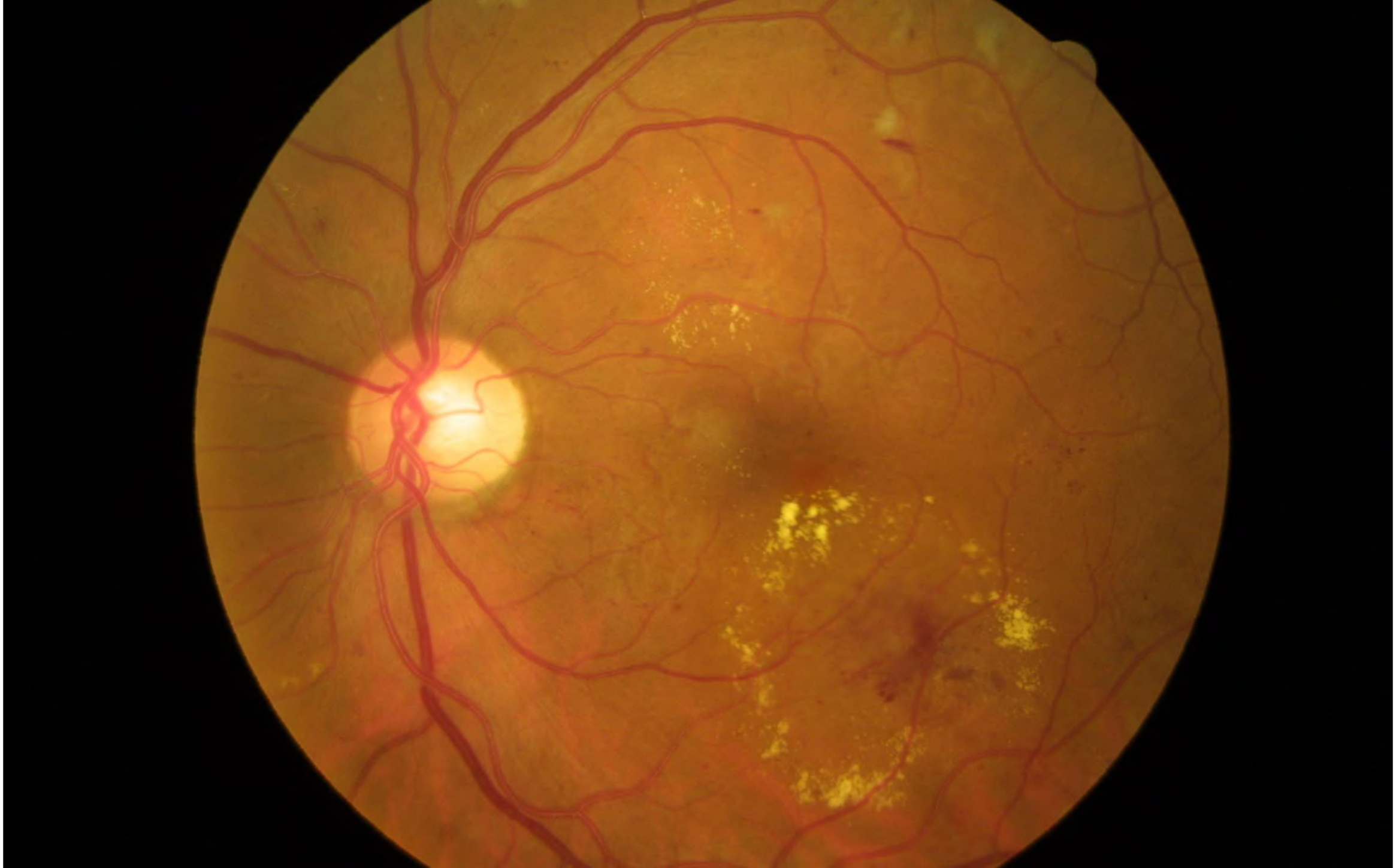
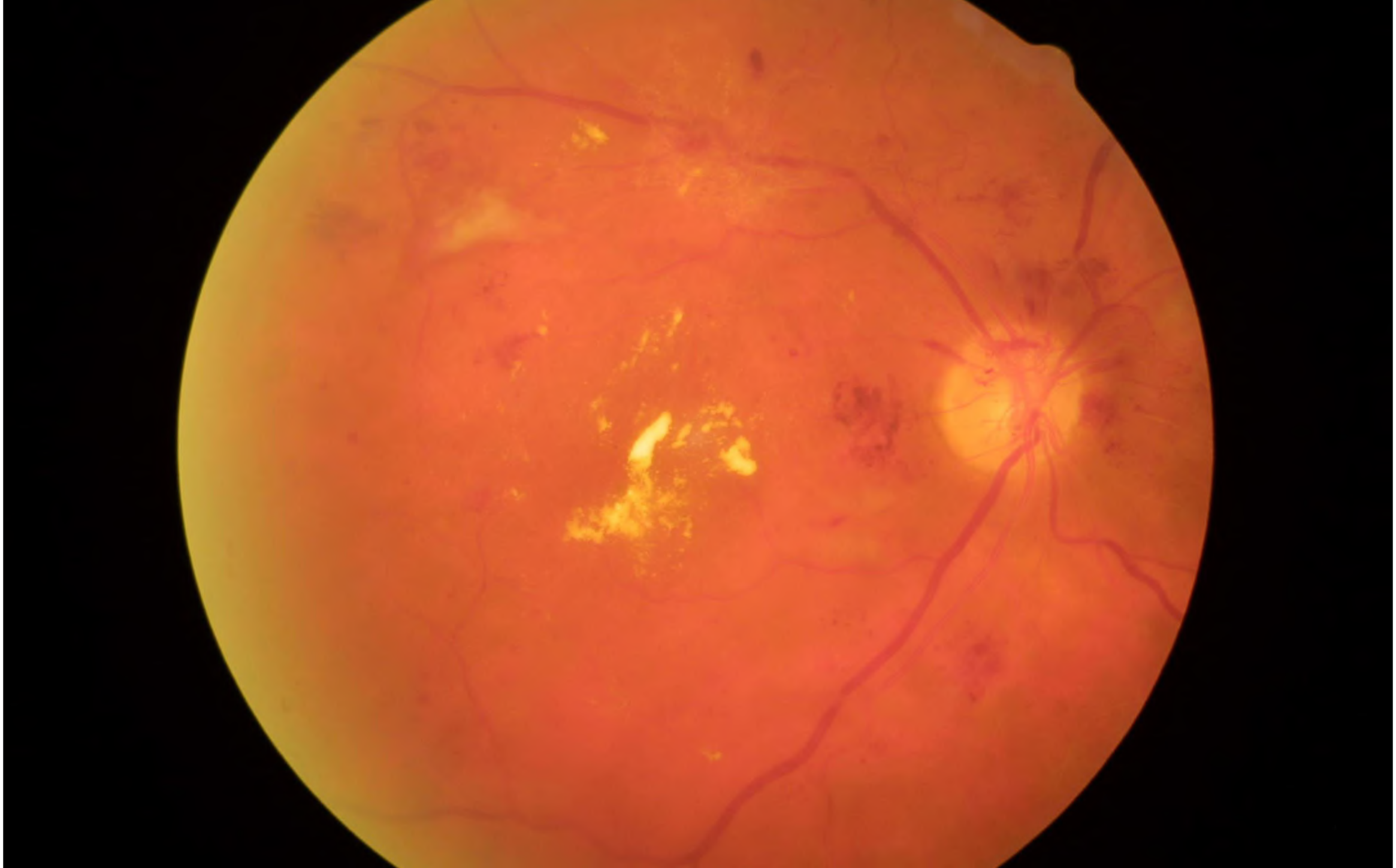
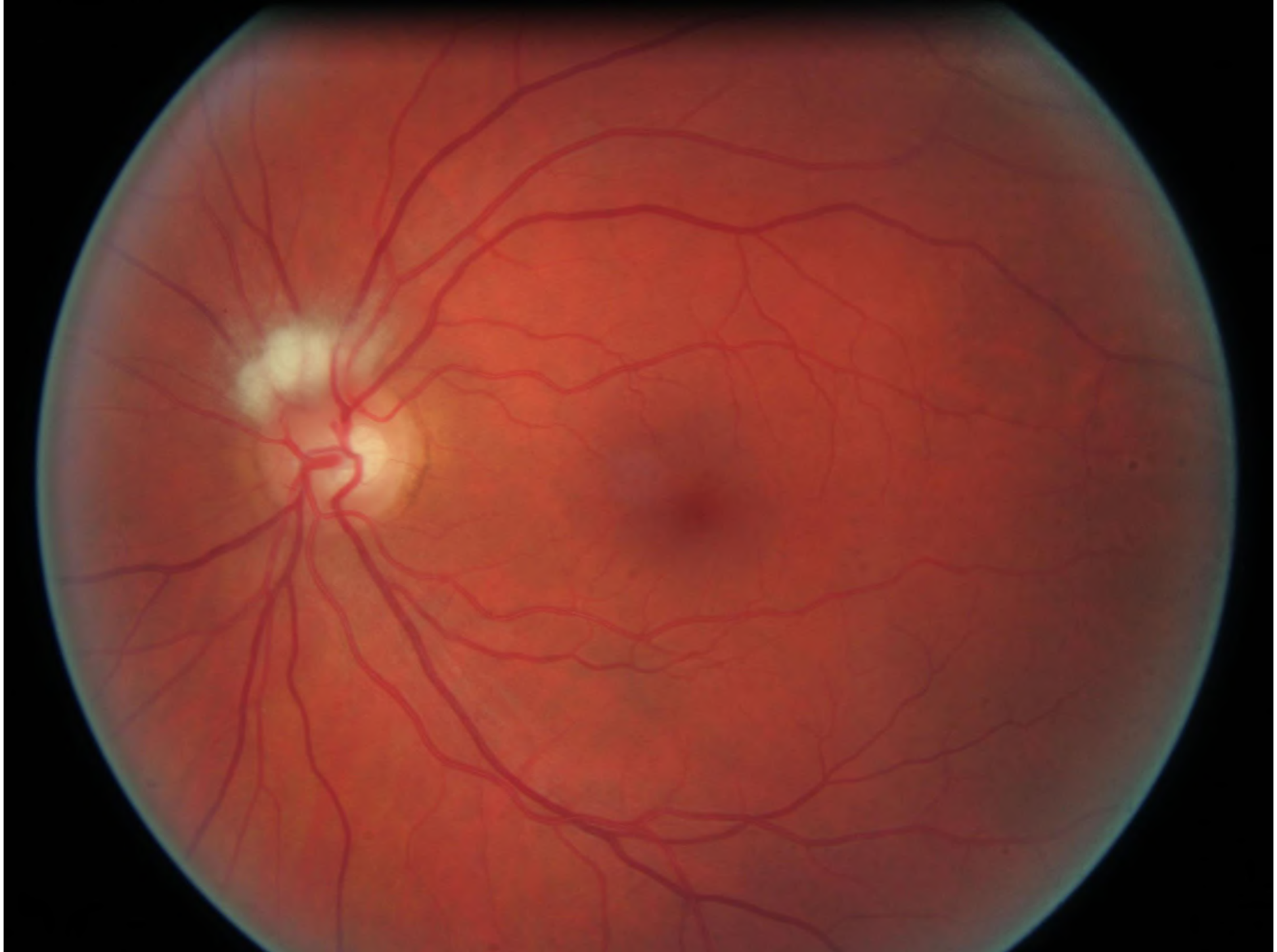
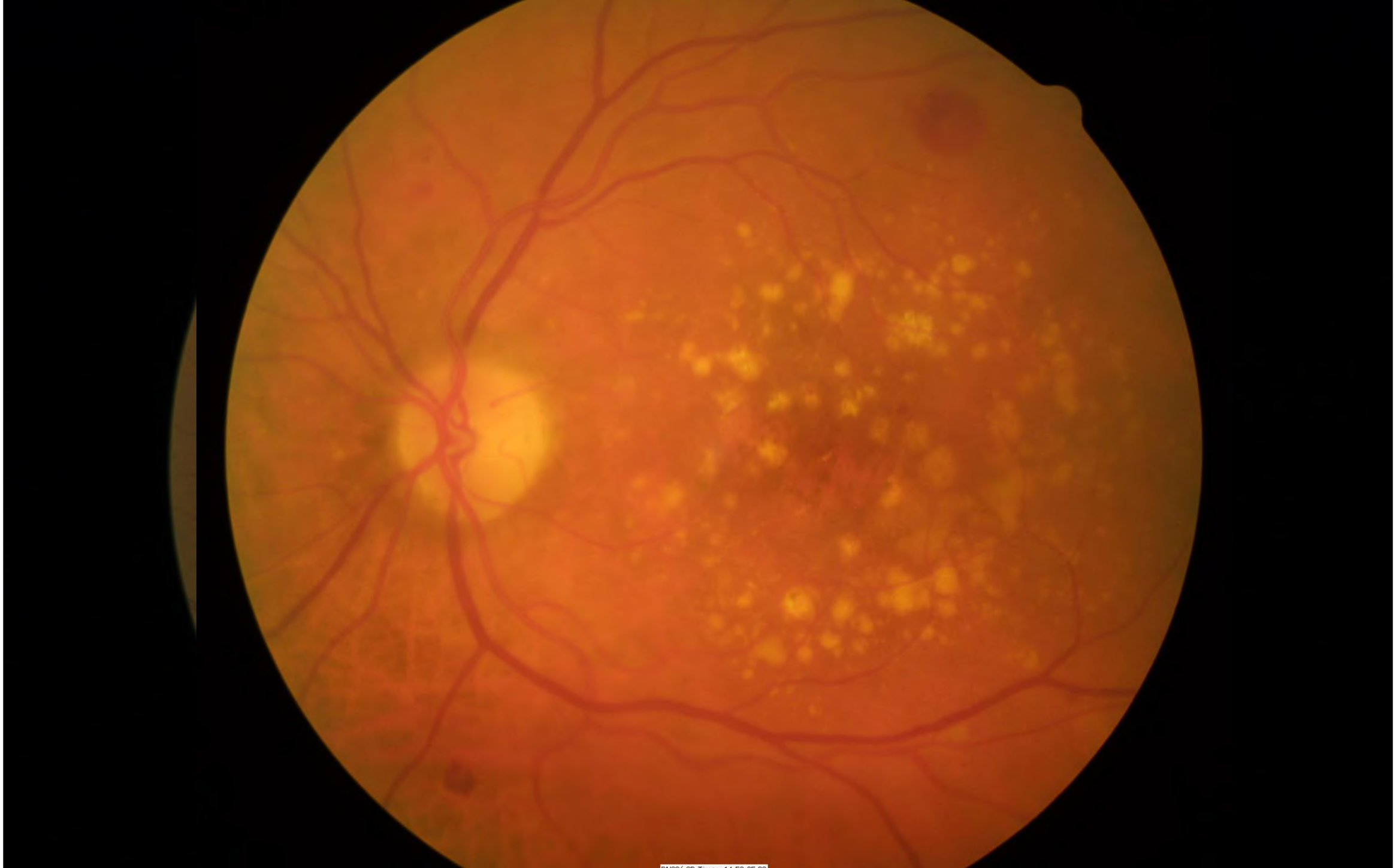


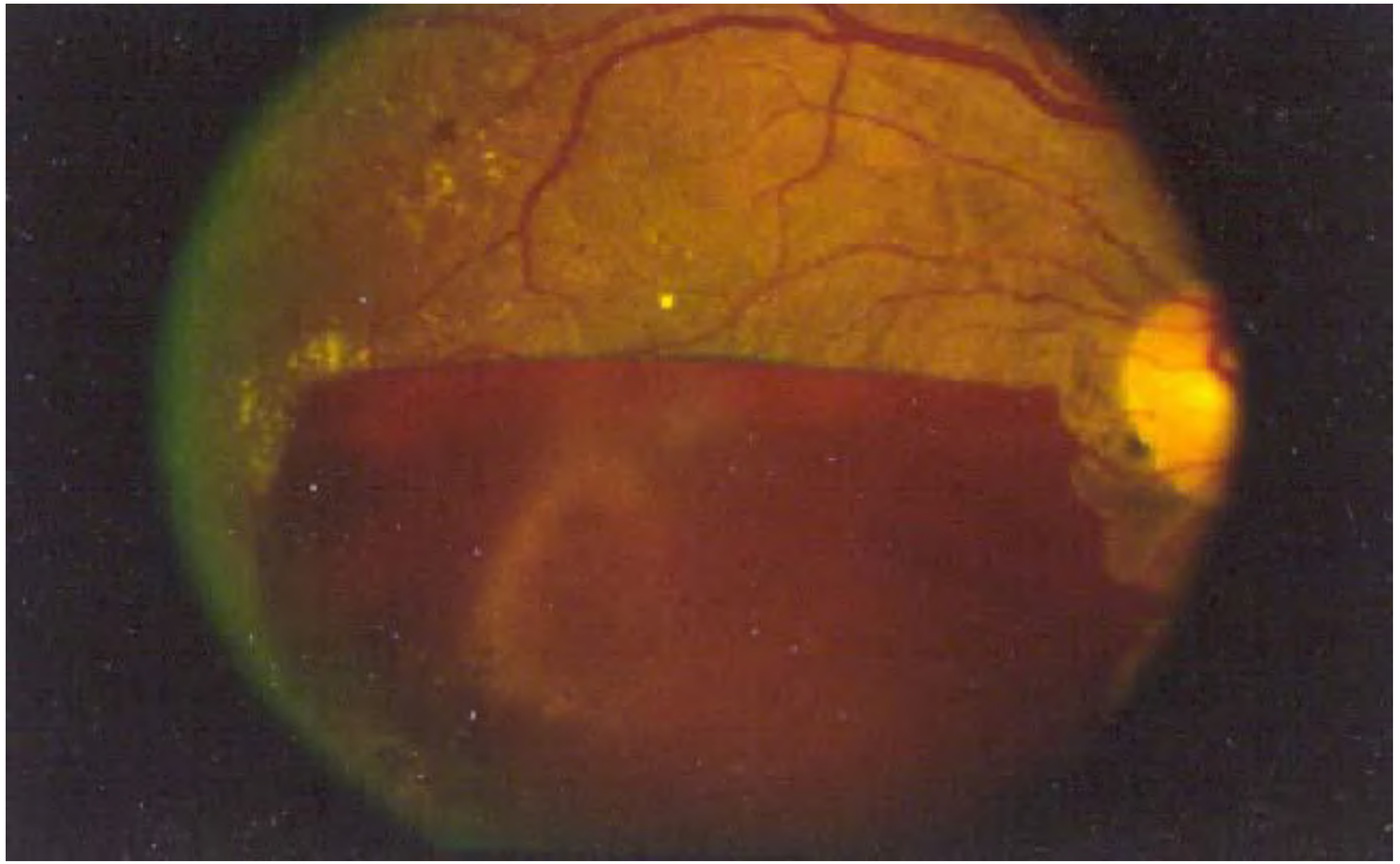


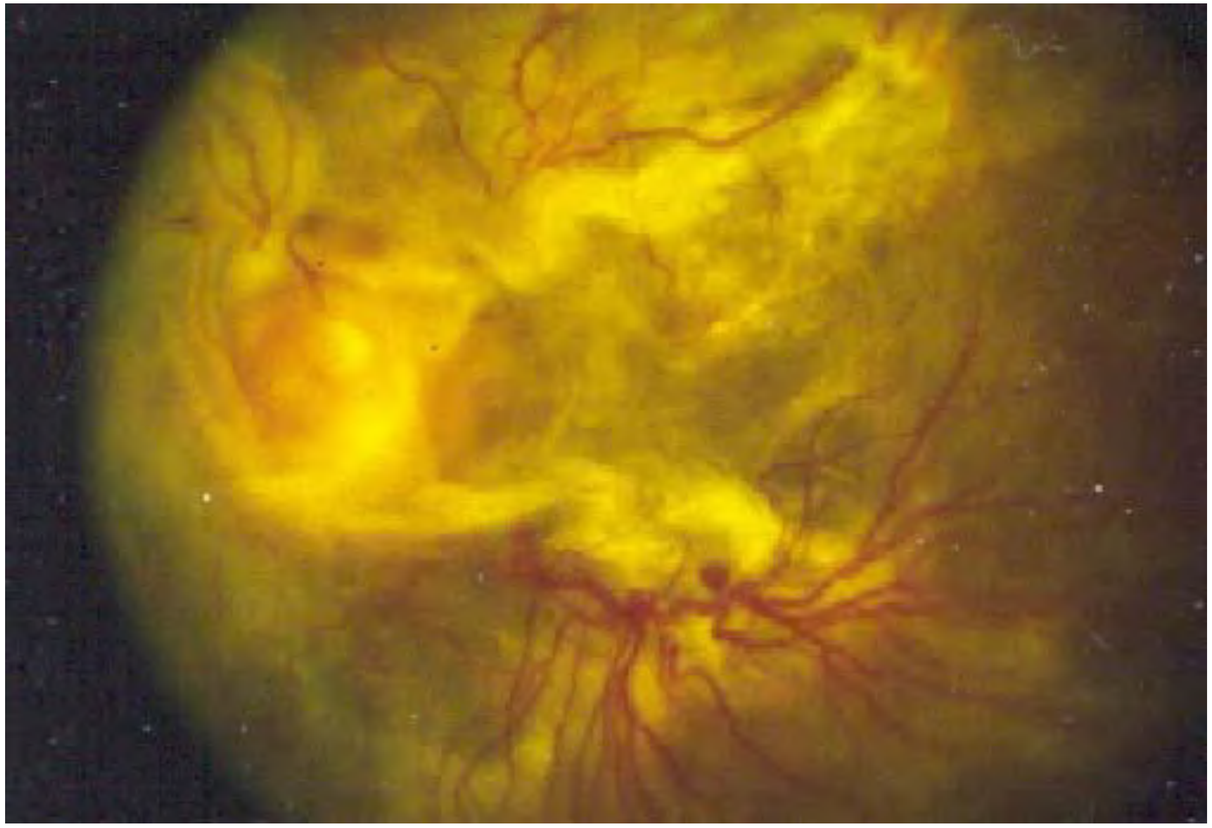








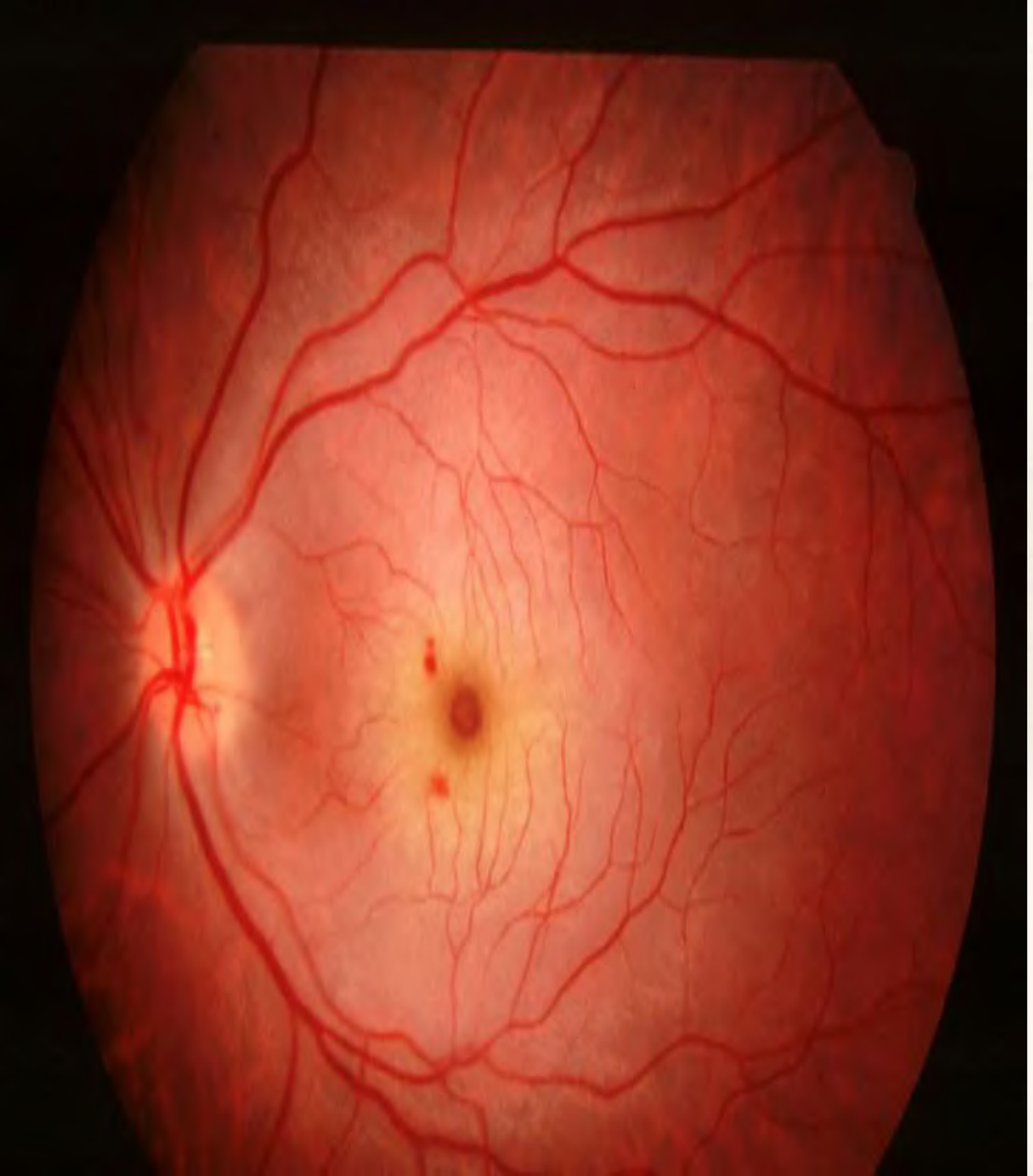


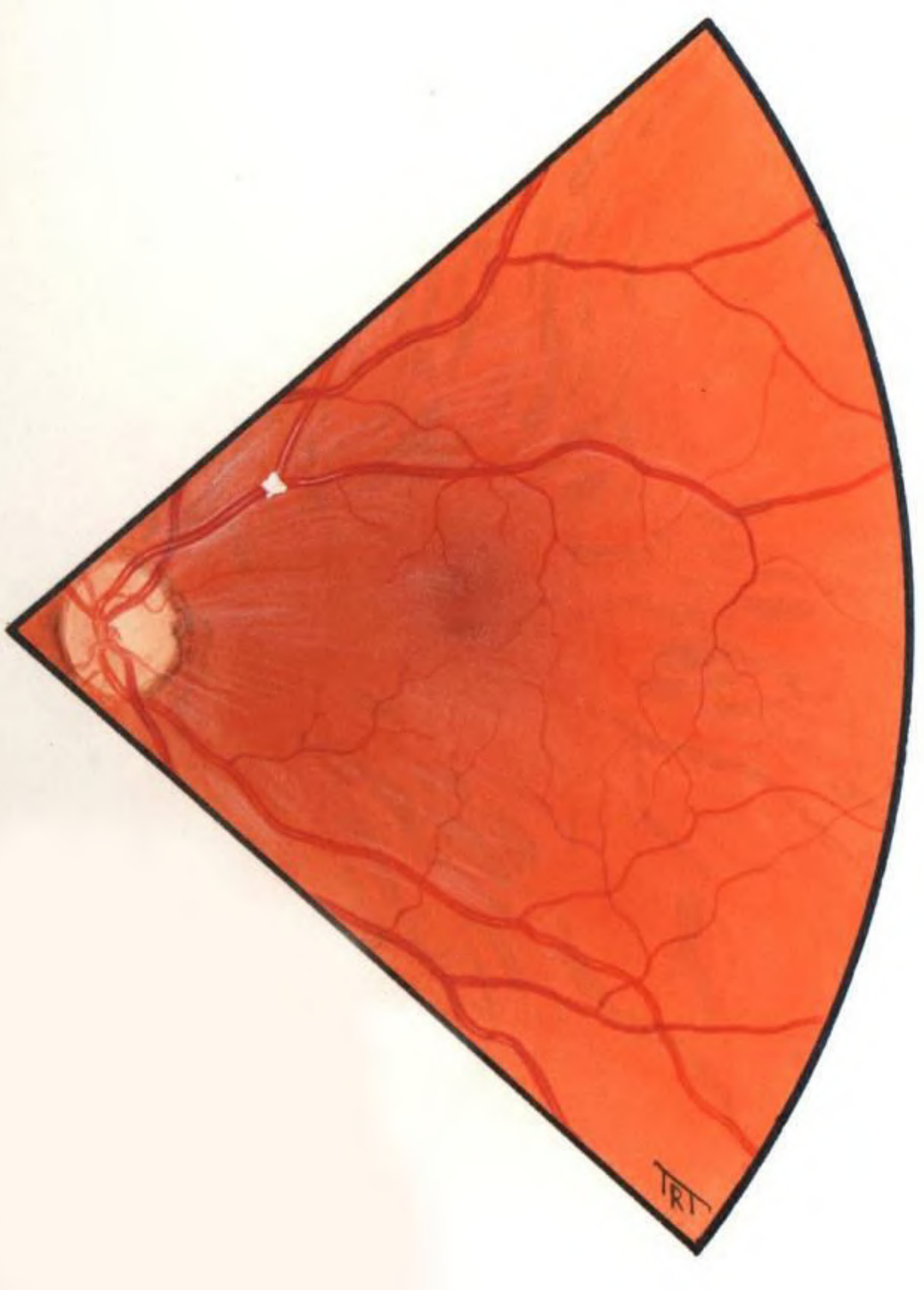


Sudden Painless Loss of Vision

- **Retinal vascular Occlusions**
- **Retinal Detachment**
- **Vitreous /Pre-retinal bleed due proliferative retinopathy (DR,RVO, Eale's disease), PVD, Valsava retinopathy, Raised BP etc**
- **AION**

Most important people in Life

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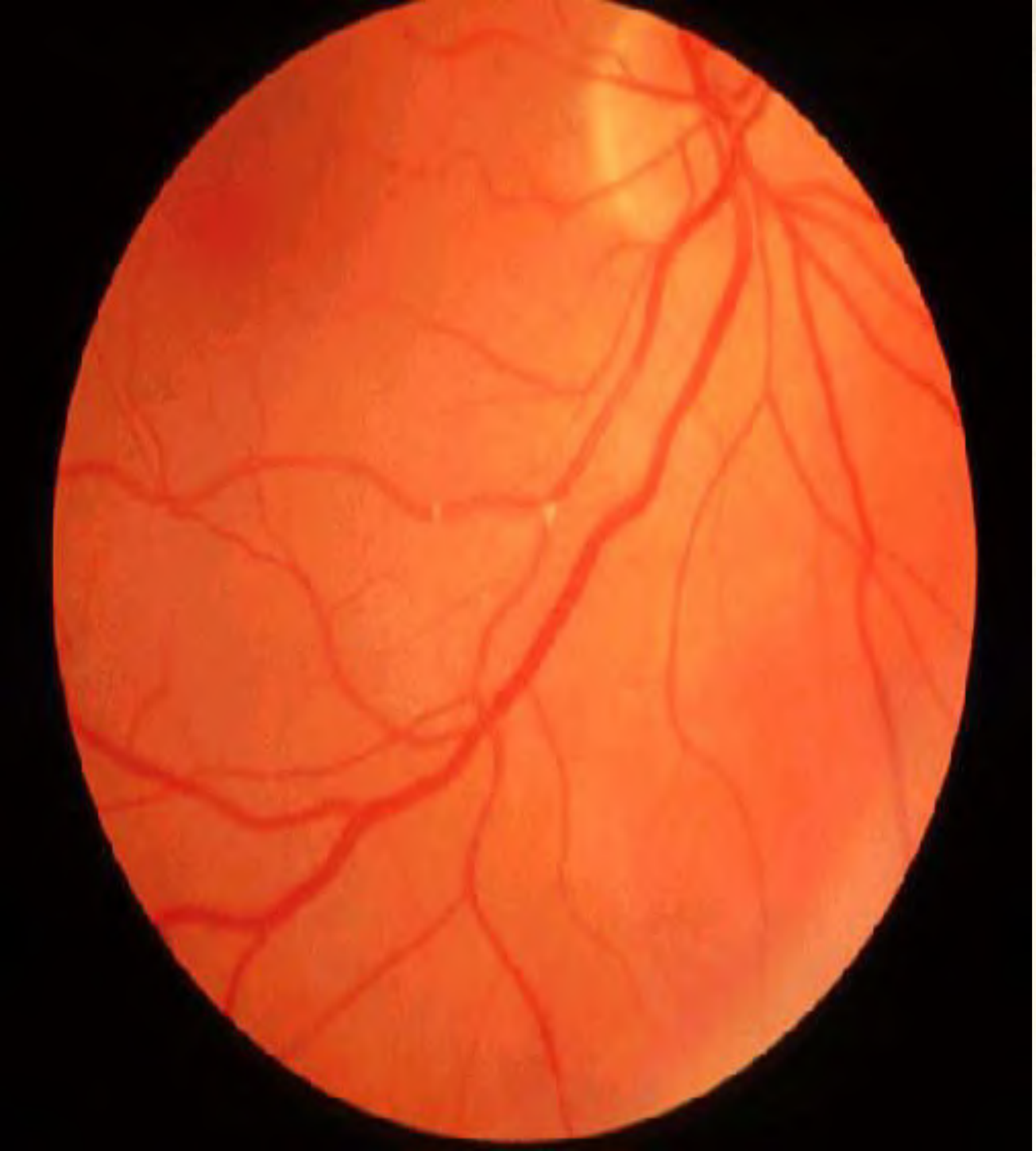
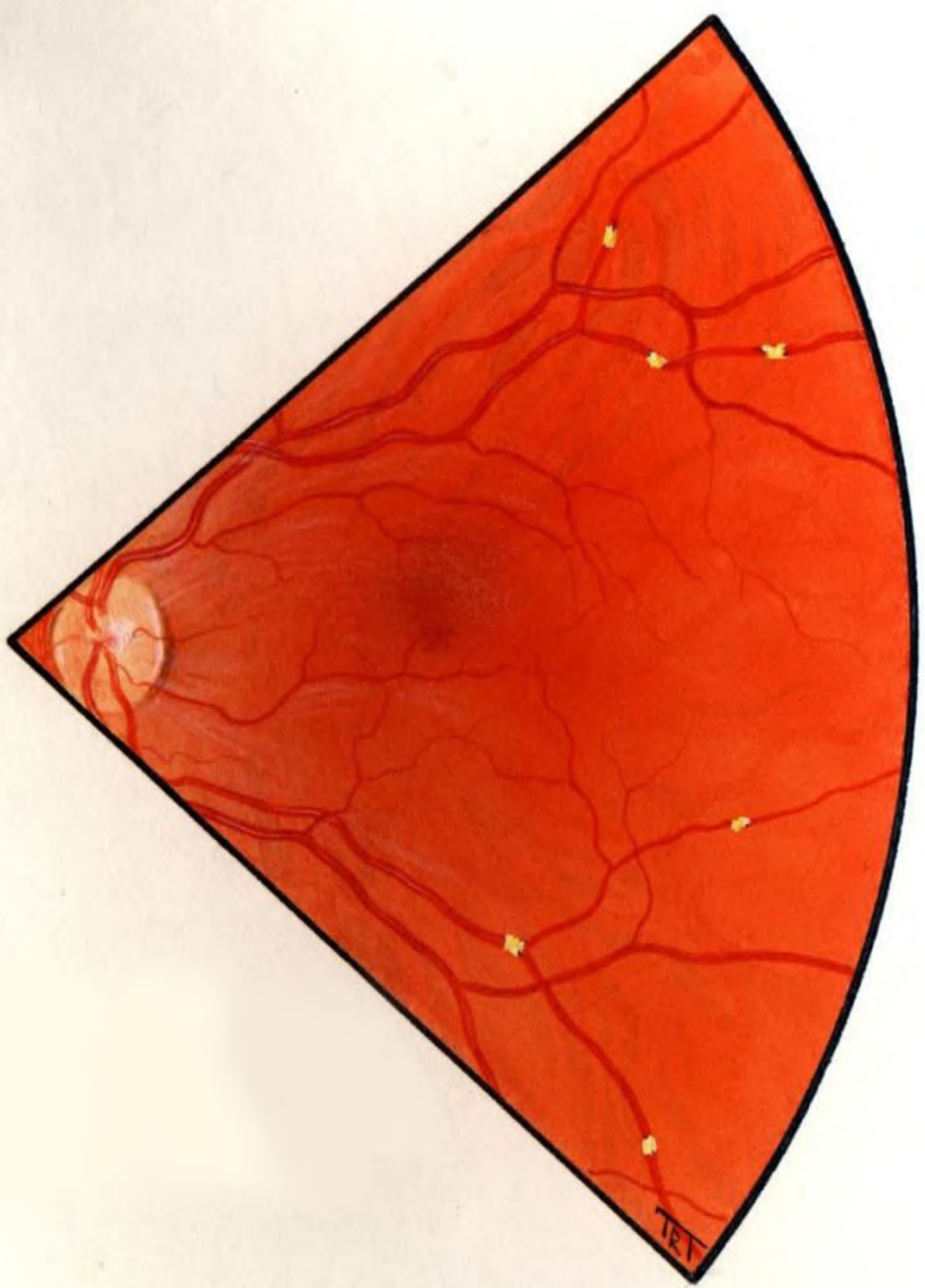




Retinal Artery Occlusions

- **Central RAO:**
- 50%: idiopathic
- 33% carotid artery disease
- 10% Giant cell arteritis

- Blockage is within optic nerve substance (obstruction site not visible on ophthalmoscope)

- **Branch RAO** – Obstruction distal to Lamina Cribrosa
- Visible blockage.

Retinal Artery Occlusions

Atherosclerosis - Most common cause of CRAO --- 80% cases

Mostly by thrombus formation (Localized intimal damage due to Artherosclerosis - incites thrombus)

Embolization: Common cause of CRAO - Ophth artery-first branch of internal carotids.

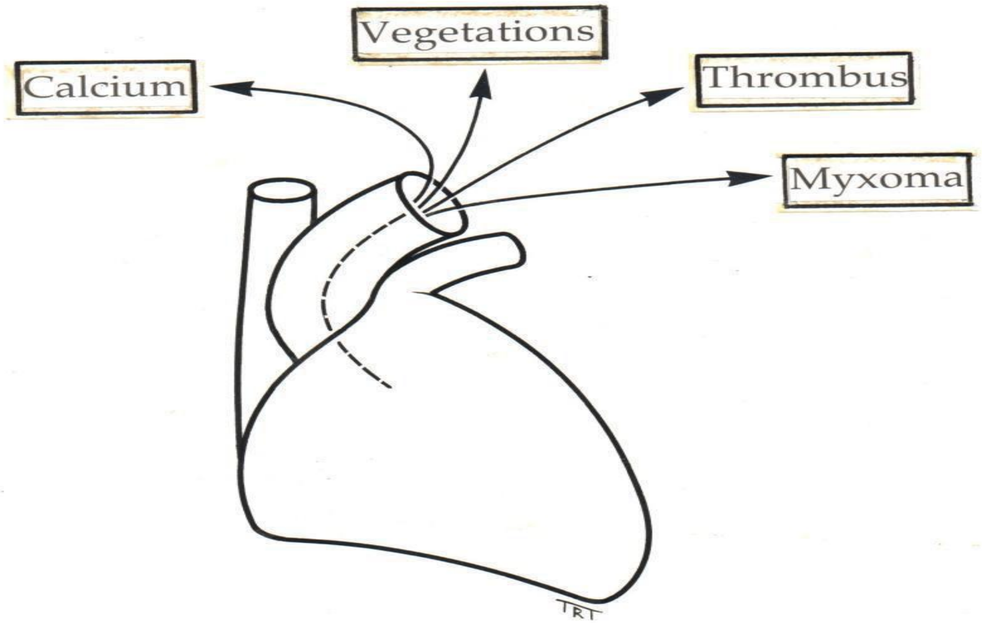
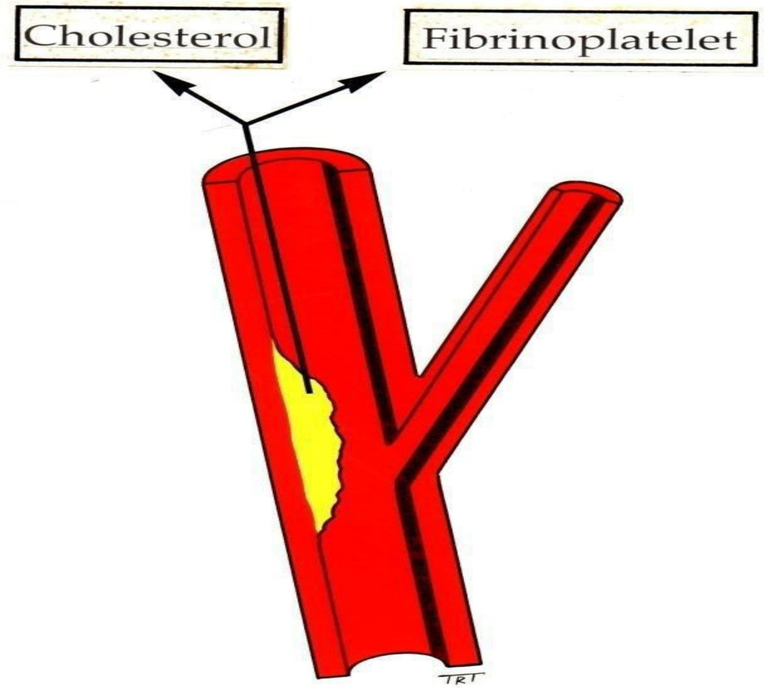
Carotids---emboli from atheromatous plaques at carotid bifurcation.

Heart:

Calcific	Aortic/mitral valves,
Vegetations	Bacterial endocarditis,
Thrombus	MI (Lt side of heart), M.stenosis with Atrial fibrillation
	Myxomatous - Atrial myxoma

Retinal Artery Occlusions

Types of emboli

Cardiac	Carotid
	

Retinal Artery Occlusions

- **Carotid artery disease:** Leading cause of morbidity and mortality.

  Carotid bifurcation- Vulnerable to atheromatous ulceration and stenosis

- **Cholesterol (Hollenhorst Plaques)**

  Rarely causes significant obstruction. Frequently symptomatic

  Refractile golden to yellow orange crystals-usually at bifurcation of arteries.

Retinal Artery Occlusions

- **Fibrinoplatlet Emboli:** Dull grey elongated particles-usually multiple-may fill entire luman.

Causes:

Retinal Transit Ischemic Attacks - Amaurosis fugax: usually but occasionally complete obstruction

Retinal TIA-Painless unilateral loss of vision-few min – Recovery

May be associated ipsilateral **cerebral TIAs** with contralateral signs.

Retinal Artery Occlusions

- **Calcific:** From atheromatous plaques in ascending aorta/carotid arteries, calcific heart valves- usually single white-usually close to disc more dangerous.

- **Periarteritis:** Systemic vasculitis, polyarteritis nodosa, S.L.E, Optic neuritis, Behcet's, syphilis, G.C.A and other collagen diseases, mucormycosis.

- **Blood disorders**: Protein S or Protein C deficiency, sticky platelet syndrome.
- Hypercoagulative states- ploycythemia, sickle cell disease.

Retinal Artery Occlusions

- **COMPRESSIVE:** External compression by tumor, hemorrhage, inflammation.
- **TRAUMA:** Direct damage to O.N and vessels.
- **Retinal Migraine:** Rare - Exclude other causes.
- **MISCELLANEOUS:** BP, Diabetes, Retro bulbar anesthesia etc.

Central Retinal Artery Occlusion:

Size / Location of obstructed vessel.

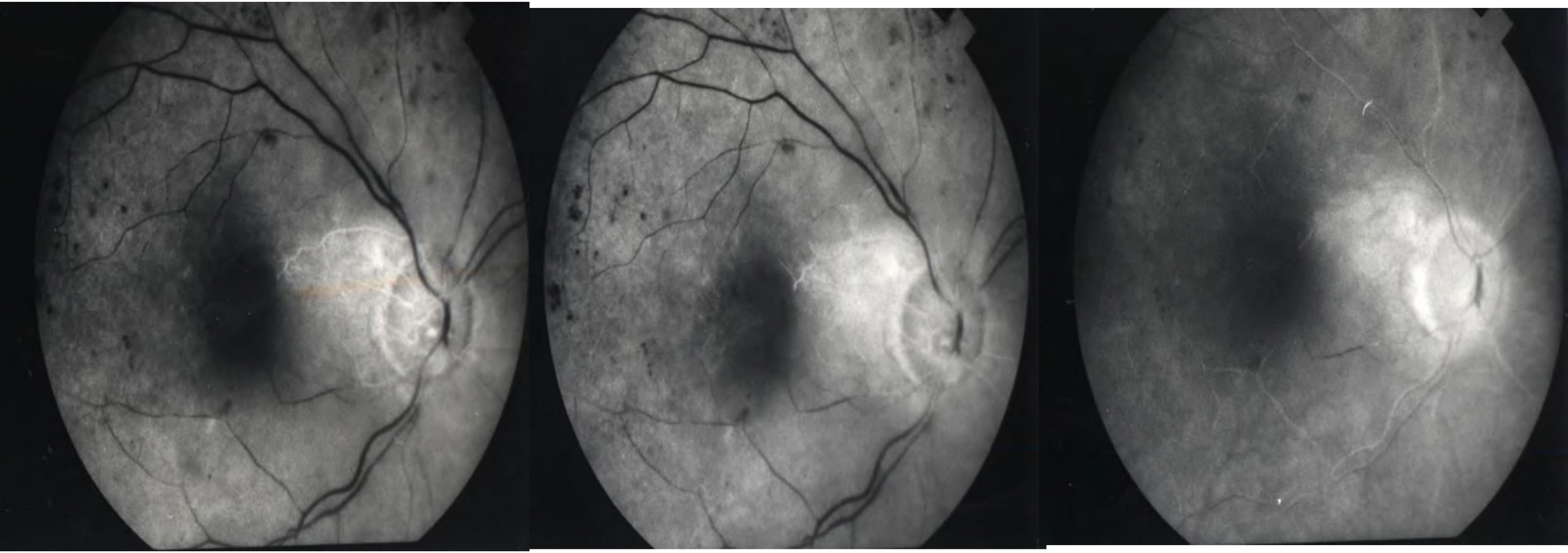
Severity / Duration of obstruction.

Usually atheroma – but can be calcific emboli.

FEATURES: within seconds

- Acute / profound visual loss
- Relative APD – Marcus Gun pupil
- White retina + cherry red spot within minutes
- 20 % individuals Cilio-retinal arteries from ciliary circulation – spares macula
- Retinal vessels – Narrowing – sludging and segmentation of blood column(wks)

FA of central retinal artery occlusion

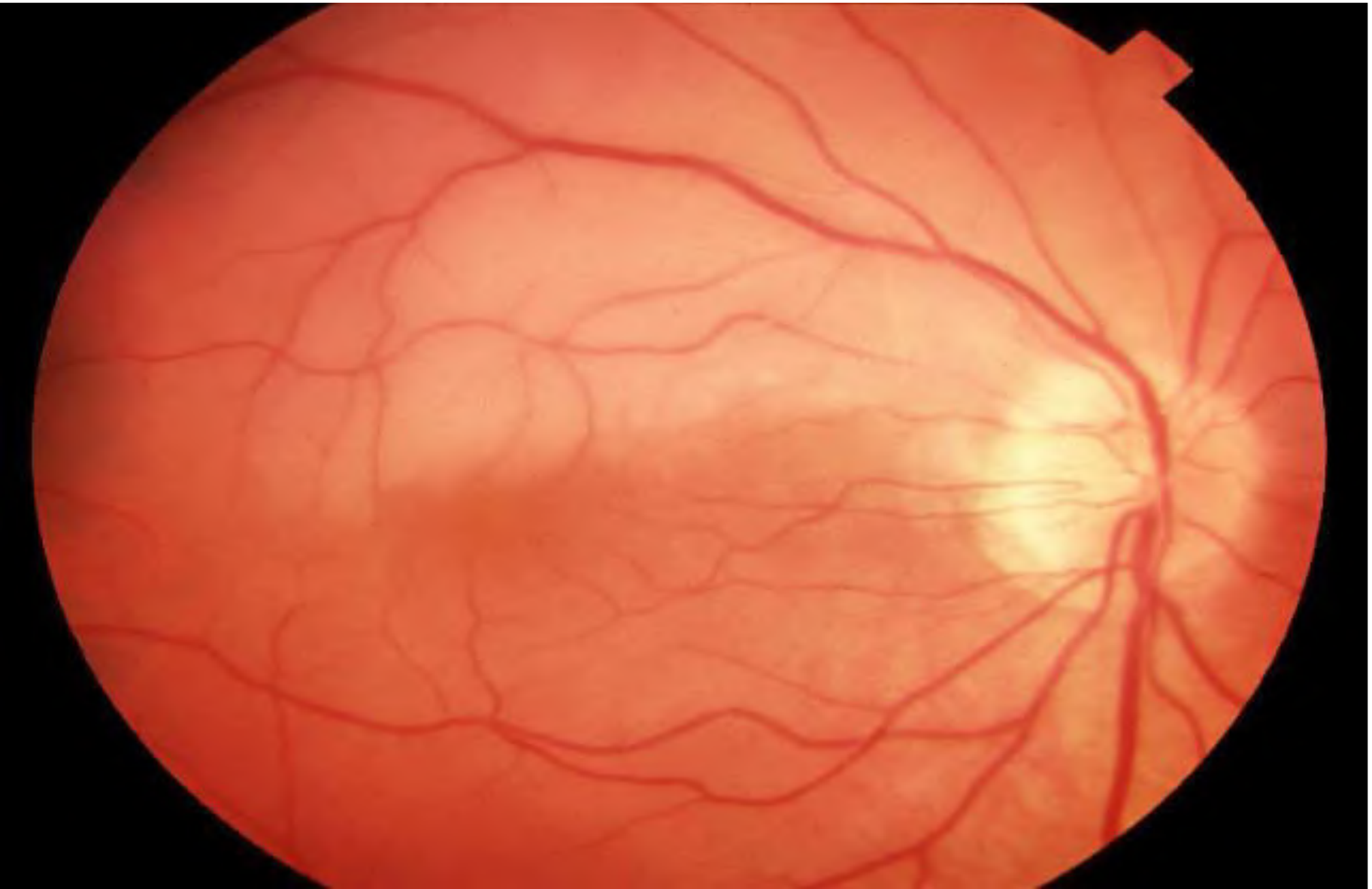


Early filling of cilioretinal artery

Non-filling of other vessels

Late staining of vessel walls

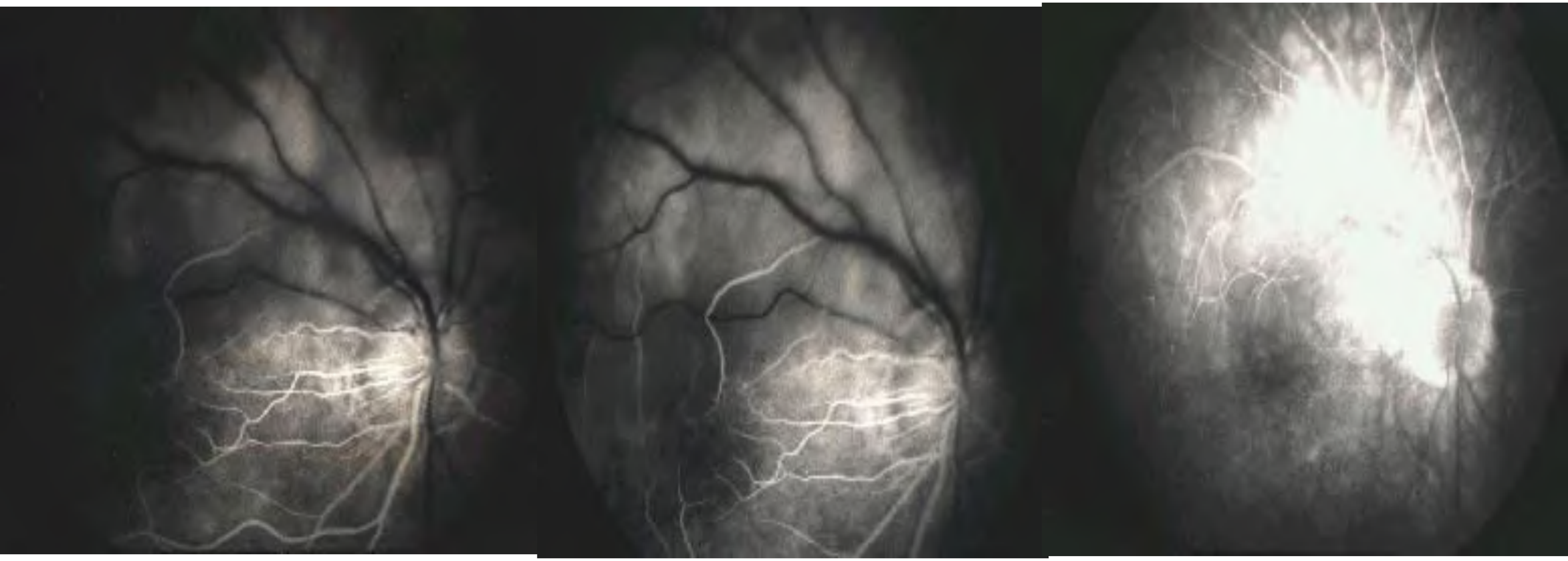
Branch retinal artery occlusion (BRAO)



- VA - variable
- APD - mild or absent
- Retina whitening
- Arteriolar narrowing

White cloudy swelling clears (Permanent sectorial visual field defect-atrophy of inner retinal layers).
Re-canalization of obstructed vessel – only subtle or absent ophth signs

FA of branch retinal artery occlusion

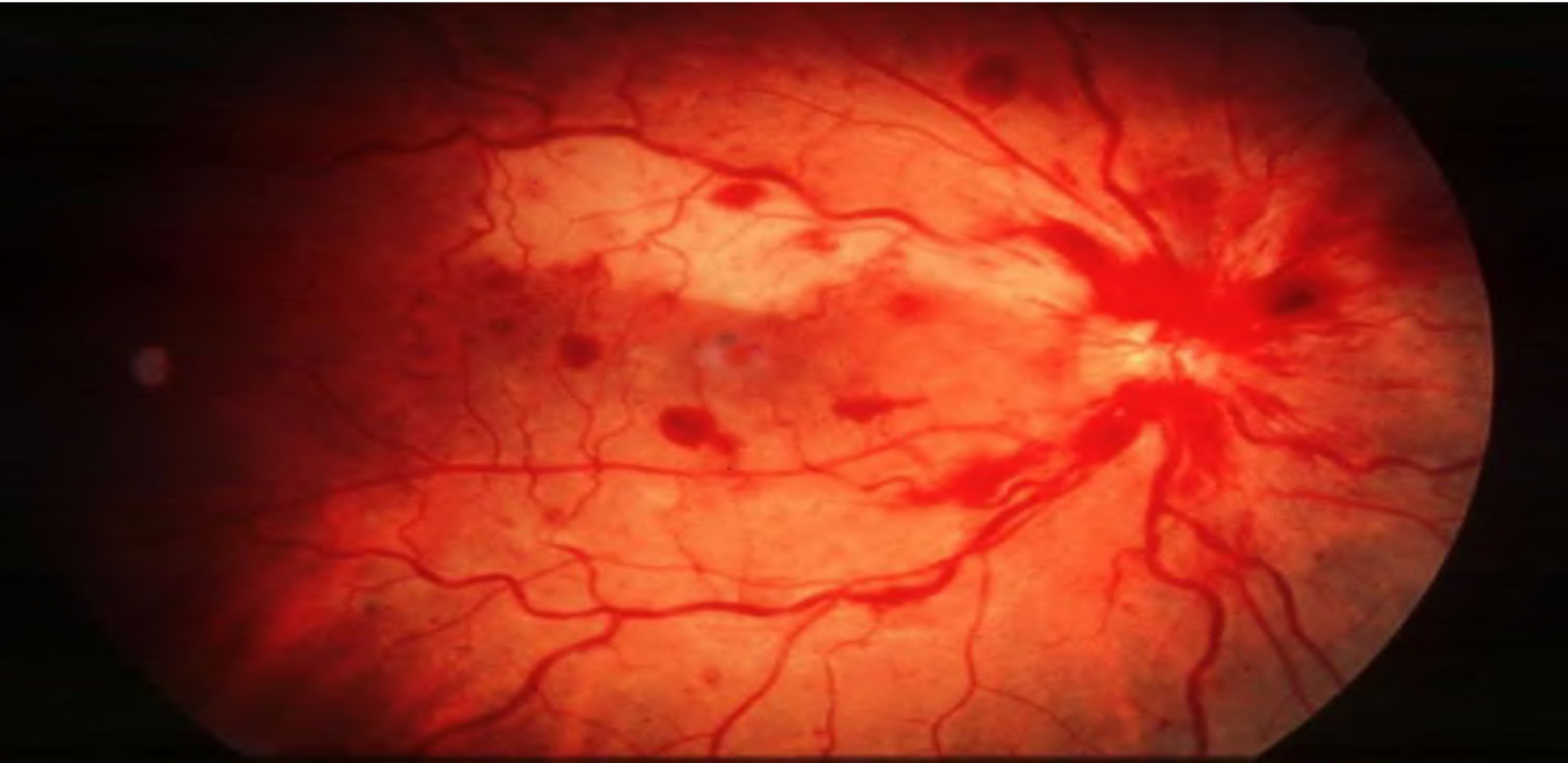


Early masking

Extreme delay of
Arterial phase

Late staining of
arterial walls

CRVO/CRAO



EVALUATION: Clinical

- **F.F.A**
- **Color Doppler U/S evaluation** - orbital circulation degree of obstruction differentiate ophthalmic artery obstruction from CRAO
- BRAO/CRAO- Pts at risk to have obstruction in other eye 10%-bilaterality

  Evaluate for embolic sources:

  Cardiovascular exam

  Carotid artery exam

  (Echocardiogram, carotid non-invasive testing)

  Rule out G.C.A- Pts above 50 years

  Do E.S.R-Biopsy if needed.
- **Cherry red spot** (CRAO)- Ischemic visual loss, age, associated systemic disease, check surrounding vessels and retina, other causes-storage diseases

TREATMENT

- Retina-Highly metabolic organ -very sensitive to ischemia

- CRA is end artery -No true normal anastomosis - No regeneration – Ischemia - 90 min- cell death due to hypoxia

No proven Rx available

Try with in 48/72 hours

❖ **Dislodging emboli distally:**

- A/C Paracentesis, ocular massage, medications to lower IOP

❖Dissolving thrombi:

Clot dissolving medications

streptokinase, urokinase, heparin, tPA

Systemic-I/V infusion

Local -via ophth artery with catheter

Initial reports-encouraging-risks only reserved for cases within 48 hours and only CRAO not BRAO.

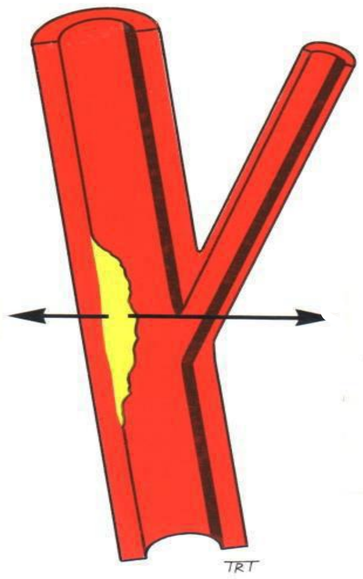
❖Increasing oxygenation to retina::

Carbogen - 95% O2 + 5% Co2

For 10 min every 2 hours for 1-2 days. Avoid in chronic obstructive lung disease.

❖Protecting surviving retinal cells from ischemic damage?

Treatment options for carotid disease



Antiplatelet therapy

- **Aspirin 75 mg daily**

Aspirin + dipyridamole (Persantin)

- **Clopidorel (Plavix) 75 mg daily**

Anticoagulants

if antiplatelet therapy ineffective

Carotid endarterectomy

- **Patients with other risk factors for stroke**
- **Symptomatic carotid stenosis > 70%**

Retinal Artery Occlusions

Neo-vascularization is uncommon compared to venous occlusions

Proliferative retinopathy:

NVE /NVD

- **Laser**
- **Anti VEGF**


© Jamie Wiseman

Thanks

Retinal Venous Occlusions

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No financial disclosures

Never reply when you are angry. Never make a promise when you are happy. Never make a decision when you are sad.

RETINAL VEIN OCCLUSION

Predisposing Factors:

SYSTEMIC:

Age: Increasing age – {6th –7th decades }

Diabetes / B.P

Blood dyscrasia: Hyper viscosity – chronic leukemia, Polycythemia, Changes in plasma proteins – macroglobulinaemia , Sickle cells disease –BRVO

❖If bilateral CRVO – check for blood dyscrasia

Drugs – Oral contraceptives

RETINAL VEIN OCCLUSION

Predisposing Factors

OCULAR:

Raised IOP

Hyperopia

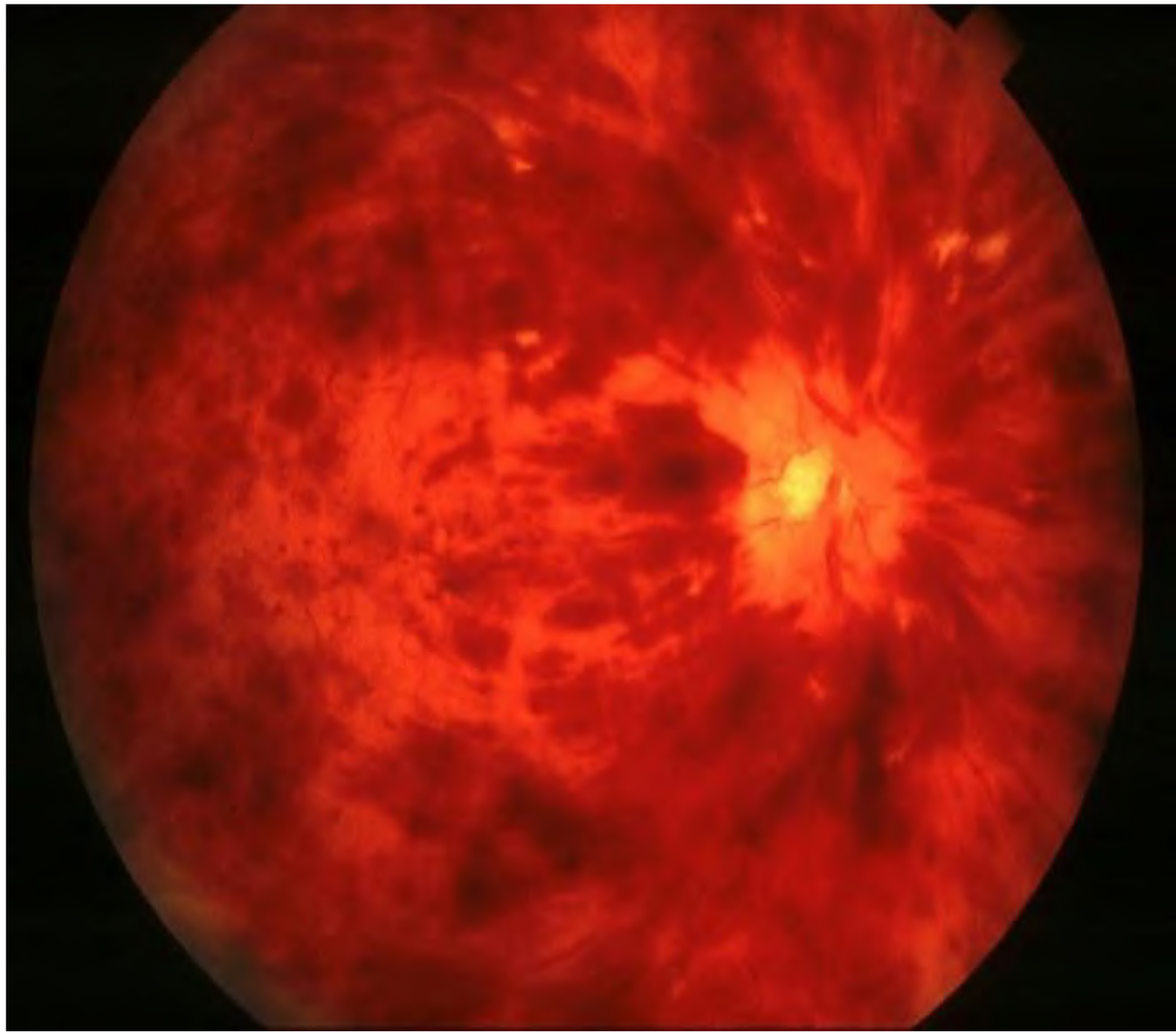
Congenital anomaly of CRV – usually young pts

Periphlebitis – Sarcoidosis, Behcet's disease, retinal vasculitis

Trauma

RVO: Pathogenesis

- Venous blockage----
- Back pressure on capillaries/ Stagnation---
- Hypoxia (Retinal ischemia) -----
- Endothelial junction dysfunction____
- Leakage of fluid & blood------
  (edema / hemorrhages)
- Severe non-perfusion leads to ischemia



CENTRAL RENTINAL VEIN OCCLUSION

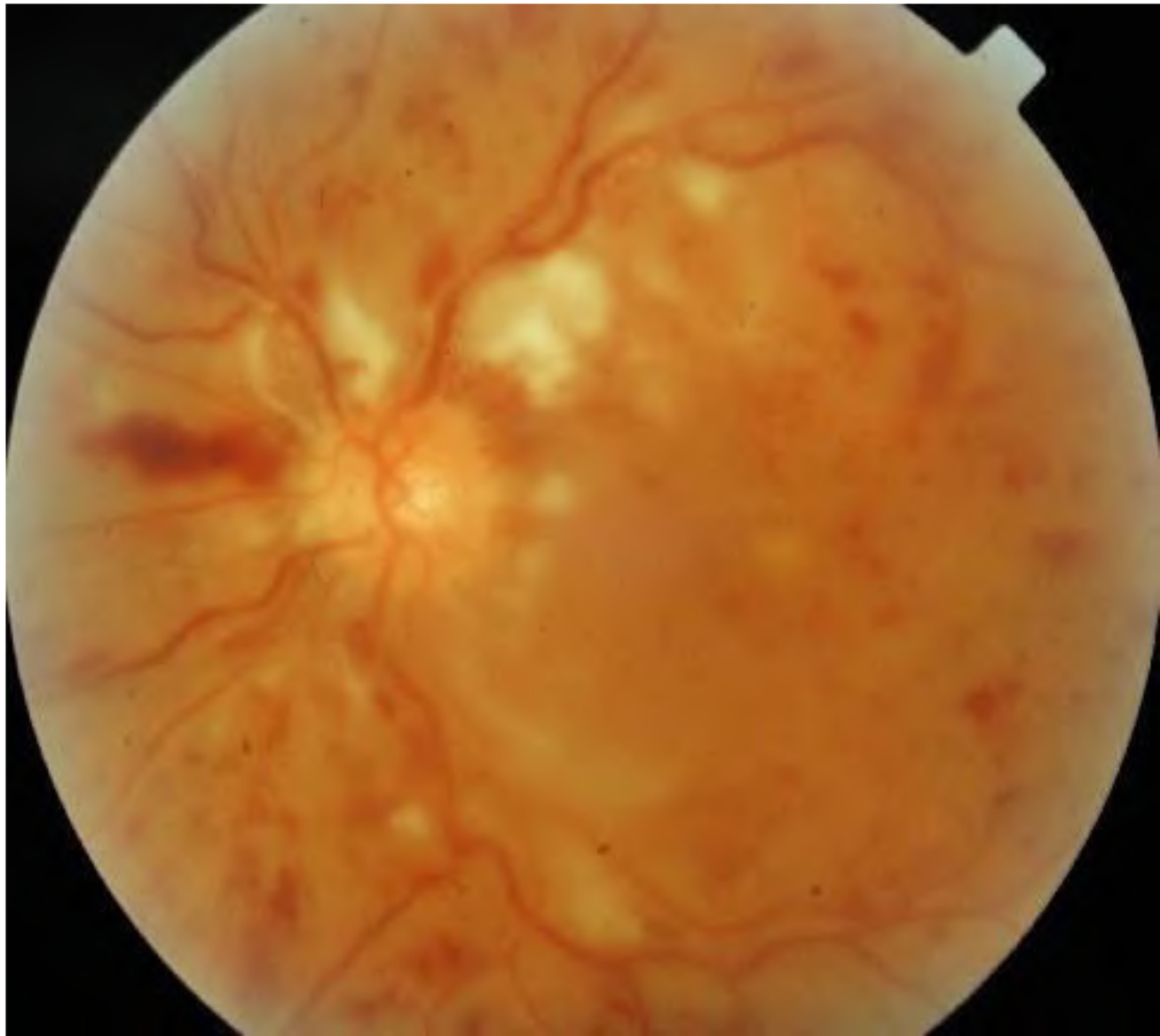
- **NON ISCHEMIC CRVO**
- **ISCHEMIC CRVO**
- **PAPILLO PHLEBITS**

CENTRAL RENTINAL VEIN OCCLUSION

NON ISCHEMIC CRVO:

Most common – 75%

- **Painless – sudden – Marcus Gunn pupil – slight**
- **Venous dilation and tortuosity – mild visual loss**
- **Retina hemorrhages (dot – blot and flame shaped) – all over – also in periphery**
- **Cotton wool spots – few**
- **Disc edema – macular edema (Mild to Moderate)**



CENTRAL RENTINAL VEIN OCCLUSION

Non-Ischemic

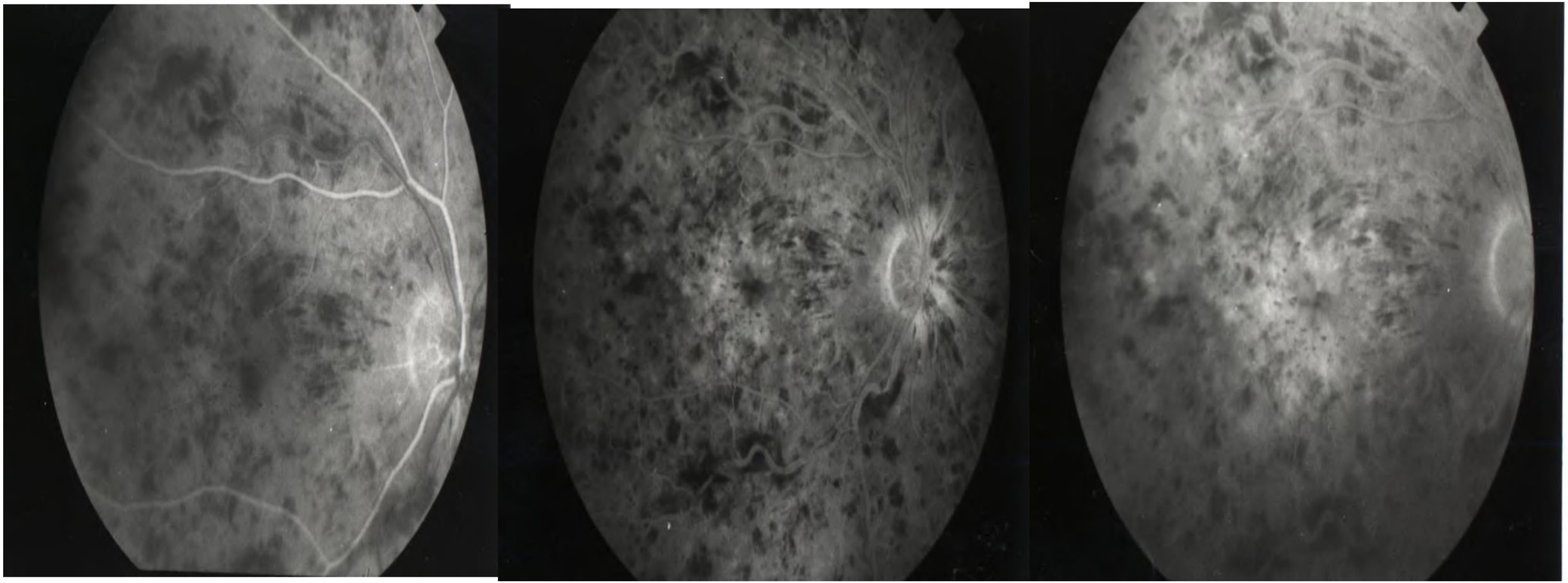
❖**Features severity:** **mild to moderate severity**

→ Hemispheric or Hemi central RVO

- **F.F.A** Retinal stasis + good capillary perfusion. (less than 10DD of capillary non perfusion)

- → Acute signs – resolves – 6 – 12 months
- →**Hard exudates, Disc collaterals + Epiretinal membrane formation + pigmentary changes at macula**

FA of non-ischemic Central Retinal Vein Occlusion



Good retinal capillary perfusion

• CENTRAL RENTINAL VEIN OCCLUSION
Non-Ischemic

COMPLICATIONS:

❖Vision decreased – macular edema

❖Conversion to ischemic CRVO

15% - 4 months,

34 %- 3 years

PROGNOSIS:

- Recovery – Normal / Near normal – 50%
- Poor vision – mainly chronic cystoid macular edema
- Initial V.A – good – better recovery

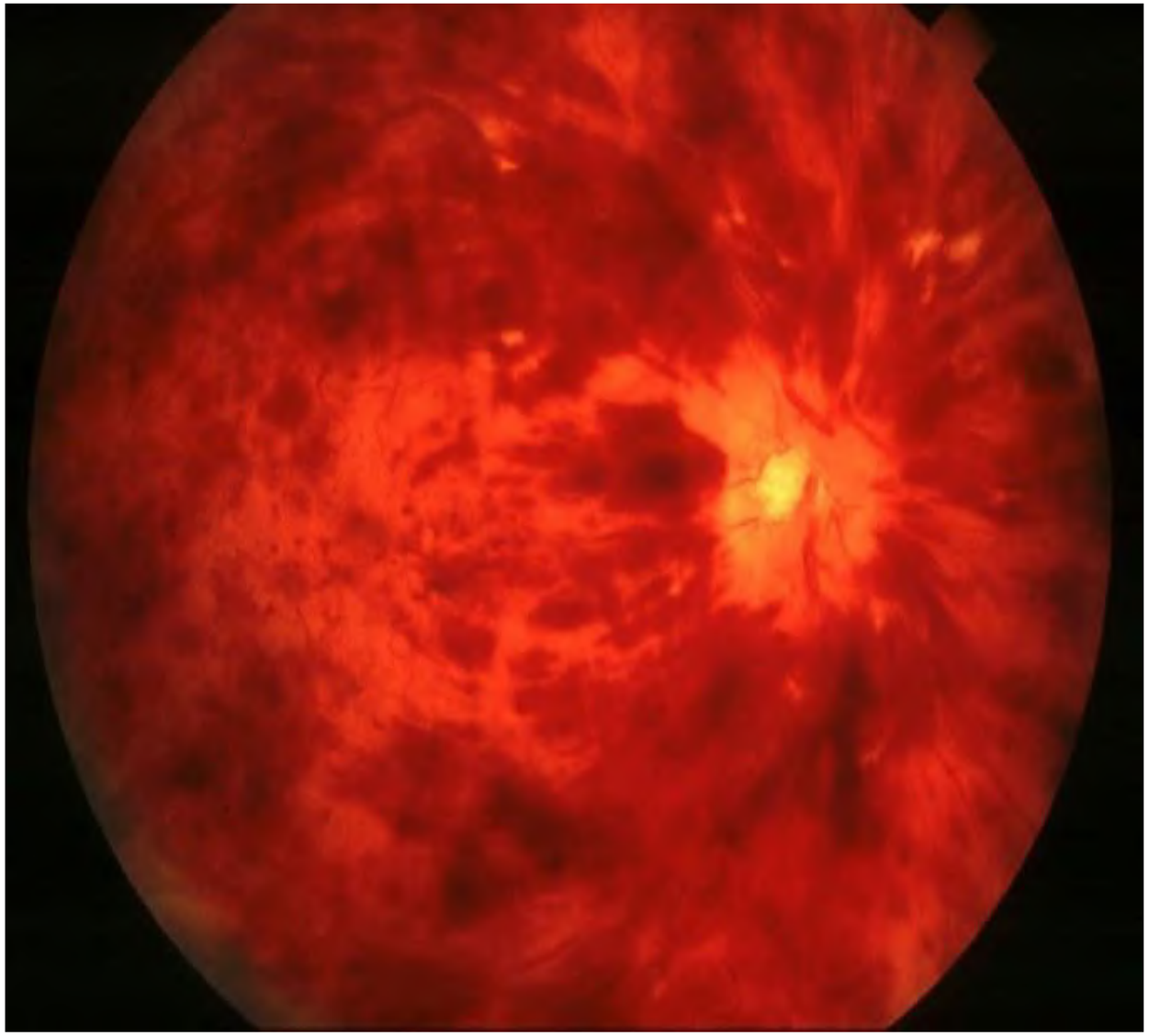
Rx: Treat the cause/risk factors, Anti VEGF, No laser to macular edema, Laser induced chorio-retinal anastomosis

CENTRAL RENTINAL VEIN OCCLUSION

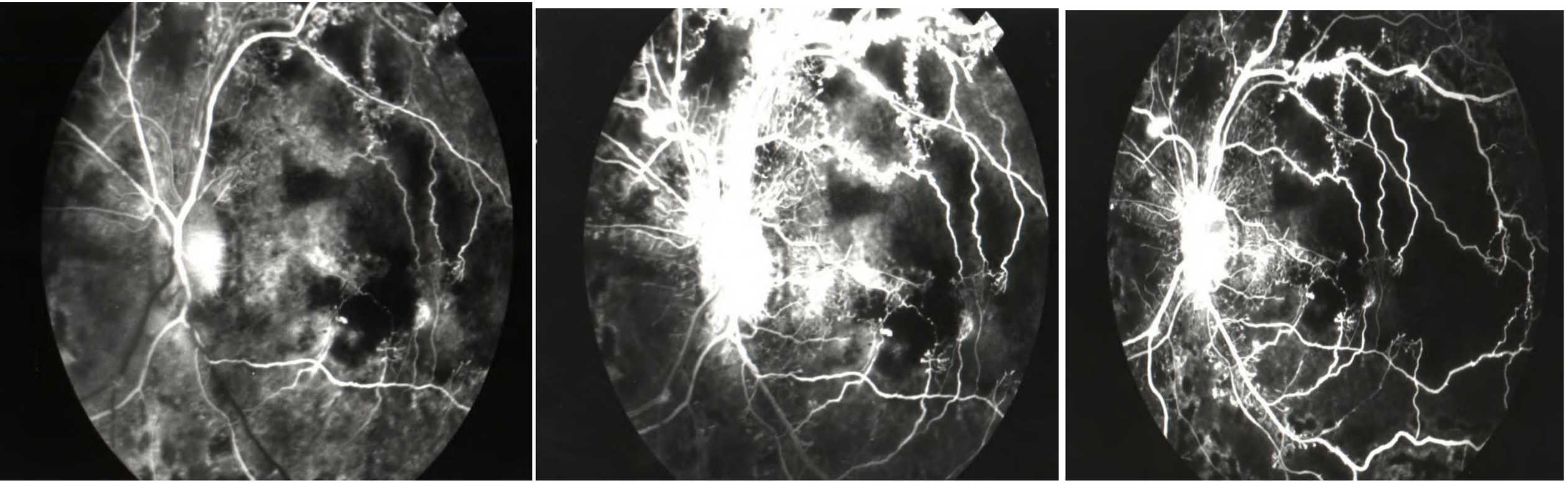
Ischemic

- Less common
- Severe visual loss (usually < 6/60)
- Sudden – painless

- **SIGNS:** All are Severe



FA of ischemic central retinal vein occlusion



F.F.A Central masking of retinal vascular bed – by hemorrhages + Extensive capillary non perfusion > 10 Disc diam

CENTRAL RENTINAL VEIN OCCLUSION

Ischemic

Complications:

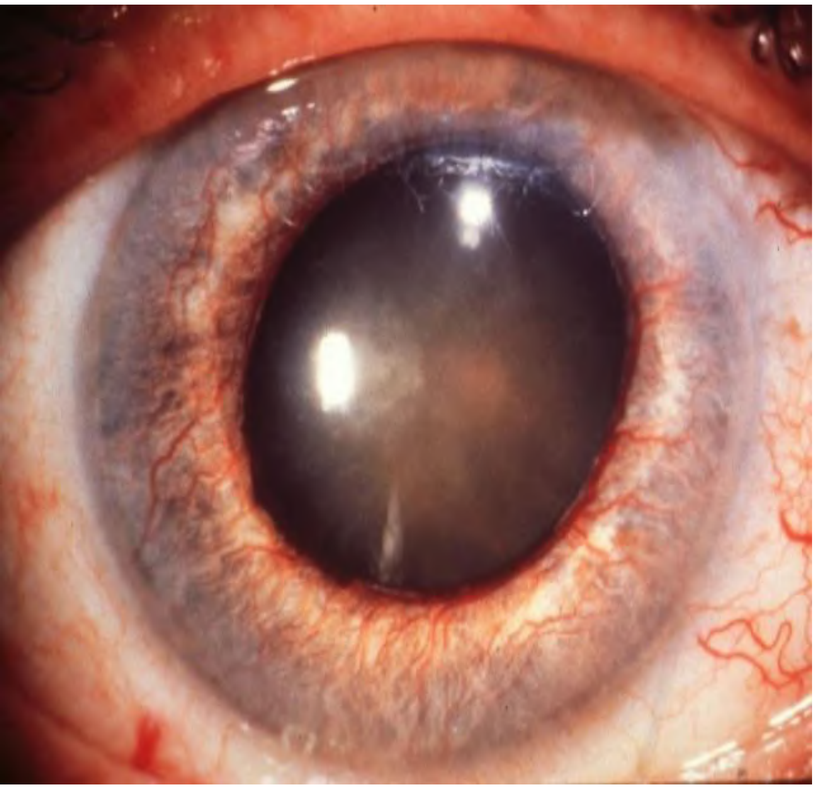
- Macular ischemia – Visual compromise (Permanent)
  (Macular edema, Ischemic maculopathy)

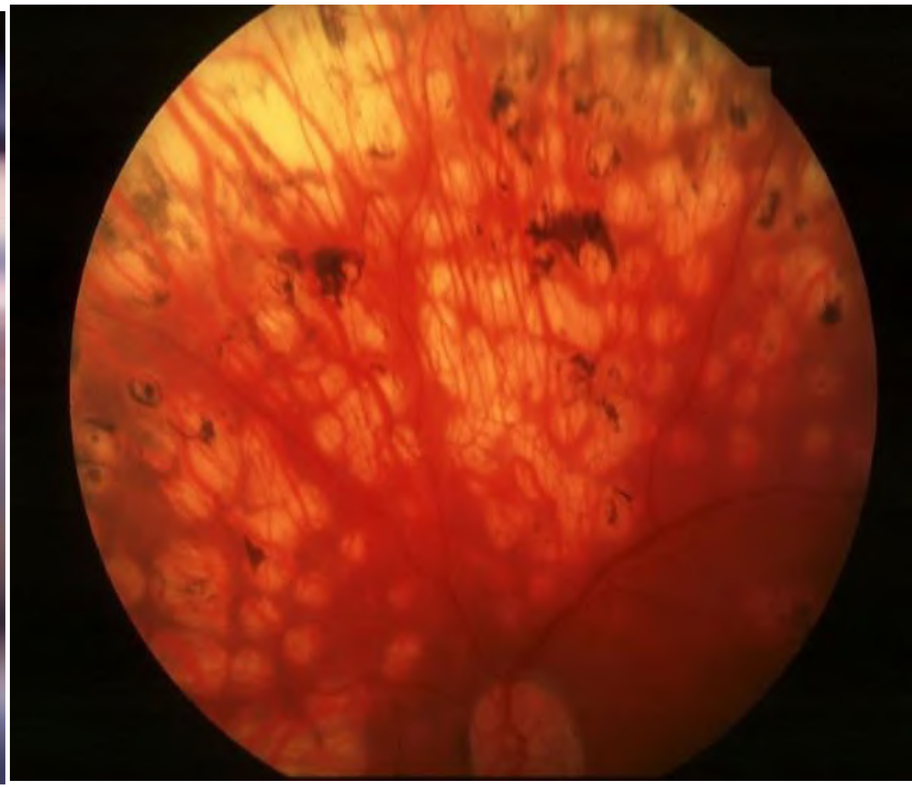
- Rubeosis 50% - **90/100 day glaucoma** – NVG

- **15% NVE / NVD**, Vitreous haemorrhage, Tractional R/D.

Ischemic central retinal vein occlusion:

Management: Anti VEGF / PRP Laser





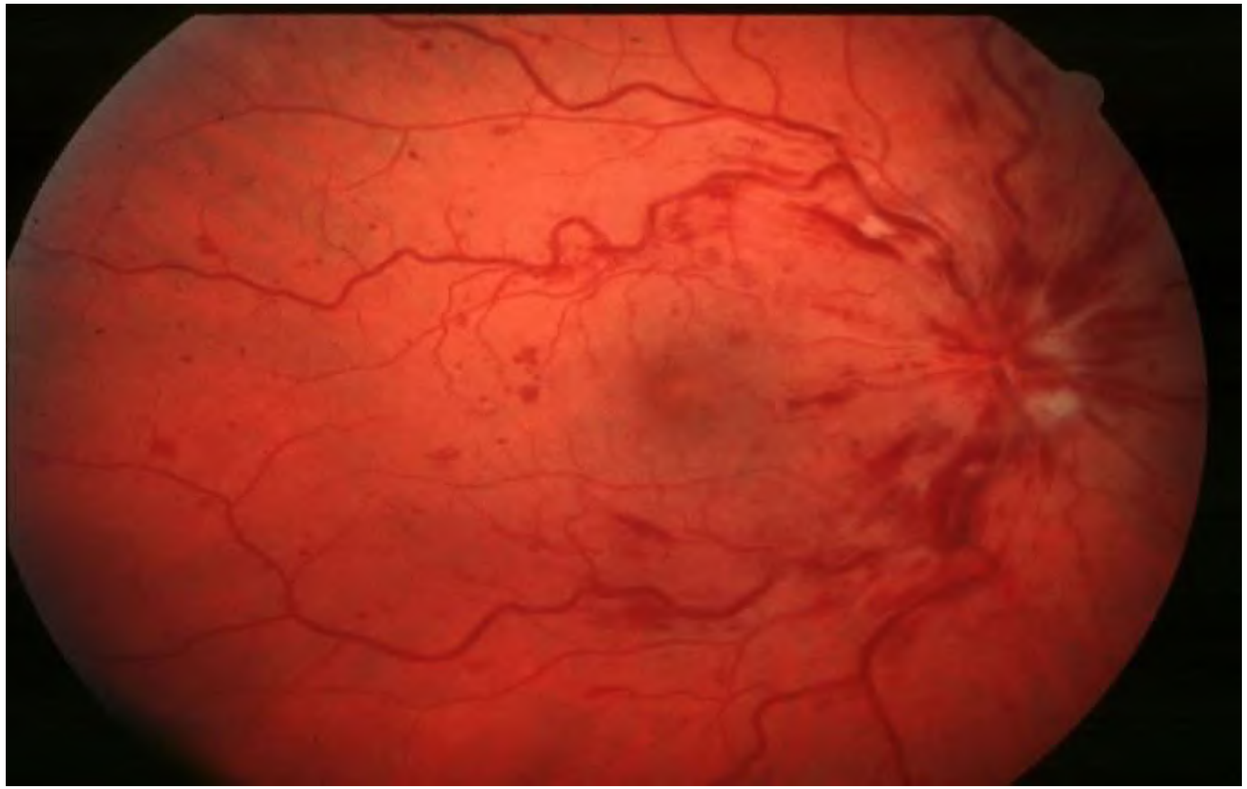
- Check every month for 6 months
- Look for rubeosis and angle new vessels

Treat neovascularization by panretinal photocoagulation

CRVO: Papillophlebitis

Uncommon – young adult Optic disc vasculitis / CRVO in young.

Affects healthy patients < 50 years



Mild blurring – worse in morning

- **VA – slight decrease**
- **RAPD - absent**
- **Venous tortuosity and dilatation**
- **Variable cotton-wool spots and haemorrhages**
- **Severe disc edema**
- **Very good prognosis in 80% (6/12 and better)**

CRVO: Treatment options

- Lasers
- Steroids
- Thrombolytic therapy
- Surgical Options

- Anti-VEGF

- Combination therapy

TREATMENT OPTIONS

- Anti-VEGF: Lucentis (Ranibizumab),

Avastin (Bevacizumab), Eylea (Aflibercept)

- **Ozerdex (Dexamethasone implant)**
- **Flucinolone acetate implant**
- **IVTA: Intra-Vitreal Triamcinolone**
- **Lasers**

- **Optic nerve sheathotomy / Neurotomy with PPV (Non-ischemic CRVO, ? Ischemic CRVO).**

- **PPV with cannulation & infusion of tPA in Non-ischemic CRVO.**

CRVO:TREATMENT OPTIONS

- LASERS:
  - (CVOS)
    - **Grid Laser reduced macular edema but no effect on vision**
    - **PRP (Angle/rubeosis/NVD/NVE)**
    - **Prophylactic PRP (Poor Compliance)**

Steroids

- **SCORE study: (Standard Care vs Corticosteriods for REtinal VO study)**

Observation vs 1mg IVTA vs 4mg IVTA (4mnthly)..

(BCVA gain of 15 letters or more…12 mnths)……. 7% vs 27% vs 26%

- **GENEVA Trial** (Global Evaluation of implaNtable dExamethasone in RVO with macular edemA):

  0.7mg vs 0.35mg dexamethasone implant vs sham.

  41% vs 40% vs 23% .. 15 letters improvement…. 90th day

  (Efficacy not sustained …… 180th day)

Ip MS et al. A randomized trial comparing the efficacy & safety of IVTA with observation to treat vision loss associated with ME secondary to CRVO:the Standard care vs Corticosteriods for RVO(SCORE) study report 5. Arch Ophthalmol 2009;127

Haller JA et al. Dexamethosone intravitreal implant in patients with ME related to BRV or CRVO 12 months study results.Ophthalmology2011;118

Treating CRVO: Anti-VEGF Ranibizumab

CRUISE study (Central R V occlUsIon Study: Evaluation of efficacy and safety).

Ranibizumab

HORIZON STUDY: Extension trial of CRUISE study (87% CRUISE patients) … **0.5mg** Ranibizumab (PRN)

RETAIN study (Ranibizumab 0.5mg)

- **Extension trial of Horizon {32 eyes (10.5%) of CRVO}**

Good <250 microns / Partial <10 % / Poor responders <1%
(Early AntiVEGF better outcome)

- **Brown Dm et al. Ranibizumab for ME following CRVO: 6mnths primary endpoint results of a phase III study. Ophthalmology 2010;117**
- **Heier JS et al. Ranibizumab for ME following CRVO: long term followupg in HORIZON trial. Ophthalmology 2012;119**

Treating CRVO: Anti-VEGF (VEGF Trap-Eye)

COPERNICUS Trial:

Ischemic & non ischemic / APD/duration 9mnths

Gain of 15 letters or more (2yr)

56% (treated) vs 12% (observed) / Decrease 457u vs 145u

GALILEO Trial (General Assesment Limiting InfiLtration of Exudates in CRVO with VEGF Trap-Eye:

60% (treated) vs 22% (sham) / Decrease 449u vs 169u

Boyer D et al. VEGF Trap-Eye for ME secondary to CRVO: 6mnth results of phase 3 COPERNICUS study. Ophthalmology 2012;119

Holz FG et al. VEGF Trap-Eye for ME secondary to CRVO: 6mnth results of phase 3 GALILEO. Br J Ophthalmol 2013; 97

Treating CRVO: Anti-VEGF (Bevacizumab)

- **Systemic associations**

  HTN (47%)

  DM = (23.5%)

Good Visual outcome = 58.8%

Stable Visual outcome= 41.2%

Mean BCVA (Initial)= 1.79 SD 0.87

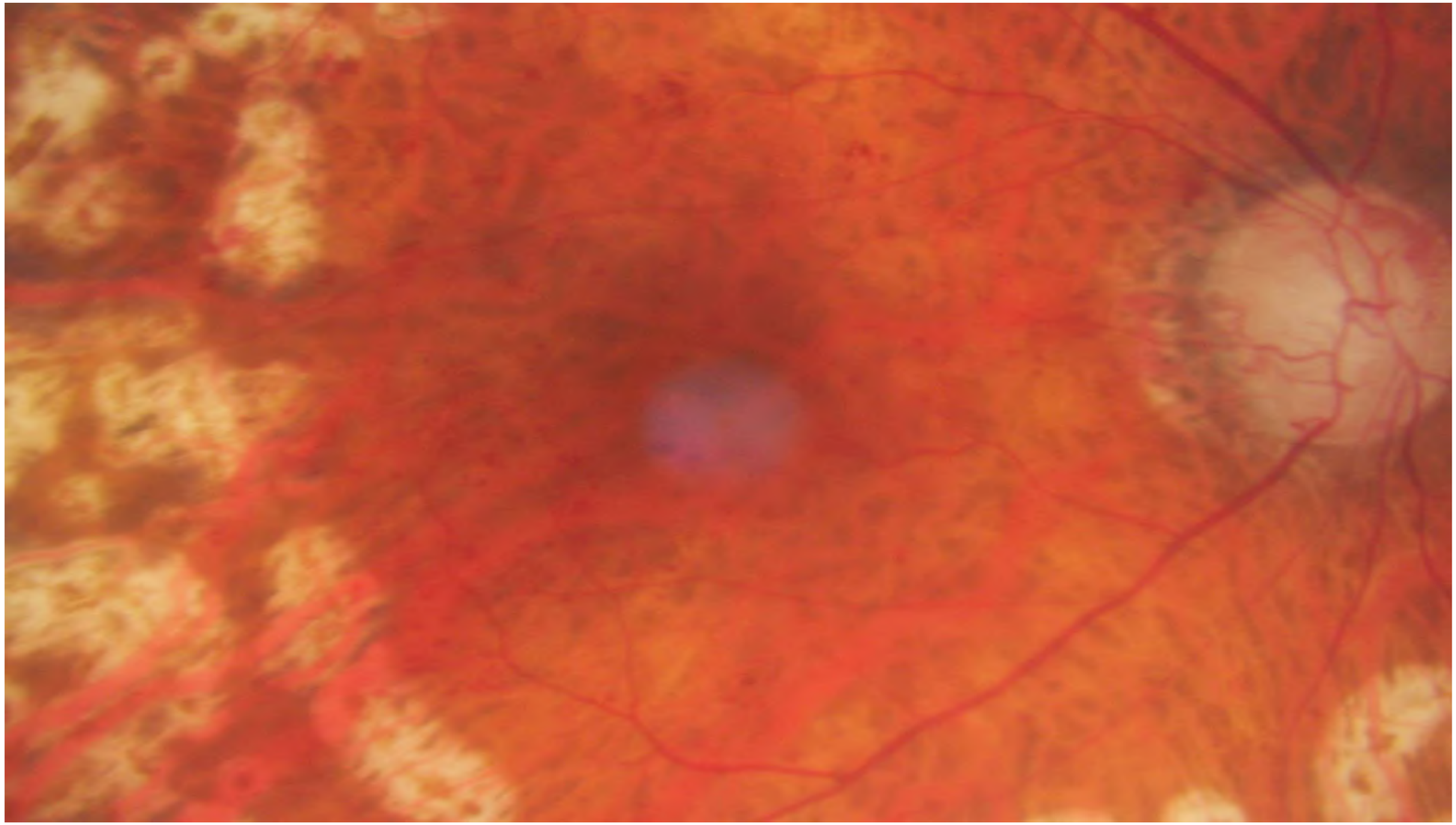
Mean BCVA (Final)= 1.18 SD 0.77

Mean Improvement = 0.63 SD 0.84 (P-value 0.008)

Jan S et al. Anti VEGF (Bevacizumab) in CRVO: An interventional case series. Pak J Med Research 2010; 49 (2):39-43

Treating CRVO: Anti-VEGF

Disc Collaterals



On the mountains of truth you can never climb in vain: either you will reach a point higher up today or you will be training your powers so that you will be able to climb higher tomorrow
“Friedrich Nietzsche”

BRANCH RETINAL VEIN OCCLUSION

- 6th – 7th decade
- Sudden onset – usually blurred vision
- Meta-morphosia
- Peripheral – occlusion – occasionally – No visual effects

Nasal BRVO may be asymptomatic

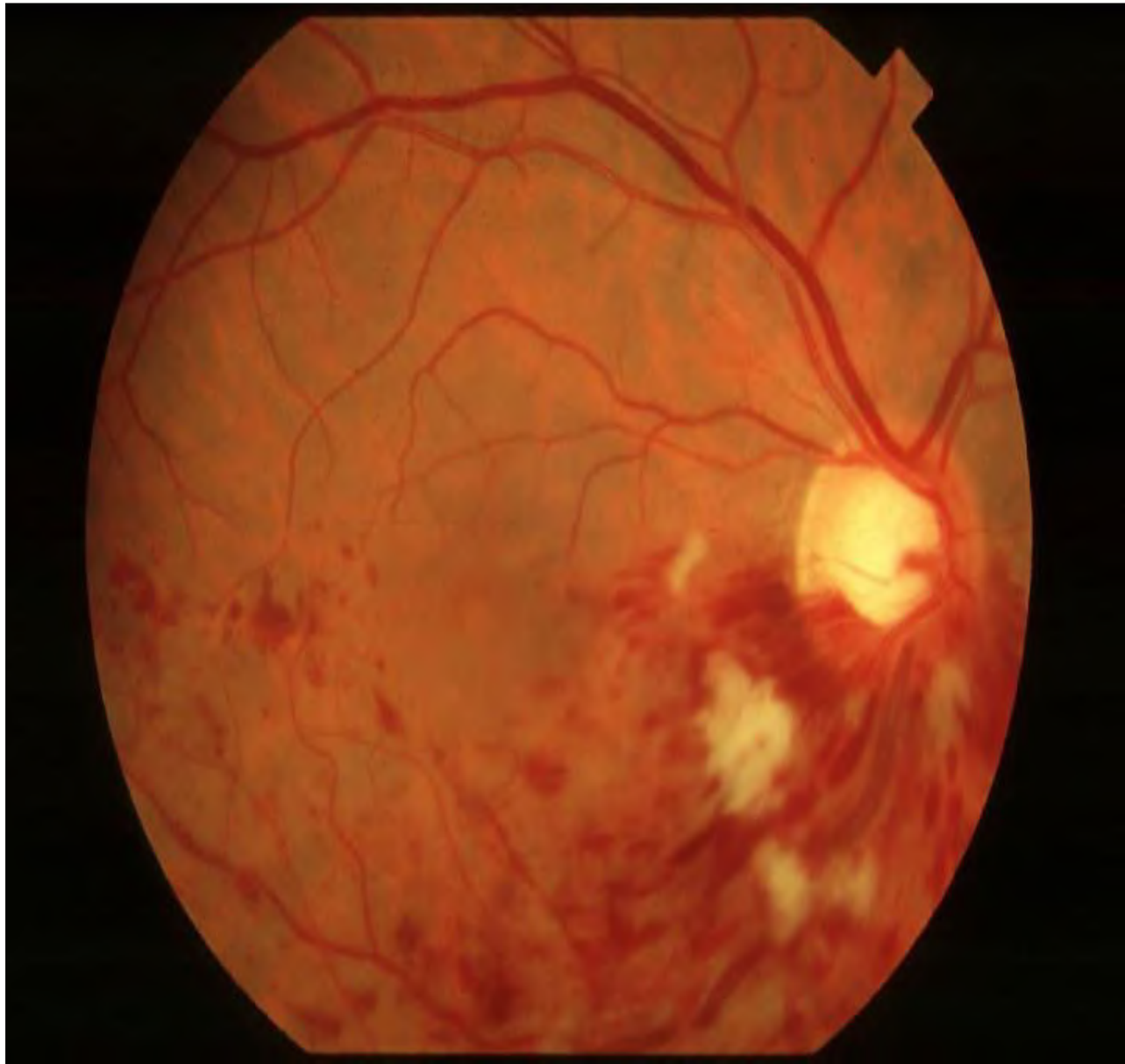
Mahsood YJ, Nazim M**, Sanaullah Jan**. **Comparison of visual outcome after intravitreal bevacizumab with standard and alone management protocol in branch retinal vein occlusion. Ophthalmology Update.** January-March 2016; 14 (1): 27-32.

Branch retinal vein occlusion (BRVO)

Signs of acute BRVO

- Most common Supero temporal
- Venous tortuosity and dilatation
- Retinal Edema
- Flame-shaped and ‘dot-blot’ haemorrhages
- Cotton-wool spots and retinal oedema

All in part of retina drained by affected vein



Branch retinal vein occlusion (BRVO)

LATER ON:

Hemorrhages start resolving --- Hard exudates start forming

OLD OCCLUSION:

- Vascular sheathing – Collaterals - Hard exudates
- Cholesterol crystals deposition may be present
- RPE degeneration at macula

Initially V.A decrease – hemorrhage / macular edema

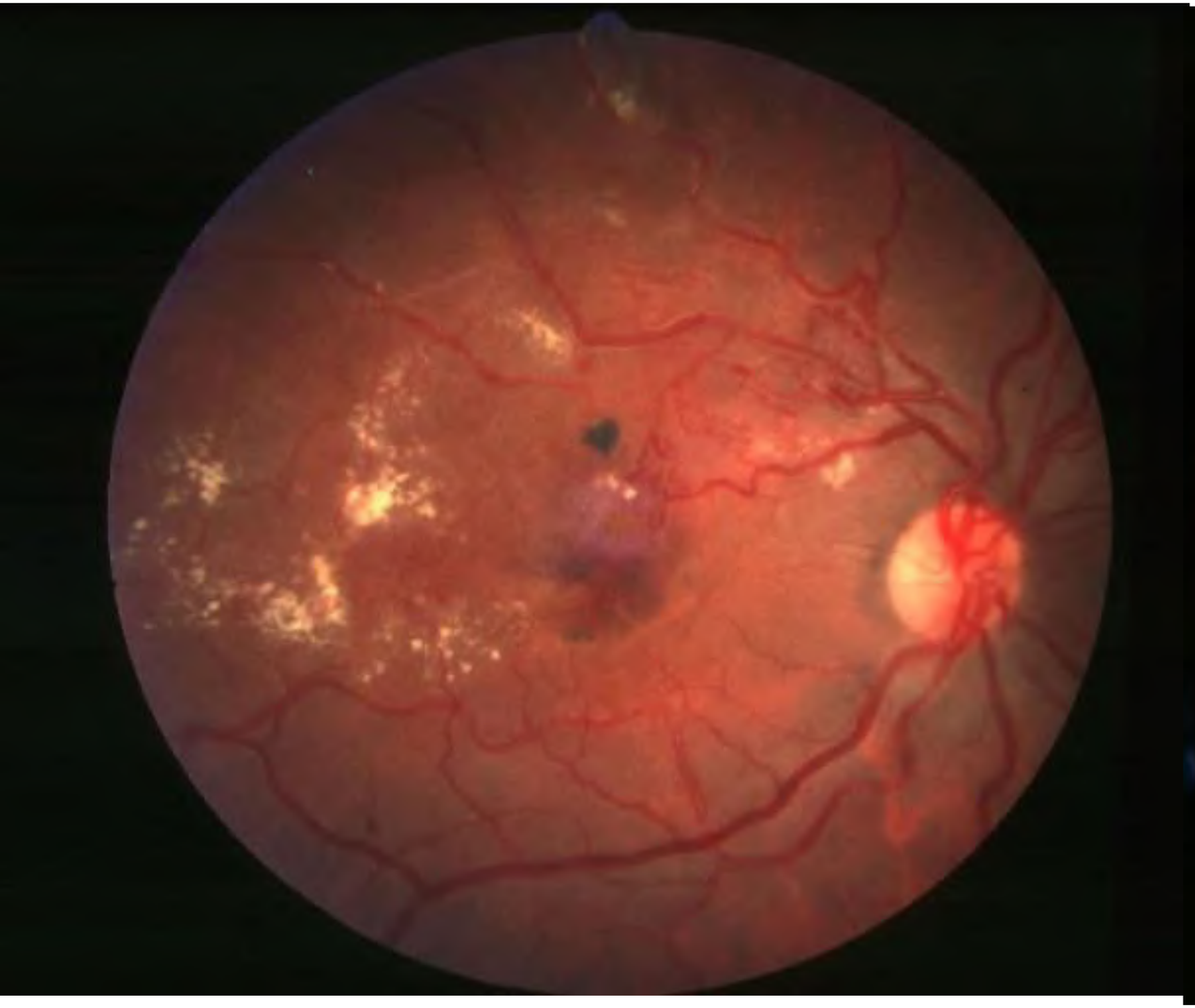
Risk of CRVO/ BRVO in 2nd eye – 10%

COMPLICATIONS: Chronic macular edema, Upto 60% of BRVO-Neovascularization

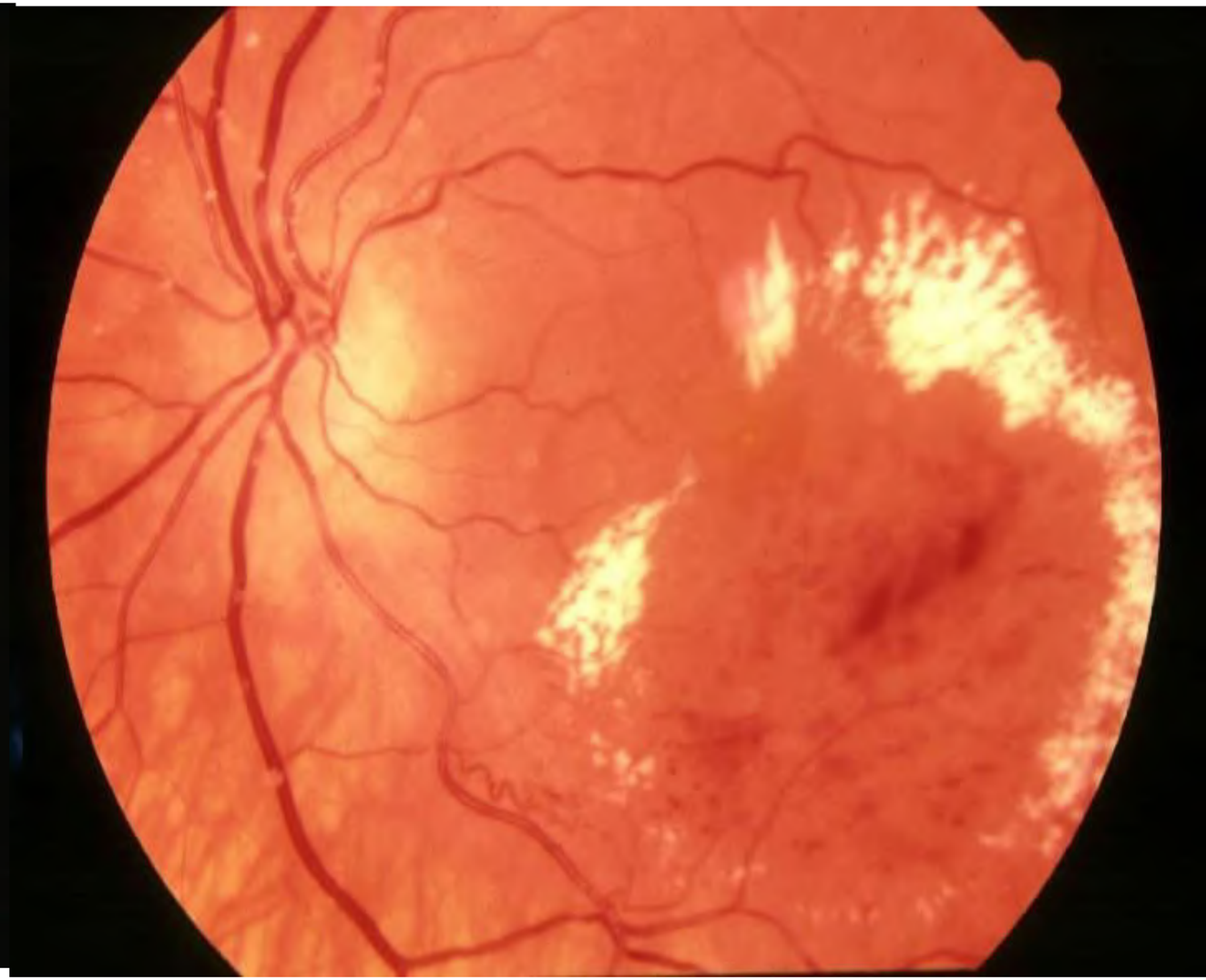
May Present with floaters & defective vision

With 6 months – 50% eyes develop collaterals with return of V.A to 6/12 or better

Signs of old branch retinal vein occlusion

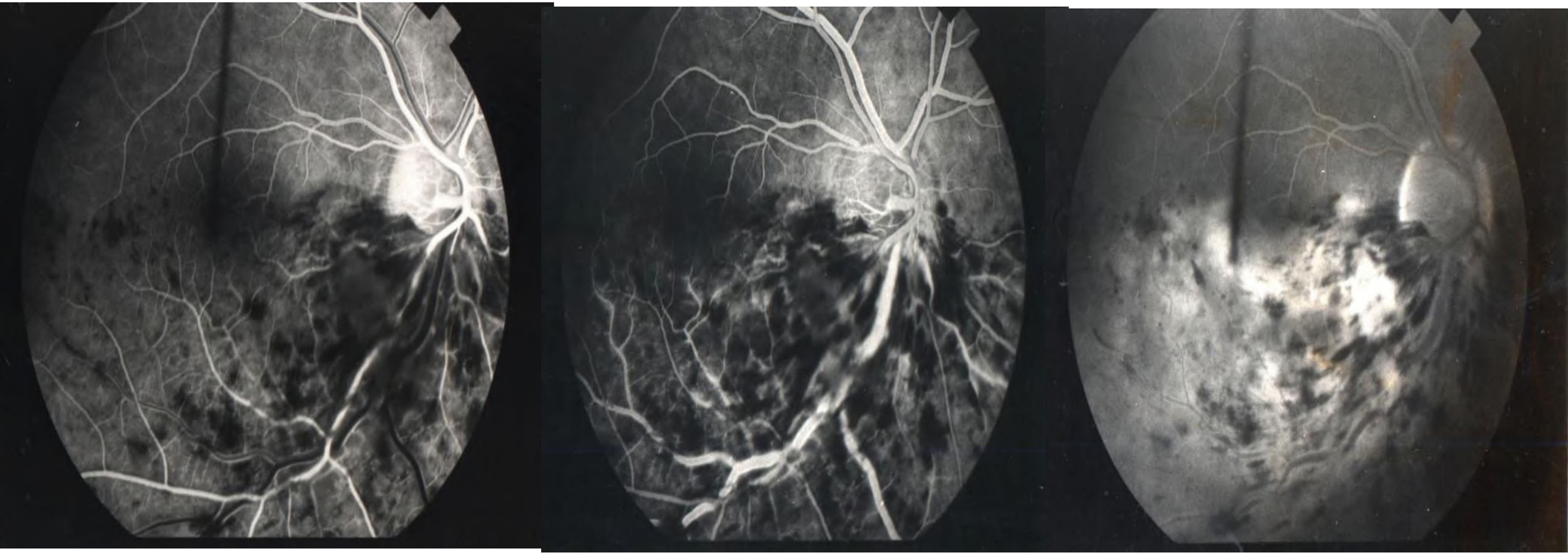


Vascular sheathing and collaterals



Hard exudates

FA of branch retinal vein occlusion



Early - blocked background fluorescence due to haemorrhage

Late – hyperflurorescence due to diffuse edema

Branch retinal vein occlusion

CHRONIC MACULAR EDEMA:

Most common cause of persistent V.A decrease after BRVO

Now standard treatment is Anti VEGF

B V O Study

→ Wait for 6 – 12 wks F.F.A

→ If macular non-perfusion – No Rx.

→ If macular edema with VA 6/12 or worse at 3mnths

→ Grid laser / laser leaking areas

→ Don’t Rx collaterals.

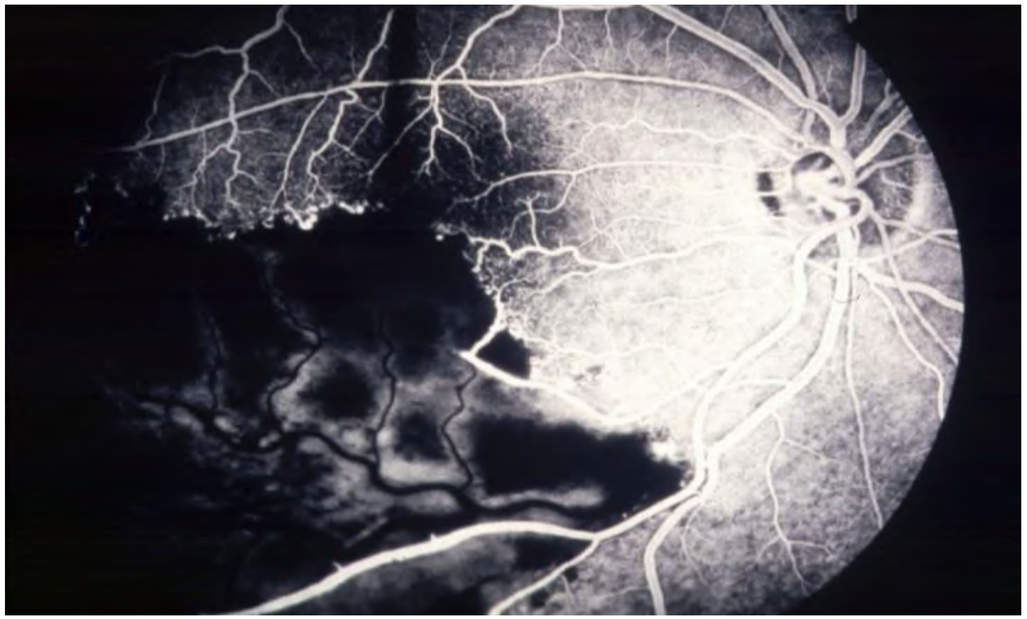
Follow up after 3mnths – if macular edema persists – Re Rx.

Management of chronic macular oedema

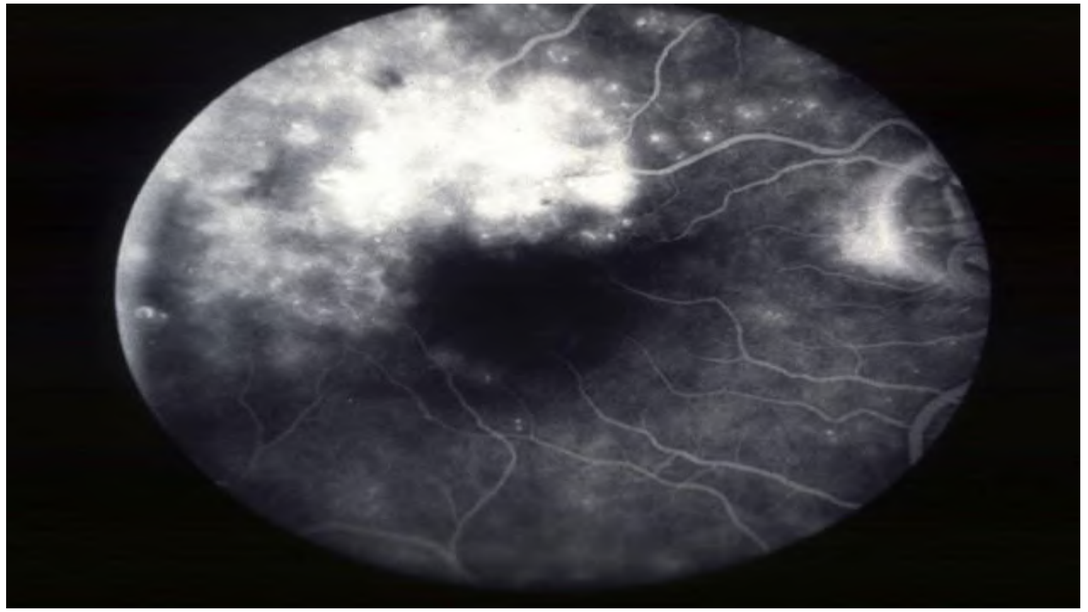
- **BVOS**

•Most common cause of persistent poor VA

- **Wait 6-12 weeks and perform FA**



Macular non-perfusion - no treatment



Good macular perfusion and VA 6/18 or worse after 3 months - consider laser photocoagulation

NEOVASCULARIZATION: NVD/NVE 30-50%

ISCHEMIC BRVO: > 5 disc diam non-perfusion

NON-ISCHEMIC BRVO: < 5 disc diam non-perfusion

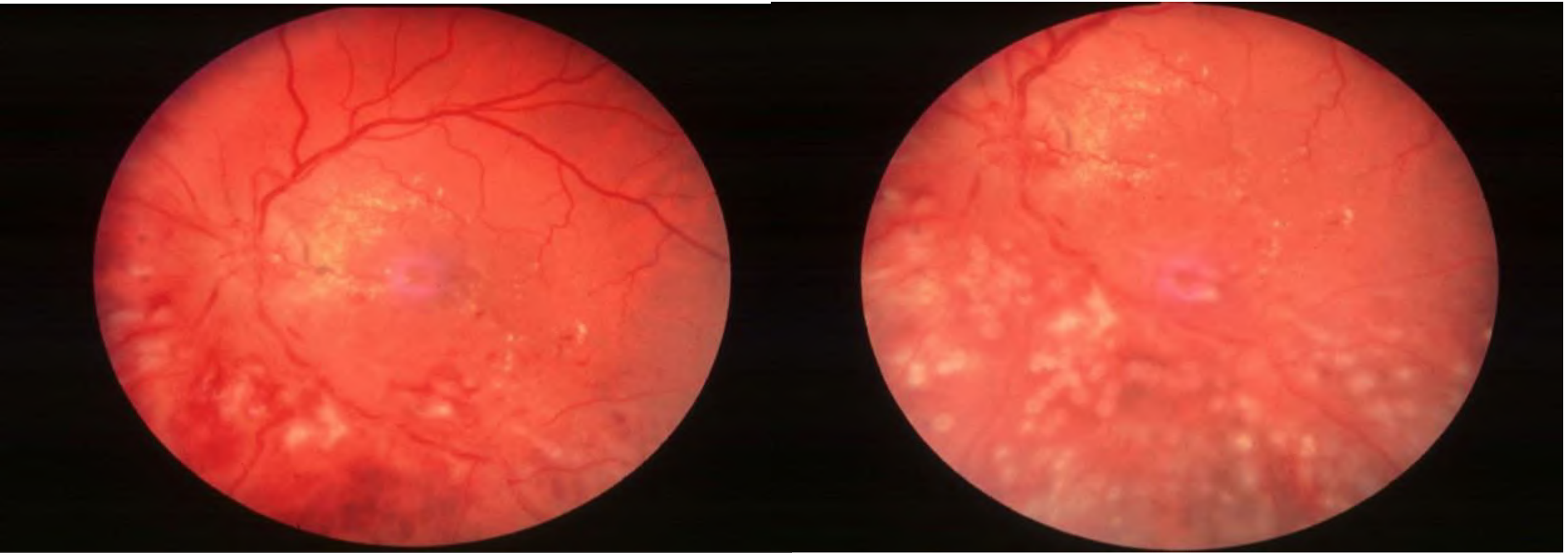
NVD – Eye with extensive non-perfused area

Neo-vessels – usually during initial 6-12months but any time with in first 3 years

F.F.A Ischemic BRVO – 4 monthly follow up

If NVD, NVE, Rubeosis :
Argon laser to affected segment / Anti VEGF.

Management of neovascularization



- Occurs in about 30-50% of eyes
- Most frequently after 6-12 months

- Perform laser photocoagulation to involved segment

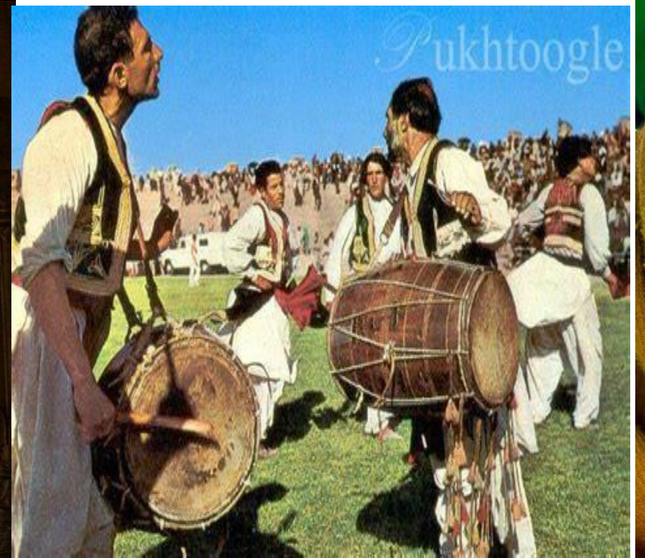
TREATMENT OPTIONS

- Anti-VEGF: Lucentis (Ranibizumab),

Avastin (Bevacizumab), Eylea (Aflibercept)

- **Ozerdex (Dexamethasone implant)**
- **Flucinolone acetate implant**
- **IVTA: Intra-Vitreal Triamcinolone**
- **Lasers**

Thanks



RETINAL DETACHMENT

Dr Sanaullah Jan

16th Khyber EyeCon 2023 in conjunction with 7th PVRS conference

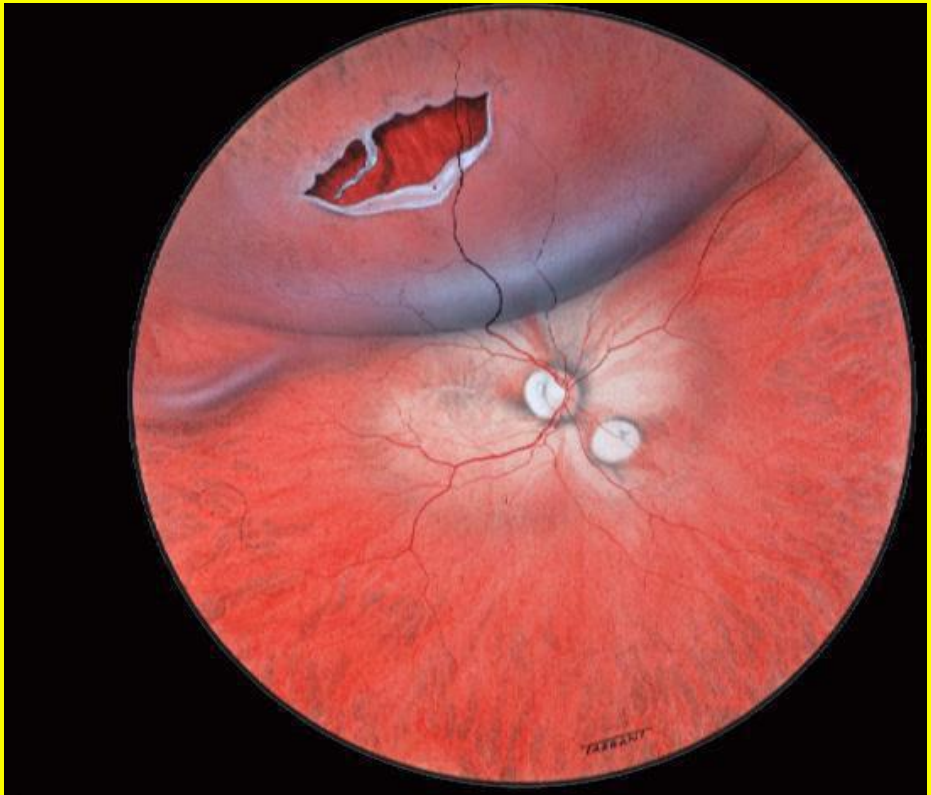
- 7th – 9th Feb 2025
- Serena Hotel, Peshawar

- Under Graduate Session
- Poster Presentation

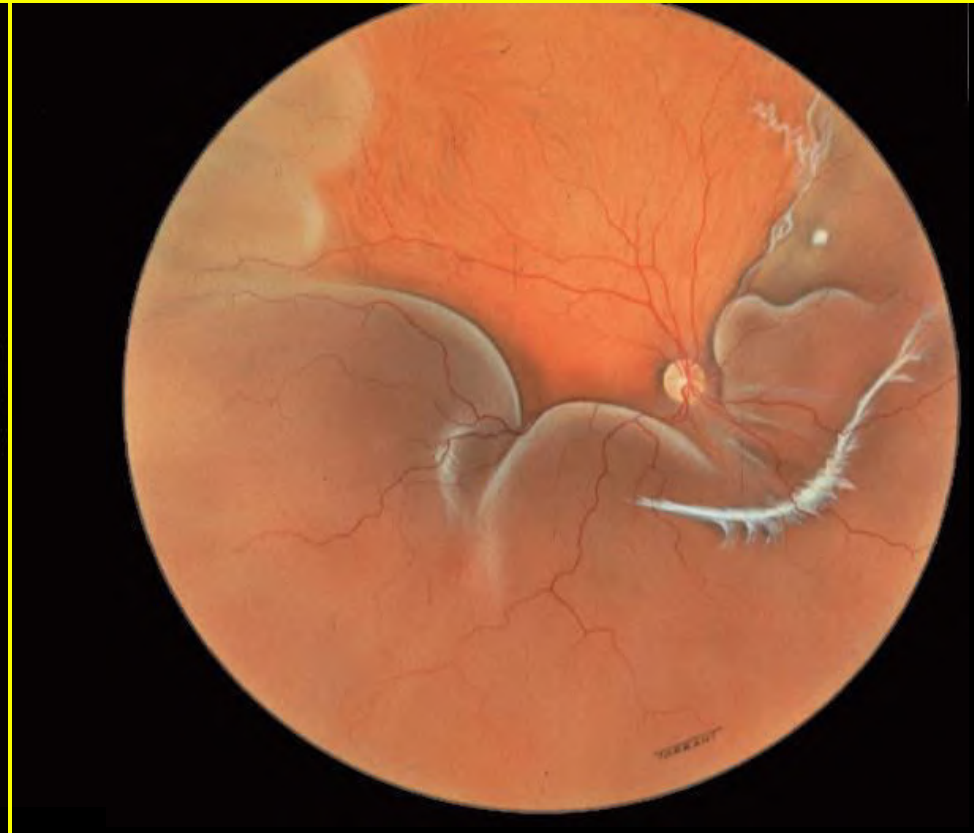
Best Presentation/Poster Award

Retinal detachment (RD)

- Separation of sensory retina from RPE by subretinal fluid (SRF)



Rhegmatogenous - caused by a retinal break

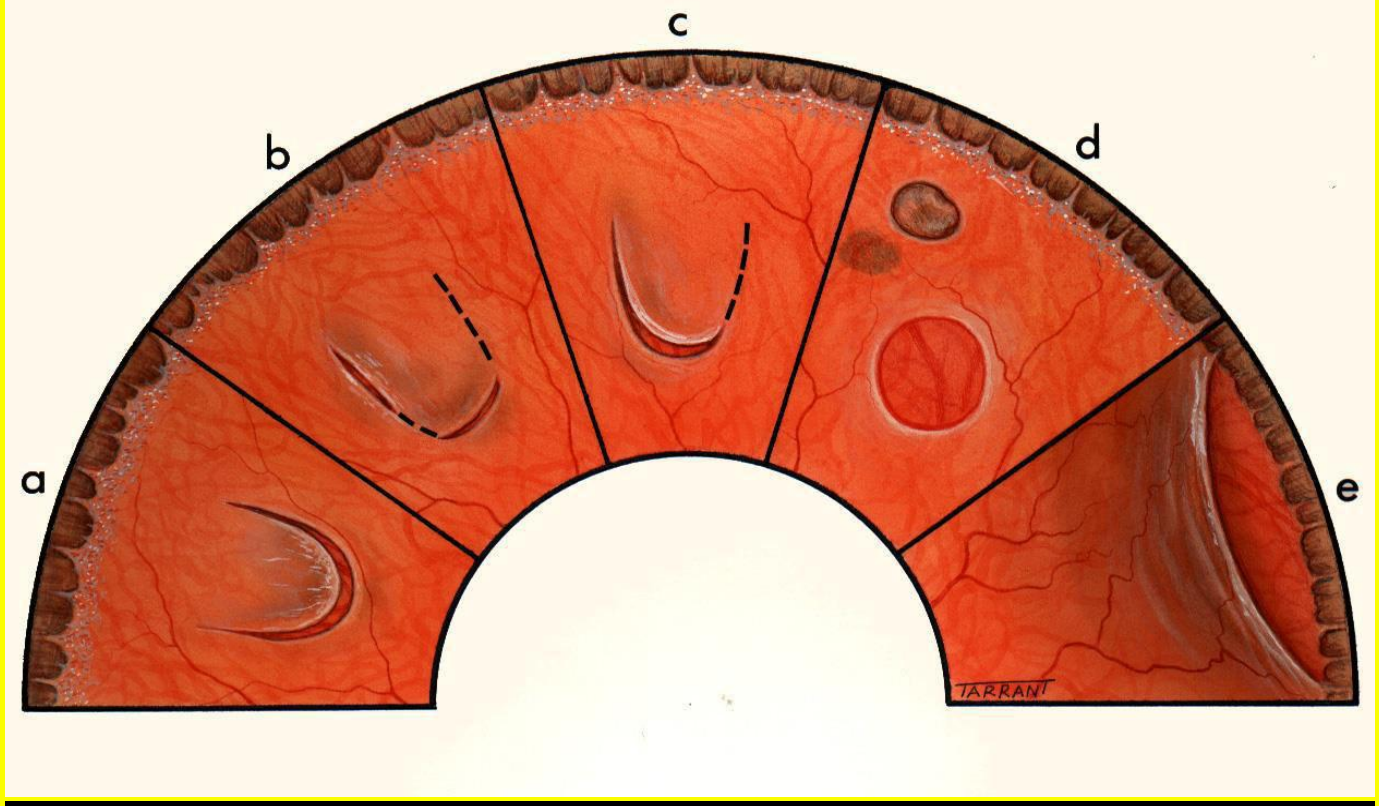


Non-rhegmatogenous - tractional or exudative

Definition and classification

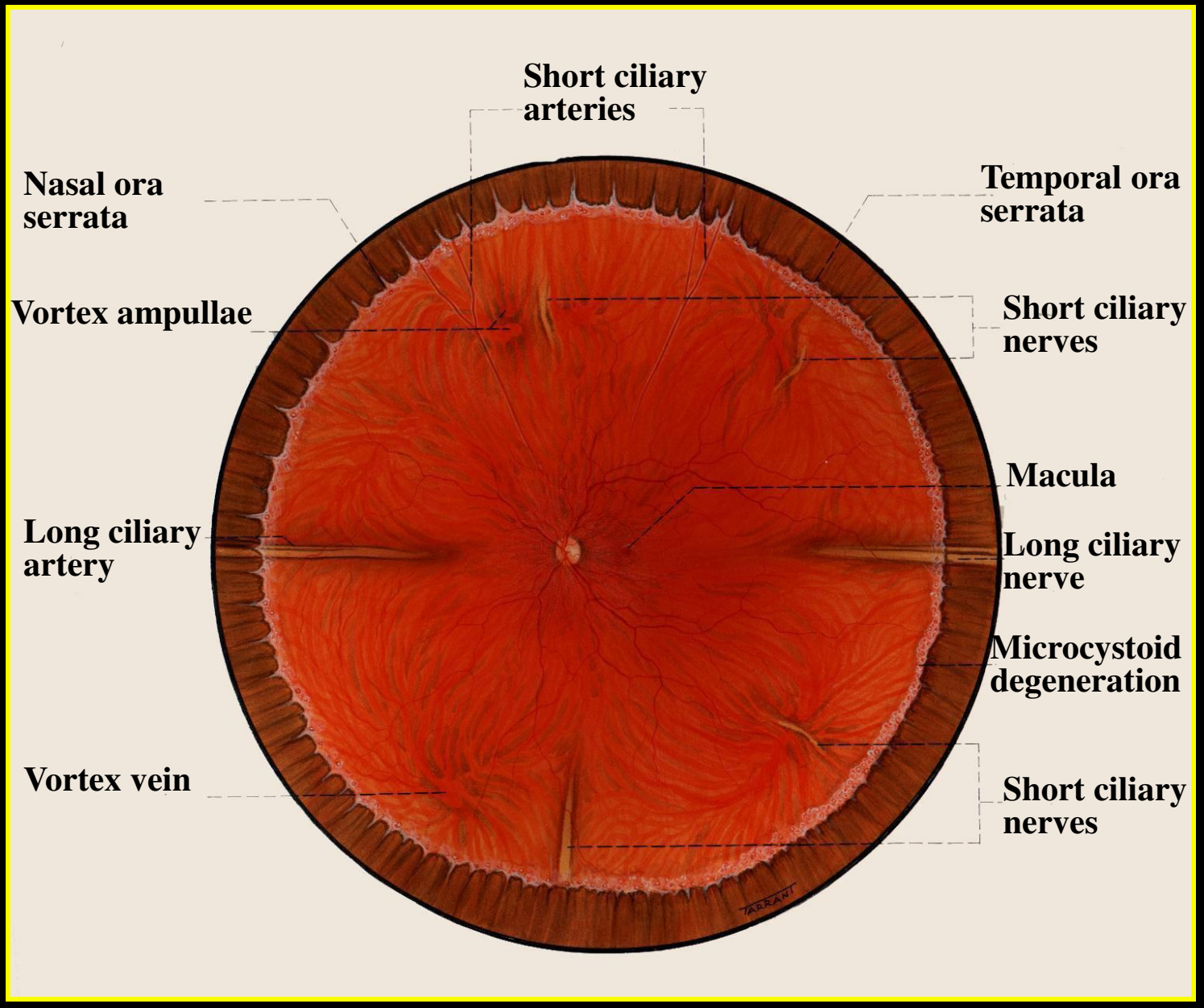
- Break - full-thickness defect in sensory retina
- Hole - caused by chronic retinal atrophy
- Tear - caused by dynamic vitreoretinal traction

Morphology of tears

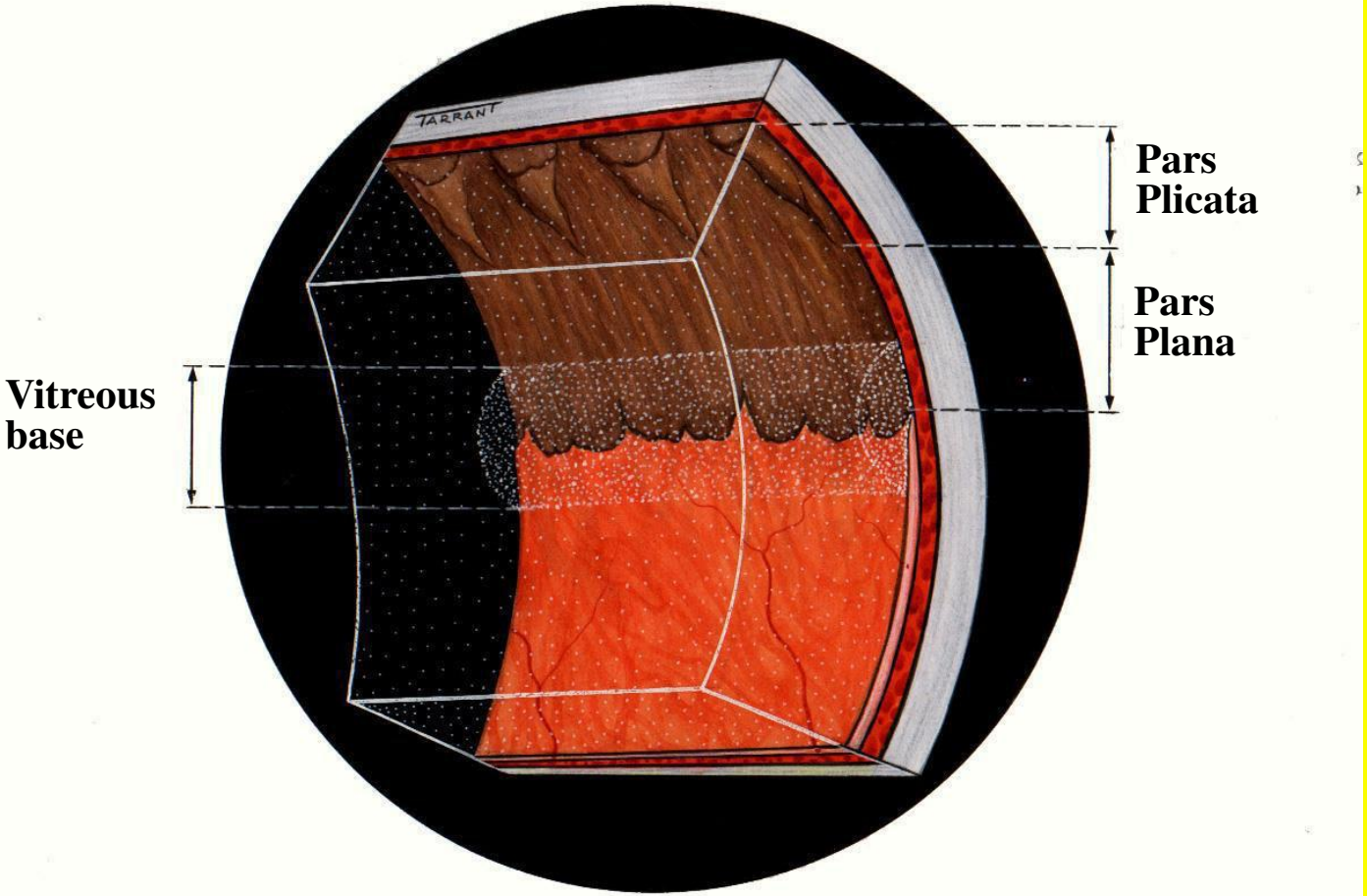


a. Complete U-tear
b. Linear
c. Incomplete L-shaped
d. Operculated
e. Dialysis

Normal anatomical landmarks



Anatomy of vitreous base



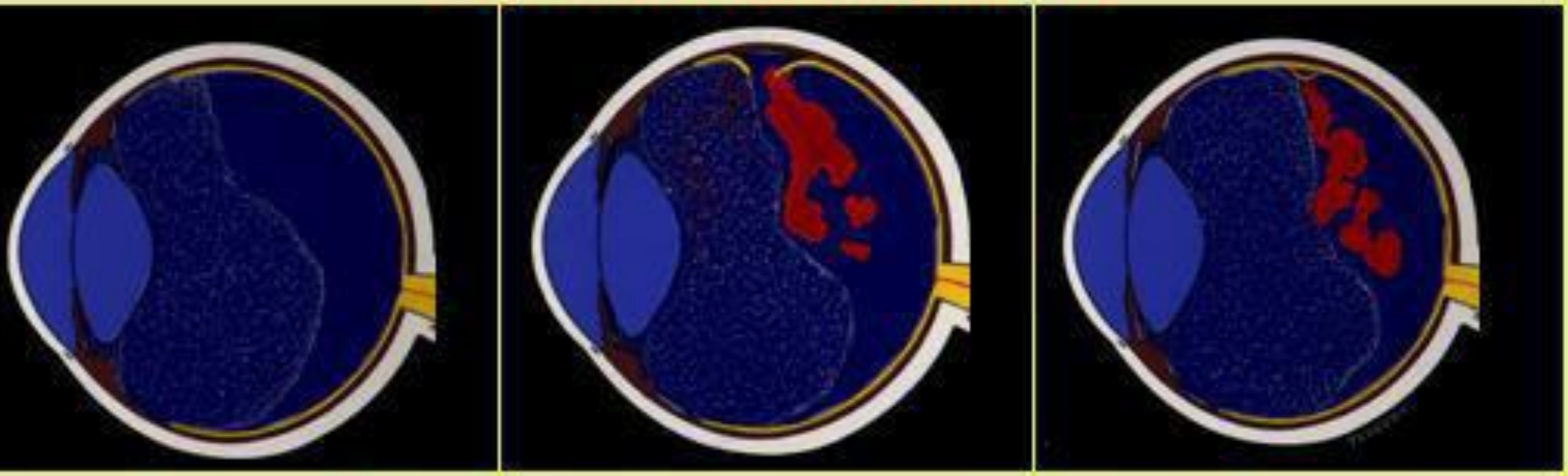
- 3-4 mm wide zone straddling ora serrata
- Strong adhesion of cortical vitreous
- Anterior limit of posterior vitreous detachment

Pathogenesis of rhegmatogenous RD

Two components for retinal break formation

- Acute posterior vitreous detachment (PVD)
- Predisposing peripheral retinal degeneration

Possible sequelae of acute PVD



Uncomplicated PVD (85%)	Retinal tear formation and haemorrhage (10-15%)	Avulsion of retinal vessel and haemorrhage (uncommon)

Retinal Detachment

Annual incidence 10/10,000

Bilaterality 10 %

Symptoms:

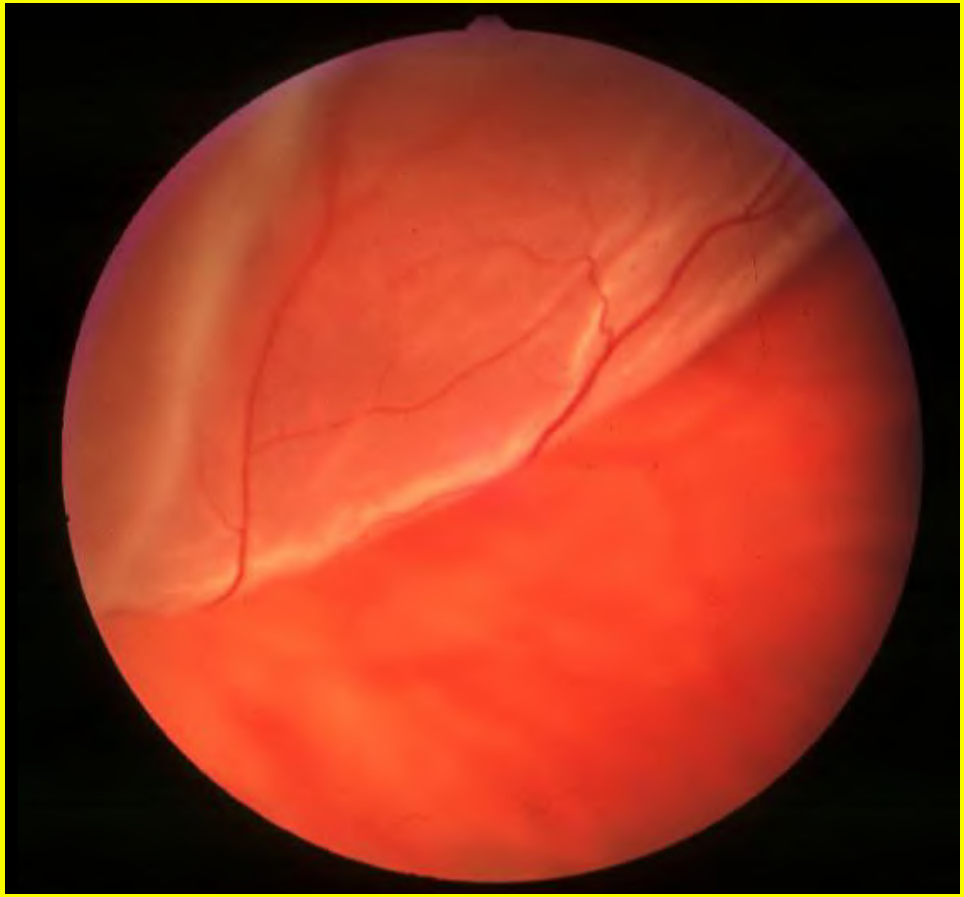
- Flashes
- Floaters
- Visual field defect
- Visual acuity

Retinal Detachment

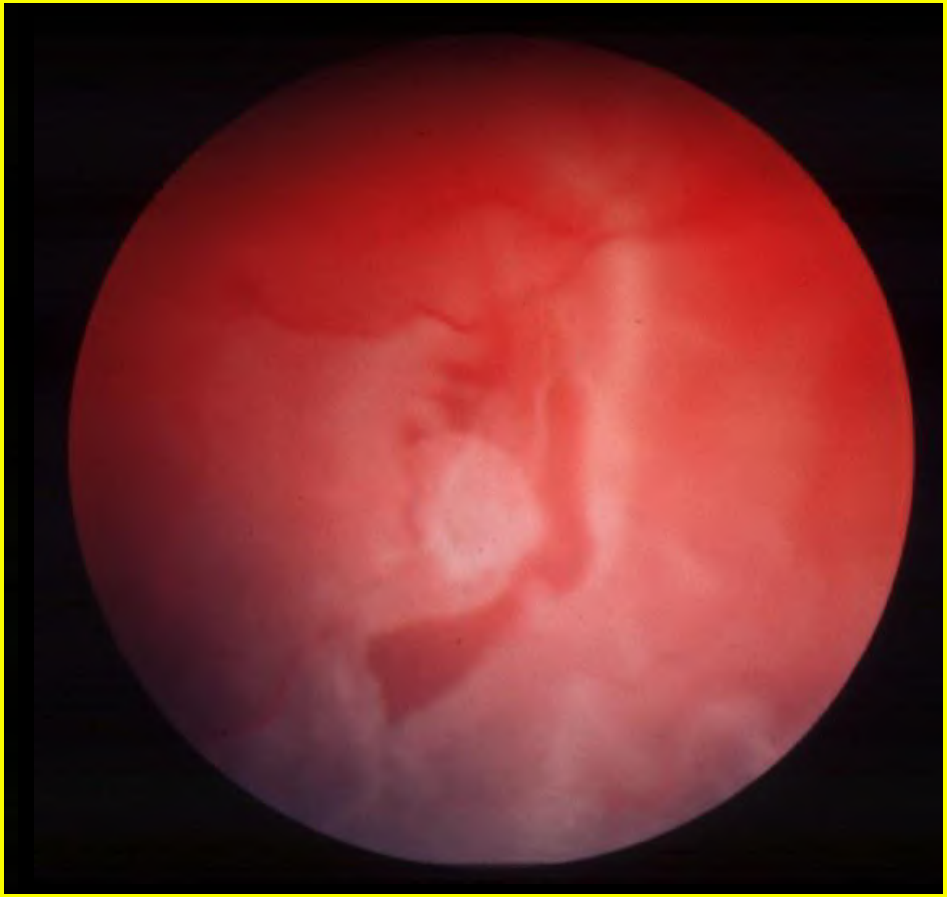
- BCVA
- Visual field analysis
- A/C (cells+)
- Tobacco’s dust
- PVD (Weis ring)
- IOP (hypotony/raised)

Fresh rhegmatogenous RD – signs

- Annual incidence - 1:10,000 of population
- Eventually bilateral in 10%

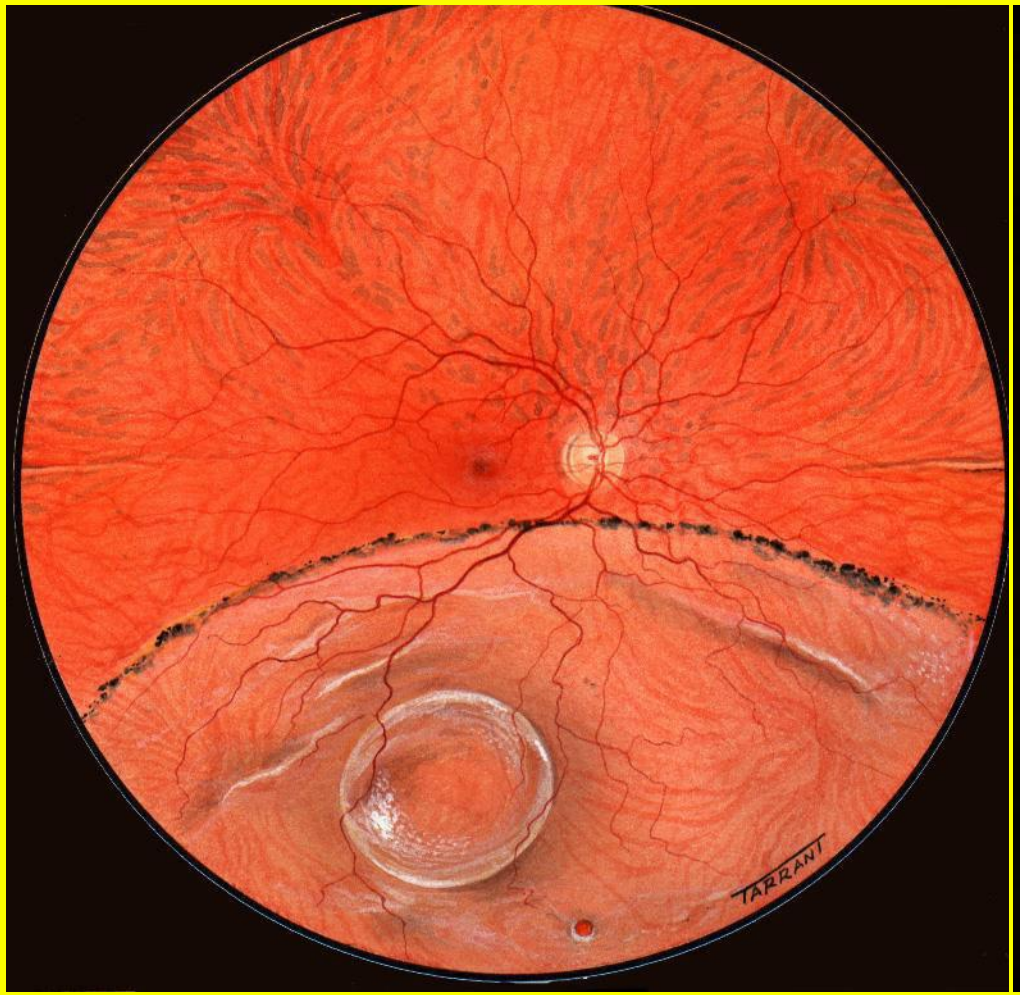


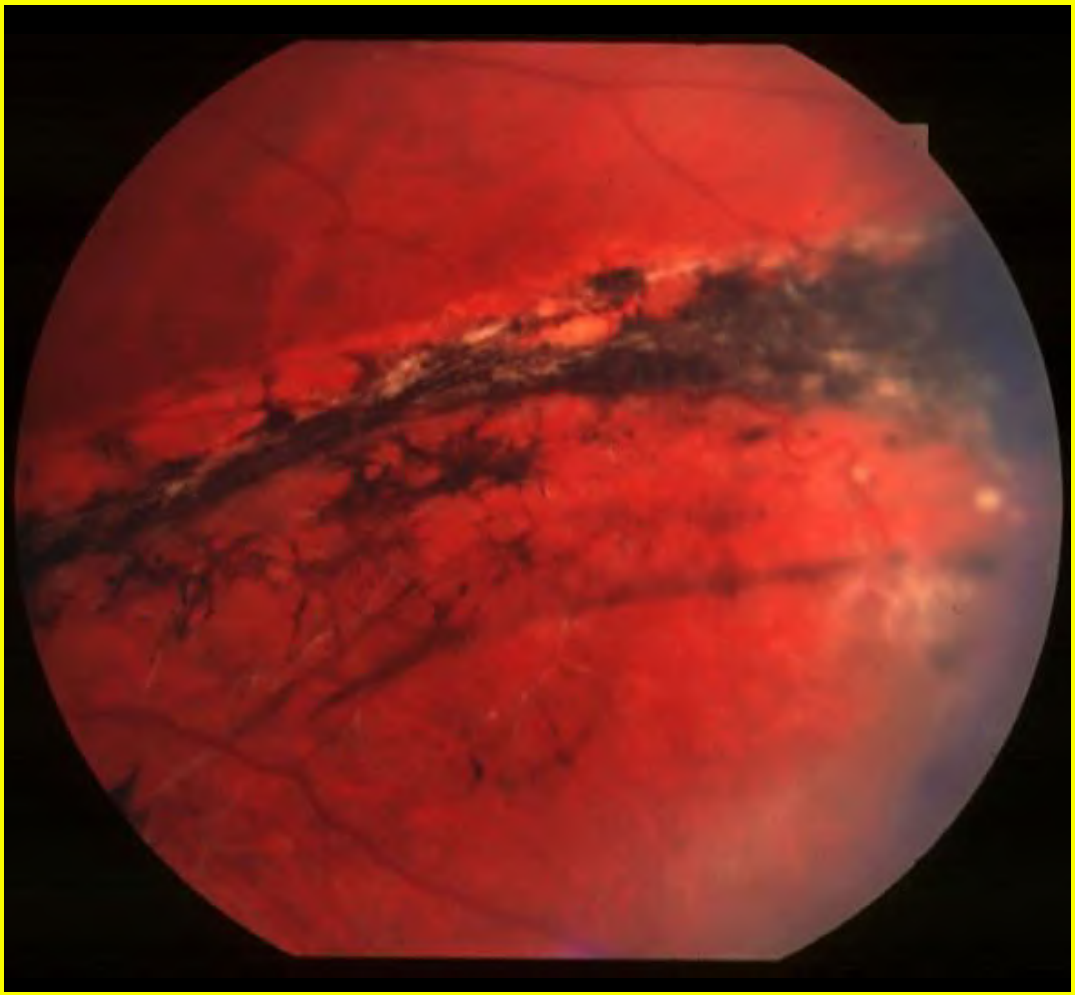
- Convex, mobile elevation extending to ora serrata and disc
- Slightly opaque with dark blood vessels



- Loss of choroidal pattern
- Retinal breaks

Longstanding rhegmatogenous RD - signs

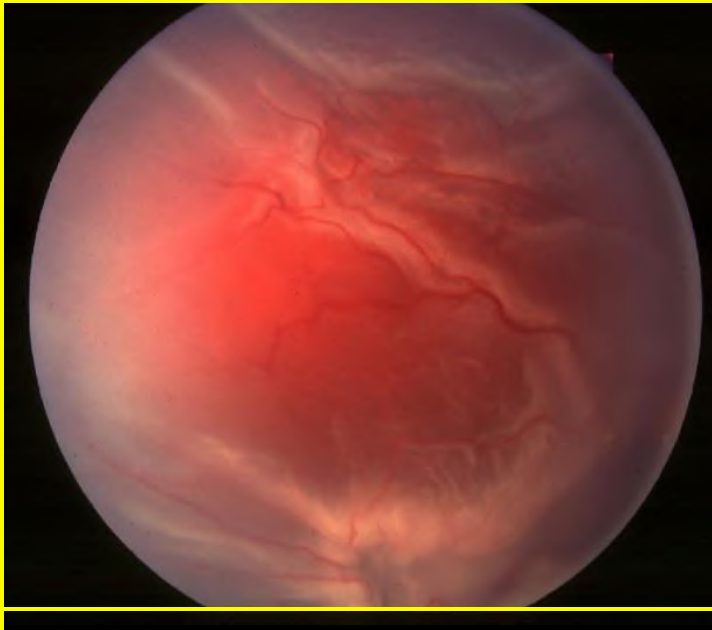
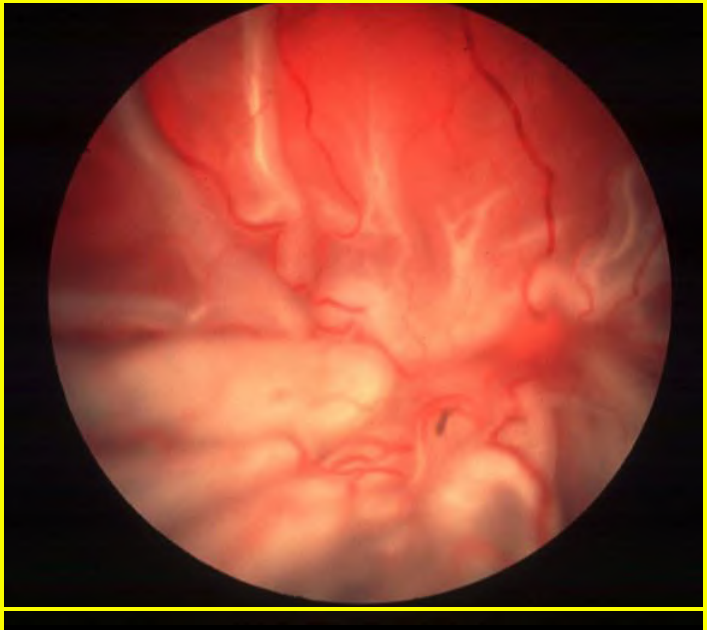




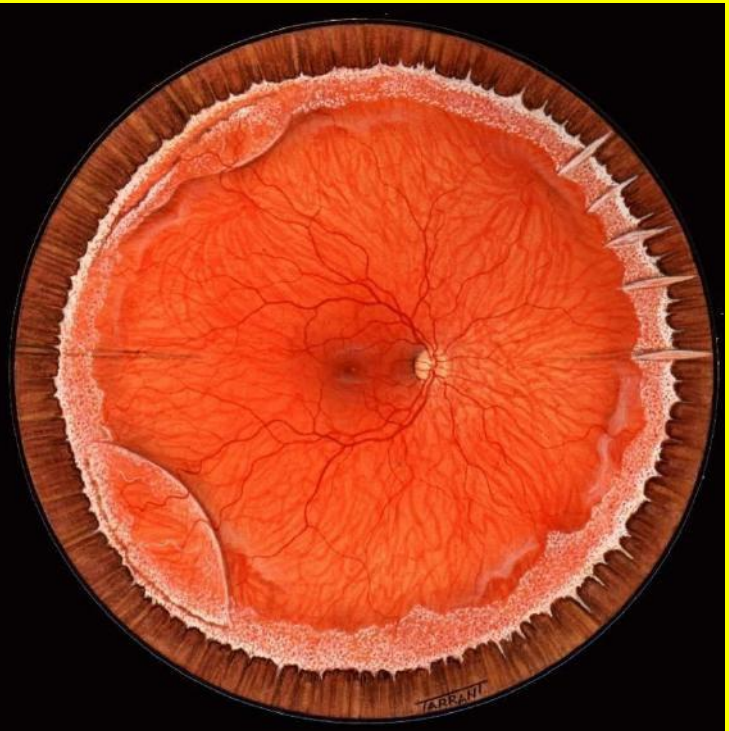
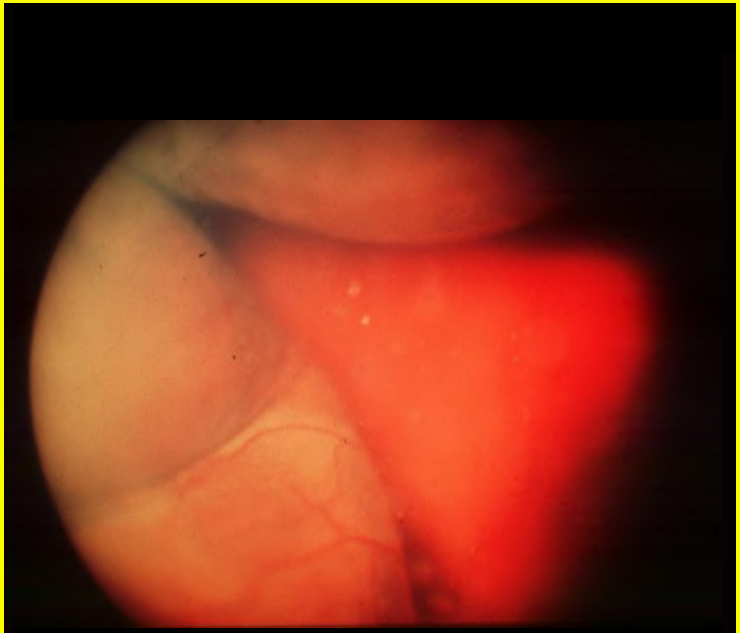
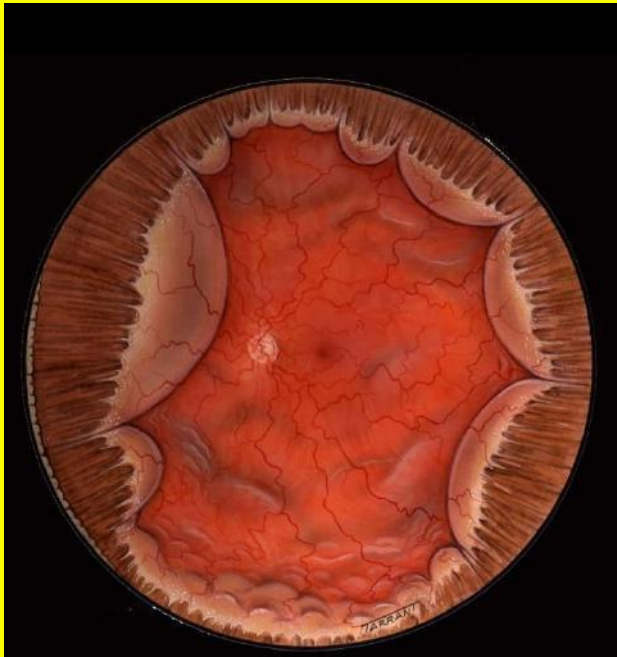
- Frequently inferior with small holes
- Very thin retina
- Secondary intraretinal cysts

- Demarcation lines (high-water marks)

Proliferative vitreoretinopathy

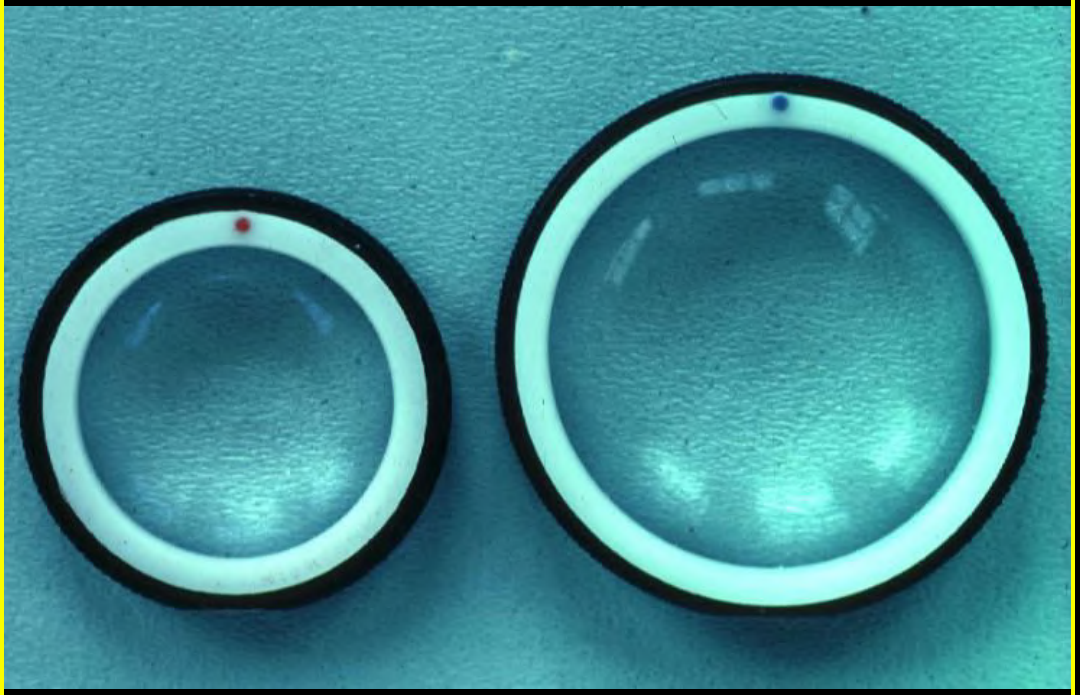
Grade A (minimal)	Grade B (moderate)	Grade C (severe)
		
• Vitreous haze and tobacco dust	• Retinal wrinkling and stiffness • Rolled edges of tears	• Rigid retinal folds • Vitreous condensations and strands

Differential diagnosis of RD

Degenerative retinoschisis	Choroidal detachment	Uveal effusion syndrome
		
• Frequently bilateral • Smooth, thin and immobile • Occasionally breaks in one or both layers	• Associated with hypotony • Unilateral, brown, smooth, solid and immobile • Ora serrata may be visible	• Idiopathic • Rare, unilateral • Combined choroidal and exudative detachments

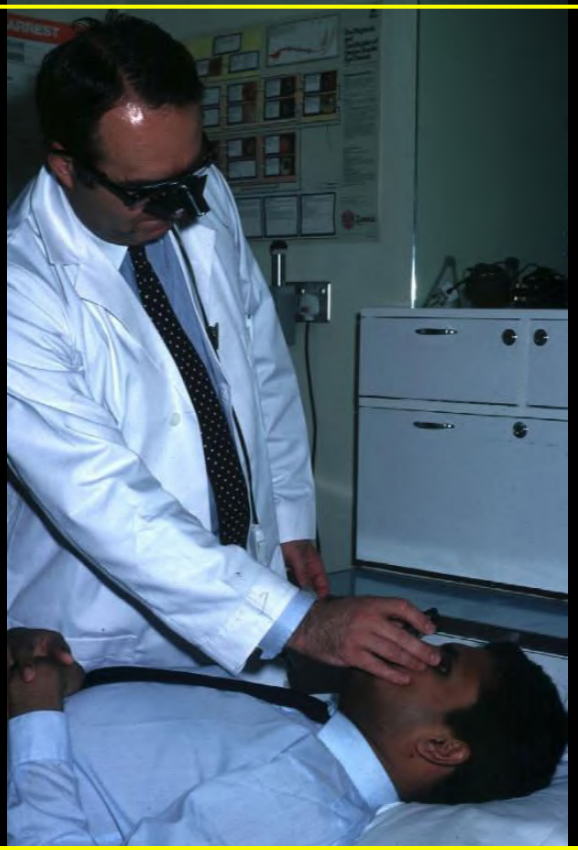
Indirect ophthalmology

Condensing lenses



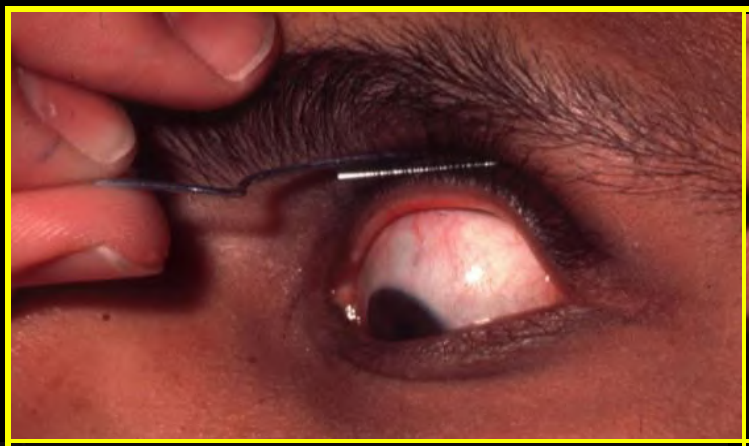
- The higher the power, the less the magnification, the shorter the working distance but the greater the field of view

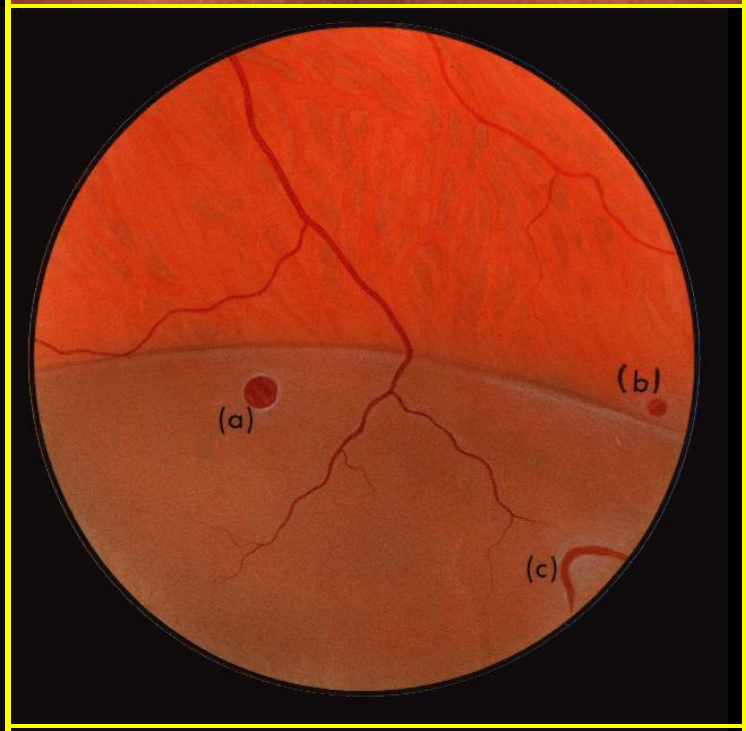
Technique

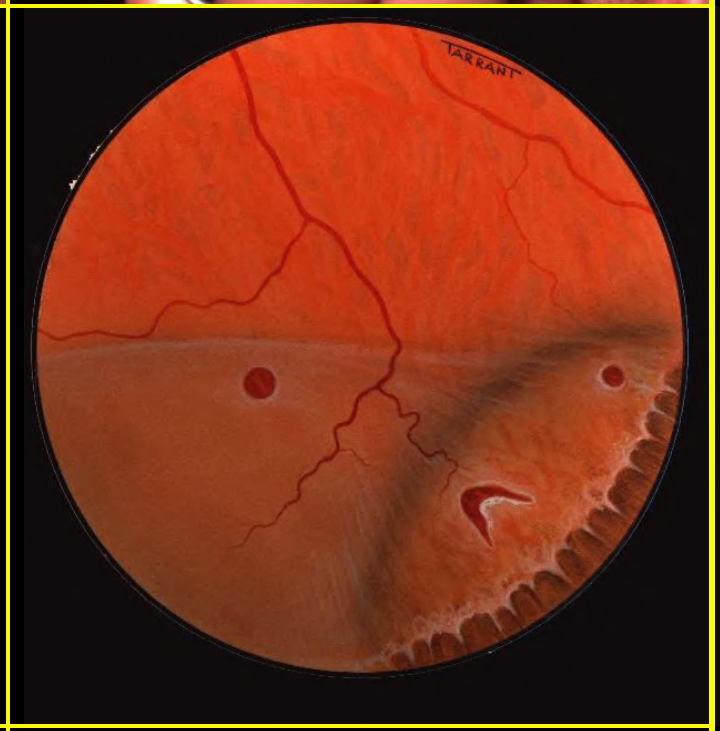


- Keep lens parallel to patient's iris plane
- Avoid tendency to move towards patient
- Ask the patient to move eyes and head into optimal positions for examination

Scleral indentation







Retinal breaks in detached retina without indentation

Enhanced visualization of breaks with indentation

Fundus drawing

Technique

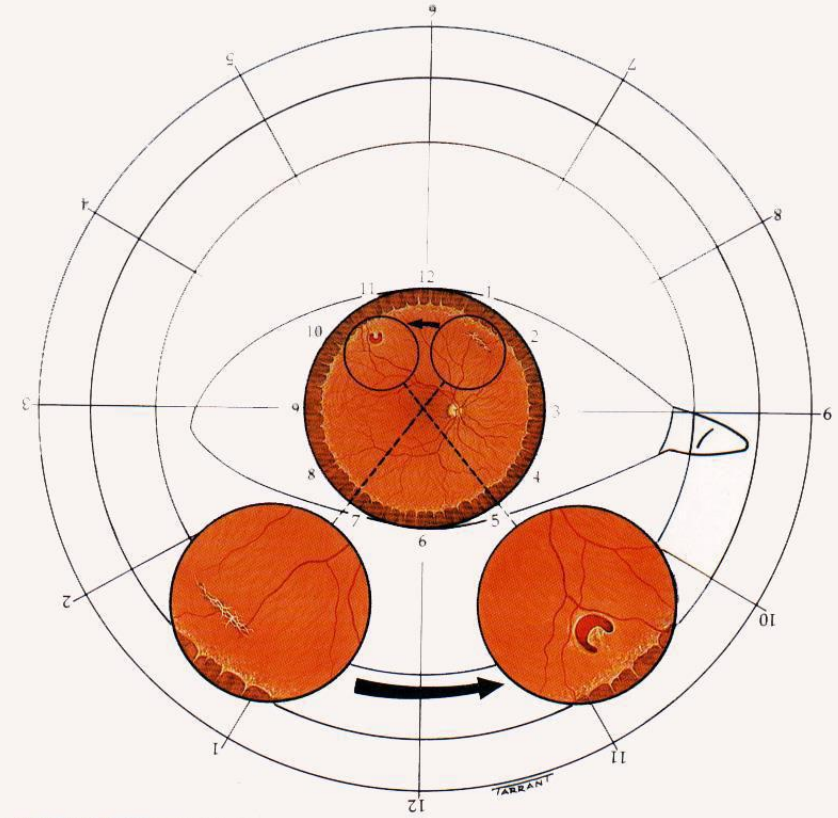
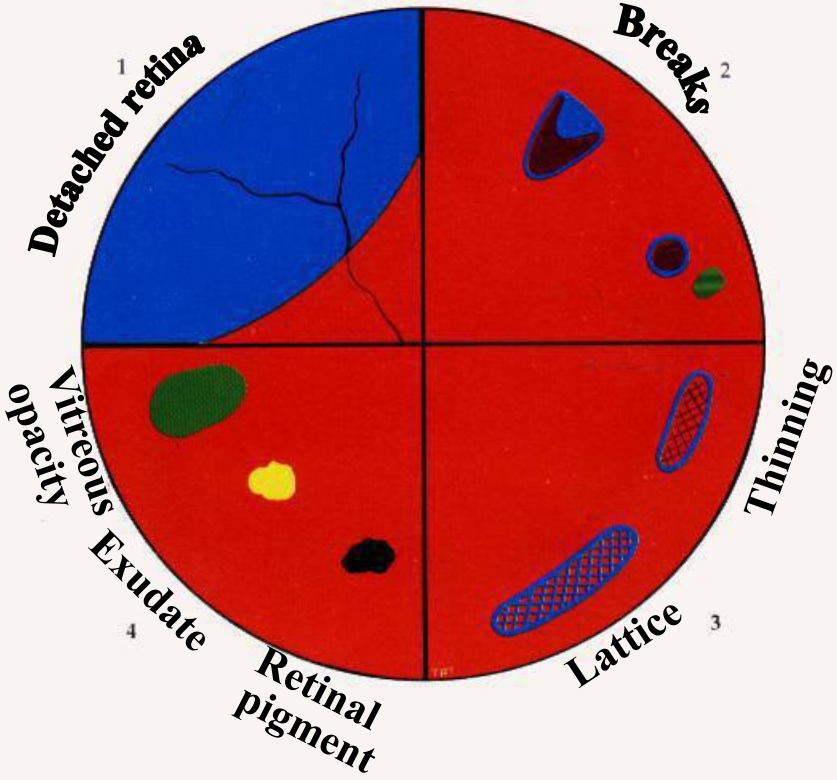


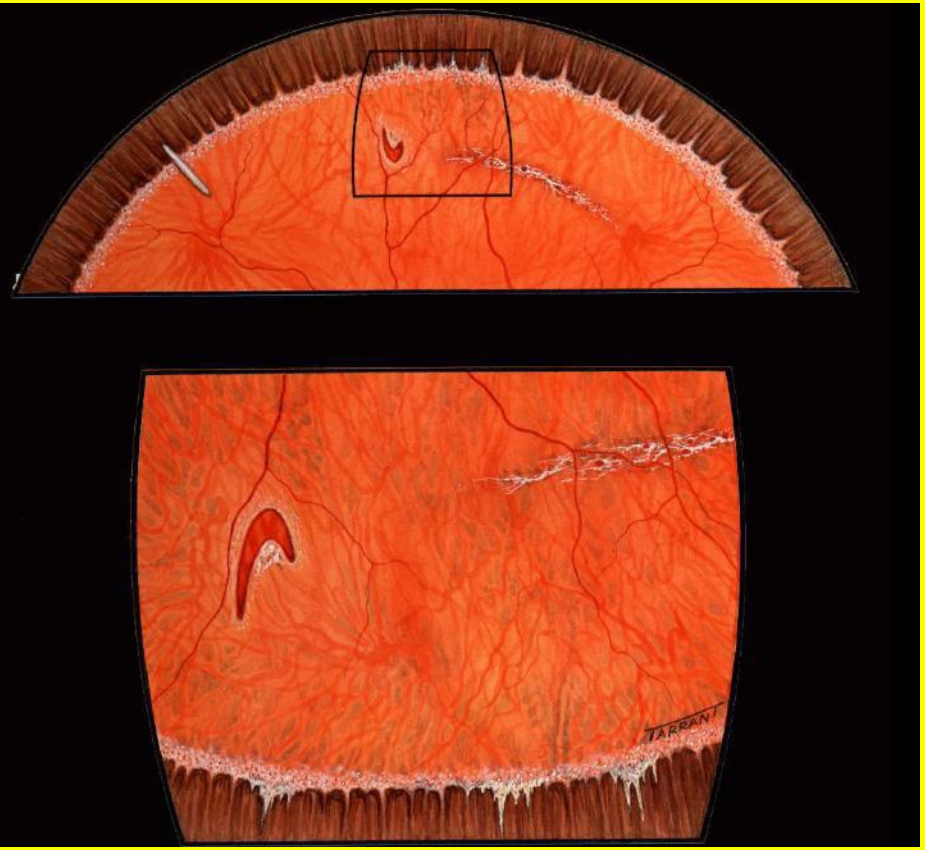
Figure 9.6 Drawing retinal lesions

Colour code



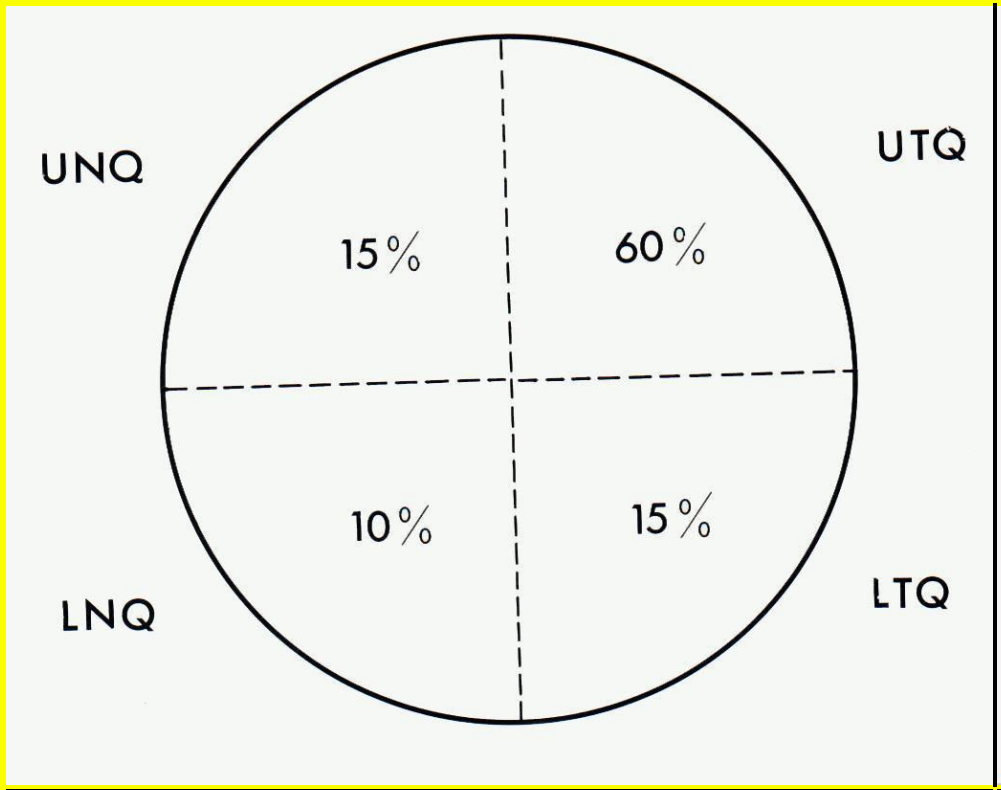
- Place chart upside down
- Draw what you see (Quadrant near you)

Slitlamp biomicroscopy

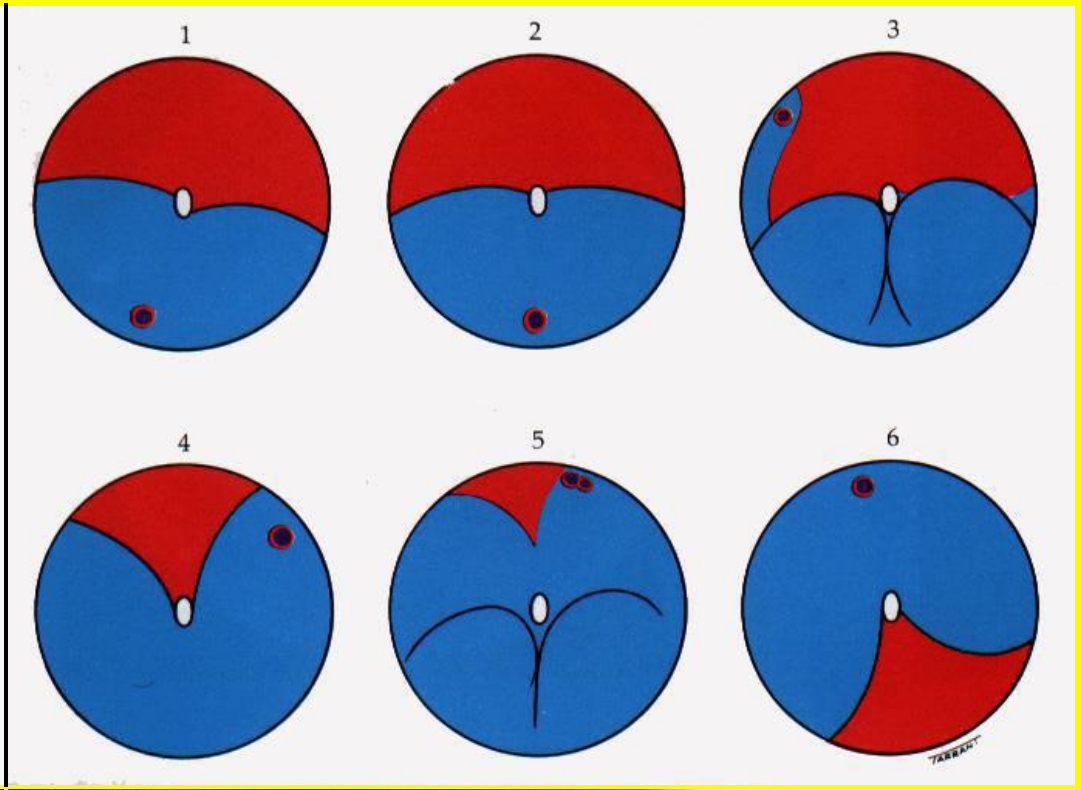
Goldmann triple-mirror lens	View of peripheral fundus
	
• Equatorial mirror (largest and oblong) - from 30 degree to equator • Peripheral mirror (square) - from equator to ora serrata • Gonioscopic (smallest)	• Vertical meridian's Image is upside down But not laterally reversed

Primary retinal break

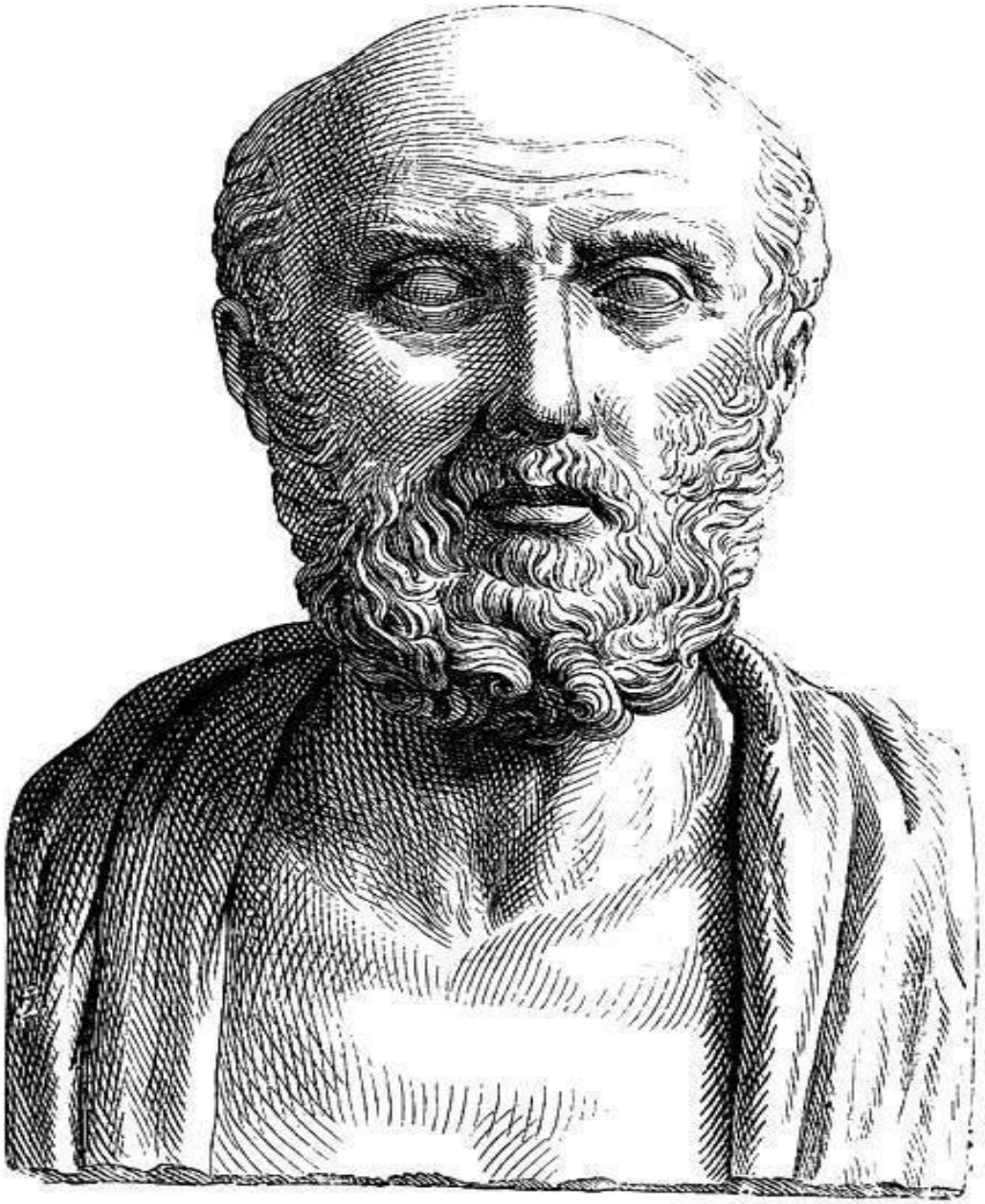
It is responsible for RD and determines configuration of SRF

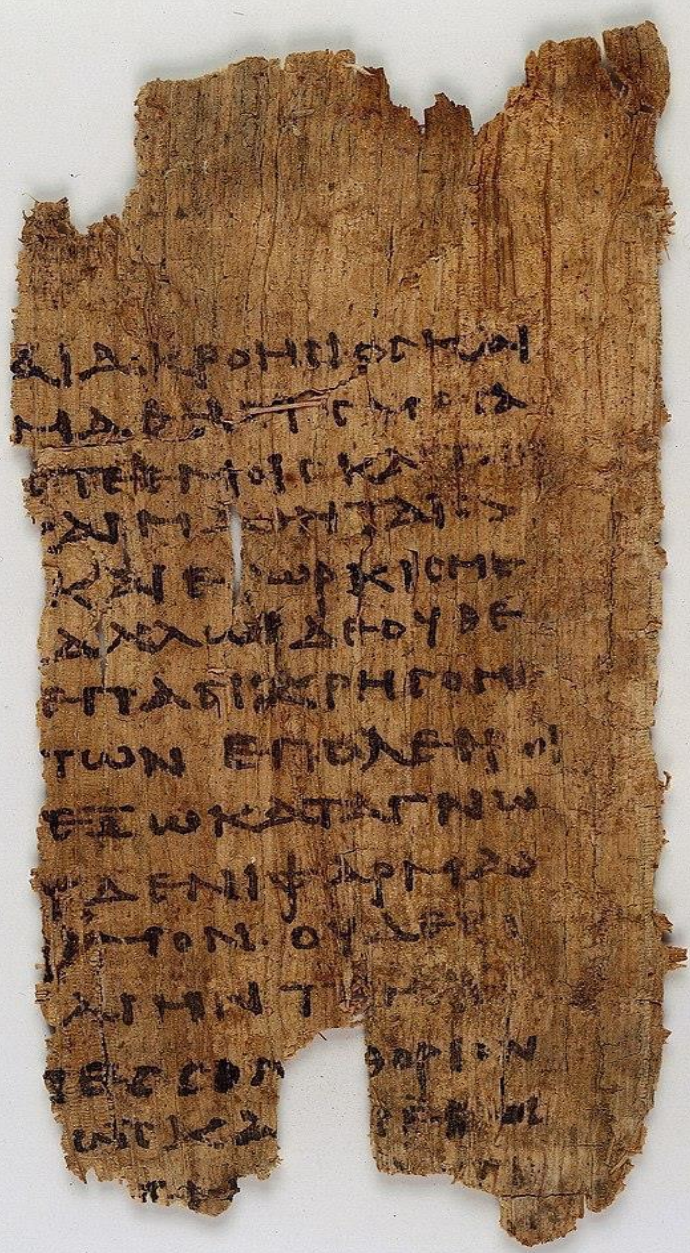


Quadratic distribution of breaks in eyes with RD



Configuration of SRF in relation to primary break

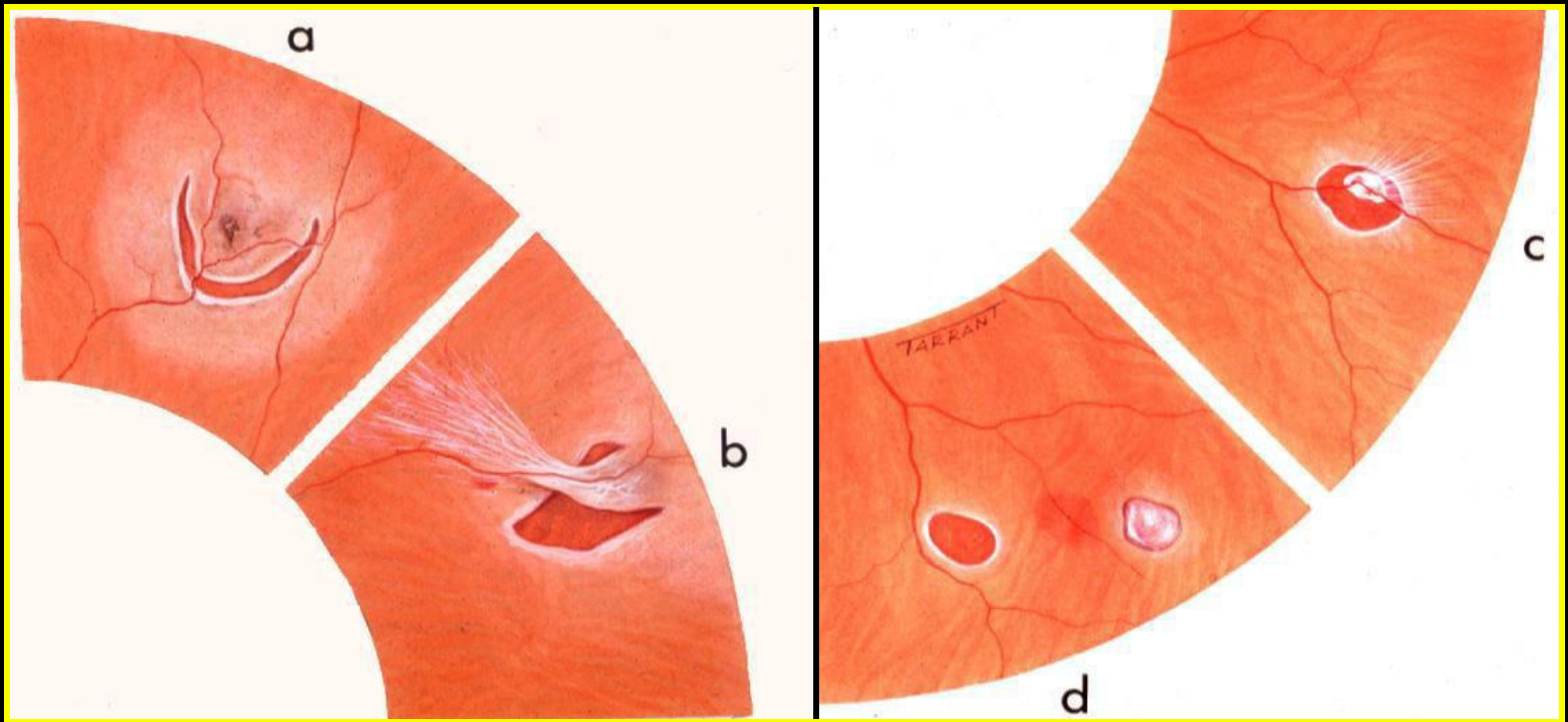




PROPHYLAXIS OF RHEGMATOGENOUS RETINAL DETACHMENT

1. **Retinal breaks**
2. **Predisposing degenerations**
   - Lattice
   - Snailtrack
   - White-without-pressure
3. **Treatment modalities**
   - Laser photocoagulation
   - Cryotherapy
4. **Benign peripheral degenerations**

Retinal breaks



a - Large U-tear with
‘ subclinical RD ’
- treat

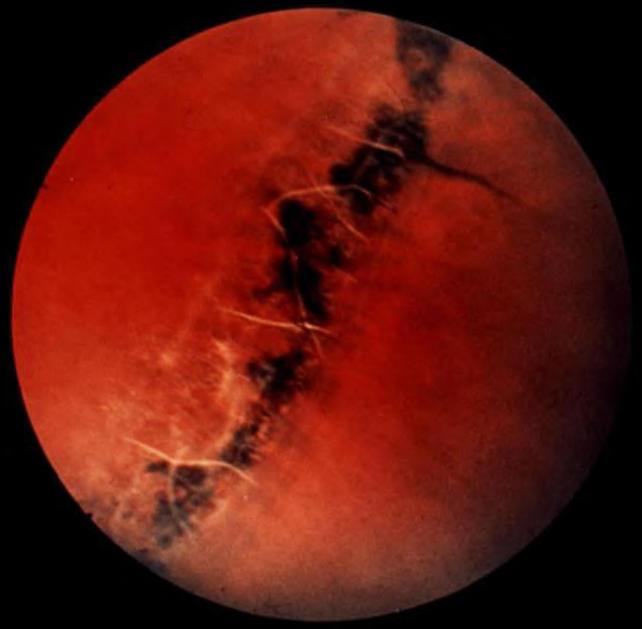
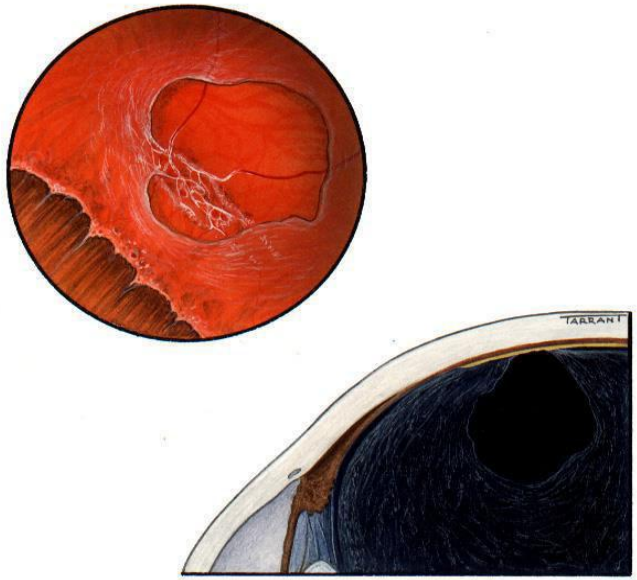
b - Large symptomatic U-tear
- treat

c - Operculated tear bridged by blood vessel
- treat

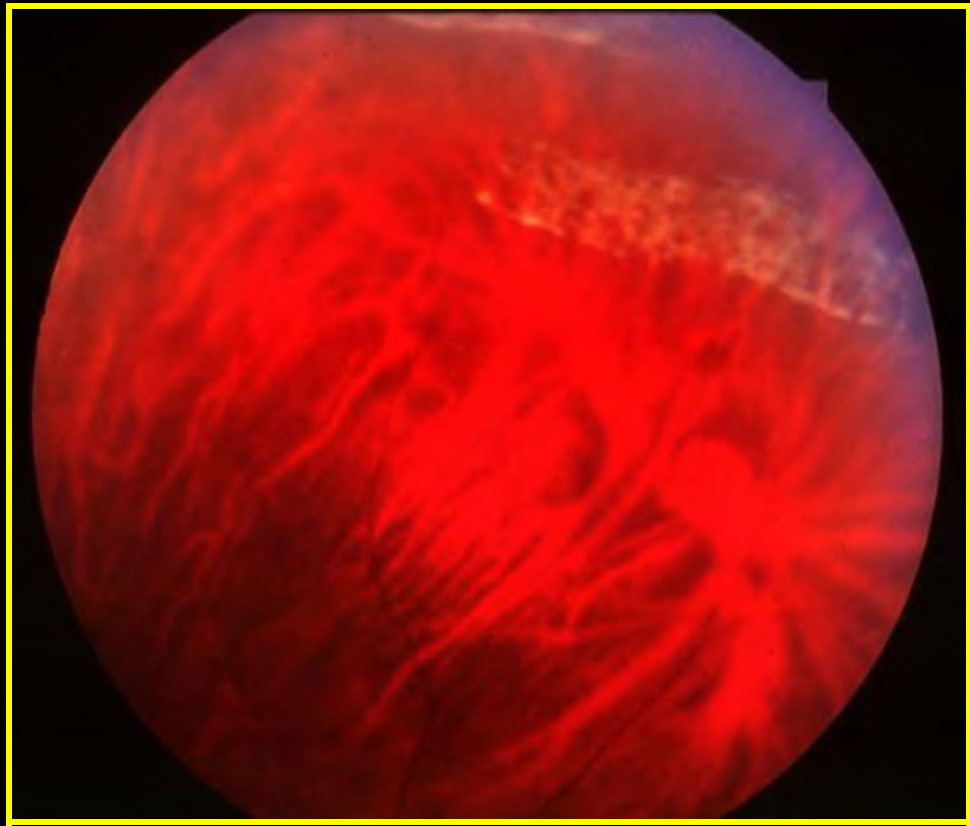
d - Asymptomatic operculated tear
- do not treat

Typical lattice degeneration

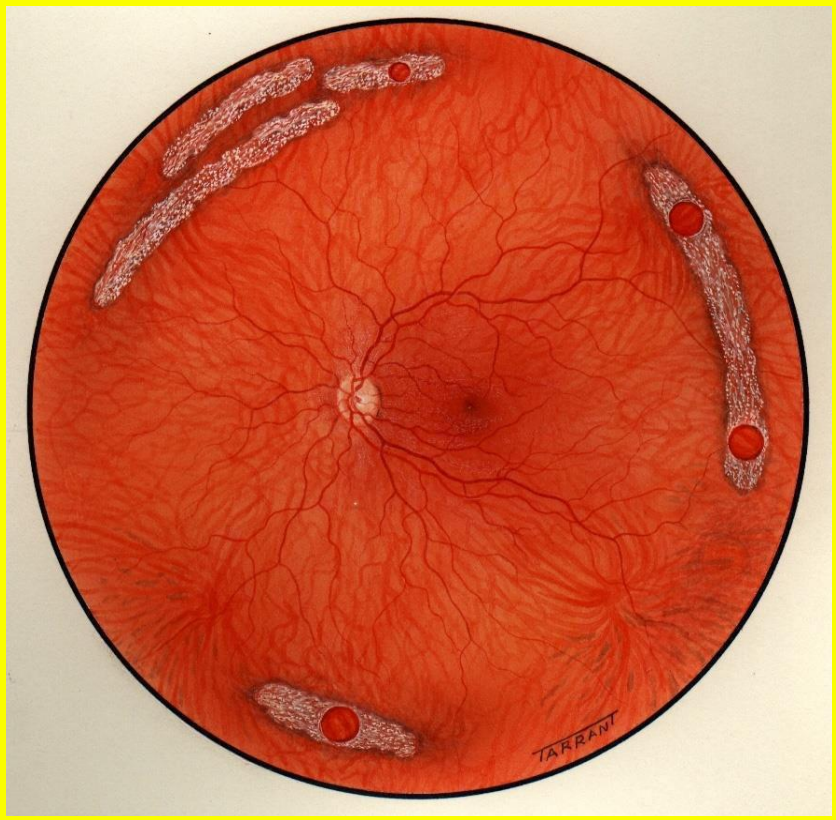
- Present in about 8% of general population
- Present in about 40% of eyes with RD

Retina	Vitreous
	
• Spindle-shaped islands of retinal thinning • Network of white lines within islands • Variable associated RPE changes • Small round holes within lesions are common	• Overlying vitreous liquefaction • Exaggerated attachments around margin of lesion

Snailtrack degeneration



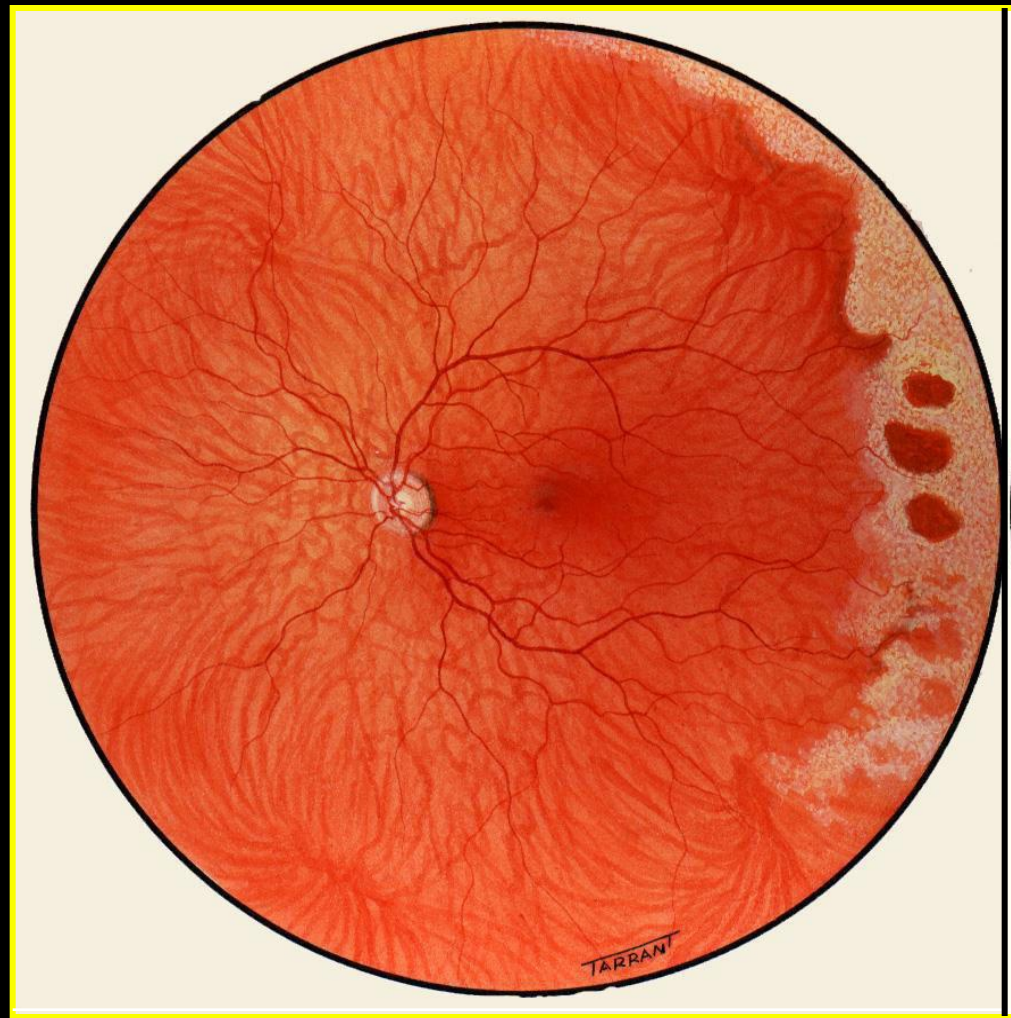
Sharply demarcated, frost-like bands which are longer than lattice



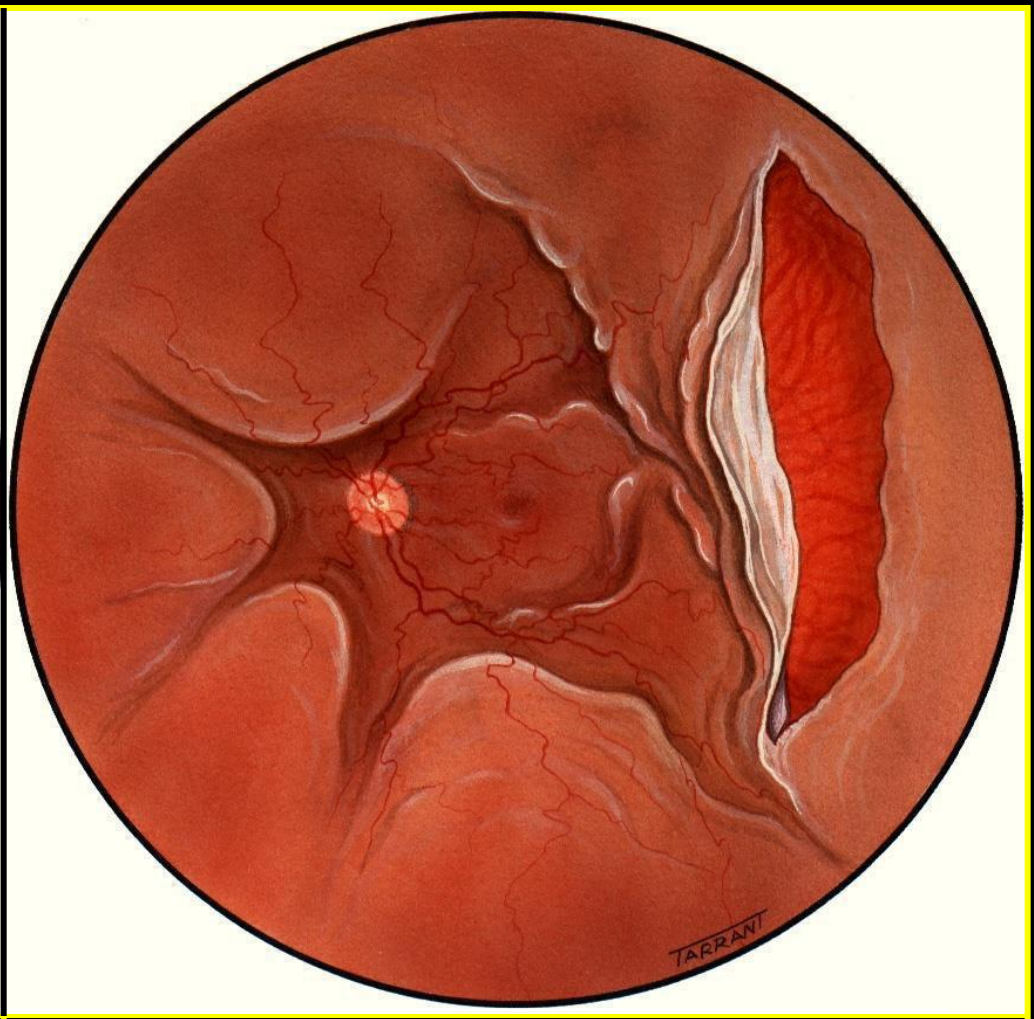
Large round holes which carry high risk of RD

Indications for prophylaxis - presence of holes

White-without-pressure



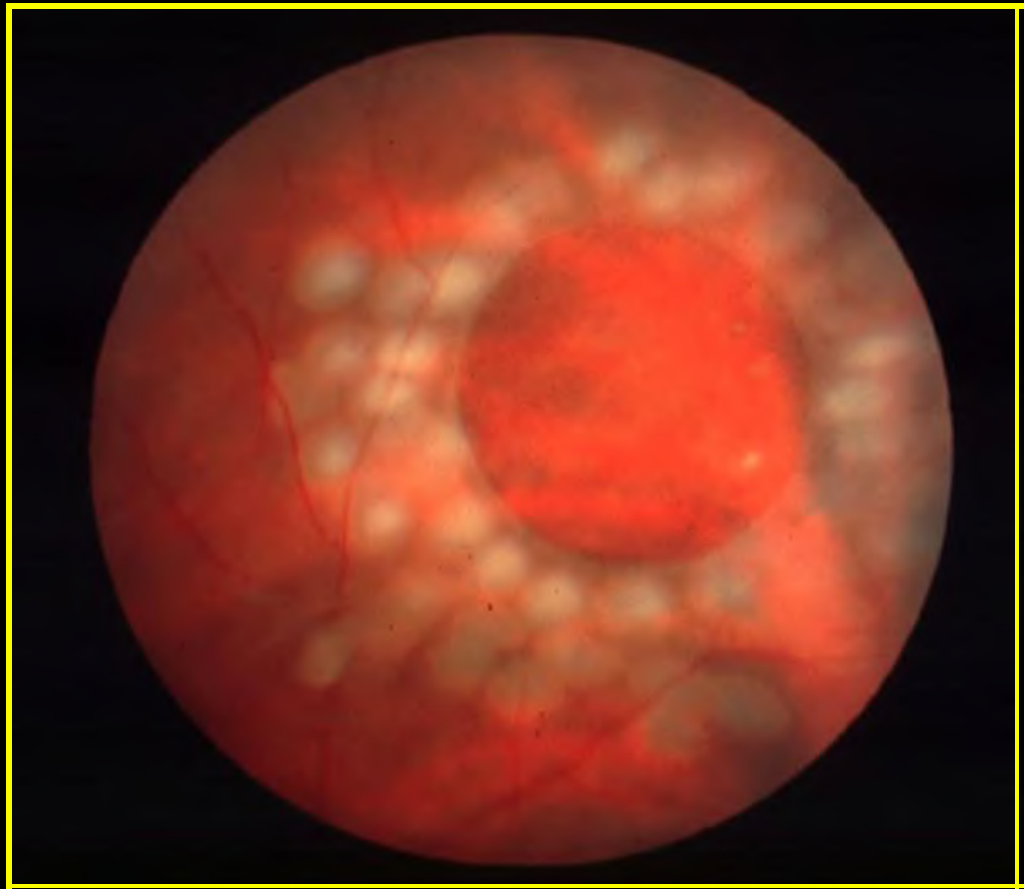
Translucent grey appearance of retina



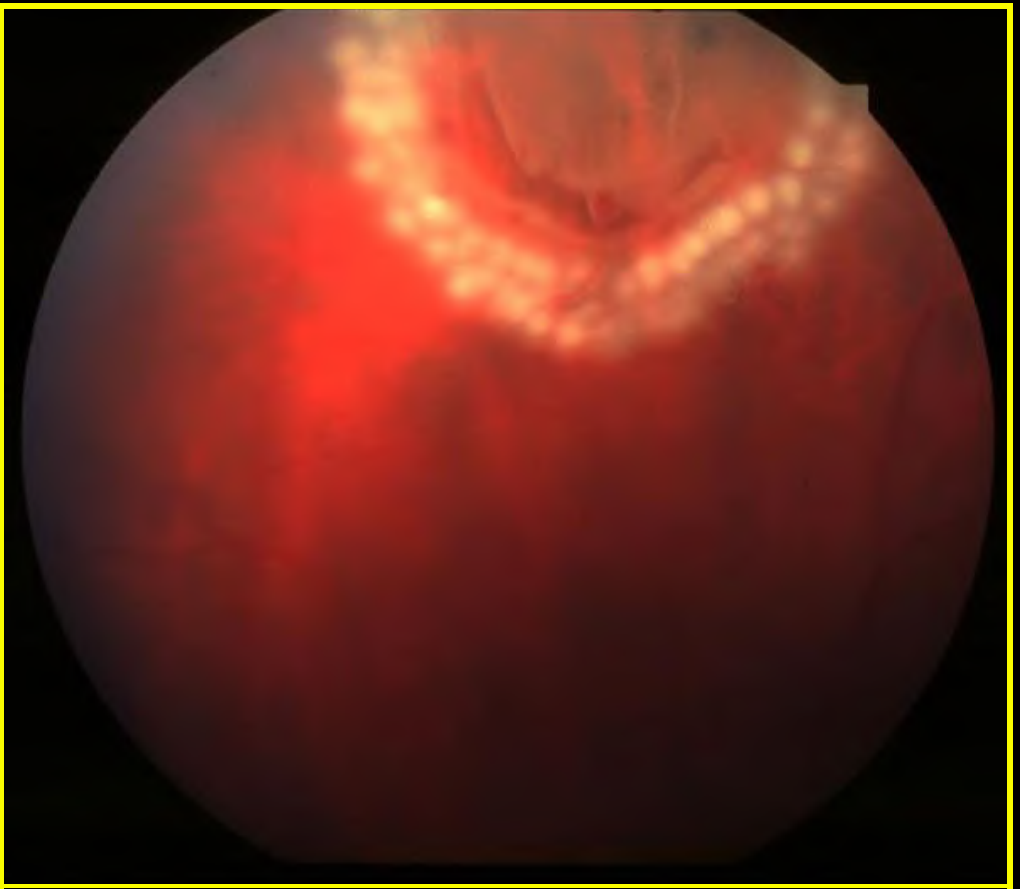
Occasional giant tear formation along posterior margin of lesion

Indications for prophylaxis - giant tear in other eye

Technique of laser photocoagulation

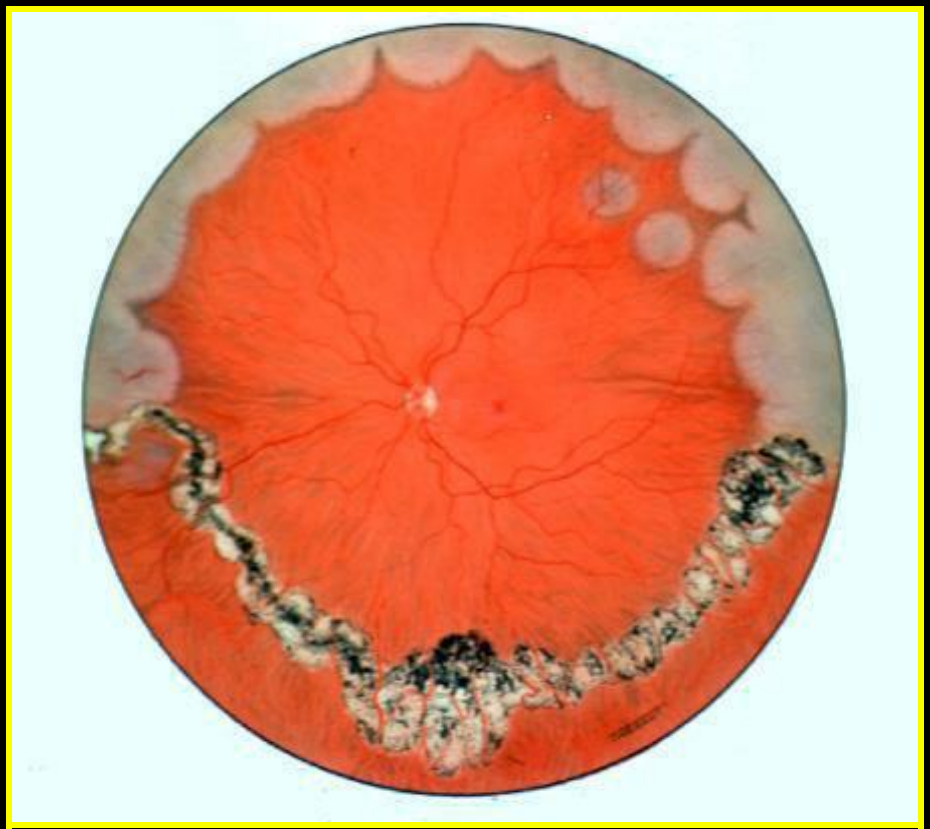


Surround lesion with two rows of confluent burns



Difficult for anterior lesions and if media hazy

Technique of cryotherapy



- Surround lesion with single row of cryo-applications
- Preferred for treatment of large areas

PRINCIPLES OF RETINAL DETACHMENT SURGERY

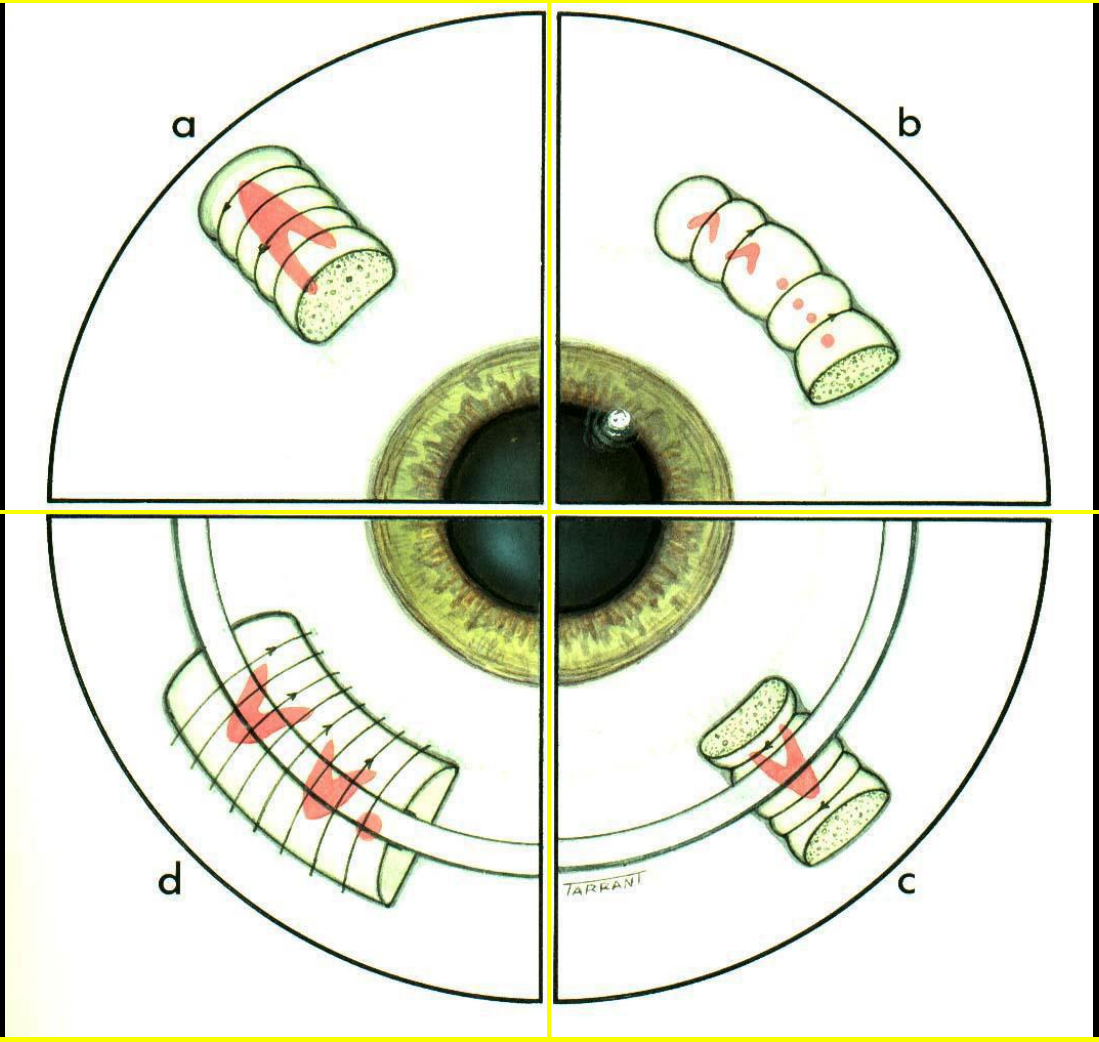
1. Scleral buckling

- Localization of breaks
- Aseptic inflammation (Cryotherapy/Laser)
- Sealing the break (Local explant/buckle)
- Encircling procedure
- Drainage of subretinal fluid

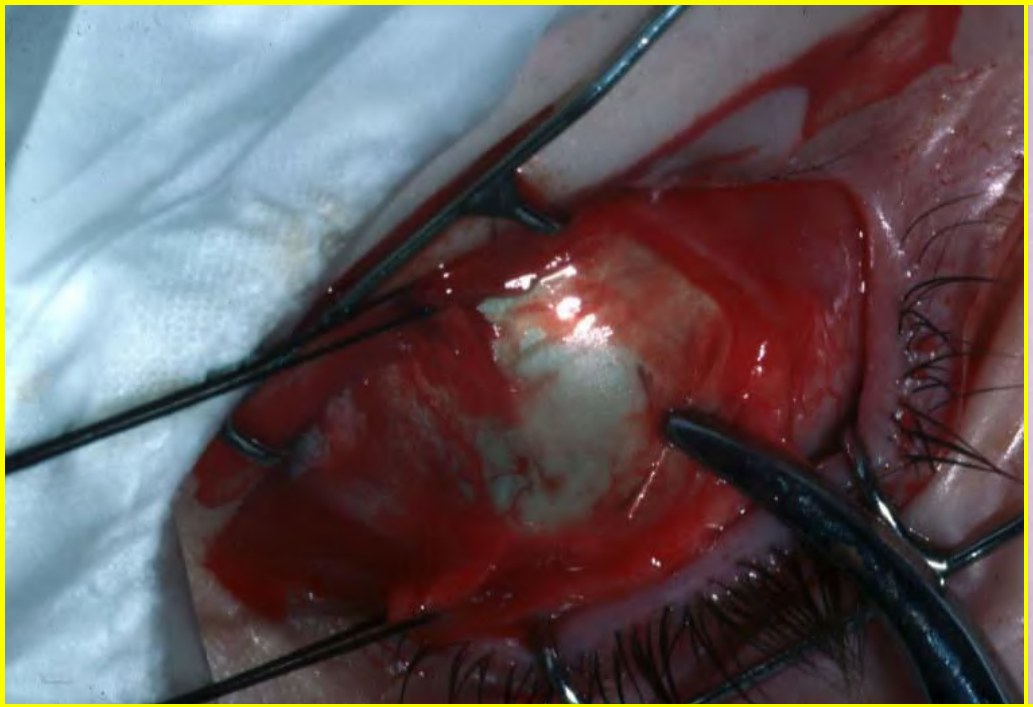
2. Vitrectomy

- Giant tears
- Proliferative vitreoretinopathy (PVR)
- Diabetic tractional RD
- Combined RD

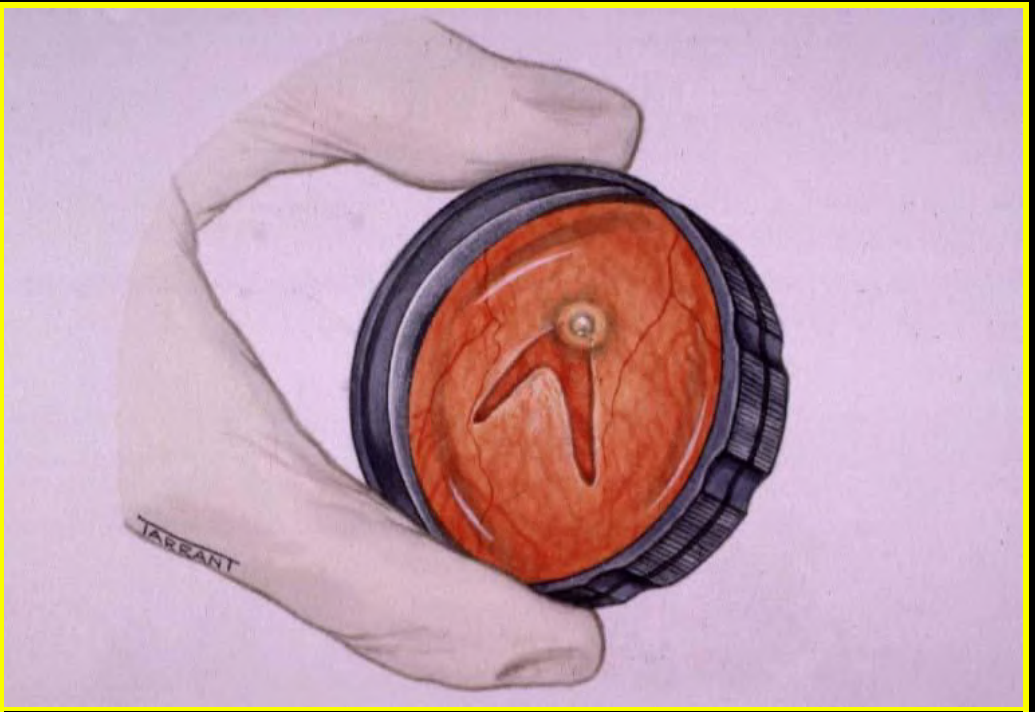
Configuration of scleral buckles

Radial		Segmental circumferential
Encircling augmented by solid silicone tyre		Encircling augmented by radial sponge

Localization of breaks

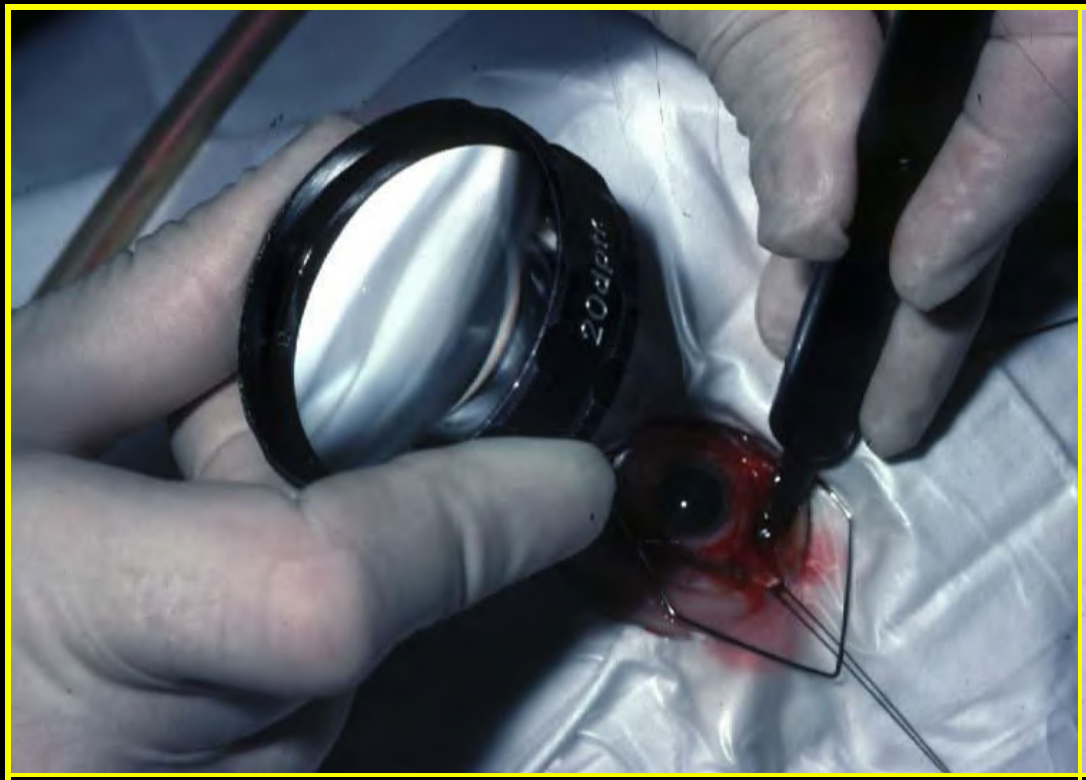


- Insert 5/0 Dacron scleral suture at site of apex of break
- Grasp cut suture with curved mosquito forceps close to knot



- While viewing with indirect ophthalmoscope check position of indentation in relation to break

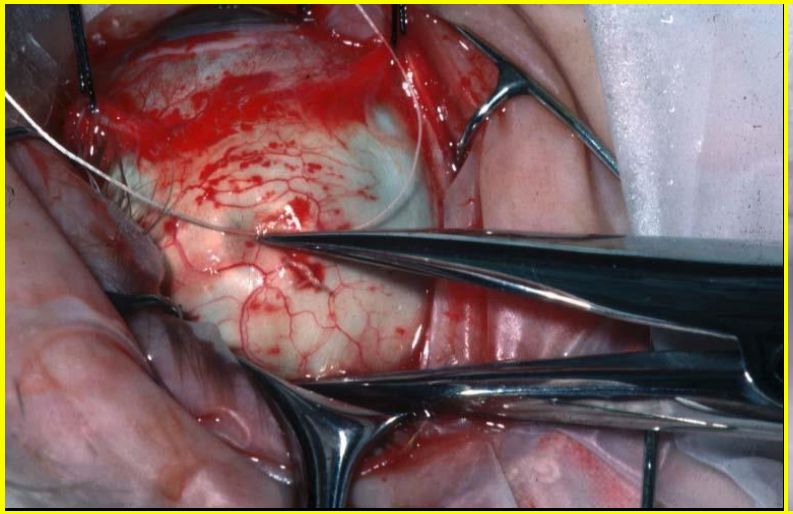
Cryotherapy



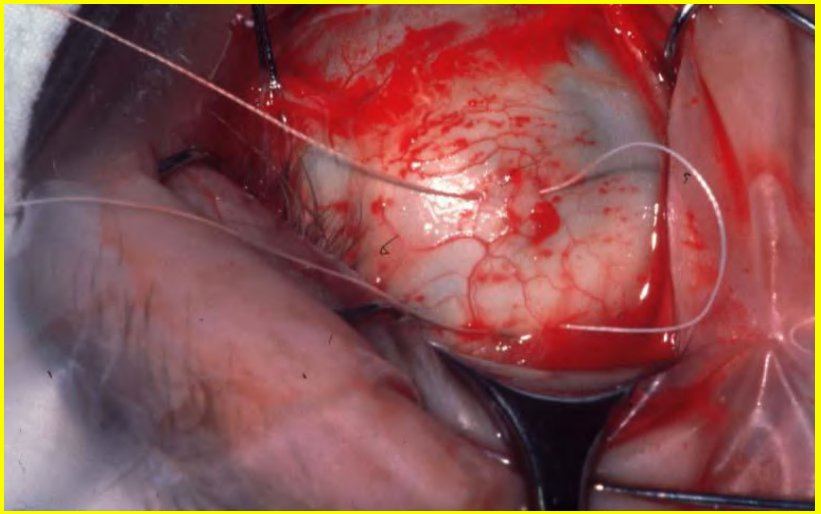
While viewing with indirect ophthalmoscope indent sclera gently with tip of cryoprobe

Freeze break until sensory retina just turns white

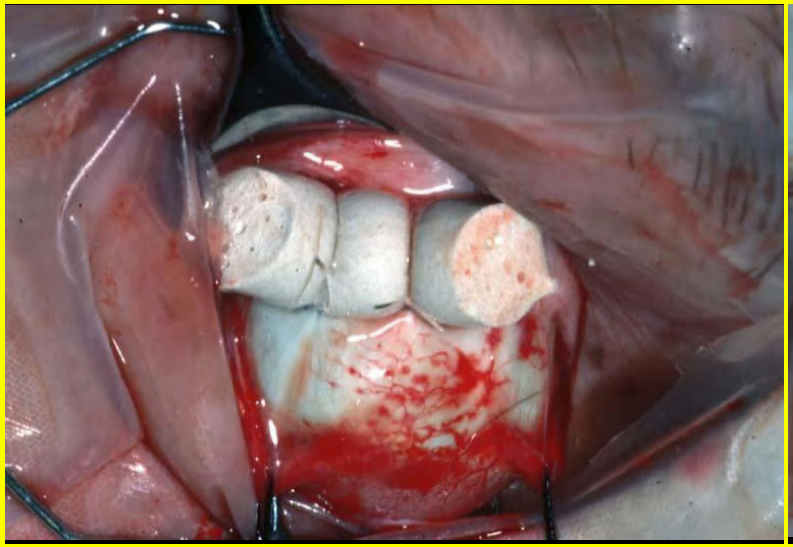
Insertion of local explant



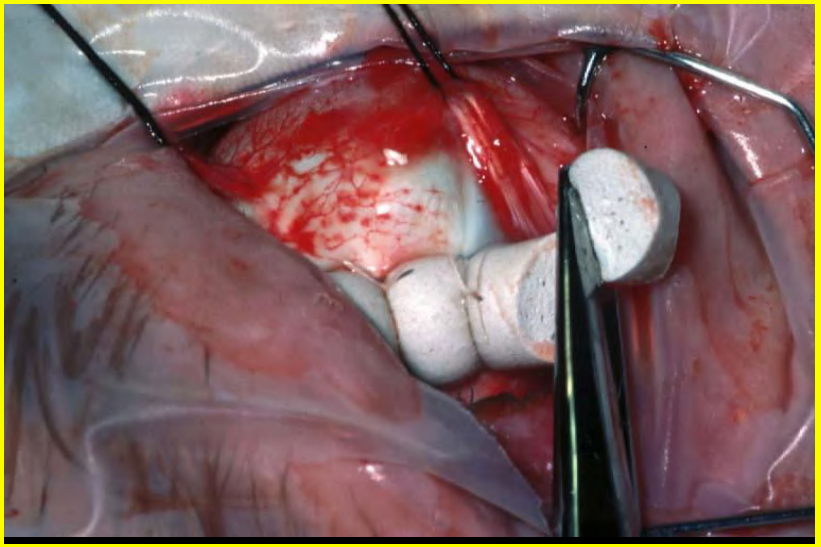
Distance separating sutures measured and marked



Insertion of mattress-type suture



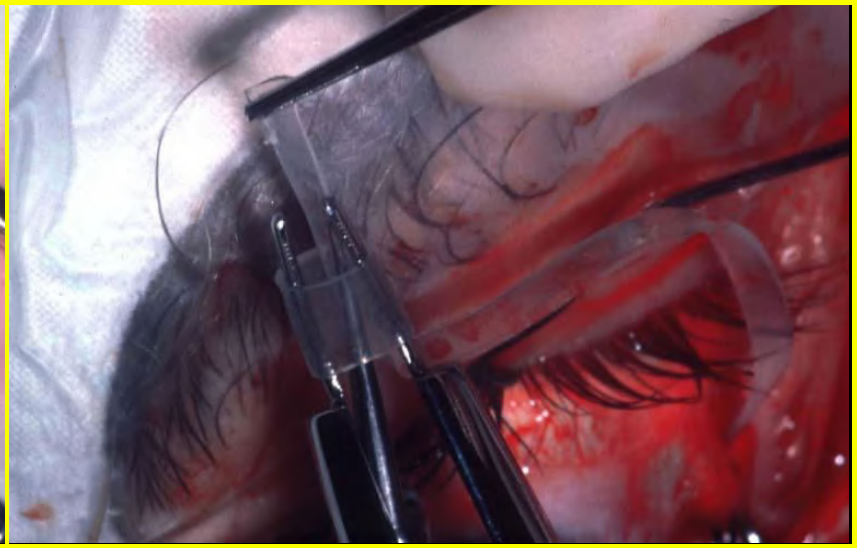
Sutures tightened over explant



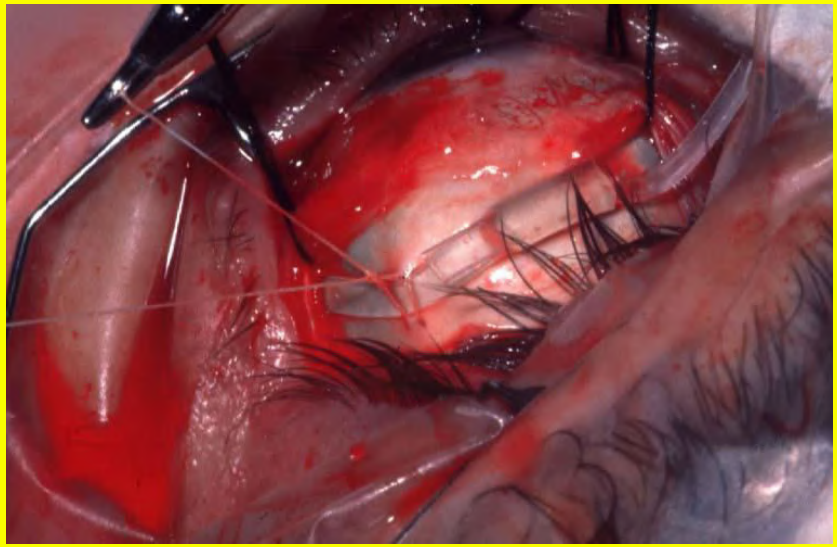
Ends trimmed

Encircling procedure

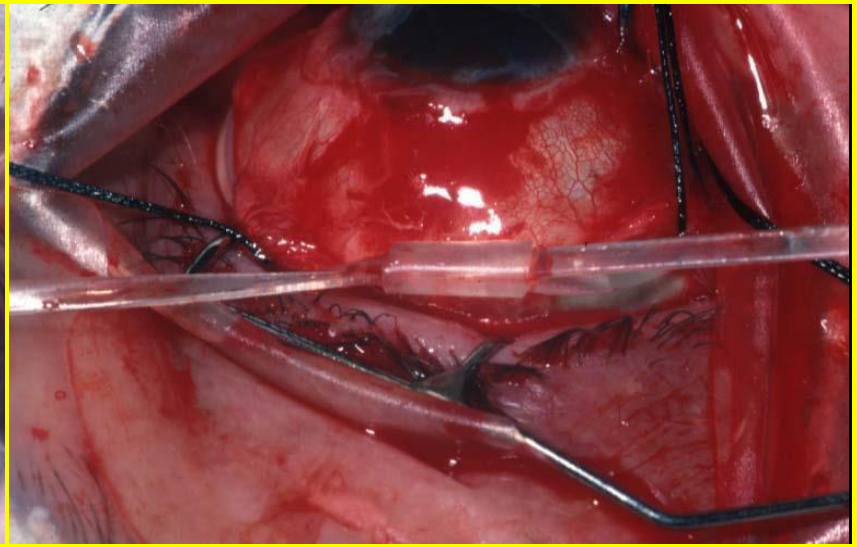
Strap fed under four recti



Ends secured with Watzke sleeve



Strap slid posteriorly and secured in each quadrant



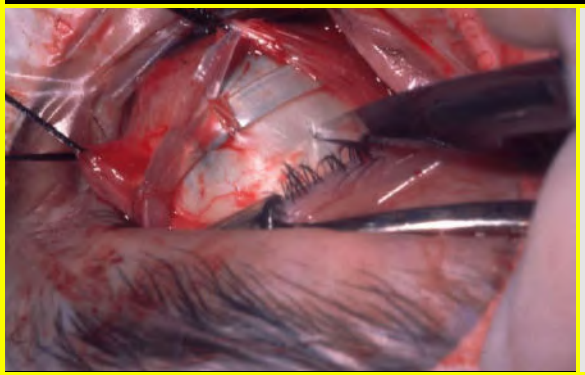
Strap tightened to produce required amount of internal indentation

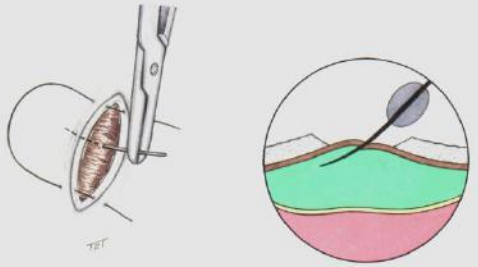
Drainage of subretinal fluid

Indications

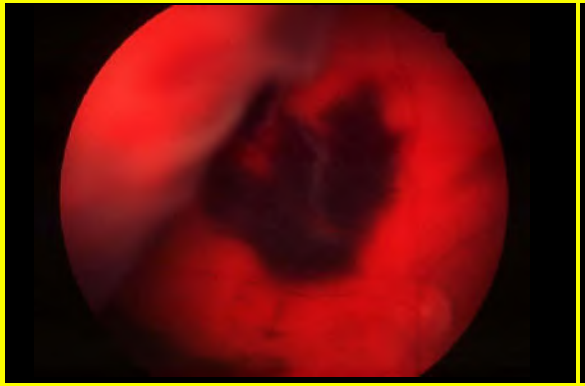
- Difficulty in localizing break
- Immobile retina
- Longstanding RD
- Inferior RD

Technique

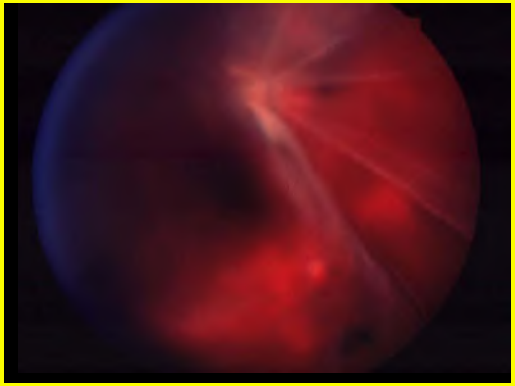




Complications



Haemorrhage



Retinal incarceration

Scleral Buckling
Surgical Principles/Tips

- Localization of breaks (Primary/Secondary)
- Aseptic inflammation: Cryotherapy, Laser
- Sealing of break: Buckle (Trye, Sponge, Solid rods, BB)
- SRF drainage
- Check CRA status

Scleral Buckling: Art To Learn

Our Experience

- Conventional retinal re-attachment surgery
- Success 80% (Including high PVR/PPK/Less experience)…. 2003

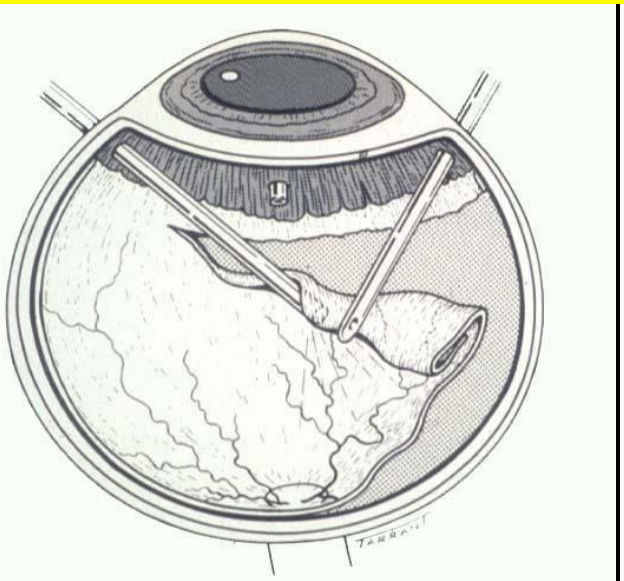
- Scleral buckling for primary rhegmatogenous RD
- Successful re-attachment in 85.7% cases….. 2010

- Scleral Bucling RD with retinal dialysis
- Surgical reattachment 95.8% …… 2015

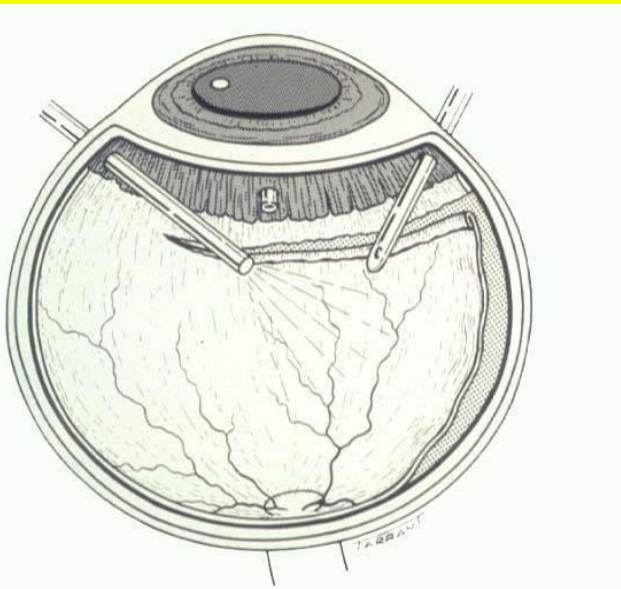
- Non drainage Scleral Buckling surgery
- Primary surgical success rate was 91% 2017

- Sanaullah jan, et al. Conventional retinal reattachment surgery. Journal of college of Physicians and surgeons Pakistan. August 2004;14(8): 470—473.
- Khan MT, Sanaullah Jan, et al. Outcome of scleral bucking procedures for primary rhegmatogenous retinal detachment. J Pak Med Ass. Sept 2010; 60(9): 754-7.
- Sanaullah jan, et al. Retinal detachment due to retinal dialysis: Surgical outcome after scleral buckling. Asia-Pacific J Ophthalmol. Sep-Oct 2015; Volume 4(5):259-262.
- Sanaullah Jan, et al. Non Drainage Scleral Buckling Surgery. OA J Ophthalmol 2017; 2(2):000123.
- Sanaullah Jan, et al. Complications of SRF drainage in Scleral Buckling. J Clin Community Ophthalmol. Jan-Jun 2023 1(1):11-14.
- Abdullah AS, Sanaullah Jan,et al. Complications of conventional scleral buckling during and after the treatment of rhegmatogenous RD. J Physicians & Surg Pak. May 2010, Vol 20 (5): 321-326.

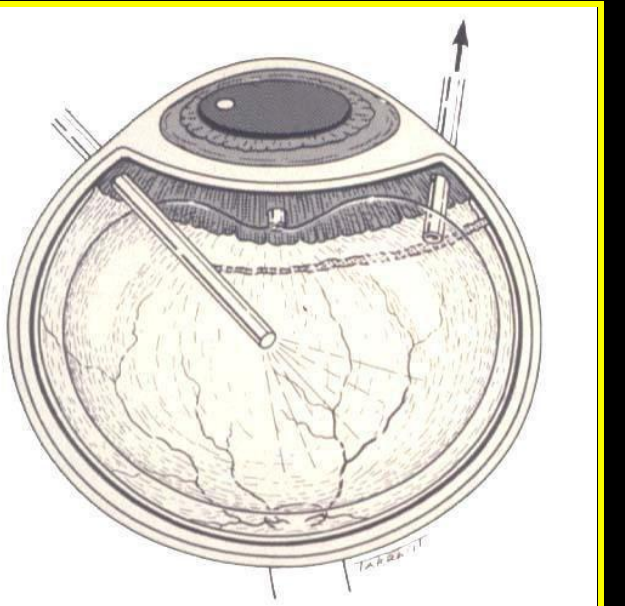
Vitrectomy for giant tears



Unrolling of flap with light pipe and probe

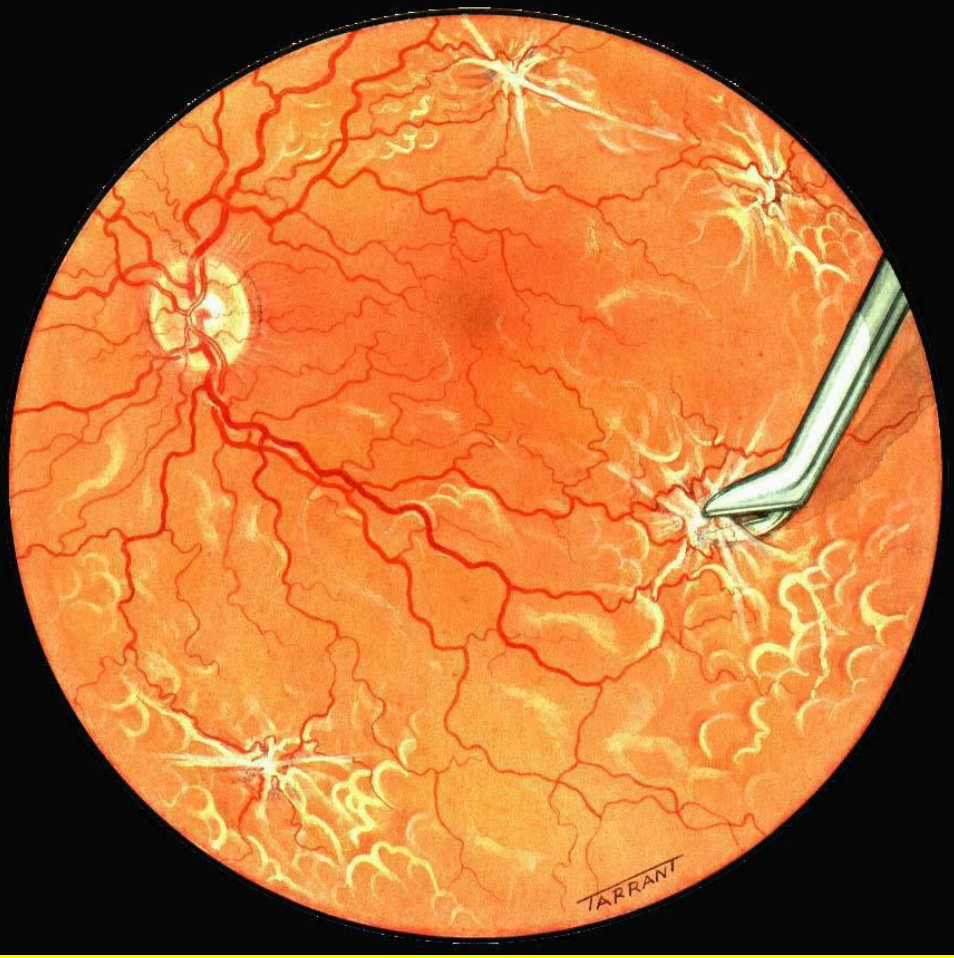


Completion of unrolling



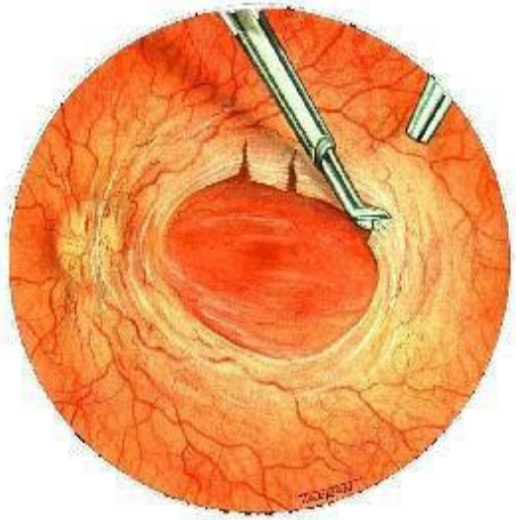
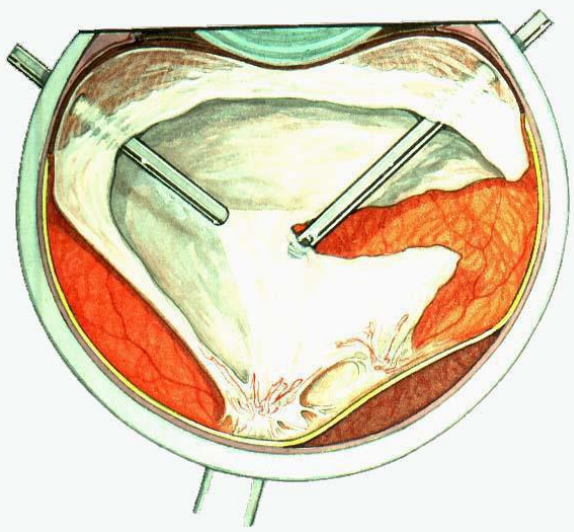
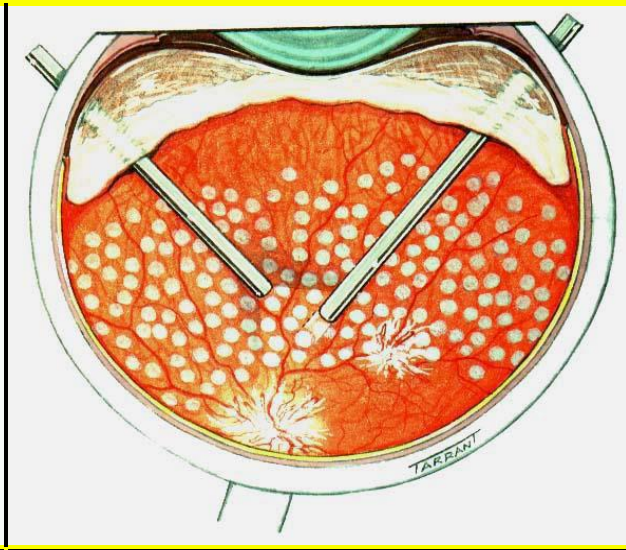
Injection of silicone oil or heavy liquid

Vitrectomy for PVR



- Dissection of star folds and peeling of membranes
- Injection of expanding gas or silicone oil

Vitrectomy for diabetic tractional RD

		
Release of circumferential traction	**Release of antero-posterior traction**	**Endophotocoagulation**

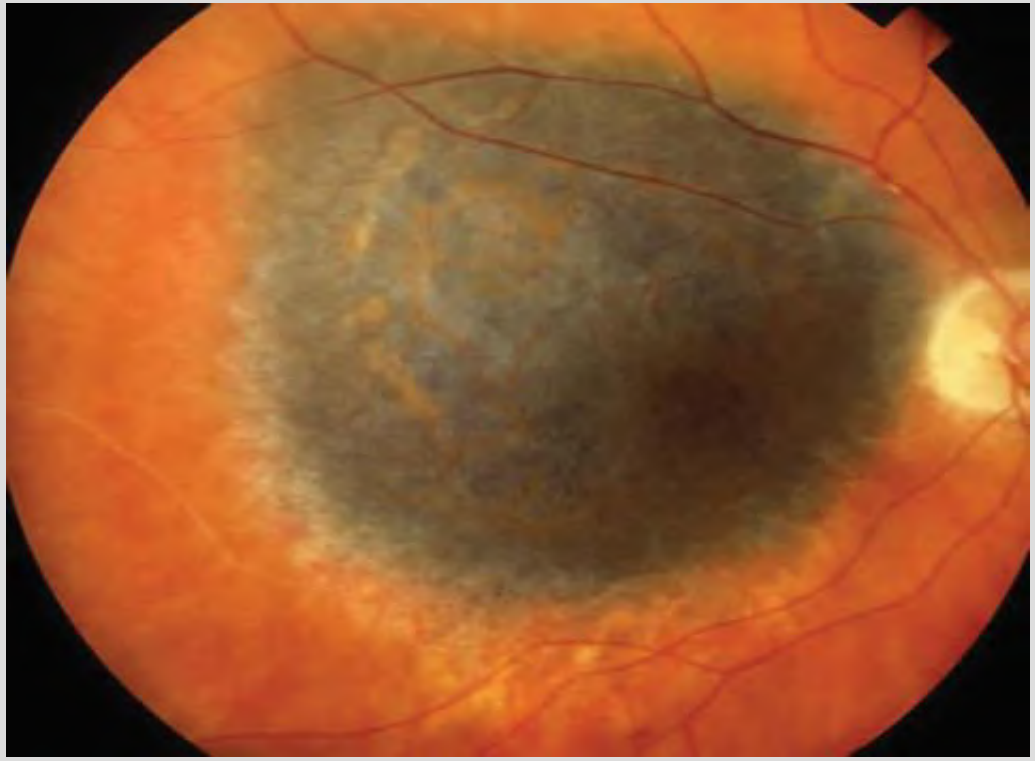
THANK YOU

CHOROIDAL MELANOMA

DR UMER KHAN ORAKZAI

Associate Professor of Ophthalmology

KGMC/HMC



Choroidal melanoma is the most common primary intraocular malignancy in adults

Accounts for 80% of all uveal melanomas
Six cases per 1million population

Presentation peaks at around the age of 60 years.

Most cases occur sporadically.

PREDISPOSING RISK FACTORS

- fair skin
- lighter iris colour,
- numerous and/or atypical (dysplastic) cutaneous naevi,
- choroidal naevus,
- congenital ocular and oculodermal melanocytosis (naevus of Ota)
- uveal melanocytoma.
- Chronic sunlight exposure and arc welding are environmental risk factors

HISTOPATHOLOGY

- Histopathology reveals spindle and epithelioid cell types

CLASSIFICATION

- Spindle shaped cells
- Epithetheliod cells
- Mixed cells

CLASSIFICATION

- Small diameter less than 10mm
- Medium diameter 10 to 15mm
- Large more than 15mm

SPREAD

- Lesions may penetrate Bruch membrane and the retinal pigment epithelium (RPE) with herniation into the subretinal space, classically assuming the shape of a collar stud
- Scleral channel and vortex vein invasion can lead to orbital spread
- Metastasis is commonly to the liver, bone and lung

MORTALITY

- Mortality is up to 50% at 10 years.

SYMPTOMS

- **Symptoms are often absent, with a tumour detected by chance on routine fundus examination**.

- A range of visual disturbance can occur depending on tumour characteristics.

SIGNS

- A solitary elevated subretinal grey-brown or rarely amelanotic dome-shaped mass.
-
- About 60% are located within 3 mm of the optic disc or fovea.
- Clumps of overlying orange pigment are common

- Associated haemorrhage and subretinal fluid are common

- Other signs can include sentinel vessels, choroidal folds, inflammation, rubeosis iridis, secondary glaucoma and cataract.


Choroidal Melanoma

DIFFERENTIAL DIAGNOSIS

PIGMENTED LESIONS

- Choroidal naevus
- RPE Hypertrophy
- Sub retinal hemmorhhage

NON PIGMENTED

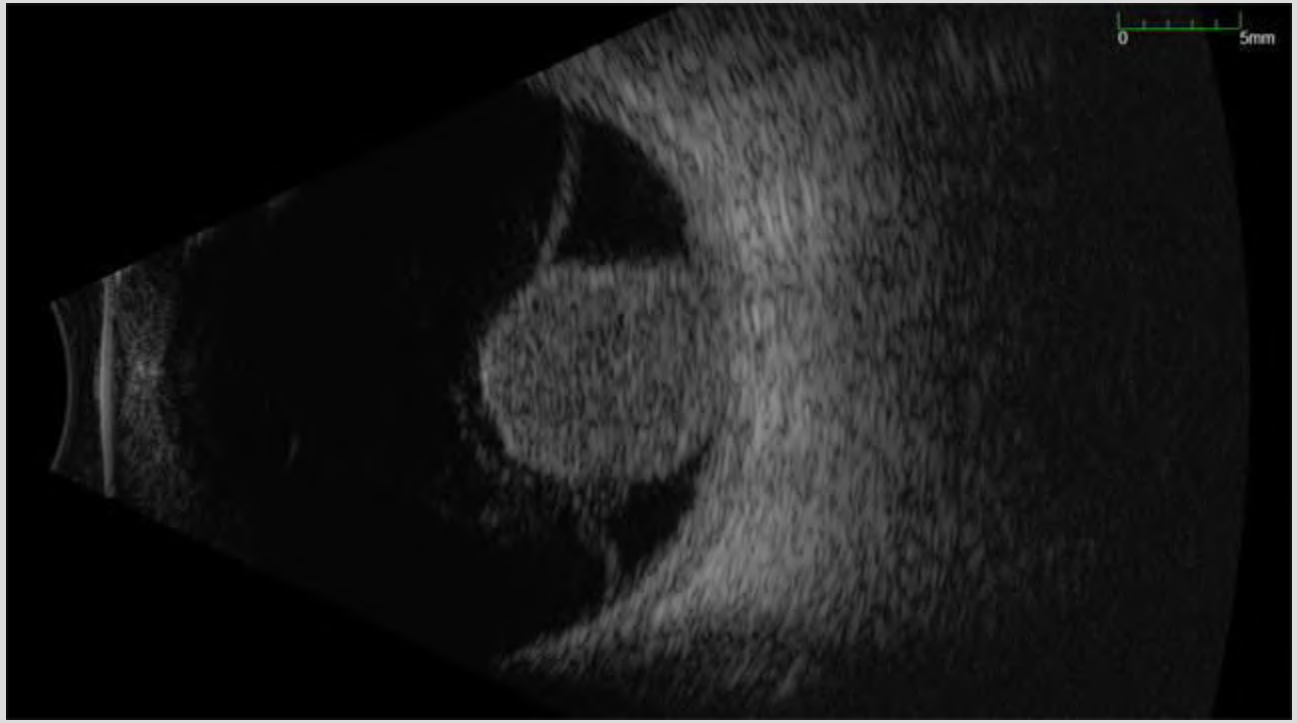
- Hemangioma
- Granuloma
- metastasis

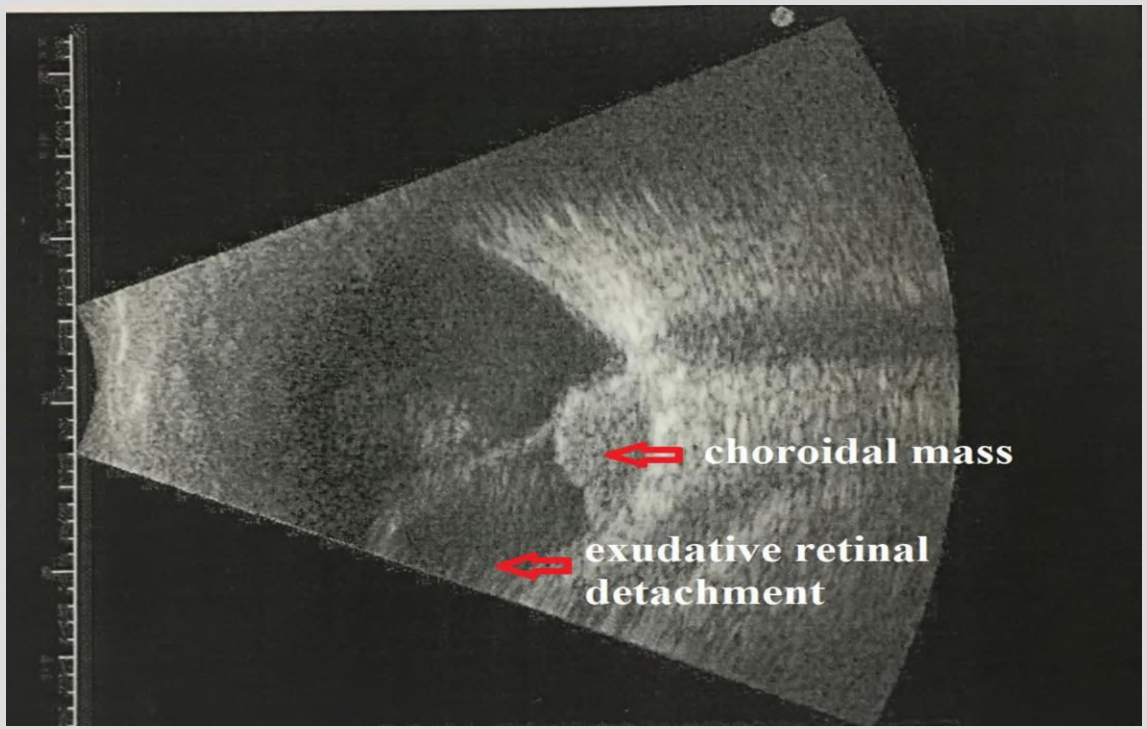
INVESTIGATIONS

- **Examination is sufficient for diagnosis in the majority of cases.**

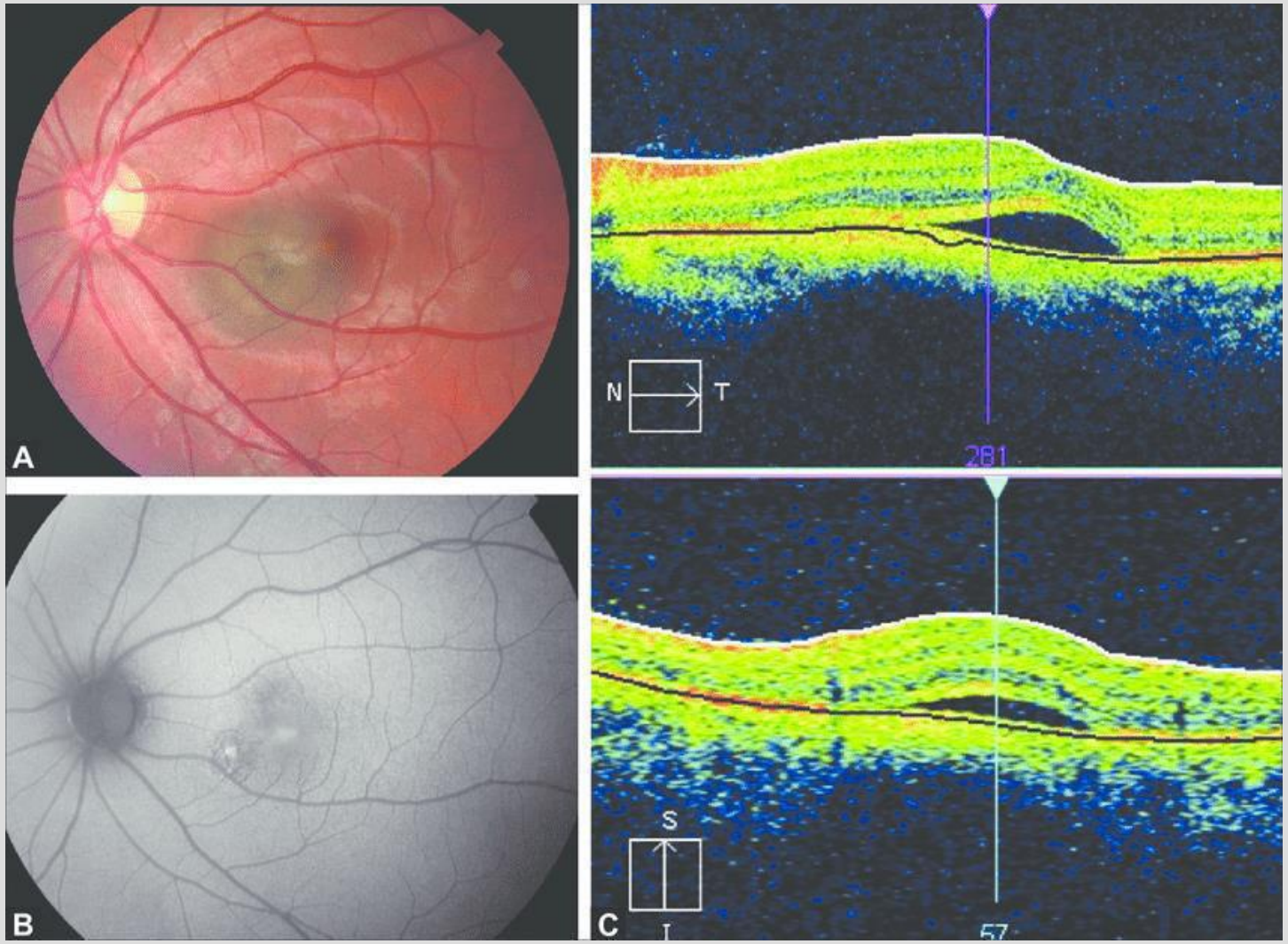
- **FFA is of limited diagnostic value**. The most common findings are an intrinsic tumour ('dual') circulation
- Pinpoint hyperflourescence at the apex of tomuour.

- **Ultrasound is used to measure lesion dimensions** and to detect tumours through opaque media and exudative retinal detachment and may also demonstrate extraocular extension. The characteristic findings are internal homogeneity with low to medium reflectivity, choroidal excavation and orbital shadowing





OCT



- **Magnetic resonance imaging (MRI)** is useful to demonstrate extraocular extension and may be of some help in differential diagnosis.

- **Biopsy is useful when the diagnosis cannot be established** by less invasive methods. It may be performed either with a fine needle or using the 25-gauge vitrectomy system

- **Genetic tumour analysis** is becoming increasingly important in management, particularly with regard to prognosis, as metastasis occurs almost exclusively with certain genetic profiles

- **Systemic investigation is directed principally towards detecting** metastatic spread

TREATMENT

- Brachytherapy
- External beam radiotherapy
- Trans pupillary thermotherapy
- Transcleral choroidectomy
- Enucleation

Thank You

Hypertensive Eye diseases

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Hypertension

- ACC and AHA
- Blood pressure more than 120 systolic and 80 diastolic labelled as high
- Changes in life style
- Medications

- Retinopathy
- Choroidopathy
- Optic neuropathy
- Vein occlusion
- Artery occlusion

RETINOPATHY

- **Arteriolar narrowing**
- **Cotton wool spots**
- **Vascular leakage**
  - flame-shaped retinal haemorrhages and retinal oedema
- **Arteriolosclerosis**
  - involves thickening of the vessel wall characterized histologically by intimal hyalinization, medial hypertrophy and endothelial hyperplasia

Pathophysiology

Vasoconstrictive Phase:

- due to the elevated luminal pressures, local autoregulatory mechanisms cause retinal arteriole narrowing and vasospasm to reduce flow.

Sclerotic Phase:

- the layers of the endothelial wall undergo changes such as intimal thickening, medial hyperplasia, worsening arteriolar narrowing, AV crossing changes, and silver and copper wiring.

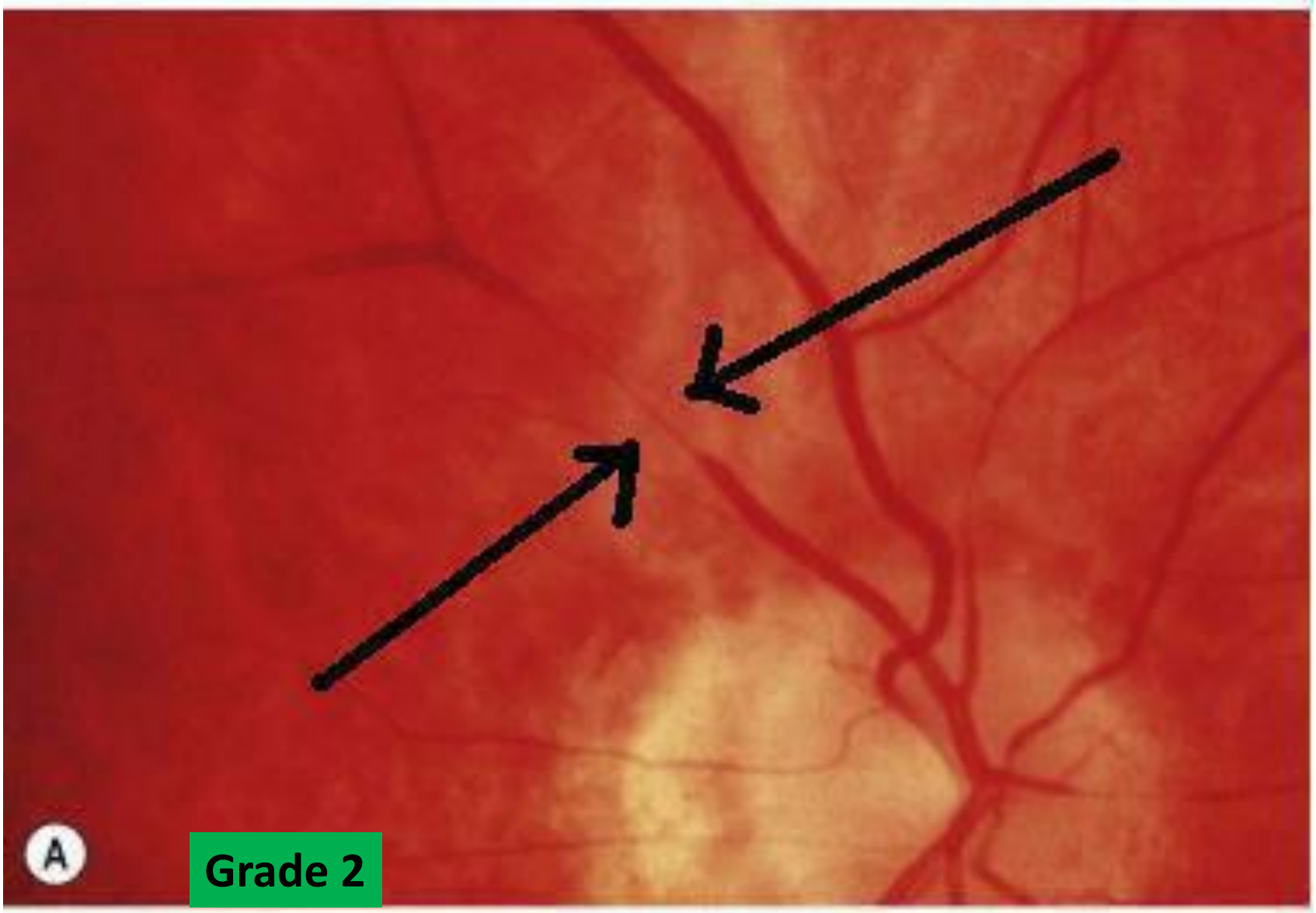
Exudative Phase:

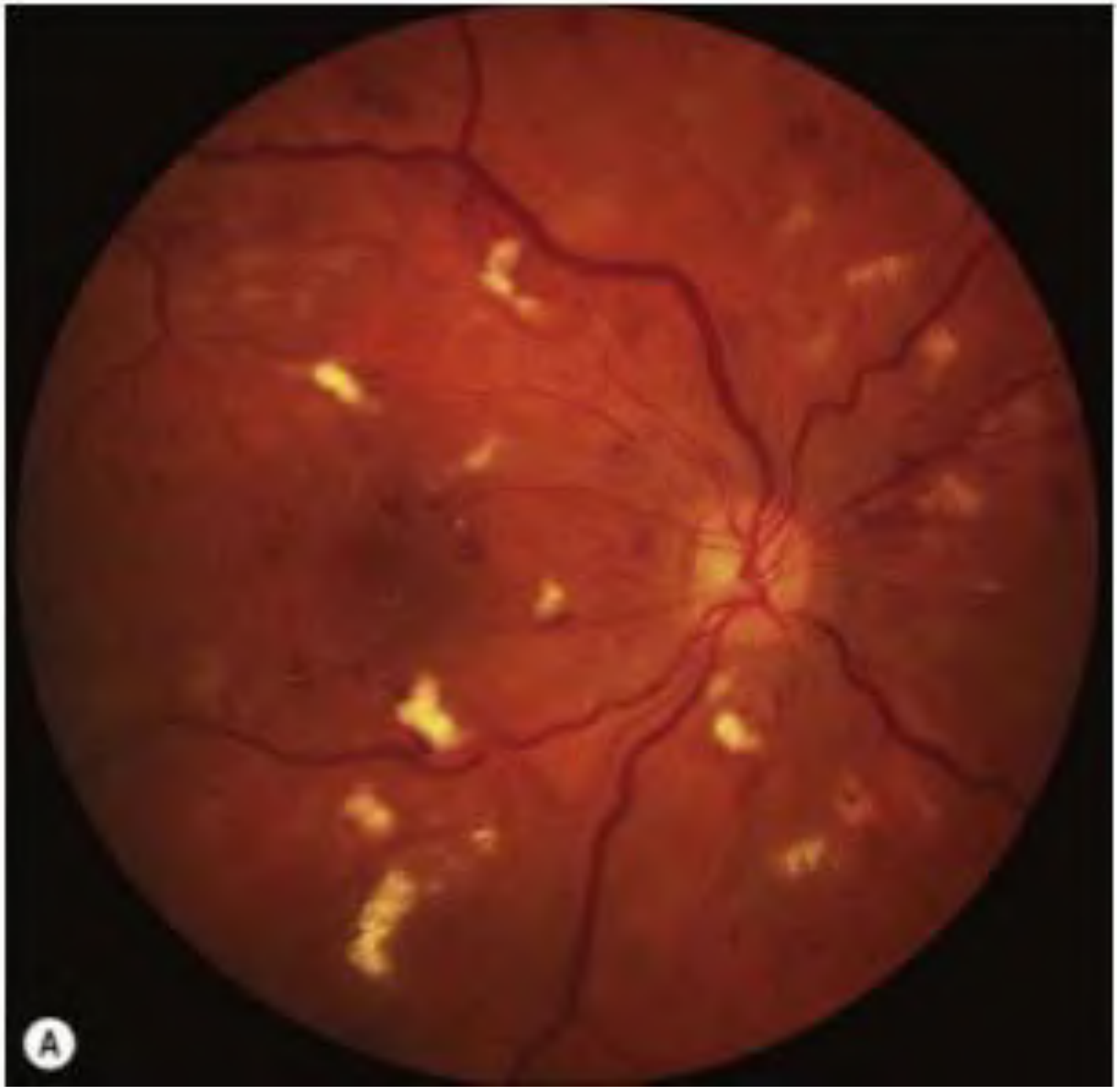
- there is a disruption of the blood-retinal barrier and leakage of plasma and blood causing retinal hemorrhages, hard exudates, retinal ischemia, and necrosis of smooth muscle.

Grading

Grading of hypertensive changes

Grade 0: No changes
Grade 1: Barely detectable arterial narrowing
Grade 2: Obvious arterial narrowing with focal irregularities (Figure 1)
Grade 3: Grade 2 plus retinal hemorrhages, exudates, cotton wool spots, or retinal edema (Figure 3)
Grade 4: Grade 3 plus papilledema (Figure 4)


A
Grade 2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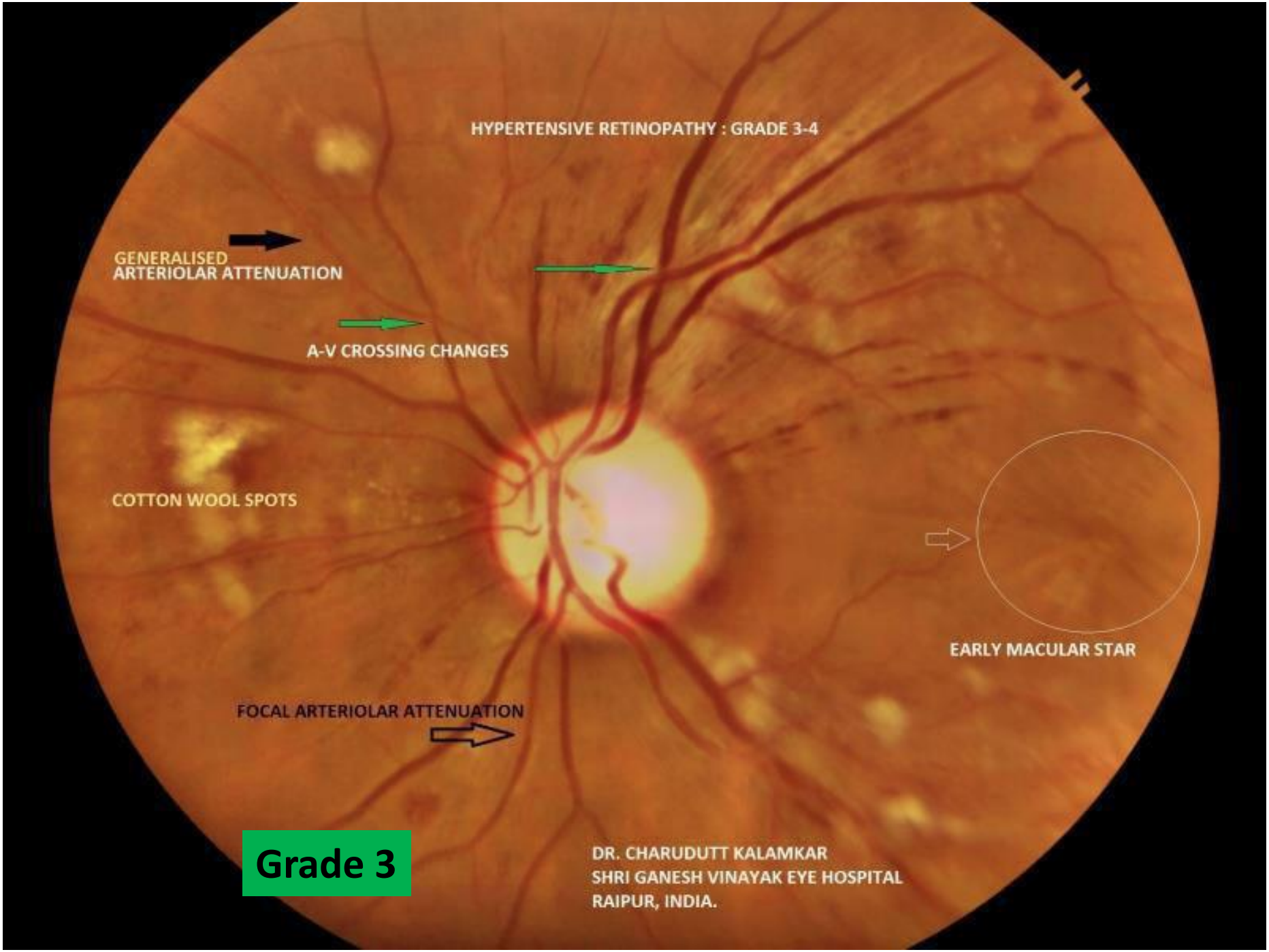
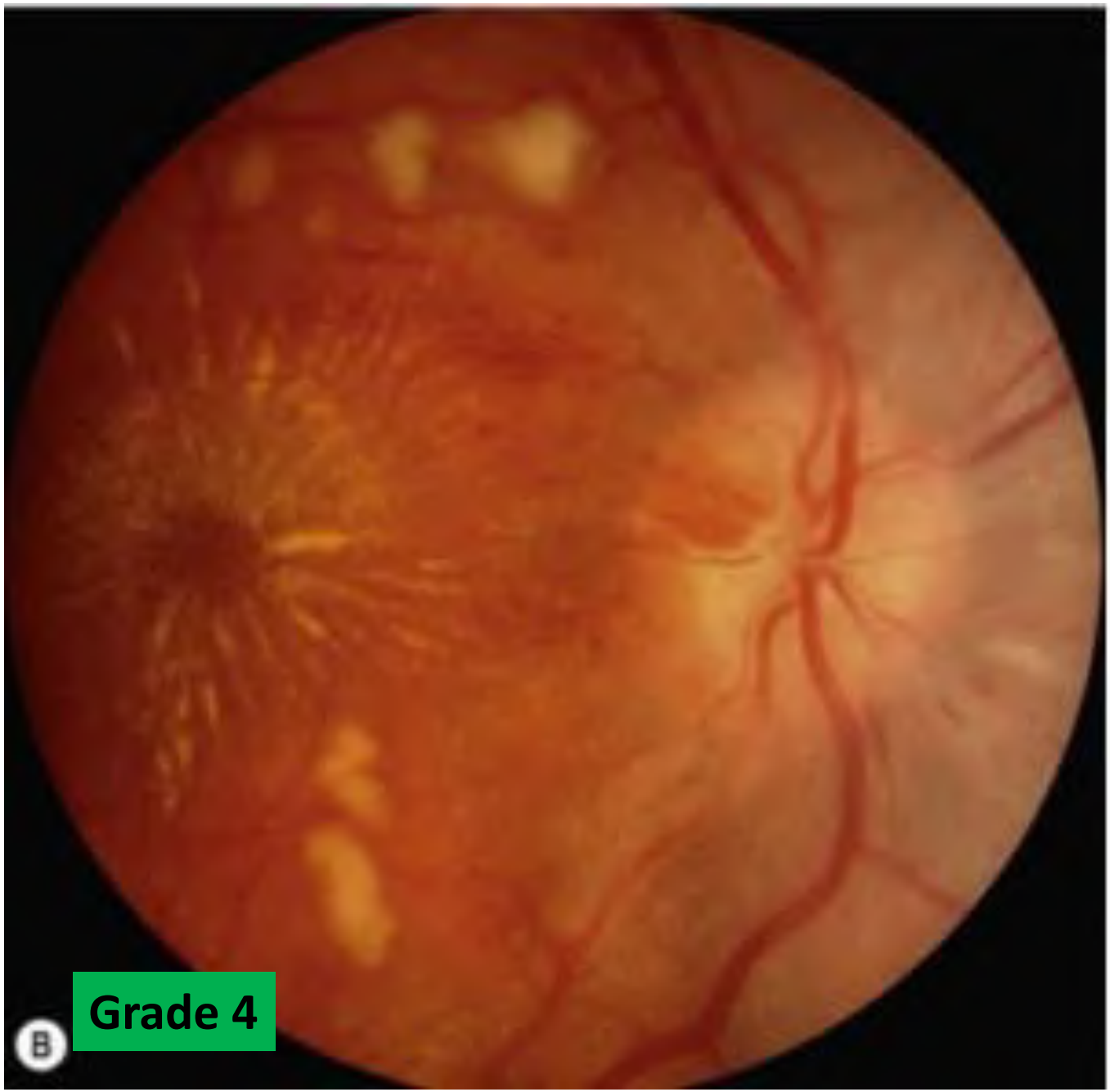

HYPERTENSIVE RETINOPATHY : GRADE 3-4
GENERALISED ARTERIOLAR ATTENUATION
A-V CROSSING CHANGES
COTTON WOOL SPOTS
EARLY MACULAR STAR
FOCAL ARTERIOLAR ATTENUATION
Grade 3
DR. CHARUDUTT KALAMKAR
SHRI GANESH VINAYAK EYE HOSPITAL
RAIPUR, INDIA.


B
Grade 4

RISK FACTORS
duration of elevated blood pressure
degree of blood pressure elevation over normal
high salt diet,
obesity,
tobacco use,
alcohol,
family history,
stress,

INVESTIGATIONS

- FFA
- OCT MACULA

TREATMENT

- Control blood pressure and other associated risk factors
- Treatment of occular complications if any.

OTHER EFFECTS OF HYPERTENSION ON EYE

- Choroidopathy
- Optic neuropathy
- Artery occlusion
- Vein occlusion

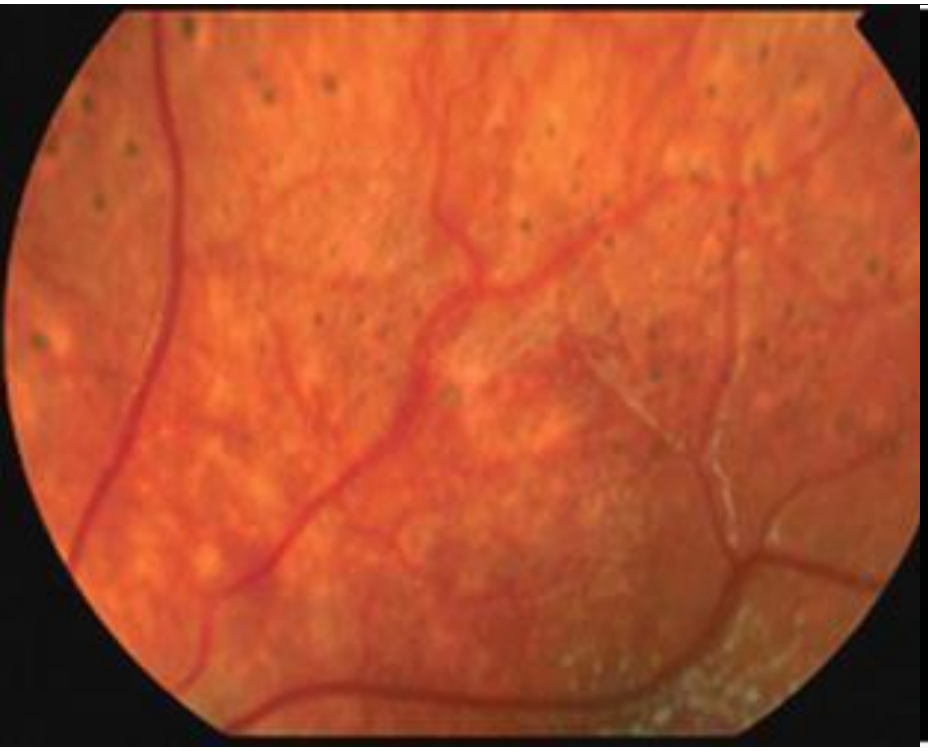
Choroidopathy

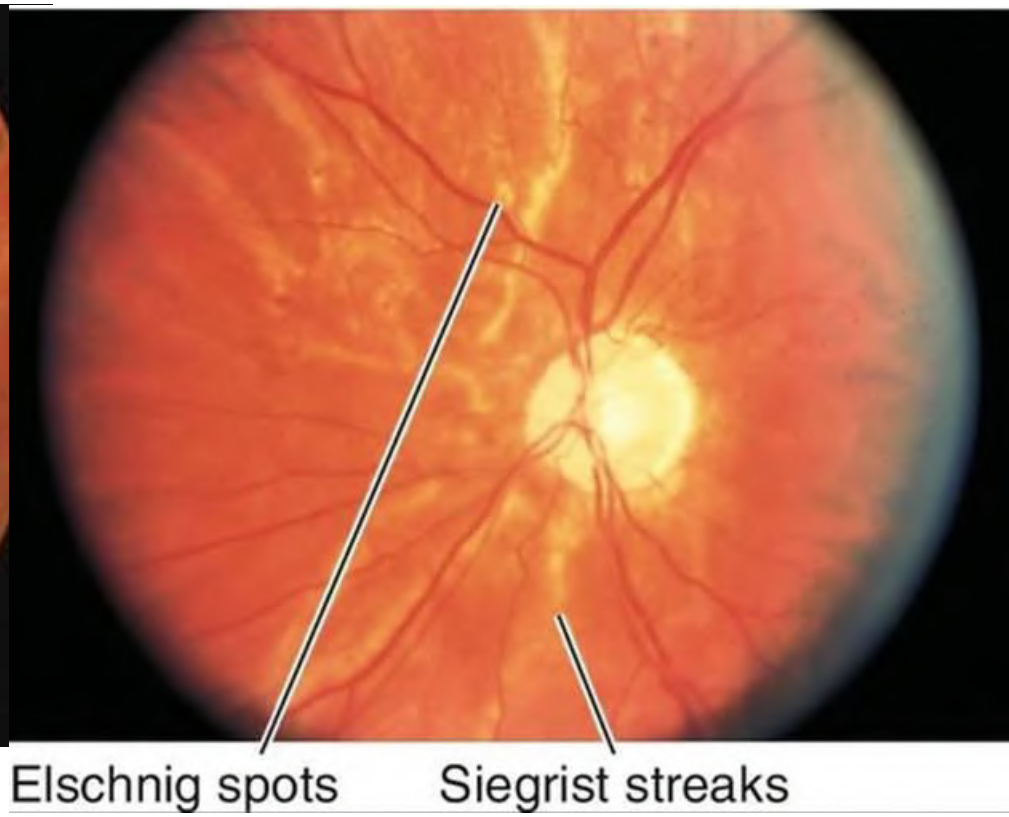
- Choroidopathy is rare but may occur as the result of an acute hypertensive crisis in young adults.

- **Elschnig spots** are small black spots surrounded by yellow halos which represent focal choroidal infarcts.

- **Siegrist streaks** are flecks arranged linearly along choroidal vessels indicative of fibrinoid necrosis associated with malignant hypertension

- **Exudative retinal detachment,** sometimes bilateral, may occur in severe acute hypertension such as that associated with toxaemia of pregnancy.



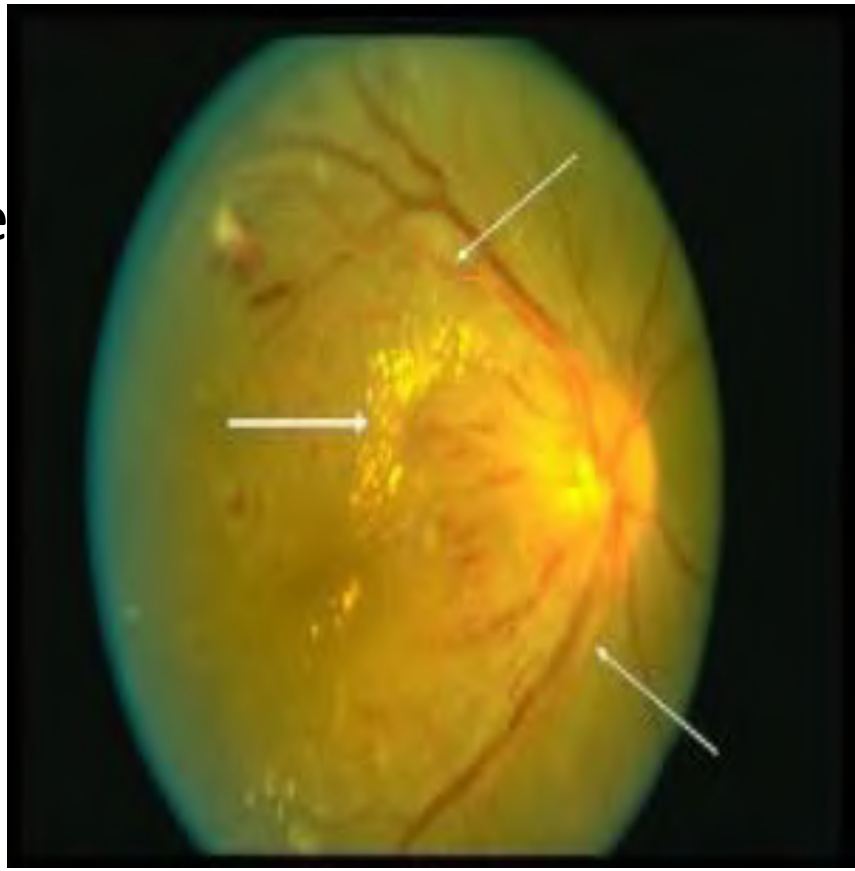


Elschnig spots

OPTIC NEUROPATHIES

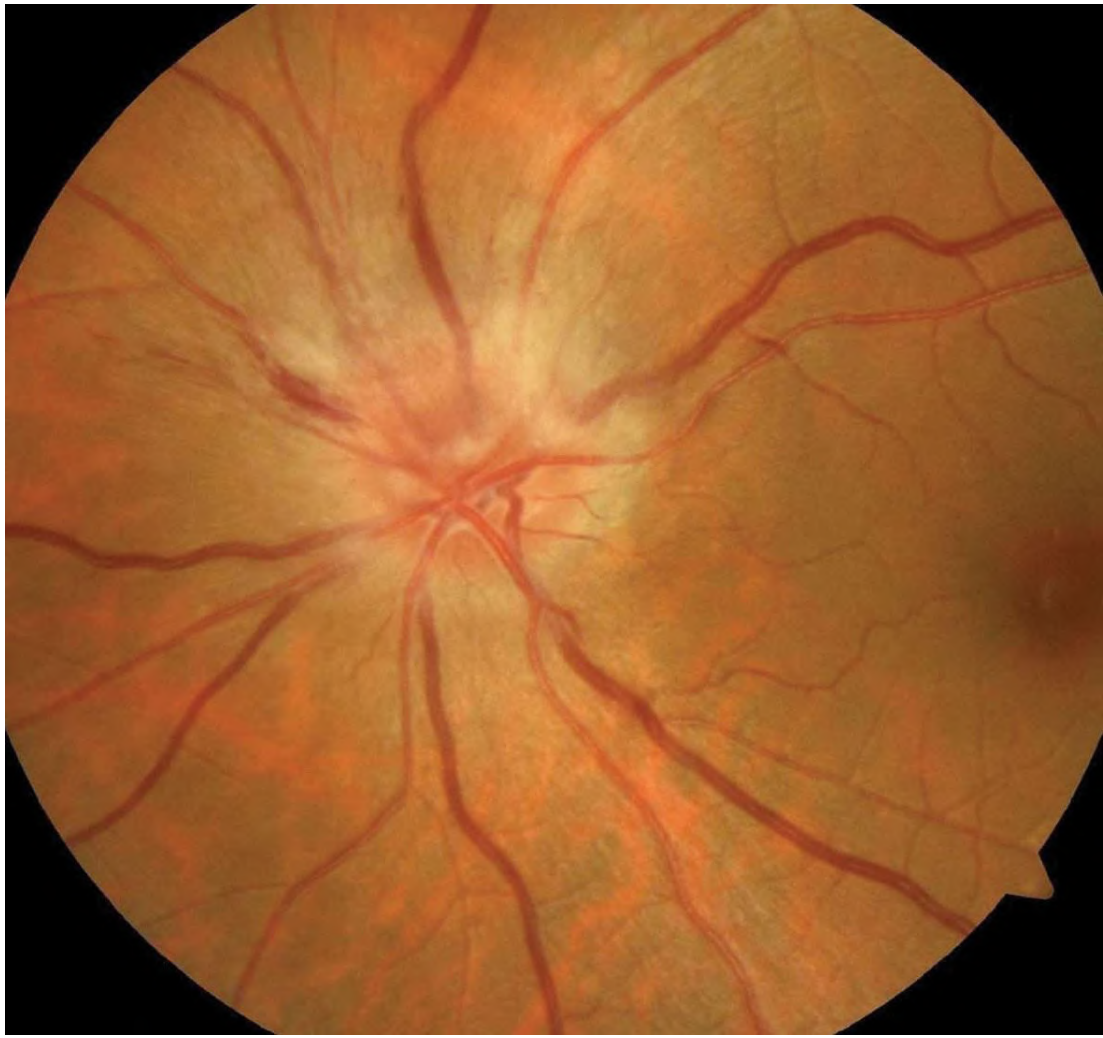
OPTIC DISC SWELLING

- flame shaped hemorrhages at the disc margin,
- blurred disc margins,
- congested retinal veins,
- papilledema
- secondary macular exudates

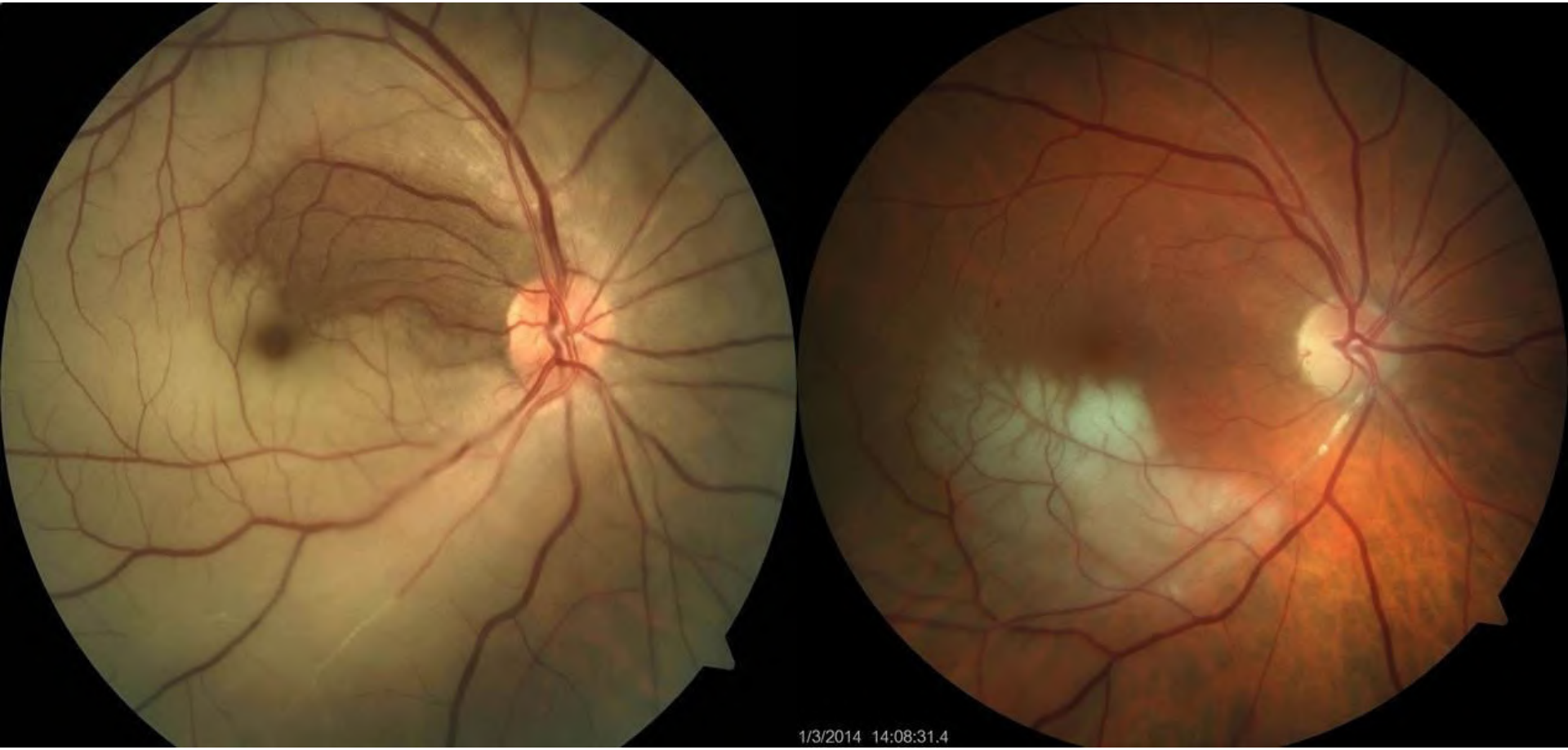


Non-arteritic anterior ischemic optic neuropathy (NAION)

- Sudden painless loss of vision or visual field problem
- Sectoral or total swollens disc with hemorrhages



ARTERY OCCLUSIONS



CRAO

BRAO

SYMPTOMS

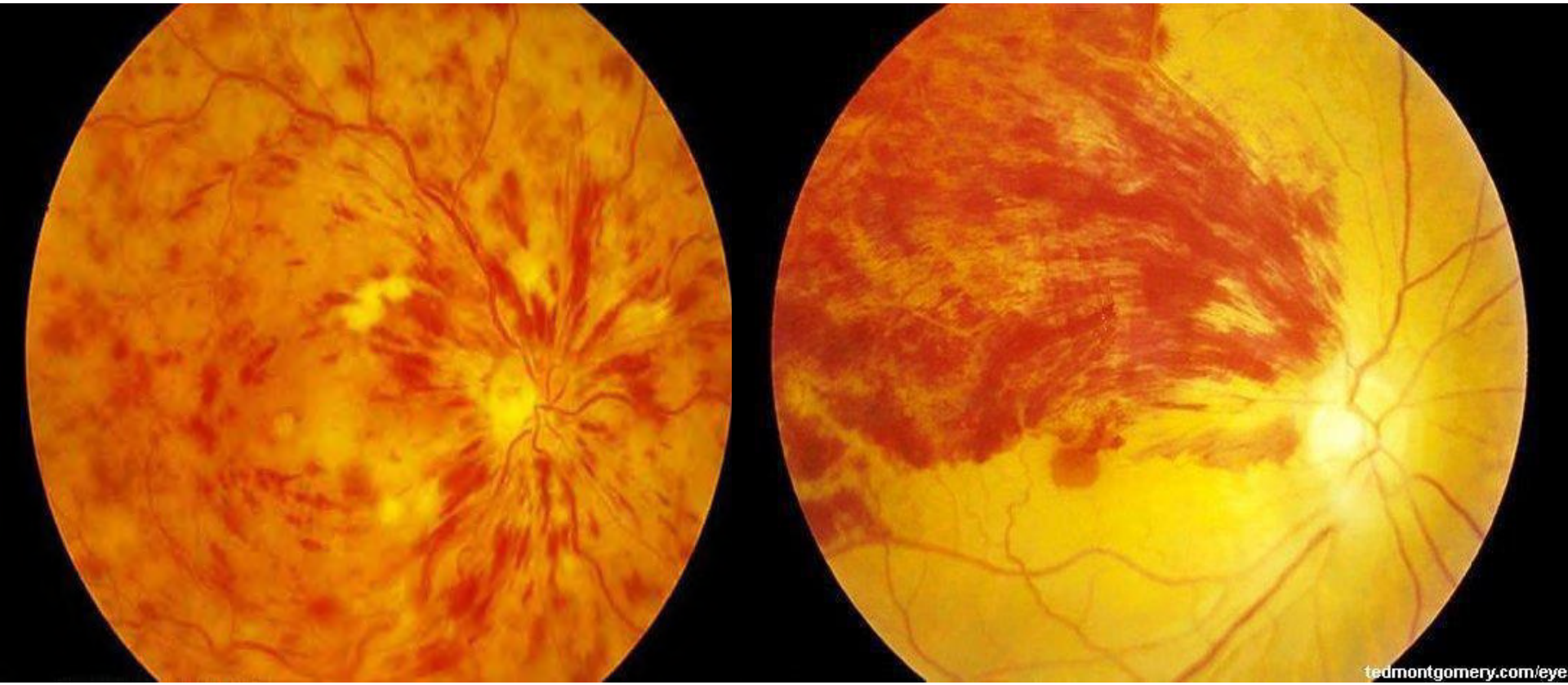
- PAINLESS SUDDEN UNILATERAL LOSS OF VISION. (PL or NPL)

- AMAUROSIS FUGAX

SIGNS

- LARGER ARTERIES THREAD LIKE AND ARTERIOLES ARE INVISIBLE.
- VEINS NORMAL .
- FEW HOURS THE RETINAL LOSES ITS TRANSPARENCY AND BECOME MILKY WHITE.
- CHERRY RED SPOT

VEIN OCCLUSIONS



CRVO

BRVO

SYMPTOMS

- PAINLESS SUDDEN UNILATERAL LOSS OF VISION.

FUNDUS

- MILD TORTUOSITY AND DILTATION .
- H’AGES-DOT n BLOT , FLAME SHAPED H’ GES.
- COTTON WOOL SPOTS
- DISC EDEMA.
- MACULAR EDEMA

Thank you

Night Blindness

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Associate Professor
KGMC/HMC

Etiology

Night Blindness may exist from birth or be caused by injury or malnutrition (for example, vitamin A deificiency).

Causes:

- Vitamin A deficiency
- Retinitis Pigmentosa & other retinal degenerations
- Congenital night blindness
- Pathological myopia
- Peripheral cortical cataract & Corneal opacities
- Advanced Primary open angle glaucoma

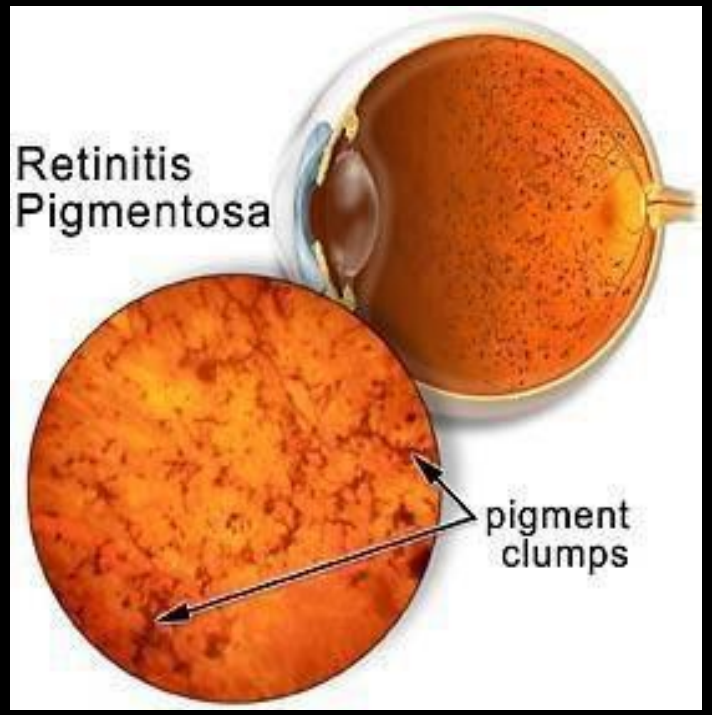
Retinitis Pigmentosa

- A disorder in which the rod cells in the retina gradually lose their ability to respond to the light.
- Patients suffering from this genetic condition have progressive nyctalopia and eventually their daytime vision may also be affected (Hemerlopia).

Mode of Transmission

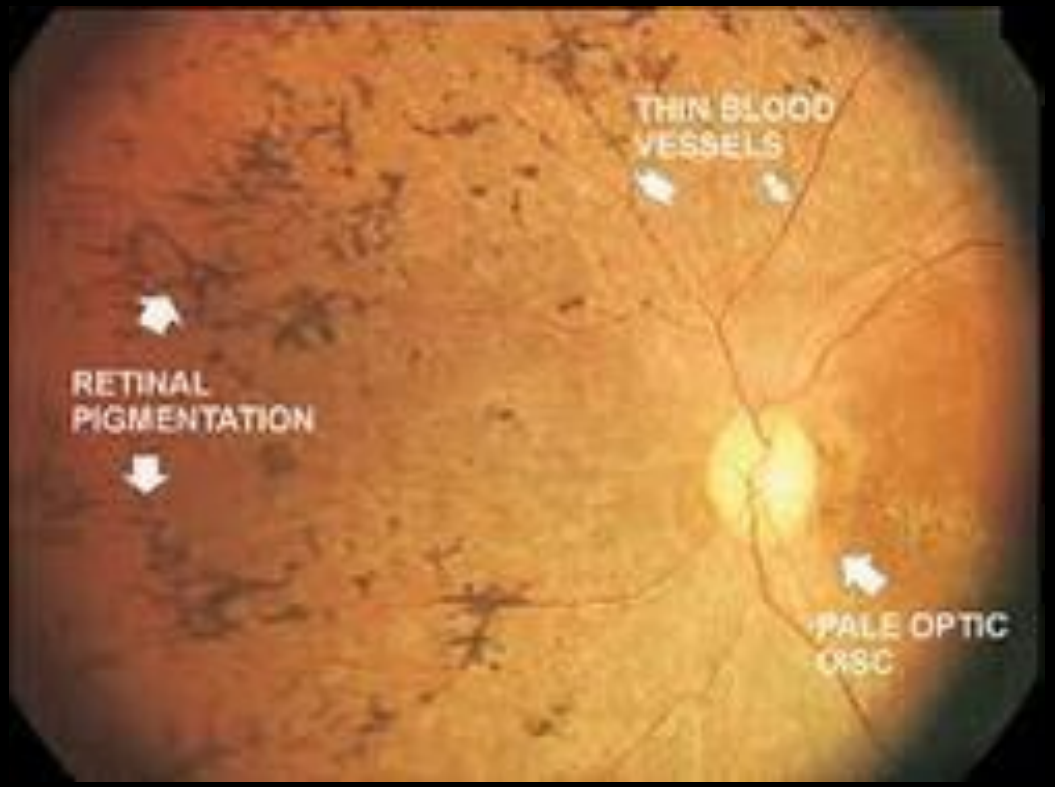
- Autosomal dominent
- Autosomol Recessive
- X – linked
- Sporadic

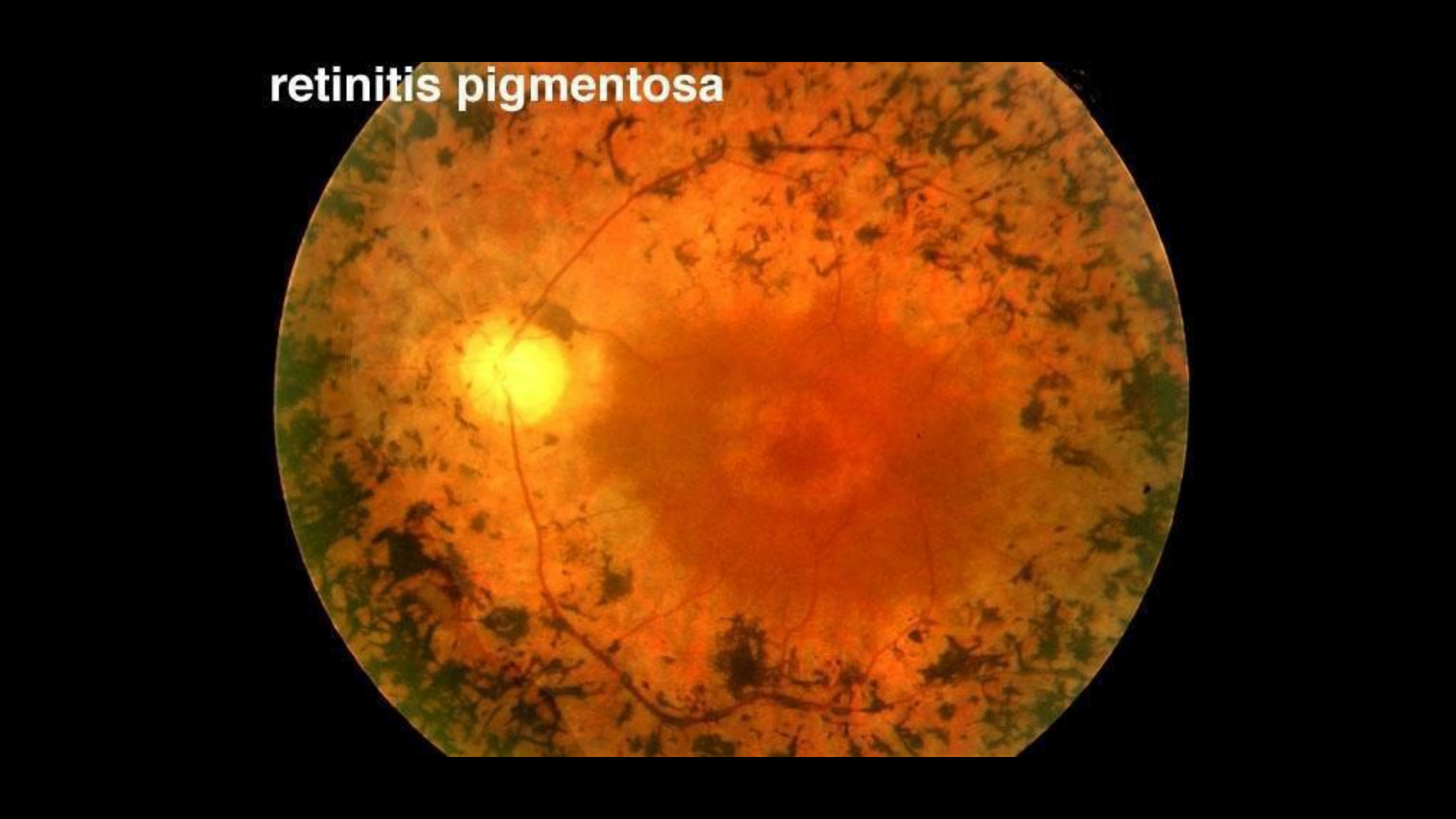
- Signs and Symptoms
  - Difficulty seeing dim lighting
  - Tendency to trip easily bump into objects when in poor lighting
  - Gradual loss of peripheral vision
  - Glare
  - Loss of contrast sensitivity
  - Eye fatigue (from straining to see)



Detection and Diagnosis

- Retinitis pigmentosa is usually diagnosed before adulthood.
- Often the patient complains of difficultly with night vision.
- The diagnoses by examining the retina with an ophthalmoscope
  - Waxy Pale Disc
  - Arteriolar attenuation
  - The classic sign of RP "bone-spicules.“
- Electroretinography (ERG).


THIN BLOOD
VESSELS
RETINAL
PIGMENTATION
PALE OPTIC
DISC


retinitis pigmentosa

Associations of RP

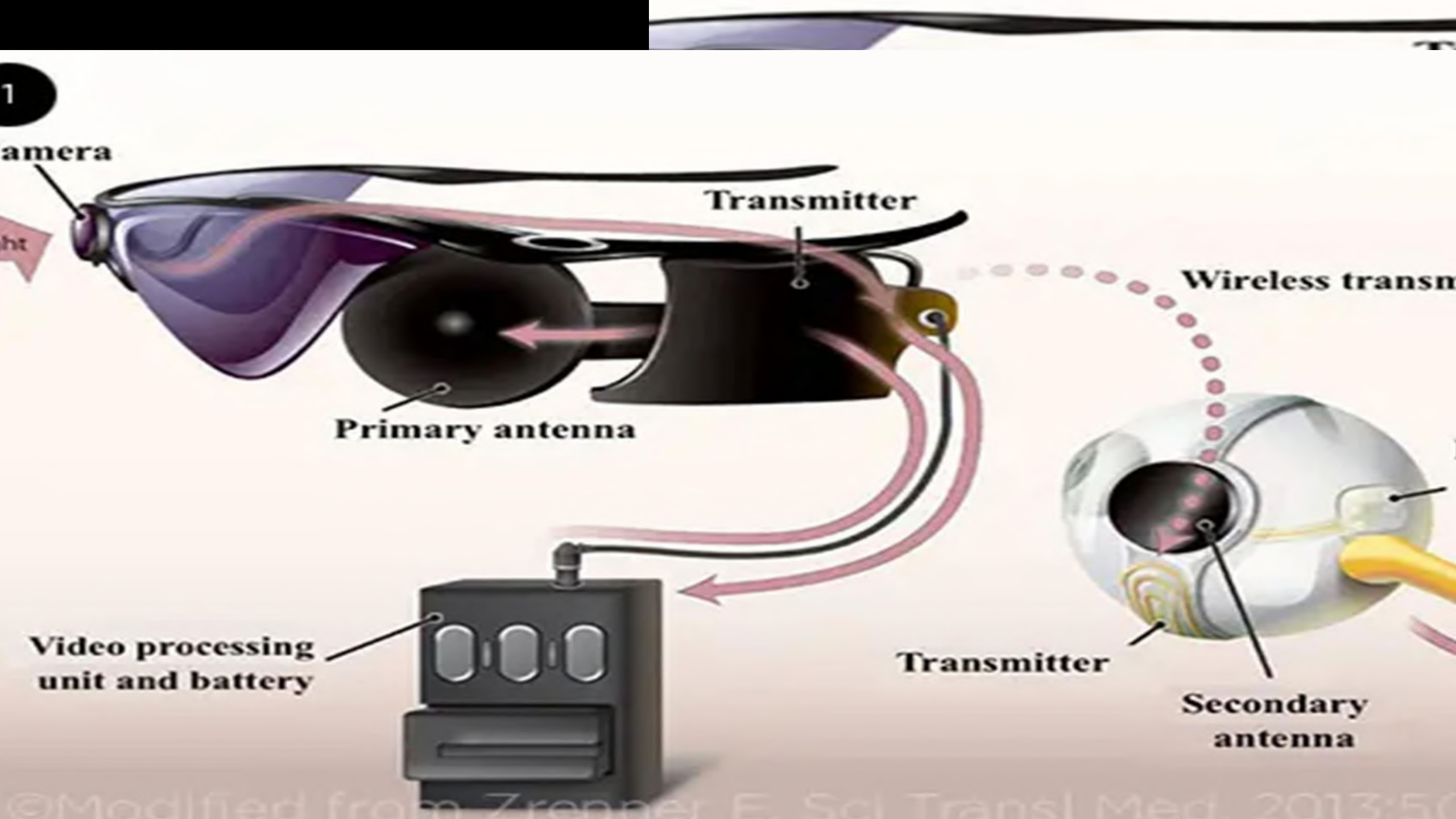
- Myopia
- Open angle glaucoma
- Macular oedema
- Retinal detachment
- Keratoconus
- Bardet Biedel syndrome
- Refusum disese
- Neuropathy
- Ataxia

Treatment

- There is currently no standard treatment or therapy for retinitis pigmentosa
- However, scientists have isolated several genes responsible for the disease.
- Once RP is discovered, patients and their families are encouraged to seek genetic counseling and trait.
- Low vision aids
- Diet and supplements

Treatment

- Vitamin A 15000 IU per day
- Omega 3 supplement
- Argus 2 Retinal prosthesis
- Gene Therapy (luxturna RPE65gene)
- Registration with Blind Association
- White stick
- Guide dogs
- Dark glasses
- Well illuminated bright light in the room.


amera
Transmitter
Wireless transm
Primary antenna
Video processing
unit and battery
Transmitter
Secondary
antenna

Vitamin A Deficiency

- Inhibits the production of rhodopsin (the eye pigment responsible for sensing low light situations) found in the retina, composed of retinal (active form of Vitamin A)
- The decreased amount of rhodopsin in the eye implies that there is inadequate retinal to bind to opsin
- Therefore, night blindness results

Functions of Vitamin A: Vision

- Retinal is a necessary structural component of rhodopsin or visual purple, the light sensitive pigment within rod and cone cells of the retina.
- If inadequate quantities of vitamin A are present, vision is impaired.

Causes

- Ineduate intake
- Fat malabsortion
- Liver diseases

Night Blindness

- Lack of vitamin A causes night blindness or inability to see in dim light.
- night blindness occurs as a result of inadequate pigment in the retina.
- It also called tunnel vision.
- Night blindness is also found in pregnant women in some instances, especially during the last trimester of pregnancy when the vitamin A needs are increased.

Night blindness

In dim light, you can make out the details in this room. You are using your rods for vision.

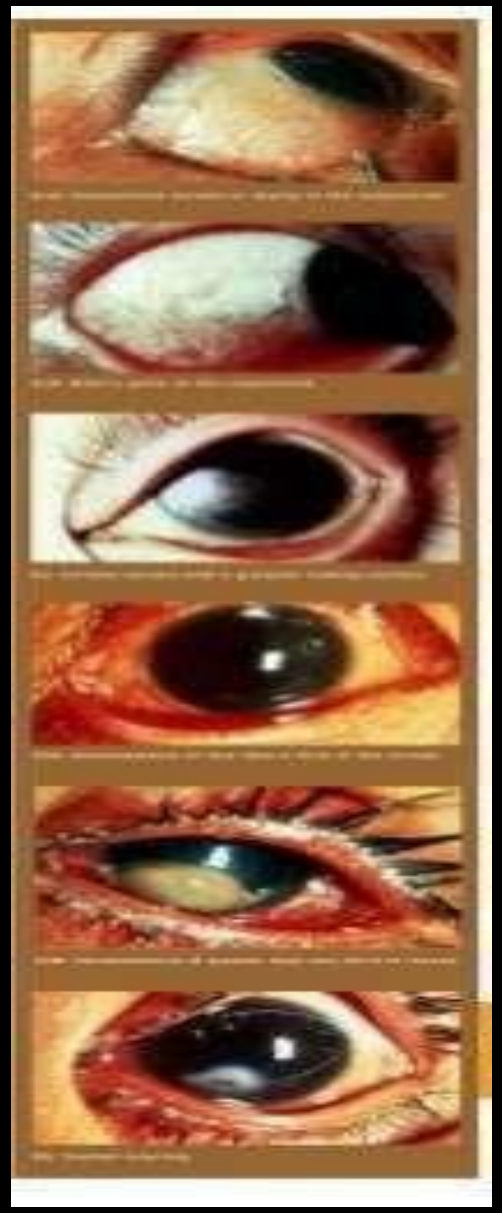
A flash of bright light momentarily blinds you as the pigment in the rods is bleached.

You quickly recover and can see the details again in a few seconds.

With inadequate vitamin A, you do not recover but remain blinded for many seconds.

Classification of xerophthalmia

- XN Night blindness
- X1A Conjunctival Xerosis
- X1B Bitot’s spot
- X2 Corneal Xerosis
- X3A Corneal ulceration/keratomalacia (< 1/3 corneal surface)
- X3B Corneal ulceration/keratomalacia (≥ 1/3 corneal surface)
- XS Corneal scar
- XF Xerophthalmic fundus

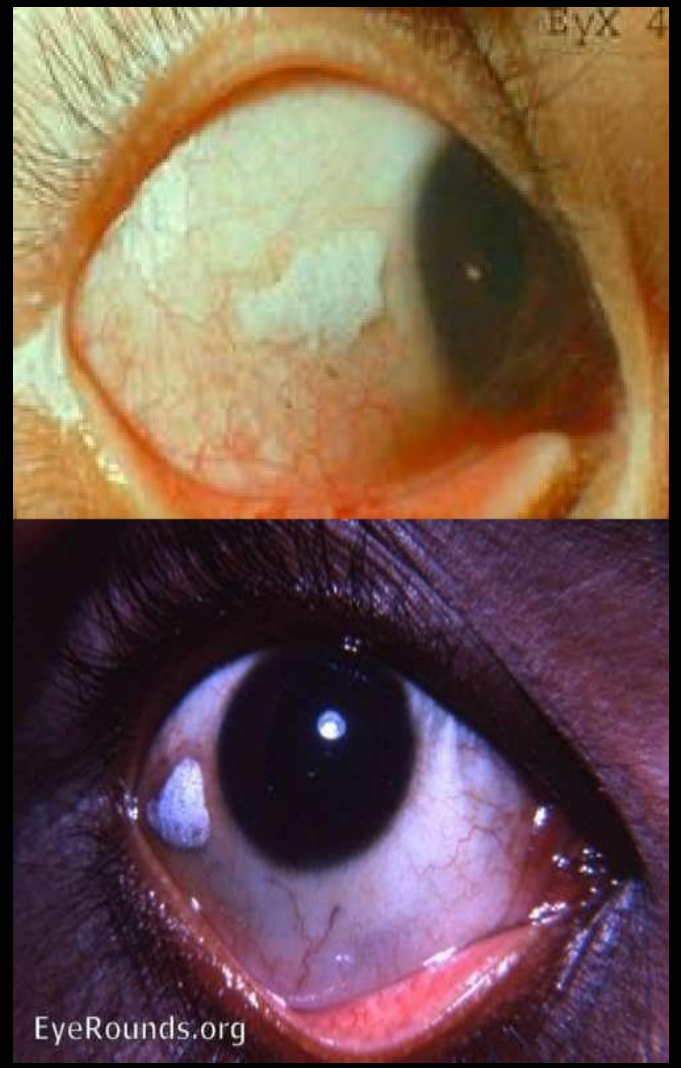


Conjunctival Xerosis

- Conjunctiva becomes dry and non wettable.
- Instead of looking smooth shiny it appears muddy &wrinkled.

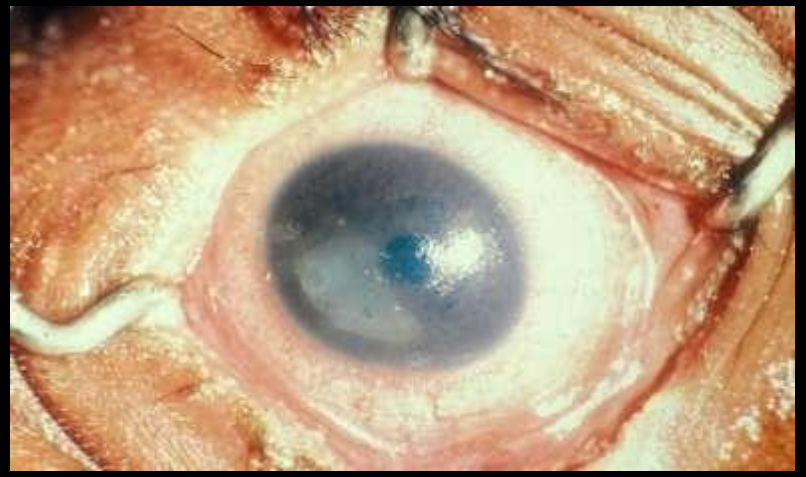
Bitot’s Spot

- These are foamy and whitish cheese-like tissue spots that develop around the eye ball, causing severe dryness in the eyes.
- These spots do not affect eye sight in the day light.



Keratomalacia

- One of the major cause for blindness.
- Cornea becomes soft and may burst.
- The process is rapid.
- If the eye collapses vision is lost.



Recommended treatment schedule

	6 -12 months	> 1 yr
Immediately	100,000 IU	200,000 IU
Next day	100,000 IU	200,000 IU

- 2–4 weeks later 100,000 IU 200,000 IU
- Severe Protein-Energy Malnutrition (PEM) Monthly until PEM resolves
  100,000 IU 200,000 IU

Summary

- Night Blindness
- Vitamin A deficiency
- Retinitis Pigmentosa

Any Questions?

MCQ

1. A 25 years old boy presents with complaints of night blindness since childhood which is progressive. There is no past history of glasses use or trauma. On examination his fundi shows waxy pale disc, arteriolar attenuation and pigmentary changes (bone spicules).

What is your most likely diagnosis in this case?

1. Cataract
2. Myopia
3. Retinitis Pigmentosa
4. Primary Open Angle glaucoma
5. Vitamin A deficiency

Ans: 3

OPTIC NEURITIS

DR UMER KHAN ORAKZAI

Associate Professor

KGMC/HMC

- An acute inflammatory disorder of the optic nerve
- Typically presents with sudden monocular visual loss and eye pain
- In young adults, more commonly in women
- A common initial manifestation of multiple sclerosis (MS)

OPTIC NEURITIS

CLASSIFICATION:

ETIOLOGICAL:

a. Idiopathic

b. Demyelination

c. Infections (viral) etc.

d. Para infections/Post viral syndrome

e. Toxic-Drugs

f. Intraocular inflammations

g. Contiguous inflammations (Sinus, Orbit)

h. Systemic disease-sarcoid, T.B, Syphilis

❖Para infectious-after viral infections, immunization

❖Infections-viral infections, cat scratch fever, syphilis, Lyme disease

AIDS usually causes-Neuroretinitis

- Drugs-Ethambutol, Isoniazid, Interferon, Chloramphenicol
- I.O. Inflammations: Uveitis, APMPPE
- Sinus related-Headache + ENT opinion

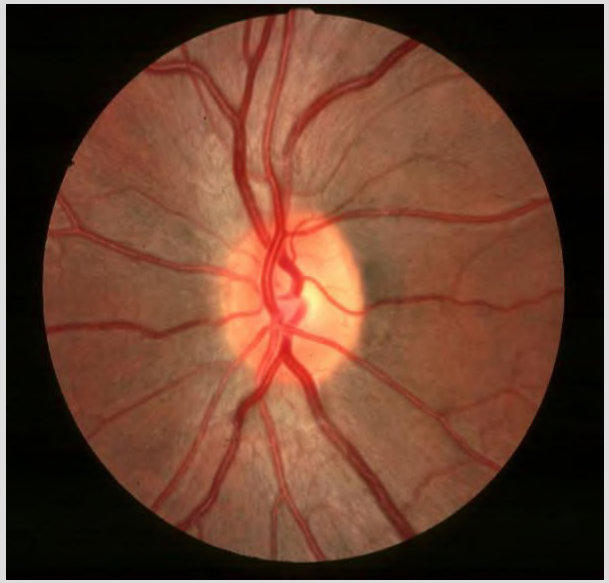
OPTIC NEURTIS

CLINICAL TYPES

- Retrobulbar (1/3rd cases)
- Papillitis (1/3rd cases)
- Neuroretinitis

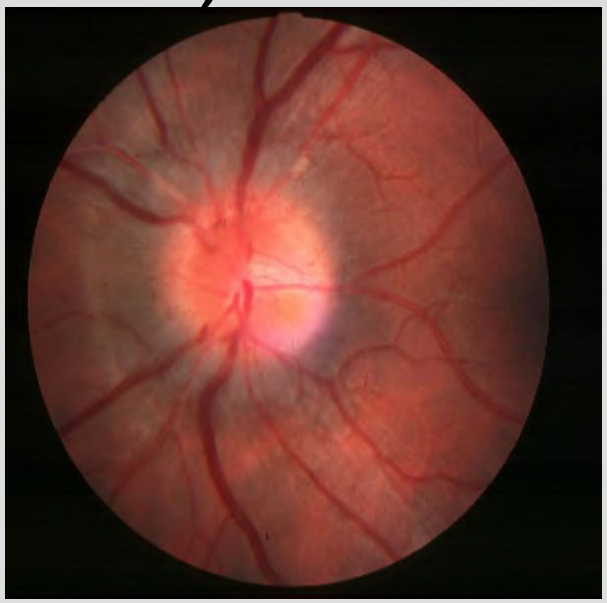
Classification of optic neuritis

Retrobulbar neuritis (normal disc)



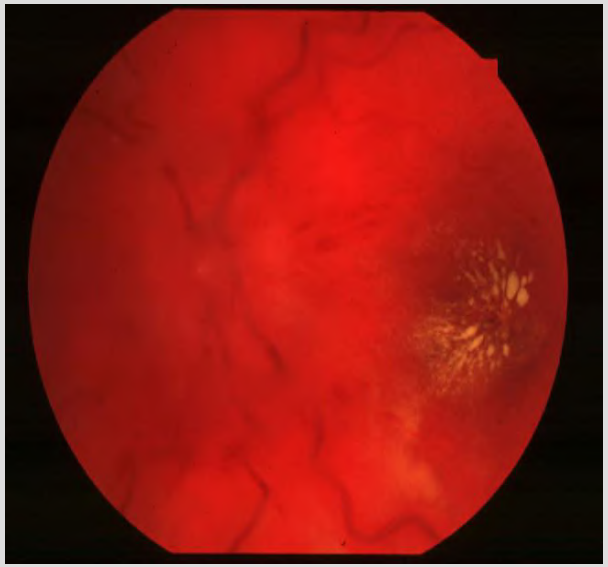
- Demyelination - most common
- Sinus-related (ethmoiditis)
- Lyme disease

Papillitis (hyperemia and oedema)



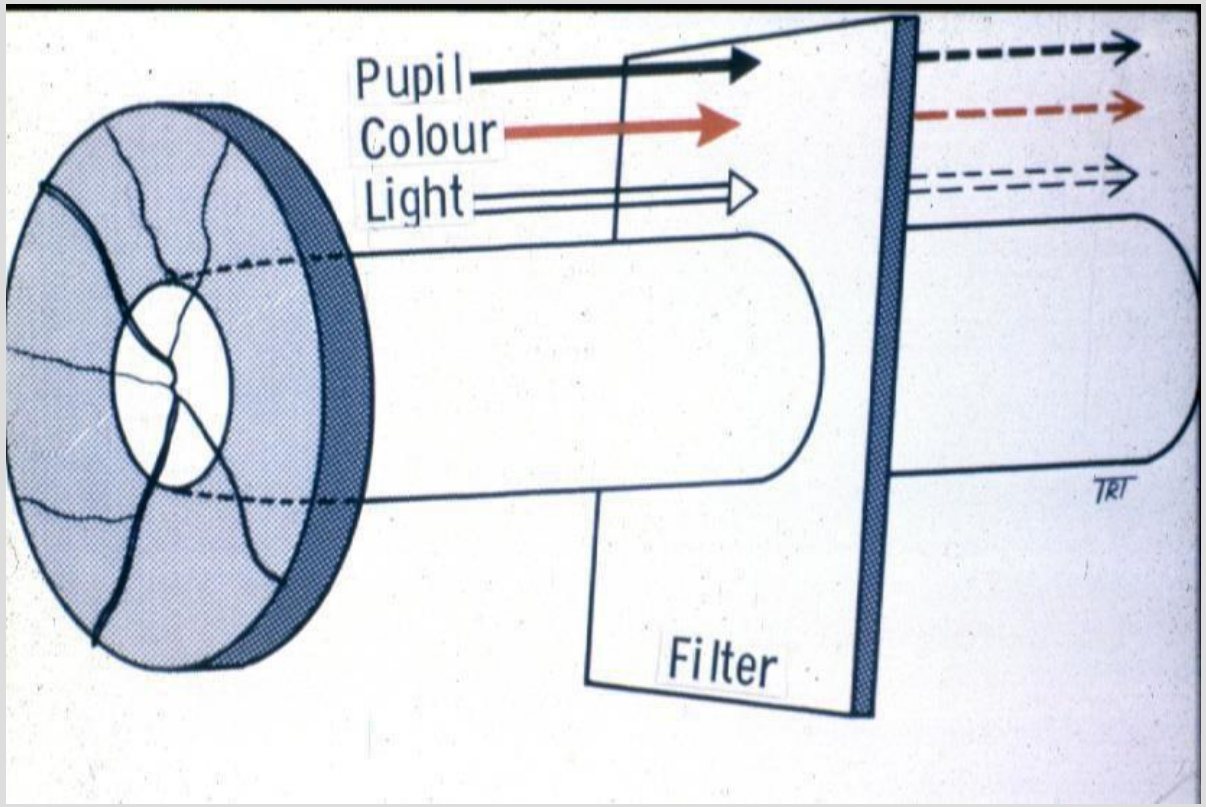
- Viral infections and immunization in children (bilateral)
- Demyelination (uncommon)
- Syphilis

Neuroretinitis (papillitis and macular star)



- Cat-scratch fever
- Lyme disease
- Syphilis

Signs of optic nerve dysfunction



- Reduced visual acuity
- Afferent pupillary conduction defect
- Dyschromatopsia
- Diminished light brightness sensitivity

SYMPTOMS: Triad

- ❖Loss of vision
- ❖Ipsilateral eye pain
- ❖Dyschromatopsia

70% - initially unilateral

30% - 2nd eye may get involved

(1) Loss vision: 58% - isolated symptom

Range – 6/6 – PL-ve

Vision decreases for 10-14 days

Stabilize – Improvement (2-3 months)

(II) PAIN: Ipsilateral eye pain – 88%
with eye movements – 21% "Dull ache" in/behind the eye – 62% headache (involved region) 22% generalized headache- 13%
Pain – cause unknown with in 24 hrs. then recovers in 48-72 hrs.

O.N.T.T. (Pain) – 92% cases.

Persistent Pain 5 days – Atypical

(III) DYCHROMATOPSIA: Impaired color vision
Color desaturation – No macular lesions highly suggestive of O.N disease.
Color defect – worse than expected.

OTHER FEATURES:

1) **RAPD:-** Ipsilateral – Neutral density filter

2) **CONTRAST SENSITIVITY:** Most sensitive (Even if V.A 6/6)

3) **VISUAL FIELD DEFCTS:** Central scotoma – may be altitudinal, arcuate, diffuse etc.

4) **PULFRCH EFFECT:** Pendulum movements appears elliptical when observed in front of eyes. [Delayed transmission + weak stimulation]

5) **UHTHOFF'S SYMPTOM:** episodic transit worsening of vision with exertion [Exertion, hot food/drink, Tired end day] Recovery – within 5-6 min/sometimes 2hrs. Pts with Uhthoff's – Higher incidence of MS, May be present in lebers optic N, Toxic optic N (Chloramphenicol).

6) **INVERSE UHTHOFF's SYMPTOM** – Improved vision with exercise, beer etc.

.

7) Visual obscuration in bright light: O.N Pts vision in bright light – see better in dim light.

8) **MOVEMENT PHOSPHENES:**

Phosphenes: Seeing brief flash of light (1-2 sec). Almost exclusively and horizontal eye movements better seen in dark/dim room with closed eyes ipsilateral and unilateral suggests demyelination

9) **SOUND PHOSPHENES:** Produced by sudden noise when pt. is resting in the dark. May occur in optic neuritis or compressive neuropathy

PROGNOSIS: 75 – 6/9 OR BETTER V.A

- 85% 6/12 OR BETTER V.A

- Color vision, contrast sensitivity, light brightness appreciation often
- remain abnormal. Mild RAPD may stay and optic atrophy may start.

OPTIC NEURTIS TREATMENT TRIAL:

- Multicentered, Randomized, Prospective clinical trial – 457 cases of
- O.N (81-45 years- Acute O.N for 8 days + visual)
- Field defects + RAPD – Included (Typical)

OPTIC NEURITIS TREATMENT TRIAL

- I): Oral prednisone – 1 mg/kg/day – 14 days
- II): I/V Methyl prednisolone (1000 mg/day) for 3 days f/b oral prednisone (1mg/kg/day) for 11 days.
- III): Oral placebo for 14 days.

OPTIC NEURITIS TREATMENT TRIAL

Results

- Oral steroids – recurrence of O.N in affected or contralateral eye.
- I/V steroids → Fast recovery of vision for the first year but then after no difference.

TREATMEMT:

- Mild cases – No treatment
- Cases with V.A > 6/12 I/V steroids f/b oral steroids – speed up recovery and lower the incidence of MS in first two years.

No long-term benefit on the final V.A.

WORKUP

- → All patients with O.N – MRI – if normal no further work.
- → MRI show 2 or > 2 typical lesion then I/V steroids f/b oral may incidence of MS in first two years.

ASSOICATION OF MS & OPTIC NEURITIS:

1) 15 – 20% pts with MS will present with O.N.
2) 35 – 40% pts with MS will develop optic neuritis during course of their disease.
3) 74% female and 34% male with optic neuritis will develop MS when followed up to 15 years

4) 50% -70% of clinically isolate optic neuritis have abnormal MRI similar to that seen in MS.
5) Risk of MS is increased in pts of optic neuritis when there is winter onset, HLA DR2 positively and Uhthoff's phenomenon.
6) 36 eyes of 18 pts – when alive only 8 of 18 pts had diagnosis of unilateral or bilateral optic neuritis.

On Autopsy35 eyes out of 36 eyes showed evidence of demyelination.

Thank You

Optic Neuropathy

Dr Umer Khan
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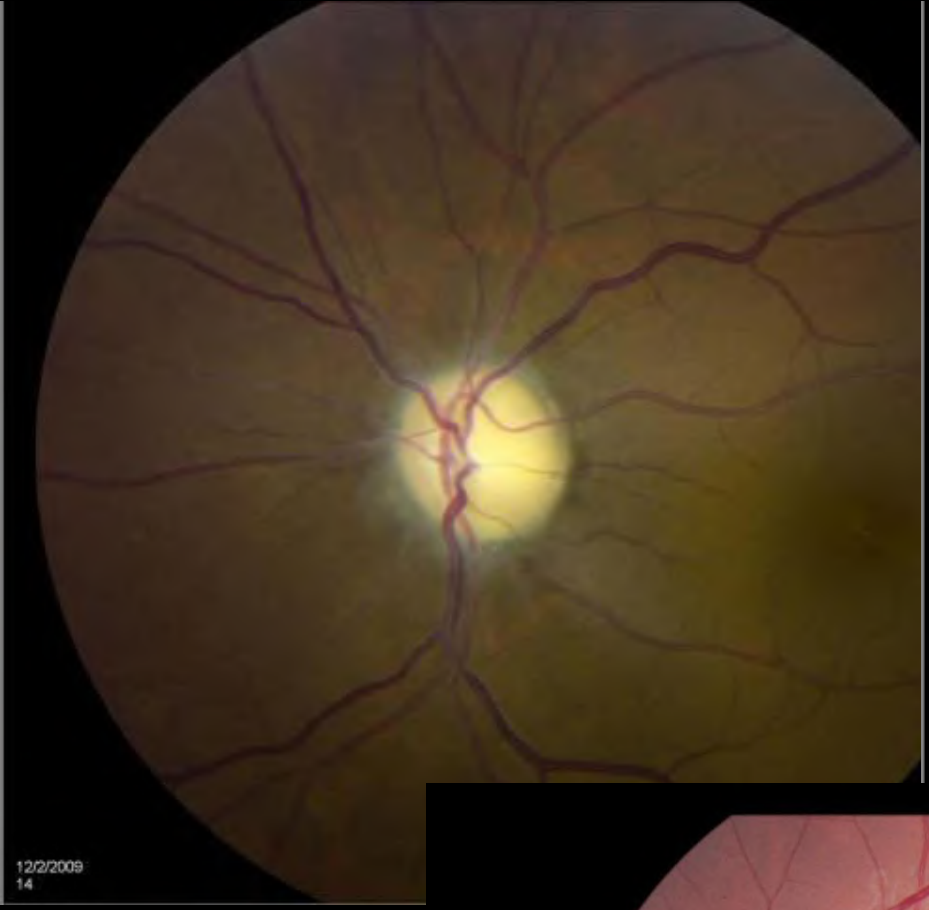
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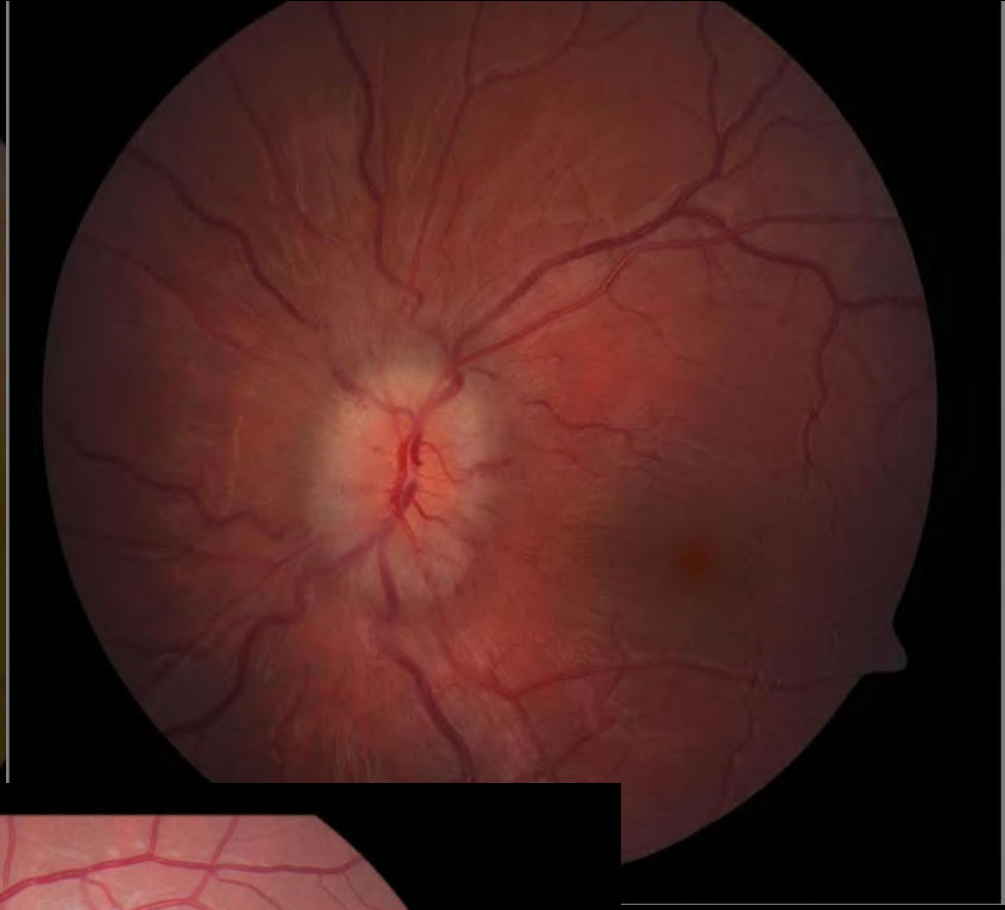
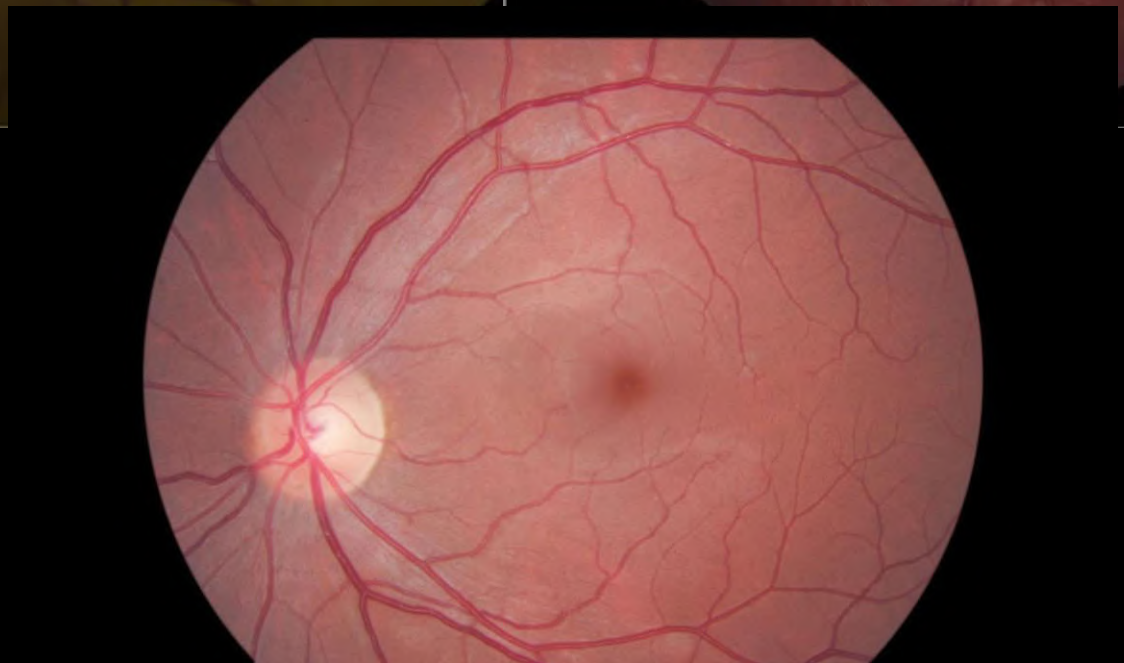
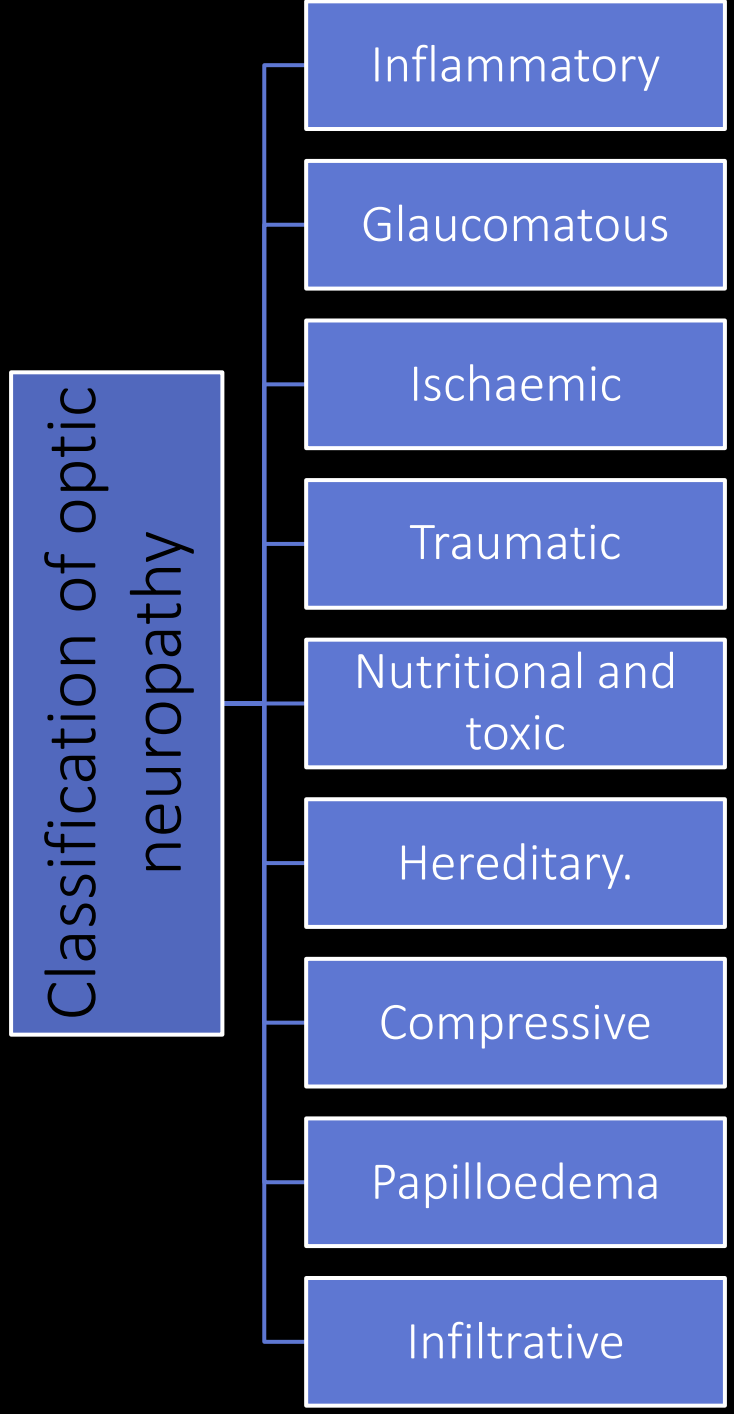




Optic neuropathy

Optic neuropathy is damage to the optic nerve from any cause.
Damage and death of these nerve cells, or neurons, leads to characteristic features of optic neuropathy.


Classification of optic neuropathy
Inflammatory
Glaucomatous
Ischaemic
Traumatic
Nutritional and toxic
Hereditary.
Compressive
Papilloedema
Infiltrative

Optic Neuropathies : Causes

- **Demyelinating**
- Inflammatory
- Non-arteritic Ischemic
- Arteritic Ischemic
- Traumatic

Rapid onset

- Infiltrative
- Compressive
- Hereditary
- Radiation
- Paraneoplastic
- Toxic/nutritional

Gradual onset

Hereditary Optic Neuropathy

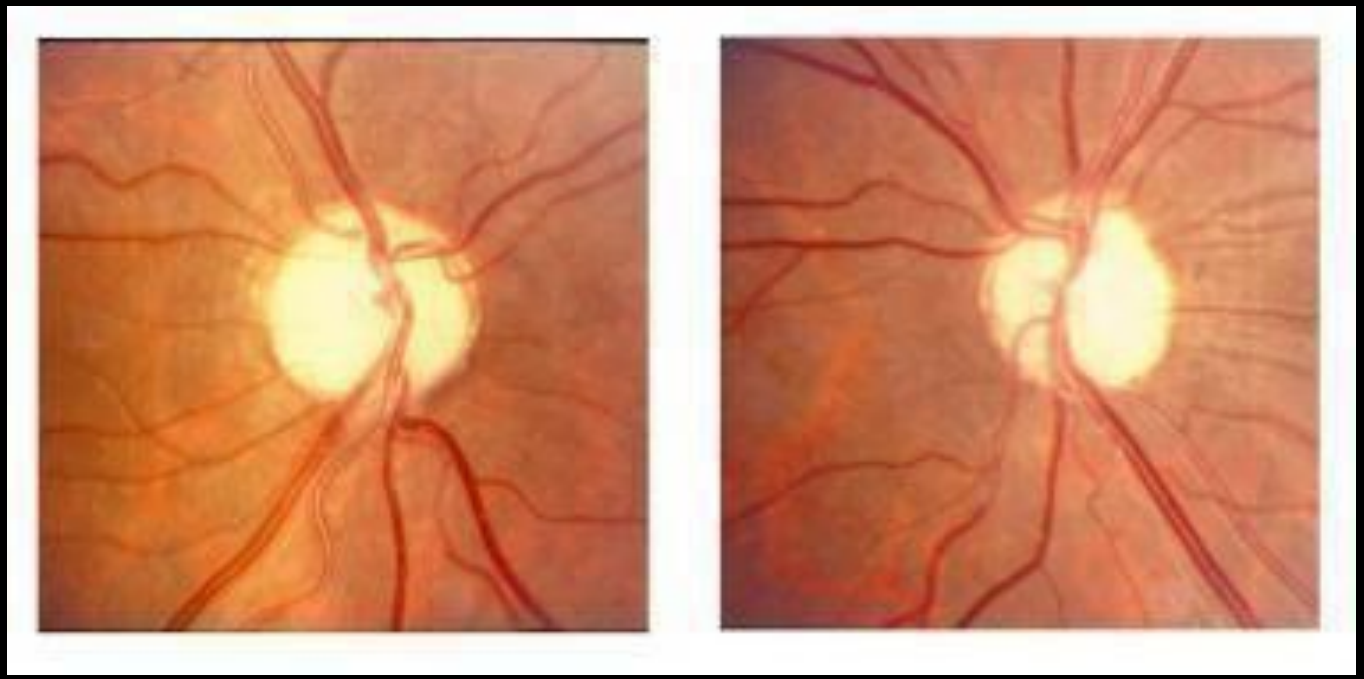
- AD
- AR
- XL

HEREDITARY OPTIC NEUROPATHY: AD (KJERS' TYPE)

- 1st decade of life
- Bilateral symmetric visual loss.
- Bilateral central or cecocentral scotomas.
- Color vision deficit .

HEREDITARY OPTIC NEUROPATHY: AD (KJERS' TYPE)

- The optic disc : temporal pallor and in some cases severe excavation and cupping.



HEREDITARY OPTIC NEUROPATHY: BEHR SYNDROME

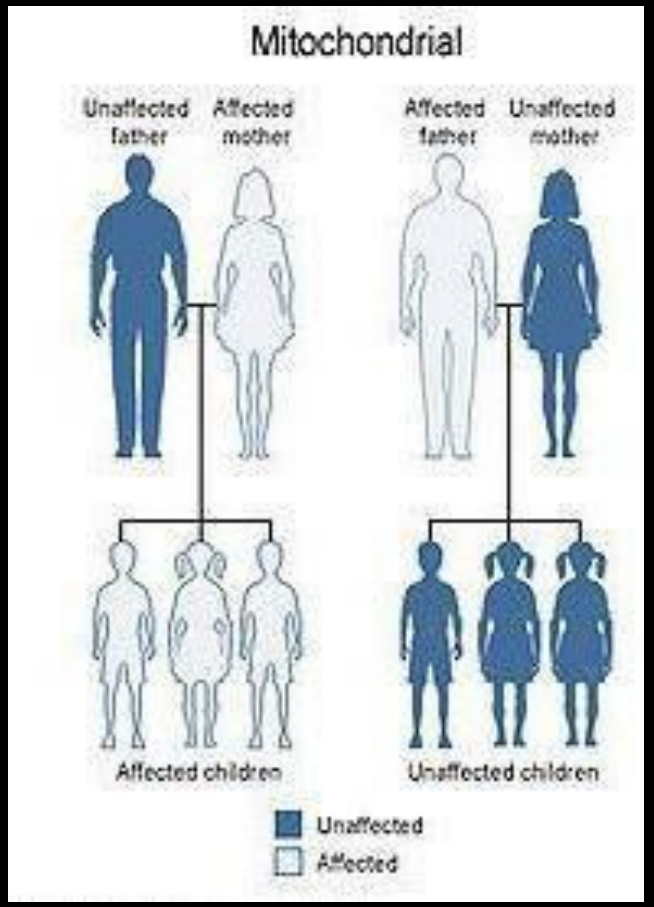
- Inheritance is AR
- Presentation is in early childhood with reduced vision.
- Optic atrophy is diffuse.
- Prognosis is variable, with moderate to severe visual loss and nystagmus.
- Systemic abnormalities include spastic gait, ataxia and mental handicap.

HEREDITARY OPTIC NEUROPATHY; WOLFRAM SYNDROME

- also referred to as DIDMOAD (diabetes insipidus, diabetes mellitus, optic atrophy and deafness).
- 1st year of life
- inheritance being AR, AD or via the maternal mitochondrial line.
- Optic atrophy is diffuse and severe and may be associated with disc cupping.

HEREDITARY OPTIC NEUROPATHY: LHON

- leber hereditary optic neuropathy
- LHON has 4 primary mitochondrial mutations
- M>F
- The frequencies of mutation may vary across different countries



HEREDITARY OPTIC NEUROPATHY: LHON

- Acute unilateral, painless, visual loss.
- some cases may stay asymptomatic or have a chronic course
- Sequential bilateral involvement may occur weeks or months later.

May demonstrate neurological as peripheral neuropathy, ataxia, Dystonia and cardiac conduction defects

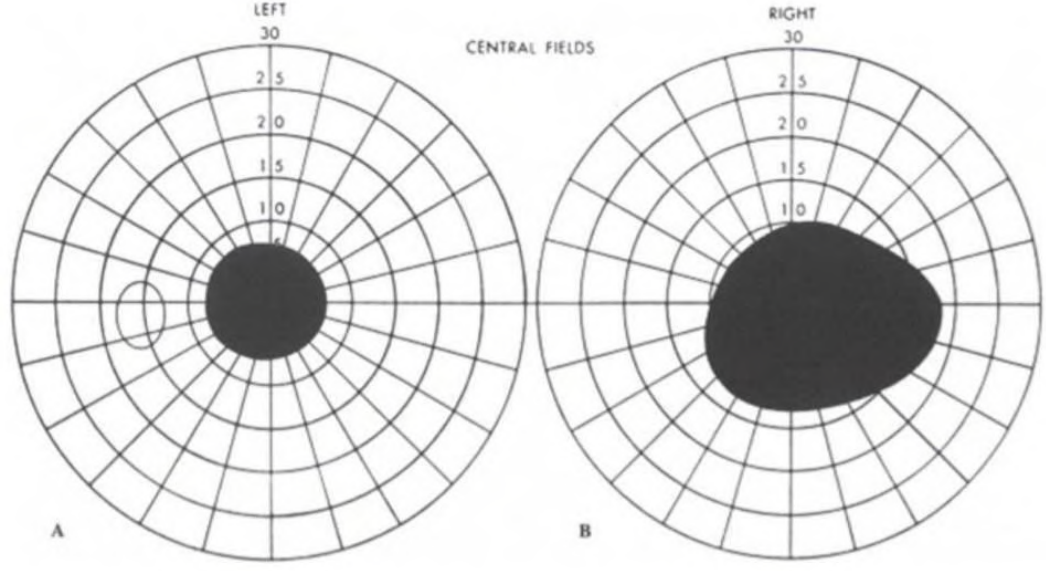
HEREDITARY OPTIC NEUROPATHY: LHON

- Occasionally, optic nerve pallor can be seen initially. Because of the wide age range (6–80 years old) at which LHON may present, it is frequently misdiagnosed

- Young patients are often diagnosed as optic neuritis and older patients as ischemic or infiltrative optic neuropathy.

HEREDITARY OPTIC NEUROPATHY: LHON

- Visual filed defects tend to be central or cecocentral as the papillo-macular bundle is first and most severely



HEREDITARY OPTIC NEUROPATHY: LHON

- Fundoscopy may show disk swelling, thickening of the peripapillary retinal nerve fiber layer

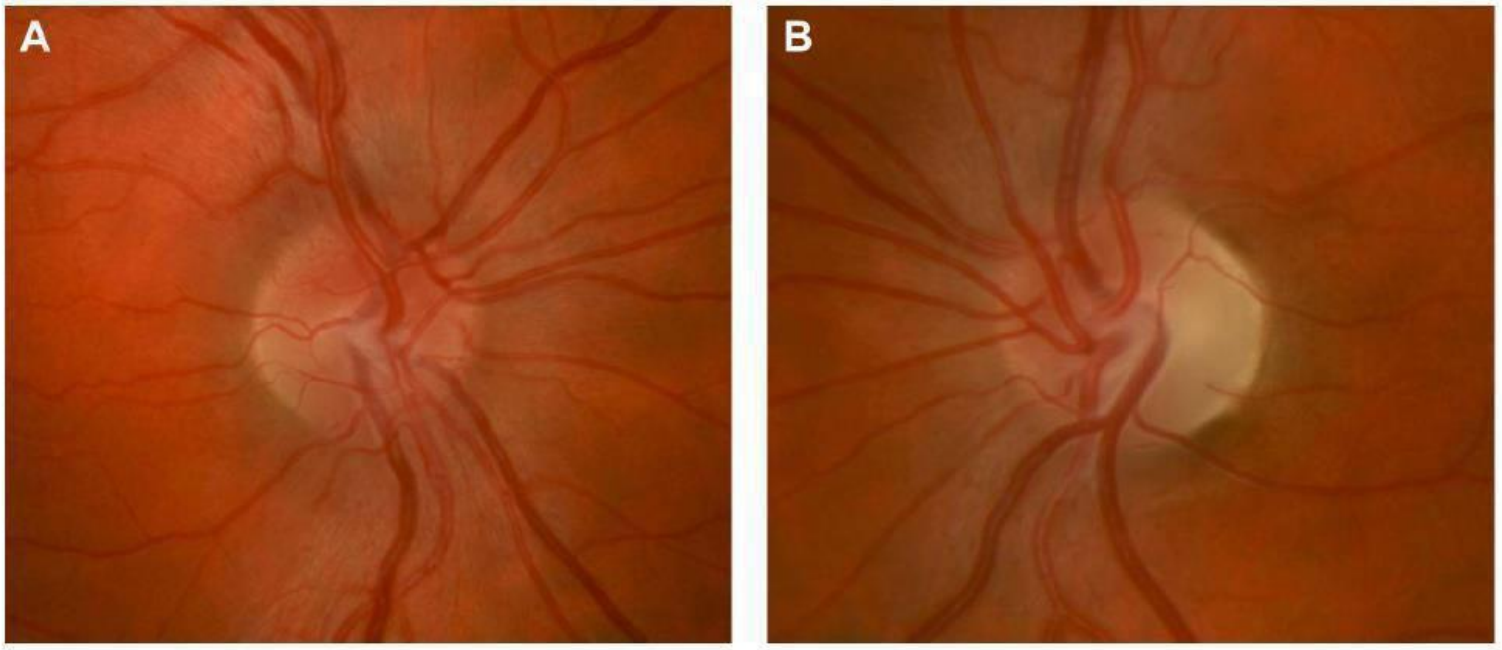


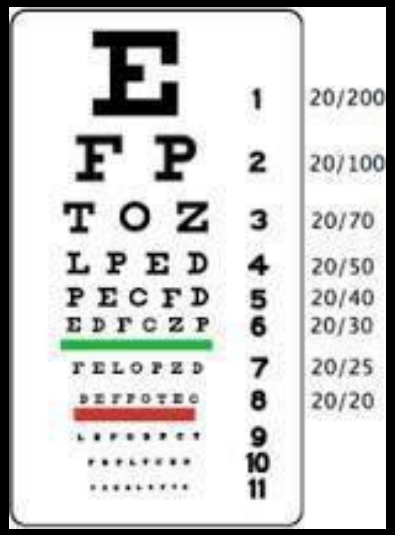
Figure 2 Right optic nerve (**A**) of a patient with acute LHON-related vision loss showing mild hyperemia, blurring of the disc margin, and elevation of the optic nerve head from swelling of the peripapillary retinal nerve fiber layer. LHON-related vision loss in the left eye had occurred 6 months prior leading to prominent temporal optic nerve pallor (**B**) from atrophy of the retinal nerve fiber layer.
Abbreviation: LHON, Leber hereditary optic neuropathy.

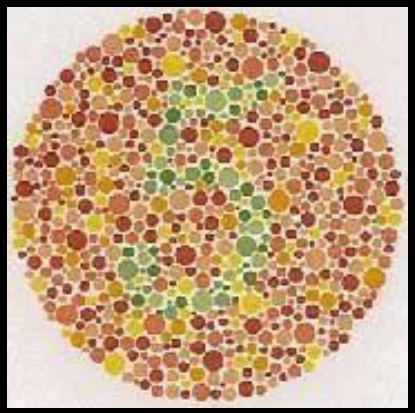
TREATMENT •

- Apart from symptomatic measures such as low vision aids, treatment is generally ineffective.
- Dietary deficiencies should be avoided, particularly of B12.
- Smoking and excessive alcohol consumption should be discouraged, theoretically in order to minimize mitochondrial stress.
- Idebenone appears to be neuroprotective in this condition and may have a role to play in some patients.
- Gene therapy is under active investigation

Toxic Optic Neuropathies

- Visual acuity may vary from minimal reduction to no light perception (NLP) in rare cases.
- Most patients have 20/200 vision or better.
- Color vision should be assessed because dyschromatopsia is a constant feature in these conditions.



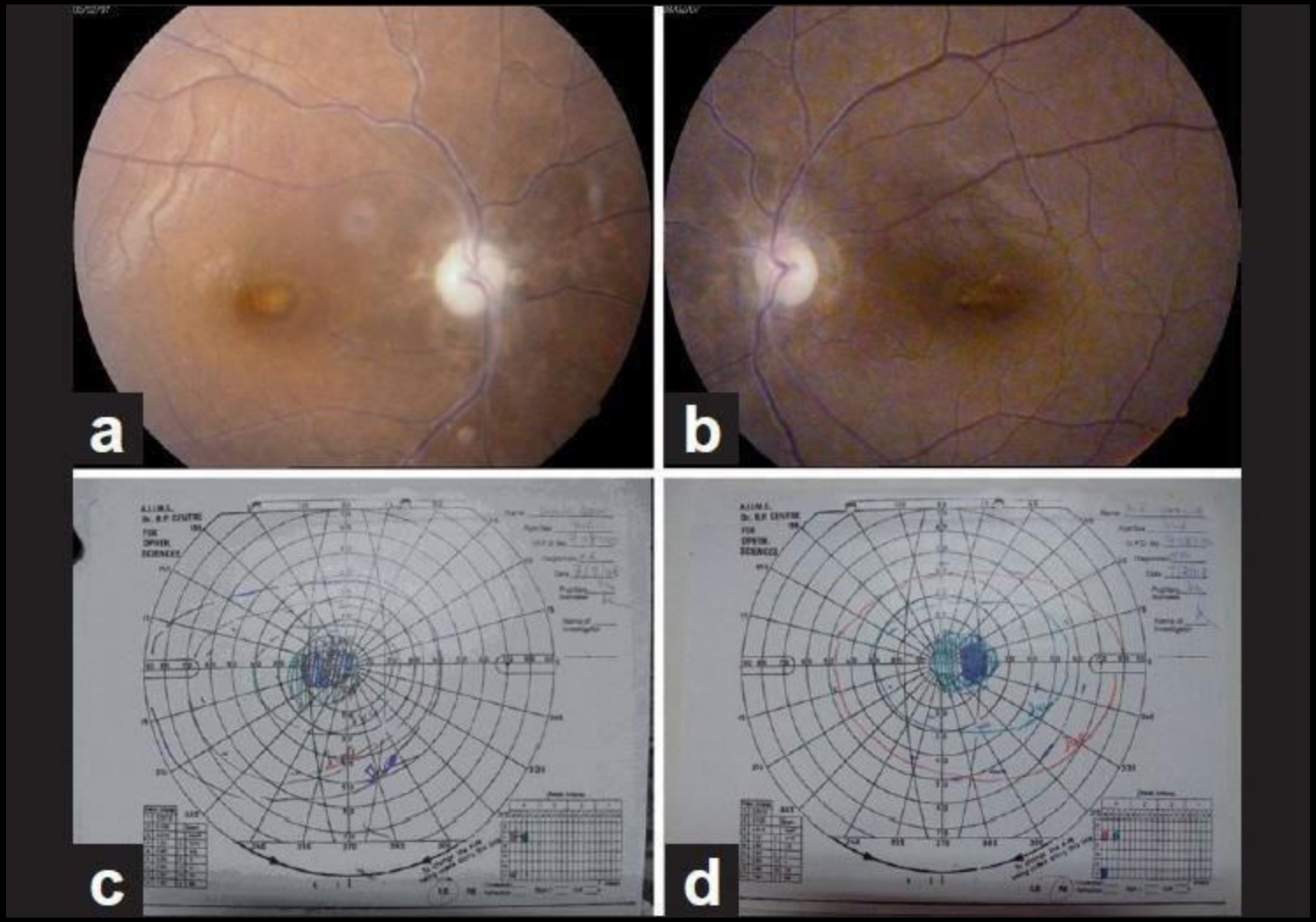


Toxic Optic Neuropathies

- In the early stages of toxic optic neuropathies, most patients also have normal-appearing optic nerves, but disc edema and hyperemia may be seen in some intoxications, especially in acute poisonings.

- Papillomacular bundle loss and optic atrophy develop after a variable interval depending on the responsible toxin.

TOXIC OPTIC NEUROPATHIES



TOXIC OPTIC NEUROPATHIES

Alcohols: Methanol, ethylene glycol (antifreeze)

Antibiotics: Chloramphenicol, sulfonamides, linezolid

Antimalarials: Chloroquine, quinine

Antitubercular drugs: Isoniazid, ethambutol, streptomycin

Antiarrhythmic agents: Digitalis, amiodarone

Anticancer agents: Vincristine, methotrexate

Heavy metals: Lead, mercury, thallium

Others: Carbon monoxide, tobacco

Nutritional Optic Neuropathies

THE CLINICAL PRESENTATION AND BASIC PATHOPHYSIOLOGY ARE SIMILAR TO TON

Most often, they present as a non-specific retrobulbar optic neuropathy.

Currently, the treatment is limited to the intensive use of vitamins with variable results in individual cases, and to the implementation of preventive measures, when feasible.

Nutritional Optic Neuropathies

- Optic disc may be normal or slightly hyperemic in the early stages.
- In a small group of patients with hyperemic discs, small splinter hemorrhages on or off the disc.
- Several months to years later , papillomacular bundle dropout and temporal disc pallor, followed by optic atrophy.

Nutritional Optic Neuropathies

- Thiamine (vitamin B1)
- Cyanocobalamin (vitamin B12)
- Pyridoxine (vitamin B6)
- Niacin (vitamin B3)
- Riboflavin (vitamin B2)
- Folic acid

Nutritional Optic Neuropathies

TOBACCO ALCOHOL AMBYLOPIA (TAA)

- TAA is an old term for a constellation of elements that can lead to a mitochondrial optic neuropathy.

- The classic patient is a man with a history of heavy alcohol and tobacco consumption.

Nutritional Optic Neuropathies

TOBACCO ALCOHOL AMBYLOPIA (TAA)

- Combined nutritional mitochondrial impairment, from vitamin deficiencies (folate and B-12) classically seen in alcoholics, with tobacco- derived products, such as cyanide

- It has been suggested that the additive effect of the cyanide toxicity,and deficiencies of thiamine, riboflavin, pyridoxine, and b12 result in TAA

Toxic Optic Neuropathies: Other agents

- Hypovitaminosis A – night blindness (nyctalopia), keratomalacia.

- Hypervitaminosis A – yellow skin and conjunctiva, pseudotumor cerebri (papilledema), retinal hemorrhage.

TREATMENT

- Treatment Consideration should be given to co-management with a general physician or neurologist
- Dietary revision with formal nutritional advice, incorporating increased fruit and leafy green vegetable intake.
- Abstention from alcohol and tobacco is a priority
- Vitamins. A daily multivitamin preparation, plus thiamine (100 mg twice daily) and folate (1 mg daily). • Intramuscular hydroxocobalamin (vitamin B12) injections.
- Exposure to the identified agent should be discontinued immediately in cases due to medication or environmental toxicity

THANK YOU

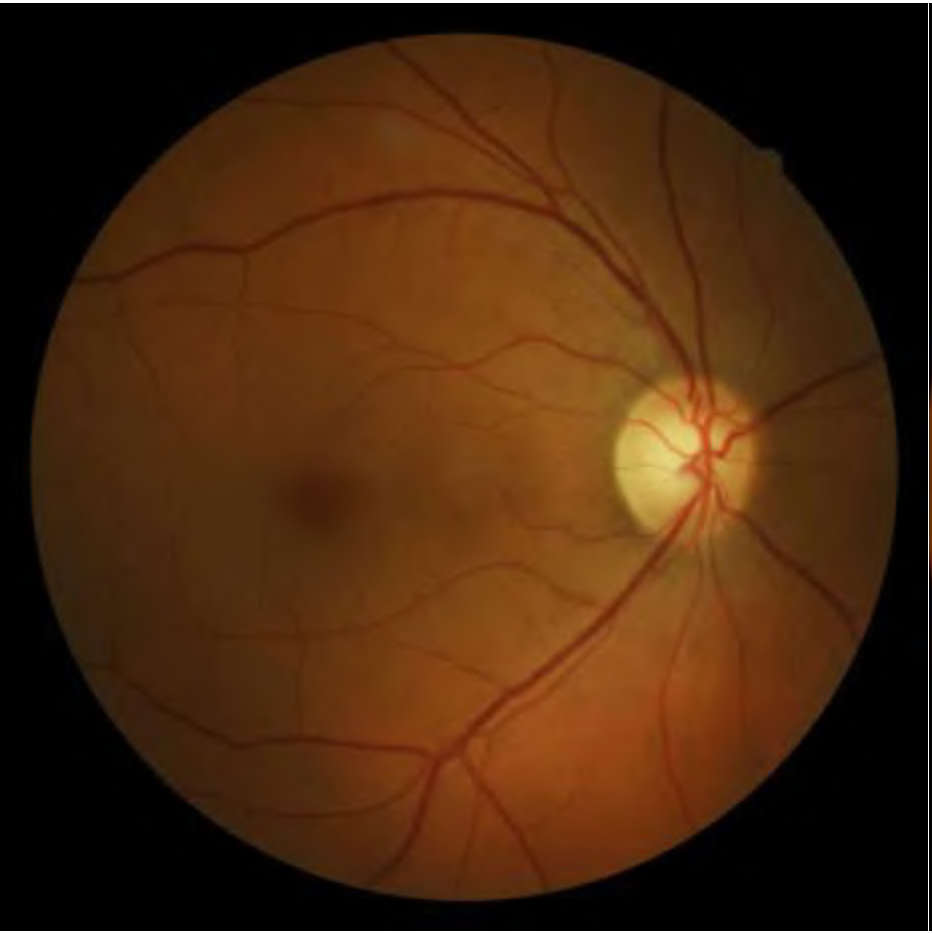
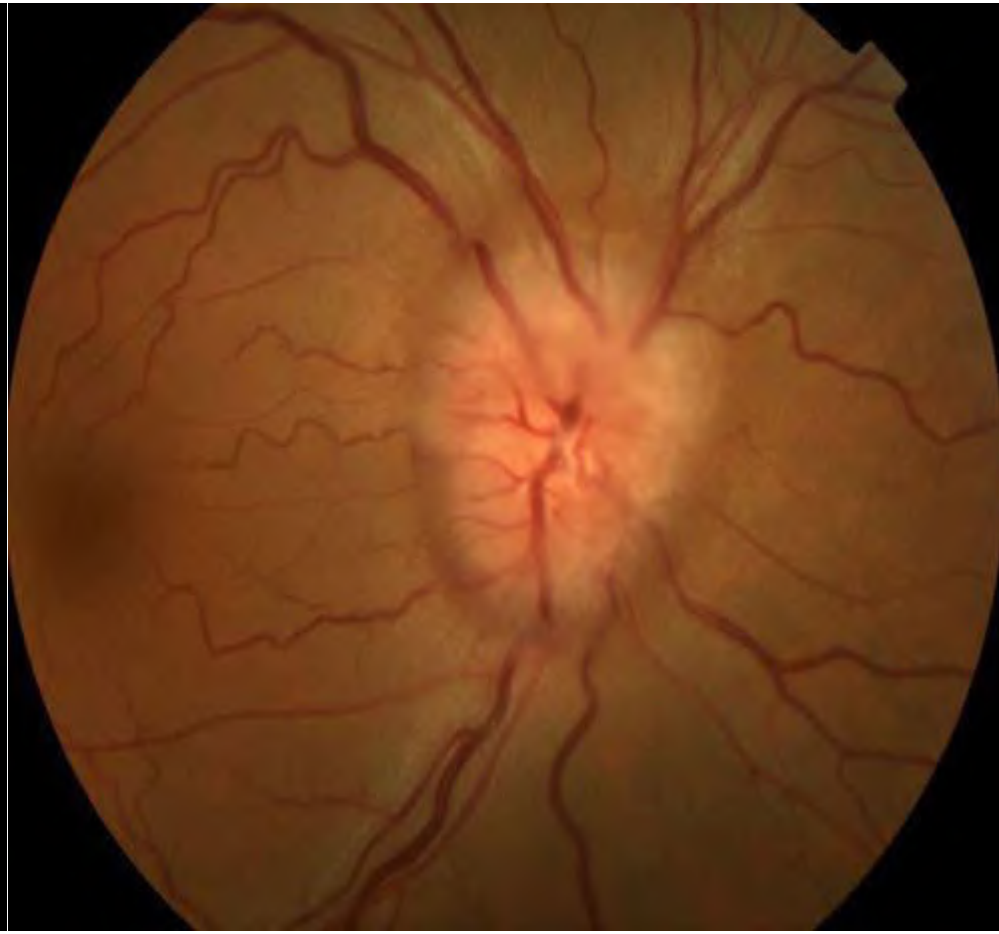
OPTIC DISC SWELLING/ PAPILLOEDEMA

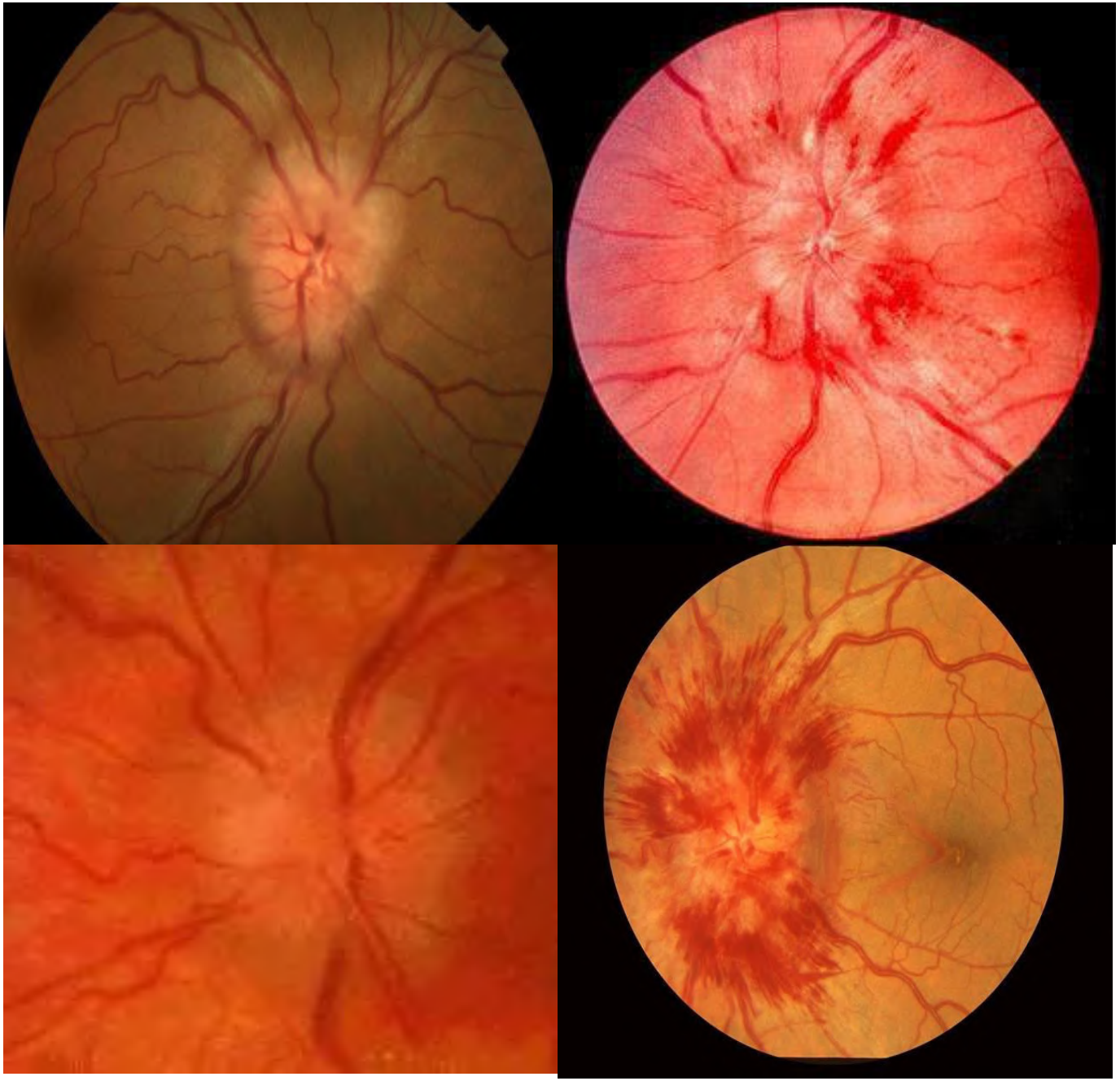
Dr Umer Khan

Associate Professor KGMC/HMC

- A 19- year- old woman has new- onset headaches and transient visual obscurations. She has severe acne and uses a topical medi cation prescribed by her dermatologist. She weighs 110 pounds and is 5 feet, 2 inches tall. Visual acuity is 20/20 OU with normal color vision. Static perimetry shows blind- spot enlargement bilaterally, and ductions are full. What ONH finding is expected to be seen on ophthalmoscopy?

1. mild hyperemia with telangiectatic vasculature
2. bilateral pallor
3. bilateral edema
4. normal architecture



- Passive hydrostatic non inflammatory swelling of optic nerve head secondary to raised intracranial pressure.

- Usually bilateral ; may be unilateral.

- Optic disc swelling in the absence of raised intracranial pressure is referred to as optic disc edema.

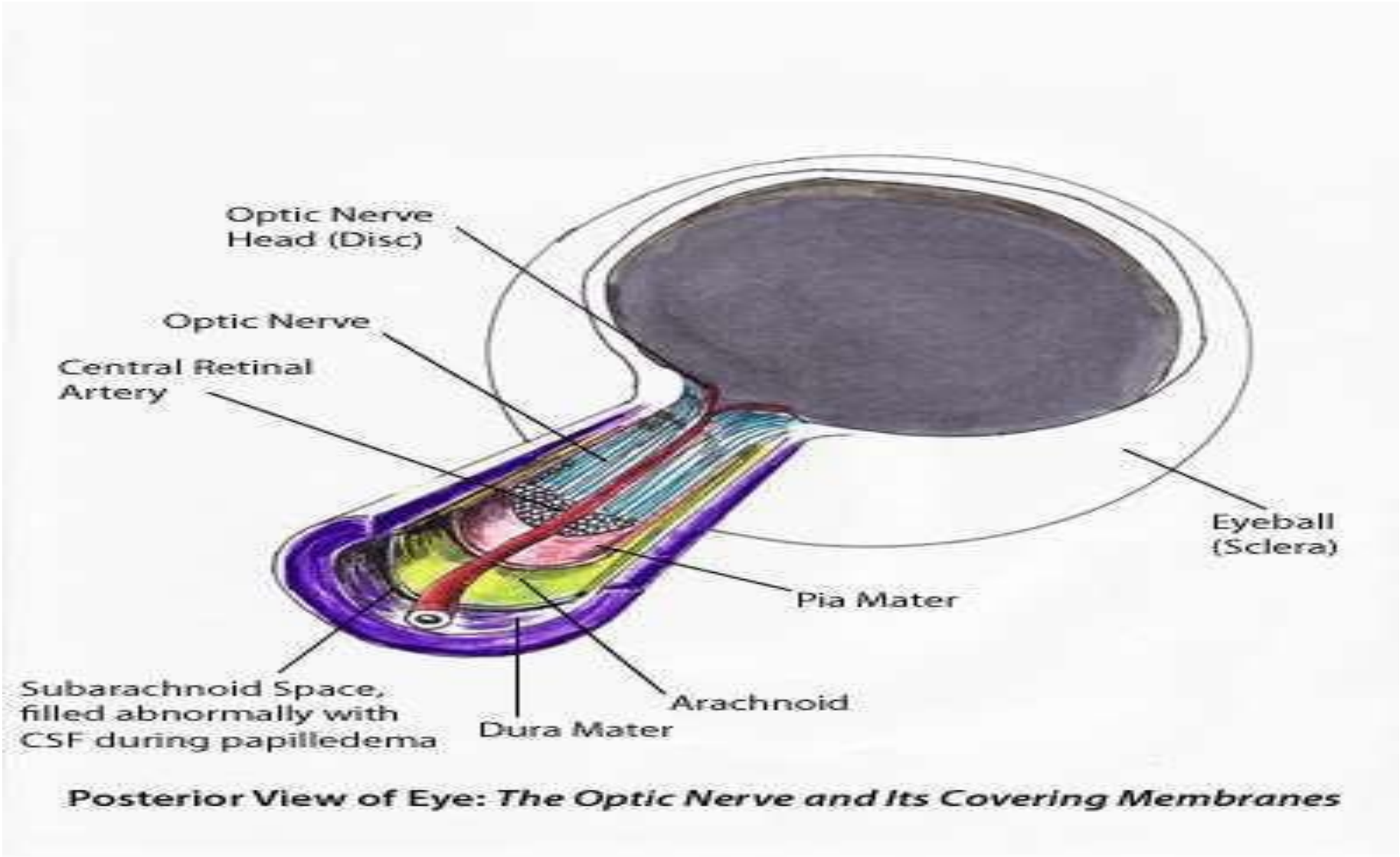
True disc swelling	Papilledema	↑ICP	Tumors, etc. (Table 16.9)
	Local disc swelling	Inflammatory	Optic neuritis Uveitis Scleritis
		Granulomatous	Tuberculosis Sarcoid
		Infiltrative	Leukemia Lymphoma
		Vascular	AION CRVO Diabetic papillitis
		Tumors	Of optic nerve (meningioma, glioma) Of orbit
		Hereditary	LHON

No true disc swelling	Pseudopapilledema	Structural	Disc drusen Tilted discs Hypermetropic discs Myopic discs Myelinated peripapillary nerve fibers

Table 16.9 Causes of raised intracranial pressure

Mass effect	Tumor Hemorrhage Trauma (hematoma/edema)
Increased CSF production	Choroid plexus tumor
Reduced CSF drainage	Stenosis of formen/aqueduct (congenital or secondary to tumor, cyst, infection, etc.) Damage to arachnoid granulations (meningitis, subarachnoid hemorrhage) Idiopathic intracranial hypertension
Other	Malignant hypertension

Drugs	Tetracycline
	Corticosteroids
	OCP
	Vitamin A derivatives
	Nalidixic acid
Endocrine	Hypoparathyroidism
	Adrenal adenomas
Habitus	Obesity
	Obstructive sleep apnea syndrome
Hematological	Cerebral venous thrombosis



Posterior View of Eye: *The Optic Nerve and Its Covering Membranes*

Symptoms(Ocular)

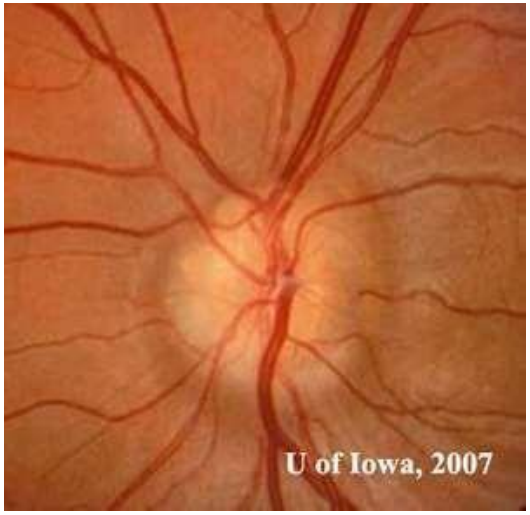
- Visual Acuity
- Transient obscuration of vision.
- Central vision affected late.
- Horizontal Diplopia.

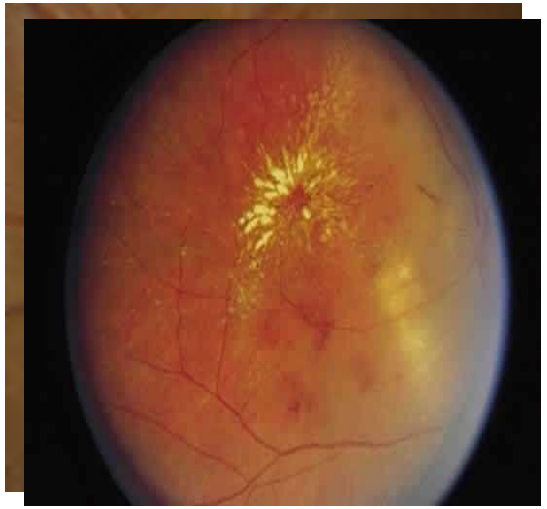
Symptoms(General)

- Headache more in the morning, intensifies with head movement, coughing or straining.
- Projectile vomiting.
- Loss of consciousness/ generalized motor rigidity.

Signs(Mechanical)

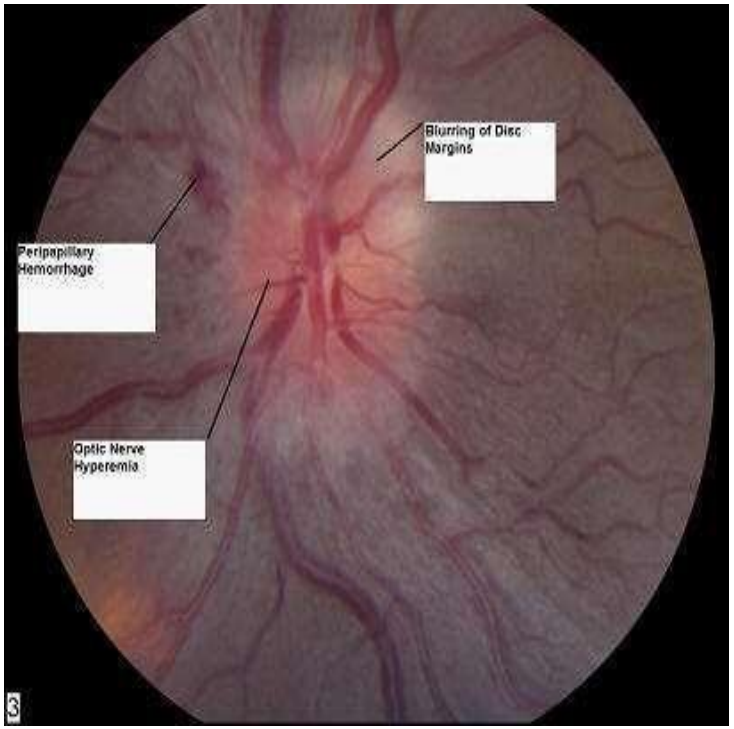
- Elevation of the optic disc.
- Blurring of the optic disc margin.
- Filling in of the physiological cup.
- Edema of the peripapillary nerve fiber layer.
- Retinal or choroidal folds(Paton's lines)
- Macular fan.





Signs(Vascular)

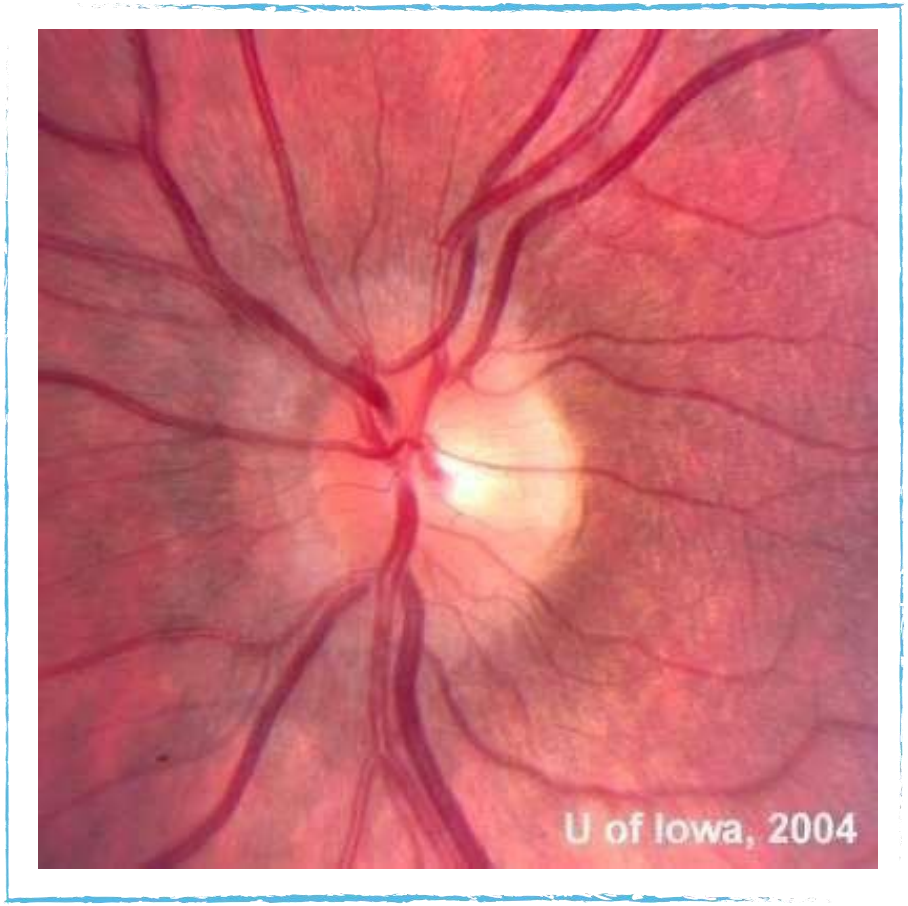
- Hyperemia of the optic disc.
- Vascular congestion.
- Peripapillary haemorrhage.
- Exudates in the disc or peripapillary area.
- Nerve fiber layer infarcts.



Grading of Papilledema

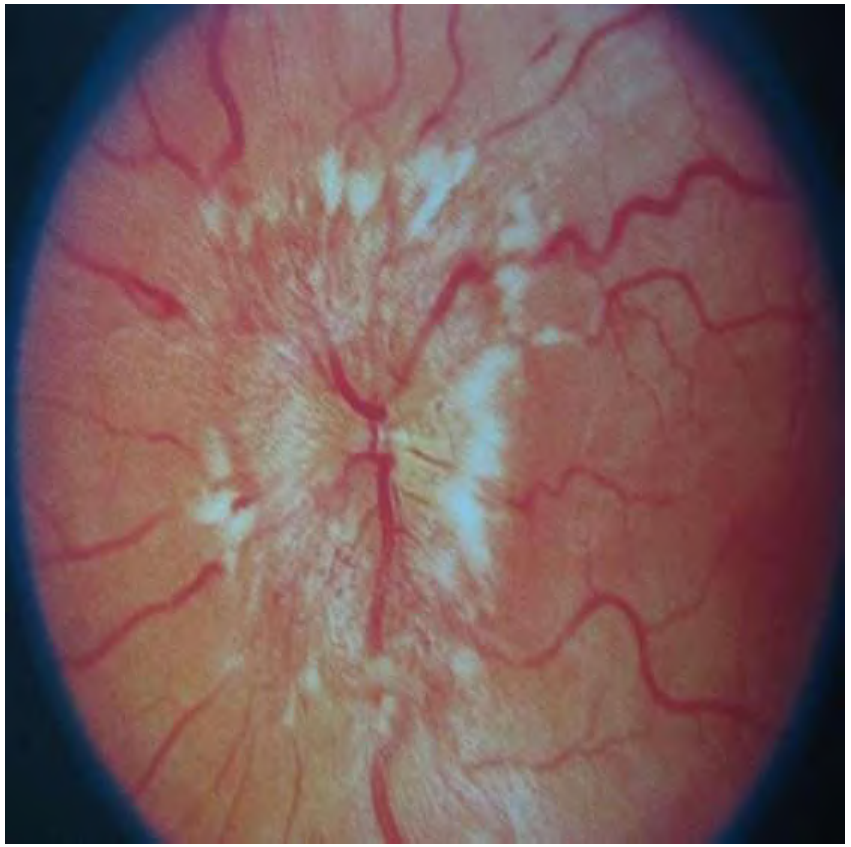
Early Papilledema

- Disc elevation.
- Venous distention and tortuosity.
- Obscuration of the normal disc margin and overlying retinal vessels.
- Absence of spontaneous venous pulsations



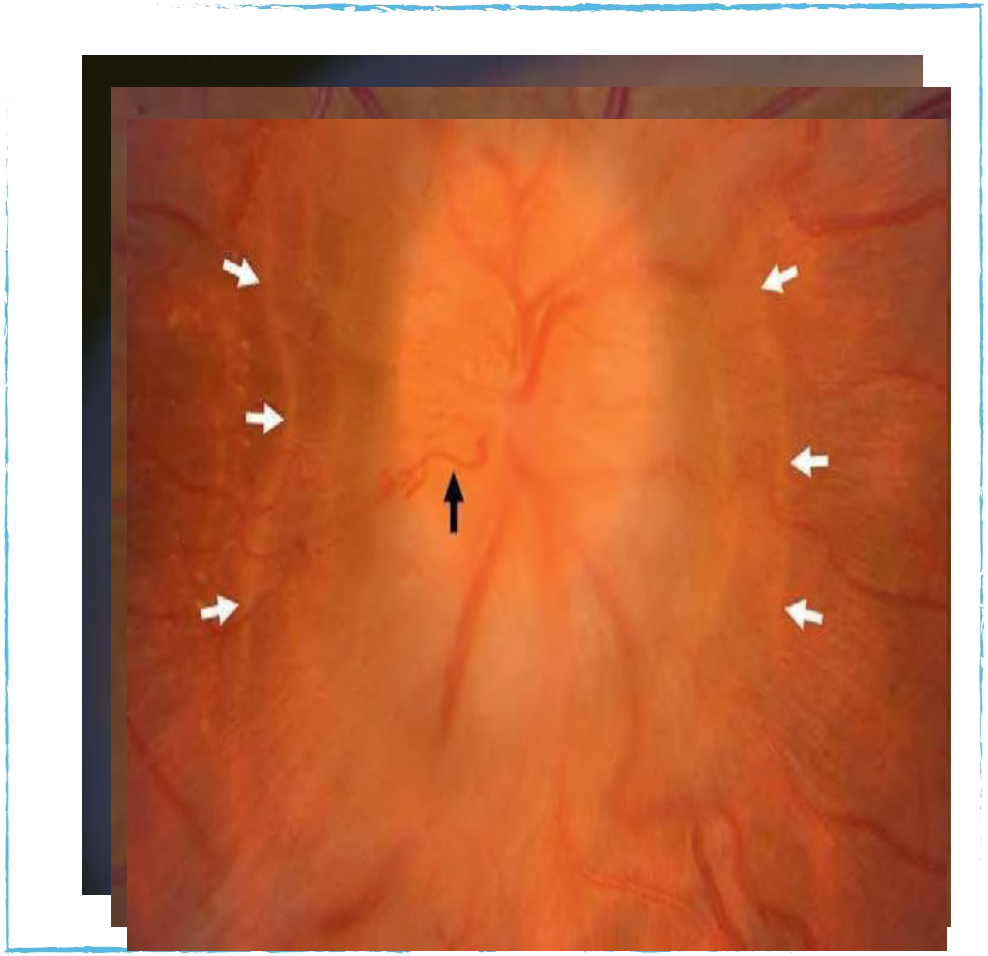
Established Papilledema

- Marked elevation of nerve head with blurring of margins.
- Engorged tortous venules.
- Peripapillary splinter hemorrhages.
- Cotton wool spots.
- Hard exudates over the disc and macular area.



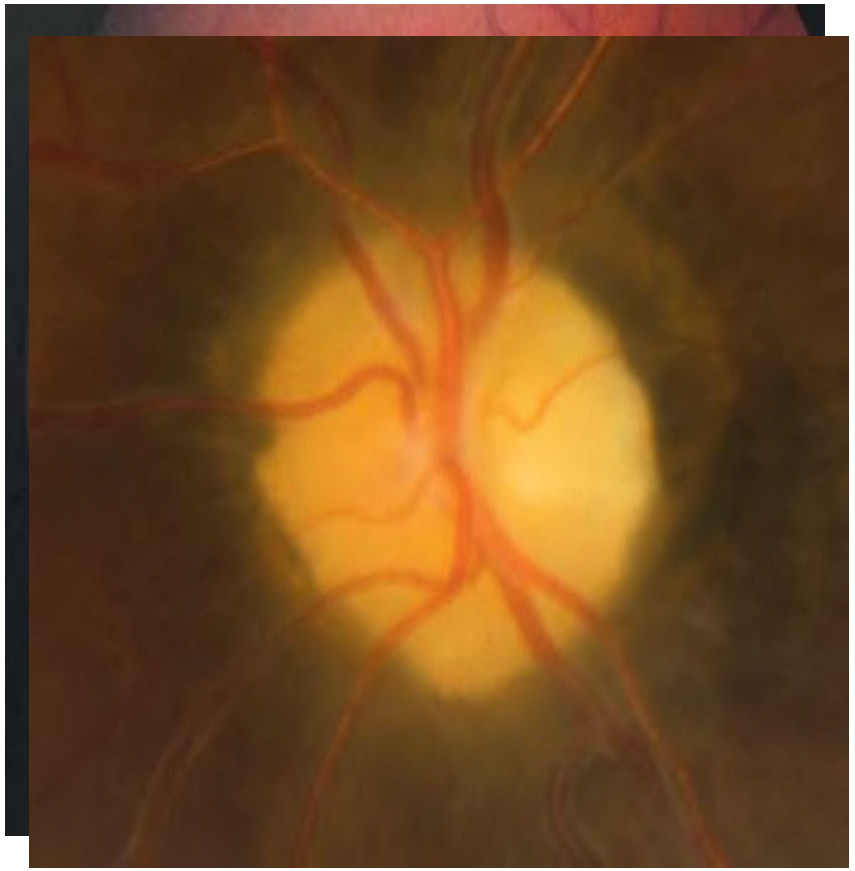
Chronic Papilledema

- disc hyperemia decreases and disc progressively appears pale in color.
- Opticociliary shunts and drusen like deposits may be present on the disc.
- High water mark.



Atropic Papilledema

- Onset of optic disc pallor (secondary optic atrophy) .
- Decrease in disc haemorrhage.
- Narrowing of blood vessels
- Optic disc appears dirty white and blurred



Investigations

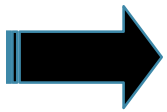
- History and physical examination including blood pressure measurement.
- Ophthalmic examination - In addition to fundus examination, assessment of
- visual acuity, pupillary examination, ocular motility & alignment, and visual fields.
- MRI with or without contrast is the best investigation of choice.

CT Scan/ MRI

- To rule out
  - Intracranial lesions.
  - Obstructive hydrocephalus

- Can detect
  - Subarachnoid, epidural & subdural hemorrhages.
  - Acute infarctions.
  - Cerebral edema.

Lumbar puncture



- Therapeutic procedure
  - Pseudotumor cerebri

- CSF for microbial and infectious studies.

- Diagnostic
  - Recording opening pressure.

Treatment

- Treat the cause
  - Craniotomy to remove tumor.
  - Stop toxic drugs
  - Correct anemia , HTN
  - For BIH, weight reduction, acetazolamide, surgery (Shunts)

Thank You

LEUKOCOREA AND RETINOBLASTOMA

Dr Irfan Ullah Khattak

- Leukocoria → “**white pupil**”
- Greek → “leukos” (white) and “kore” (pupil)
- It refers to the reflection of white light seen upon direct illumination of the fundus through the pupil, in contrast to the usual red glow

Etiology

- Tumors
  - Retinoblastoma
- Congenital malformations
  - Persistent Fetal Vasculature
  - Chorioretinal or optic nerve coloboma
  - Retinal dysplasia
- Media opacities
  - Cataract
  - Corneal opacity
  - Organizing vitreous hemorrhage

- Vascular diseases
  - Retinopathy of prematurity
  - Coats disease
  - Familial exudative vitreoretinopathy
- Inflammatory diseases
  - Ocular Toxocariasis
  - Congenital Toxoplasmosis

Retinoblastoma

- malignant (cancer) cells form in the tissues of the retina
- Heritable
- Non heritable

Clinical Features

- Leukocorea
- Strabismus
- Decreased Vision
- Incidental finding
- Redness
- Orbital Cellulitis

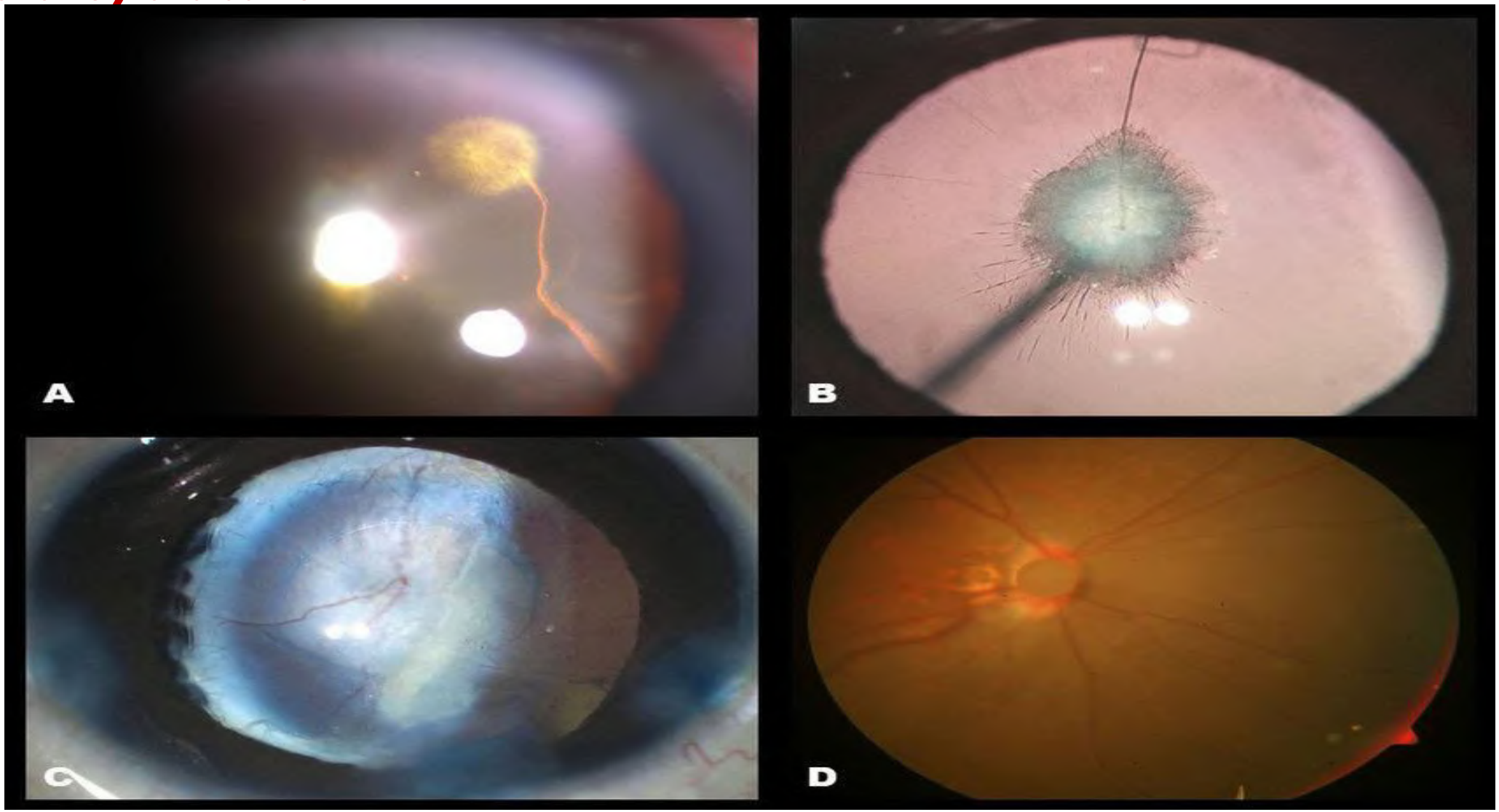
Investigations

- Ophthalmoscopy
- B Scan
- MRI
- CT

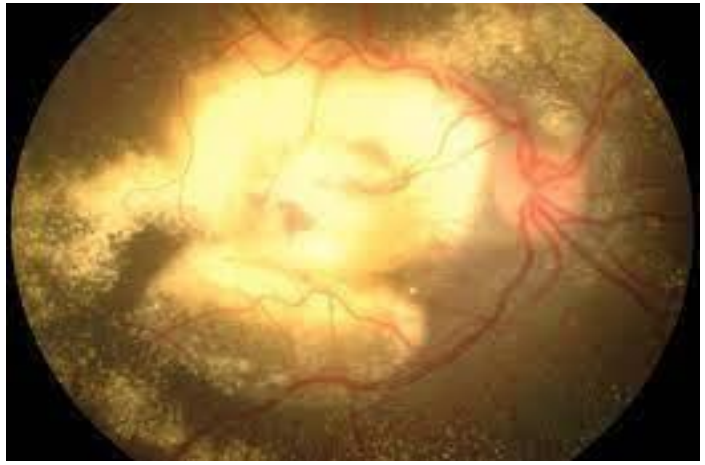
Treatment

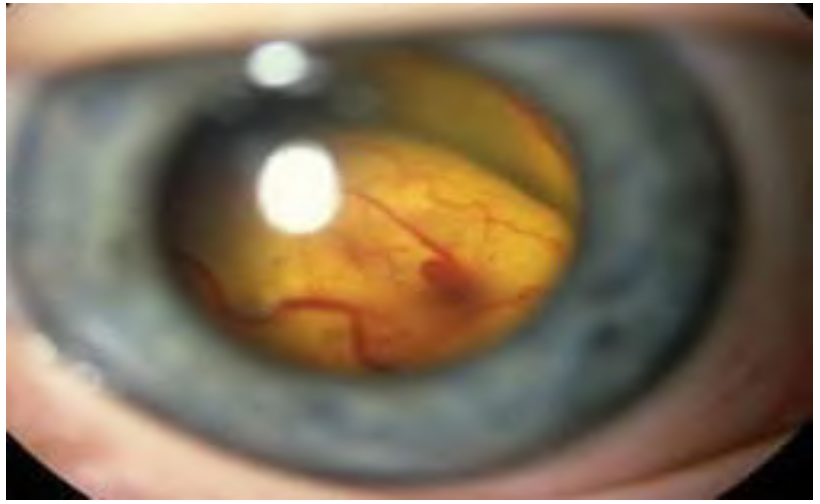
1. Chemotherapy
2. Laser photocoagulation
3. Thermotherapy
4. Brachytherapy
5. Enucleation
6. Exentration

PFV/PHPV



COATS DISEASE





TOXOCARIASIS

ROP

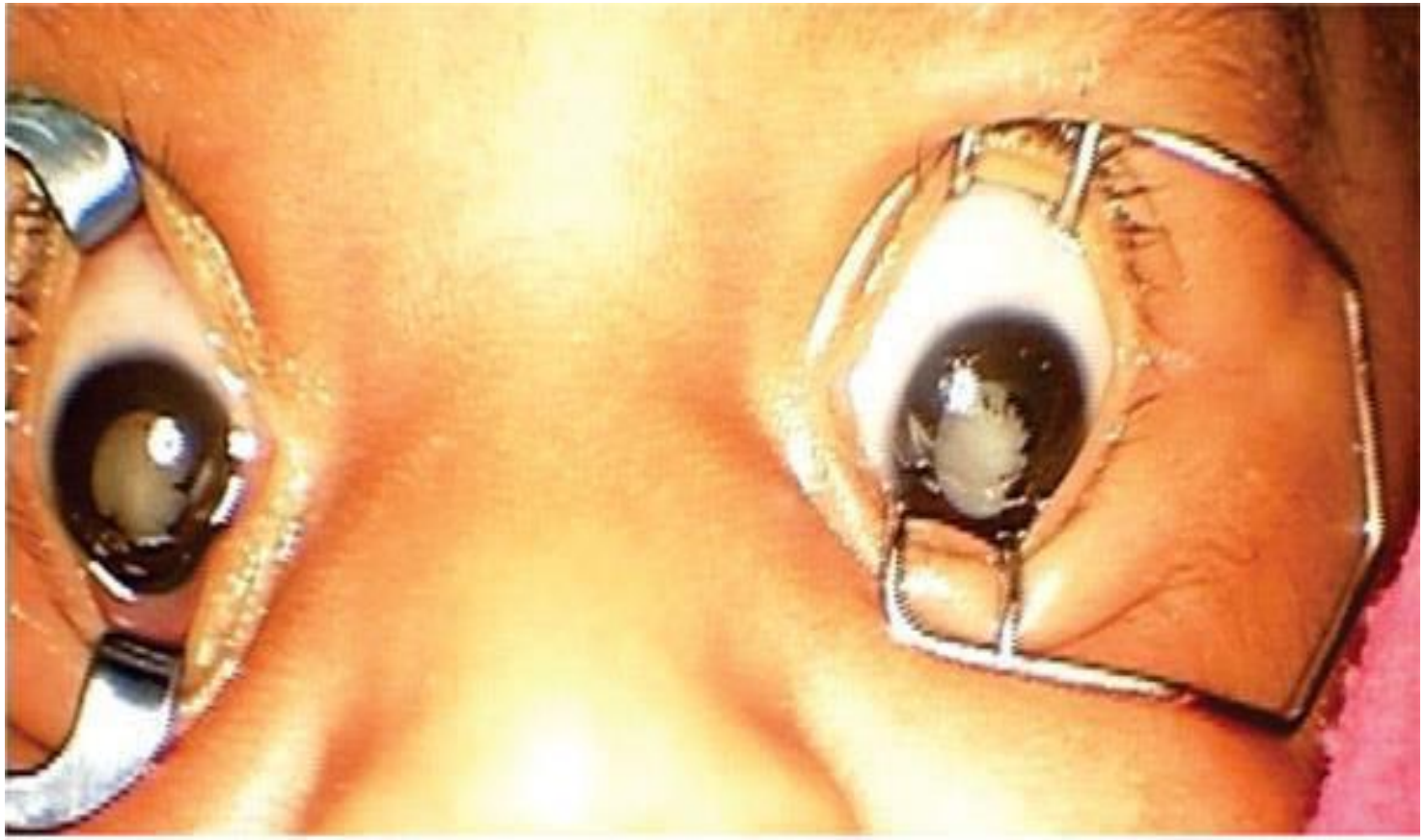
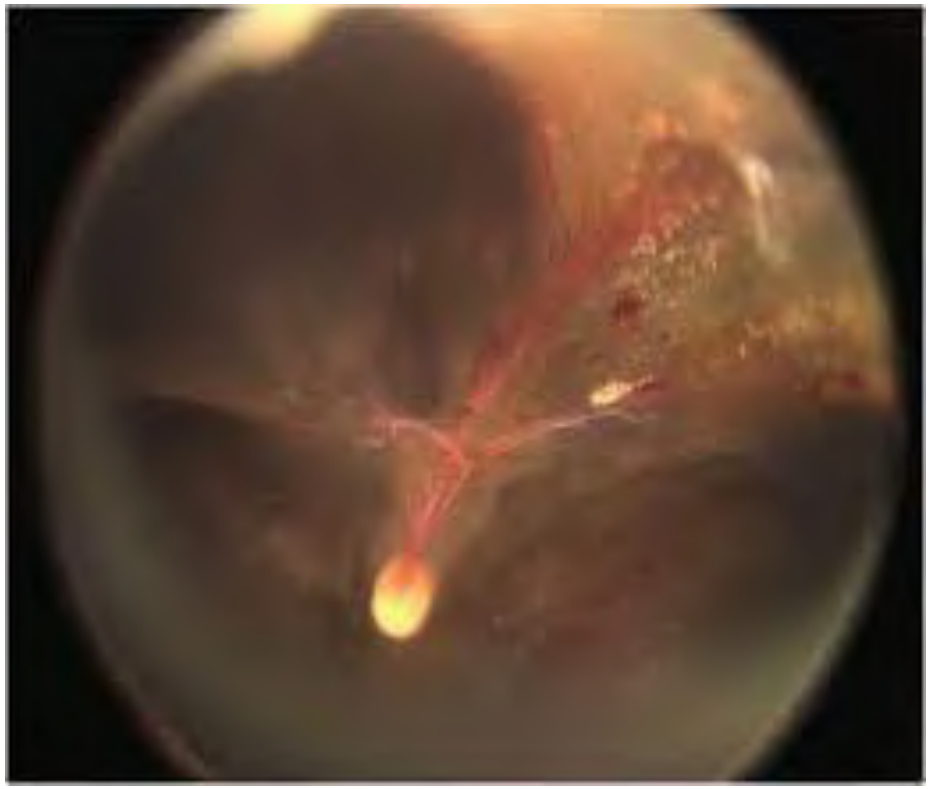


Figure 5: Showing total retinal detachment in both eyes (ROP Stage 5).

FEVR




thank you

RETINOBLASTOMA

BY

Associate prof DR afzal Qadir

Head of department of pediatric ophthalmology

Objectives

- History
- Introduction
- Inheritance
- Clinical feature
- Pattern of growth
- Clinical Manifestations
- Differential diagnose
- Treatments

History

- It was first mention by petras in 1597.
- Then venroff describe the origin from undifferentiated retinal cell named Retinoblastoma in 1900.
- American ophthalmology society has first adopted the word retinoblastoma in 1926.

Introduction

- Retinoblastoma is the primary malignant neoplasm of the retina that arises from immature cell of retina .
- Most common primary intraocular malignancy of child hood.
- Might be unifocal \ multifocal
- May be unilateral or bilateral

Inheritance

- Unilateral has only one effected which range from 55 to 65%
- Bilateral has effected both eyes which range from 25% to 35%
- In bilateral cases multifocal tumors in both eyes are the rule .

Clinical feature

- Leucocoria
- Strabismus
- Red painfull eye
- Poor vision
- Orbital cellulitis
- Hyphema
- Unilateral or bilateral mydriasis
- proptosis

Leukocoria

Strabismus in RB

Pattern Of Growth

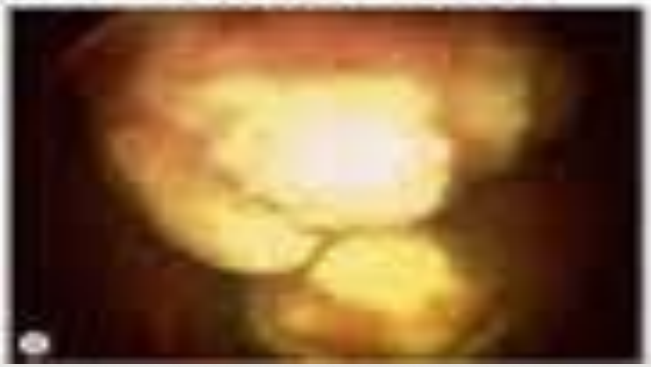
Growth pattern

ENDOPHYTIC

- Into vitreous cavity
- No overlying retinal vessels
- Simulate endophthalmitis
- Present as pseudohypopyon, nodules at pupillary border.

EXOPHYTIC

- Grows outwards into subretinal space
- Retinal vessels seen over it
- Retinal detachment
- Simulate coats disease



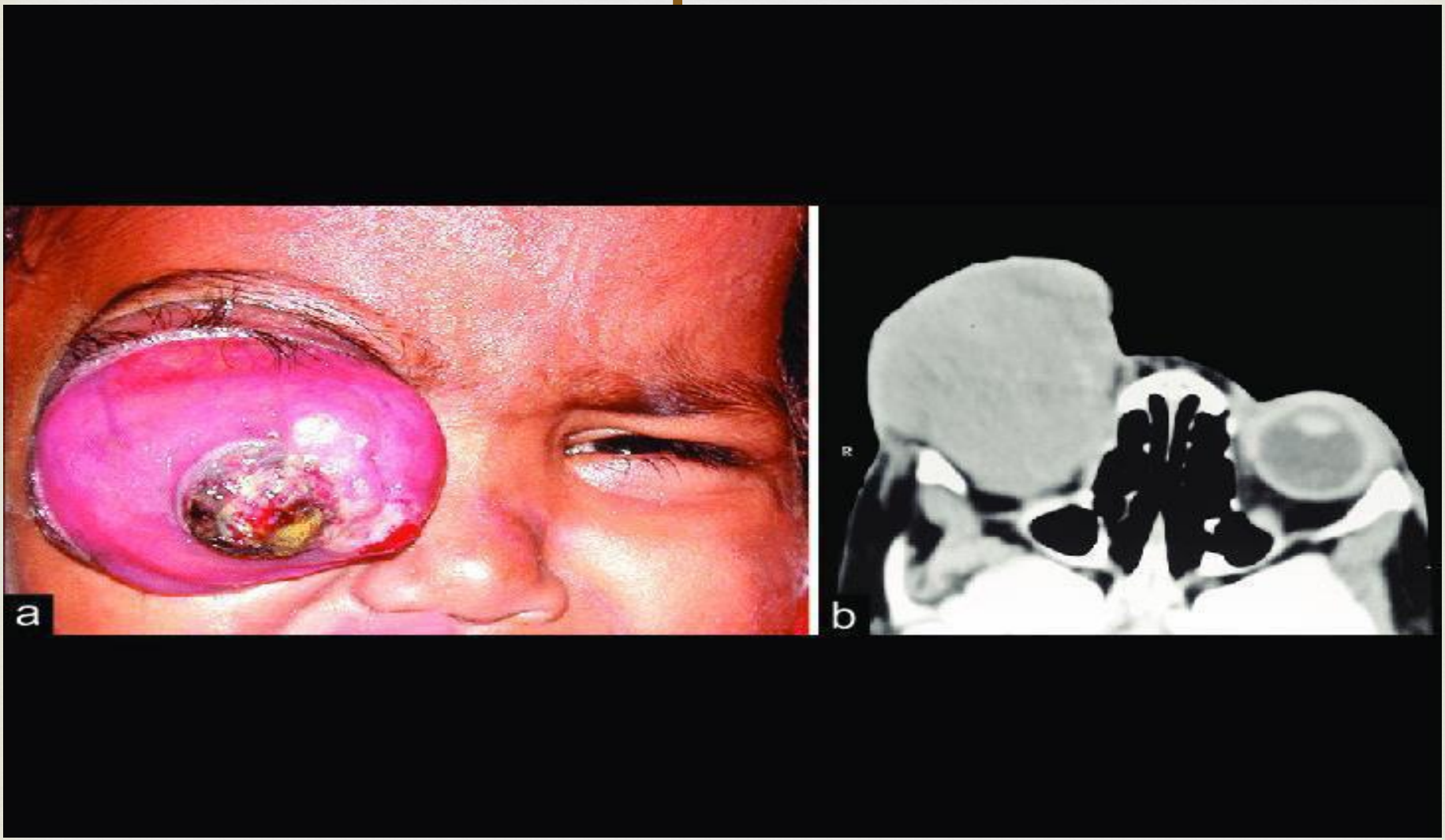
Grades of RB

- Grades A: Small tumors (no more than 3 millimeters [mm] across) that are only in the retina and are not near important structures such as the optic disc (where the optic nerve enters the retina) or the foveola (the center of vision).
- Grade B: All other tumors (either larger than 3 mm or close to the optic disc or foveola) that are still only in the retina.

CONT

- Grade C: Well-defined tumors with small amounts of spread under the retina (subretinal seeding) or into the jelly-like material that fills the eye (vitreous seeding).
- Grade D:Large or poorly defined tumors with widespread vitreous or subretinal seeding. The retina may have become detached from the back of the eye.

Proptosis



Clinical Manifestation

- Clinical presentation depend upon the stage of disease
- Translucent white fluffy retinal mass
- Strabismus if tumor involves macula which reduceed the VA

Clinical Manifestations

- **Endophytic:** grows in to vitreous cavity . Mass fullfil the vetreous cavity and seeds.
- **Exophytic:** tumor toward retina and detached the Retina (RD)
- **Diffuse Infiltrating Tumor:** diffusely involve retina Placoid thickness of retina.

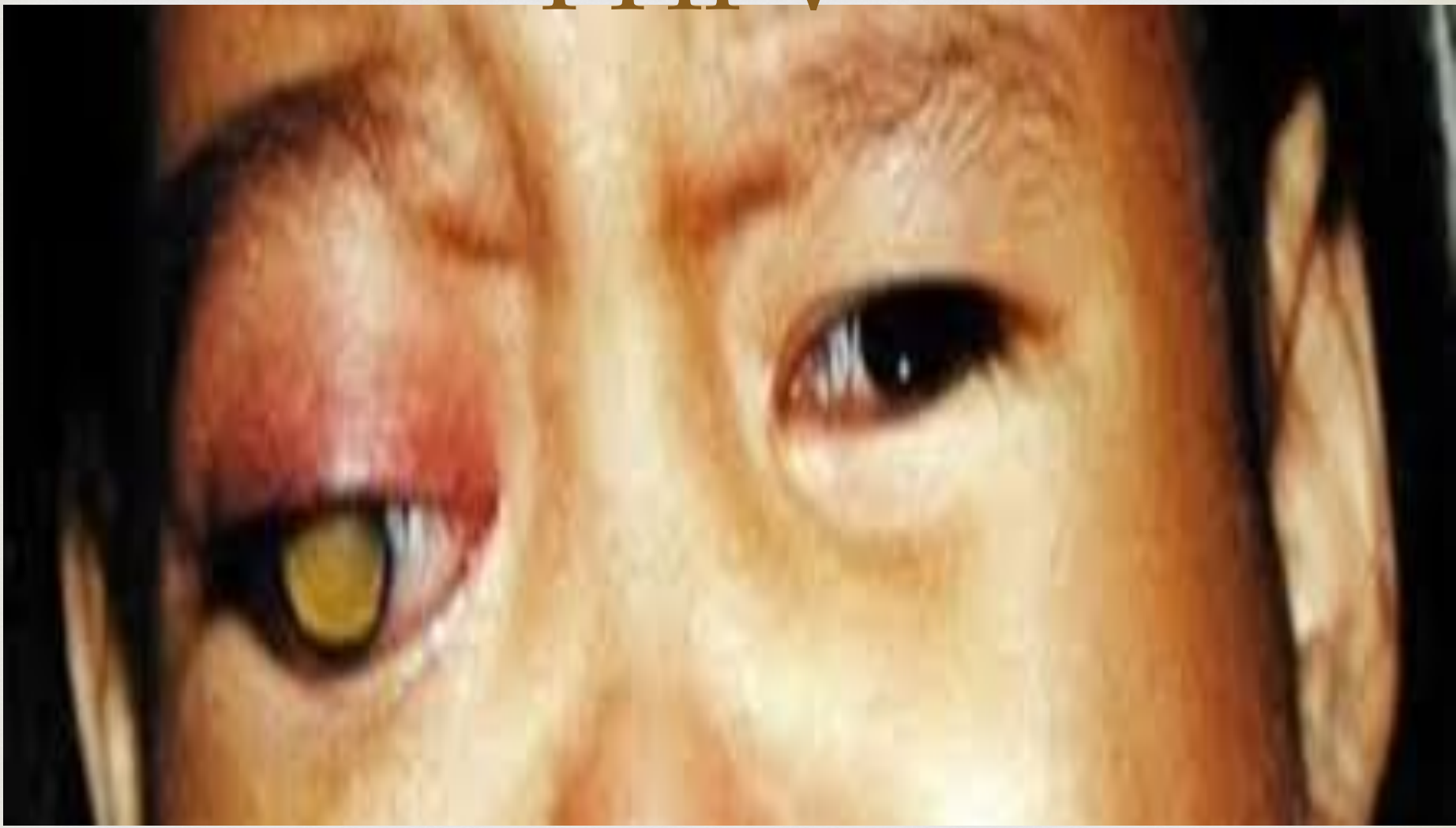
Retinal Detachment

Differential diagnose

- Congenital cataract
- PHPV (persistent hyperplastic primary vitreous)
- Coloboma (uveal tract coloboma)
- Coats disease
- ROP(Retina of prematurity)

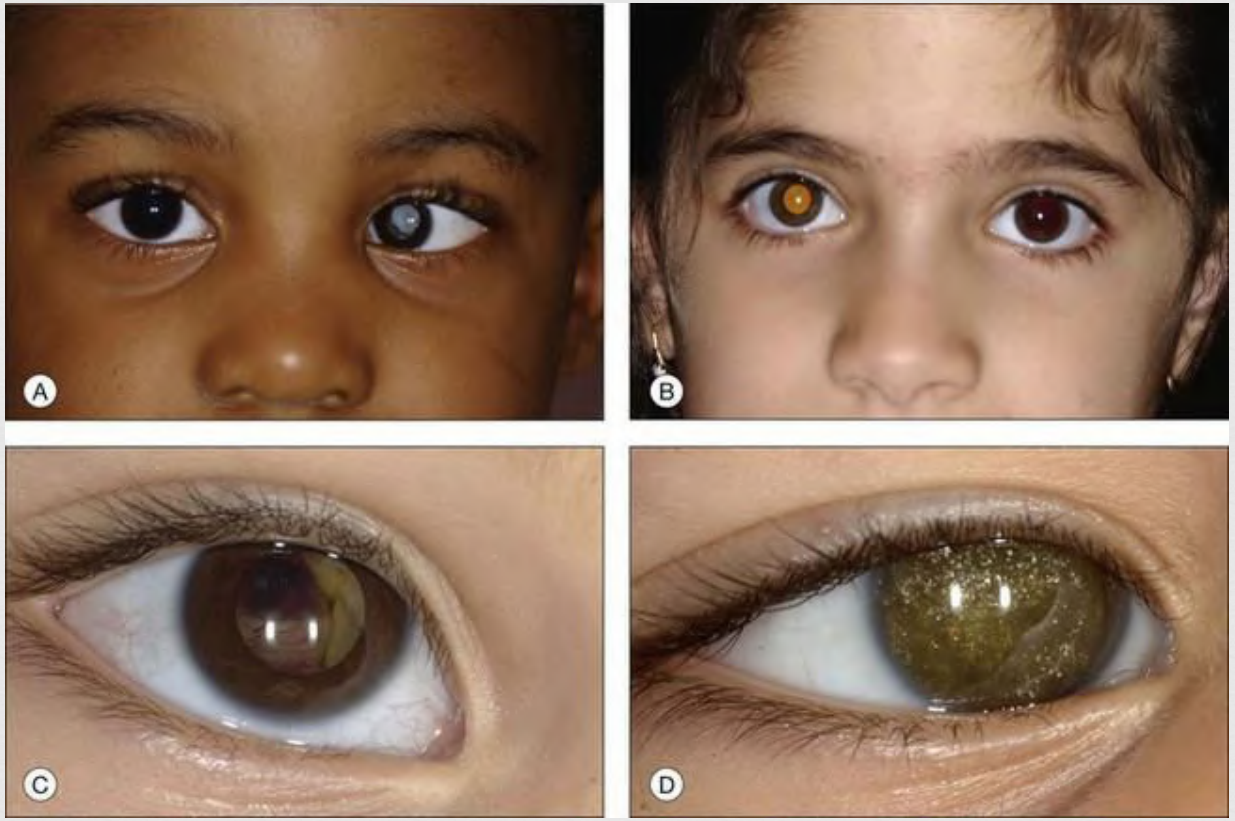
Congenital Cataract

PHPV

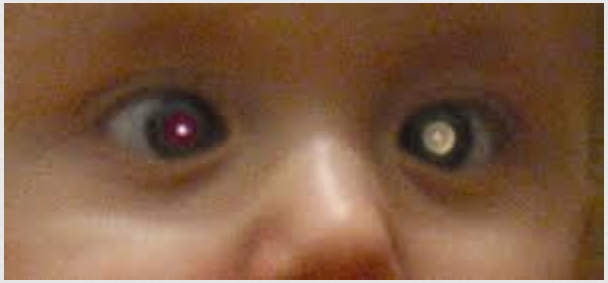


Coloboma

Coats Disease



ROP



Treatment

- External beam radiation therapy
- Cryotherapy
- Thermotherapy
- Chemotherapy
- Intravitreal chemotherapy
- Surgery such as

I. Enucleation
II. Exantration

External beam therapy radiation

- External beam radiation therapy comes from a machine that aims radiation at your cancer. It is a local treatment, which means it treats a specific part of your body.
- As our eyes treatment and other parts.
- Types
  - Photon
  - Protons
  - Electrons

Photons

- Most radiation therapy machines use photon beams. Photons are also used in x-rays, but x-rays use lower doses. Photon beams can reach tumors deep in the body. As they travel through the body, photon beams scatter little bits of radiation along their path. These beams do not stop once they reach the tumor but go into normal tissue past it.

Protons

- Protons are particles with a positive charge. Like photon beams, proton beams can also reach tumors deep in the body. However, proton beams do not scatter radiation on their path through the body and they stop once they reach the tumor. Doctors think that proton beams might reduce the amount of normal tissue that is exposed to radiation. Clinical trials are underway to compare radiation therapy using proton beams with that using photons beams. Some cancer centers are using proton beams in radiation therapy, but the high cost and size of the machines are limiting their use.

Electrons

- Electrons are particles with a negative charge. Electron beams cannot travel very far through body tissues. Therefore, their use is limited to tumors on the skin or near the surface of the body.

Side effects of (EBRT)

- Fatigue
- Hairs loss
- Memory problems
- Nausea and vomiting
- Skin rashes
- Headache
- Blurred vision (if this is to other parts of the body)

Cryotherapy

- Cryotherapy is the use of extreme cold to freeze and remove abnormal tissue. Doctors use it to treat many skin conditions (including warts and skin tags) and some cancers, including prostate, cervical and liver cancer. This treatment is also called cryoablation.

Side effects CT

- Bleeding
- Bone marrow effect
- Nerve damage
- Swelling

Thermotherapy

- Thermotherapy (heat therapy) is a procedure that involves the application of superficial heat to injured or damaged body parts. The heat alters tissue temperatures in targeted regions. These temperature increases make the tissues more extensible. Patients report experiencing pain relief and faster healing.
- Side effect.
  - Skin rash or skin burn

Chemotherapy

- Chemotherapy is a cancer treatment where medicine is used to kill cancer cells. There are many different types of chemotherapy medicine, but they all work in a similar way. They stop cancer cells reproducing, which prevents them from growing and spreading in the body.

Chemotherapy medicine

- Abraxane (chemical name: albumin-bound or nab-paclitaxel)
- Adriamycin (chemical name: doxorubicin)
- carboplatin (brand name: Paraplatin)
- Cytoxan (chemical name: cyclophosphamide)
- daunorubicin (brand names: Cerubidine, DaunoXome)
- Doxil (chemical name: doxorubicin)

Side effects of Chemotherapy

- Fatigue
- Nausea and vomiting
- Infection (weakness of autoimmune system)
- Anemia
- Diarrhea

Intra vitreal chemotherapy

- Treatment in which anticancer drugs are injected directly into the vitreous humor (gel-like fluid inside the eye). Intravitreal chemotherapy is used to treat retinoblastoma that has spread to the vitreous humor and has come back or has not gotten better after other treatment.

Intra arterial chemotherapy

- Intra-arterial (IA) chemotherapy for the treatment of intraocular retinoblastoma, also referred to as superselective intra-arterial chemotherapy and chemosurgery.
- **NOTE**: It is not given to the patient of RB whether his or her extra ocular muscle involves.

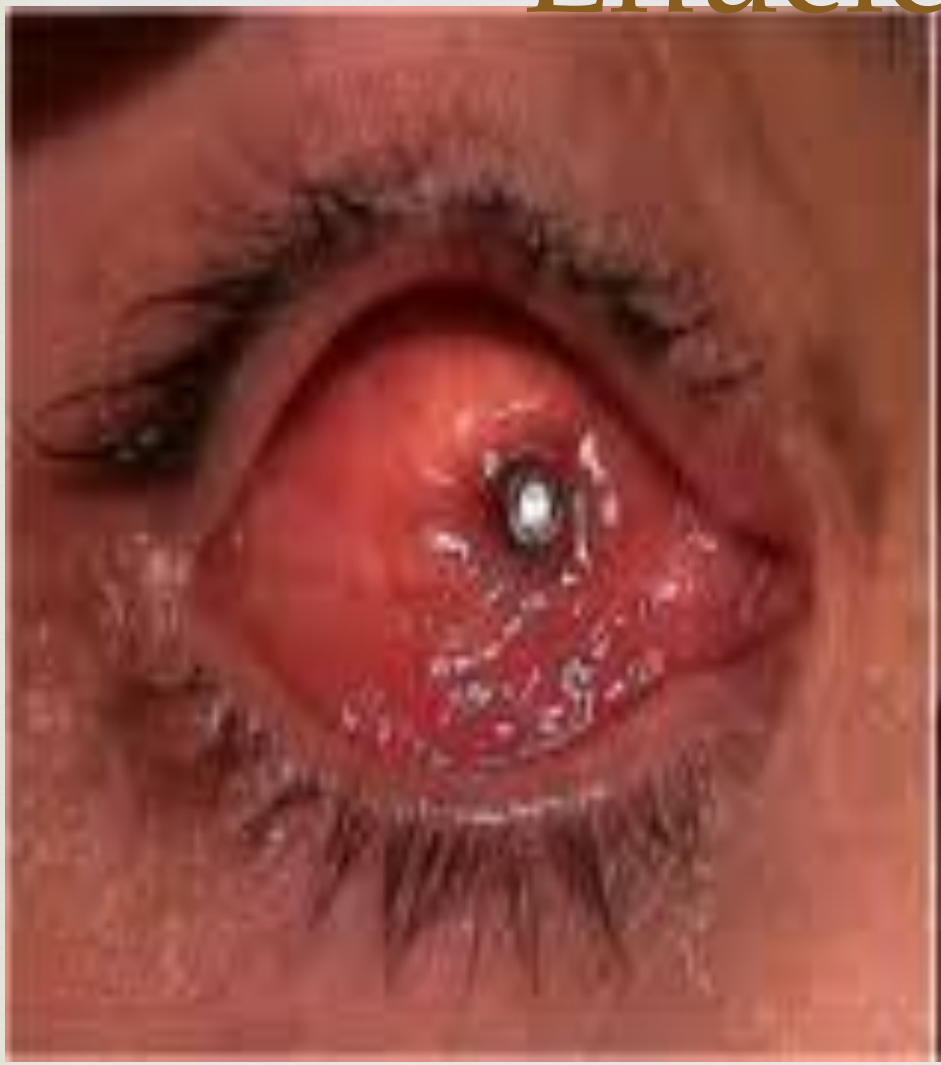
Side effects of intra arterial chemotherapy

- Swelling of the optic nerve
- RD
- Bleeding
- Irreversible loss vision

Surgical Management

- **Enucleation**:Enucleation is the removal of the eye from the orbit and involves the separation of all tissue connections between the globe and the orbit. This is one of the oldest operations within the field of ophthalmology and is one of the most challenging therapeutic decisions to make.

Enucleation



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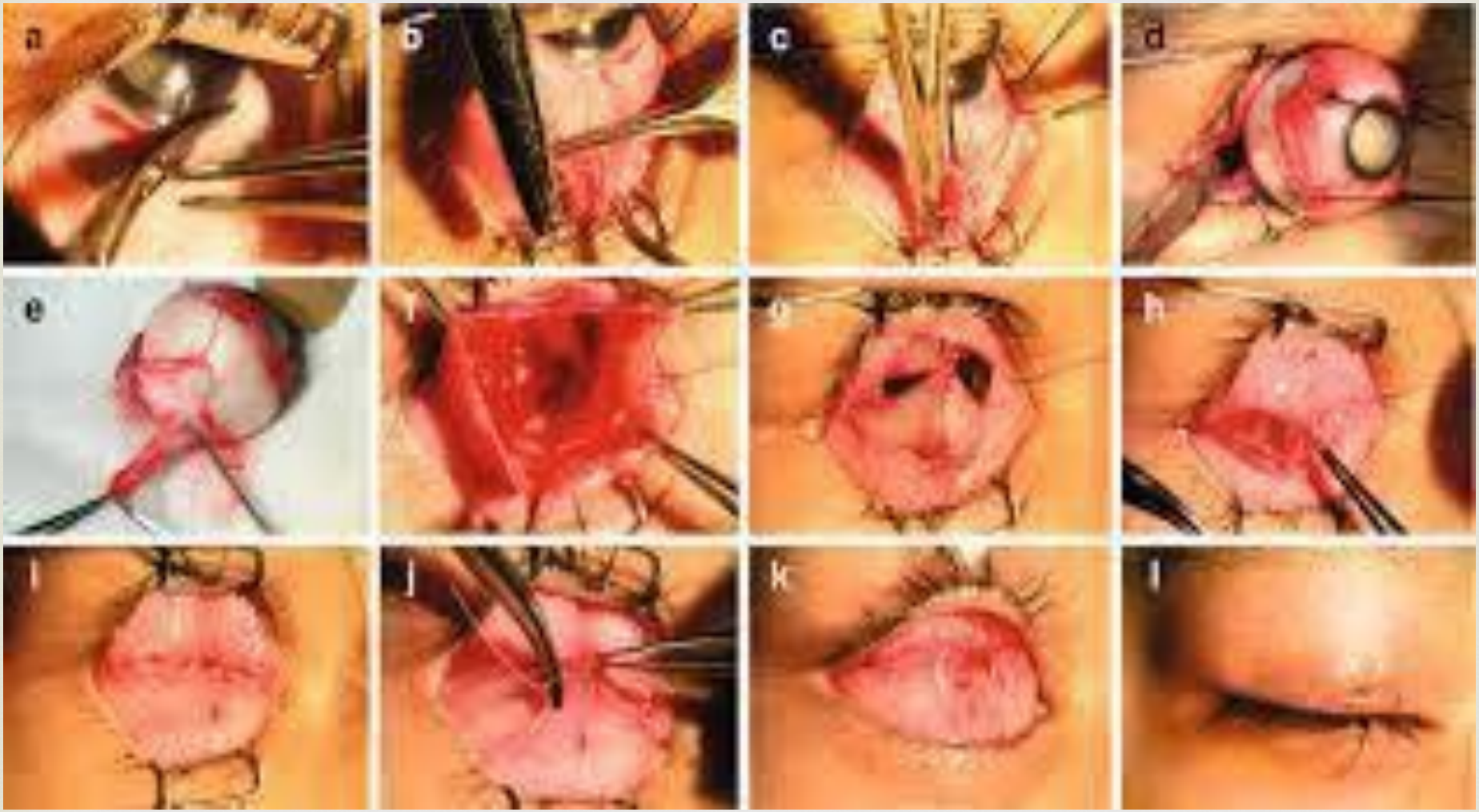
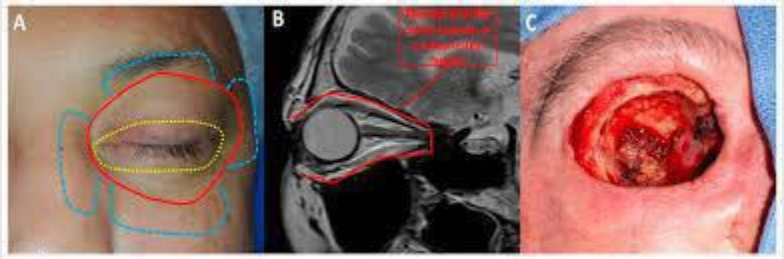


Enucleation eye

Exantration

- **Exantraion :** Orbital exenteration is defined as removal of the entire contents of the bony orbit, including the globe, extraocular muscles and periorbital fat, and many times includes the eyelids, in contrast to enucleation, which involves only removal of the globe.

Exantration of eye



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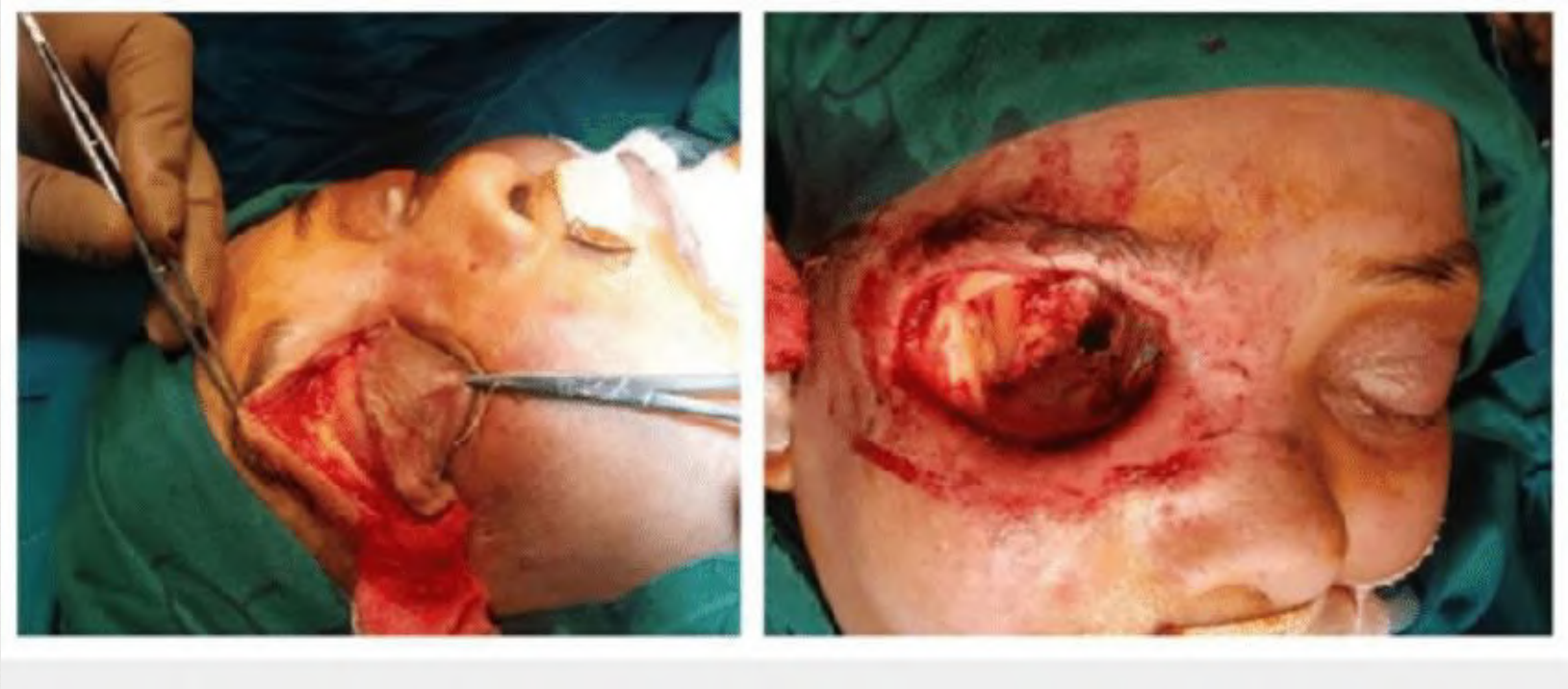


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Amblyopia / Lazy Eye

Dr AFZAL QADIR

Associate Professor Pediatric Ophthalmologist

KGMC, HMC

Greek word Amblys= “dull” ops= “eye”

- **Amblyopia** is the unilateral, or rarely bilateral, decrease in best corrected visual acuity (BCVA) caused by form vision deprivation and/or abnormal binocular interaction, for which there is no identifiable pathology of the eye or visual pathway. (From birth to 8 years of age).

- Suppression temporary active cortical inhibition of the image of an object formed at the retina of the squinting eye. (with both eyes open)

Complex visual processing disorder

- Accommodation accuracy and facility,
- Fixation stability,
- Pursuit and saccadic accuracy,
- Location in space,
- Reduce contrast sensitivity,
- Dark adaptation abnormalities,
- Visual field abnormalities,
- Occasionally relative afferent pupillary defect (RAPD).

Cont;

Normal Binocular Vision

- Our eyes work together as visual cortex integrates the images form each eye to produce a single binocular stereoscopic image.
- The cortical merging of two images is called binocular fusion

Two types

1. **Motor fusion;** Keep the eyes aligned together on a visual target
2. **Sensory fusion;** Brain merging images from each eye into single stereoscopic image

Primary brain processing centers are the lateral geniculate nucleus (LGN) & striate cortex.

Brain centers process information from more than 1 million retinal axon from each eye.

Requirement of normal visual development

- Clear retinal image
- Equal image clarity
- Proper eye alignment
- Continues clear cortical active image integration

Visual Development & Amblyopia

- At birth visual system is immature & (VA) approximately 20/400. VA improves and stereopsis develops during first months of life.
- Normally developing infants is the ability to accurately fixate **ON & follow small objects by 6 months of age .**
- **Healthy infants may occasionally show delay visual maturation.**
- **Persistence of strabismus after 3months of age may indicate ocular pathology.**
- Myelination of the optic nerves, development of the visual cortex, & growth of (LGB) occur during first two years of life.
- Fovea reaches to maturity at approximately 4 years of age.

- Prevalence of amblyopia 3% to 6%.
- Importance of detection and treatment of amblyopia
- psychological effects of amblyopia on amblyopic child & their parents.
- Self-image, work, school, & friendships were negatively impacted.
- While treatment of amblyopia has a positive impact on psychological disorders.
-

RISK FACTORS TO DEVELOP AMBLYOPIA

- Low birth weight and prematurity
- Delay milestones and CNS disorders
- Positive family history
- Maternal smoking

What are the types of amblyopia?

- The nature of amblyopia differs depending on the cause:-
  - Refractive amblyopia
  - Anisometropic amblyopia
  - Meridonial amblyopia
  - Strabismic amblyopia
  - Visual deprivation amblyopia
  - Toxic amblyopia

Classification of amblyopia

Functional Amblyopia

- Not due to the diseases in the eye
- unilateral/bilateral of the eye
- Reversible
- Examples:
  - Refractive amblyopia
  - Anisometropic amblyopia
  - Meridonial amblyopia
  - Strabismic amblyopia

Structural/Pathological Amblyopia

- Due to lesion in the eye or visual pathway
- unilateral/bilateral of the eye
- Irreversible
- Examples:
  - Visual deprivation amblyopia
  - Toxic amblyopia

What are the sign and symptoms of amblyopia?

Symptoms	Signs
• No symptoms • Blurred vision • Reduced vision • Reduced contrast sensitivity	• No obvious sign, unless severe abnormality is present. • Rubbing or squinting of eyes • Misaligning eyes • Reduced VA • Droopy eyelid

Pathophysiology

- **Changes** have been found in the lateral geniculate nucleus (LGN) and visual cortex.it has been shown that amblyopic eye functions at its best in **mesopic and scotopic** conditions and at its worst under photopic conditions.

- Significant **reduction** in relative **cortical blood flow and glucose metabolism** during visual stimulation of the amblyopic eye during a position emission tomography scan.

Diagnosis

- • Visual Acuity Testing
- Preschool children, Liner acuity is more desirable than single optotype (crowding phenomena), Allen picture figures, LEA figures, HOTV, illiterate E game, wright figures.

- • Fixation testing for amblyopia
- Preverbal children monocular or binocular fixation preference
- Monocular fixation testing: normally developed child more than 2 to 3 months of age should show central fixation with accurate smooth pursuit and saccadic refixation eye movement. test for central fixation by covering one eye then move a target slowly back and forth in front of the child to observe the accuracy of fixation.

Eccentric fixation

- Extrafoveal part of the retina
- Looking to the side, not directly at the fixation target
- Poor smooth pursuits, donot accurately follow a moving target.
- Para foveal 2* off the fovea
- Para macular 2-4* from the center of fovea
- Centrocaecal > 4* b/w macula and optic disc
- Para central around the optic disc.

- **Visuscope (direct ophthalmoscope)**
- Cooperative children

Fixation behavior testing

- Moving a visual target through the child's visual space.
- Each eye tested separately.
- Accuracy is improved if the test is repeated several times.
- The ability to follow past the midline develops at approximately 2 months of age;
- vertical eye movements typically develop around 3 months.

Equal fixation prefrrence

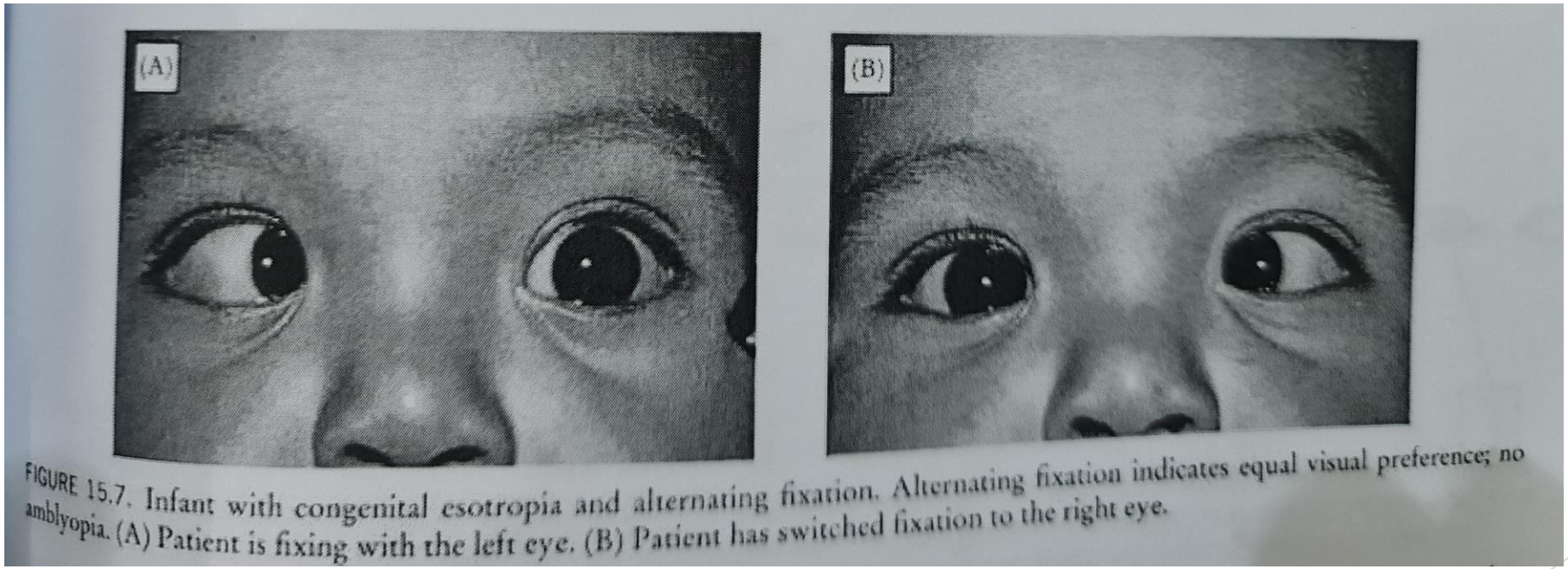
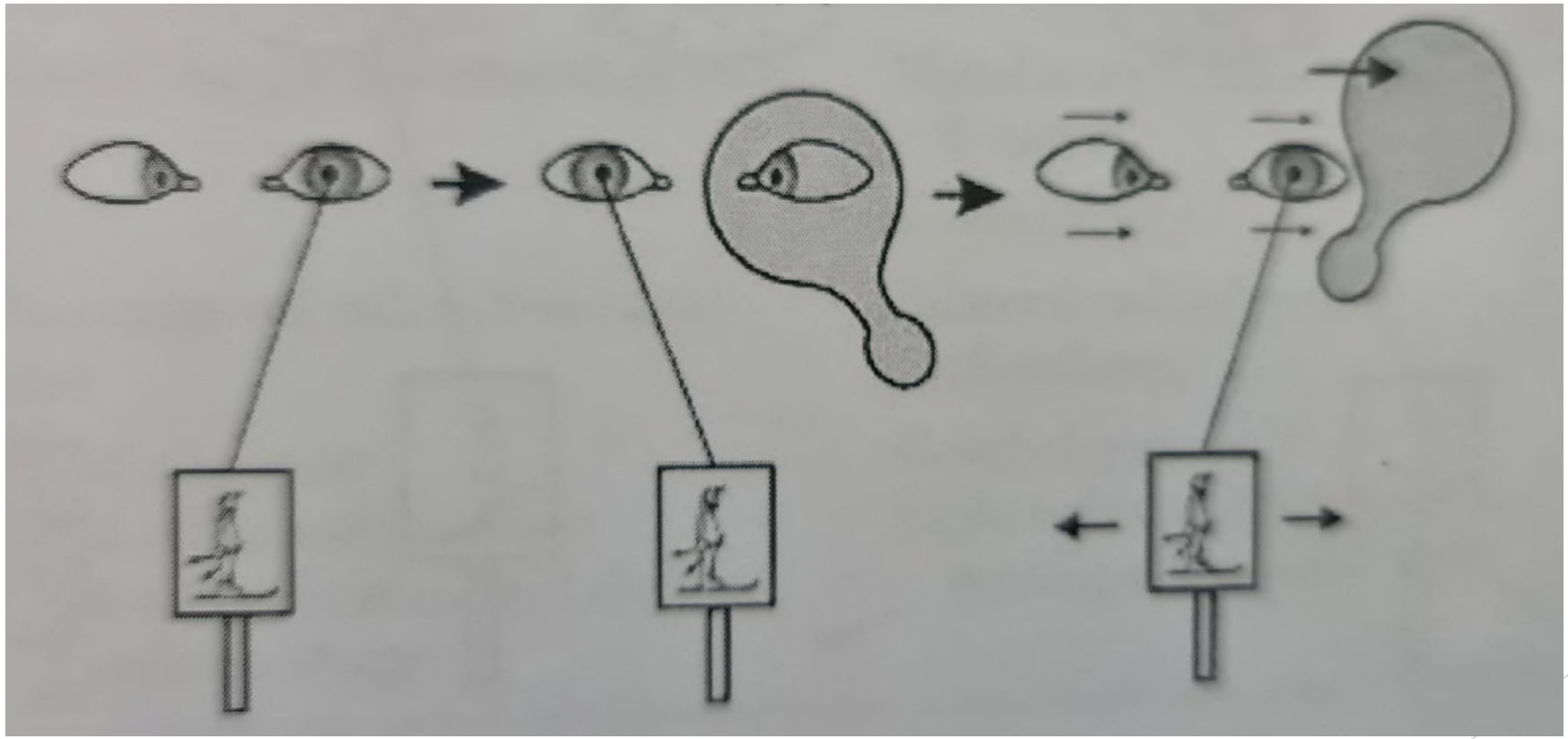


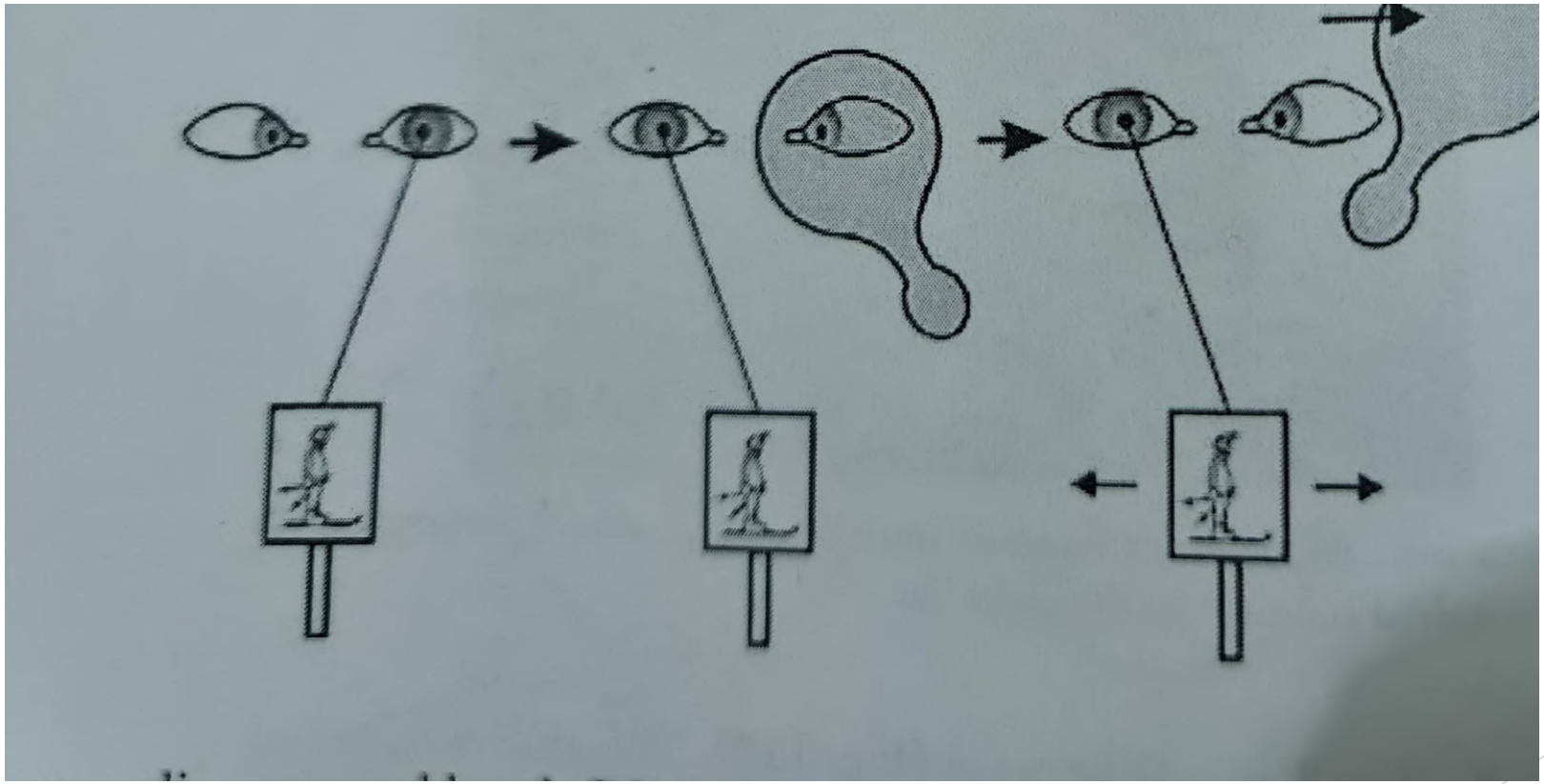
FIGURE 15.7. Infant with congenital esotropia and alternating fixation. Alternating fixation indicates equal visual preference; no amblyopia. (A) Patient is fixing with the left eye. (B) Patient has switched fixation to the right eye.

Strong fixation preference

amblyopia of the deviated eye



Non-preferred eye.
No amblyopia



Vertical prism test (induced Tropia test)
10 PD Fixation test

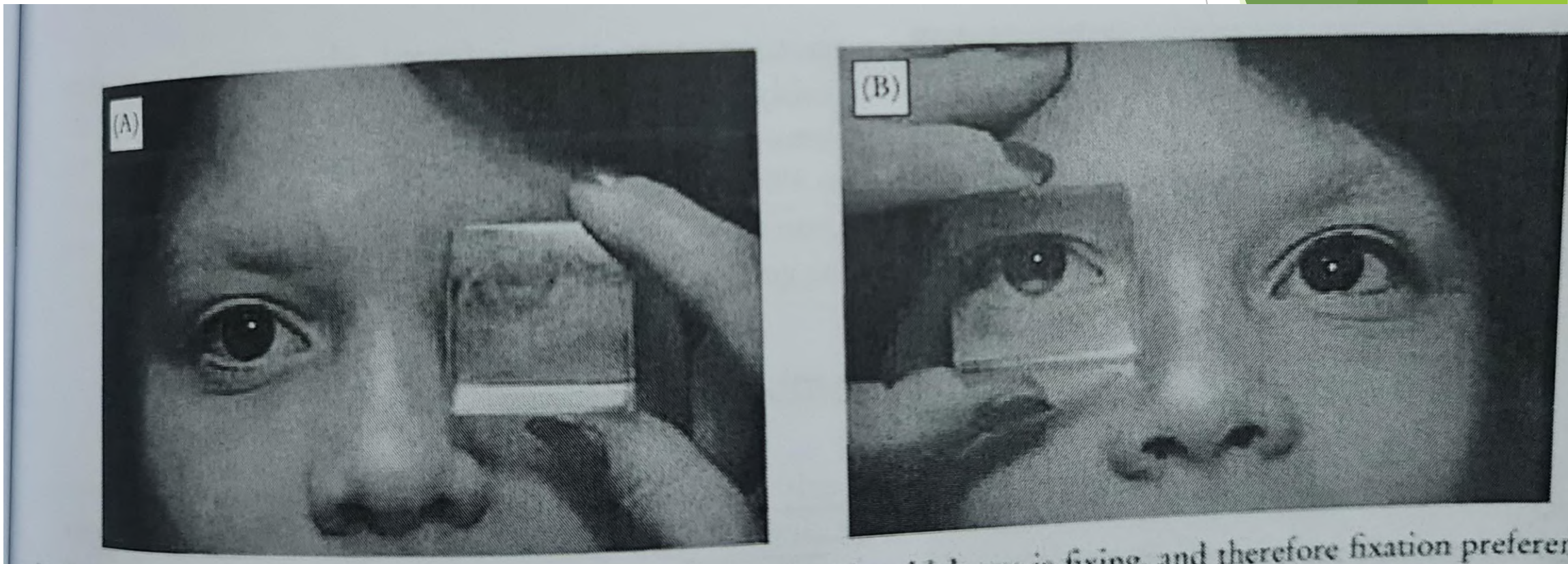


FIGURE 15.11. Vertical prism is placed in front of one eye to identify which eye is fixing, and therefore fixation preferen
determined. (A) One can identify that the right eye is fixing because the right eye is in primary position and the patient i
the vertical displaced image in the left eye. (B) Patient is still fixing with the right eye. Both eyes shift upward because the r
viewing through the prism. This is a base-down prism, so the eyes move up.

Cross-fixation

- Large angle esotropia & tight MR muscle difficulty bringing the eyes to primary position … stay adducted
- “cross fixation”
- Rt fixate on object in lt gaze
- Lt fixate on object in rt gaze
- Ability to hold fixation past midline or to hold fixation through smooth pursuit with either eye…. Equal vision

Latent nystagmus

- Pts with strabismus often have latent nystagmus, horizontal jerky nystagmus
- Occurs or get worse in both eyes if one occluded
- Occlusion of one eye increase nystagmus & diminish VA.
- Blurring of one eye induces less nystagmus than occlusion
- +5 D lens sufficient to blur distance vision
- Take binocular visual acuity measurement in addition to a monocular acuity
- In pts with nystagmus b/c binocular vision is usually better than monocular vision.

Vision Screening
I-ARM

- I INSPECTION
- A ACUITY
- R RED REFLEX
- M MOTILITY

- RED REFLEX (Bruckner test)
  - 2 feet
  - Dim light room
- Hirschberg reflex “corneal reflex”

Amblyopia Treatment
Clear Retinal Image

- Patient with bilateral hypermetropic (>+5.00 D) should receive full hypermetropic correction, as amblyopic eyes do not fully accommodate.
- Partial correction often shows very slow or no improvement in their amblyopia.
- Patient with large astigmatism (>+2.50 D) prescribe full astigmatic correction.
- If patient has anisometropic amblyopia and straight eyes, initially prescribe just glasses and wait to start patching of the good eye.
- Most anisometropic amblyopes will respond to glasses alone or with minimal part-time occlusion of the good eye.
- Children with media opacities, such as cataract, immediate surgery with visual rehabilitation using contact lens, intraocular lens.

Correct Ocular Dominance (strabismus amblyopia)

- **Occlusion**
- covering the sound eye to stimulate the amblyopic eye. Amblyopic patients with essentially straight eyes (tropia <8 PD) and peripheral fusion are best treated with part time patching (3 to 4 hours/day).
- For anisometropic amblyopia, initially prescribe spectacle correction and follow monthly for VA improvement.
- If vision does not improve on monthly follow-ups, then part time patching or penalization therapy is started, these methods help to preserve fusion.
- If vision does not improve with part time occlusion, then full time occlusion should be tried.

Penalization

- Blurring the sound eye to force fixation to the amblyopic eye.
- Penalization only works if fixation is switched from the sound eye to the amblyopic eye.
- Blurring of the sound eye can be achieved by various methods.

- Atropine penalization 0.5%, 1% usually requires +3 or more hypermetropia in the sound eye to obtain significant blur to switch fixation.
- It is important to note that blurring the sound eye to a visual acuity lower than the amblyopic eye does not guarantee a switch fixation to the amblyopic eye.

- Young children with penalization may result reverse amblyopia (decrease vision in previously good eye) patient of 4 years of age or younger should follow closely.
- The Pediatric Eye Disease Investigator Group (PEDIG) has exclusively studied the use of atropine penalization for the treatment of amblyopia.
- Results shows that treatment with patching and atropine penalization gave similar outcome.
- Prescribing proper spectacles correction alone has been shown to be highly effective in treating both strabismic but especially anisomtropic amblyopia.
- That is not to say there is no role for patchting and penalization, but to emphasize the importance of correcting the refractive error.

Occlusive contact lens

- 92% One-line improvement of Snellen acuity but complications limited its usefulness
- Conjunctival irritation, poor contact lens fitting, high recurrence to pretreatment visual acuity 55% recurrence of amblyopia.
- Occlusive contact lens should be considered as a last resort with close follow-up.

Bilateral light occlusion

- Preventive treatment of amblyopia may be the use of bilateral light occlusion.
- Its prolongs the sensitive period of visual development.
- Neonates with hyperbilirubinemia treated under bili-lights who were patched bilaterally for several days to weeks showed that they did not have an increase incidence of amblyopia or strabismus.
- In a separate report by the author, a neonates received 17 days of bilateral patching after having 2 weeks of dense vitreous hemorrhage and hyphema.

Levodopa /Carbidopa in the treatment of amblyopia 4:1 dose ratio:

- Drugs use for Parkinson's disease.
- Levodopa 1.5mg/kg/day 1 to 7 weeks 3 divided doses

Antidepressants drugs : fluoxetine

GABA antagonist & cytidine-5-diphosphocholine.

Pleoptics

- Treating eccentric fixation association with dens amblyopia.
- A bright ring of light is flashed around the fovea to temporarily “blind” or saturate the photoreceptors surrounding the fovea, which eliminates vision from eccentric fixation point and forces fixation to the fovea.
- Pleoptics treatment are given several time a week to enhance occlusion therapy.

Active stimulation

- A high contrast spinning disc with square-wave grating was one method that has been tried (CAM) to the amblyopic eye to improve vision in the amblyopic eye.

Prognosis of amblyopia

- Depends upon age of the patient,
- Compliance,
- Severity of amblyopia and
- Type of amblyopia.
- The earlier the amblyopia occurs and the longer it remains untreated, the worse the prognosis.
- Bilateral amblyopia responds better than unilateral amblyopia.
- Myopic anisometropic amblyopia respond better than hypermetropic anisometropic amblyopia

THANK YOU

Basic Squint

Associate professor
DR Afzal Qadir
HOD
Paedritic ophthalmology

Objectives

- Definition
- Types
- Orthoposition
- Axis /Law
- Etiology
- Clinical testing
- Moments
- classification

Squint

- Definition: A “squint” is the common name for 'strabismus' which is the medical term used to describe eyes that are not pointing in the same direction, or which are misaligned.
- Squint refer to the deviation of the at primary position

STRABISMUS


NORMAL VISION


BINOCULAR SQUINT


BINOCULAR DIVERGENT STRABISMUS


MONOCULAR SQUINT


DIVERGENT STRABISMUS MONOCULAR

Types squint

- Latent squint : The condition in which the tendency of the eye to deviates is overcome by fusion reflex during binocular vision.
- Example

1. Esophoria
2. Exophoria
3. Hypophoria
4. Hyperphoria
5. In and Excyclophoria

Manifest squint

- Manifest : manifest squint is present when the eyes are open and being used.
- TYPES:

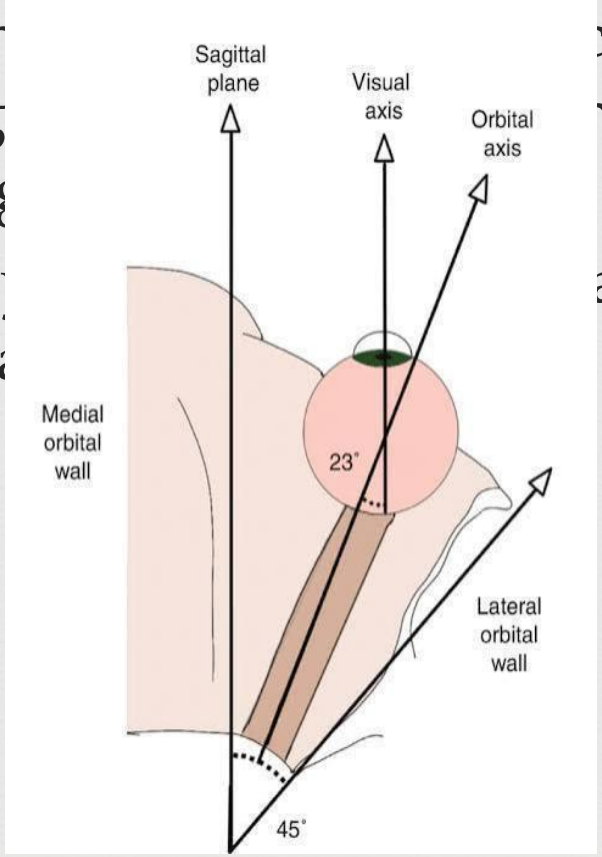
1. Concomitance of deviation .

- Comitant strabismus
- Incomitant strabismus

there maybe (Eso,Exo,Hyper,Hypo)

Orthoposition

- If there is no sh…contralateral eye, while covering …t is orthotropic (i.e. normal alig…
- A position of e…ary line of the sight intersect a…



Visual Axis

- **visual axis** :also known as the line of sight, is the imaginary line that connect the object of fixation to the fovea.
- It is path through which light reach the retina.
- **Optical axis/Pupillary axis** : optical axis is the imaginary line passing through center of the opitcal system.
- **Orbital axis**: the line passes through the apex of thr bony orbit and the center of the opening of the orbit.

Angle of kappa

- The angle between pupillary axis and visual axis.
  - **Anatomical variation**: orbital shape and size.
  - **Eye position**
  - **Corneal shape**
  - **Lens shape and position**

Etiology

- Sensory obstacles

1. Uncorrected refractive error
2. Anisometropia
3. Media opacity
4. Congenital ptosis

- Motor obstacles

1. Congenital abnormalities
2. Abnormal EOM
3. Heredity

Clinical testing

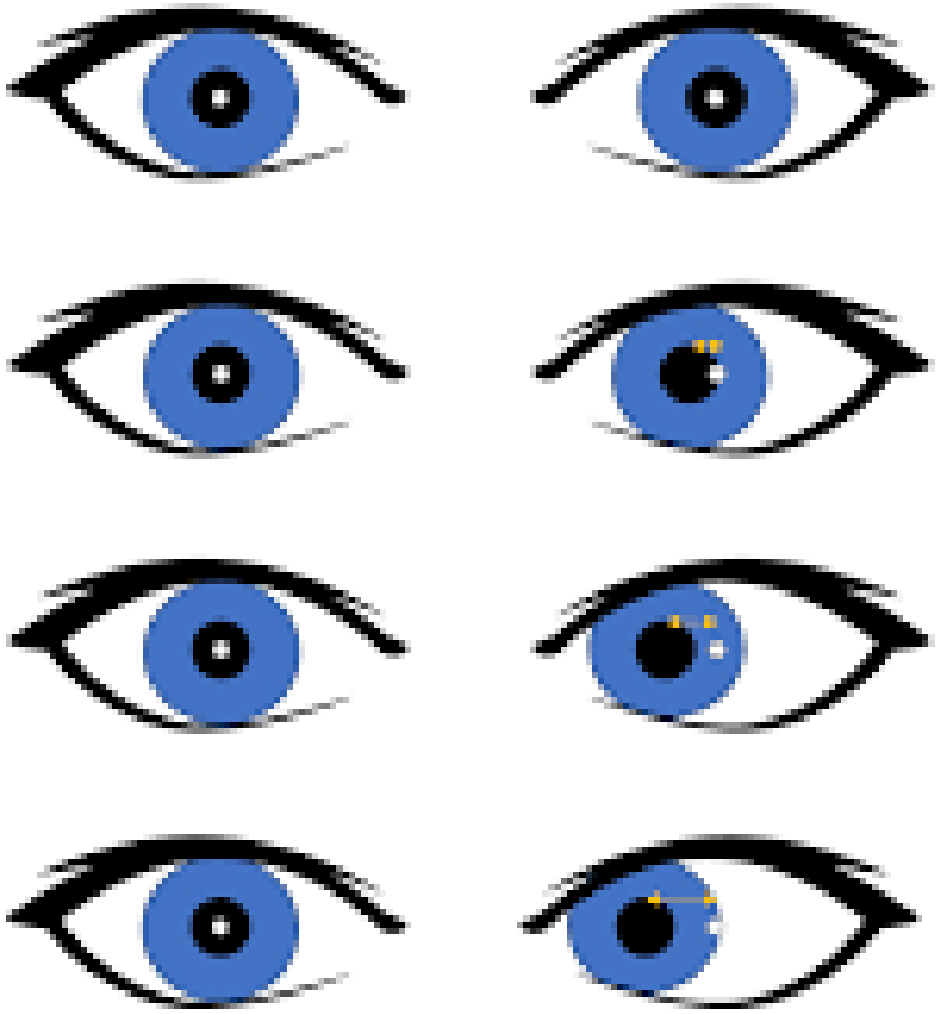
- Following clinical testing are used in squint such as given below

1. H.B
2. Cover uncover test
3. Krimsky test
4. Synoptophoria
5. Bruckner test

Hirschberg test

- The Hirschberg test (also known as the corneal light reflex test) is a quick and simple way to check ocular alignment.
- It is first very common one to rule out the deviation of the eye.

H.B degree



Normal corneal reflex

1 mm deviation ≈ 15 diopters

2 mm deviation ≈ 30 diopters

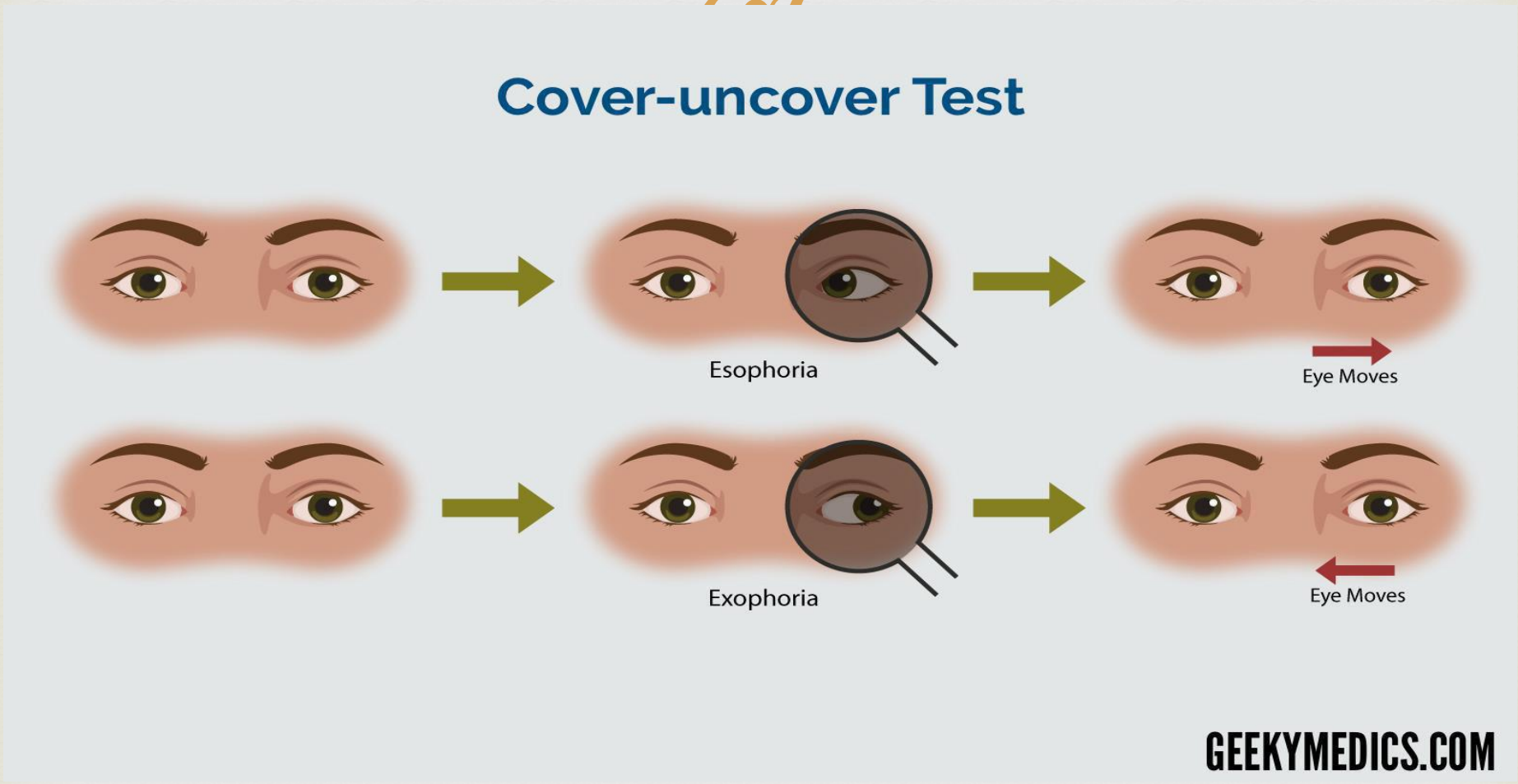
3 mm deviation ≈ 45 diopters

4 mm deviation ≈ 60 diopters

Cover uncover test

- The cover-uncover test is generally performed first. The cover-uncover test is useful to identify a tropia and differentiate it from a phoria. The test is done by using an opaque or translucent occluder to cover one eye. The occluder is held in front of the eye for a few seconds and then removed.

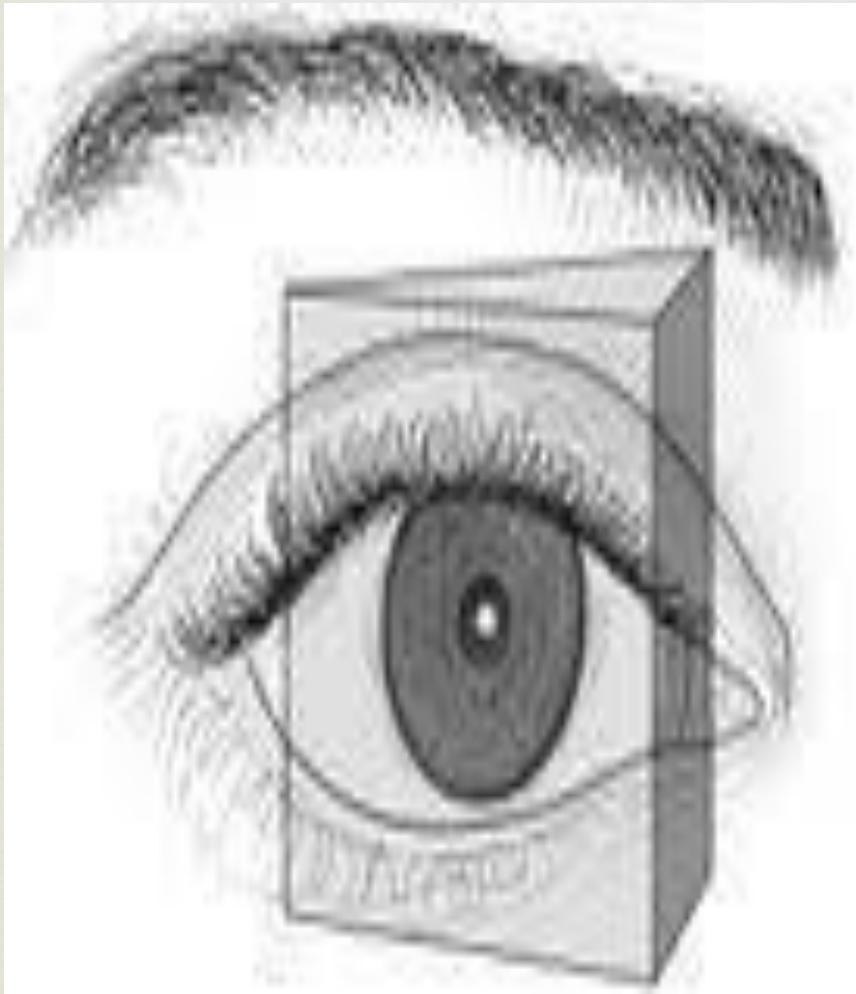
CONT

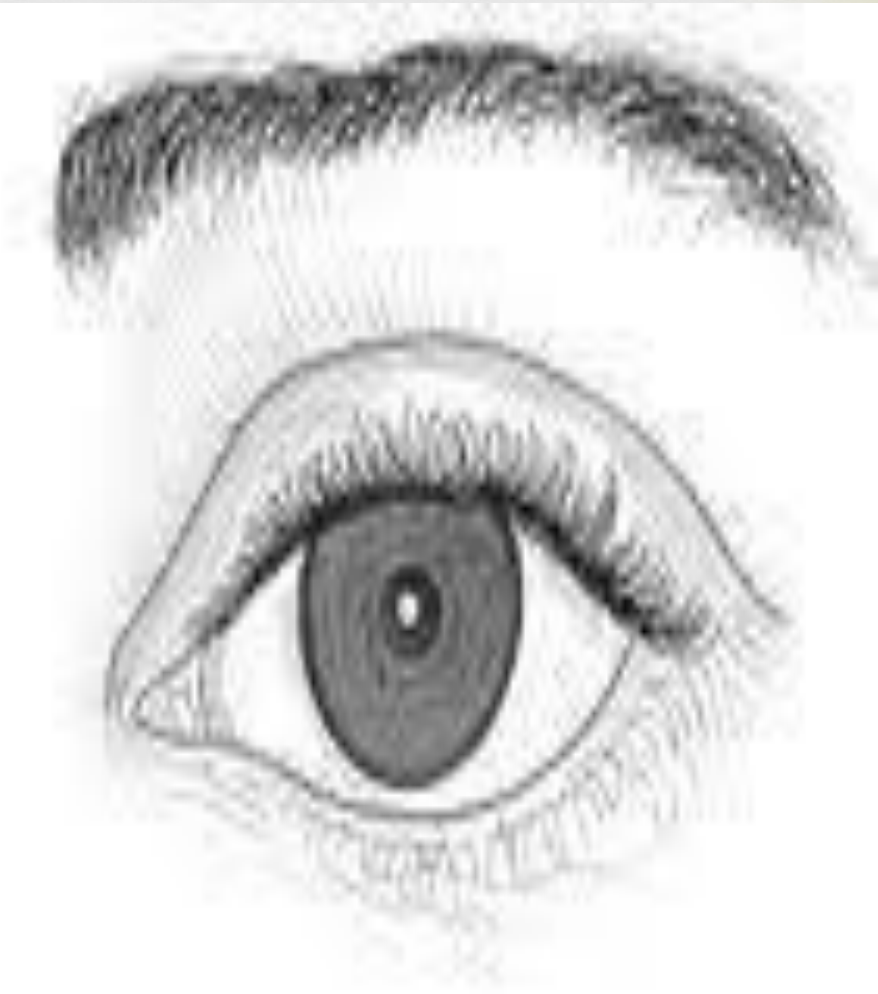

Cover-uncover Test
Esophoria
Eye Moves
Exophoria
Eye Moves
GEEKYMEDICS.COM

Krimsky test

- Krimsky test. Prism is placed in front of the deviating eye to correct the deviation of the corneal light reflex. The strabismus measurement is equal to the amount of prism necessary to center the corneal light reflex on the pupil of the deviating eye.
- The apex should toward deviation.

CONT





Synoptophoria

- Synoptophore is an instrument for assessing binocular vision, measuring angles of deviation and treating binocular anomalies by conventional orthoptic and peptic methods.

CONT

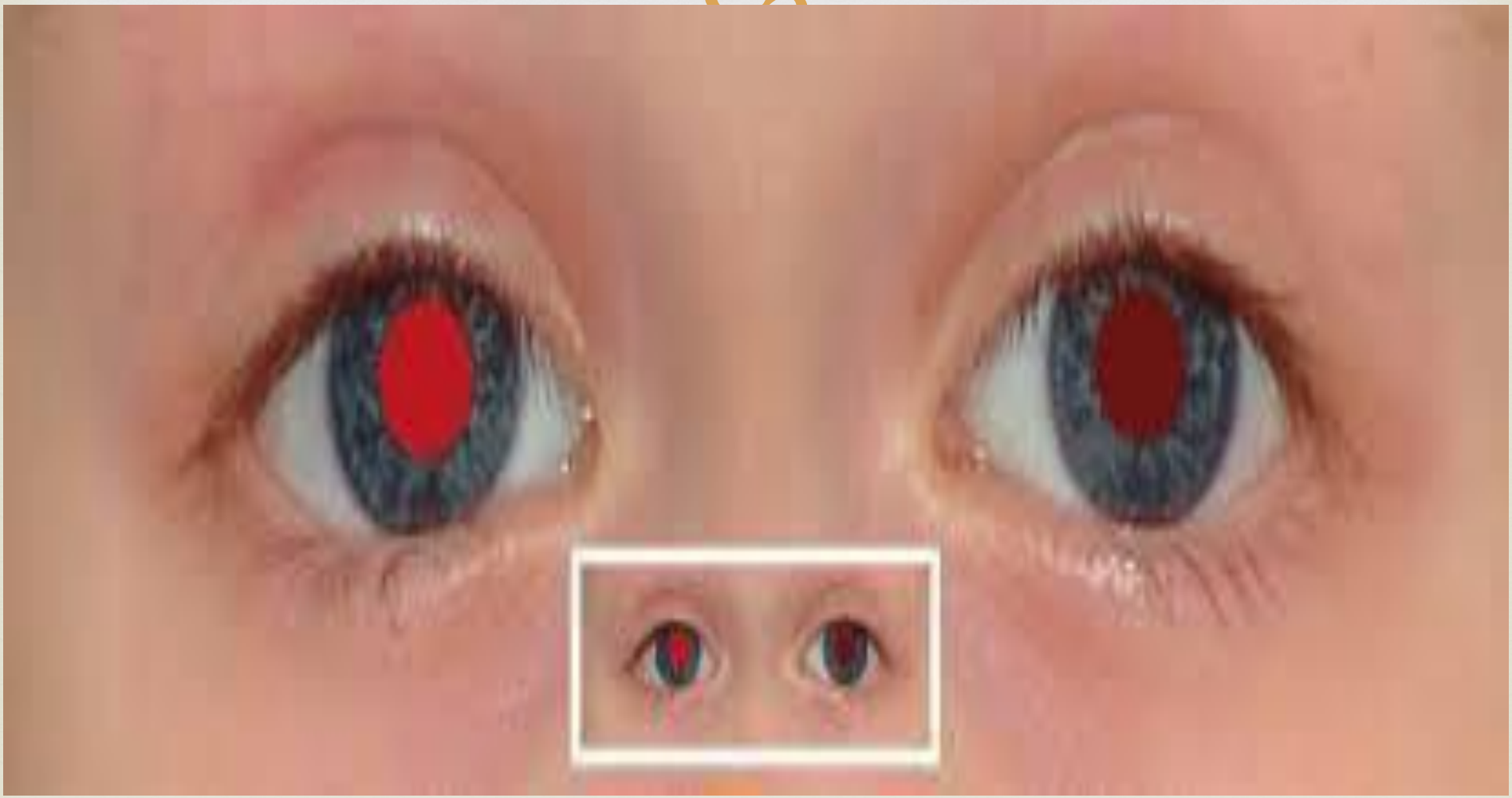
Bruckner test

- Brückner, in 1962, published a paper in German describing a “trans-illumination” test extremely useful in the diagnosis of small angle deviations and amblyopia in young uncooperative children. A bright coaxial light source, such as a direct ophthalmoscope, is used.

CONT


BRÜCKNER TEST
Md . Azizul Islam
Associate Optometrist
Oculoplasty Department (IIEI&H)

Cont



Moments

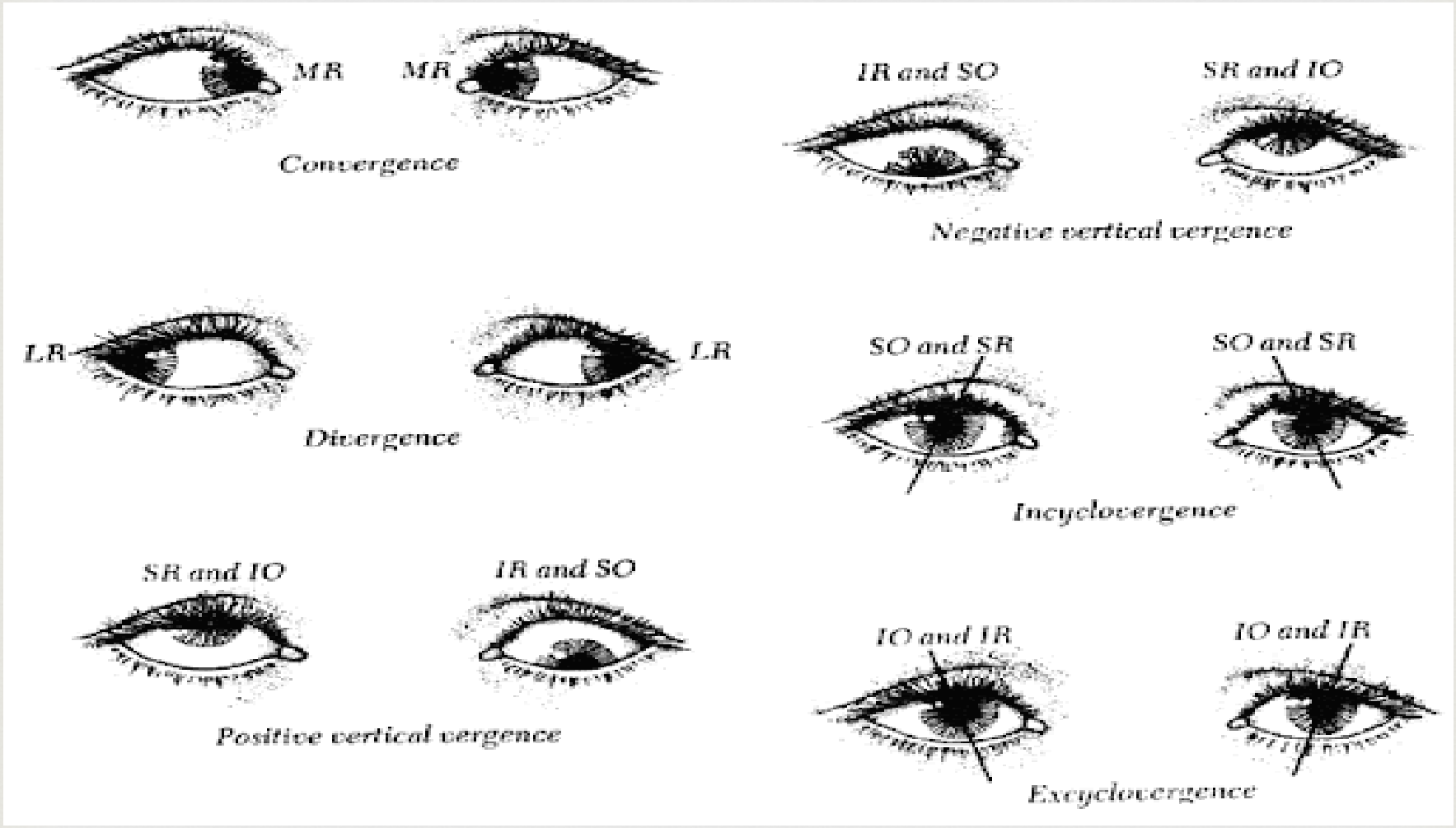
- Convergence
- Version
- vergences
- Saccade

➢ Version : Binocular eye movements are either conjugate (versions) or disconjugate (vergences). Versions are movements of both eyes in the same direction (e.g, right gaze in which both eyes move to the right). Dextroversion is movement of both eyes to the right, and levoversion is movement of both eyes to the left.

Vergences

- Vergence eye movements are disjunctive movements that move the eyes in opposite direction (i.e., convergence or divergence). Their function is to hold the images of a single object simultaneously on both foveae.

Vergences


MR
MR
Convergence
IR and SO
SR and IO
Negative vertical vergence
LR
LR
Divergence
SO and SR
SO and SR
Incyclovergence
SR and IO
IR and SO
Positive vertical vergence
IO and IR
IO and IR
Excyclovergence

Saccades Moments

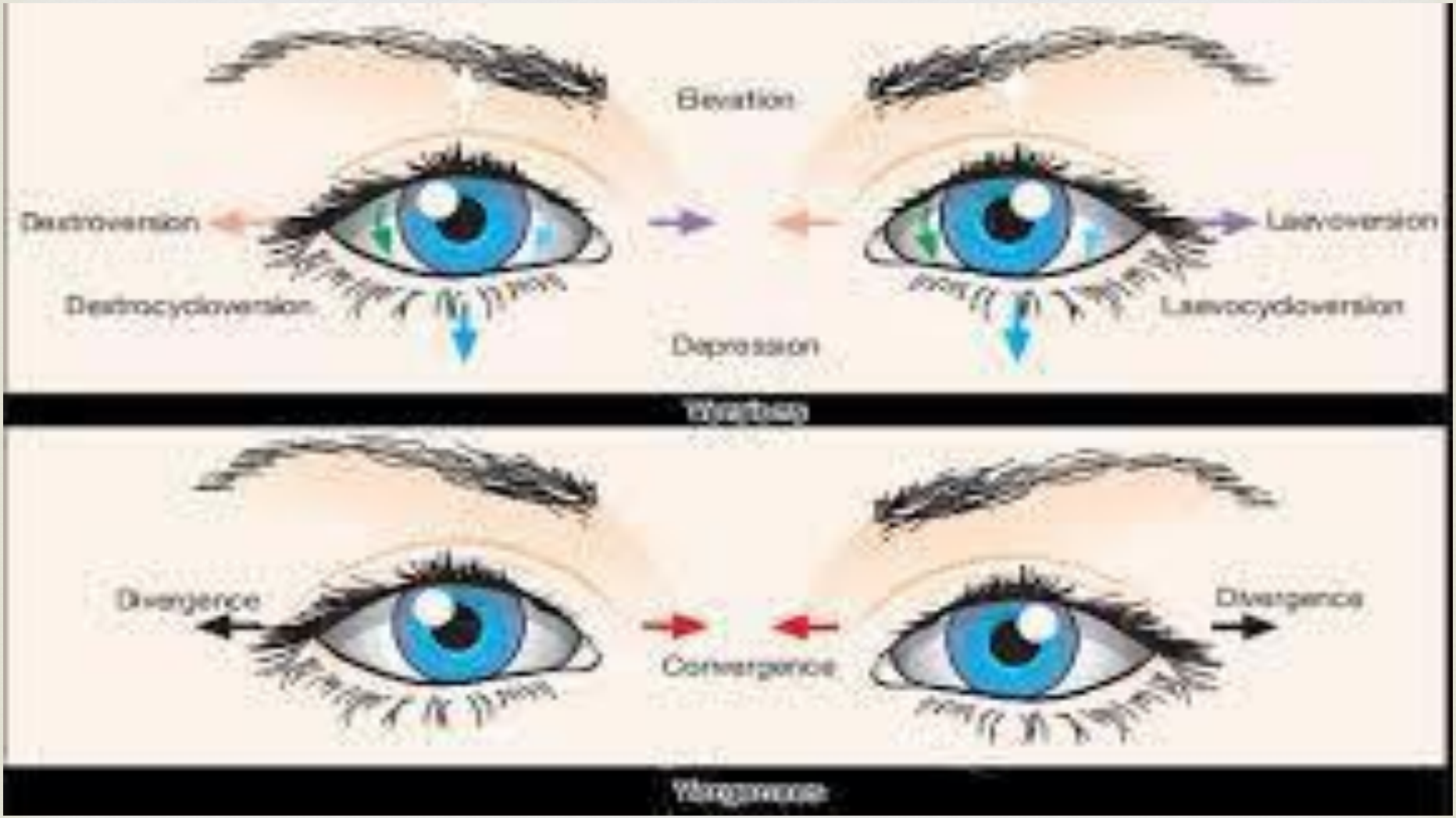
- Saccades are rapid, ballistic movements of the eyes that abruptly change the point of fixation. They range in amplitude from the small movements made while reading, for example, to the much larger movements made while gazing around a room.

CONT

Convergence

- “Convergence” describes the way your eyes move together and point inward when you look at nearby objects, such as books, tablets or smartphone screens. With convergence insufficiency, there's an eye coordination problem, in which your eyes instead drift outward as you look at objects close-up

CONT


Elevation
Dextroversion
Laevoversion
Depression
Divergence
Convergence
Divergence

Classification

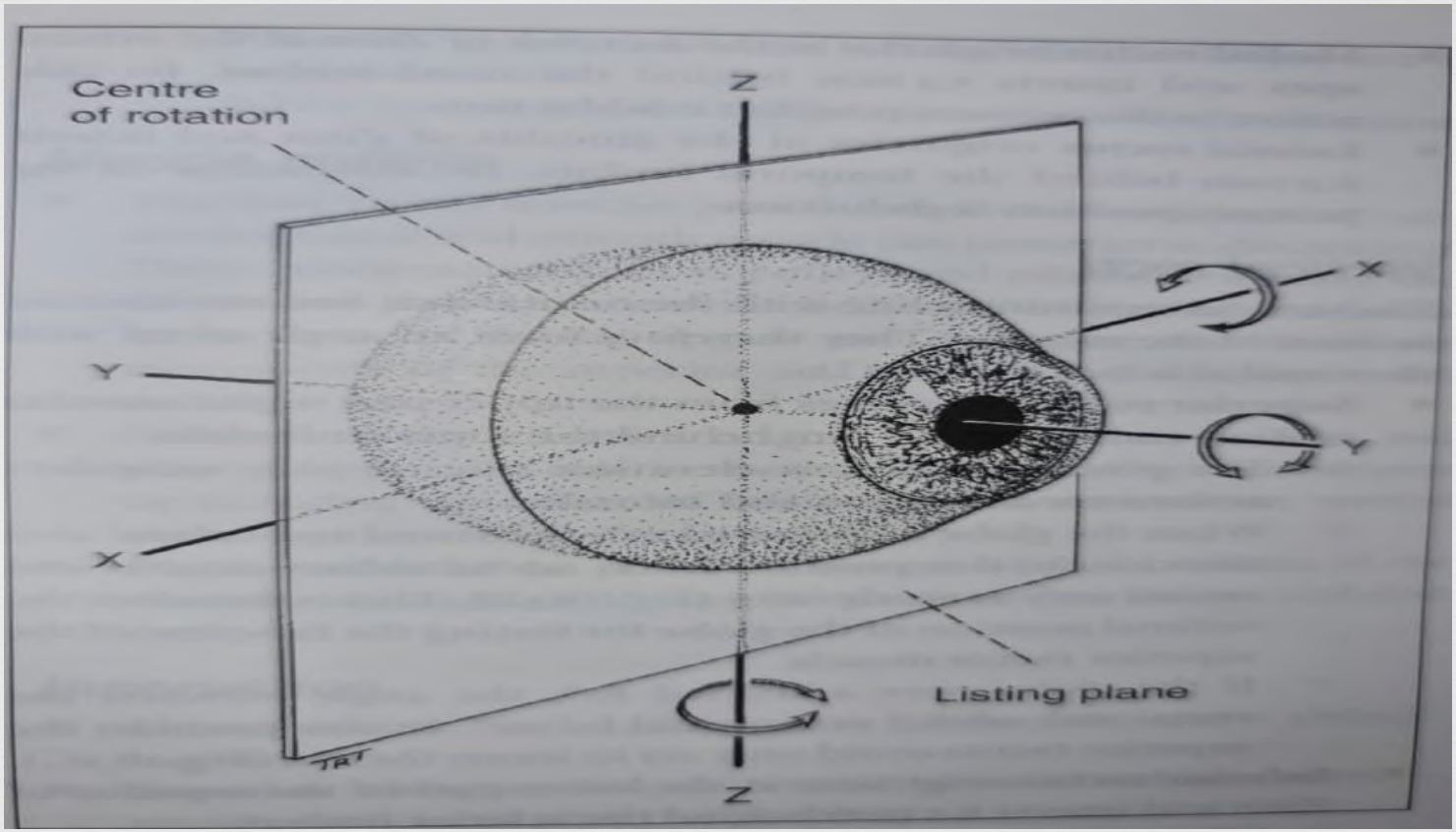
- Squints can be classified according to the direction of the turn of the eye: esotropia (convergent) refers to an eye that turns inwards towards the nose; exotropia (divergent) refers to an eye that points outwards; hypertropia is when the eye is in an upward direction

Esotropia

Exotropia

Hypertropia

Hypotropia


Centre
of rotation
Z
X
Y
Y
X
Z
Listing plane

ANY
QUESTIONS

THANK YOU

CLINICAL EVALUATION OF STRABISMUS

HISTORY

- Age of onset
- Variability
- General health
- Birth history
- Family history

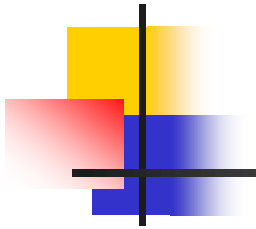
Examination

- Visual acuity testing
- Tests for stereopsis
- Tests for sensory anomalies
- Measurement of deviation
- Motility tests
- Refraction and fundoscopy

Visual acuity

- Minimum angle of separation (subtended at the nodal point of the eye) between two objects that allow them to perceive as one

- Normal min angle of separation is 1 min or less, and corresponds to letters on the 6/6 line of the snellen chart, when viewed from 6 meters

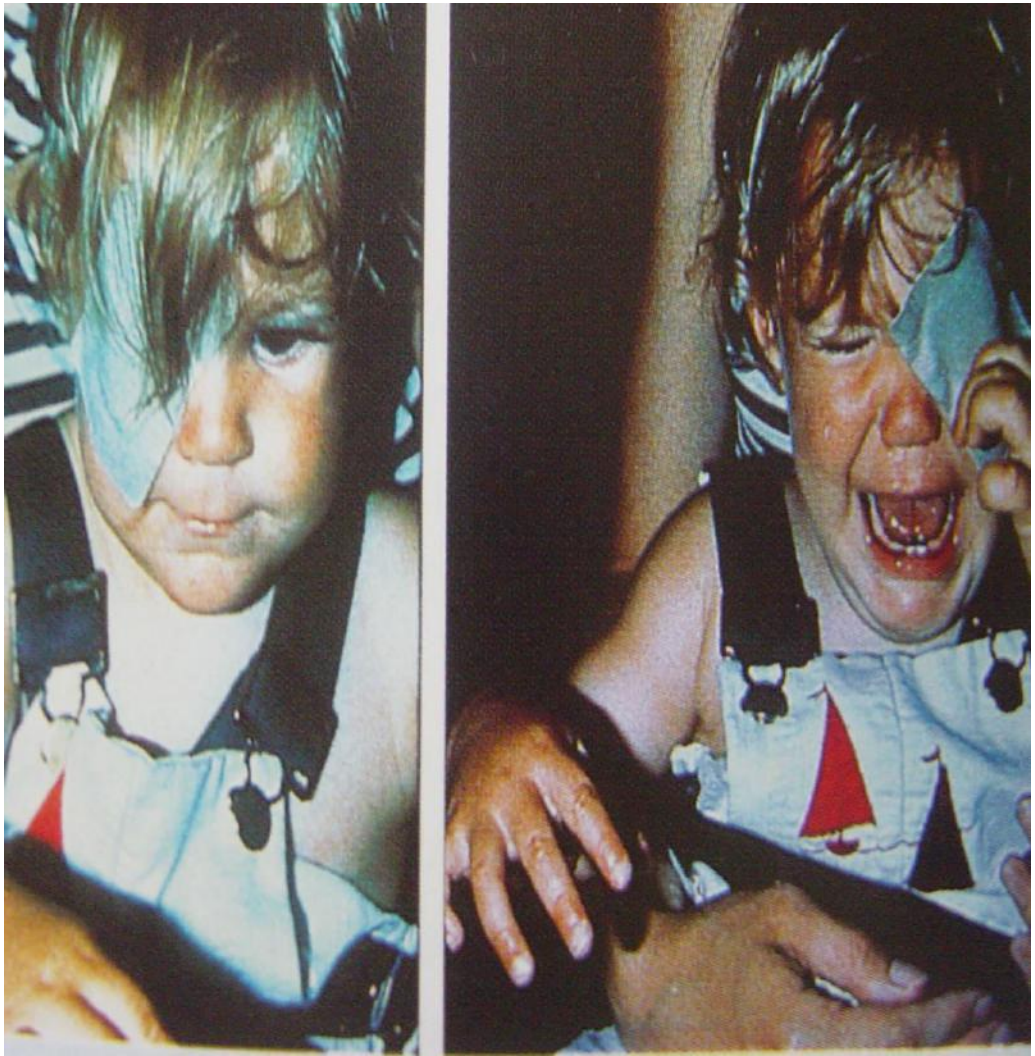


- Testing VA in preverbal children
- Testing VA in verbal children

Testing VA in preverbal children

Simple examination and observation of the child

Occlusion of one eye



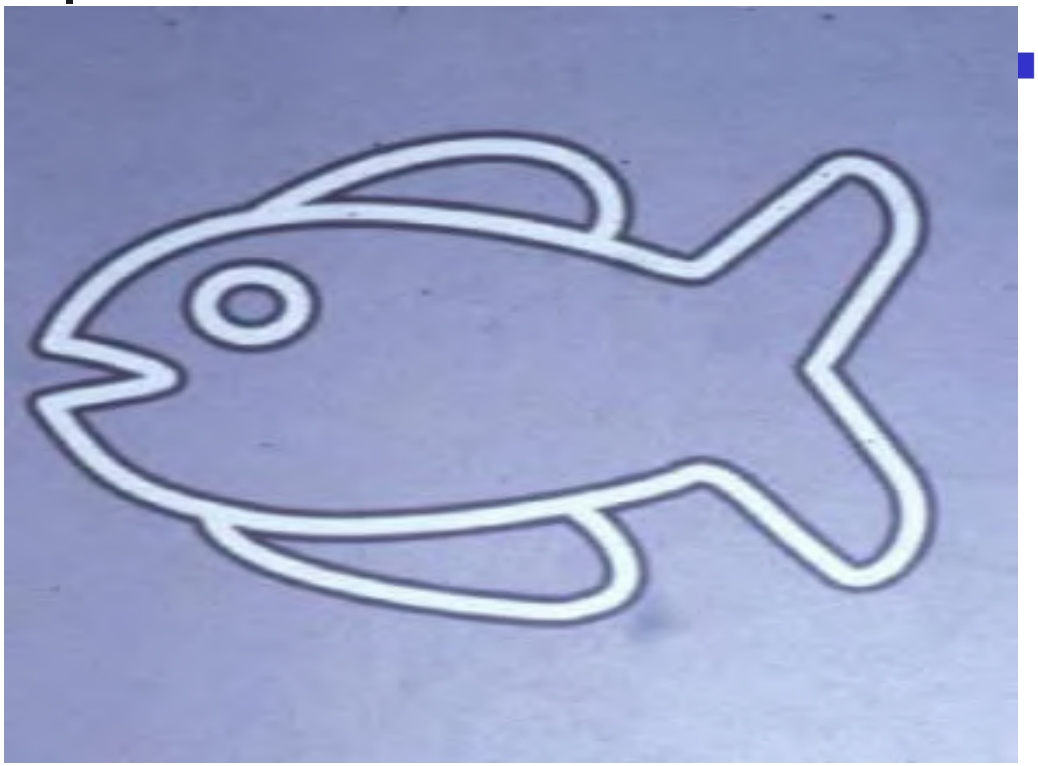
Hundreds and thousands test

If a child is able to see and pick up small sweets at 33cm VA is at least 6/24

Rotation test

- Gross qualitative test of the ability to fixate with both eyes open
- Examiner hold the kid facing him and rotates briskly through 360^0
- If VA is normal eyes will deviate in the direction of rotation under the influence of vestibul-ocular response
- The eyes will flick back to normal position to produce nystagmus
- When rotation stops nystagmus also stops due to suppression of post-rotatory nystagmus by fixation
- If viion is poor the post rotatory nystagmus does not stop as vestibulo-ocular response is not blocked by visual feed back

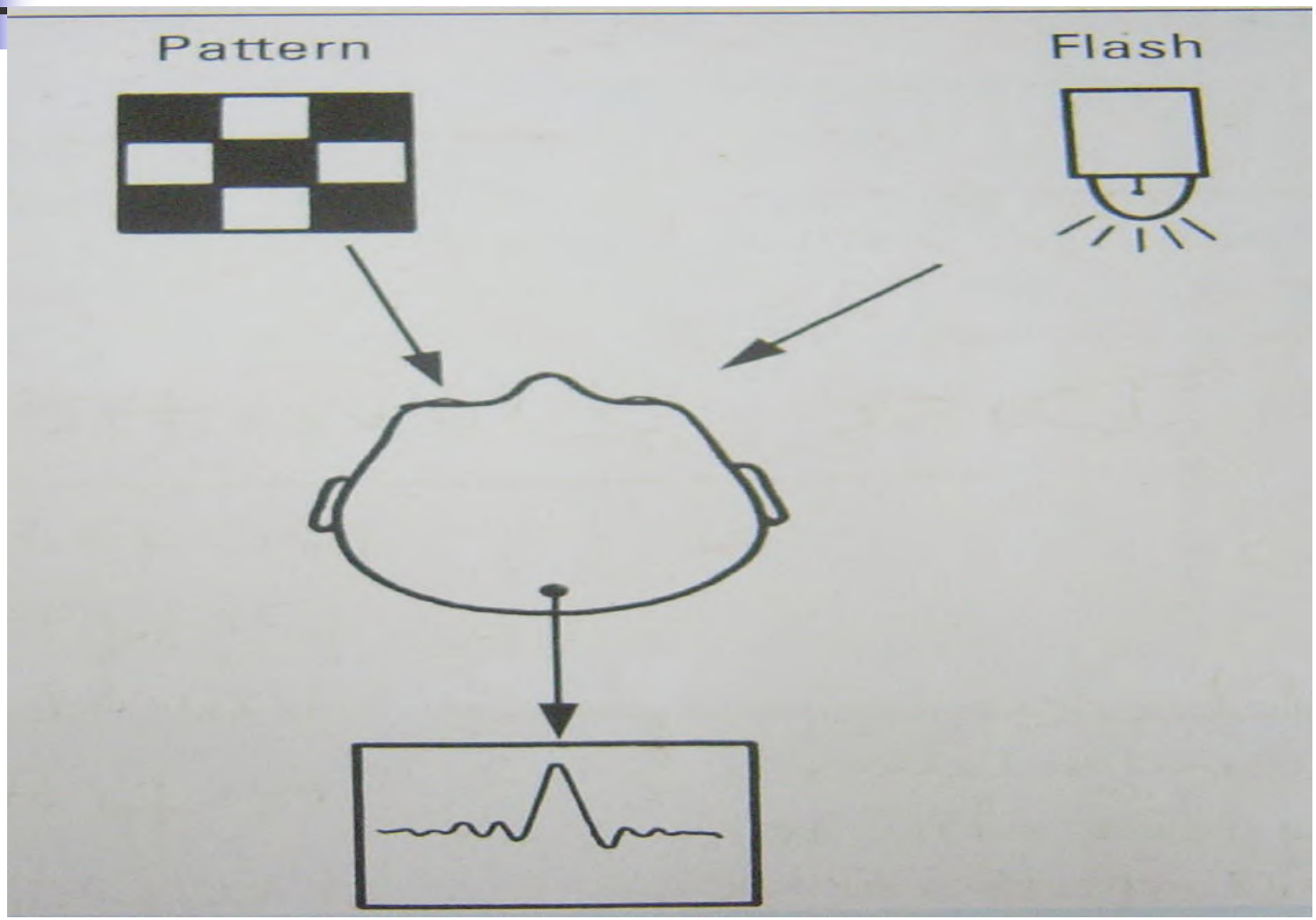
Preferential looking



- They are based on the fact that infants prefer to look at a pattern rather than homogenous stimulus

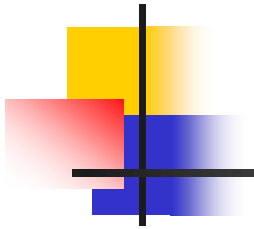
  (cardiff acuity cards)

Pattern visual evoked potential



Fixation test

- A 16 base down prism over one eye and occlude the other eye
- The eye behind the prism is forced to elevate and take up fixation
- Observe the eye behind the prism
- Fixation is guarded as central non central steady and non steady
- Uncover the other eye and note its fixation
- If fixation immediately returns to the uncovered eye then VA is impaired , if it is maintained after a blink then VA is good
- Repeat with the other eye



- Remember fixation should be

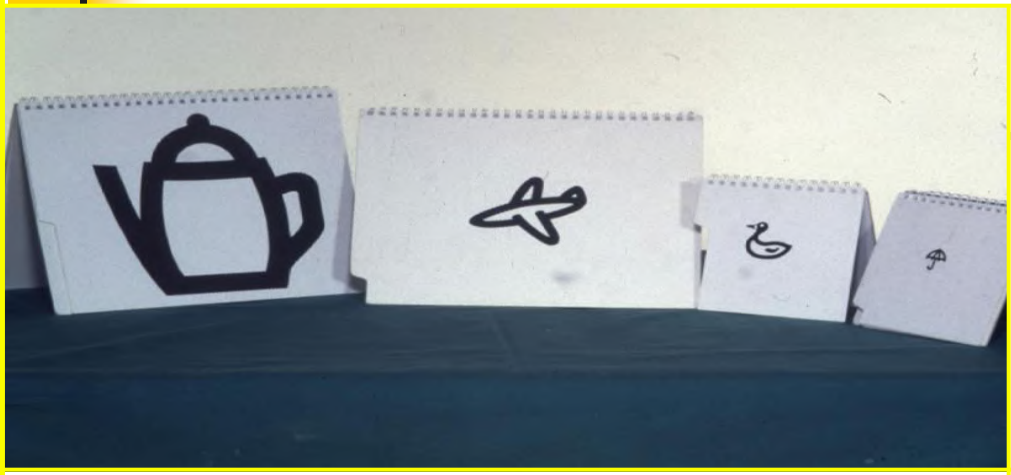
C entral

S teady

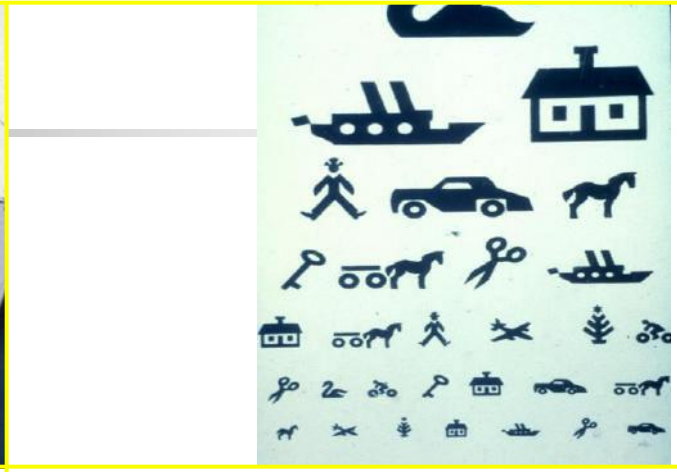
M aintained

VA testing in verbal children

At age 2 years (naming pictures)



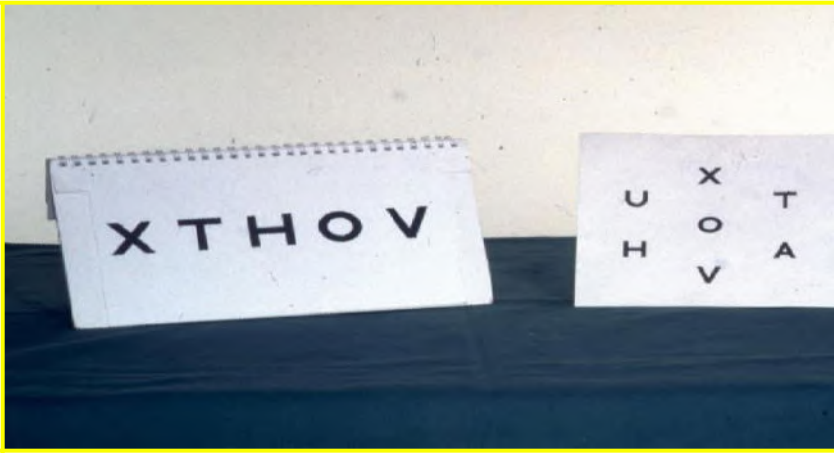
Kay single picture



Multiple pictures

At age 3 years (matching tests)

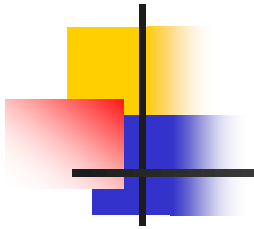
Sheridan-Gardiner



Sonksen-Silver

KEELER LOG Mar Crowded test

Sheridan-Gardiner test



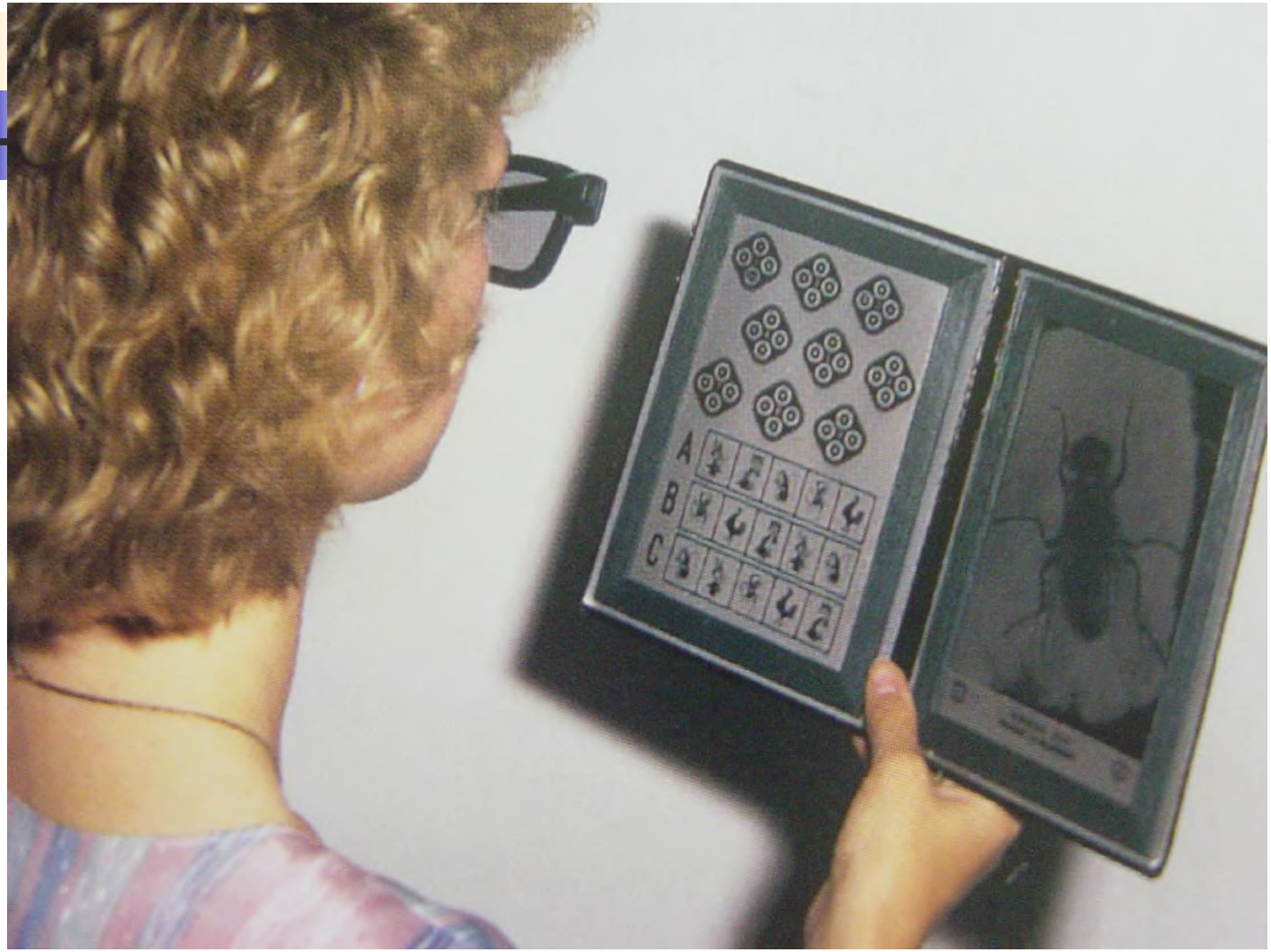
- At the age of 4 years they can perform on Snellen acuity chart

Tests for stereopsis

- It is measures in seconds of arc (1° = 60 minutes of arc ; 1 minute =60 seconds of arc
- Normal stereoacuity is 60 seconds, the lower the value the better the acuity

Titmus test

- 3 dimensional polaroid vectograph viewed through polaroid spectacles
- Fly is for gross stereopsis (3000 sec of arc)
- Circles there are 9 squares with 4circles (800 -40 sec of arc)
- Aimals (400-100 sec of arc)


A
B
C

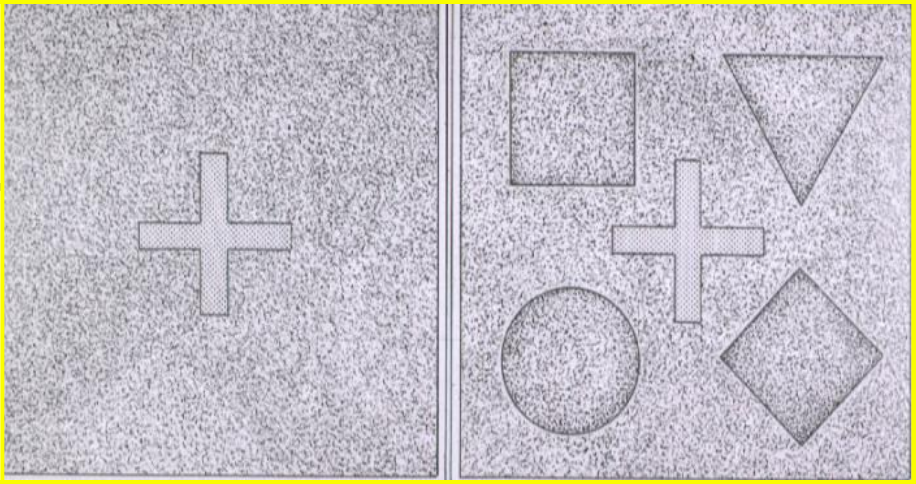

1
2
3
4
5
6
7
8
9
A
B
C

Tests for stereopsis

Titmus

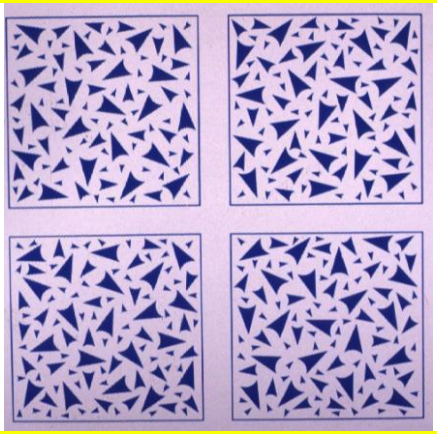
- Polaroid spectacles
- Figures seen in 3-D

TNO random dot test



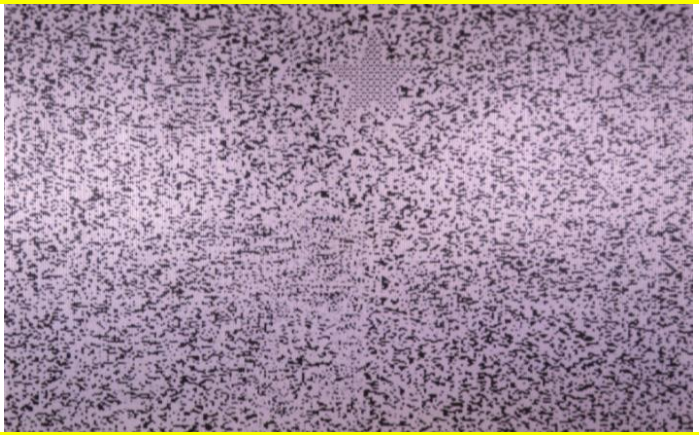
- Red-green spectacles
- 'Hidden' shapes seen

Frisby

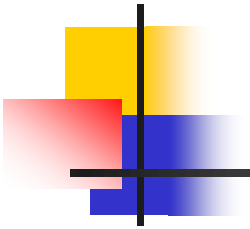


- No spectacles
- 'Hidden' circle seen

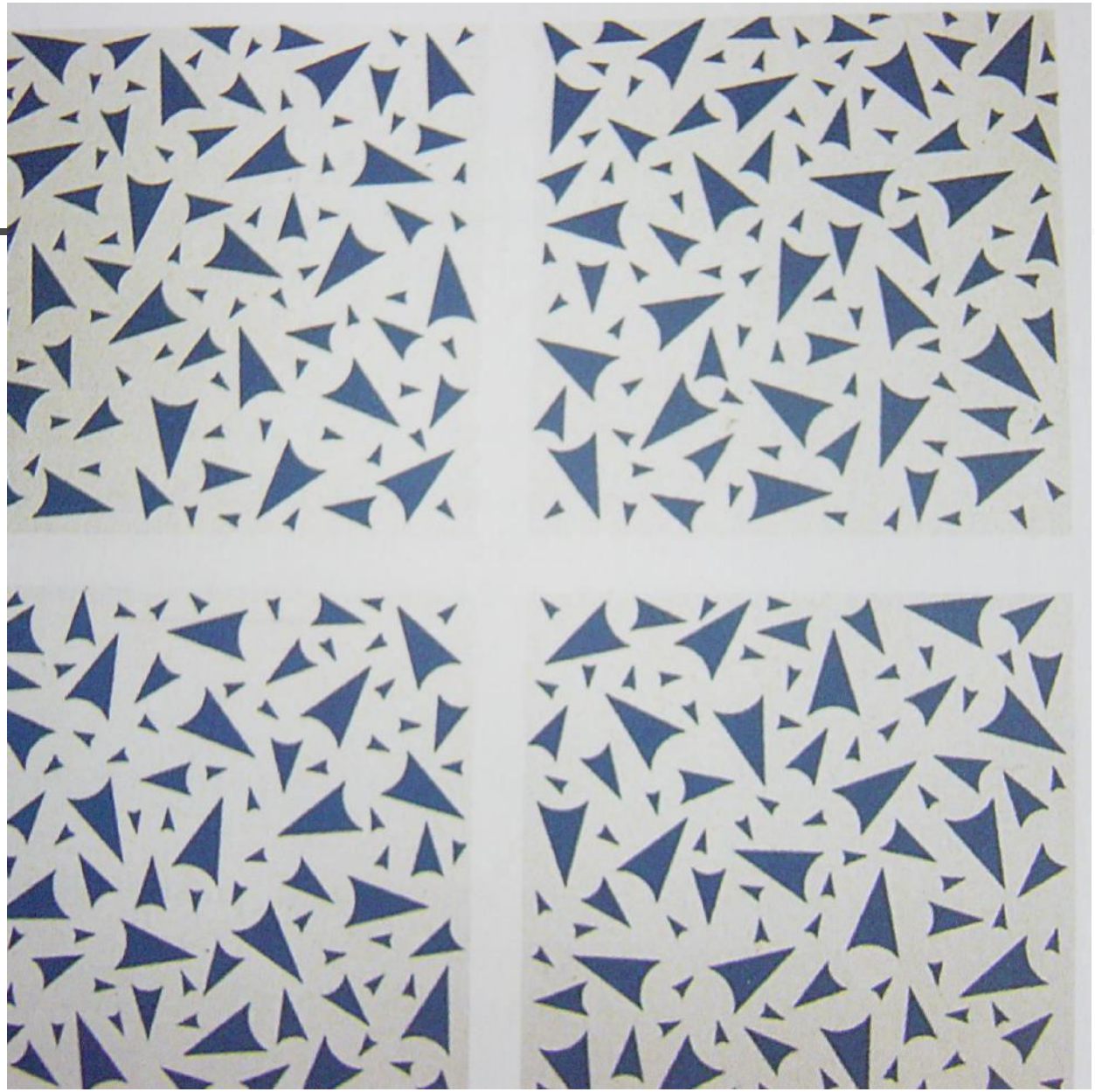
Lang



- No spectacles
- Shapes seen

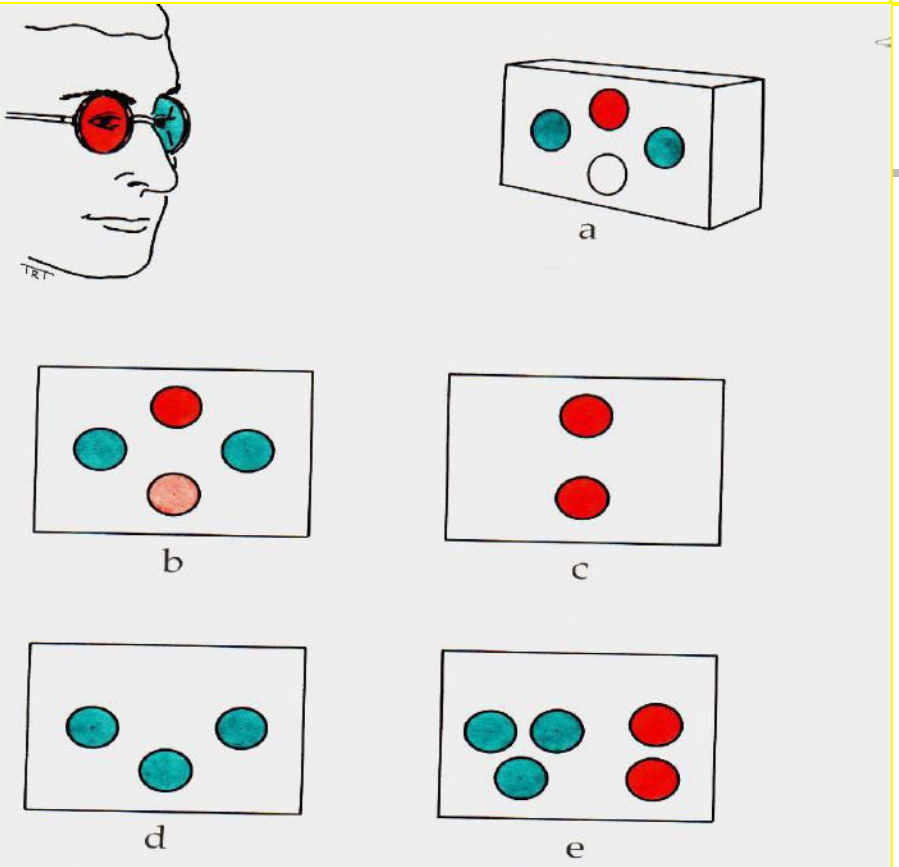


- TNO Test is a true measure of stereopsis (480 -15 sec of arc)
- LANG Test especially useful in babies as they instinctively reach out to touch shapes
- FRISBY Test disparity is created by thickness of plates (600-15 sec of arc)



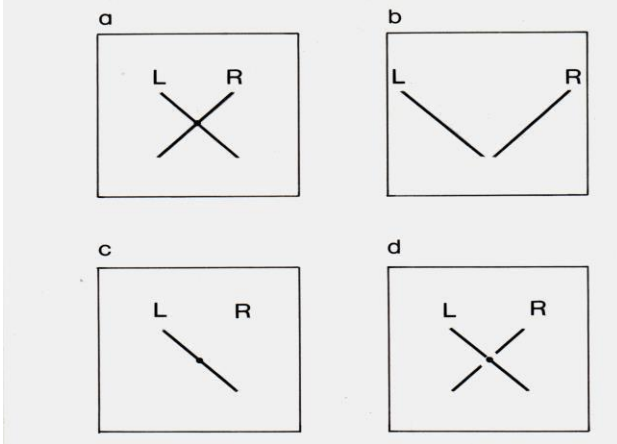
Tests for sensory anomalies

Worth four-dot test

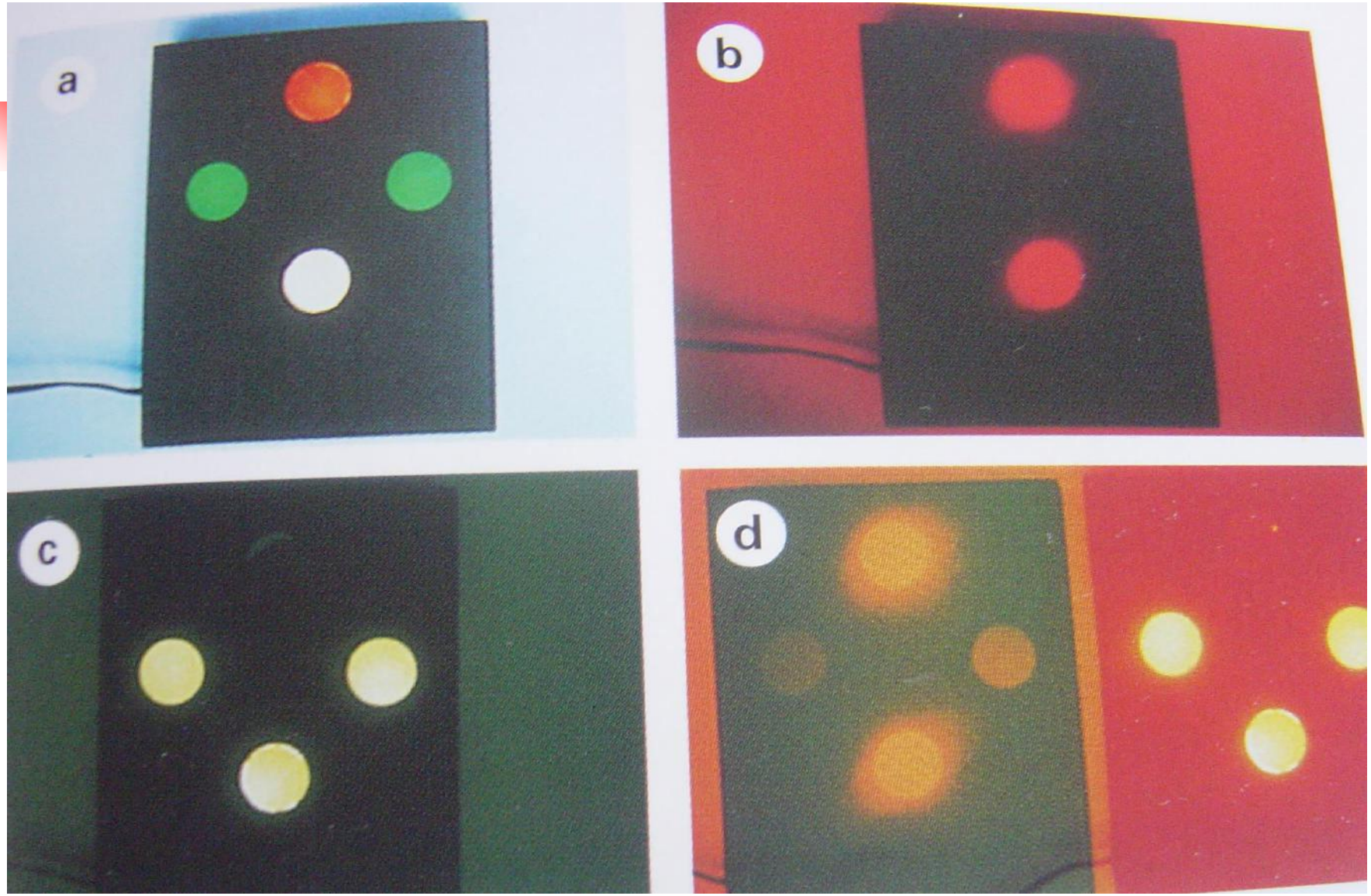


a - Prior to use of glasses
b - Normal or ARC
c - Left suppression
d - Right suppression
e - Diplopia

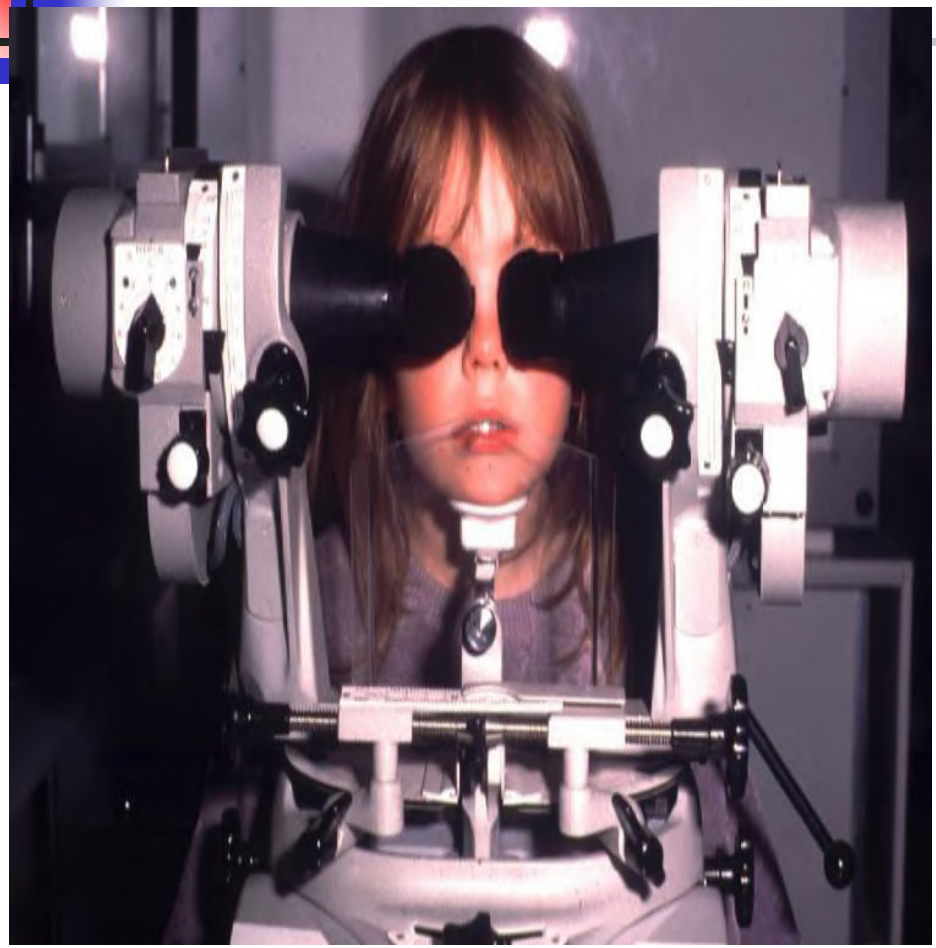
Bagolini striated glasses



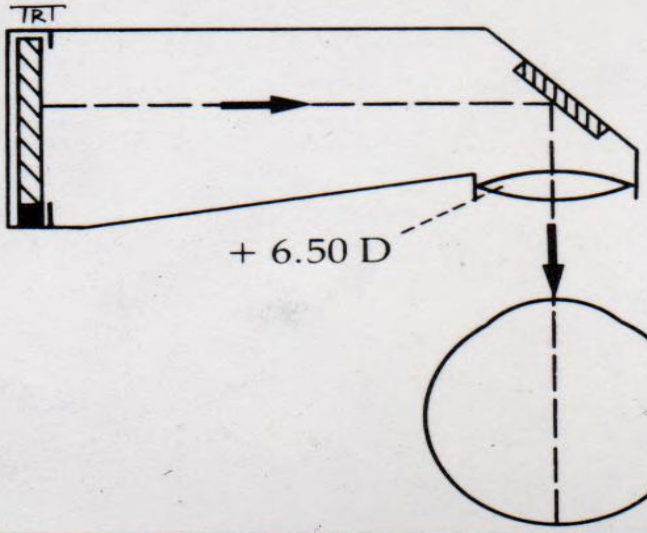
a - Normal or ARC
b- Diplopia
c - Suppression
d - Small suppression scotoma

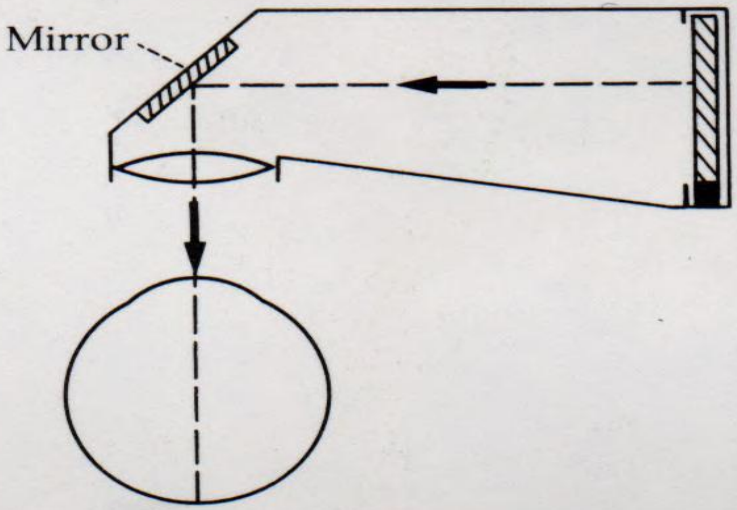

a
b
c
d

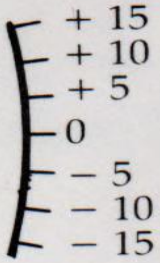
SYNOPTOPHORE

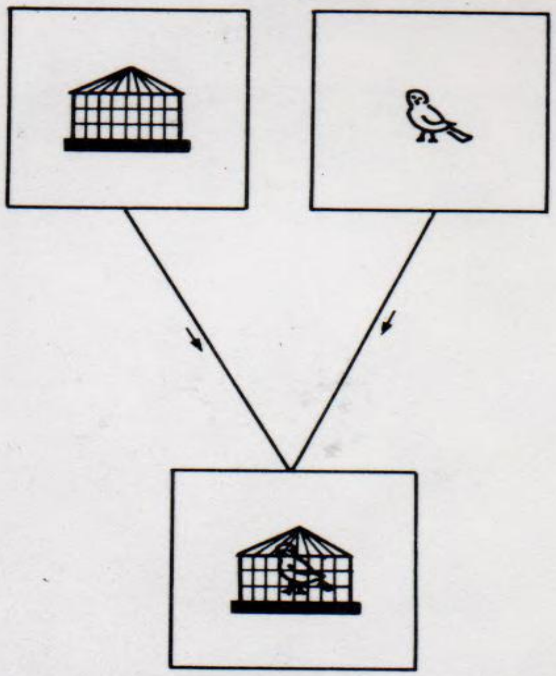


- Grading of binocular vision
- Detection of suppression and ARC
- Measurement of angle

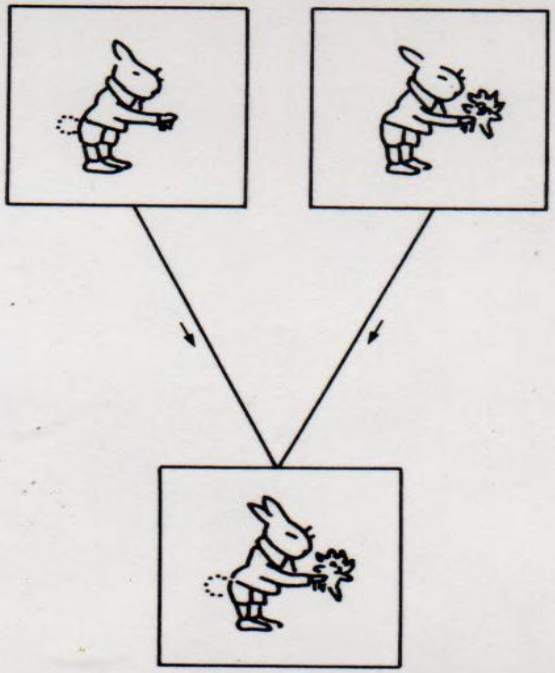




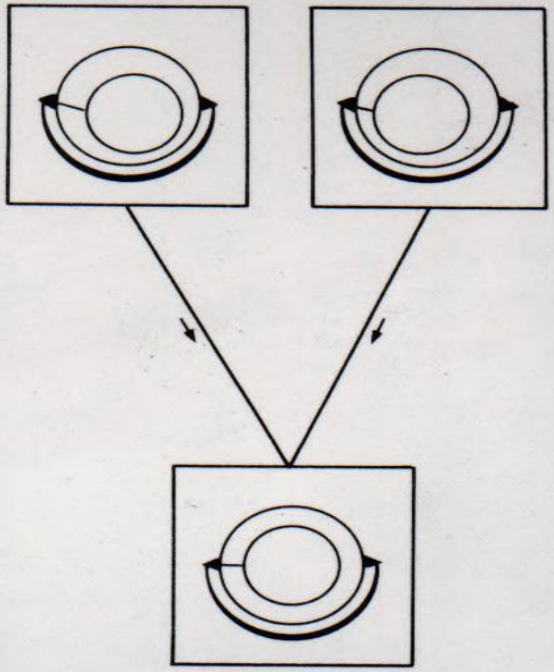




Simultaneous perception



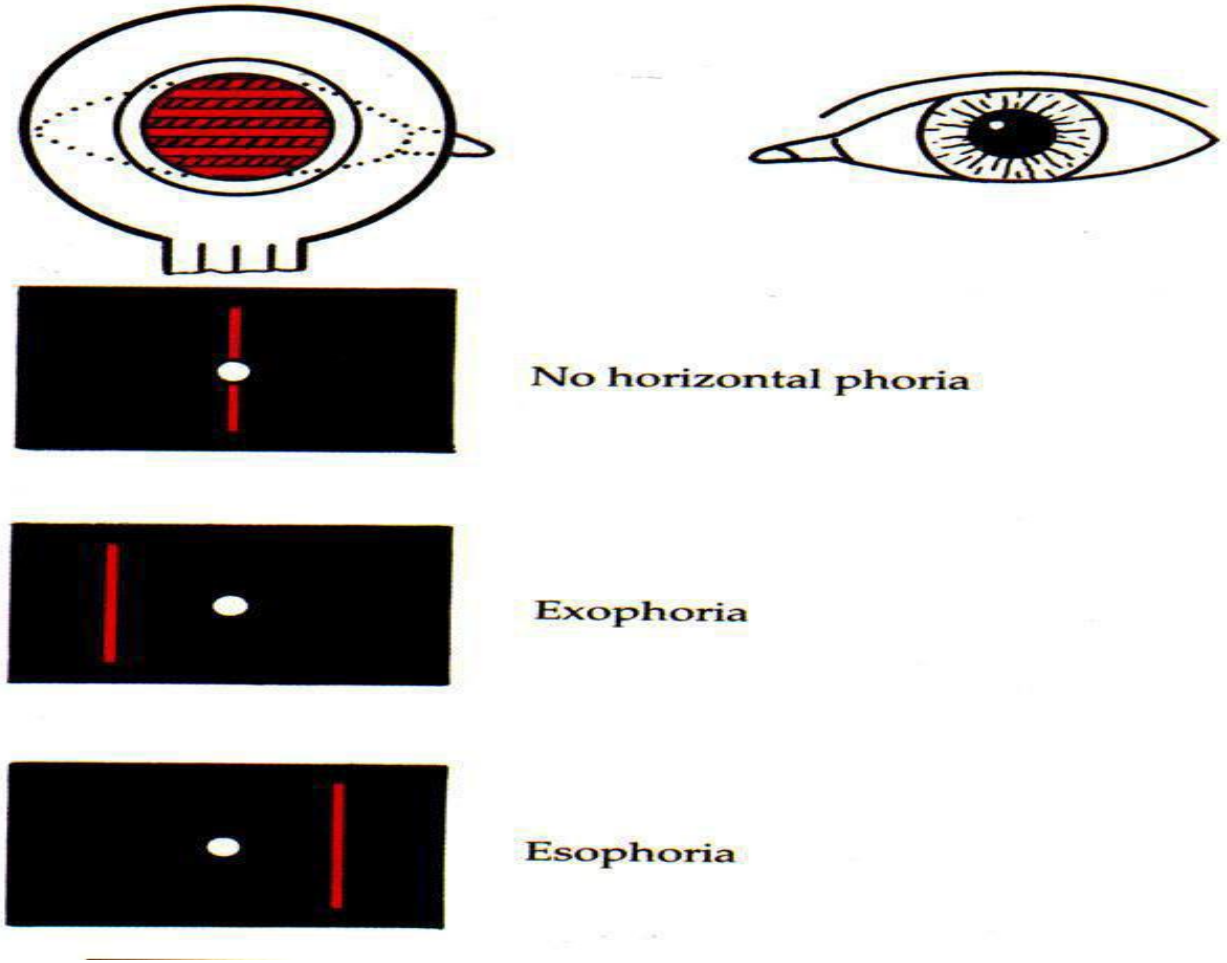
Fusion



Stereopsis

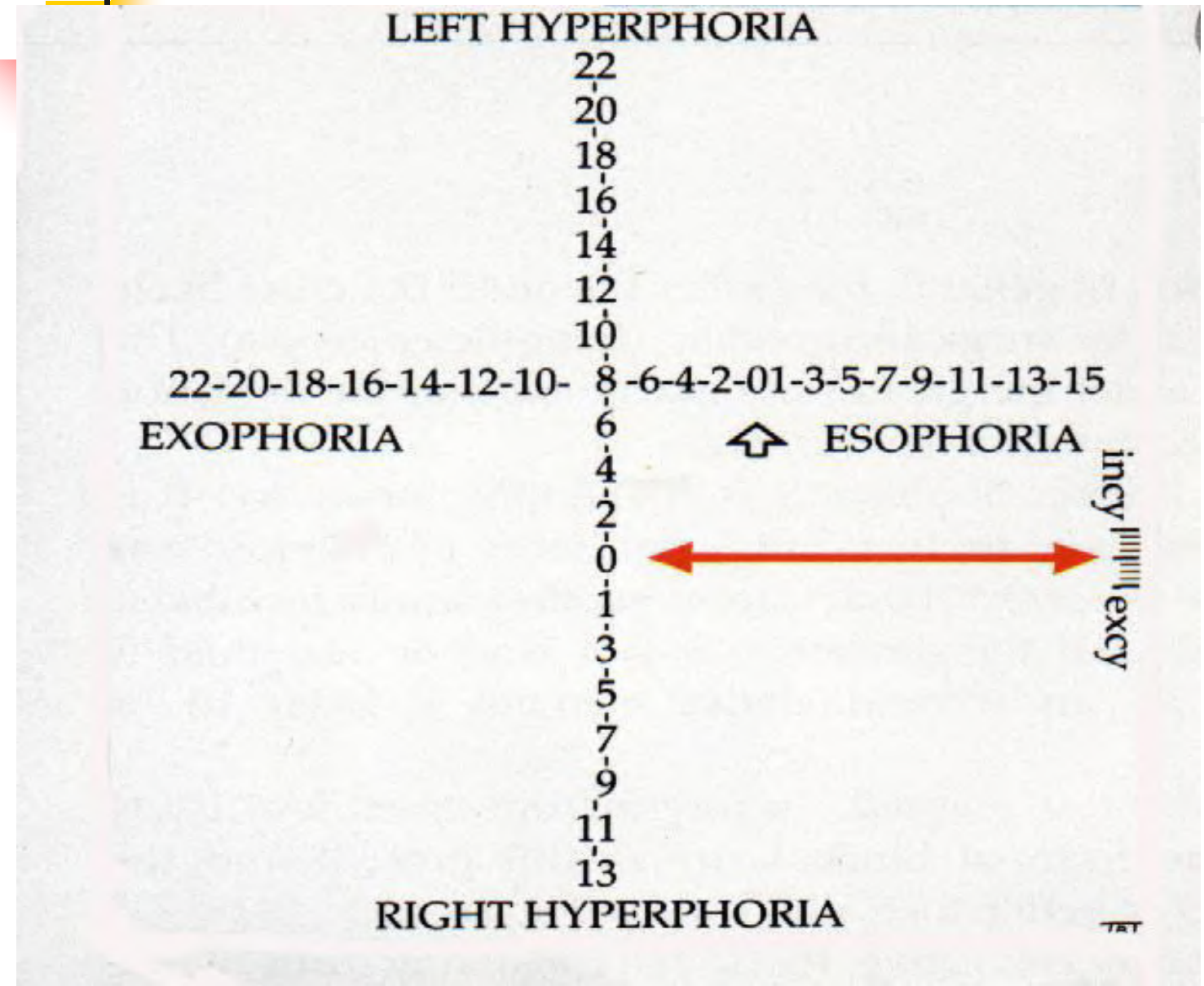
DISSIMILAR IMAGE TEST

MADDOX ROD TEST



Put prisms to measure deviation and the apex should be in direction with the deviation

MADDOX WING TEST

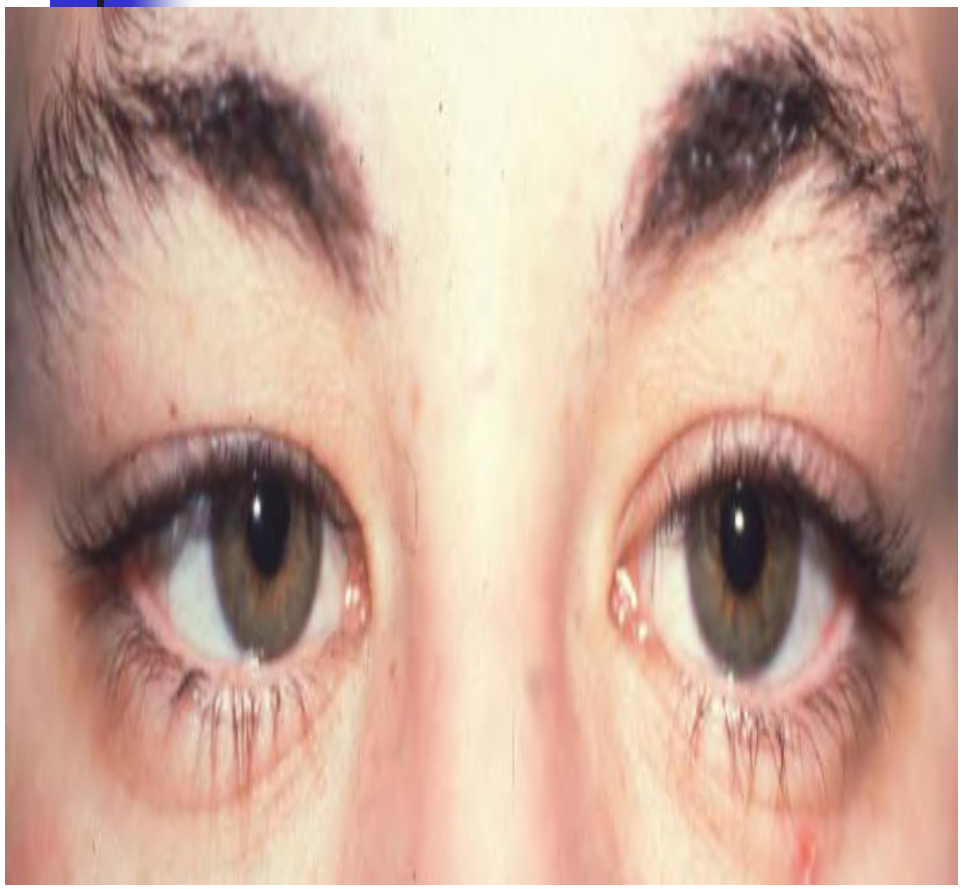


Is done for near vison for phorias

Measurement of deviation

- Hirschberg test
- Krimsky test
- Cover tests

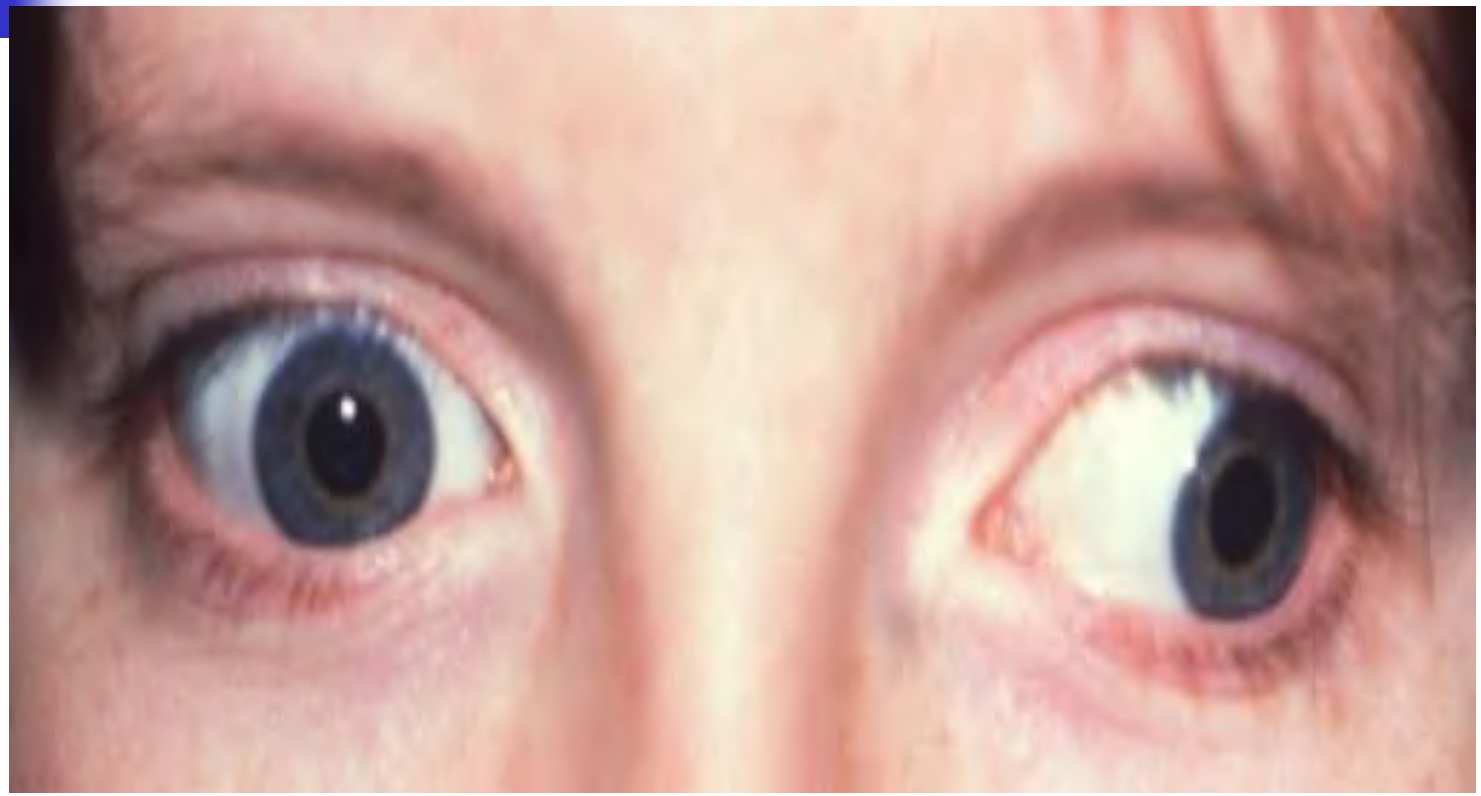
Hirschberg test



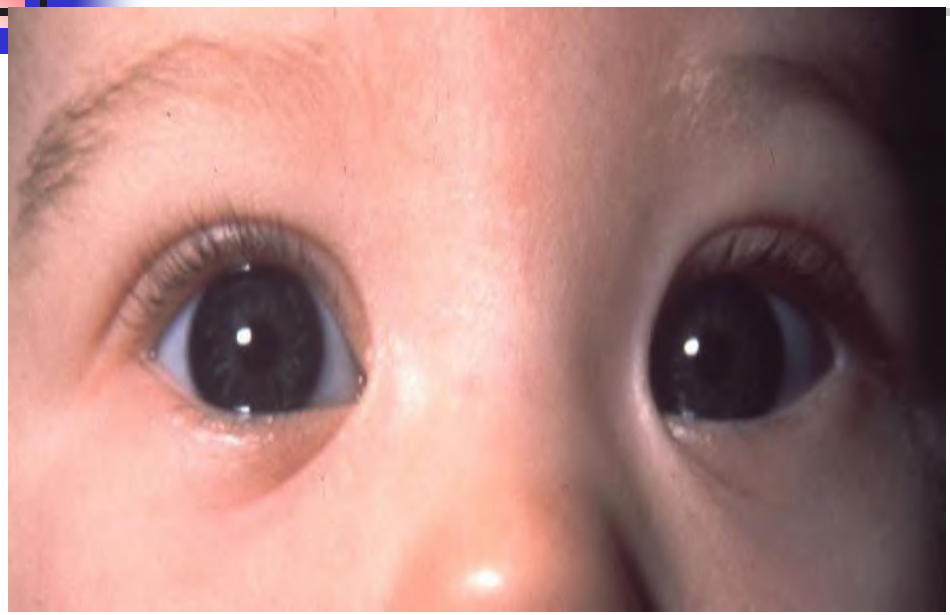
(1 mm = 7° or 15 △

- Objective measurement of the angle
- Note location of corneal light reflex
- Reflex at border of pupil =15°

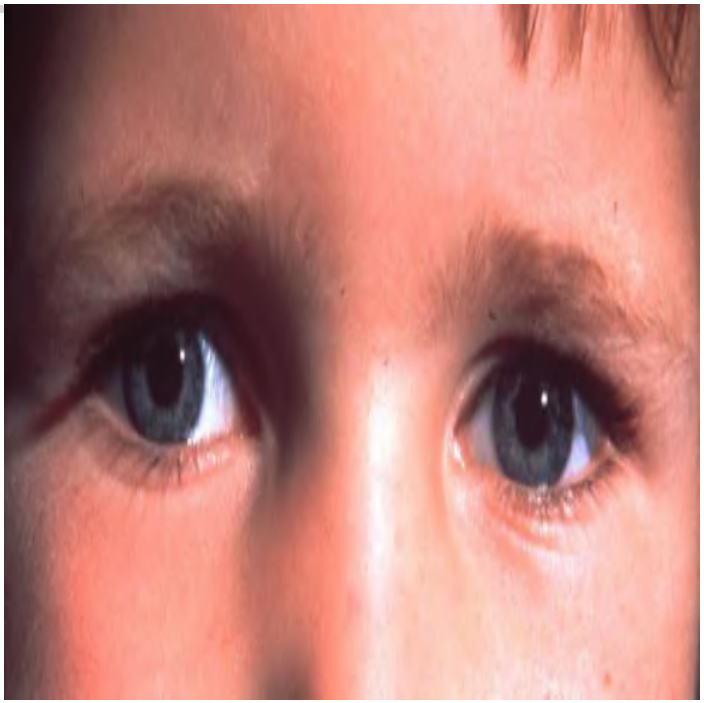
Reflex at the limbus (45° or 90 prism dioptres)



Pseudodeviations



- ***Pseudo esotropia***
- Epicanthic folds
- Short interpupillary distance

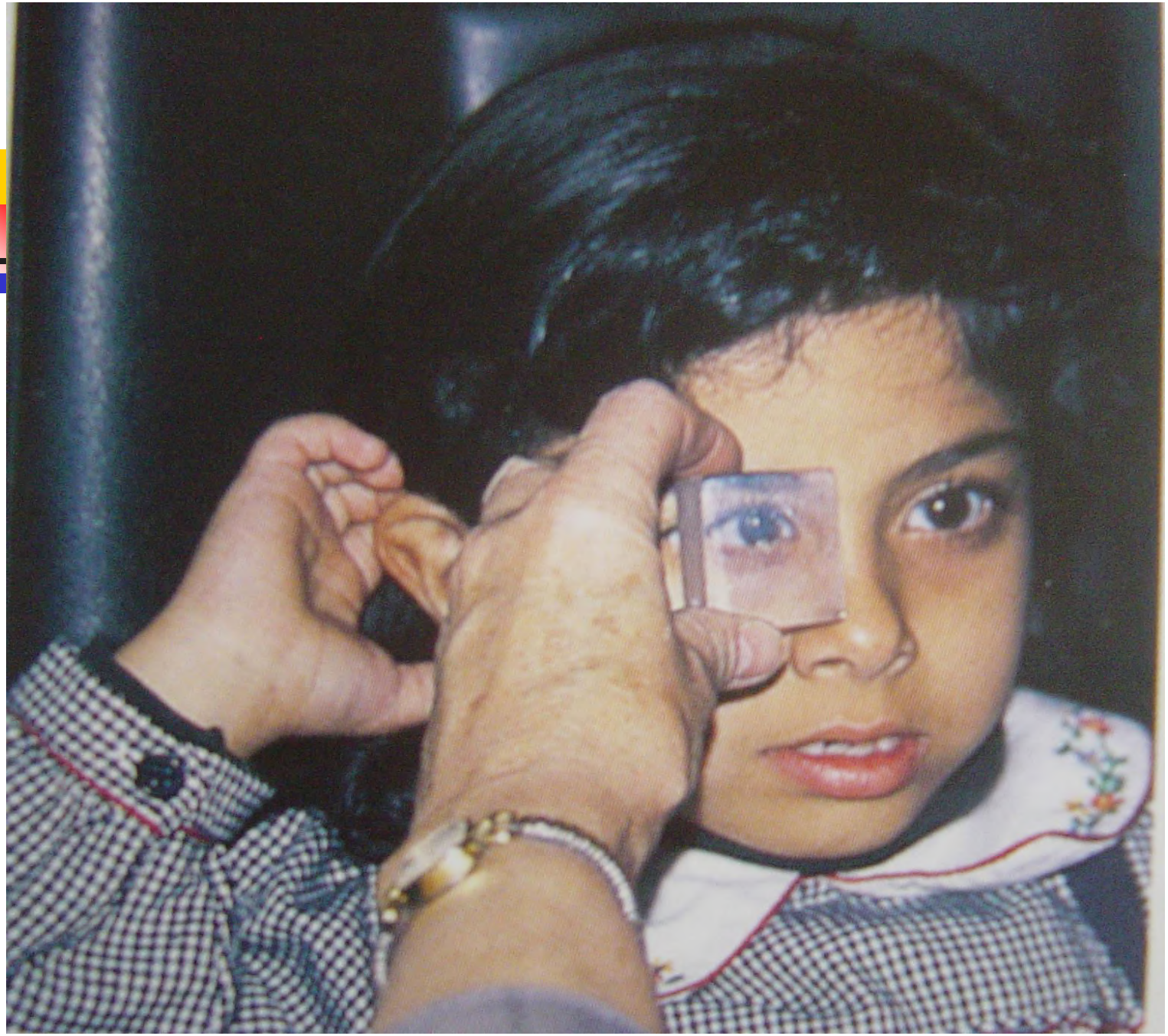


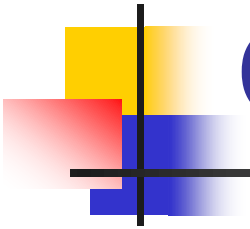
- ***Pseudo exotropia***

  wide interpupillary distance

Krimsky tests

- Prisms are placed in front of the fixating eye till corneal reflexes are symmetrical

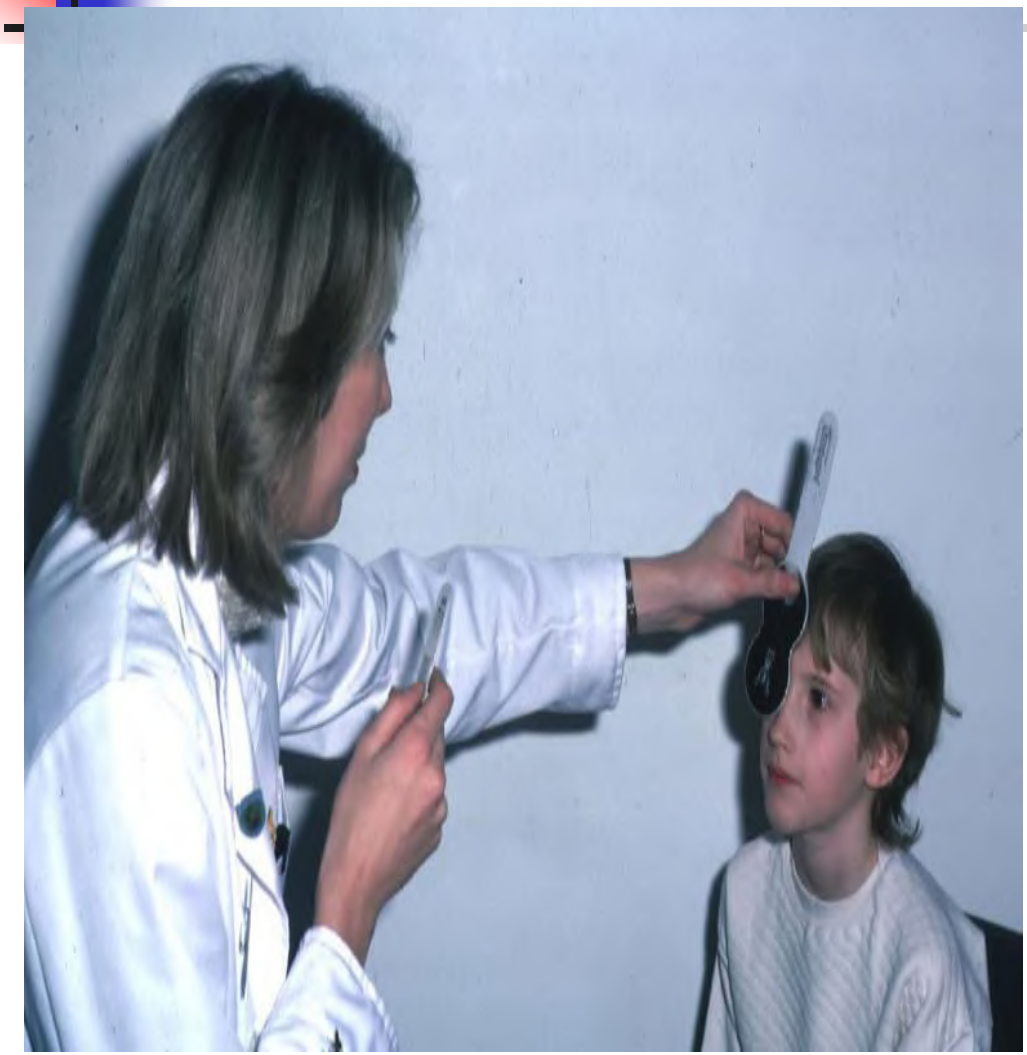




Cover tests

- Cover-uncover test
- Alternate cover test
- Prism cover test

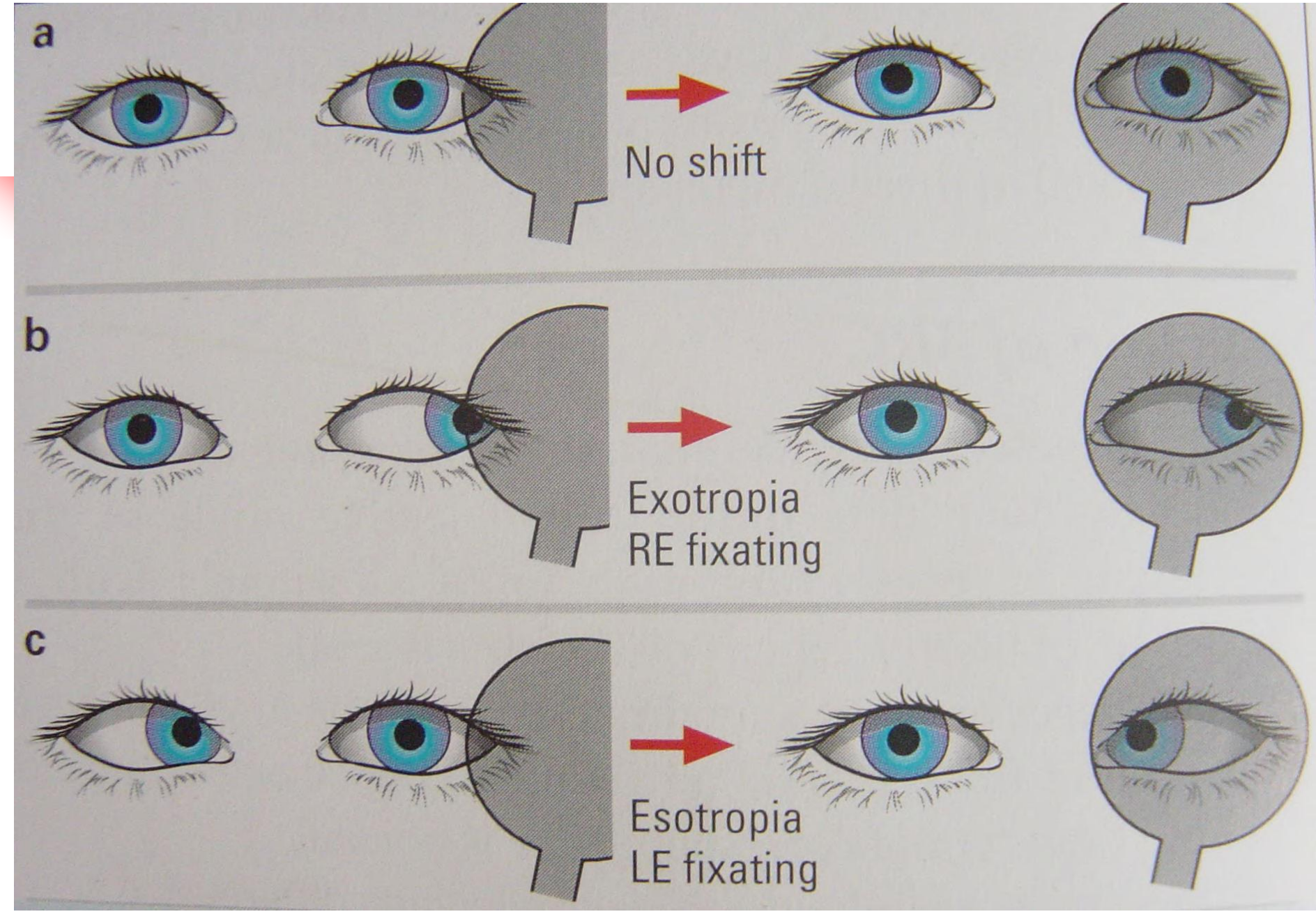
Cover tests



- Most accurate measurement of squint
- Tests are based on ability to fixate
- Differentiate tropia from phorias

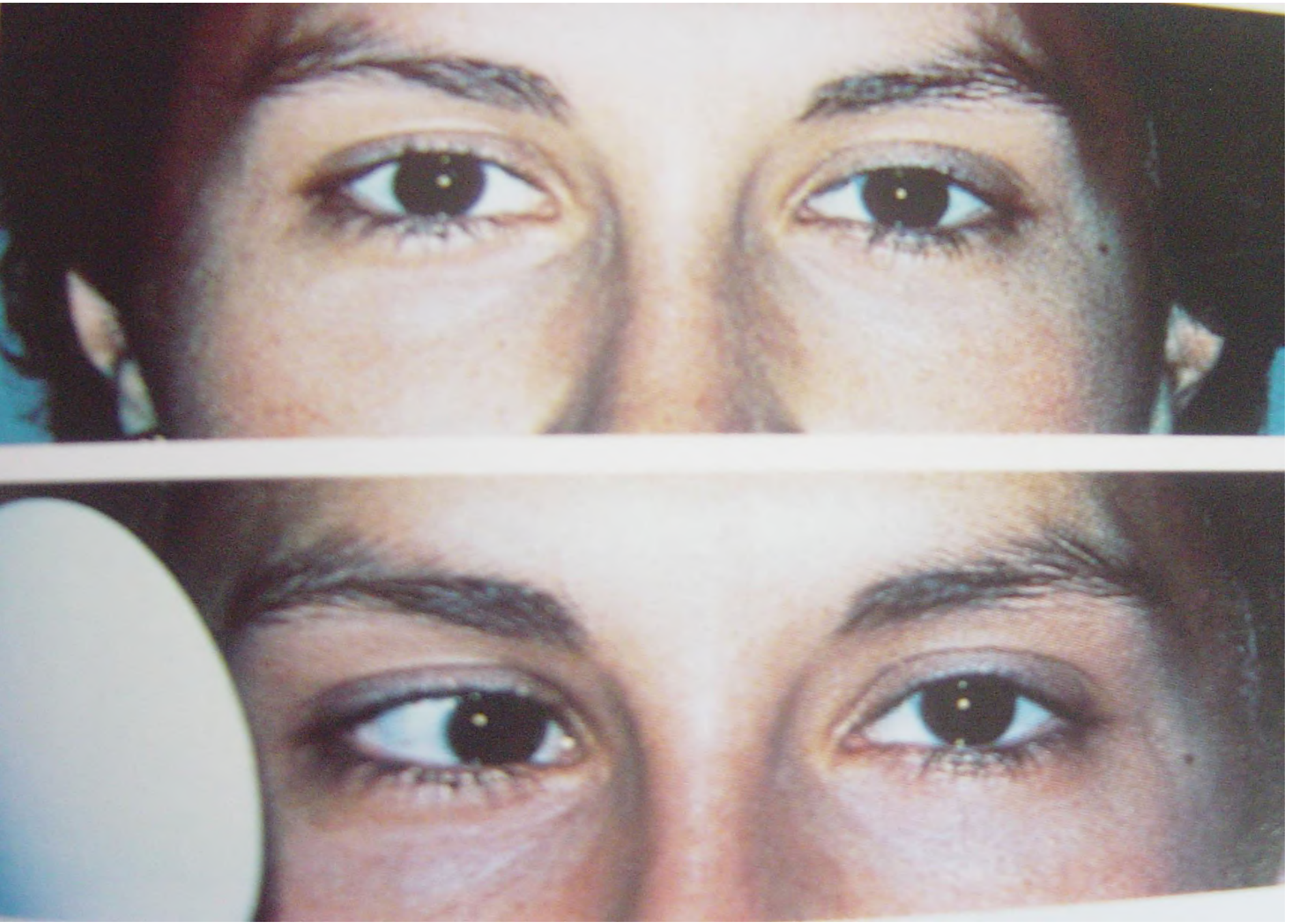
Cover tests

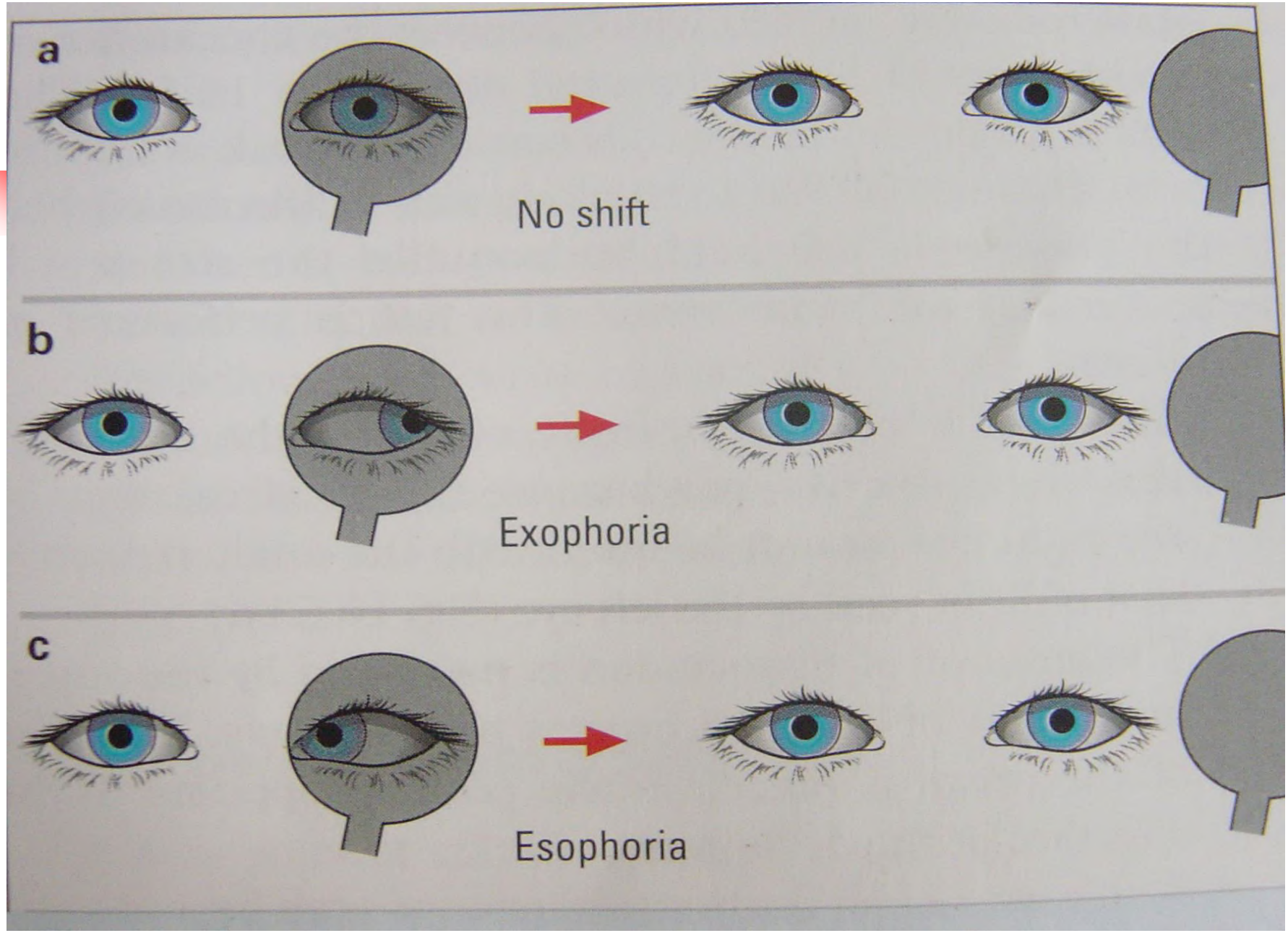
- Detect a tropia
- Performed for near and distance
- Patient looks straight ahead
- If right deviation is suspected, cover the left eye and look at the RE,
- No movement means eye is straight or LE has tropia
- Adduction of RE means exotropia, abduction means esotropia , downward movement means hypertropia and upward means hypotropia


a
No shift
b
Exotropia
RE fixating
c
Esotropia
LE fixating

Un cover test

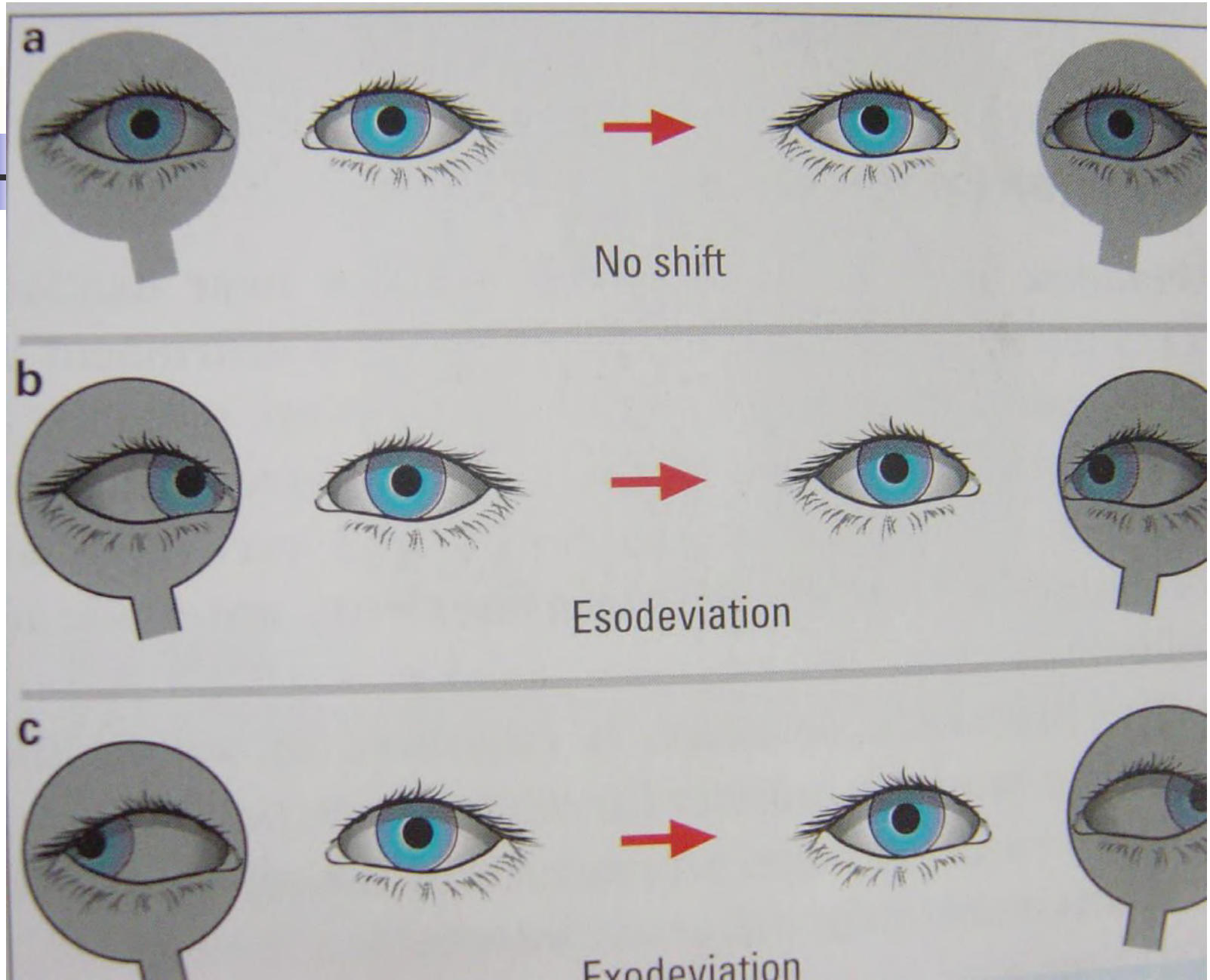
- Detect phoria
- Perform for near and distance
- Cover the RE and after few seconds remove the cover and observe the RE
- No movement means orthophoria, adduction means exophoria, abduction means esophoria




a
No shift
b
Exophoria
c
Esophoria

Alternate cover test (phoria and tropia)

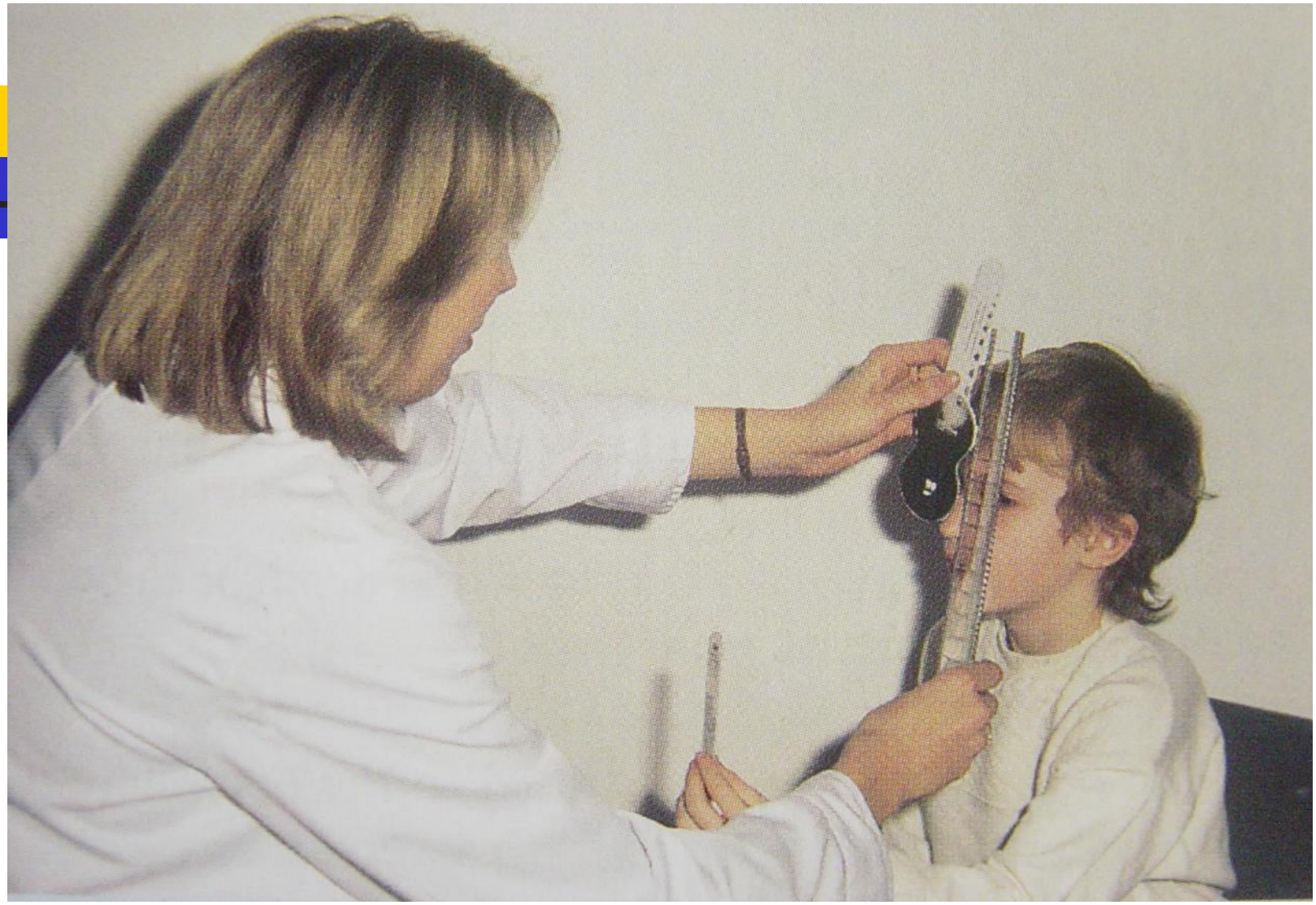
- Cover the RE for 2 sec then the LE for 2 sec and do it several times
- After the cover is removed note the uncovered eye
- A person will phoria will have straight eyes before and after and a person with tropia will have manifest deviation


a
No shift
b
Esodeviation
c
Exodeviation

Prism cover test

- Measures the angle of deviation
- Do ACT
- Place prisms in front of one eye and do ACT till the no ocular movement is seen

(base of prism is opposite to deviation)



Motility tests

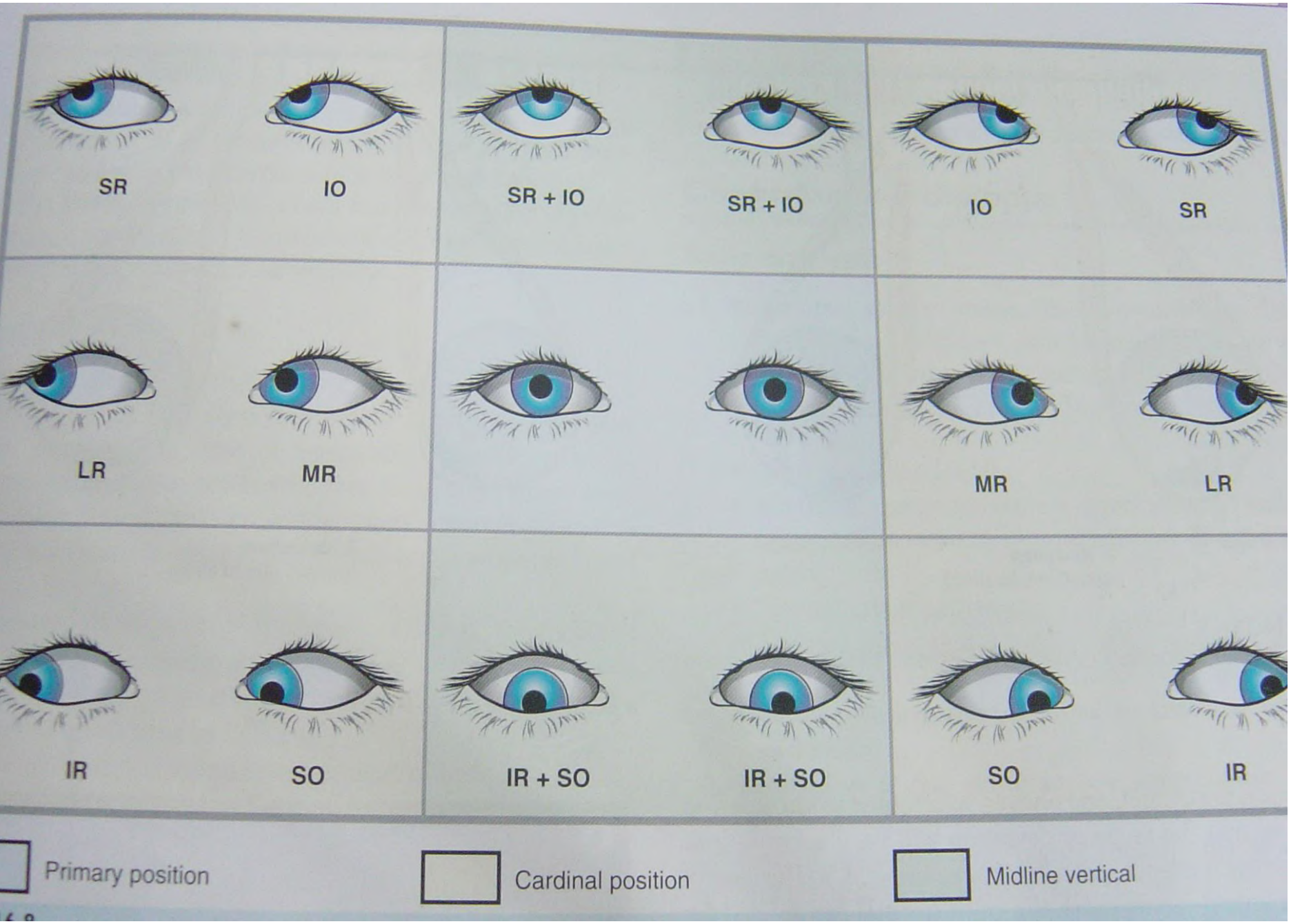
- Smooth pursuit movements
- Saccades

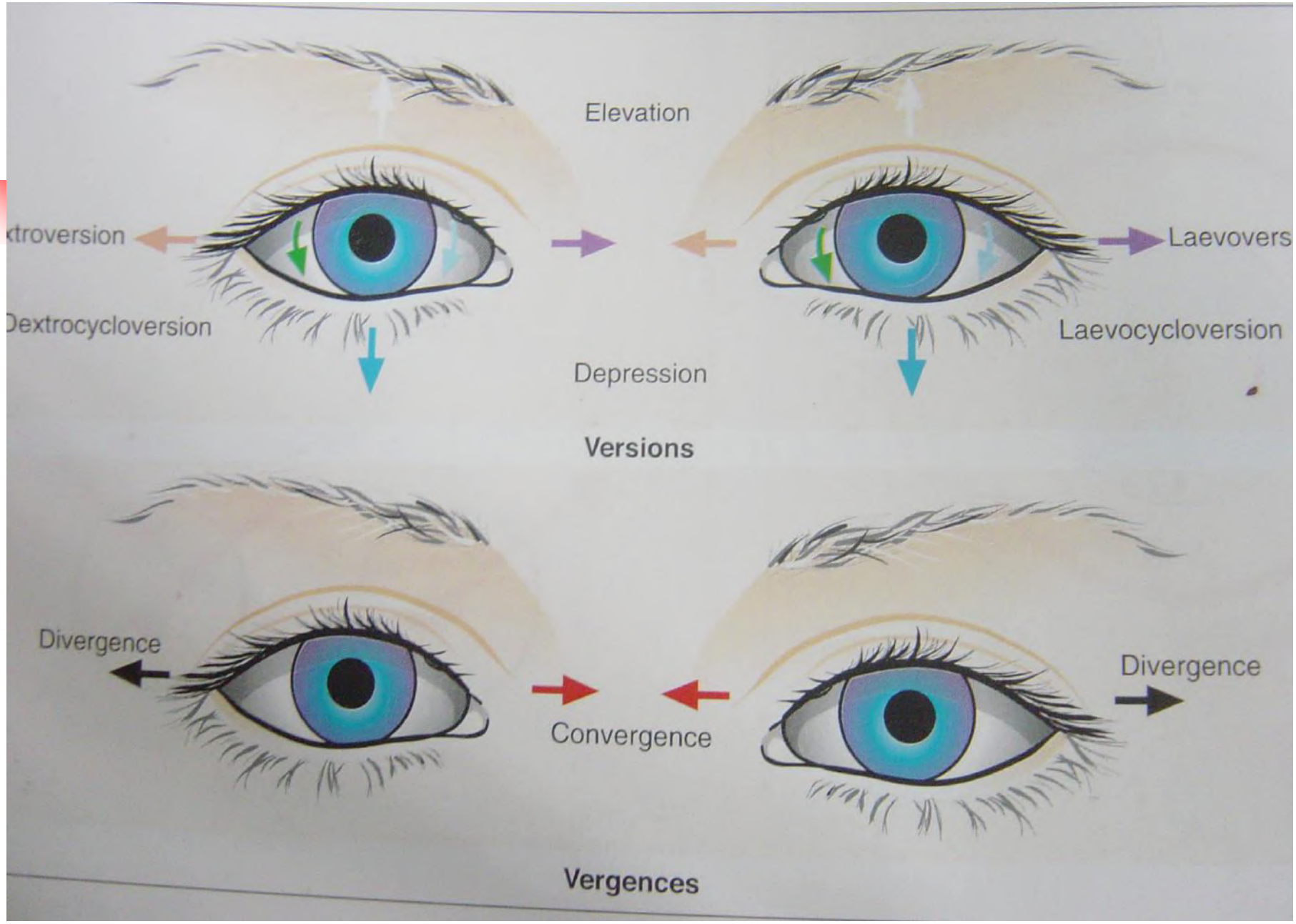
Versions

binocular, conjugate and simultaneous

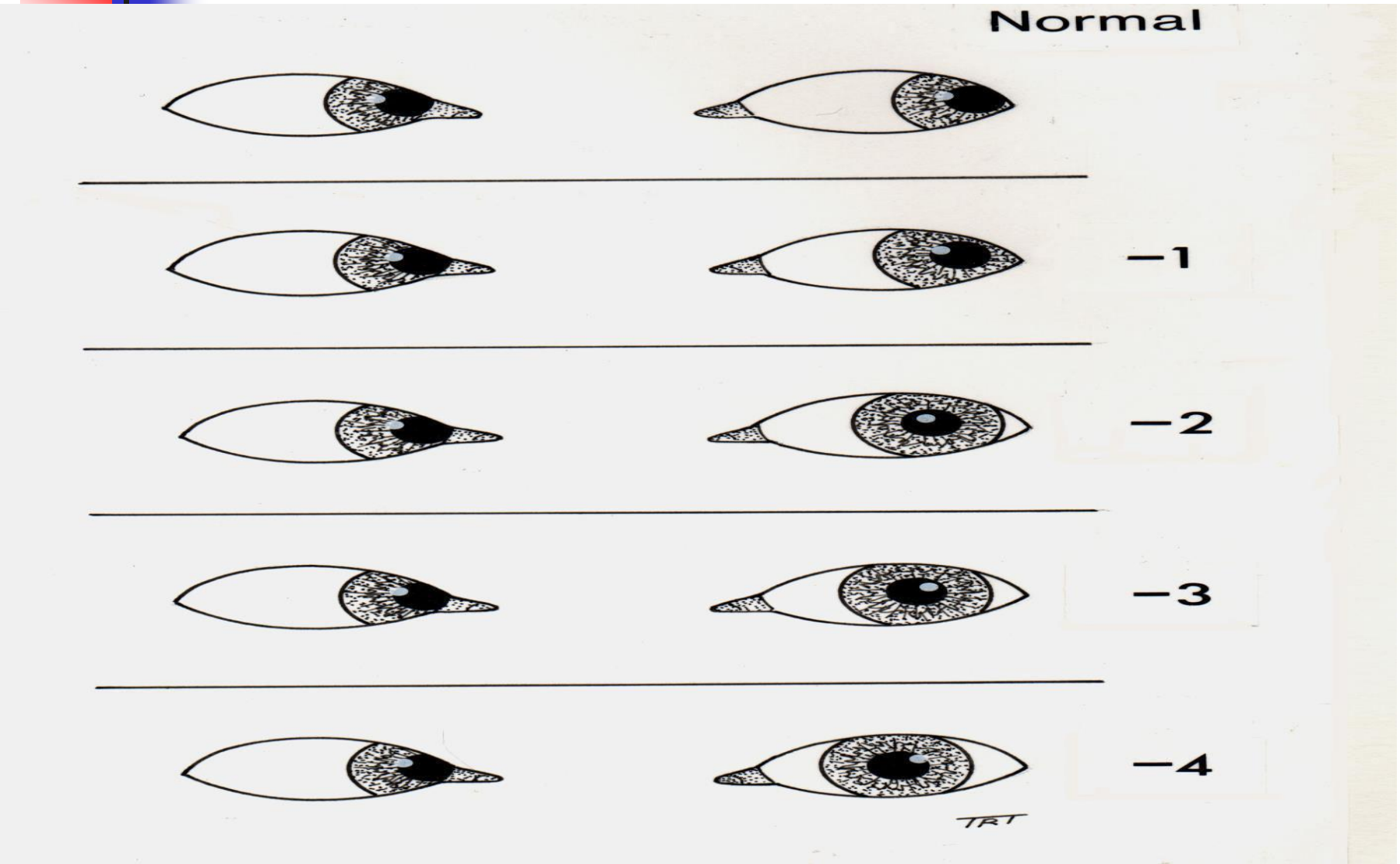
Ductions

monocular movements

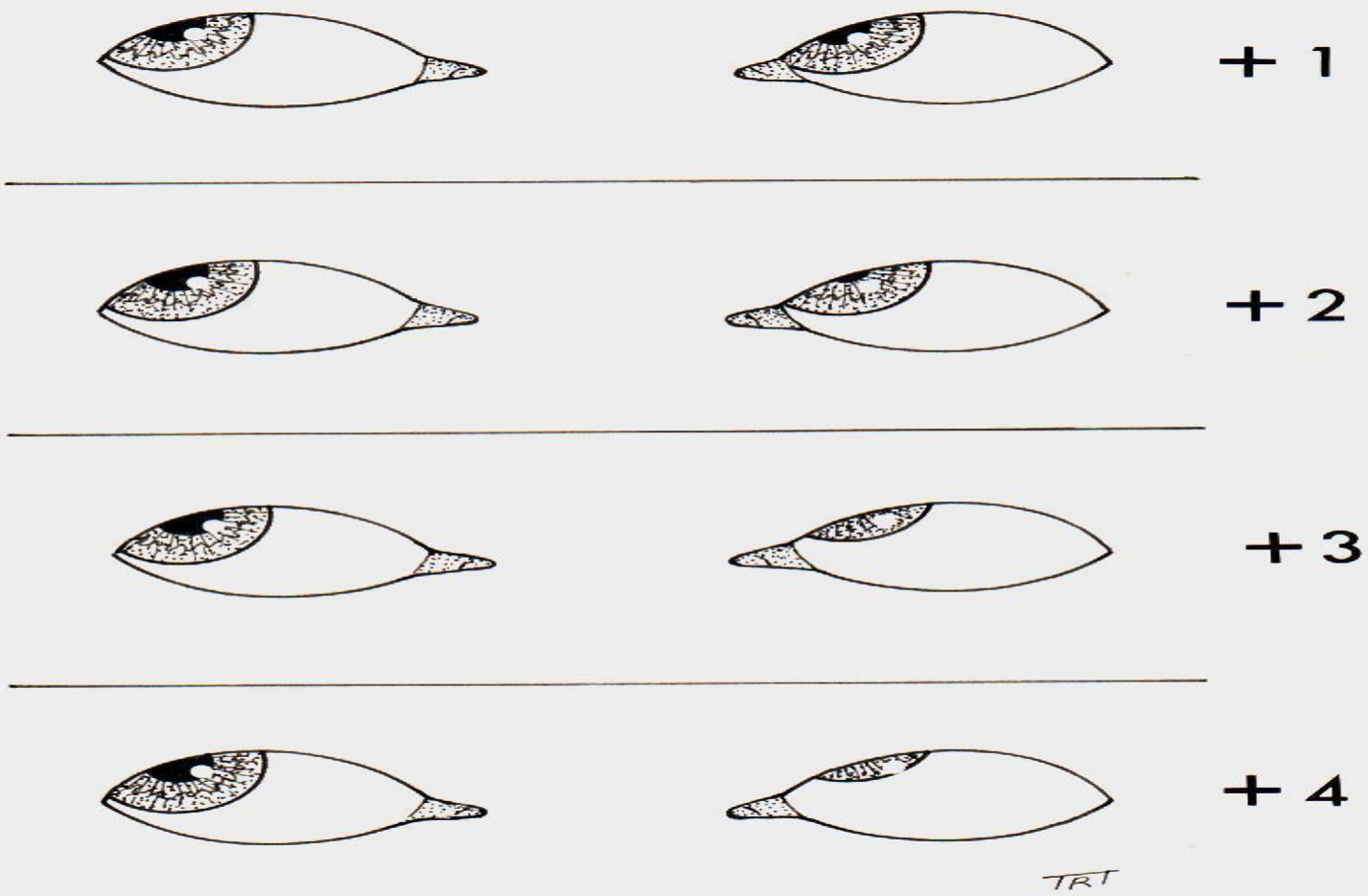

SR
IO
SR + IO
SR + IO
IO
SR
LR
MR
MR
LR
IR
SO
IR + SO
IR + SO
SO
IR
Primary position
Cardinal position
Midline vertical


Elevation
xtroversion
Laevovers
Dextrocycloversion
Laevocycloversion
Depression
Versions
Divergence
Divergence
Convergence
Vergences

Left lateral rectus underaction



Left inferior oblique overaction



Near point of convergence

- Nearest point on which eye can maintain fixation
- NPC is less than 10cm
- Noted when patient reports diplopia
- NPA is 8cm

FUNDOSCOPY

- MANDATORY (NEVER MISS)
- Squint may be due to poor vision as in

  macular scarring

  optic disc hypoplasia

  retinoblastoma (20%)

Cycloplegic refraction

- Commonest cause of squint is refractive errors
- Cycloplegia is done to negate the effect of accomodation
- Cyclopentolate 1% , 0.5%
- Atropine

TYPES OF STRABISMUS

CONCOMITANT

INCOMITANT

TERMINOLOGY

Strabismus- from Greek word "strabismos"- to look obliquely- ocular misalignment

- Orthophoria- ideal condition of ocular balance

  both eye remain aligned in all positions of gaze and all distances of the fixation point even when the fusional mechanism is disrupted

TERMINOLOGY

- Comitant (concomitant): the deviation does not vary with direction of gaze or fixating eye

- Incomitant (non-comitant); the direction varies with direction of gaze or fixating eye

  Mostly paralytic or restrictive

CONCOMITANT STRABISMUS

The variability of angle of deviation is within 5 prism dioptres in different direction of gaze

In an Incomitant squint the angle differs in various positions of gaze as a result of

Abnormal innervation

Restriction

TYPES OF CONCOMITANT STRABISMUS

- ESOTROPIA
- EXOTROPIA

ESOTROPIA (Manifest convergent squint)

- Accommodative
- Non-accommodative

AC/A RATIO

This is the amount of convergence measured in prism dioptres per unit change in accommodation

- Normal value is 3-5 PD
- One dioptre of accommodation is associated with 3-5 PD of accommodative convergence
- Abnormalities of AC/A ratio play an important role in aetiology of strabismus

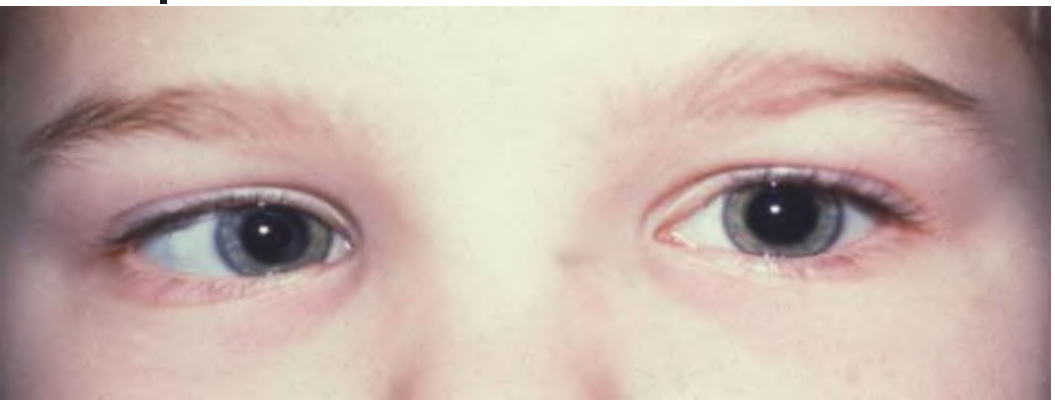
Accommodative

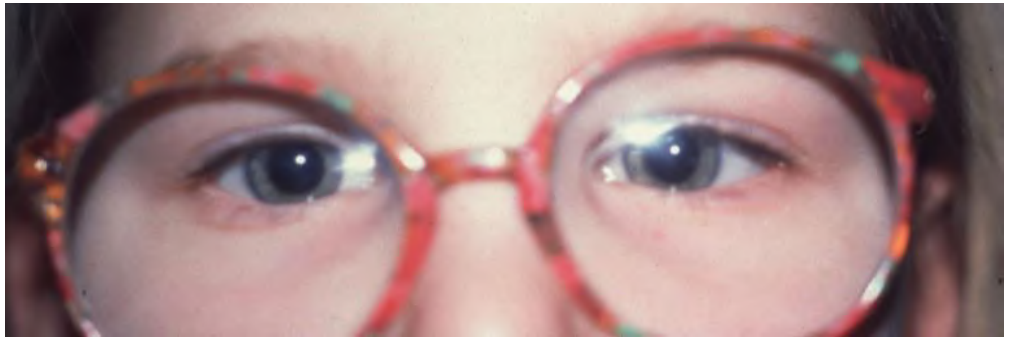
- Refractive
- Non-refractive
- Mixed accommodative

Refractive accommodative esotropia

- Presents between 18 months - 3years
- Initially intermittent
- Normal AC/A ratio
- Excessive hypermetropia and esotropia is a physiological response to it
- Hypermetropia is between +4 and +7 D
- Accommodation is accompanied by convergence which is beyond the divergence amplitude

FULLY ACCOMODATIVE





- Completely corrected by optical correction of hypermetropia,

While partially accommodative is reduced but not eliminated by correction of Hypermetropia

Non refractive accomodative esotropia (18 months to 3years)

- High AC / A ratio
- No refractive error

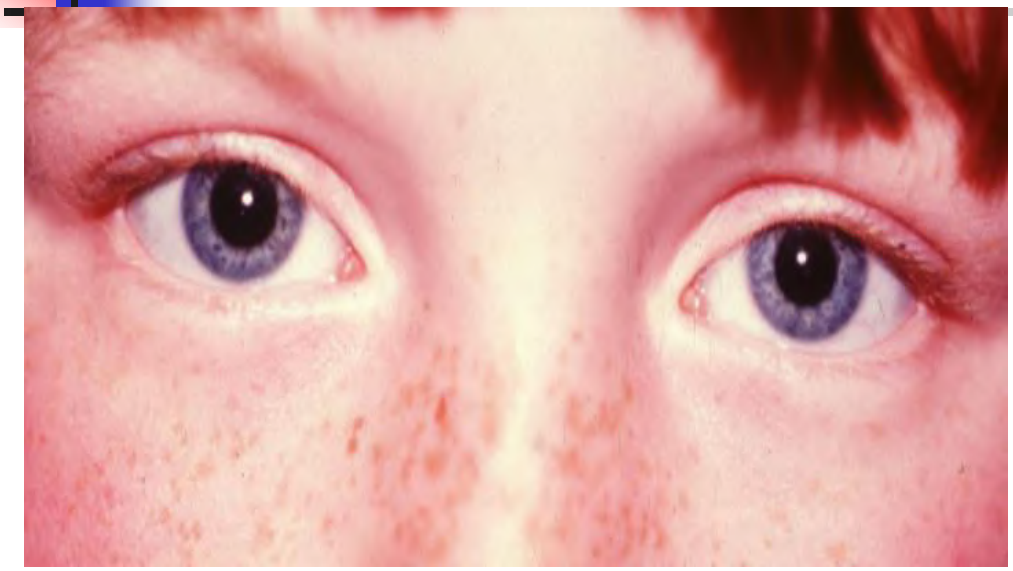
Convergence excess

High AC /A is due to increased AC

Defective accommodation

High AC /A is due to high A

Non-refractive accommodative esotropia



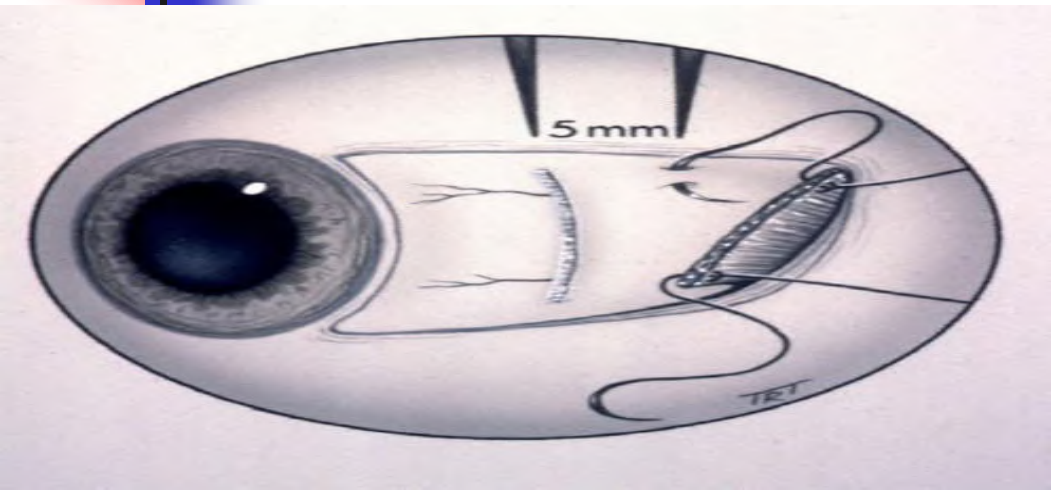
- Straight for distance

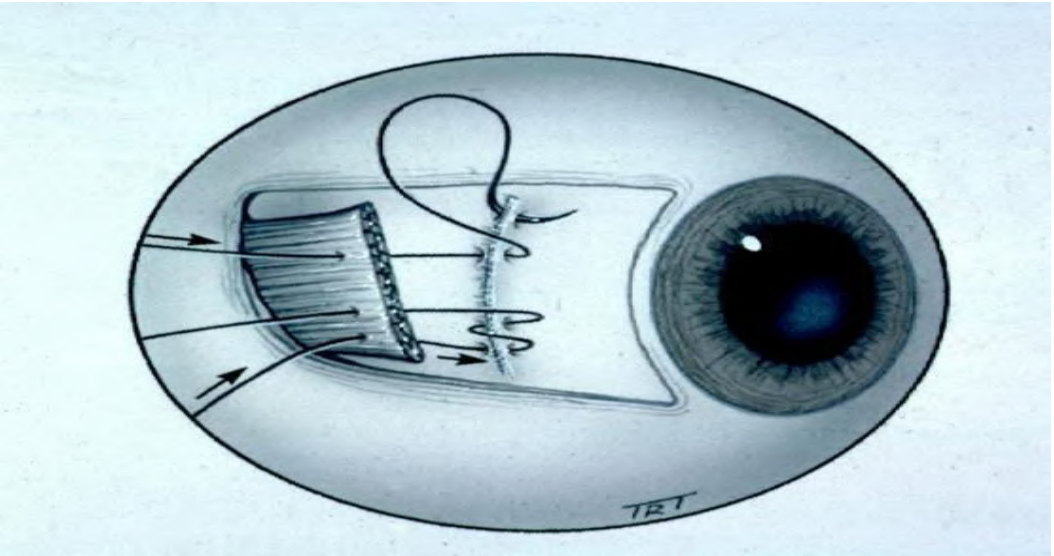
- Squint for near

Management of accommodative esotropia

- Correction of refractive error
- Miotic therapy (induce peripheral A, so less accommodative effort is needed by the patient
- Amblyopia treatment

SURGERY





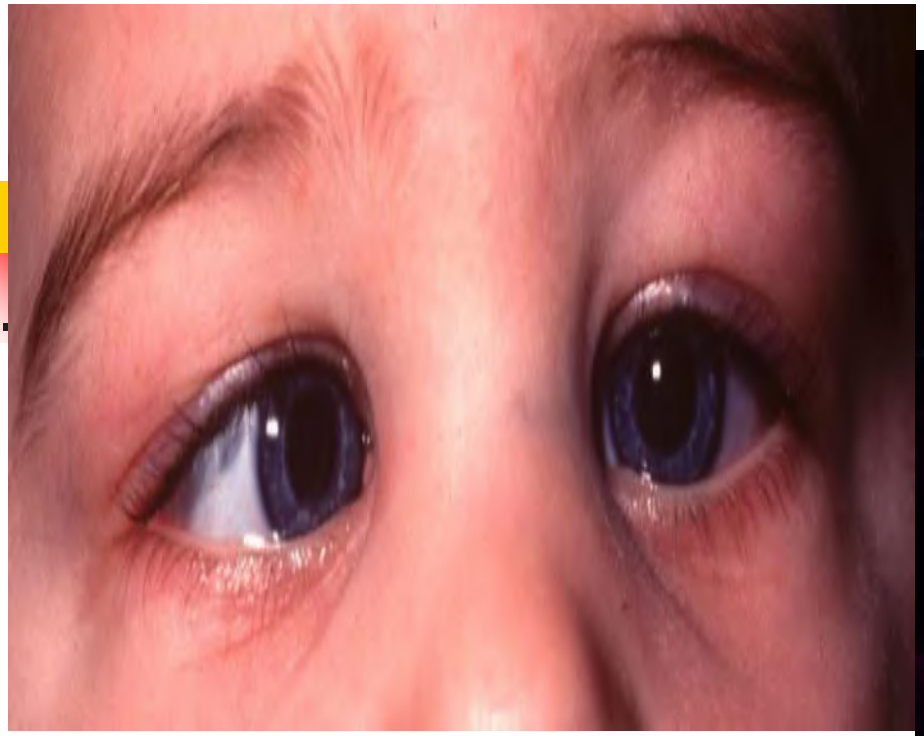
- When specs cant correct deviation
- Weaken the MR and strengthen the LR

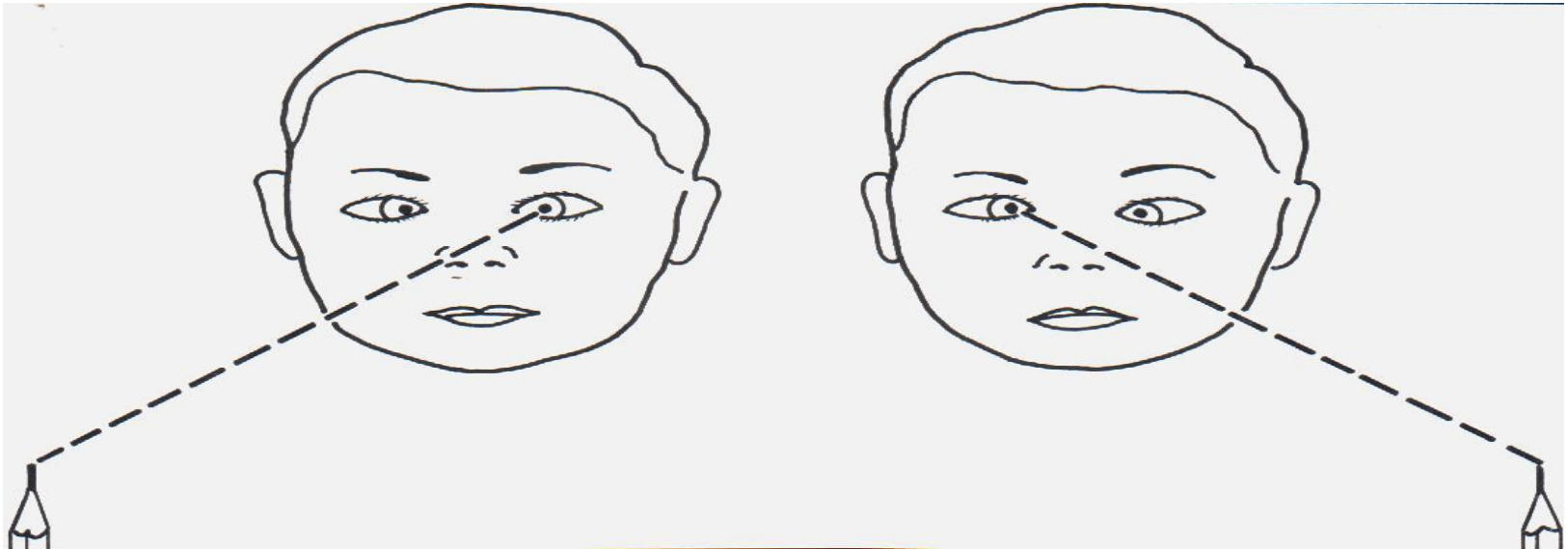
Non accomodative Esotropia

- Essential infantile
- Microtropia
- Basic
- Convergence excess
- Convergence spasm
- Divergence paralysis / insufficiency
- Consecutive
- Acute - onset

NON-ACCOMMODATIVE

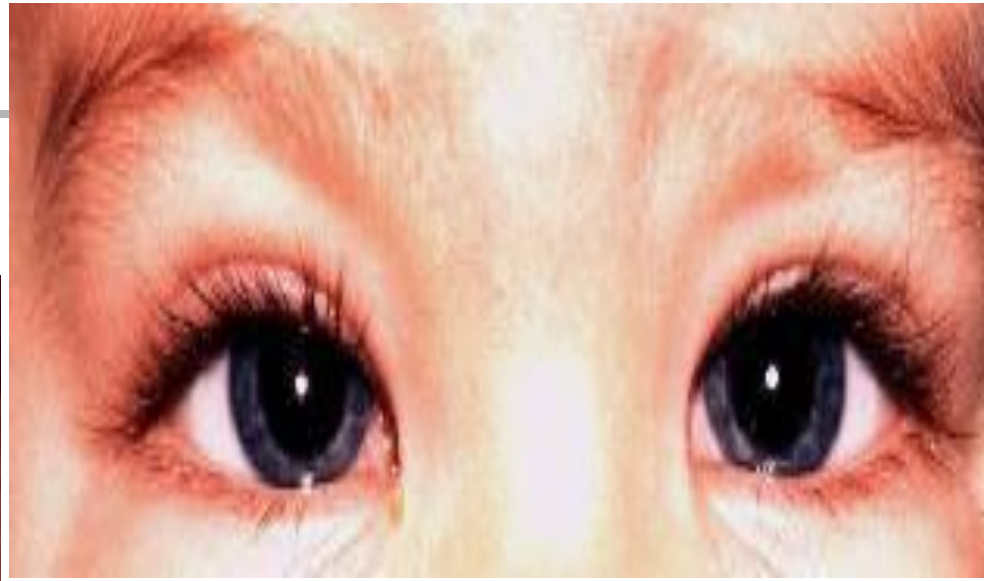
- **Essential infantile esotropia**
- Presents within first 6 months
- Angle large and stable
- Normal refraction for age
- Nystagmus in some cases
- Poor potential for BSV
- Amblyopia in about 30%

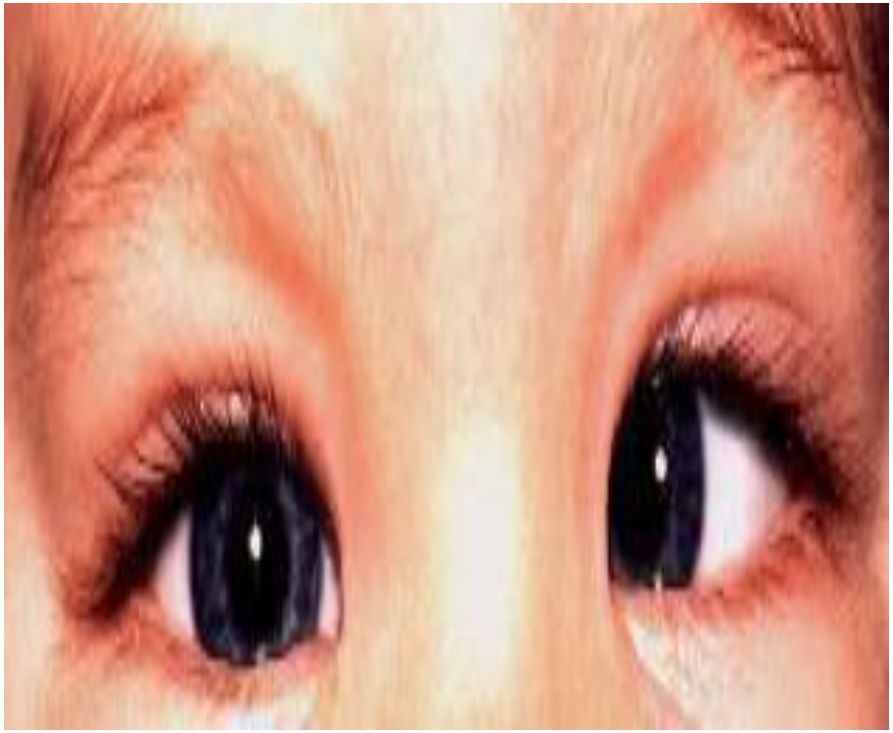




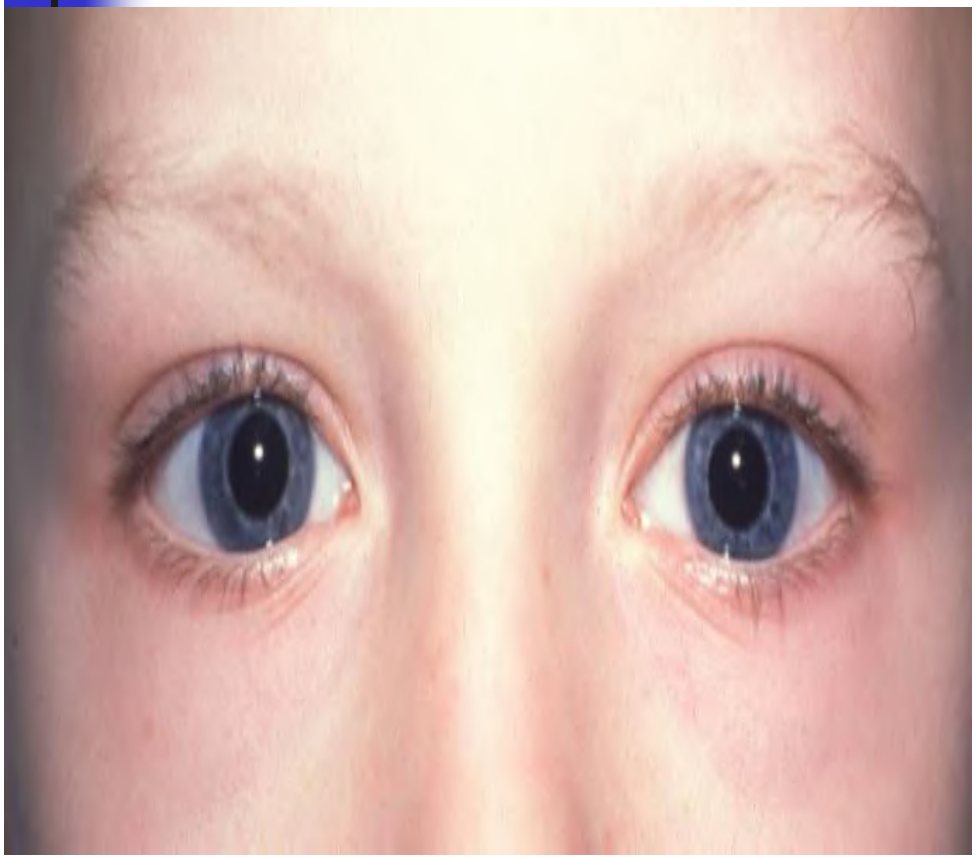
Management of essential infantile esotropia

- Correct amblyopia if present
- Surgery before age 12 months
- Bilateral medial rectus recessions





Microtropia



- Very small angle - may not be detectable on cover testing
- Anisometropia
- Central suppression scotoma

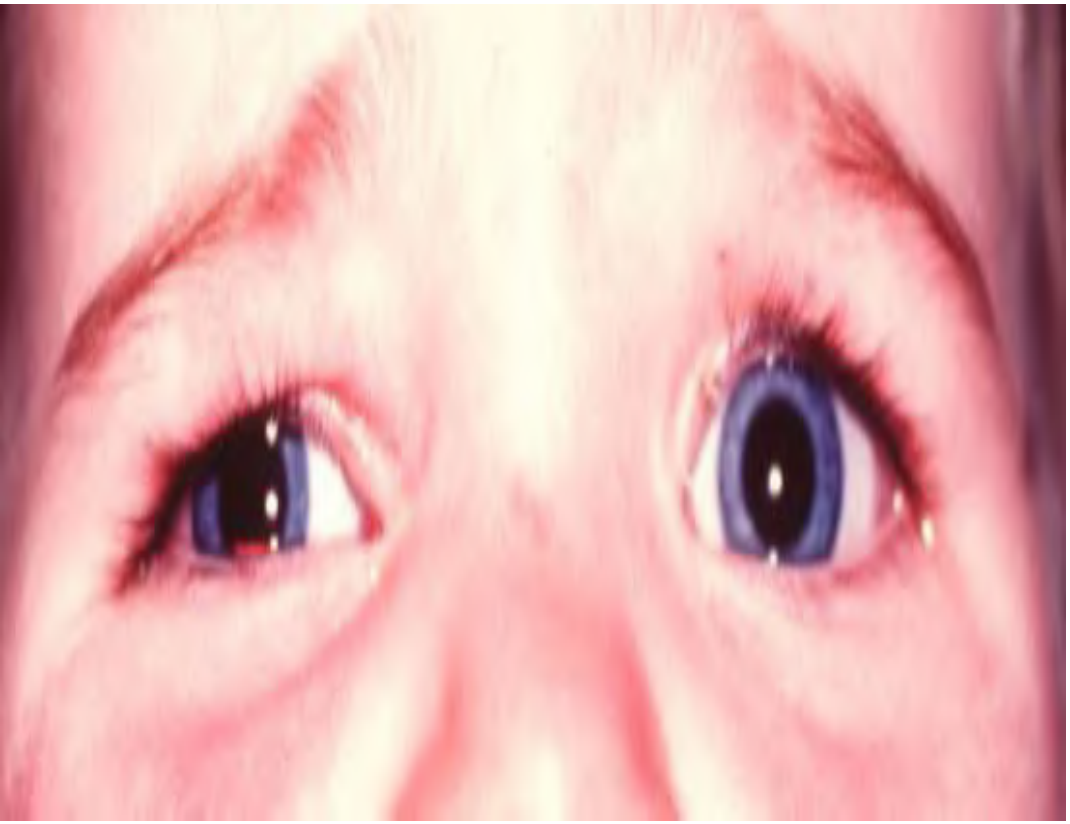
Exotropia (manifest divergent squint)

- CONSTANT
- INTERMITTENT

CONSTANT

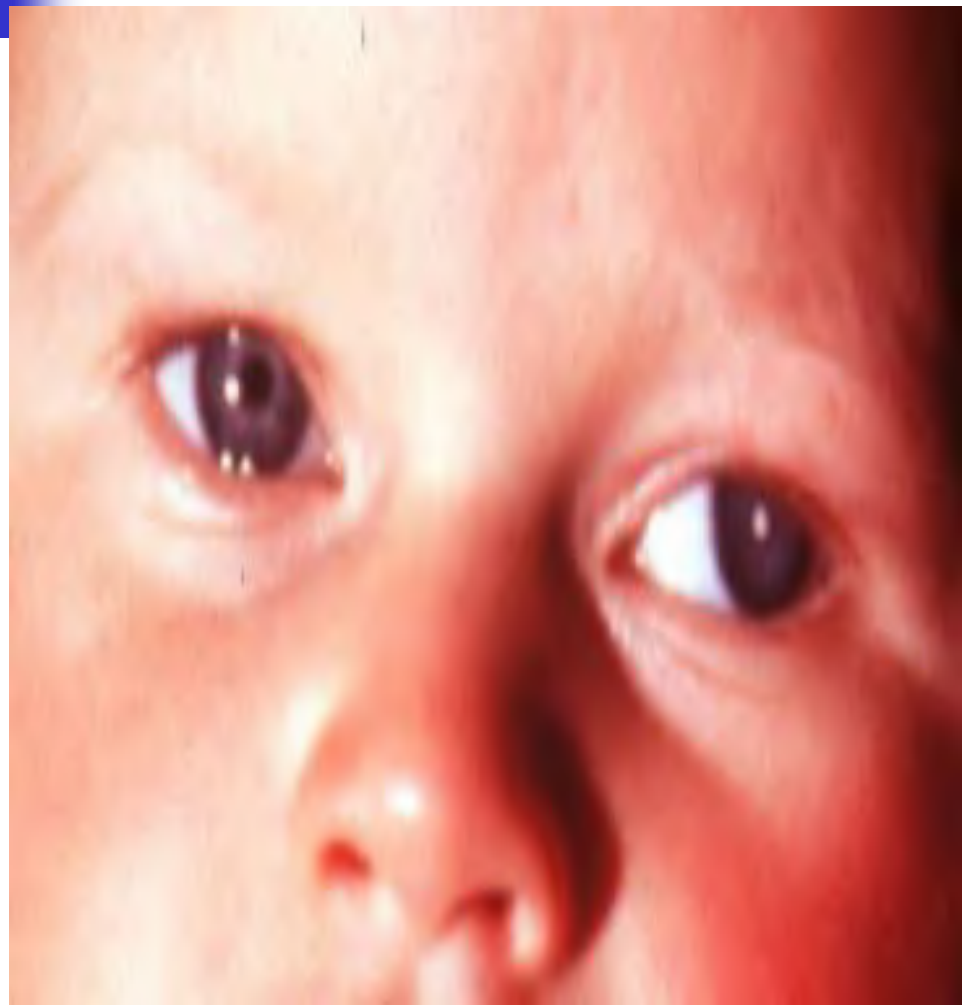
- Congenital
- Sensory
- Consecutive

Congenital



- Presents at birth
- Large angle
- Alternating fixation
- Normal refraction for age
- Neurological anomalies may be there
- Treatment is surgical

SENSORY



Disruption of binocular reflexes by

acquired lesions, such as cataract in over 5 years and in adults

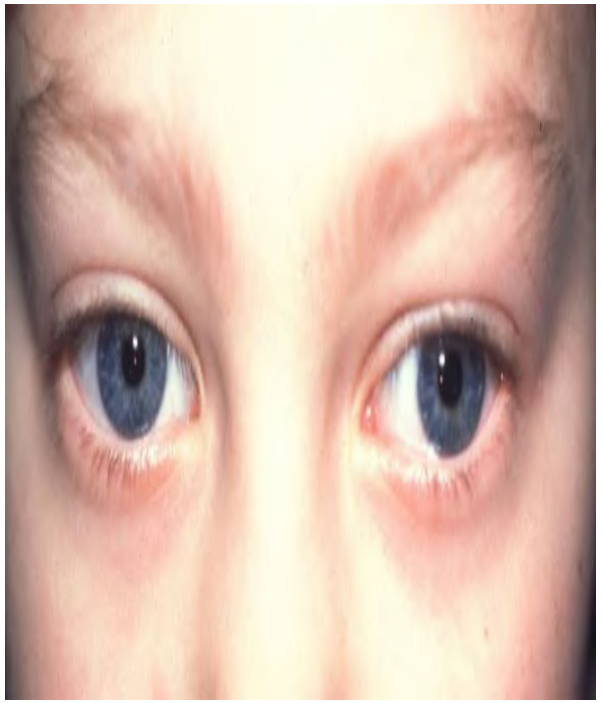
Correct the visual deficit followed by surgery

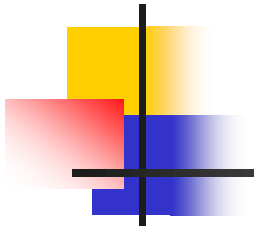
Consecutive

follows previous surgery for esotropia

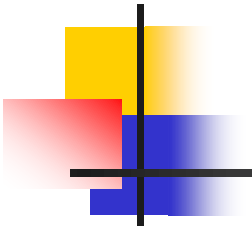
Intermittent (around 2 years)

- Basic
- Convergence weakness
- Divergence excess





- When exophoria breaks under visual inattention, bright light, fatigue or ill health



- BASIC

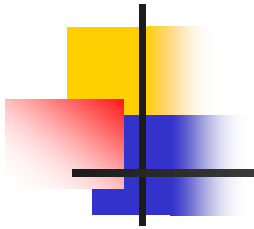
  Deviation is same for distance and near

- Convergence weakness

  adults or older kids

  angle more for near

  may be associated with Myopia



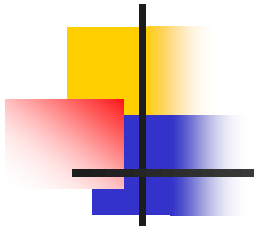
- Divergence excess
  angle is more for distance

management

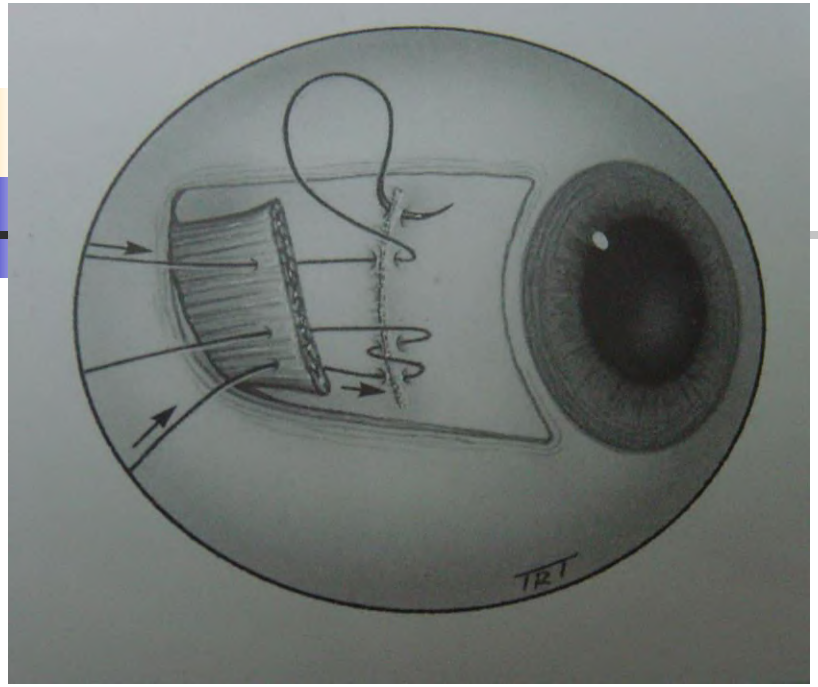
- Correct myopia
- Orthoptic treatment
- Surgery

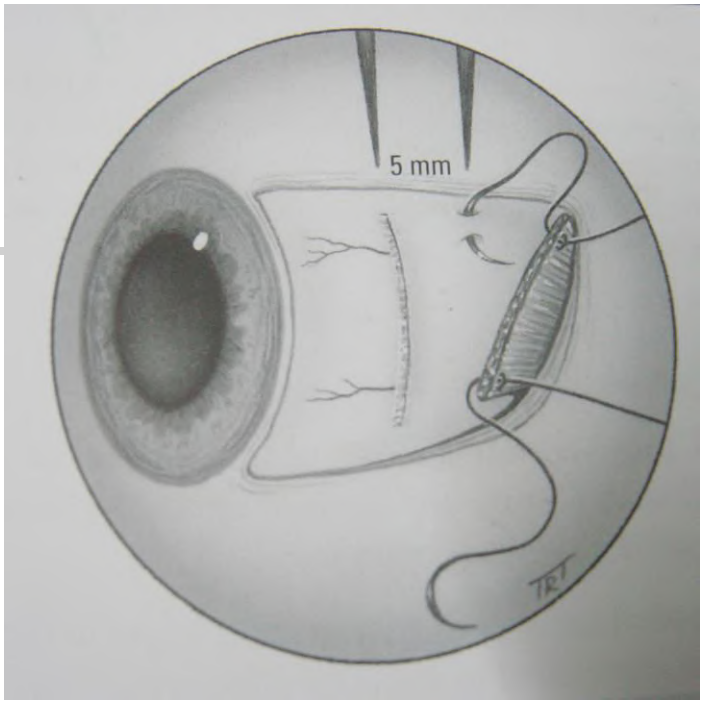
PRINCIPLES OF SURGERY

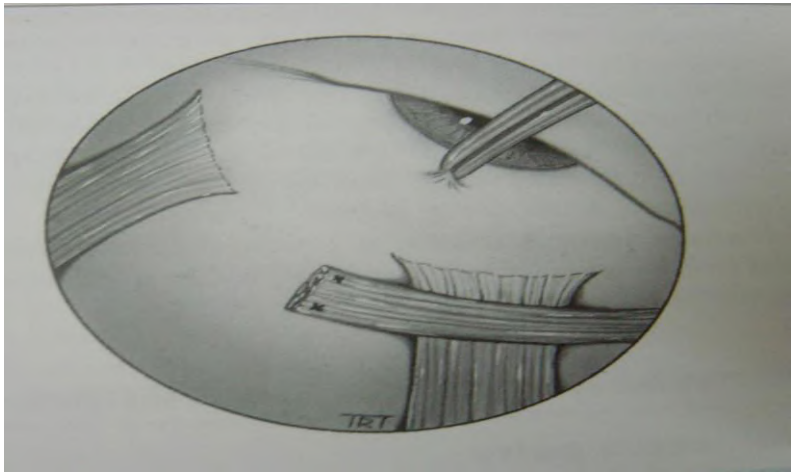
- Weakening procedures
  - Recession
  - Myectomy
  - Posterior fixation suture



- Strengthening procedures
  - Resection
  - Advancement
  - Tucking


TRT


5 mm
TRT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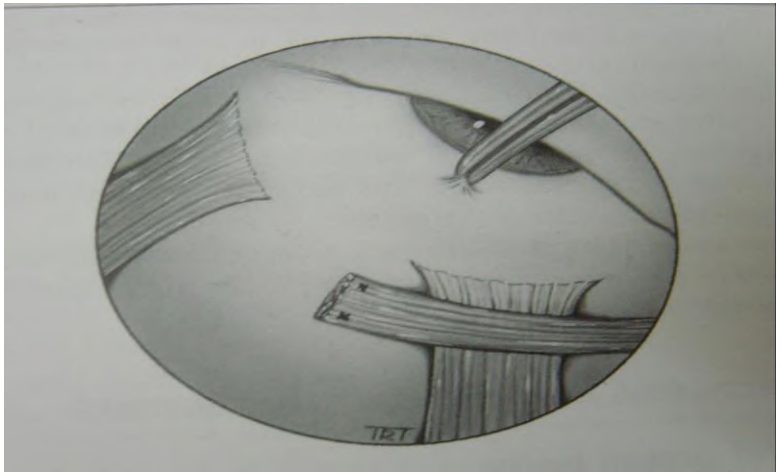
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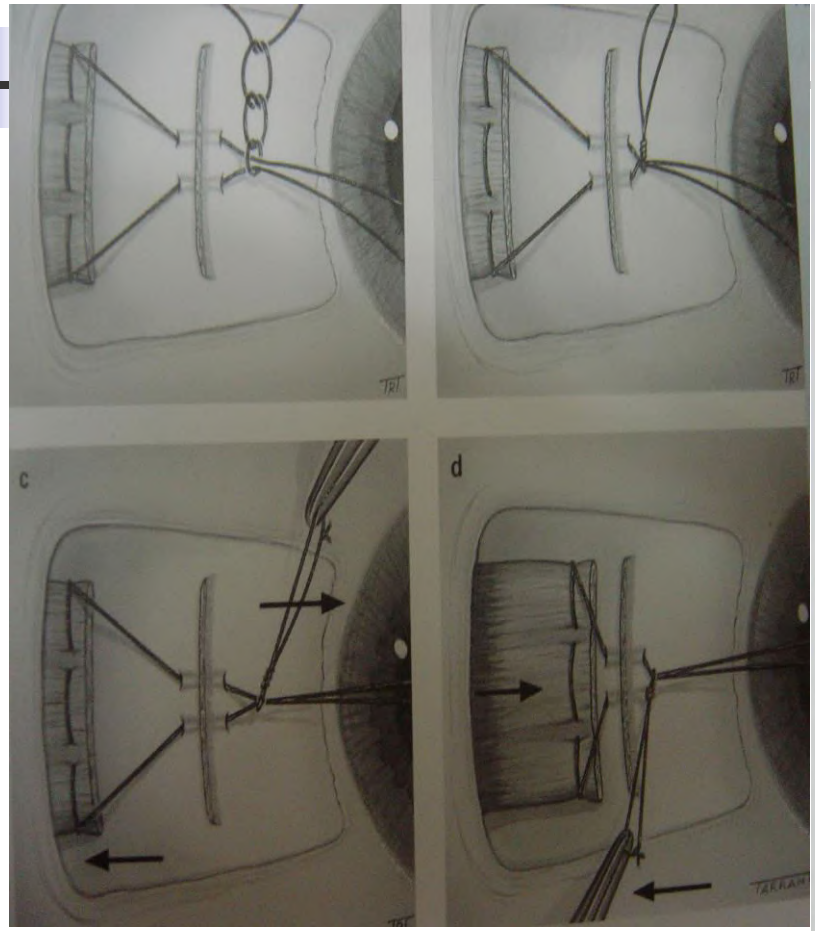
c
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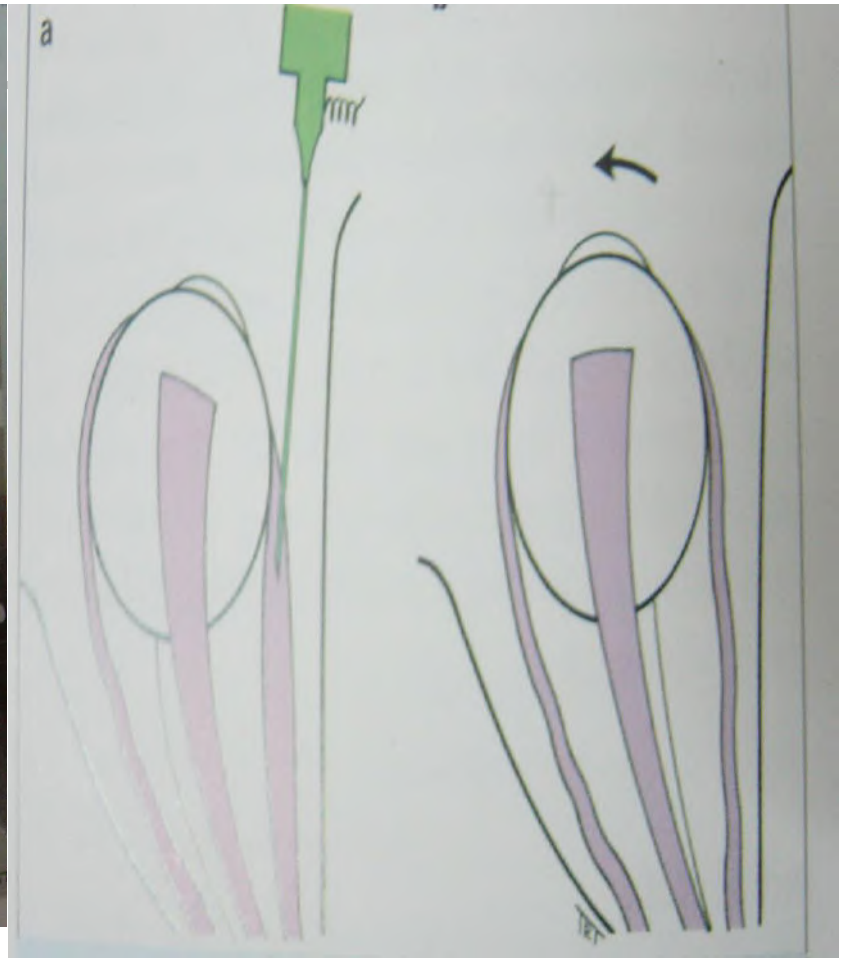
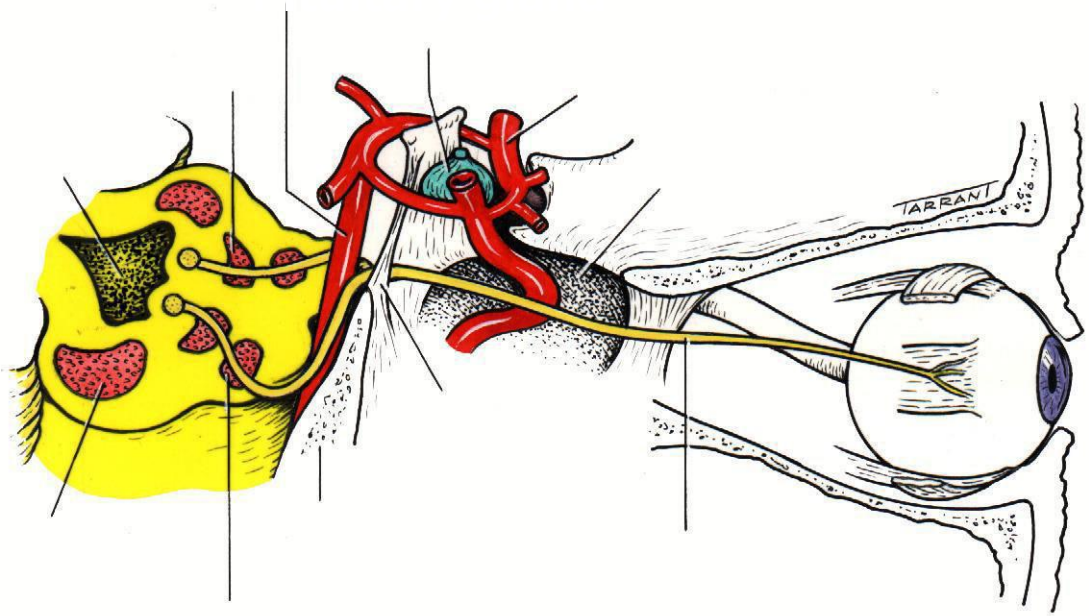

a

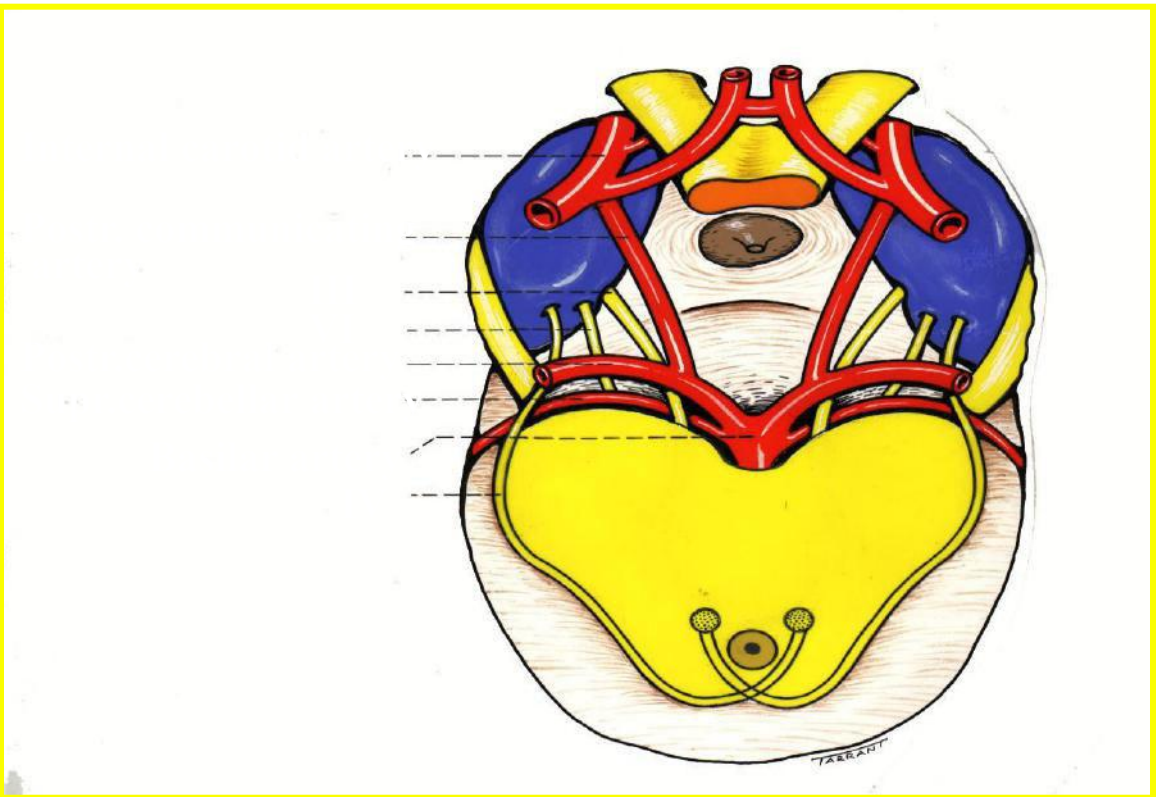
Types of Incomitant squint

- Paralytic
  - LR Palsy
  - SO Palsy
  - 3rd nerve palsy

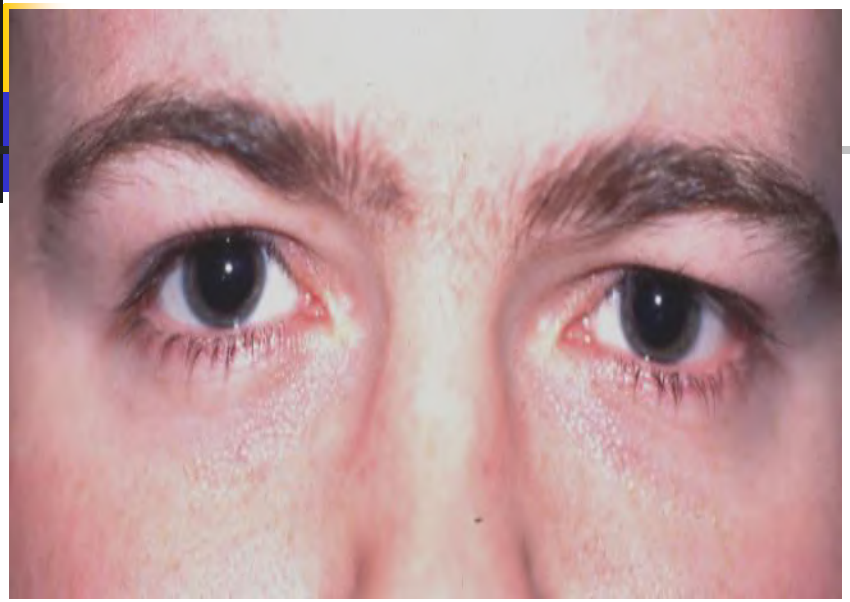
Anatomy of the 6th nerve



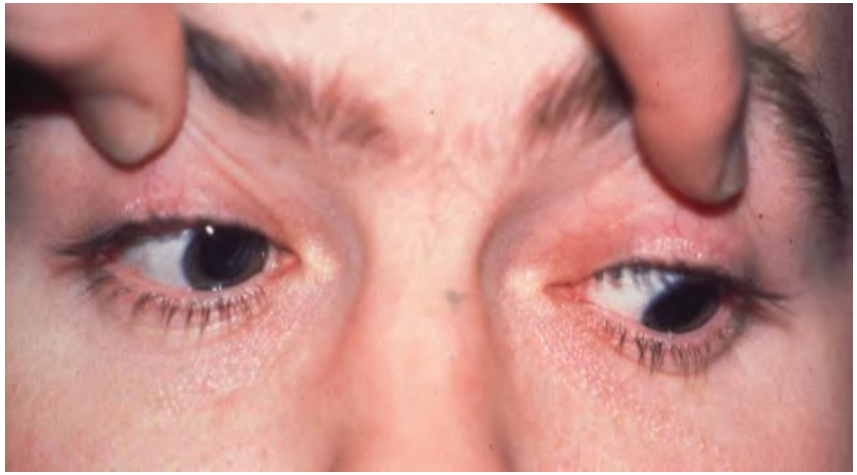
Anatomy of fourth nerve



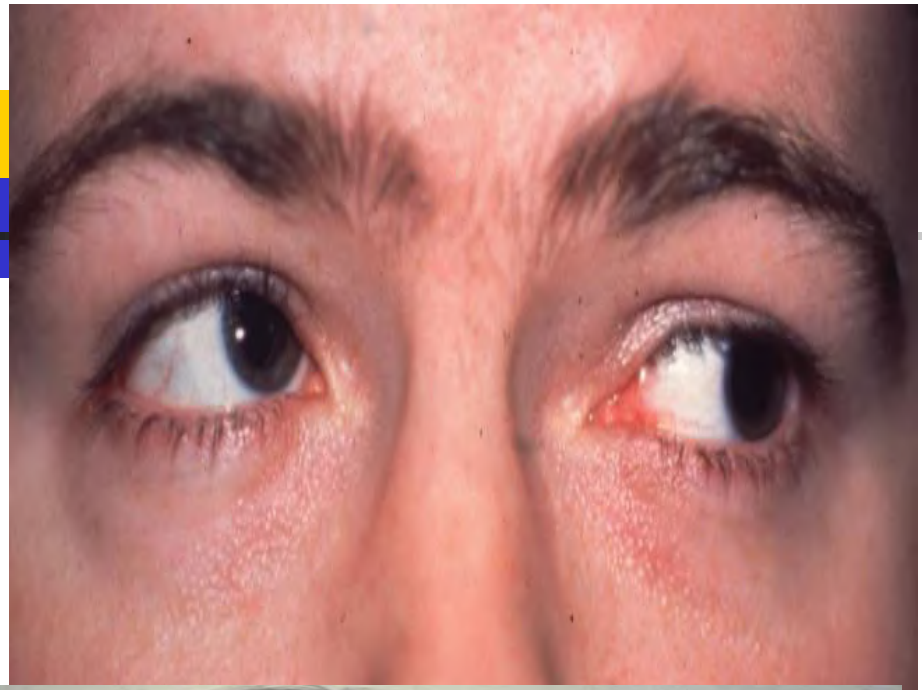
- Only cranial nerve to emerge dorsally
- Crossed cranial nerve
- Very long and slender



- Righthyperdeviation in primary position when left eye fixating



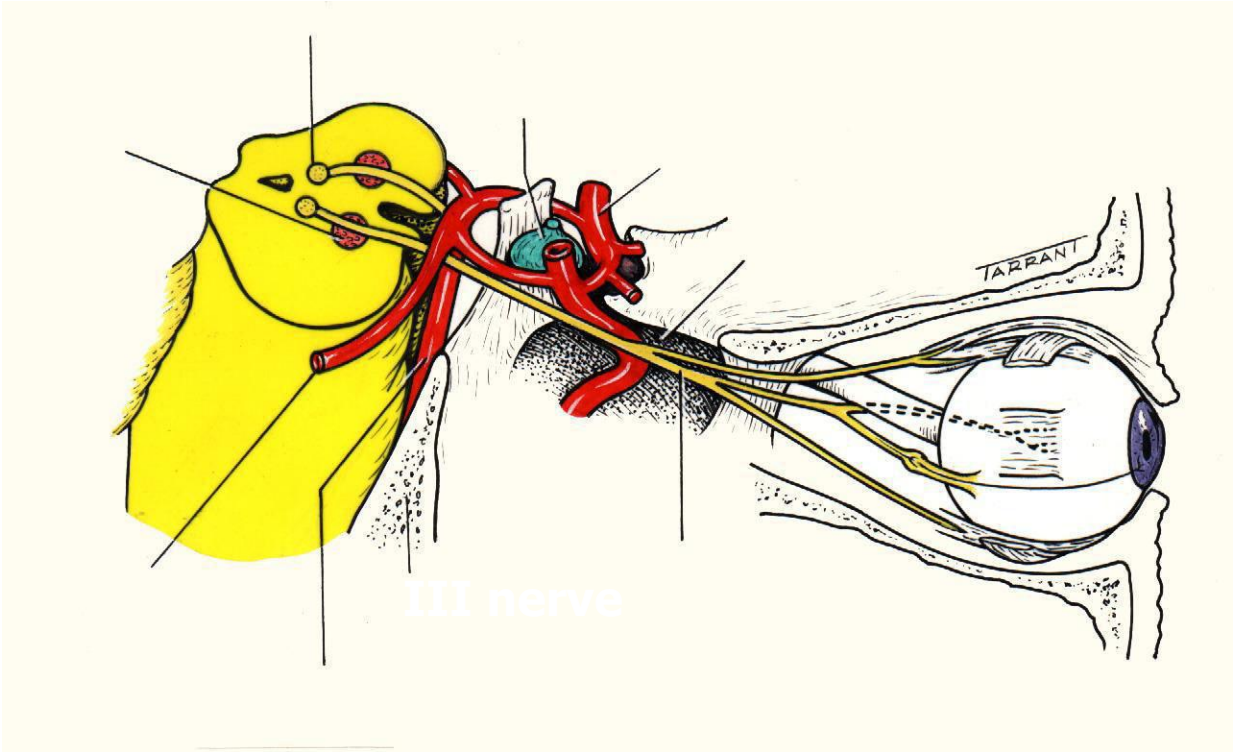
- Right underaction on depression in adduction



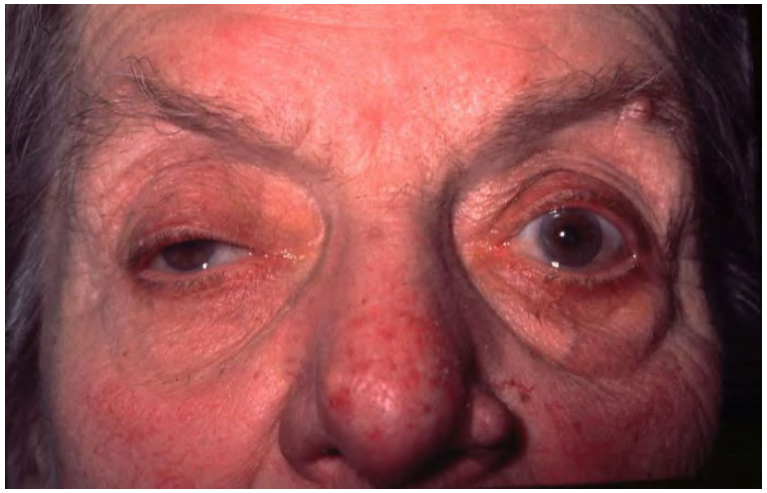
- Right overaction on left gaze

- Increase in right hyperdeviation on ipsilateral head tilt

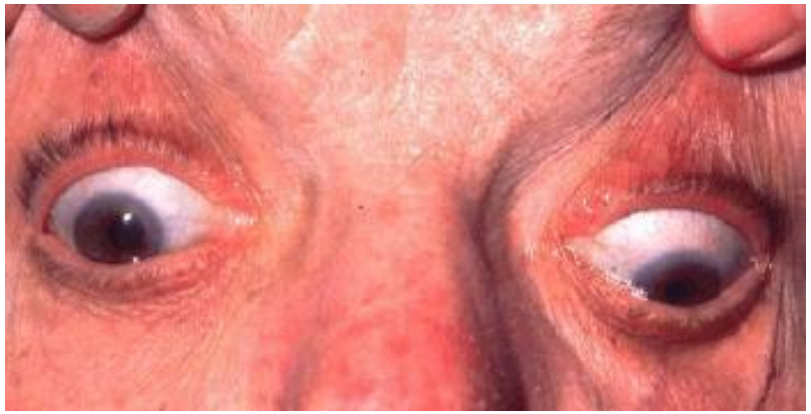
Anatomy of 3rd nerve

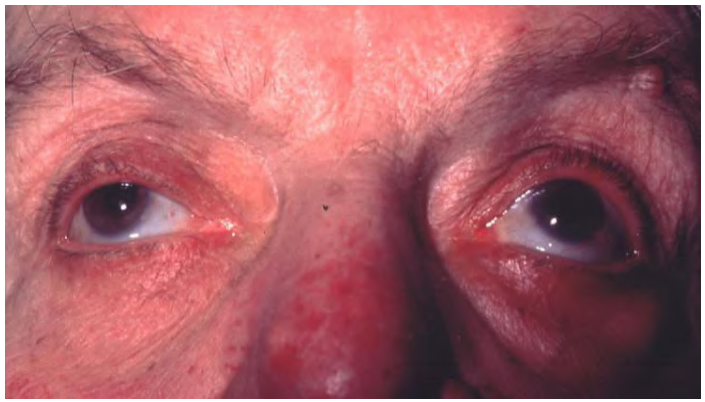


Clinical signs of 3rd nerve palsy



- **Ptosis, mydriasis and cycloplegia**
- **Abduction in primary position**
- **Limited depression and elevation**





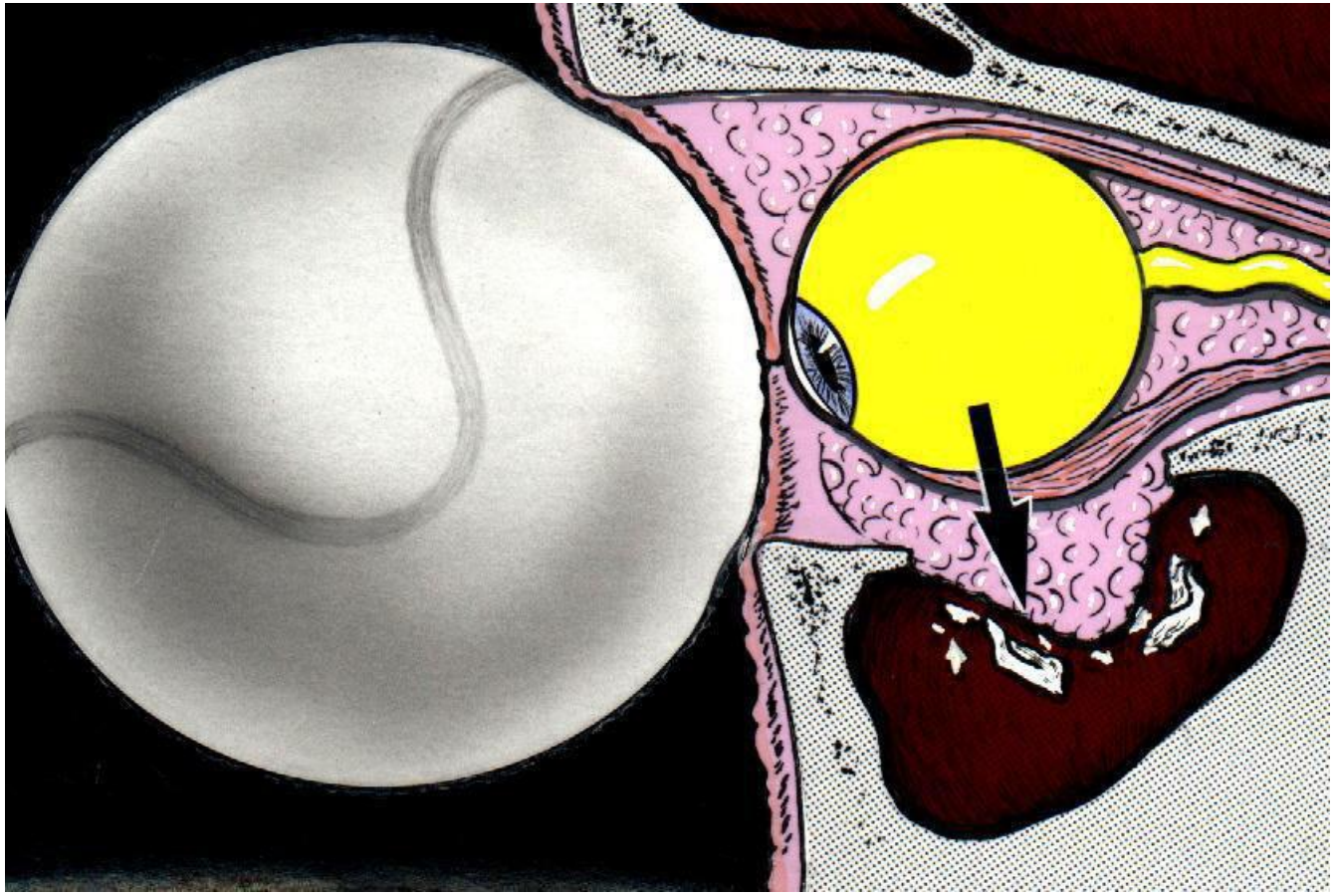
Restriction

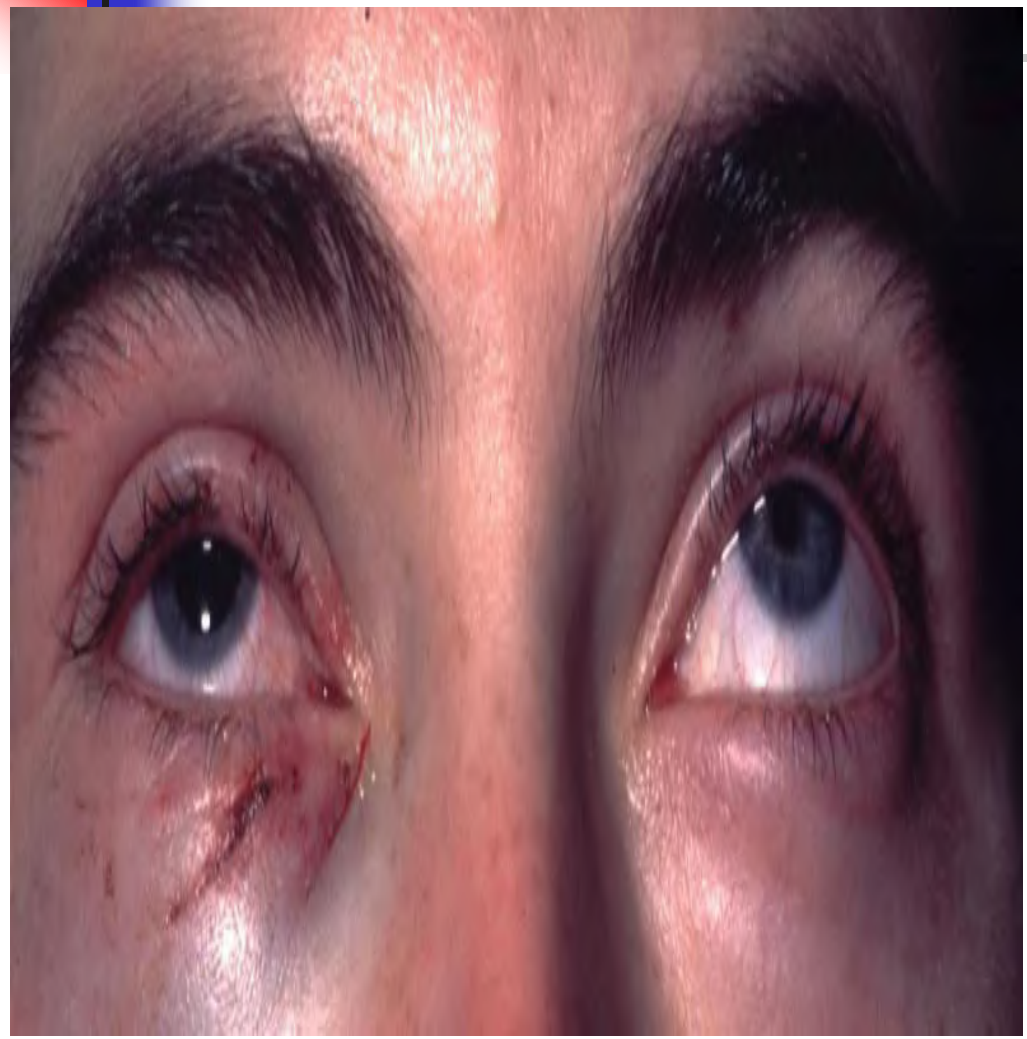
Blow out fractures

muscle entrapment

thyroid ophthalmopathy

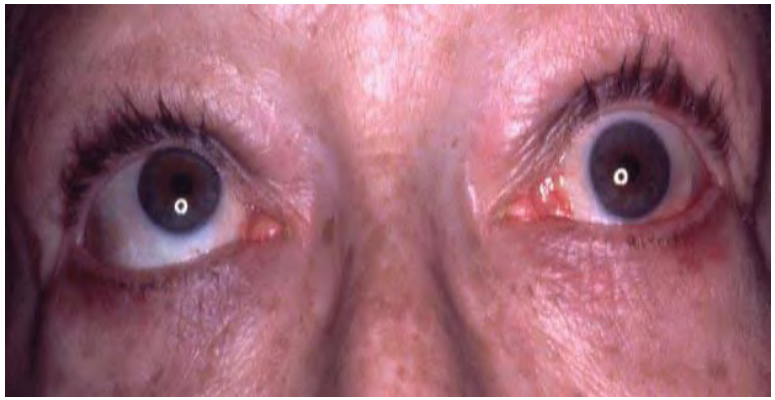
Blow out fracture of orbital floor

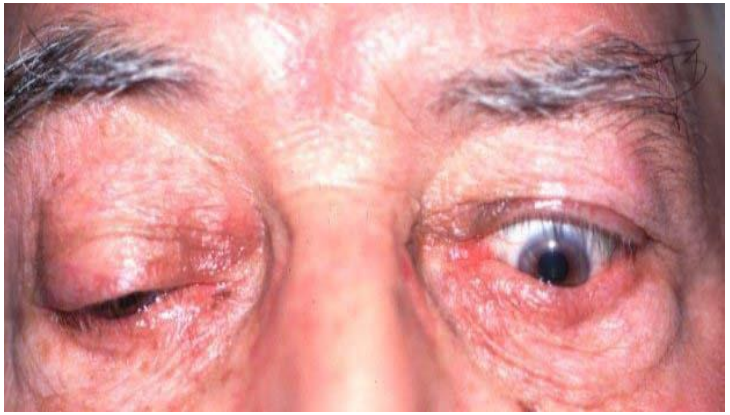


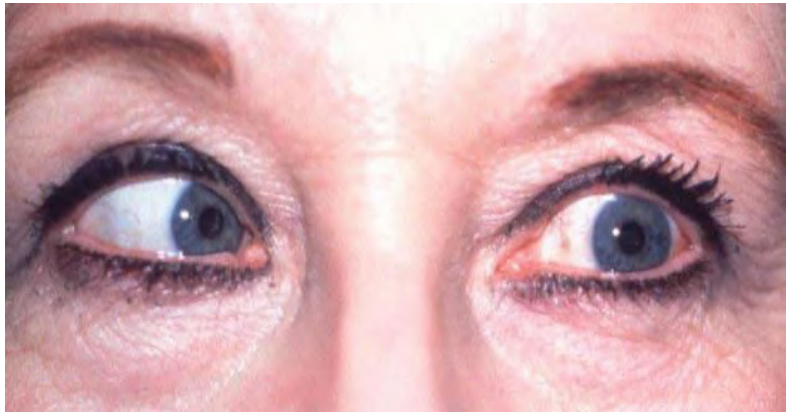


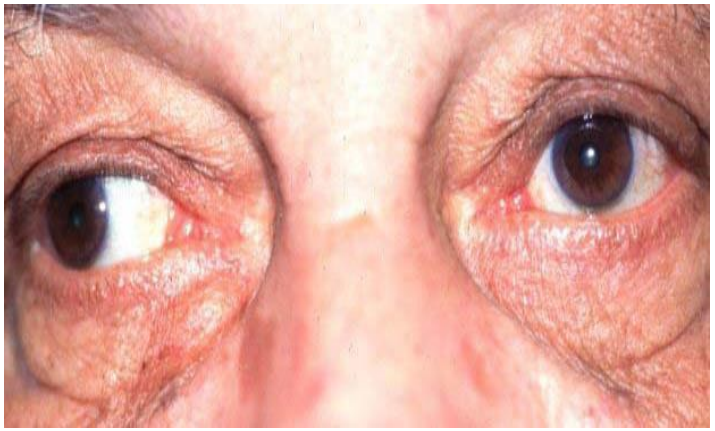
- Diplopia in up and down gaze

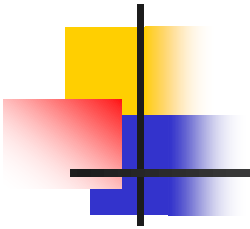
Types of restriction in thyroid eye disease







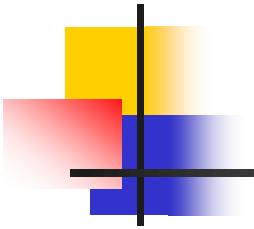




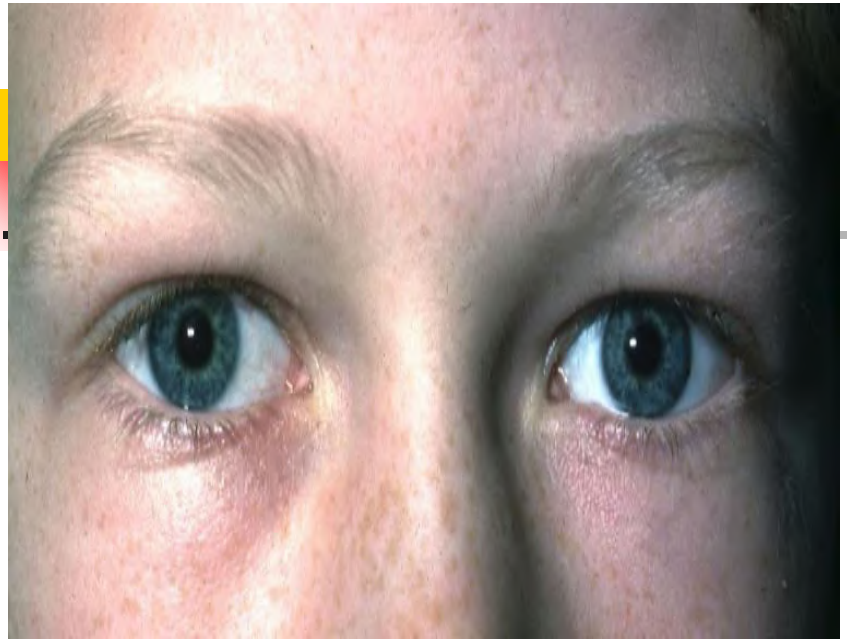
- **Special syndromes**

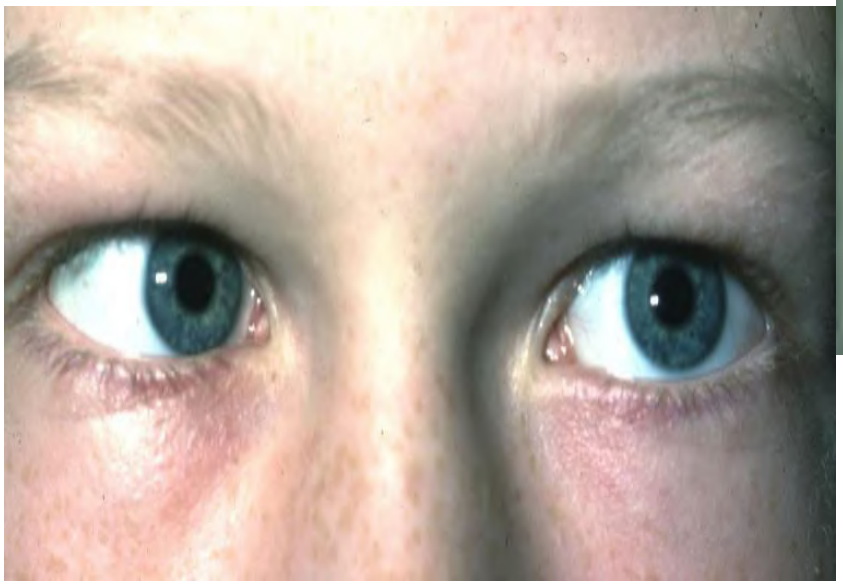
Duane syndrome

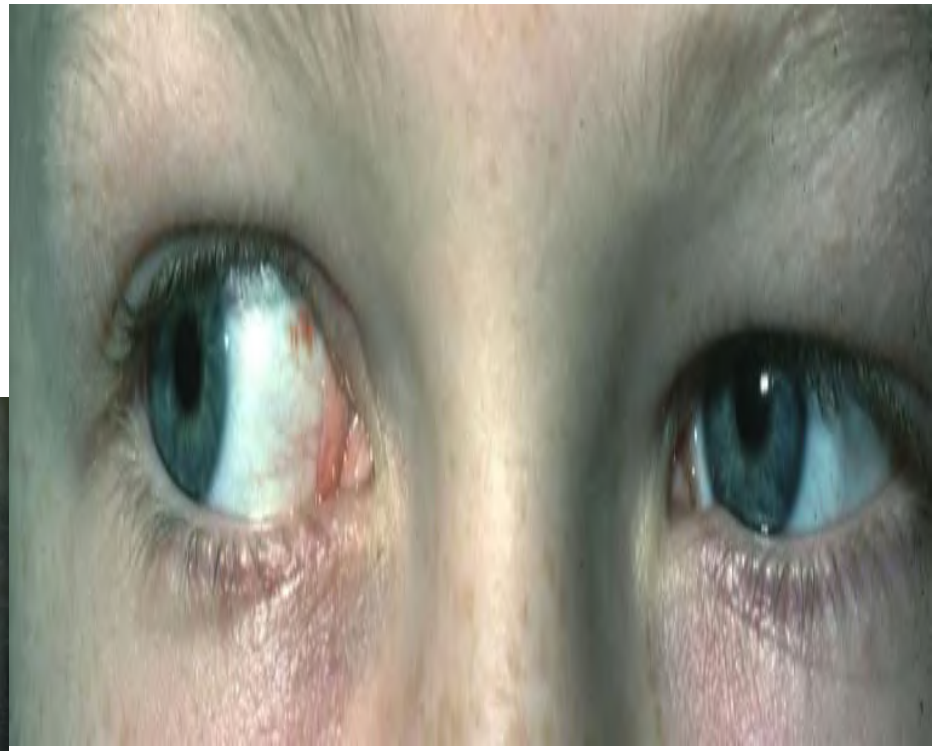
- On attempted adduction - retraction of globe and narrowing of palpebral fissure
- On attempted abduction - opening of palpebral fissure and normal globe position
- Bilateral in about 20%



- There is co contraction of both MR and LR
- There may be deafness and other congenital abn
- May be tight LR or absent Abducent nucleus







Brown syndrome

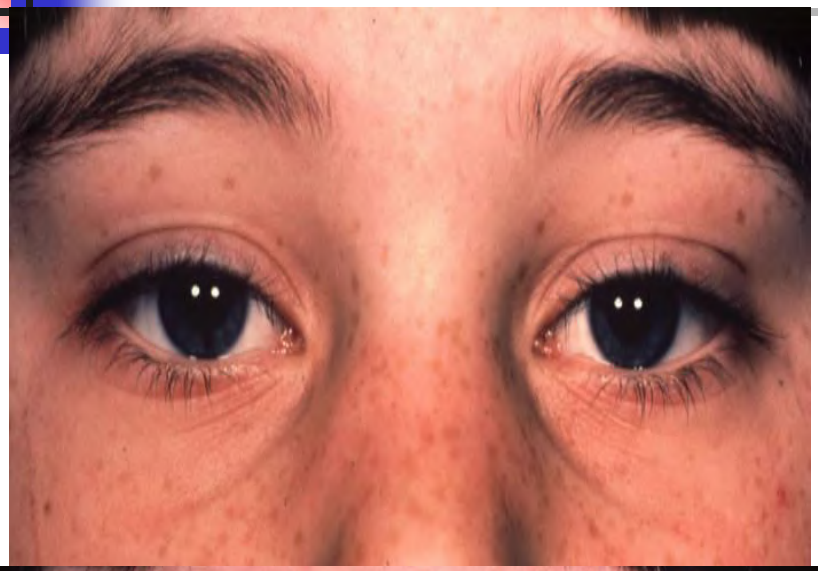
- Congenital

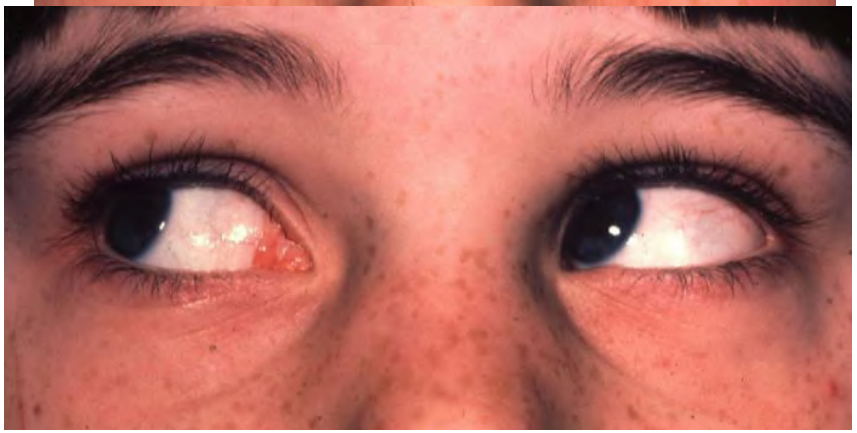
  idiopathic, congenital click syndrome
- Acquired

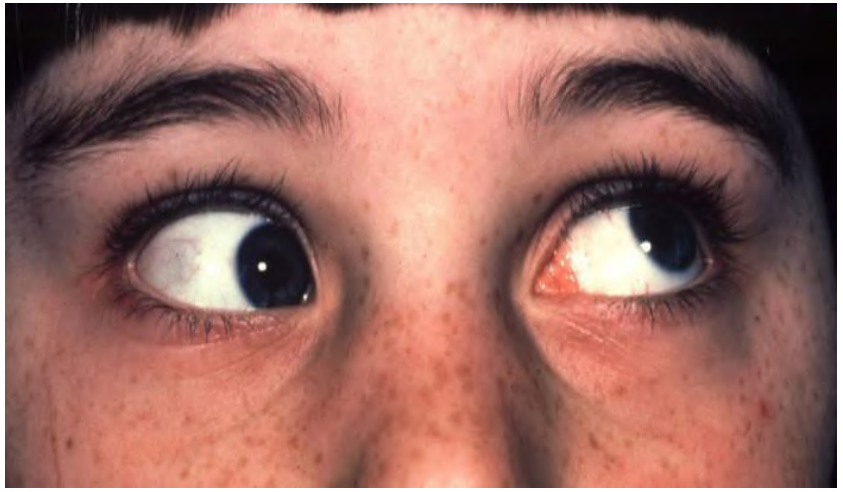
  iatrogenic

  inflammation of the tendon

Brown syndrome (right)

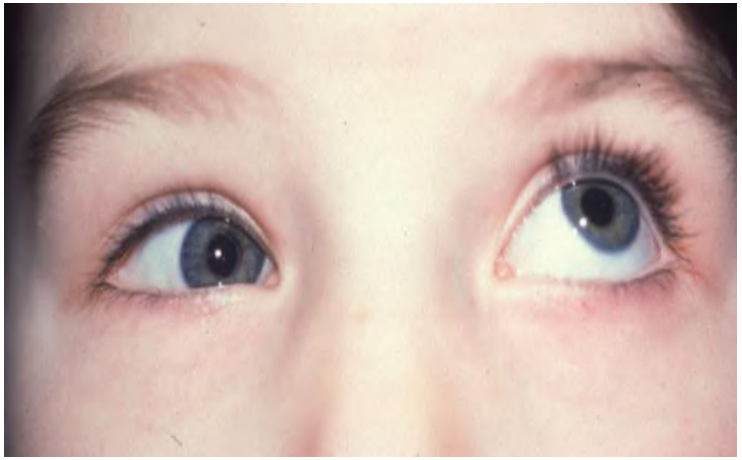


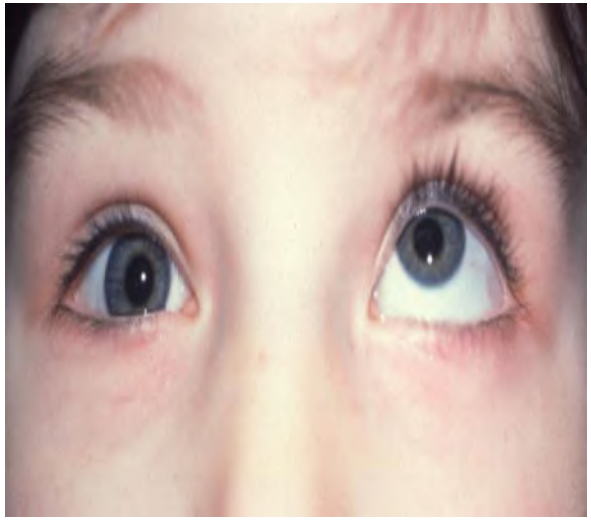




- **Limited elevation in adduction**

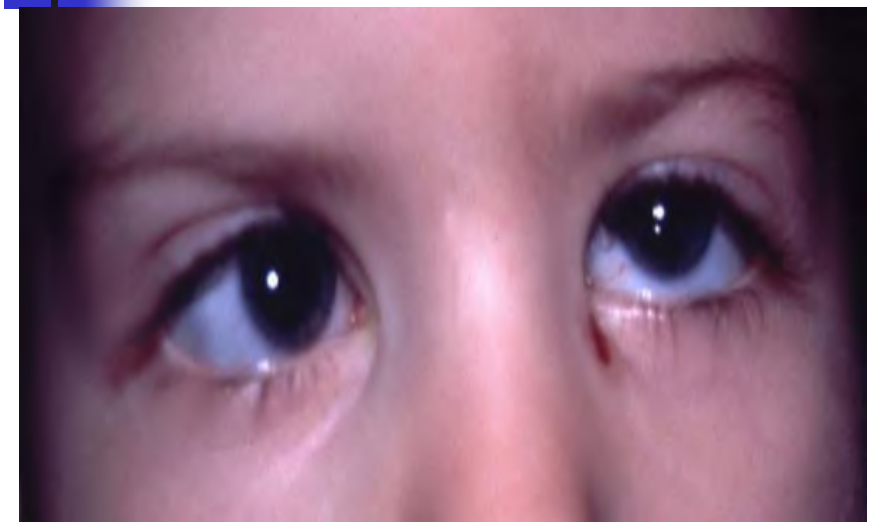
Double elevator palsy (right)

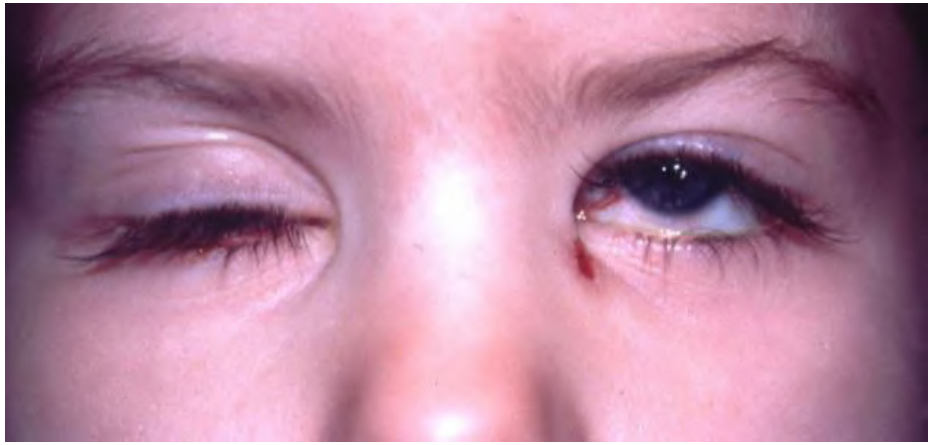




- **Unilateral elevation failure in all positions**

Mobius syndrome





- Bilateral sixth nerve palsies
- Primary position - 50% straight, 50% esotropic
- Paresis of 9th and 12th cranial nerves

Common ophthalmic drugs

Dr samina

AP Ophthalmology

- In pharmacology, a pharmaceutical drug, also called a medication or medicine,
- It is a chemical substance used to treat, cure, prevent, or diagnose, a disease or to promote well-being.

MODES OF ADMINISTRATION

- **DRUG DELIVERY IN EYES**

Topical	**Periocular**	**Intraocular**	**Systemic**
Drop	Subconj.	Intracameral	Oral
Ointment	Sub Tenon	Intravitreal	Intravenous
Gel	Peri-bulbar	Intramuscular	
	Retro bulbar		

STANDARD COLOURS FOR DRUG LABELING & BOTTLE CAP

- Yellow,, Blue : B blocker
- Red : Mydriatics and cycloplegics
- Green : Miotics
- Orange : Carbonic anhydrase inhibitors
- Brown or tan : Anti-infective agents
- Gray : NSAIDS
- Pink : Steroids

COMMON DRUGS USED IN OPHTHALMOLOGY

- MYDRIATRICS AND CYCLOPLEGICS AGENTS
- ANTI - VIRAL DRUGS
- ANTI – FUNGAL DRUGS
- ANTI-INFLAMMATORY AGENTS
- LUBRICATING AGENT AND ARTIFICIAL TEAR
- INTROCULAR AND IRRIGATING SOLUTIONS
- OTHER DRUGS AFFECTING EYE

MYDRIATRICS AND CYCLOPLEGICS AGENTS

- MYDRIATICS
  - – Only mydriasis
  - – Care should be taken in older patients
    - ➢ Phenylephrine
- CYCLOPLEGICS

– Along with mydriasis it also causes paralysis of accommodation

- Atropine sulfate
- Homatropine hydrobromide
- Cyclopentolate hydrochloride
- Tropicamide

PHENYLEPHR

- Direct acting drug

- Pupil dilation for diagnostic purpose

- **Indication**
  - COAG
  - Subacute or chronic ACG after iredectomy
  - Refused surgery cases
- **Dosage**
  - 1 or 2 drops for each refraction

- **Contraindication**
  - Narrow angle glaucoma
  - Hypertensives.
  - Type 1 diabetes mellitus.
  - Aneurysms.
  - Cardiac diseases
  - Infants
- **Adverse effects**
  - Mild stinging
  - HTN,
  - Headache
  - brow ache

ATROPINE

- Atropine sulfate is anticholinergic – 0.5%, 1%,3%
- Strongest drug in cycloplegics
- Inhibitor of muscarinic action of acetylcholine
-
- Indication
  - Mydriasis & cycloplegia
  - Pupil dilation in inflammatory condition of iris
  - Amblyopia therapy
-

- Contraindication
  - 1° glaucoma
  - Hypersensitive
- Adverse effects
  - Allergic responses
  - Blurred vision
  - Photophobia
  - Dryness of skin & mouth
  - Skin rash
- Dosage – 1 or 2 drops 0.5% to each eye, twice daily for 1 to 3 days prior to examination

HOMATROPINE

- First anticholinergic
- Directly blocks muscarinic action of acetylcholine
- Its effect last for longer time

- **Indication** –
  - Cycloplegic refraction
  - Treatment of inflammatory uveal tract condition

- **Contraindication –**
  - Tendency with occludable angles
  - Patient who have allergic to atropine
- **Adverse reaction**
  - Follicular conjunctivitis
  - Vascular congestion
  - Edema exudate
- **Dosage** – 1 or two drops repeated in 5 to 10 minute if necessary

CYCLOPENTOLATE

- Cyclopentolate hydrochloride 1% is an anticholinergic

- **Indication**
  - Cycloplegics of children under the age 12 to 20 yrs
- **Contraindication**
  - Narrow angle glaucoma
  - Hypersensitive
  - Children with emosional problems

TROPICAMIDE

- Most common anticholinergic medriatric drug
- Stronger – 1%, paralysis accomodation 0.5% produce mydriasis with slight cycloplegia
- **Indication**
    - Diagnostic process
    - Pre and post operative stages
    - Infants
    - combination with phenylephrine

- **Contraindication**
  - Narrow angle glaucoma
  - Hypersensitive
- **Adverse effects**
  - Increased IOP, blurring of vision, photophobia
  - Psychotic reaction
  - Dryness of mouth, headache or allergic reactions
- **Dosage** – One drop followed by second drop in 5 minutes of arc

ANTI - VIRAL DRUGS

- Locally given to the eye
- HSV, HZV, CMV retinitis
- **Common drugs**
  - Acyclovir
  - Vidarabine
  - Ganciclovir

ANTI – FUNGAL DRUGS

- **Common drugs**
  - Natamycin
  - Amphotericin B

ANTI-INFLAMMATORY AGENTS

- **Non – steroidal**
  - Topical :- Flurbiprofen sodium
  - Systemic :- Keterolac tromethamine
- **Steroidal**
  - Topical :- Fluoromethelone
  - Systemic :- Prednisolone

LUBRICATING AGENT AND ARTIFICIAL TEAR

- **Indication**
  - Ocular irritations in various diseases
  - Dry eyes
- **Commonly available commercial tear substitutes**
  - REFRESH TEARS
  - TEARS PLUS
  - ARTIFICIAL TEARS
- **Warning** – Discontinue using if any irritation produces
- **Direction for use** – 1 or 2 drops as frequently as required

INTRAOCULAR AND IRRIGATING SOLUTIONS

- Contain concentration of inorganic & organic constituents
- Mainly used in surgery procedure
- Keep the glob inflated

- **Indications:**
  - Cataract surgery
  - Vitrectomy & posterior segment surgeries
  - Anterior segment reconstruction

OTHER DRUGS AFFECTING EYE

Drug	Uses	Side effect
Amiodarone	A cardiac arrhythmia drug	Corneal vortex keratopathy Optic neuropathy
Digitalis	A cardiac failure drug	Chromatopsia
Chloroquine's	Malaria, Rheumatoid Arthritis, SLE	Corneal verticillata Retinopathy
Thioridazine	A psychiatric drug	Pigmentary retinopathy
Ethambutol	An anti-TB drug	Optic neuropathy
Chlorpromazine	A psychiatric drug	Corneal punctate epithelial opacities Lens surface opacities

Thanks