Structured Notes According to

OPHTHALMOLOGY

Revision friendly Fully Colored Book/Structured Notes

For Best results, watch the video lectures along with reading notes



(Author)

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LIST OF IMPORTANT TOPICS

- Retina

- Diabetic retinopatny stages, images, Mx
- Retinal detachment
- ROP staging
- Retinitis pigmentosa

Conjuctiva and cornea

- Trachoma Image, C/F, Elimination strategies
- Conjunctivitis Difference b/w etiologies
- Corneal Ulcer Fungal, Viral, Acanthamoeba

Neuro ophthalmology

- Optic pathway and its lesions
- Eye Deviation In Cranial Nerve Palsies
- Horner Syndrome
- Optic neuritis
- Papilledema

Procedures and surgeries

- Enucleation/Exenteration, Evisceration: Indications
- Keratoplasty
- Dark room procedures
- Tonometry
- Direct/Indirect Ophthalmoscopy
- Macular Function Tests
- Visual Field Defects
- EOG

Glaucoma

- Types
- Management Of Glaucoma (To be done with Pharmacology)

Tumors

Retinoblastoma, Melanoma: Stages of RB, Mx Myopia, HypermetropiaCataract: Causes, Mx





BASICS OF OPTHALMOLOGY

- Anatomy of orbit and eyeball
- Layers of the eyeball
 - Outermost layer
 - → Cornea and sclera
 - Middle layer
 - → Iris, ciliary body and choroid
 - Inner layer
 - → Retina
- Anterior and posterior chamber
- · Dynamics of aqueous humor
- · Optics and power of eye
- Accommodation



BASICS OF OPTHALMOLOGY

ANATOMY

Orbit

- Volume of eyeball/globe: 6 ml
- Volume of each orbit: 30 ml
- It is Encased in Tenon's capsule, suspended by orbit and supported by Lockwood's ligament
- Accessory eyeball structures
 - o Extraocular muscles
 - o Fat
 - Eyelids
 - Eyebrows
 - Lacrimal gland
- 3 layers
 - Outer fibrous
 - Middle vascular
 - o Inner neural layer



Layers of eye ball

EYE BALL

- Outer Most Layer consists of
 - o Anterior 1/6th: Cornea Clear & transparent
 - o Posterior 5/6th; Sclera Opaque & White
 - Junction b/w cornea and sclera: Limbus

Sclera

- Function
 - Maintains shape,
 - Site for attachment of Extra ocular muscles (EOM)



Important Information

Thinnest part of sclera lies behind the attachment of rectus muscles, thickest part of sclera is at posterior pole

- 3 layers of sclera
 - Episclera
 - o Stroma
 - o Lamina fusca
- Sclera is White and opaque due to irregular arrangement of stromal collagen
- Glistening and shiny, tough structure
- Appears yellow (icteric) in jaundice



Sclera



Previous Year's Questions

Q. Evisceration is removal of which layer of eyeball?

(FMGE JUNE 2019)

- A. Middle and inner layer
- B. Outer and middle
- C. Outer and inner
- D. All the layers

Cornea



00:06:50

- Shape of cornea
 - Prolate spheroid
 - More curved in centre than periphery
- · Keratometer measures the cornea curvature and the technique is known as keratometry
- Instruments used
 - Placido's disc
 - → In normal cornea, there is no distortion of

reflected pattern

→ In Keratoconus, distortion of reflected pattern is present



Placido's Disc

- Cornea is Transparent
 - Due to regular stromal collagen and presence of Na⁺-K⁺ ATPase pump in endothelium layer
 - o 90% light transmission
- 6 layers of cornea
 - 1. Epithelium
 - 2. Bowman's membrane
 - o Injury to this layer leads to corneal opacity or scar
- 3. Stroma
 - Thickest layer
 - 4. Predescemet's layer (Dua's layer)
 - 5. Descemet's membrane
 - Strongest layer
 - 6. Endothelium
 - Single layer of hexagonal layer
 - o Maintain corneal opacity



Previous Year's Questions

Q. Corneal transparency is decided by

(NEET 2021). (FMGE JUNE 2021)

- A. Keratan sulphate
- B. Chondroitin sulphate
- C. Heparan sulphate
- D. Hyaluronic acid



Previous Year's Questions

- Q. Which layer of cornea helps in maintaining hydration of stroma of cornea? (NEETJAN 2020)
- A. Descemet's membrane
- B. Endothelium
- C. Epithelium
- D. Stroma



Previous Year's Questions

- Q. All of the following are features of corneal epithelium except. (AIIMS JUNE 2019)
- A. Lined by stratified squamous epithelium
- B. Bowman's membrane regenerates
- C. Apical cells have microvilli
- D. Mitosis is limited to limbus
- Thickness of Cornea
 - Thinnest in centre &
 - o Thickest at periphery,
 - o Ranging from 500 -600 μ,
 - IOP depends upon central corneal thickness (CCT)
 - o Measured by Pachymetry



Pachymetry

- Most powerful refracting surface eye: 43D (70 %)
- Cornea is avascular

Glucose	Oxygen
Aqueous humor	 Air Tear film Aqueous humor

- Sensory nerve supply of cornea is ophthalmic branch of VCN
- Has highest density of nerve endings in the body

Limbus



- It is Junction of cornea and sclera
- Surgical limbus is zone of 2 mm
- Corneal stem cells are present at limbus
 - o Longitudinal parallel cells
 - o Yellowish brown in colour
 - Called as palisades of Vogt



Palisades of Vogt

- In case of deficiency of stem cells due to chemical injury (mc), prolonged contact lens wearing etc. there is decreased regeneration capacity of eye
 - Signs of Limbal stem cell deficiency
 - → Cells breakdown
 - → Inflammation
 - → Persistent epithelial defects
 - → Ulceration
 - → Neovascularization
 - → Conjunctivalisation
 - Diagnosis
 - → On Impression Cytology: Presence of Goblet cells on cornea (normally goblets cells are present in conjunctiva not in cornea)



Limbal stem cell deficiency



Important Information

 Corneal stem cells lie at limbus whereas Conjunctival stem cells lie at fornix



Previous Year's Questions

- Q. Universal limbal stem cell marker? (JIPMER NOV 2018)
- A. C-Cadherin
- B. PAX6
- C. Abcg2
- D. P63

MIDDLE LAYER OF EYEBALL

Uvea

- Consists of
 - 1. Iris
 - 2. Ciliary Body
 - 3. Choroid

Iris

- Most anterior part of the uveal tract
- Divided by collarette into central pupillary zone and peripheral ciliary zone

00:19:51

- Two muscles
 - o Sphincter pupillae
 - Dilator pupillae



Collarette

- Function
 - o Controls entry of light by dilating / constricting pupil
- Two arterial circles
 - o Major arterial circle
 - → Lies at root of iris where it attaches to the ciliary body
 - → If it bleeds leads to hyphema



Hyphema

- o Minor arterial circle
 - → Lies at collarette



Arterial circle



Important Information

- John Daugman patented iris pattern scans.
- · Iris patterns are distinct for each person.

CILIARY BODY



- 3 parts
 - 1. Pars Plicata: Anterior half with folds
 - 2. Pars Plana: Posterior half without folds
 - 3. Ciliary Muscle: Enclosed by Pars Plicate & Pars Plana
- Root of iris is attached to ciliary body
- Thinnest part of iris
- Rupture leads to iridodialysis
- Characteristic D-shaped pupil are seen in Iridodialysis

Function

- Aqueous humour production
- Accommodation
- Pars plana only way to access vitreous humour
- Maximum risk of sympathetic ophthalmitis occurs on injury to ciliary body

CHOROID



- Posterior most structure of uvea
- Most vascular structure of eye
- Choroid circulation: 85% of ocular blood flow
- Vortex Veins: Drains entire uvea but particularly choroid)

Function

- Supplies nutrients to outer retina,
- Thermoregulation

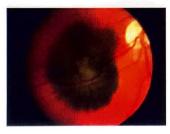
4 layers

- Chorio-capillaries laver
- Bruch's membrane
- Haller's layer and Sattler's layer



Important Information

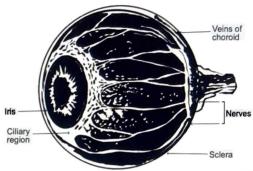
- M/C ocular tumour in children is retinoblastoma
- M/c ocular tumour in adults: Malignant melanoma of choroid



Malignant melanoma of choroid

THE GRAPES OF WRATH



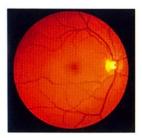


INNER MOST LAYER OF EYEBALL

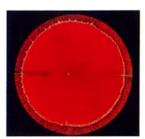


Retina

- Innermost layer stretches from central fovea to peripheral ora serrata
- Present only posteriorly
- Central retina: Macula, fovea, optic disc
- Centre of retina occupied by: Macula
- Centre of Macula Occupied by: Fovea
- Fovea is most sensitive structure to light so brightest and sharpest image forms here



- Peripheral retina
- Retina
- → serrated ora serrata
- → Thinnest



Peripheral retina: Serrated ora serrata

Unusual → light passes through retina → photoreceptors

in outer retina, neural signals \rightarrow reverse order to inner retina \rightarrow Optic nerve

- Microscopic Structure of Retina
 - Neurons: 5 Types
 - Neuroglia [supporting cells]: 3 Types

Neuron Types

- 1. Photo Receptors (Rods/Cones) (1st Order neuron)
- 2. Bipolar Cells (2nd Order neuron)
- 3. Ganglion Cells (3rd Order neuron)
- 4. Amacrine Cells
- 5. Horizontal Cells

Neuroglia cells

- 1. M Muller's
- 2. A-Astroglia
- 3. M-Microglia

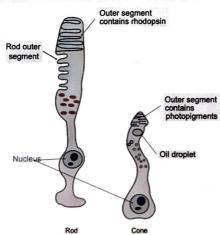


How to remember

MAM

PHOTORECEPTORS

THOTORECEI TORS	
Rods	Cones
 Peripherally located 120 million in number 1 type Responsible for Peripheral vision Black & white vision Night Vision 	 Centrally located 6 million in number Red, Green & Blue types of cones + Responsible for Central Vision Colour Vision Day light vision
	Outer segment



Photoreceptor cell



Previous Year's Questions

- Q. First order neuron in visual pathway. (AIIMS MAY 2019)
- A. Bipolar cells
- B. Ganglion cells
- C. Photoreceptors
- D. Lateral geniculate body

ANTERIOR CHAMBER (AC)



00:43:23

- Space between cornea and iris
- Filled with aqueous humor
- Depth
 - Measured from centre of cornea to centre or anterior lens capsule
 - o 3 mm
 - o Shallow AC
 - → Women Elderly children (WEC)
 - → If less than 2.1 mm at risk of developing angle closure glaucoma
 - o Deep AC: Young males
 - o Volume: 250 µl
 - Anterior chamber angle (AC Angle) is space between iris/ endothelium at limbus, estimated by Van Herick's method

Structures lying in AC (from iris to corneal side)

- I Iris
- Can't CB band
- See Scleral spur
- This Trabecular meshwork
- Stuff Schwalbe's line



How to remember

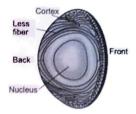
- · I Can't See This Stuff
- Immune privileged site: ACAID (Anterior Chamber Associated Immune Deviation) \(\psi\) immune defence mechanism

CRYSTALLINE LENS



00:54:23

It is Biconvex, transparent, avascular structure



Crystalline Lens

Contains

- 1. Nucleus: centre of lens
- 2. Cortex: periphery of lens
- 3. Capsule: completely covers the lens on all sides
- Thinnest part of capsule: Posterior Capsule
- LEC's (Lens Epithelial Cells)
 - Only Present in anterior capsule
 - Responsible for secondary/ after cataract
- Lens fibers
 - Forms bulk of lens
 - On cut section resembles layers of onion

Composition

- Water: 65%
- Proteins: 35%, has highest concentration of proteins among all the structures of body
- 90% lens proteins are crystalline in nature



Important Information

Lens has highest concentration of proteins among all the structures of body (35%)

Function

- Transmits and focus light on retina
- Accommodation
- Lens contributes 19D of the 60D of power of eye (30%)
- Lens derives nutrition and oxygen from aqueous humor
- Lens suspended by Zonules / Suspensory Ligaments from ciliary body

IRIS LENS DIAPHRAGM INCLUDES

Iris
 Ciliary body
 Zonules

Moves together

4. Lens



CB Zonules Lens complex

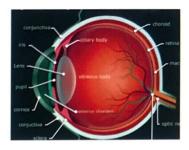
POSTERIOR CHAMBER (PC)

- Space b/w the Iris & Iens
- Contains agueous humor
- AC & PC Connected with each other by pupil

VITREOUS HUMOUR



- Glassy transparent gel, Space b/w posterior surface of lens & Retina
- Volume: 4 ml single largest structure inside the eye



Composition

- 99% H₂O
- Hvaluronic acid
- Collagen

Important Information

- Intra-ocular Structure having highest amount of hyaluronic acid Vitreous humor
- Body Structure having highest amount of hyaluronic acid-cartilage

Function

- Shock absorber
- Optical media
- Maintains shape



- o Produced at birth
- Attached to disc margin, macula, vitreous base (strongest), Posterior Capsule by Weiger's ligament
- o Vitreous base avulsion occurs in ocular trauma
- Only safe way to enter vitreous cavity is through pars plana

AQUEOUS HUMOUR

· Produced from non-pigmented epithelium of pars plicata

Mechanism of production

- 1. Secretion (70%), active transport, requires energy
- 2. Ultrafiltration, passive
- 3. Diffusion, passive
- Rate of production: 2.50 µL/minute
- Slows down at night but never stops
- Outflow rate: C value 0.20 µL/mm Hg /minute
- Composition is very similar to blood

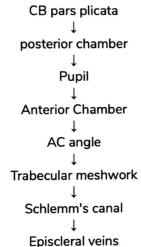
Blood Plasma Vs Aqueous Humor

Blood	Aqueous humor
† Glucose† Protein	 80% of blood Glucose Almost protein Free ↑ Ascorbate ↑Lactate

Function

- Supplies glucose and oxygen to cornea and lens,
- Removes waste products (Macrophages, blood, debris)

Circulation of Aqueous Humour





Circulation of Aqueous Humour

Aqueous Vs Vitreous

Aqueous Humor	Vitreous Humor
• Aqueous = H ₂ O	• Vitreous = Glass
• Solution	• Gel
 Provides nutrition 	 Provides shock absorption
• Produced at 2.5 µl/min	Produced at the time of birth
Entry through limbus	 Entry through Pars Plana only
 Composition is Similar to Blood plasma with few exceptions 	 Composition 98% H₂O Hyaluronic acid Type II Collagen

OPTICS

- Optic Curved Surfaces can bend light
- Curvature α Bending

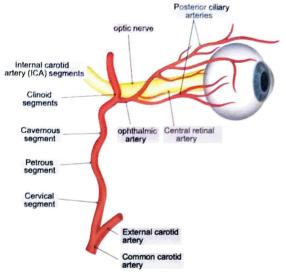
Power

- Total Power of eye: + 60 Dioptres (exactly 58.6 D)
 - Power of Cornea: 43 D (70%)
 - o Power of Lens: 19 D (30%)
- Plus indicates convergence
- Minus indicated Divergence
- Power of Eye Contributed by
 - 1. Anterior Surfaces of Cornea (Maximum Contribution)
 - 2. Posterior Surfaces of Cornea
 - 3. Anterior Surfaces of Lens
 - 4. Posterior Surfaces of Lens

VASCULATURE OF THE EYE



- Principal artery of eye is Ophthalmic Artery (10 branches)
- Most critical: Central Retinal artery (CRA) is 1st branch of ophthalmic artery



Blood supply of eye

- Ophthalmic Artery is first branch of Internal Carotid Artery
- Principal veins are Central Retinal Vein / Vortex veins
- Superior Ophthalmic vein is the largest and principal vein

NERVE SUPPLY OF EYE



- 6 Cranial nerves: II, III, IV, V, VI, VII
- Autonomic nervous system
 - o Parasympathetic: Ill nerve Miosis, accommodation
 - Sympathetic: V nerve Mydriasis, Muller's muscle, Inferior tarsal muscle, sweat glands

BASIC OPHTHALMIC MEASUREMENTS



Orbital volume	30 ml
Eyeball volume	6 ml
AC volume	250 µL
Conjunctival sac volume	35 µL
Eyedrop volume	50 μL
Aqueous humour formation	2.5 µL/minute
Basal tear formation	1.2 µL/minute
Fovea and Optic disc	1.5 mm
Monocular visual field	S-60N-60I-70T-100

VISUAL ACUITY

Ö 01:47:32



Distant vision: 6/6

6 m (distance between patient & chart)

6 m (distance at which a normal person reads the chart)

- We use snellen's chart to study distant vision
- Visual acuity 6/60 means patient standing at 6 metres is able to read a line which normal person can read from 60 metres



Important Information

- Angle subtended by each letter onto the human eye is 5 min / letter
- Angle subtended by each part of letter onto the human eye is I mi



Previous Year's Questions

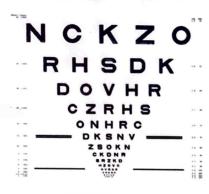
Q. What is the angle subtended by the biggest Letter of Snellen's chart at the nodal point of eye? (AIIMS NOV 2019)

- A. 5 minutes
- B. 20 minutes
- C. 30 minutes
- D. 50 minutes



Snellen's Letter

 Most accurate chart for visual acuity is early treatment diabetic retinopathy study (ETDRS) chart



(ETDRS) chart

- Landolt's Rings
 - o Chart used for illiterate people



Landolt's Rings

- Picture charts
 - For preschool children (< 3 years of age)



Picture charts

- Allen Chart
 - For children



Allen Chart

- Optokinetic drum
 - o For preverbal children (<2 years of age)



Optokinetic drum

NEAR VISION



- Optimal near vision: N6
- Near vision depends on
 - 1. Accommodation
 - 2. Convergence
 - 3. Miosis

Near reflex / Triple response/ Accommodational reflex

Accommodation

- Change in curvature of lens to increase power of the lens to look at nearer objects
- Minimum distance for light rays to become parallel is 6m
 - o Nearer the object, rays are more divergent
 - o Distant object, rays are parallel
- ↑ Curvature of lens
 - ↑ Power of lens (19D + additional 16D = 35D)
 - o Rays of light can be focused on retina
- Mechanism of Accommodation

- Increased curvature of anterior surface of lens
- Anterior movement of lens
- Equatorial diameter ↓ AP diameter ↑

Convergence

- Simultaneous inward movement of both eyes towards each other to maintain binocular single vision
- Both medial recti muscles involved



Convergence

Miosis

Constriction of pupil

INTRAOCULAR PRESSURE (IOP)



- Goldmann equation: IOP = (F/C+P)
 - Where F is rate of formation of aqueous humor, C is rate of drainage of aqueous humor and P Episcleral venous pressure (~10 mmHg)
- Normal IOP: 10 21 mm of Hg (average 16mm Hg)
- IOP is measured by Tonometer: Tonometry / Tonography
- Most accurate tonometer: Goldmann Applanation tonometer
- IOP dependent on central corneal thickness (CCT)





Goldmann Applanation tonometer

Tonopen

Important Information

- Thick cornea measure IOP falsely high
- Thin cornea measure 10P falsely low
- So, people with thin corneas are at higher risk of developing glaucoma

VISUAL FIELD



- "An island of vision in a sea of darkness"
- An area of space which is visible without moving the gaze from a central target

Monocular field limits

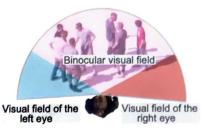
- Superior 60°
- Nasal 60°
- Inferior 70°
- Temporal 100°



Important Information

Most important field defects lie within central 30°





Monocular Temporal Crescent

Monocular Field

Binocular Field

Blind spot of Mariotte

- Physiological, absolute scotoma corresponding to scleral canal where the optic disc lies or from where the optic nerve goes out
- No rods and cons are present over optic disc, so image formed on optic disc cannot be seen by retina
- Lies temporally, 15 degrees from fixation, 7.5 degrees in diameter, 1.5 degrees below horizontal meridian



Important Information

 Optic disc lies nasal side on retina so blind spot is present temporally in visual field

Humphrey's Perimeter

Instrument that measures visual field



Humphrey's Perimeter

- · Cannot be tested by Snellen chart
- Contrast sensitivity test is the ability to distinguish object without clear outlines/ discriminate objects from their background



Contrast Sensitivity

- VA charts test high contrast letters, daily visual tasks require resolution of low/medium contrast
- Single most important factor in visual functioning is contrast sensitivity
- Activities which require contrast sensitivity
 - o Driving at night
 - o Rain/fog
 - o Difficulty reading newspaper
 - o Pour coffee into a dark mug
- Spatial frequency (SF): determines clarity and gradation of bright/dark areas
- Highest (SF): 30 cycles / degree, 6cpd most critical

Contrast sensitivity decreases in

- Glaucoma
- Diabetic Retinopathy
- Amblyopia
- Cataract
- Macular degeneration
- Keratoconus

Three test of contrast sensitivity

1. Pelli - Robson Chart lowest contrast where correct response on 2/3 letters – Contrast Sensitivity Threshold

V R S K D R N H C S O K S C N O Z V



Mars Test

3. FACT: Functional Acuity Contrast Test

COLOUR BLINDNESS: DALTONISM 6 00

- Most common x linked recessive disorder
- 8% males colour blind: 1/12 males ,1/200 females
- Colour blind people Confuse colours

Problems with Daltonism

- · Can't pick up ripe fruits
- · Can't make out meat is cooked
- Wear mismatched clothing



Colour blindness

Types of colour blindness

 Trichromatism: sensitive to all 3-cone system (Red, Green, Blue), no colour deficiency



Trichromatism

- Anomalous trichromatism: one colour \(\psi \) sensitivity
 - o Protanomaly: Red reduced

Pelli - Robson Chart

- Deuteranomaly: Green reduced
- Tritanomaly: Blue reduced
- Dichromatism: One colour cone system is absent, sensitive to other two
 - o Protanopia: Red cones missing
 - Deuteranopia: Green cones missing
 - Tritanopia: Blue cones missing



 Monochromatism sensitive to only one colour cone system

	Tritanomaly	Tritanopia
•	Blue colour reduced sensitivity	Blue cones missing

Clinical problems

- Risk of † accidents in driving, particularly protanopia
- Face discrimination in exams
- · Limited career options



Important Information

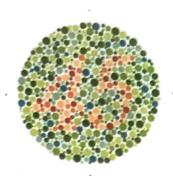
M/c form of colour blindness: Red Green colour blindness

Tests for colour blindness

Holmgren's wool



· Ishihara pseudoisochromatic chart



- FM 100 Hue test
- Hardy Rand Rittler plates



Colour Vision Testing Made Easy



Nagel's Anomaloscope: gold standard

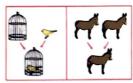
GRADES BINOCULAR SINGLE VISION © 00:24:06

Three grades

- 1. Simultaneous macular perception SMP
- 2. Fusion
- 3. Stereopsis

Simultaneous macular perception

- Visual signals transferred from two eyes to brain cortex are perceived at the same time, ability to see two dissimilar objects simultaneously
- Indicates presence or absence of suppression



Simultaneous macular perception

Fusion and Stereopsis

- Fusion: Two images fused, with effort to maintain fusion despite difficulty
- Stereopsis: Two images blended to produce depth
 - Perception and stereoscopic effect crucial for driving, sports and motor control
- Depth perception: Ability to see in 3 D and judge distance of objects
- Stereopsis is the highest grade of binocular vision

2Dvs3D



2D vs 3D

LENSES USED IN OPHTHALMOLOGY



OFTITIALMOLOGI	
Convex lens	Concave lens
 Centre is thick and periphery is thinner 	 Centre is thin and periphery is thicker
• + power lens	• - power lens
 Converges rays of light 	 Diverges rays of light
Magnifies1D = magnifies by2%	Minifies1D = minifies by2%



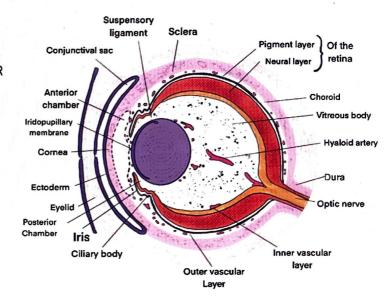


- Q. A 66-year-old male who was homeless was brought to Ophthalmology OPD. On examination, a dense haemorrhage in the posterior chamber of the right eye was found, involving the visual axis. The normal anatomical contents of this chamber is/are:
- A. Retinal vessels
- B. Aqueous humour
- C. Vitreous humour
- D. All

Answer: B

Solution

- BOTH AC AND PC CONTAINS AQUEOUS HUMOR
- Posterior chamber(PC) space between posterior surface of iris & anterior surface of lens.
- · Posterior chamber contain aqueous humor
- Posterior segment contain vitreous humor



Reference: Comprehensive Ophthalmology; A K Khurana, 6th edition page 4

- Q. A 27-year-old, 38 weeks pregnant woman with a height of 130 cm and a weight of 51 kg diagnosed case of cephalopelvic disproportion with fetal distress was posted for emergency cesarean section. A lower segment cesarean section was performed and a live male infant 1.8 kg with an APGAR of 8/9 was delivered. The eye of this newborn would have
- A. Hypermetropic with regular astigmatism
- B. Hypermetropia
- C. Hypermetropic with irregular astigmatism
- D. Myopia

Answer: B

Solution

- At birth, the eye is hypermetropic by +2 to +3D and usually becomes emmetropic by the age of 5 to 7 years
- This is because of the smaller eyeball
- · Eyeball length at birth is 16mm.

Reference: AK KHURANA PG NO. 35

- Q. A 28-year-old woman who had just delivered a baby 24hrs before was very concerned after she read an article on google about a baby's visual acuity not being normal at birth. You can reassure the mother, that the baby attains a normal level of visual acuity is at:
- A. 6 months
- B. 1 year
- C. 3 years
- D. 6 years

Answer: C

Solution

Eye in the postnatal period

- Fixation starts developing by 4-6 weeks. The critical period for the development of fixation reflex is 2-4 months. Development of fixation is completed by 6 months.
- · Fixation development is completed in 6 months.
- The macula is fully developed by 4-6 months.
- Fusional reflex, stereopsis and accommodation are well developed by 4-6 months.
- Cornea attains normal adult diameter by 2 years of age.
- The lens grows throughout life.
- Full visual acuity (6/6) is attained by 3 years of age.

Land to the second seco	THE THE PERSON AND ADDRESS OF THE PERSON ADDRESS OF
Age	Visual acuity
Newborn	6/240
1 month	6/180 - 6/90
4-6 months	6/18 - 6 /9
3 Years	6/6

Reference: Comprehensive Ophthalmology Fourth Edition by A.K.Khurana, Pg-12





EMBRYOLOGY OF EYE

- Derivatives of surface ectoderm in eye
- Derivatives of mesoderm in eye
- Derivatives of neuroectoderm in eye
- Derivatives of Secondary mesoderm in eye



2

EMBRYOLOGY OF EYE

Eye has derivatives from

(5) 00:01:20

- 1. Ectoderm
- 2. Mesoderm
- 3. Neural crest cells



Important Information

No Endoderm derivatives in eye

ECTODERM DERIVATIVES

- Surface Ectoderm
 - o L-Lens
 - o E Epithelium (except epithelium of iris & ciliary body)
 - V Vitreous
 - L Lacrimal apparatus



How to remember

- LEVeL
- Neuro Ectoderm

00:02:29

00:01:20

- M Muscles of pupil (sphincter & dilator pupilae)
- o O-Optic nerve
- R Retinal pigment epithelium / Retina
- o E Epithelium of ciliary body & iris
- o Vitreous



How to remember

MORE

MESODERM DERIVATIVES



- M Muscles (extraocular) of eye
- E Endothelium of all ocular of orbital blood vessels
- S Sclera & Schlemm's canal
- O VitreOus



How to remember

MESO

NEURAL CREST CELLS DERIVATIVES



- Corneal stroma & Endothelium cells
- Trabecular "meshwork"
- Ciliary muscles
- Melanocytes



Important Information

Vitreous is derived from all the three layers





- Q. On examination of the crystalline lens of the human eye under light microscopy, you were able to visualise the gross suture patterns of lenses. These lens sutures are formed in:
- A. Fetal nucleus
- B. Embryonic nucleus
- C. Infantile nucleus
- D. Adult nucleus

Answer: A

Solution

Development of Lens

Surface ectoderm

Classification of nucleus formed during lens development

- Embryonic (0-3 months)
- Fetal (3 months to gestation)
- Infantile (from birth to puberty)
- Adult (adult life)

Sutures

- Seen in fetal nucleus
- Anterior Y shaped
- Posterior inverted Y shaped

Reference: A K Khurana Anatomy and physiology of eye



LEARNING OBJECTIVES

Orbit & Adnexa

- Anatomy of orbit
 - Bones
 - Muscles
 - Nerves
- Blow out fracture
- Orbital cellulitis
- Cavernous sinus thrombosis
- ORBITAL APEX DISORDERS
- GRAVES OPHTHALMOPATHY
- ENOPHTHALMOS
- DISORDERS OF THE EYELIDS
 - PTOSIS
 - ENTROPION
 - ECTROPION
 - BLEPHARITIS
 - CHALAZION and Stye
 - o BASAL, SQUAMOUS and SEBACEOUS CELL CARCINOMA of eyelid



3

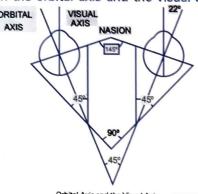
ORBIT & ADNEXA

ORBITAL ANATOMY



- Volume of each orbit: 30 mL
- Volume of each eyeball: 6 mL
- Angle between two lateral walls: 90 degrees
- Angle between lateral and medial wall: 45 degrees
- · Two medial walls parallel to each other
- Angle between the orbital axis and the visual axis: 20

degrees



ORBIT



 Orbit has two bony cavities, pear/pyramidal shaped base, 4 walls, apex



Important Information

- Rule of 7: In orbit there are
 - o 7bones.
 - o 7 muscles.
 - o 7 Nerves

7 bones

- S Sphenoid
- E Ethmoid
- L Lacrimal
- F Frontal
- Z Zygomatic
- P Palatine
- A And
- M Maxillary



How to remember

SELFZPAM

7 muscles

- Superior rectus
- Inferior rectus
- Medial rectus
- Lateral rectus
- Superior oblique
- Inferior oblique
- LPS

7 nerves

- II, III, IV, VI,
- 3 nerves from ophthalmic branch of V nerve
 - Nasociliary nerve
 - Lacrimal nerve
 - Frontal nerve

NASAL BONE

NASAL BONE

SUPRAORBITAL FORAMEN

ZYIGOMATIC PROCESS OF FRONTAL

OPTIC CANAL

SUPERIOR ORBITAL
FISSURE

ORBITAL PROCESS
OF ZYGOMATIC

Orbital cavity

- Thinnest wall of the orbit is medial wall
- Infection from the ethmoid sinus enters the orbit causing orbital cellulitis
- Roof separates anterior cranial fossa from orbit

Two Parts

- 1. Anterior: Eyeball
- 2. Posterior: Muscles, vessels, nerves all supported by fatty tissue
- Function of the orbit is protection of eyeball
- Orbit is a Rigid box, can only expand forwards → proptosis
- Orbital septum is the boundary between lids and orbit



BLOW OUT FRACTURE

- **Ö** 00:10:10
- Fracture of orbital walls without involving orbital rim
- M/c type of orbital fracture
- Size of blunt object > size of orbital aperture
- Causes of Blow out fracture are Falls, traffic accidents, personal violence



Important Information

- . Thinnest wall of the orbit is medial wall
- · M/c wall to fracture is inferior wall: Orbital floor

Triad of Blow Out Fracture

- Enophthalmos
- Diplopia
- Infraorbital anaesthesia
- Oculocardiac reflex is seen



Important Information

Triad of Oculocardiac reflex

- Nausea
- Bradycardia
- Syncope

Diagnosis

Tear Drop Sign on X Rays

Management

- Surgical repair is only done if
 - Fracture involving at least 50% of orbital floor: Within 2 weeks
 - o Enophthalmos > 2 mm
 - o Diplopia with limitation of up gaze or down gaze
- Urgent repair is done in
 - Paediatric blowouts with entrapment of EOM, or activation of ocular cardiac reflex (OCR)

ORBITAL CELLULITIS



- Acute infection of the orbit behind the orbital septum there is no involvement of the globe, only soft tissue within the orbit is involved
- Most commonly in children
- M/c cause Ethmoidal Sinusitis, dental abscess, middle ear infection
- M/c infecting organism is Staph aureus (MRSA) > Streptococci

Clinical features

- Proptosis
- Chemosis
- Restriction of ocular movements



Important Information

- MC cause of U/L proptosis in a child → Orbital cellulitis
- MC cause of B/L proptosis in a child → Neuroblastoma metastasis
- MC cause of U/L proptosis in an adult
- MC cause of B/L proptosis in an adult

Thyroid Eye Disease (TED)

Thyroid related ophthalmopathy (TRO) /Grave's Ophthalmopathy (GO)

Signs of Orbital Cellulitis

 Erythema and well demarcated lid edema extending beyond eyelid margin



Orbital Cellulitis

- Loss of vision
- Pain with ocular movements

Diagnosis

Investigation: MRI/Contrast enhanced CT

Management

- Uncomplicated cases with antibiotics alone
- IV Vancomycin + Ceftriaxone
- Surgical: Drain involved sinus/orbital abscess



Previous Year's Questions

Q. Axial proptosis is seen in?

(JIPMER Dec 2019)

A. Hyperthyroidism

- B. Optic nerve sheath Meningioma
- C. Orbital floor fracture
- D. Lacrimal gland tumour

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CAVERNOUS SINUS THROMBOSIS (**)



 Cavernous Sinus is Pair of Dural venous sinuses in middle cranial fossa on either side of the Turkish saddle, divided by septa into 'caves'

Referimages 3.1

 Rare, life-threatening condition (30%): Complicating facial infection (nasal furuncle) sinusitis, orbital cellulitis forming bacterial embolus (Staph Aureus) leading to thrombosis

Symptoms

- Headache (M/c)
- Swinging fever
- Chills
- Rigors
- Cranial Nerve signs
- · Periorbital edema
- Chemosis
- Painful ophthalmoplegia
- Proptosis, EOM paralysis
 - Earliest Sign: 6th nerve palsy
 - o B/L within 48 hours



Cavernous Sinus Thrombosis

CNS Symptoms

- Disorientation
- Drowsiness
- Coma
 - 6th palsy with Horner's syndrome (HS) → CS Parkinson's Sign

Treatment

- i/v Antibiotics
- i/v Heparin
- Steroids

?

Previous Year's Questions

Q.What is the diagnosis for a patient with unilateral proptosis with bilateral 6th nerve palsy with chemosis and euthyroid status?

(NEET Jan 2020)

A. cavernous sinus thrombosis

- B. Thyroid ophthalmopathy
- C. Retinoblastoma
- D. Orbital pseudotumour

ORBITAL APEX DISORDERS



Disorders caused by inflammation of structures close to orbital axis. These include

- Cavernous Sinus Syndrome (CSS)
- 2. Superior Orbital Fissure Syndrome (SOFS)
- Orbital Apex Syndrome (OAS): Loss of vision due to optic nerve involvement

1. CAVERNOUS SINUS SYNDROME (CSS)

 CSS is resulting from compromise of Cranial nerve (III, IV, VI & 2 branches of V) which lies within Cavernous Sinus

Symptoms

- Ophthalmoplegia
- Proptosis
- Trigeminal sensory loss
- Horner's Syndrome
- o M/c cause are tumours: Schwannoma's (m/c)

Vascular causes

- Carotid cavernous fistula
- Cavernous sinus thrombosis

Inflammatory causes

Tolosa Hunt Syndrome granulomatous inflammation of CS

2. SUPERIOR ORBITAL FISSURE SYNDROME

Due to compression of structures in Superior orbital fissure



Clinical Features

- Ophthalmoplegia: Damage to 3,4,6 nerves
- Ptosis: LPS palsy due to 3rd CN damage
- Proptosis: \(\) tension of the EOM (globe retractors)
- Fixed, dilated pupils: \(\parasympathetic \) parasympathetic (3rd CN)
 - Corneal anaesthesia: Nasociliary nerve damage
 - Forehead/eyelid anaesthesia: Frontal/lacrimal N \

Difference between CCS and SOFS CCSSOFS

ccs	SOFS
1. Bilateral	1. Unilateral
 Ophthalmic(V₁) & maxillary (V₂) branches of trigeminal (V) nerve affected 	 Only Ophthalmic (V₁) branch of trigeminal (V) nerve affected
3. Horner syndrome present	3. Horner syndrome absent

3. ORBITAL APEX SYNDROME

- Orbital apex incorporates the Superior orbital fissure and optic canal
- Cranial nerves involved with loss of vision

Symptoms

- Proptosis
- Ophthalmoplegia
- Loss of vision
- RAPD
- Ptosis
- Hypoesthesia of forehead
 - o SOFS in front orbital apex, CSS behind it: B/L
 - o Differentiating point is loss of vision

PEDIATRIC ORBITAL TUMOURS



- M/c primary benign orbital tumour of children: Dermoid cyst>capillary hemangioma - 2nd
 - o M/c location of dermoid cyst superotemporal location



- M/c primary malignant tumour of orbit in children
 - o Rhabdomyosarcoma



- Most common secondary tumours of orbit in children: Uncommon
- Metastases from
 - Neuroblastoma (Raccoon sign)



- Ewing's Sarcoma
- o Wilm's tumour
- o Chloromas in AML: Granulocytic sarcoma

ORBITAL TUMOURS IN ADULTS



- Most common primary benign tumour of the orbit in adults
- Cavernous hemangioma
- M/c primary malignant tumour orbit: Lymphoma NHL B cell origin
- Most common secondary tumours of adult orbit
- Metastasis from
 - Breast carcinoma
 - Lungs
 - Prostate



Enophthalmos



Important Information

 Breast ca metastasis to eye often cause enophthalmos rather than exophthalmos due to fibrosis of EOM

GRAVES OPHTHALMOPATHY/ TED/TRO



Autoimmune inflammatory disorder

Pathology

Antibodies leading to †production of thyroxine hormone

in blood

- Graves' disease is the m/c cause of hyperthyroidism
- Hyperthyroid: 90%, Euthyroid 6%, hypothyroid
- Stimulation of orbital fibroblasts leads to stimulation of inflammatory cytokines like (TNF α) → GAG / Hyaluronic Acid → gets deposited in periorbital tissue and all over eyes
- Thyroid stimulating immunoglobin is the closest functional biomarker of TED

Graves triad

- Hyperthyroidism
- Ophthalmopathy
- Pretibial myxoedema

Werner's (NOSPECS) Classification



- 1. Class 0: N No signs, no symptoms
- 2. Class 1: O Only signs
- Two signs
 - Von Graefe's sign: Lid lag due to overstimulation of Muller muscle (sympathetic system) which contracts and pulls the lid up



- o Lid retraction: m/c sign of TRO
 - → It also Occurs because of ↑sympathetic tone on Muller's
- 3. Class 2: S Soft tissue signs conjunctival injection



- 4. Class 3: P Proptosis (exophthalmos)
- 2nd m/c sign
- On CT scan: Fusiform enlargement of muscle belly sparing the tendons is seen



5. Class 4: E - Extra ocular muscle restriction



Important Information

Order of involvement of EOM in Thyroid Eye Disease

1-Inferior rectus

am-Medial rectus

So-Superior rectus

Lucky - Lateral rectus



How to remember

I am So Lucky

6. Class 5: C - Corneal exposure keratopathy



7. Class 6: S - Sight loss due to optic nerve compression



How to remember

NOSPECS

ENOPHTHALMOS

Posterior displacement of globe within the orbit (pulled inwards)



Enophthalmos

Two causes

- 1. † Volume of orbit
- 2. I volume of orbital contents
- Causes of \uparrow volume orbit: M/c is orbital fractures, NF type

1

- Causes of \(\psi \) Volume orbit: Microphthalmos, orbital fat atrophy (post-surgical, post radiation)
- Scirrhous ca breast metastasis: M/c rule out female with enophthalmos



Important Information

 Any female come with Enophthalmos rule out metastasis due to breast carcinoma

Triad

- Ptosis
- Enophthalmos
- Restricted ocular movements



On examination

- Deep set eyes
- Deep sulcus
- Ptosis



Visual inspection

- · Chin up, looking upwards from down position
- Difference of >2 mm between two eyes on Hertel's exophthalmometry

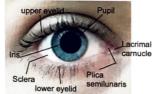
DISORDERS OF THE EYELIDS



 Covers the eyes, lids are moveable folds of skin and muscle that can close over the eyeball

Functions of eyelids

- Protection of ocular surface
- Tear film distribution
- Tear drainage
- Sweeping mechanism to clear debris from cornea

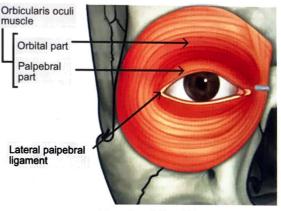


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Important Information

- · Muscle opening eye lid
 - I. LPS (Levator Palpabrae Superioris)
 - 2. Muller's muscle (doesn't elevate > 2mm)
 - 3. Frontalis muscle
- Muscle closing eye lid
 - 1. Orbicularis oculi

Medial palpebral ligament



Muscle of Eye lid

Layers of Eyelid



- From superficial to Deep
- 1. Skin/Subcutaneous tissue
- 2. Orbicularis
 - Concentric bands which close eyelids
 - Function is to assists in tear drainage → Facial N
 - a. Palpebral: Gentle closure (blinking): Has two parts
 - → Pretarsal (tear drainage).
 - → Preseptal
 - b. Orbital: Forced, tight closure
- 3. Orbital septum divides eyelid from orbit
- 4. Fat: 2 fat pads in upper lid, 3 pads in lower
- 5. Levator palpebrae superioris
 - Elevates upper eyelid
 - Innervated by cranial nerve III
- 6. Superior Tarsal (Muller's) muscle
 - o Smooth muscle
 - Sympathetic innervation
 - o Elevates eyelid for 2 mm
- 7. Tarsus: Plate of dense connective tissue, scaffolding, Meibomian glands lie within this tarsal plates
- 8. Conjunctiva: Transparent membrane contains goblet cells, accessory lacrimal glands of Krause and Wolf ring
 - → basal aqueous layer of tear film



Important Information

- Basal aqueous layer of tear film: Accessory lacrimal glands of Krause and wolfring
- · Reflextear: Main Lacrimal gland



Previous Year's Questions

Q. Levator palpebrae superioris is supplied by?

A. 2nd cranial nerve

(JIPMER Dec 2019)

B. 3rd CN

C. 4th CN

D. 6th CN

PTOSIS

· Drooping of upper eyelid





Ptosis

 In resting position Upper Lid covers 2 mm of cornea and Lower Lid is at inferior limbus

Classification of Ptosis

- · Congenital Ptosis: Within 1 year of birth
- Isolated congenital ptosis due to developmental myopathy of LPS



Congenital Ptosis

- 2. Blepharophimosis syndrome
 - o Ptosis,
 - Telecanthus (distance b/w two medial canthus is more than normal)
 - Epicanthus inversus,
 - Shortening of Horizontal palpebral fissure
- 3. Marcus Gunn/jaw winking syndrome
 - Synkinesis → chewing → elevation of ptotic lid

- Congenital ptosis with abnormal movements of upper lid with movements of the jaw
- Due to abnormal connections between 5th nerve controlling the jaw and 3rd nerve supplying the LPS
- Simultaneous contraction of LPS and external pterygoids

Treatment

· Excision of LPS with frontalis sling surgery



Previous Year's Questions

Q. 3-year-old child presents with drooping of upper lid since birth. O/E, the palpebral aperture height is 6 mm and with poor LPS function what is the procedure recommended?

(NEET JAN 2019)

- A. Observation
- B. Mullerectomy
- C. Fasanella servat operation
- D. Frontalis Sling surgery



Previous Year's Questions

Q. Appropriate treatment for mild Congenital ptosis is.

(FMGE DEC 2019)

A. LPS resection

- B. Antibiotics and hot compresses
- C. Tarsalfracture
- D. Wedge resection of conjunctiva
- Acquired Ptosis: Occurs after 1 year of age
- 1. Aponeurotic/Involutional/Senile ptosis
 - o Causes
 - →Trauma
 - → Prolonged contact lens usage
 - →Post-surgical: Stretching of levator aponeurosis leading to †lid crease



Senile ptosis

- 2. Neurogenic Ptosis
 - o CN Ilipaisy

- o Horner's syndrome
- 3. Myogenic Ptosis
 - Myotonic dystrophy
 - Chronic progressive external ophthalmoplegia (CPEO)
 - o Myasthenia Gravis (MG)



Myogenic Ptosis

- 4. Mechanical Ptosis
 - o Chalazion
 - Neurofibromatosis (NF)
 - o Edema: Excessive weight of upper lid



Chalazion

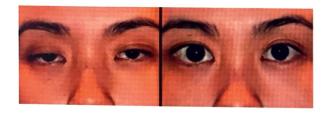
- 5. Traumatic Ptosis: Due to blunt/penetrating
- 6. Pseudoptosis
 - No real ptosis
 - o Examples: dermatochalasis enophthalmos

Clinical Features

- Cosmetic appearance
- Visual field obstruction

Management of Ptosis

- Surgery is done for \u00edvisual fields and cosmetic improvement
- Surgical approaches
- 1. LPS advancement: Done if LPS has good action



- 2. Frontalis sling: Done if LPS has poor action, CPEO, Marcus Gunn ptosis, myotonic dystrophy
- Muller's muscle conjunctival resection (MMCR): For mild to moderate ptosis with good levator action, Horner's syndrome





 Phenylephrine test is done before MMCR to predict success of the surgery.



Previous Year's Questions

Q. A lady presents with ptosis, on eating sipping fluid her ptosis decreases. What is the most likely diagnosis?

(FMGE JUNE 2021)

- A. Mechanical ptosis
- B. Horner's syndrome
- C. Complicated ptosis
- D. Blepharophimosis syndrome

ENTROPION

Inward rolling of eyelids against the eyeball



Entropion

Inward rolling of only eye lashes: Trichiasis

Types

- 1. C-Congenital: Very rare → resolves with time
- 2. C-Cicatricial: Uncommon
- Causes trachoma, OCP, post radiotherapy
- 3. I Involutional: M/c → horizontal lid laxity, disinsertion of lower lid retractors
- 4. S-Spastic: Acute ocular irritation



How to remember

CCIS

Clinical features

- Foreign body sensation
- Photophobia
- Blepharospasm
- **Epiphora**
- Redness
- Discharge

Diagnosis

Snap back test: assesses horizontal lid laxity

Treatment

- Temporary: Lid taping below lower lid
- Everting sutures: Quickert procedure



Jones retractor plication (definitive treatment)

ECTROPION



01:47:22

Outward rolling of eyelids away from eyeball



Ectropion

Types

- 1. C Cicatricial: Uncommon causes are trauma, burns, radiotherapy
- 2. I-Involutional: M/c, horizontal lid laxity
- 3. M Mechanical: Uncommon tumours displace lid away from alobe
- 4. P-Paralytic: Uncommon VII CN palsy



How to remember

CIMP



Previous Year's Questions

Q. A patient presents with complaints of itching of the lid and swelling for the past one month. O/E there are crusting. scaling with small ulcers at the eyelid margin. If untreated it can lead to.

(JIPMER MAY 2018)

- A. Orbital cellulitis
- B. Chalazion
- C. Ectropion and epiphora
- D. Orbital abscess

Clinical features

- Eversion of lid margin
- Irritation
- Congestion
- **Epiphora**
- Recurrent infections
- Keratinization

Treatment

- Horizontal lid shortening using lateral strip procedure
- Retractor reinsertion

BLEPHARITIS



01:50:22

Inflammation of lid margin

Types

- Anterior
 - o Squamous/Seborrheic: 1/3
 - Ulcerative/Staphylococcal: 1/3
 - Mixed: 1/3
- Posterior: Meibomian Gland Disease (MGD)



Blepharitis

Associated conditions

- Dry eyes
- Acne rosacea
- Seborrheic dermatitis
- Demodex mites

1. SQUAMOUS BLEPHARITIS

- Hyperemia of lid border with greasy scales on eyelashes
- Collarettes on base of eyelashes, erythema lid margin



Squamous Blepharitis

Associated with seborrheic dermatitis of scalp

Symptoms

- Burning
- Itching
- Red eyes
- Crusting
- Symptoms worse in mornings

Treatment

- Lid hygiene, warm compresses
- Baby shampoo: Detergent for debris removal

2. ULCERATIVE/STAPHYLOCOCCAL **BLEPHARITIS**

Infection by staph: Ulcers at base of eyelashes

Symptoms

- Burning
- Pain
- Gritty sensation



Ulcerative Blepharitis

On examination

- Injected lid margins
- · Matted hard crusts that bleed when removed: Loss of eyelashes

Treatment

- Topical antibiotic steroid combinations
- Lid hygiene
- Warm compresses

3. MEIBOMIAN GLAND DISEASE (POSTERIOR)

 25 & 30 Meibomian glands in upper & lower tarsus respectively

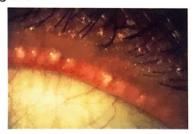


Meibomian Glands

- Meibum is made up of 100 complex lipids, 90 proteins and electrolytes.
- Meibum helps in stabilization of tear film by preventing its evaporation.

Pathology

Chronic, diffuse abnormality of Mg's leads to duct obstruction with qualitative/ quantitative changes in the secretion of gland



Meibomian Gland Duct Obstruction

Obstruction leads to hyposecretion

Symptoms

- **Burning**
- Discomfort
- Tear film instability → visual fluctuation

Treatment

- Warm compresses and lid hygien
- Lubricants
- Dietary changes Omega 3: Omega 6 ratio 1:1
- Oral Doxycycline, Oral Azithromycin (DOC)

SUMMARY OF BLEPHARITIS

-			
(1)	0	2:	0

2:46

Seborrheic	Ulcerative	MGD
Anterior eyelid	Anterior	Posterior
Loss of lash rare	Frequent	None
Lid margin greasy	Fibrin crusts	Foam
Lid ulceration none	Frequent	None

3 glands present in eye

1. Meibum gland

modified sebaceous gland

2. Zeis gland

3. Moll gland → modified sweat gland



Important Information

 Meibomian glands are longitudinal parallel glands opening at posterior margin of eyelid

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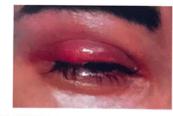
EXTERNAL HORDEOLUM/STYE



- Acute staph infection of Zeis glands at the base lashes
- Seen most commonly in Diabetics and people with uncorrected refractive error due to poor ocular hygiene

Clinical features

- Painful
- Red
- Hot
- Swollen eyelid lump
- Pointing pustule



EXTERNAL HORDEOLUM

Treatment

- Topical antibiotics
- Hot fomentation

INTERNAL HORDEOLUM



Acute staph infection of Meibomian glands within tarsal plates

Symptoms

- Pain
- Redness
- Diffuse eyelid swelling

Treatment

- Warm compresses
- Systemic antibiotics
- Incision and drainage

CHALAZION



Chronic inflammatory granuloma of Meibomian glands

Pathology

 Obstruction of Meibomian orifices prevents exit of secretions leading to lipogranulomatous reaction

Risk factors

- Poor lid hygiene
- Acne rosacea
- Blepharitis

Symptoms

- Painless lump on lid
- Possible blurred vision



CHALAZION

Treatment

- Hot fomentation
- Intralesional triamcinolone injection
- Incision and curettage

Summary

Stye	Internal Hordeolum	Chalazion
Acute	Acute	Chronic
Painful	Very painful	Not painful
Attacks zeis glands	Attacks meibomian glands	Attacks meibomian glands
Rx topical antibiotics	Systemic antibiotics Incision drainage	Incision curettage



Previous Year's Questions

Q. The lipogranulomatous chronic inflammation of the meibomian gland is called.

(JIPMER MAY 2018)

- A. Hordeolum Internum
- B. stye
- C. xanthelasma
- D. Chalazion



Previous Year's Questions

Q. Chronic granulomatous inflammation in Upper lid (Painless swelling) is:

(FMGE DEC 2019)

- A. Internal hordeolum
- B. External hordeolum
- C. Chalazion
- D. Trachoma
- E. None

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Previous Year's Questions

Q. Internal hordeolum is due to inflammation of?

(FMGE Dec 2019)

- A. Meibomian glands
- B. Zeis glands
- C. Lacrimal glands
- D. Moll glands

LID TUMOURS



- Benign: 80%
- Malignant: 20%



Important Information

Signs causing concern for malignant transformation of nevus to melanoma

- A Asymmetry: Two halves of lesion not symmetric
- B-Borders: Irregular borders, pigmentation
- C Colour: Uneven colour changes, white, grey, blue
- D-Diameter: Enlarging size or >6mm
- E Elevation



How to remember

ABCDE

BASAL CELL CARCINOMA

Basal cell carcinoma: M/c lid malignancy

Site

- Lower lid: Medial canthus
- Upper lid: Lateral canthus

Risk Factors

- †age
- Sun exposure
- Whites

On Examination

- Nodule
- Rolled pearly edges
- surface ulceration



Treatment

Wide local excision

SQUAMOUS CELL CARCINOMA

- 2nd m/c eyelid malignancy
- risk of metastasis due to significant tissue destruction

Sine

Prefers lower lid: Medial canthus

Risk Factors

- Sun exposure
- white skin
- X-rays and chemical exposure
- Smoking
- Can arise from Bowen's disease: SCC in situ

On Examination

- 1. Scaly lesion
- 2. Hyperkeratotic patch
- 3. Irregular margins



Treatment

Wide local excision

SEBACEOUS CELL CARCINOMA

- Rare, (5%) aggressive, ↑ mortality rate
- Arises from MG s, Zeis glands
- M/c site Upper lid, caruncle

Risk factors

- †age
- Females

On examination

- Painless
- Solitary
- Firm nodule
- Yellowish

- "Resembles chalazion (Do biopsy of recurrent chalazion)"
 - Hallmark: Pagetoid spread- intraepithelial growth often on conjunctiva, presents as congestion
 - Unresolving u/I conjunctivitis suspect SCC

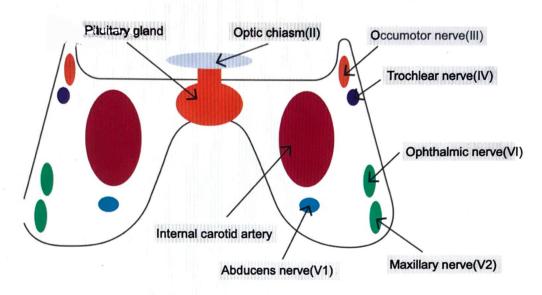


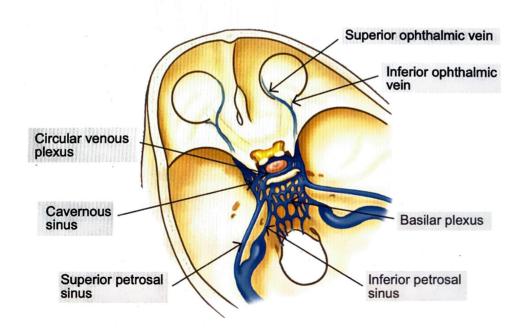
Treatment

MOSH micrographic excision with frozen sections

Images 3.1

Anatomy of the cavernous Sinus









Q. A 7-year-old girl presented to Pediatric OPD with a bump on her eyelid. Her mother reported that it started as a small lesion that has been progressively increasing in size over several weeks. On examination, a lipogranulomatous inflammation was noted. Your probable diagnosis would be

A. Fungal infection

B. Tuberculosis

C. Chalazion

D. Viral infection

Answer: C

Solution

Chalazion

- Lipogranulomatous inflammation.
- In case of recurrent chalazion or seborrheic blepharoconjunctivitis rule out any sebaceous cell carcinoma.
- Incision and curettage is done from conjunctival side.
- Horizontal incisions are avoided to minimize damage to meibomian ducts.

Tuberculosis, Fungal and viral agents cause infection and associated inflammation but not lipogranulomatous inflammation

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, Chapter 2 - Eyelids, page 39

- Q. A 40-year-old female presented with a chief complaint of four days history of right eyelid swelling and redness. The patient denied any trauma, discharge, or change in vision, and reported no systemic symptoms or other complaints. On examination, a non-tender nodule in the tarsal plate of the eyelid was noted. The sclera was clear and there was no sign of conjunctivitis. Which of the following is not true about the treatment of this patient:
- A. Intralesional triamcinolone injection
- B. Oral tetracycline
- C. Curettage is done from skin side
- D. Vertical incisions are given to avoid damage to meibomian ducts

Answer: C

Solution

Treatment for Chalazion

- 1. Intralesional triamcinolone injection in a recent-onset chalazion.
- 2. Oral tetracycline should be used for prophylaxis in recurrent cases.
- 3. Curettage is done from the conjunctival side with the help of a chalazion scoop.
- 4. Vertical incisions are given to avoid damage to meibomian ducts.

Reference: Comprehensive ophthalmology A K Khurana 7th edition Pg 384

- Q. A 64-year-old male came to GH with the complaint of a painless nodule in the lower eyelid for almost a year now. On examination, a nodule with rolled pearly edges with surface ulceration was noted near the medial canthus of the lower eyelid. All of the following are true about the given condition except:
- A. Most common type of malignant eyelid tumor
- B. More common in lower eyelid
- C. Treatment is local surgical excision of the tumour along with a 3 mm area of normal skin with primary repair
- D. Radiotherapy and cryotherapy should be given in operable and inoperable cases

Answer: D

Solution

Basal Cell Carcinoma is the most common type of malignant lid tumour



Basal Cell Carcinoma

- Most common malignant eyelid tumor
- Rodent ulcer/ Pearl ulcer
- More common in lower evelid
- IOC: Biopsy
- Rx: local surgical excision of the tumour along with a 3 mm surrounding area of normal skin with primary repair is the treatment of choice

Radiotherapy and cryotherapy should be given only in inoperable cases for palliaton.

Reference: Comprehensive ophthalmology A K Khurana 7th edition Pg - 401

- Q A 16-year-old female patient presented to an ophthalmology OPD with complaints of loss of eyelashes for the past 3 weeks. All of the following could be a cause for this condition in this patient, except:
- A. Addison's disease
- B. Hypothyroidism
- C. Hyperparathyroidism
- D. Thioridazine toxicity

Answer: A

Solution

Madarosis:

- Loss or decrease in the number of cilia of eyelashes and or eyebrows.
- The common causes include ocular causes such as
 - A. blepharitis, trachoma, local trauma (mechanical, thermal or following radiotherapy or cryotherapy) of the eyelids,
 - B. Tumours of the eyelids and systemic causes like hypothyroidism, hyperthyroidism, hypoparathyroidism,

hyperparathyroidism, hypopituitarism, leprosy, syphilis.

- C. Rubella
- D. Congenital Syphilis; Congenital Leber amaurosis
- E. Batten Mayou Disease
- F. Thioridazine toxicity

Reference: Clinical Methods in Ophthalmology Second Edition, Pg-15

- Q. An 18-year-old male patient presented to ophthalmology OPD with drooping of the upper eyelid. On examination, ptosis was noted with 4 mm of the cornea covered by the upper eyelid. The grade of Ptosis in this patient is?
- A. Mild
- B. Moderate
- C. Severe
- D. Profound

Answer: A

Solution

- In unilateral cases of ptosis, the difference between the vertical height of palpebral fissures of the two sides indicates the degree of ptosis.
- In bilateral cases, it can be determined by measuring the amount of cornea covered by the upper eyelid and then subtracting 2 mm.
- Measurement of the degree of ptosis by measuring the amount of cornea covered by the upper eyelid and subtracting it by 2mm (the normal value)
- Depending on this, ptosis is classified as:
 - a. Mild ptosis: 2 mm
 - b. Moderate ptosis: 3 mm
 - c. Severe ptosis: 4 mm.
- In this question, 4 mm of the cornea is covered by the upper eyelid. Subtracting 2 mm from this means there is 2 mm of ptosis, i.e. mildgrade

Reference: Clinical Methods in Ophthalmology Second Edition, Pg-114



LEARNING OBJECTIVES

Lacrimal apparatus

- LACRIMAL GLANDS
- TYPES OF TEARS
- DRAINAGE OF TEARS
- BLOCKAGE OF LACRIMAL APPARATUS
 - DIAGNOSTIC TESTS
 - PROBING
- NASO LACRIMAL DUCT OBSTRUCTION (NLDO)
 - Types and management
- DACRYOCYSTITIS



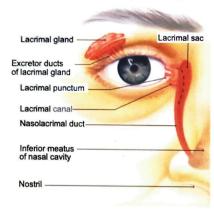
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LACRIMAL APPARATUS

LACRIMAL GLANDS

Ö 00:00:14

- · 2 cm long
- They have 2 parts
- 1. Orbital
- 2. Palpebral
- Function of lacrimal glands is to produce tears
- Tears drain into superior conjunctival fornix
- Tears spread over cornea by blinking



Lacrimal Apparatus



Important Information

- Lacrimal gland in the outer superolateral quadrant of the globe
- Lacrimal sac in medial part of globe

THREE TYPES OF TEARS

1. Basal tears

Produced by accessory glands of Krause and wolfring

2. Reflex tears

Produced by Main Lacrimal gland

3. Psychic tears

- Produced by emotional stimuli
- Secretomotor fibers of lacrimal gland: Greater petrosal nerve (GPN) branch of facial nerve
- Basal tear secretion → 1.2 µL/min
- 90% of tears are reabsorbed from NLD mucosa only 10% get drained out

?

Previous Year's Questions

- Q. Lacrimal gland is supplied through parasympathetic system. It is supplied by which ganglion? (FMGE DEC 2019)
- A. Ciliary ganglion
- B. Otic ganglion
- C. Pterygopalatine ganglion
- D. Submandibular ganglion

DRAINAGE OF TEARS

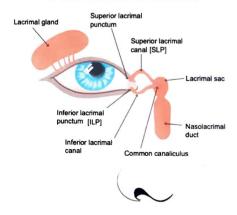


Upper & Lower punctum → upper and lower canaliculus

Common canaliculus

NLD (12-14mm) ← lacrimal sac

Interior meatus of the nose



Drainage of Tears

BLOCKAGE OF LACRIMAL APPARATUS



Epiphora

- Excessive watering of eyes
- Cause
 - 1. Excessive lacrimation
 - 2. Blockage of lacrimal apparatus (m/c)
- Common sites of blockage
 - 1. NLD (M/c)

- 2. Common canaliculus
- 3. Upper/Lower canaliculus



Important Information

- Hasner's valves are present at junction of NLD with inferior meatus
- These don't get canalized and leads to obstruction of NLD

DIAGNOSTIC TESTS FOR SITES OF OBSTRUCTION



- 1. Syringing
- 2. Probing
- 3. Jones test 1 & 2



Important Information

Syringing

- Clear fluid regurgitates from same Punctum: LCB / UCB
- Clear fluid regurgitates from both puncta: Common canalicular block
- Fluid mixed with mucopurulent secretions regurgitates from both puncta: NLD block



Syringing



Previous Year's Questions

- Q. In canalicular blockage, which is true about Jones test findings? (JIPMER MAY 2018)
- A. First test positive, second test negative
- B. First test negative, second test positive
- C. Both positive
- D. Both negative

PROBING

1. Soft stop: stoppage of probe d/t non canalization of valve

- Soft stop can occur due to inferior canaliculi blockage (when passed through ILP) Or Superior canaliculi blockage (when passed through SLP)
 - Common canaliculi blockage



Probina



Important Information

- Canaliculi blockage distinguished by length of the probe
- <10 mm: Lower / upper canaliculi block
- >10 mm: Common canalicular block
- 2. Hard stop: d/t blockage of nasolacrimal duct

JONES TEST 1

Method

 Put 2% fluorescein dye drops in eye, place cotton bud at interior meatus and wait for 5mins

Results

Positive: stained bud → Patent passages



Negative: No stain → Do jones test 2

JONES TEST 2

Method

 Place cotton bud, and do lacrimal syringing and flush the dye

Results

Positive → Stained bud → Lacrimal pump failure



Jones Dye Test 2 (Secondary) positive

Negative → No staining → Mechanical obstruction

NASO LACRIMAL DUCT OBSTRUCTION (NLDO)



- Nasolacrimal duct obstruction is m/c disorder of lacrimal system
- 2 types
 - Congenital
 - Acquired

Acquired NLDO

- M/c cause is inflammation / fibrosis leading to obstruction other causes – Trauma, Sx, Tumor
- Primary acquired NDO-PANDO no cause found
- ROPLAS test: Regurgitation on pressure over lacrimal sac confirms NLDO



Naso Lacrimal Duct Obstruction

MANAGEMENT OF ACQUIRED NLDO Ø 00:31:20

- 1. Canalicular obstruction
 - Conjunctival Dacryocystorhinostomy (DCR) with jones tube placement
- 2. Canalicular DCR
 - At least 8mm of upper canaliculus / lower canaliculus should be patent
- 3. NLD block
 - DCR (Gold standard)

Congenital NLDO



- Almost 6%-20% children are born with obstruction of distal end of NLD [Hasner's valve not canalized]
- Therefore, there is infection of stagnant tears in sac
- · Leading to epiphora, discharge, crusting
- But it usually does not cause any discomfort
- Spontaneous resolution occurs by 1st year for 90% of children

- D/D of congenital NLDO: Congenital glaucoma (photophobia)
- Treatment
 - Lacrimal sac massage
 - → Crigler massage
 - → 3 times/day up to 6 months





Crigler massage

- o Therapeutic probing → If no resolution by 6-10 months
- o DCR (connect sac to the middle meatus of nose)

After 3yrs of age



Previous Year's Questions

Q.A 3-month-old baby presents with mucous exuding from eye on pressing the lacrimal sac. what treatment should be given? (FMGE JUNE 2021)

A. Lacrimal sac massage

- **B. Syringing**
- C. Probing
- D. DCR



Previous Year's Questions

Q. A 5-year-old child presents with chronic epiphora. what is the next step of management? (FMGE JUNE 2021)

- A. Lacrimal sac massage
- B. Tarsal fracture
- C. Lacrimal probing
- D. Dacryocystorhinostomy

DACRYOCYSTITIS



Acute inflammation of lacrimal sac

Cause

Congenital stenosis → stasis of tear

↓ Secondary infection

Dacryocystitis

Symptoms

- Edema
- · Redness and warmth
- · Pain below medial canthus

Diagnosis

 ROPLAS Test: Pressure on the swelling → leads to purulent discharge from punctum & patient winces with pain

Treatment

- Antibiotics: Oral clindamycin
- Incision & drainage for lacrimal sac abscess





Q. True about the test being performed in the given image:



- A. Need instillation of anesthesia before doing the test
- B. Need a ph meter to interpret
- C. Color changes if there is mucin deficiency
- D. Color changes from white to red on contact with tear film and is used to measure the dryness status of eye

Answer: D

Solution

Phenol Red Thread test is the test to detect dry eye severity. When in contact with tear film it changes its colour from white to yellow to red.

Its main advantage over Schirmer test is:

- It is Less time consuming as compared to Schirmer test(15 sec)
- Anaesthesia of cornea is not required
- On the contact of tear strip with the tear it turns red
- Can be done in children easily
- Reflex tearing is minimal
- Differentiates between aqueous deficient & non aqueous deficient eyes

Reference: Satinder VashishtandSativir Singh. Evaluation of Phenol Red Thread test versus Schirmer test in dry eyes: A comparative study. Int J Appl Basic Med Res.; 1(1): 40–42.

- Q. A 21-year-old male patient presented with a complaint of a gunshot wound in the right side of his cheek caused by a bullet fired from an air gun. CT showed a foreign body located on the right side of the skull. It was detected that the pellet penetrated through the anterior and posterior walls of the maxillary sinus without any noticeable injury to the blood vessels or nerve damage. Injury to which of the following structure can cause damage to lacrimal secretion in this patient:
- A. Ciliary ganglion
- B. Pterygopalatine ganglion
- C. Optic nerve
- D. Oculomotor nerve

Answer: B

Solution

Damage to Pterygopalatine ganglion can cause damage to lacrimal secretion

Nerve Supply of Lacrimal Gland

- Sensory Supply: lacrimal nerve
- Sympathetic supply: Carotid plexus of cervical sympathetic chain
- Secretomotor fibres: Salivary nucleus → greater petrosal nerve → synapse at pterygopalatine ganglion → zygomatic nerve → lacrimal nerve-→ lacrimal gland

Reference: Comprehensive Ophthalmology 6th edition Pg 387, AK Khurana

- Q. 26-year-old woman presented to Eye OPD with complaints of foreign body sensations in her eyes. On examination the cornea was clear but tear film deficiency was noted. In general, which of the following could not be a cause for tear film deficiency:
- A. Infiltrative disease of lacrimal glands
- B. Post corneal transplant
- C. Systemic Vitamin C deficiency
- D. congenital absence of meibomian gland

Answer: C

Solution

Causes of Dry Eye Aqueous Deficiency



Aqueous deficiency dry eye also known as keratoconjunctivitis sicca (KCS). Its causes include:

- a. Sjogren's syndrome (Primary keratoconjunctivitis sicca).
- b. Non-Sjogren's keratoconjunctivitis sicca. Causes can be grouped as below:
- 1. Primary age-related hyposecretion is the most common cause.
- 2. Lacrimal gland deficiencies as seen in congenital alacrima, infiltrations of lacrimal gland, e.g., in sarcoidosis, tumours, post-radiation fibrosis of lacrimal gland and surgical removal.
- 3. Lacrimal gland duct obstruction as seen in old trachoma, chemical burns, cicatricial pemphigoid and Stevens-Johnson syndrome.
- 4. Reflex hyposecretion (neurogenic causes) as seen in Familial dysautonomia (Riley-Day syndrome), Parkinson disease, reflex sensory block, reflex motor blade, 7th cranial nerve damage, reduced corneal sensations after refractive surgery and corneal lens wear.

Lipid Abnormality

- Absent meibomian Glands: ectodermal Dysplasia
- Blepharitis
- Meibomianitis

Mucin Deficiency

- Goblet cell destruction: Chemical burns, cicatricial pemphigoid
- Vitamin A deficiency
- Drugs: practolol, Echothiophate

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Other causes of dry eye

Lid problems

- Exposure keratitis
- Entropion
- Ectropion
- Symblepharon
- Large lid notches
- Lagophthalmos
- Keratinised lid margins

Reference: Comprehensive Ophthalmology, 6th edition, pg 389, AK Khurana

Q. A 43-year-old female presented to the eye OPD for evaluation of 2–3 months of right facial swelling. From the examination findings of enlarged lacrimal and parotid glands, a diagnosis of Mikulicz's syndrome was made. Which of the following could not be a cause for this syndrome:

A. Leukemia

B. Lymphosarcoma

C. Sarcoidosis

D. Diphtheria

Answer: D

Solution

Diphtheria is not a recognised cause for Mikulicz's syndrome

Mikulicz's syndrome

Bilateral symmetrical enlargement of lacrimal and salivary glands

Causes

- Leukemia
- Lymphosarcoma
- Benign Lymphoid hyperplasia
- Hodgkin's disease
- Sarcoidosis
- Tuberculosis

Reference: Comprehensive ophthalmology A K Khurana 6th edition Pg 399



LEARNING OBJECTIVES

Lens and Cataract

- BLINDNESS
- Classification of cataract
 - Based on Cause
 - Based on Morphology
- STAGES OF CATARACT
- SYMPTOMS OF CATARACT
- TREATMENT
 - Glasses
 - Surgery
 - → ICCE (intracapsular cataract extraction)
 - ightarrow ECCE With IOL (extracapsular cataract extraction with intraocular lens)
 - → Phacoemulsification
 - → SICS (small incision cataract surgery)
 - → MICS (micro incision cataract surgery)
 - → FLACS (femto laser assisted cataract surgery)
- INTRAOPERATIVE COMPLICATIONS
- POST OP COMPLICATIONS
 - POSTERIOR CAPSULAR OPACIFICATION
 - CYSTOID MACULAR EDEMA
 - POST OPERATIVE ENDOPHTHALMITIS
- PEDIATRIC CATARACT SURGERY
- AMBLYOPIA / LAZY EYE SYNDROME



5 LENS

BLINDNESS



 According to WHO, Blindness is Visual acuity less than 3/60 with best possible correction in better eye



Important Information

Mc cause of blindness in world: Cataract

- 70% of blindness in India
- · 50% of blindness in world

2nd MCC of blindness in world: Glaucoma 3rd MCC of blindness in the world: ARMO MC infectious cause of blindness: Trachoma

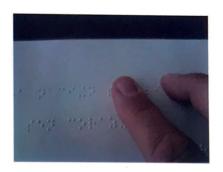
2nd MC infectious cause of blindness: Onchocerciasis

(River blindness)

MCC of blindness in children: Vit A deficiency MCC of ocular morbidity: Refractive errors

 According to NPCB (National Program for Control of Blindness).

6/6 - 6/18	Normal Vision	
6/18 - 6/60	Low vision	
< 6/60	Blind (changed now)	
< 3/60 with best possible correction in better eye	Blind (New definition)	
< 6/60	Economic Blindness	
< 3/60	Social Blindness	
< 1/60	Manifestation Blindness	
No light perception (PL -ve)	Absolute Blindness	





Previous Year's Questions

Q. Most common cause of blindness in India?

(FMGE JUNE 2019)

- A. cataract
- B. Refractive error
- C. Trachoma
- D. Glaucoma



Previous Year's Questions

Q. A patient with VA >1/60, but < 3/60 in his better eye. What type of blindness does he have?

(FMGE JUNE 2021)

- A. Low vision
- B. Economic blindness
- C. Social blindness
- D. Manifest blindness



Previous Year's Questions

Q. A person comes with right eye 6/60 and left eye 3/60. He should be categorized into which type of blindness? (FMGE DEC 2019)

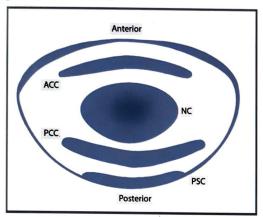
- A. Manifest
- B. Social
- C. Economic
- D. No blindness

CATARACT

- 00:11:19
- Opacification of Lens of capsule
- zommonest cause of blindness

Classification Based on Cause

- 1. Age Related Cataract (>55 years)
 - Previous term: Senile cataract
 - M/C cause of cataract
 - Age group: 50 60 yrs.
 - MC risk factor: UV light exposure
 - Sunglasses provide protection
- 2. Congenital/Developmental Cataract
- 3. Traumatic Cataract
- 4. Complicated Cataract
- 5. Metabolic Cataract
- 6. Heat Cataract
- 7. Radiation cataract
- 8. Drug induced cataract



Types of Cataract

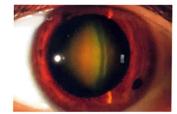
Classification Based on Morphology

- 1. Nuclear Cataract
- 2. Cortical Cataract
- 3. Posterior Sub Capsular Cataract

NUCLEAR CATARACT

(1) 00:14:55

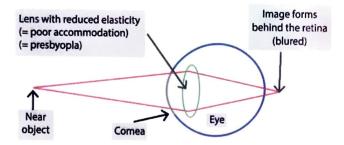
Sclerosis and hardening of nucleus with †yellowing

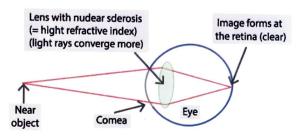


Nuclear cataract

Clinical Features

- Most commonly seen in elderly population
- Hemeralopia
 - Day blindness d/t constriction of pupil
- Decreased distant vision
 - D/t index myopia (distant > near)









Cortical cataract

Hemeralopia

- Second sight
- Recovery of near vision
- Presbyopia resolves itself as nuclear cataract ↑ ses refraction index



Important Information

- Of all the cataracts best vision is preserved in:
 Nuclear cataract
- Second Sight is seen only in: Nuclear cataract
- Of all the cataracts Max visual handicap is seen in: Posterior subcapsular cataract

CORTICAL CATARACT / CUNIEFORM CATARACT



Clinical Features

- Nyctalopia (Night blindness)
- Cuneiform cataract (wedge shaped cataract)

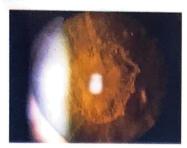
POSTERIOR SUB CAPSULAR / CUPLIFORM CATARACT



Clinical Features

Cause maximum visual handicap

- Have maximum glare (as they are closet to nodal point)
- **Nodal Point**



Posterior Sub Capsular Cataract

- Lies close to the posterior capsule
- Light rays passing through nodal point does not bend
- More posterior the cataract, more drop in vision
- Aka Cupuliform Cataract (Cup shaped cataract)
- |sed near vision [Both Visions are affected (near > distant)]
- Due to miosis in near vision



Glare

AGE RELATED CATARACT



- M/c type of cataract
- † with age

Risk Factors

- UV light exposure: Sunglasses provide protection
- Oxidative stress: Free radicals
- **Smoking**
- Gender: Female
- High body mass index
- Diarrheal/dehydrational crisis
- Nutritional supplements do not prevent cataracts

ZONES OF LENS



Nucleus: 84%, cortex: 16%

Cortex: Peripheral part

Nucleus

- **Types**
 - o Embryonic nucleus: Innermost, represents 1-3 months of gestation
 - Fetal nucleus: 3 months till birth
 - Infantile nucleus: Birth to puberty
 - Adult nucleus from puberty onwards

CONGENITAL/DEVLOPMENTAL CATARACT



00:38:53

- Coralliform Cataract
 - Coral like
 - o No profound loss of vision
- Congenital cataracts are Broadly divided into syndromic and non-syndromic cataracts
- 50% genetic: Mostly autosomal dominant



Coralliform cataract

Types

- Polar, zonular, total, membranous
- a. Polar cataract. Anterior /posterior, limited visuals loss. <3mm in diameter.
- b. Zonular: Lamellar/nuclear/sutural-lamellar
 - o Most common cataract causing loss of vision in children. Most common among zonular is lamellar
- c. Total cataract
 - o Entire lens opaque
 - ↓Visual acuity
 - Nystagmus
 - o Seen in Down's syndrome & congenital rubella syndrome



Zonular cataract

Infectious Causes of Cataract

- **TORCHES**
- M/c cause: Congenital rubella syndrome



Rubella cataract



Important Information

Triad of congenital rubella syndrome

- · Salt & pepper retinopathy
- · Glaucoma
- · Nuclear cataract.

Foveal Fixation

· Critical period for development of fixation: 3 months



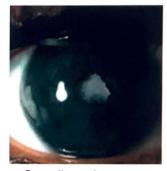
TRAUMATIC CATARACT

- **Ö** 00:46:34
- Mostly by Blunt & penetrating trauma
- Blunt trauma coup and contrecoup injury
- Coup → direct impact → vossius ring
 - Vossius ring is the ring of pigment on anterior lens capsule due to imprint of iris striking the lens capsule
- Contrecoup → shock waves → posterior cortical opacification

Rosette cataract or stellate cataract

COMPLICATED CATARACT

- 00:49:10
- Secondary to intraocular disease
- Caused by disease of eye
 - Uveitis (m/c)
 - o retinitis pigmentosa
 - o Glaucoma
 - o High myopia
 - o Leber's congenital amaurosis
 - o High myopia
 - o Retinal detachment



Complicated cataract

Characteristics

- Mostly posterior subcapsular cataract
- Bread crump appearance
- Shows polychromatic lustre

METABOLIC CATARACTS

00:51:43

Causes

- Diabetes: 5 times more common
- Non-enzymatic glycation of lens proteins
- Oxidative stress
- Polyol pathway activation

Types

Diabetic cataract

Early senile

- Early 40's
- Type 2 DM
- Slow progressive
- Post. Subcapsular

True diabetic /snow flake

- Young patients with uncontrolled diabetes
- Type 1 DM
- Abrupt onset, acute progressive
- Snowflake / snow storm like

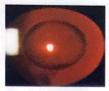


Snowflake cataract

Systemic diseases	Cataract		
Willson's disease	Sunflower Cataract		
Galactosemia	Oil droplet cataract		
Myotonic dystrophy	Christmas tree Cataract		
Fabry's disease	Propeller / Spoke like Cataract		
Atopic dermatitis	Shield Cataract		

 Keyser – Fleisher ring → Characteristic of Wilson's disease







Sunflower cataract

Oil drop cataract

Christmas tree cataract

00:58:24

HEAT CATARACT

- True exfoliation Cataract
- Glass blower's Cataract
- Iron Worker's Cataract





Important Information

Important Causes of Posterior Sub Capsular Cataract

- Steroids
- Complicated Cataract
- Radiation induced Cataract

RADIATION CATARACT



- Ionizing radiation causes cataract
 - o Mc cause is X rays
- Posterior sub-Capsular cataract
- Most sensitive to radiation: Lens



Previous Year's Questions

Q. Most sensitive to radiation is. (JIPMER MAY 2019)

- A. Retina
- B. Optic nerve
- C. Lens
- D. Cornea

DRUG INDUCED CATARACT



- Systemic Steroids (MC cause)
- Posterior Subcapsular Cataract

- Chlorpromazine: Causes stellate Cataract
- Chloroquine
- Amiodarone
- Busulphan
- Penicillamine
- Gold
- Ecothiophate (Strong miotic)



Chlorpromazine cataract



Important Information

- A patient with bronchial asthma came with slow painless loss of vision.
- Diagnosis Steroid induced cataract

STAGES OF CATARACT



- 1. Incipient
- 2. Immature
- 3. Mature
- 4. Hypermature

Incipient Cataract

Earliest stage

Clinical Features

- · Loss of Contrast Sensitivity
- Diplopia (mild)
- Glare
- Minimal Loss of vision (usually 6/6 on Snellen's chart)







Loss of contrast sensitivity

Immature Cataract

- Partial opacification of lens
- Yellowish grey color
- Iris shadow seen
- Vision decreased, VA > 6/60



Immature Cataract

Mature Cataract

- Totally opaque
- White
- Often intumescent (Swell with water)
- Can cause glaucoma
- VA < 6/60



Mature cataract

Hyper Mature Cataract

- Small white lens
- Lens shrinks as water comes out
- Capsule wrinkles
- Phacodonesis (rocking movement of lens) present
- Subluxation/dislocation occurs



Hyper mature cataract



Important Information

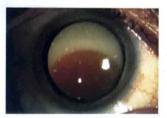
Stage of cataract producing maximum glaucoma is -Mature stage

Types of Hypermature Cataract

Morgagnian cataract

· Cortical Cataract: Hyper mature

- Bag of milky fluid with nucleus floating inferiorly
- Cortex liquefies Nucleus Falls into it inferiorly



Morgagnian cataract

Sclerotic cataract

Nuclear Cataract: Hyper mature

- Cataracta Brunescent
 - Brown in colour due to urochrome
- Pigment
 - 2nd hardest cataract
- CATARACTA NIGRA
 - Black in Color
 - Hardest cataract



Cataracta Brunescent

Slow painless loss of vision

Caused by

- Cataract
- Refractive errors
- Open angle glaucoma
- ARMD
- Diabetic retinopathy

SYMPTOMS OF CATARACT



O 01:21:17

- Slow, progressive, painless loss of vision
- Diplopia/Polyopia
- Coloured Halos (always abnormal)
- Glare
- Loss of contrast
- Changes in colour perception
- More browns and ochre, less blues, and greens



Coloured Halos

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Slit Lamp Examination

- Can be examined from eye lids & eye lashes to anterior 1/3rd of vitreous
- Differentiates different types of cataract
- Color and size of lens is seen



Slit Lamp



Important Information

On slit lamp examination of posterior 2/3rd of vitreous and retina can be done by using supplementary lens like 90D lens or 78D lens.

TREATMENT



- 1. Glasses
- 2. Surgery
 - Indication depends on visual Handicap
 - If there is no problem with daily activities, surgery not indicated

Surgery

- 1. ICCE (intracapsular cataract extraction)
- 2. ECCE With IOL (extracapsular cataract extraction with intraocular lens)
- 3. Phacoemulsification
- 4. SICS (small incision cataract surgery)
- 5. MICS (micro incision cataract surgery)
- 6. FLACS (femto laser assisted cataract surgery)

ICCE (INTRA CAPSULAR CATARACT (5) 01:30:00 EXTRACTION)

- Cataract Lens + Capsule removed
- Leads to aphakia
 - High powered convex lens prescribed
 - For each 1D, convex lens †ses magnification by 2%
 - o For each 1D, concave lens minimizes by 2%
 - Leads to Diplopia due to non-fusion of different sized images





Subluxated cataract

?

Previous Year's Questions

Q. Intraocular lens is implanted in a young adult after cataract surgery which was uneventful. When will you remove the IOL?

(AIIMS MAY 2019)

- A. Remove after 10 years
- B. Remove after presbyopia
- C. After secondary cataract develops
- D. Never removed

APHAKIA

Clinical features

- Deep anterior chamber
- Jet black pupil
- Iridodonesis (trembling movement of iris)
- Diplopia
- Jack in box scotoma
- Pin cushion defect



Jet black pupil

ECCE (WITH PCIOL)



- Extra Capsular Cataract Extraction with Posterior Chamber IOL
- Lens removed; capsule left in situ
 - Anterior capsulotomy done
 - o Cataract lens removed
 - o Artificial Intra ocular lens placed in posterior chamber
- 6/18 vision restored (compared to finger counting at 1mtr in ICCE)
- Leads to Pseudophakia

IOL

- 1. PMMA (Poly Methyl Meth Acrylate) IOL
 - Can transmits about 4 time the normal light

Convex lens glasses

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- Monofocal IOL
- Most commonly used
- One focusing distance, mostly calculated for clear distance vision
- Patient has to wear glasses for near vision



Unifocal IOL

2. Multi Focal IOL

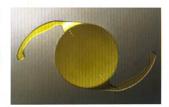
- Expensive
- Have more side effects: ↓ contrast
- Used in selective patients who don't want to wear glasses
- Both near and distance focus, at same time, different powers at different zones



Multifocal IOL

3. TORICIOL

- · Indicated for patient with astigmatism
- Implanted according to corneal markings



Toric IOL

- 4. Accommodative IOL
- Moves back & forth & provides pseudo-accommodation



Accommodative IOL



Important Information

IOL was invented by Sir Harold Ridley



Previous Year's Questions

Q. Advantage of lens over spectacles? (FMGE DEC 2019)

A. Less prismatic effects

B. Protection from UV rays

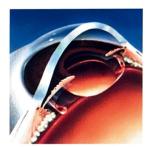
C. Decrease infection

D. Decrease inflammation

PHACO EMULSIFICATION



- Stitch less
- Incision
 - o Less than 3mm in size
 - Valvular/multiplanar incision
 - o Self-Sealing
 - Scleral/Corneal tunnel incision
- Lens emulsification by ultrasonic frequency (40,000/s)
- Foldable IOL: Silicone / Acrylic IOL is used
- Less time consuming; 6/6 vision restored
- Costly



Phacoemulsification



Important Information

Stitching of wound leads to Astigmatism due to irregular curvature of cornea So stitchless surgery is preferred.



Foldable IOL

PHACO INCISIONS

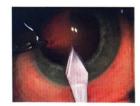
Two types

1. Scleral incision

- 2-3 mm behind limbus
- Preferred in SICS
- Jendothelial count
- incidence of endophthalmitis
- † hyphema

2. Clear corneal incision

- Shorter
- Faster visual recovery
- Wound stability
- Jincidence of hyphema
- †incidence of endophthalmitis
- Wound leaks occur
- †loss of endothelial cells

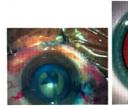


Phaco Incisions

STEPS OF PHACO EMULSIFICATION © 01:57:16

1. Anterior capsulotomy

- Continuous curvilinear capsulorhexis (CCC)
- Creates a central 5-6 mm
- · Capsulotomy large enough to remove cataract,
- Small enough to stabilize IOL.
- Ideal size of capsulorhexis: 5.5 mm
- · Trypan blue dye injected in absence of red reflex





Continuous curvilinear capsulorhexis

2. Hydro dissection

- Injection of Balanced salt solution (BSS) under the anterior capsule
- To separate cortex from capsule
- · Permits rotation of nucleus



3. Hydro delineation

- Injection of BSS/ water into nucleus
- Splits nucleus into epinucleus and endonucleus
- Epinucleus act as protective shell to confine the ultrasonic energy
- Nucleus fragmentation techniques
 - Divide & conquer: Safest, most efficient
 - Phaco chop
 - Stop and chop

4. Irrigation & Aspiration

5. Foldable IOL insertion

- Foldable IOL material
 - o Hydrophobic acrylic: Most preferred
 - Lowest incidence of secondary cataracts
 - o Glistening: Fluid filled microvacuoles within IOL optic
 - o Hydrophilic acrylic: Risk of calcification
 - Silicone IOL: Reduced dysphotopsias
 - → Disadvantage: Avoided in patients undergoing vitreoretinal Sx. Silicone oil may coat IOL

MSICS – MANUAL SMALL INCISION (*) 02:08:25 CATARACT SX

- Alternative to phaco emulsification
- Faster
- Cost effective
- Excellent, outcomes, low complication rates
- Self- sealing cornea scleral tunnel → deliver cataract with IOL implantation
- Sutureless
- Curvilinear partial thickness incision (6-7 mm), depth 0.3 mm
- Large capsulorhexis to prolapse nucleus

Irrigating wire Vectis

Residual cortex aspirated

PCIOL implanted







MSICS

Irrigating wire Vectis

SMALL INCISION CATARACT SX. (SICS) VS PHACOEMULSIFICATION



• 3 mm incision
 Machine is used
 Foldable IOL
 Suture less
 6/6 vision restored
 Costly



Important Information

CSR - No. of cataract surgeries performed per million population. It is 4000-6000 in developed countries.

PHACONIT/MICS/BIMANUAL PHACO



- · Cataract surgery through sub 1 mm incision
- Standard phacoincision < 1.9 mm not possible because of diameter infusion sleeve.
- Titanium tip of phaco hand piece 0.9 mm in diameter, but surrounded by infusion sleeve allowing fluid to pass into eye, which cools the tips to prevent corneal burns



Phaconit

- Sleeve removed from phaco-tip
- Tip passed into eye through 0.9 mm incision
- Side port irrigating chopper held in left hand cold BSS poured continuously on incision site
- Ultra-thin Rollable IOL was implanted through 1.2 mm incision



- Useful in
 - o Mitotic pupils
 - Corneal endothelial disease
 - o PXF
 - Weak zonule

FEMTO LASER ASSISTED CATARACT © 02:19:22 SURGERY (FLACS)

- Femto = 10⁻¹⁵
- Wavelength (): 1053 mm causes photo disruption
- Corneal incision: Limbal 2.2 mm at 3 planes



FLACS

Advantages

- Limbal relaxing incisions to correct astigmatism < 1.5 D with arcuate incisions.
- 2. Anterior capsulotomy: Thousands of small laser spots to create curvilinear opening
- 3. Nucleus fragmentation: Laser induced cavitation bubbles

PHACO VS FLACS



PHACO	FLACS	
 Incision – 2.2 mm keratome Capsulotomy – capsulorhexis Nucleus division – ultrasound 	 Femto laser makes corneal incision Laser shots create opening Laser cavitation bubbles 	

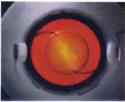
INTRAOPERATIVE COMPLICATIONS © 00:00:54

- 1. Posterior capsule rupture with vitreous loss
- Clear the vitreous from anterior chamber and maintain all capsule possible
- Ideally place IOL in capsular bag
- Other Places for IOL include
- Ciliary sulcus
- ACIOL
- Scleral fixated IOL

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ACIOL

Scleral fixated IOL



Important Information

Important Complication of ACIOL UGH Syndrome

- · Uveitis
- Glavcoma
- Hyphema

POST OP COMPLICATIONS



00:09:44

SECONDARY/AFTER CATARACT/ © 00:10:30 POSTERIOR CAPSULAR OPACIFICATION

- MC (20-50%)
- · Younger the age: Faster the occurrence,
 - o 50-75 years patients develops within 6-8 months
 - o 20-25 years patients develops within 3 months
 - o 6 months 1 vr child develops within 3 weeks
- Other risk factors
 - o DM, uveitis, RP

Pathology

Trauma to LECs (Lens Epithelial Cells)

↓ Stimulate mitosis

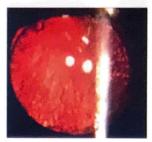
Proliferation of Cells

Migrates & deposits at the center of posterior capsule

Slow & painless loss of vision following cataract surgery

Symptoms

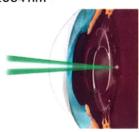
- t contrast, glare, difficulty in reading, slow painless blurring of vision
- No after cataract in ICCE
- Types
- 1. Elshing's Pearls (more common)
- 2. Sommering's Ring



Elshing's Pearls

Treatment

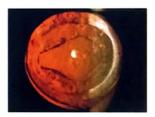
- ND YAG Laser Capsulotomy
 - o Components of ND Yag Laser
 - → ND Neo Dymium
 - → Y-Yttrium
 - → A-Aluminum
 - → G-arnet (mineral)
 - Wave length: 1064 nm



ND YAG LASER

Prevention

- Through cortical clean-up with manual polishing of capsule →Remove all LEC's
- 2. Capsulorhexis diameter slightly smaller than optic IOL
- Use square truncated optic edge IOL design → This mechanically provides a barrier effect preventing LEC growth





Previous Year's Questions

Q. A 60-year-old man underwent phacoemulsification surgery and placement of foldable IOL of left eye a year back has come back with complaints of hazy vision now in that eye. There is no associated redness or pain or watering of eye. The probable cause (JIPMER MAY 2018)

A. Posterior capsular opacification

- B. Cystoid macular edema
- C. Neovascular glaucoma
- D. Chorioretinitis

CYSTOID MACULAR EDEMA

- 00:24:36
- M/c cause of loss of vision following cataract surgery
- Causes slow painless vision, hyperopic shifts, \(\pm \) contrast sensitivity, \(\pm \) reading speed
- · Can occur in 1st month
- Predisposing factors: Diabetes, uveitis, ERM, vein occlusion, posterior capsular rupture.
- Irvine Gass Syndrome: CME after uncomplicated cataract surgery, peaks at 4 to 6 weeks
- Treatment: Topical NSAIDS, topical steroids.



CME

POST OPERATIVE ENDOPHTHALMITIS



- Infection of vitreous cavity (surgical emergency)
- Hall mark: Progressing vitritis
- · Earliest sign: Retinal periphlebitis

Early Onset Endophthalmitis

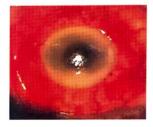
Late Onset Endophthalmitis

- < 6 weeks (mc within 3 odays)</p>
 - 6 weeksCauses
- Cause

- Propionibacterium acnes [mc]
- o Staph, epidermidis (MC)
- Staph aureus

Clinical Features

- Immense pain,
- Redness
- Lid edema
- Hazy Cornea
- Hypopyon
- Hazy media
- Dramatic loss of vision (< 72 hrs, from 6/6 vision to hand movements)



Endophthalmitis

Treatment

- DOC
 - Intravitreal Vancomycin (For Gram positive organisms)
 - Intravitreal Ceftazidime (For Gram negative organisms)/Amikacin
- Intracameral (into anterior chamber) route can also tried
- Pars plana vitrectomy (when vision is very poor VA < PL)
- Most important Precaution: Eye lid & Eye lash cleansing with povidone lodine



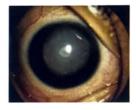
Important Information

Maximum macular toxicity: GENTAMYCIN

PEDIATRIC CATARACT SURGERY



- Surgery is only done for cataracts with
 - o Central opacity > 3mm,
 - Nystagmus
 - Strabismus
- Timing of Surgery depends on
 - o Bilateral Cataract
 - → 6-8 weeks, one week apart
 - → after foveal fixation
 - Unilateral Cataract
 - → ASAP, 4-6 weeks of birth
 - → Prone for AMBLYOPIA / LAZY EYE



Pediatric cataract

Syndrome

- Surgery: Lens aspiration / Lensectomy with posterior capsulotomy with anterior vitrectomy
- IOL implantation: after 1 year of age
- Preferred IOL material is hydrophobic acrylic
 - Least incidence of secondary cataract



Pediatric Cataract



Important Information

Formula for correction in case of pediatric cataract

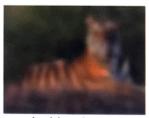
- Under corrected 20% for < 2 years of age
- Under corrected 10% for 2-8 years of age

AMBLYOPIA/LAZY EYE SYNDROME (†) 00:48:26

- M/c cause visual loss in children
- JVA is not attributable to structural Abnormality of eye



Normal eye



Amblyopic eye

- Lesions present in LGB
- Risk factor
 - premature baby
 - o small for GA
 - developmental delay

Definition

- · Best corrected visual activity (BCVA) 6/12 one eye, difference BCVA 2 lines or more
- Critical period: 8 years
- Blurred image: disrupts normal visual development
- Binocular rivalry becomes abnormal

Types

- 1. Strabismic Amblyopia: Constant, U/L, esotropia
- 2. Anisometric amblyopia: Difference of 3.00 / + 1.500 / 2.0 DC b/w two eyes
- 3. Ammetropic amblyopia: 5.00/+4.50 D/2.0 DC
- 4. Meridional amblyopia: Uncorrected astigmatism > 1.0 D
- 5. Stimulation deprivation amblyopia: Cataract, corneal, opacity, ptosis

Characteristics

- J V.A, despite correcting underlying disorder
- Crowding phenomenon: Better with single optotypes
- ↓ V.A with neutral density filters less in amblyopia



Filter lens

Important Information

Rx

Upper age limit to Rx amblyopia – 8-10 years

Rx: of amblyopia

1. Glasses (refractive adaptation)

- Period of visual improvement
- Plateaus at 3 months

2. Occlusion

- Close the good eye
- Patch 6 hours a day
- 7-12 years of age: Patching

 V.A, even if Rx before
- 13-17 years of age: Patching ↑ V.A, when not treated before
- Penalization: With weekend atropine, equally active



Occlusion



Previous Year's Questions

Q. A 10-year-old child is diagnosed with amblyopia in one eye. What will be the best treatment?

(FMGE JUNE 2021)

- A. Observation
- **B. Penalisation**
- C. Occlusion
- D. None of the above

SUMMARY





Important Information

- Cataract wit max.
 Post. Sub capsular cataract visual handicap
- Cataract with highest
 Post. Polar cataract possibility of capsular rupture
- •IOL with least incidence Hydrophobic acrylic IOL of secondary cataract
- ·Cataract with max. · Intumescent cataract fluctuation of vision

ECTOPIA LENTIS

- **©** 01:12:57
- Displaced position of crystalline lens
- · Marfan's syndrome
 - M/c ocular manifestation of marfan's syndrome is ectopia lentis
 - M/c location: Supertemporal dislocation
- Homocystinuria
- · Weil marchesani syndrome



Ectopia Lentis: Supertemporal



Important Information

- Marfan's syndrome: Supertemporal dislocation of lens
- Homocystinuria: Inferonasal dislocation of lens

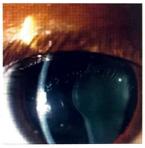
ABNORMALITIES OF LENS SHAPE



01:15:09

Lenticonus: Conical lens

- 2 types
- 1. Anterior lenticonus
 - o Anterior surface of lens become conical
 - Seen in Alport's syndrome



Anterior lenticonus

- 2. Posterior lenticonus (m/c)
 - Posterior surface of lens become conical
 - o Seen in Lowe's syndrome
- Oil droplet appearance on lenticonus



Oil droplet lenticonus

Spherophakia

- It is spherical lens
- Seen in marfan's & Weil Marchesani syndrome
- Triad of spherophakia
 - o Shallow AC
 - o High myopia
 - Angle closure glaucoma due to subluxation and pupillary block
- Inverse glaucoma: IOP † with pupil constriction

Weil Marchesani syndrome

- S-Short
- S Stubby
- S Stupid
- S Spherophakia
- S Subluxation



How to remember

• 5





- Q. A 5 month old infant is brought to ophthalmology OPD by her parents as she noticed opacification in both eyes since birth.

 On examination her eyes appeared as shown in the image. Which of the below is not a cause for this condition?
- A. Trisomy 21
- B. Stickler syndrome
- C. Lowe's syndrome
- D. Marfan's syndrome

Answer: D

Solution

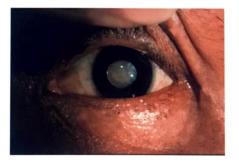
Marfan's syndrome is associated with subluxation of lens as a result of zonular weakness and not congenital cataract. Cataract may develop earlier in the subluxated lens as compared to others of same age but not congenital cataract. Stickler Syndrome is associated with quadrantic lamellar cataract though less common.

CAUSES OF CONGENITAL CATARACT:

- 1. Maternal malnutrition, infantile malnutrition (Zonular cataract).
- 2. Maternal infection e.g. (like rubella, toxoplasmosis, CMV).
- 3. Placental hemorrhage.
- 4. Drugs: Thalidomide, Corticosteroid.
- 5. Congenital Syndromes: Down's, Hallerman Streiff, Lowe, Galactosemia, trisomy 13-15, cockayne, Trisomy 21, Goldenhar, Cerebro oculo facial syndrome.
- 6. Congenital condition like Aniridia.

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, 2019, Chapter 10 - Lens, page 335-341.

Q. The patient in the image is a 70 year old male , complaints of gradual painless loss of vision. On examination, visual acuity of right eye is 3/60. Fundus is not visible . What is the most likely diagnosis?



- A. Retinoblastoma
- B. Phacomorphic glaucoma
- C. Cystoid macular edema
- D. Mature senile cataract

Answer: D

Solution

Stages of maturation of cataract

- 1. stage of lamellar separation glare is seen
- 2. stage of incipient cataract uniocular diplopia and colored halos are seen in this stage.

I. Cuneiform cataract

- This cataract starts at periphery and extend centrally so visual changes are noted at late stage
- Night blindness (nyctalopia)

II. Cupuliform cataract

- Posterior capsular
- This cataract lies right in the pathway of the axial rays and thus causes early vision loss
- Day blindness (hamarlopia)

III. Immature senile cataract

- In this stage iens may become swollen due to continued hydration. This condition is know as intumescent cataract.
- Phacomorphic glaucoma is cause by this type of cataract; it is type of secondary glaucoma and it is most common type
 of lens induced glaucoma

IV. Mature senile cataract

Lens become pearly white in colour and complete opaque. know as ripe cataract

V. Hypermature cataract

· Seen in any two of forms

I. Morganian cataract

- The cortex become liquefied and lens settles at the bottom altering its position with change in the head position
- Phacolytic glaucoma associated with morganian cataract

II. Sclerotic type

- The nucleus become diffusely cloudy or tinted due to deposition of pigments. In practice the commonly observed pigmented nuclear cataracts are either amber, brown(cataracta brunescens) or black (cataracta nigra) and rarely reddish (cataracta rubra) in colour
- Most common complication is subluxation of lens

Reference: AK khurana 7th edition pg 203

- Q. A 37-year old female presented with gradual painless loss of vision in both eyes over the past three months. She was a known case of rheumatoid arthritis and has been on steroid treatment for the past two years. Her IOP was within normal limits and red reflex is absent on both eyes. What is the most probable diagnosis?
- A. Open angle glaucoma
- B. Age-related Macular degeneration
- C. Diabetic Retinopathy
- D. Cataract

Answer: D

Solution

The diagnosis of the patient is steroid induced cataract as the patient is on medication for Rheumatoid arthritis.

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Drug induced Cataract

- Corticosteroids(Most common cause)
- Chlorpromazine
- Unioroquine
- Amiodarone
- Busulphan
- Penicillamin
- Gold

Reference: Comprehensive ophthalmology AK Khurana Pg 193



LEARNING OBJECTIVES

UVEITIS

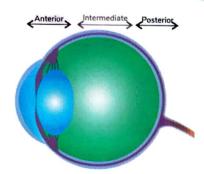
- Types of uveitis
 - Causes and signs Anterior UVEITIS (M/c ~70%)
 - → Keratic Precipitates (KP)
 - → Iris Nodules
 - → Synechiae
 - → Hypopyon
 - Causes and clinical features of Intermediate Uveitis
 - → Floaters / Muscae
 - → Snowballs & Snow Bank Appearance
 - Causes and clinical features of Posterior Uveitis
 - → Infectious and immune causes
 - → HEAD LIGHT IN FOG APPEARANCE
 - Pan Uveitis
 - → SYMPHATIC OPHTHALMITIS (PAN UVEITIS)
 - → VOGT KOYANAGI HARADA SYNDROME
- Treatment of Various types of uveitis
- Difference in diff types of uveitis
- FUCHS HETEROCHROMATIC IRIDOCYCLITIS (PHI)
- OCCULAR HIV
- DRUG INDUCED UVEITIS
- OCULAR SARCOIDOSIS



6 UVEITIS

INFLAMMATION OF UVEA





Uveitis classification

UVEA = Grape

Components	Inflammation	
• Iris	• Iritis	
 Ciliary body 	Cyclitis	
 Choroid 	Choroiditis	
 Whole Uvea 	 Pan uveitis 	

CLASSIFICATION



- 1. Anterior UVEITIS (M/c ~70%)
- Iritis
- Iridocyclitis (inflammation of iris and pars plicata of CB) (more common)
- 2. Intermediate Uveitis
- Pars Planitis
- Vitritis
- 3. Posterior Uveitis
- Choroiditis
- 4. Pan Uveitis
- Sympathetic ophthalmitis
- Vogt koyanagi Harada Syndrome



Important Information

- · Acute Uveitis: Acute onset
- · Chronic Uveitis: > 3 months
- Granulomatous Uveitis: TB, Leprosy, Syphilis, Sarcoidosis etc.
- Non-Granulomatous Uveitis

ANTERIOR UVEITIS

Causes



- 1. IDIOPATHIC (M/C, 50%)
- 2. HLAB 27 Spondylo Arthropathies
- a. Ankylosing Spondylitis (2nd M/c) 'flip flap' → Commonly seen in tall young males
- b. Inflammatory Bowel disease
 (Ulcerative colitis & Crohn's disease)
- c. Psoriatic Arthritis
- d. Reiter's Syndrome (Reactive Arthritis)
- Chlamydia is the M/c cause of reactive arthritis
- Triad
 - o C Conjunctivitis.
 - U Uveitis.
 - o R-aRthritis



How to remember

• CUR



Ankylosing Spondylitis Uveitis

Reiter's Syndrome



Important Information

- A young tall male with complaint of back pain and red painful eyes. Most probable diagnosis would be Ankylosing spondylitis
- 3. Juvenile Rheumatoid Arthritis (Jra) / Juvenile Idiopathic Arthritis
- Pauciarticular, ANA positive, Rf negative
- Contra indication for IOL implantation (as eye is already inflamed and IOL is a foreign body)
- White eye uveitis



JRA - White Eye Uveitis



Important Information

- Commonest cause of anterior uveitis in adults: Idiopathic
- Commonest cause of anterior uveitis in children: JRA

Clinical Presentation

- **Ö** 00:16:58
- Acute painful red eye with loss of vision

Signs

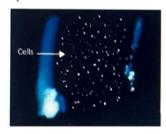
00:17:26

- 1. Circumciliary Congesion
- Bluish red in colour (as inflammation is of Anterior ciliary vessels which he in slightly deeper plane)
- Radial



Circumciliary Congesion

- 2. Cells
- WBC, neutrophils floating in Anterior Chamber
- Elicited by slit lamp examination
- Earliest sign
- Hallmark of activity



Cells in anterior chamber

3. Flare

- Protein deposition in aqueous humor
- Presents in severe uveitis

Blood vessels get inflamed

Serum protein leaks out & mixes with aqueous humor

Turbid Aqueous Humor

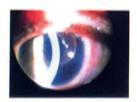
Splits the light Flare



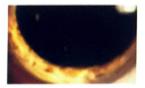
Flare

Keratic Precipitates (KP)

- Neutrophils & lymphocytes stuck on cornea
- Present in inferior half of cornea
- ARLT'S Triangle: Triangular area in the inferior part of cornea where KPs get preferentially stuck



KP's



ARLT's Triangle

- Diffuse KPs
 - KPs attached all over the cornea
 - Seen in
 - → Herpetic uveitis
 - → Fuch's Heterochronic Iridocyclitis
- Mutton Fat KPs
 - Look like mutton fat (large and greasy)
 - o Seen in Granulomatous uveitis
 - → TB, Leprosy, Syphilis, Sarcoidosis



Mutton Fat KPs

- 5. Iris Nodules
- a. Koeppe's Nodules: Present on papillary margin
- b. Busacca's Nodule: Present on surface of Iris. Seen in Granulomatous disease



Koeppe's Nodules Busacca's Nodule

- 6. Synechiae
 - a. Anterior Synechia
- Iris gets stuck with cornea
- · Blocks the angle and IOP
- May cause glaucoma
- b. Posterior Synechia
- Iris gets stuck with Lens
- May cause cataract
- Festooned Pupil
 - Small irregular pupil
- 7. Miosis
- 8. Lów lop Initially
- 9. Hypopyon
- Collection of pus at bottom of AC
- Features of severe uveitis



Festooned Pupil

Hypopyon



Previous Year's Questions

Q. Iritis is seen in all except?

(INICET NOV 2020)

- A. SLE
- B. Rheumatoid Arthritis
- C. Behcet's disease
- D. Psoriatic arthritis
- E. Ulcerative colitis

INTERMEDIATE UVEITIS

· Rare, chronic, relapsing



Intermediate Uveitis

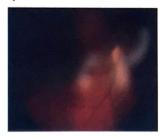
Causes

- Idiopathic (M/c~ 70%): Pars planitis
- Sarcoidosis (2nd M/c)
- Multiple Sclerosis

Clinical Features

- 1. Floaters/Muscae
- Black spots floating in front of eye

- Look like house flies: Musca
- D/t neutrophils in vitreous cavity
- Seen in
 - Normal people
 - Vitreo retinal disorders
- Loss of Vision → Commonest cause → Cystoid macular edema (CME)
- 3. Snowballs & Snow Bank Appearance
- Seen Typically in Pars Planitis



Snowballs & Snow Bank Appearance

POSTERIOR UVEITIS (CHOROIDITIS)



Causes

Infectious causes	s Immune Disorders	
 Toxoplasmosis (M/C) TB (2nd M/c) Toxocariasis, Herpes CMV, HIV 	Sarcoidosis (M/C immune)PANScleroderma	

Clinical Features

- Presents as
 - o Chorio retinitis
 - o Vasculitis
 - o Vitritis
- O/E
 - Creamy yellow patched seen → Choroiditis
 - o Chorioretinitis
 - Vitritis
 - Vasculitis
 - o head light in fog appearance seen
 - → Chorio retinitis + Vitritis
 - → Chorioretinitis → Head light
 - → Vitritis
- \rightarrow Fog
- → Occurs in Toxoplasmosis





Choroiditis

Headlight in Fog

00:35:54

TREATMENT



1. Anterior Uveitis

- a. Topical Steroids
- DOC
- Side effect
 - Glaucoma: As topical steroids inhibit the degradation of the extracellular matrix material in the trabecular meshwork leading to biological oedema and inadequate drainage of aqueous humor
 - Glaucoma occurrence depends on AIP (Antiflammatory Potency): AIP Glaucoma causation



Important Information

- Topical Steroid causes Glaucoma
- Systemic Steroid causes Cataract



Important Information

- Steroid causing Max Glaucoma: Dexa Methasone
- Steroid causing Minimum Glaucoma:
 Fluorometholone (also called as soft steroids)

b. Cycloplegics

Cycloplegics	Duration of action	
Atropine	14 days (most potent)	
Homatropine	3 days	
Cyclopentolate	1-day	
Tropicamide	6 hrs (least potent)	

- Relaxes ciliary spasm (reduces pain)
 - o DOC for Acute anterior uveitis
- Dilates the pupil, breaks synechiae thus preventing complicated cataract
- Reduces vascularity
- Homatropine is preferred drug
 - Have action for 3 days
- Atropine Ointment
- Preferred in children as they have strong ciliary tone so require drugs with strong cycloplegic action
- Atropine drops are not used as drops are:
 - o 20% absorbed in cornea

- o 80% absorbed in systemic circulation
- Leads to Atropine toxicity
- Phenylephrine is a pure dilator not a cycloplegic



Phenylephrine drops

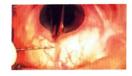
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Important Information

- DOC for Anterior uveitis: Topical steroids
- DOC for Acute Anterior uveitis: Cycloplegics

2. Intermediate Uveitis

- a. Steroid injections
- Triamcinolone
- Only injectable steroid in the eye
- By Subconjunctival route or



Subconjunctival route

 By sub tenon's route (better but riskier as more chances of scleral perforation)



sub tenon's route

3. Posterior uveitis

- a. Anti-microbials for infectious disease
 - DOC for Toxoplasmosis during pregnancy: Spiromycin
 - Highly active antiretroviral therapy (HAART) for HIV
 - Ganciclovir for CMV
 - ATT for TB

b. Systemic Steroids for Non-Infectious Causes

Not to be given for > 3 months due to side effects



Moon face with steroid

	Anterior uveitis	Intermediate uveitis	Posterior uveitis
	•lridocyclitis	Pars PlanitisVitritis	• Chorioretinitis
Cause	 Idiopathic 	 Idiopathic 	Toxoplasmosis
Symptoms	PainRednessLoss of vision	 No Pain No Redness Loss of vision Muscae / floaters 	PainRednessLoss of vision
Signs	• Cells	 Snowballs & snow banks appearance 	 Toxoplasmosis Headlight in fog appearance Viritis Vasculitis Chorioretinitis
Treatment	Topical steroids cycloplegics	• Inj. Triamcinolone	Systemic steroids anti microbiods

FUCHS HET EROCHROMATIC IRIDOCYCLITIS (PHI)

- 01:11:45
- Usually U/L Low grade anterior uveitis Seen in young age
- Idiopathic, chronic
- Rubella virus is the cause

Triad

- Heterochromia
- Cataract
- Diffuse stellate KPs



Fuch's Heterochromatic Iridocyclitis



Important Information

- KP's in uveit is are present in triangular shape called alt's triangle
- · KP's are diffusely present over all of the endothelium in FHI and Herpetic iridocyclitis
- Low grade inflammation
- No remarkable Symptoms (Only Floaters are present) until cataract formation leading to loss of vision
 - Presents as young patient with U/L cataract
- Amsler's Sign: Paracentesis (entering AC) induces Hyphema

Treatment

Topical/systemic steroids should be avoided I

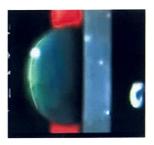


Important Information

- Vision threatening complications of FHI
- Cataract
- Glaucoma
- Vitreous opacification

POSNER SS/GLACUOMATOCYCLITIC © 01:19:30 **CRISIS**

- Rare, recurrent, unilateral attacks of uveitis in young to middle aged males
- Mild attacks of uveitis with minimal flare, white KPs
 - Marked †IOP, out of proportion as compared to the uveitis
- ↑IOP → Corneal edema → leading to mild loss of vision



Corneal edema and KP's

- Between attacks the eye is normal
- Association with CMV is seen

Treatment

- Anti-glaucoma drugs
- Topical steroids

OCCULAR HIV



- M/c ocular manifestation is Retinal microangiopathy. It includes
 - Cotton wool spots or
- Soft exudates or
 - HIV retinopathy
 - Haemorrhages, microaneurysms
- Commonest ocular infection: CMV retinitis
- M/C ocular tumor: Kaposi Sarcoma > Non-Hodgkin Lymphoma > ocular surface squamous neoplasia
- M/C systemic infection in HIV
 - M/C Cause of death in HIV

Tuberculosis

- Commonest cause of soft exudates: Diabetes / HTN Retinopathy
- Commonest ocular side effect of HAART Therapy: Immune Recovery Uveitis (anterior/intermediate)



Kaposi Sarcoma

CMV RETINITIS

- 01:27:22
- M/c cause of blindness in AIDS
- CD <50 cells /µl
- 30% in pre-HAART < 5% in HAART era
- Symptoms are
 - Floaters
 - Photopsia
 - Loss of vision
- 3 patterns
 - o Pizza pie appearance
 - o Brushfire appearance
 - o Frosted branch angiitis
- Treatment
 - HAART
 - o Oral valganciclovir



CMV Retinitis

IMMUNE RECOVERY UVEITIS



Paradoxical worsening of intraocular inflammation

Due to HAART

M/c complication of HAART



Important Information

 M/c ocular complication of HAART is Immune Recovery Uveitis (30-60%)

Pathology

Immune system recovers → attacks CMV

Ocular inflammation prominent in vitreous

Risk factors

CDH count †100 cells /µl, young patients, iv cidofovir

Symptoms

- Floaters
- **Photopsiae**
- Visual loss
- Redness
- Pain
- Posterior synechiae
- Cataract
- Vitritis
- Optic disc edema
- Epiretinal membrane (ERM)

Treatment

HIV

Periocular and intravitreal steroids

OPPORTUNISTIC INFECTIONS OF



 Tuberculosis Bacterial Syphilis Cryptococcus **Fungal** Candida

Viral (Most common)

Herpes zoster

CMV

Parasitic

Pneumocystis

Toxoplasmosis

DRUG INDUCED UVEITIS



Drugs causing uveitis	Used for treatment of
Rifabutin	MAC in HIV
• Cidofavir	CMV retinitis in HIV
• Bisphosphonates	Osteoporosis
 Prostaglandin analogues (Latanoprost) 	Glaucoma
 Metipranolol 	Most common betablocker causing Uveitis
 Sulphonamides 	Bacterial Infections, Burn dressing

OCULAR SARCOIDOSIS



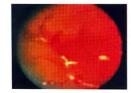
- 25% Sarcoidosis have ocular involvement
- M/c in coloured races / Scandinavians
- Bilateral granulomatous chronic uveitis
- Can cause
 - Anterior uveitis (M/C)
 - o Intermediate uveitis
 - Posterior uveitis
 - Pan uveitis



Mutton fat KP's

7 ocular signs

- Mutton fat KPs
- o Tent shaped peripheral anterior synechiae (PAS) / Trabecular meshwork (TM) nodules (Berlin's nodules)
- Vitreous opacities → 'string of pearls'
- Candle wax drippings (Tache de bougie)



Candle wax drippings



Important Information

Nodules in uveitis

- 1. Kopppe's nodules: Present on Pupillary margin
- 2. Busaca's Nodules: Present on surface of iris
- 3. Berlin's nodules: Present on trabecular meshwork

Investigation

- Chest x- ray (Single best investigation)
- Elevated ACE and soluble IL2 receptors (SIL-2R)

Treatment

Topical steroid with cycloplegic drugs



Important Information

 M/c cause of loss of vision in Sarcoidosis: Cystoid macular edema (CME)

PAN UVEITIS



SYMPHATIC OPHTHALMITIS (PAN UVEITIS)

- B/L granulomatous pan uveitis
- Caused by Trauma to One Eye
 - Injured eye: Exciting eye
 - o Other eye: Sympathising Eye

Common causes

- Penetrating/perforating injury
- Metallic foreign body
- Injury to ciliary body



Important Information

- Dangerous Area of eye is Ciliary body
- As injury to ciliary body Causes max. Sympathetic ophthalmitis

Pathology

Injury to C.B

Liberation of Uveal pigments

Traumatised pigments acts as foreign body (antigens)

Antigen - Antibody reaction

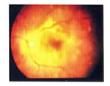
Granulations inflammation of injured eye (Uveitis)

Within 2 weeks antibodies attack ciliary of healthy eye

Uveitis of both eye

Clinical Features

- Presents at 2 weeks 2 months after injury
- Dalen Fuch's Nodules on choroid seen
- Most commonly seen in children
- Earliest symptom
 - Photophobia in good eye
 - Accommodation failure in good eye
 - o Paradoxical decrease in near vision of good eye
 - Earliest sign
 - → Retrolental flare (vitreous flare)
 - → Mutton fat KPs
 - → Hypotony



Kaposi Sarcoma

Treatment Injured EyeNormal EyeManagement

Injured Eye	Normal Eye	Management
• PL: - ive	• 6/6 Vision	 Enucleation of injured eye within 14 days of trauma
• 6/60 vision	• 6/6 vision	Repair of injured eye
• PL: - ive	 Progressed sympathetic ophthalmitis 	High dose systemic steroids for sympathetic eye



Previous Year's Questions

Q. Which out of them is a prerequisite for development of sympathetic ophthalmitis?

(NEET JAN 2020)

- A. Penetrating trauma to eye
- B. Blunt ocular trauma
- C. Chemical
- D. Infection

VOGT KOYANAGI HARADA SYNDROME



- B/L granulomatous pan uveitis with skin, neurological / auditory involvement in pigmented races, in absence of ocular trauma or Sx.
- M/c in young females (20-50 years)

Systems involved in VKH syndrome

- N Neurological
 - Auditory symptoms mainly (Loss of hearing, Tinnitus, Vertigo)
 - Neck stiffness
 - Headache
- O Ocular
- D Dermatological



How to remember

NOD

Stages

1. Prodromal Stage

- Mimics viral infection, CNS headaches, weak stiffness, hearing loss, tinnitus, vertigo
- 2. Acute Stage
 - · B/L panuveitis with serous retinal detachment
- 3. Chronic convalescence stage
 - Poliosis, vitiligo, alopecia, depigmentation of choroid: "sunset glow fundus".
- 4. Chronic recurrent stage
 - Recurrent granulomatous anterior uveitis, mutton fat KPs.



Chronic convalescence stage

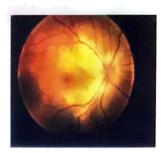
Ocular signs

- 1. B/L panuveitis
- 2. Posterior uveitis with multifocal choroiditis

Leading to serous retinal detachment

- 3. Optic disc edema and hyperemia
- Sugiura's sign: Perilimbal vitiligo





Sugiura's sign

Optic disc edema (VKH)

Treatment

· High dose systemic steroids





A 32-year-old male presented to your clinic with a complaint of a red, watery and painful left eye for 2 days. From the given history, you are suspecting Acute iritis in this patient. If you do an examination of the eye, the pupil would be:

- A. Normal
- B. Dilated
- C. Constricted
- D. Vertically oval

Answer: C

Solution

- In acute iritis (inflammation of iris) iris is irritated and the pupil constricts due to the stimulus of irritation
- A vertically oval, mid-dilated, non reacting pupil is seen in acute angle-closure glaucoma.

Reference: AK KHURANA PG NO. 159

- Q. A 44-year-old man who was recently diagnosed as a case of HIV positive was started on Highly Active Anti-Retroviral Therapy (HAART). The use of HAART in this patient can lead to the development of:
- A. Keratitis
- B. Anterior Uveitis
- C. Poterior Uveitis
- D. Optic neuritis

Answer: C

Solution

Immune Recovery Uveitis

- With the initiation of HAART, there is an upregulation of pro-inflammatory cytokines like IL-2, IL-6 and TNF alfa and the T lymphocytes fraction (increase to a range of 100 cells/mm³).
- Present understanding is that with an increase in the body's mechanism to mount an immune response, the body
 mounts a response to previously quiet and hence undiagnosed opportunistic infections such as CMV retinitis.
- Clinical manifestations can be varied and most commonly involve the posterior segment of the eye, though anterior, intermediate uveitis is also reported. Papillitis may also be a feature.

Reference: PARSON'S BOOK 22ND EDITION:- PAGE NO 247

- Q. A 55-year-old male presented to the ophthalmology OPD with complaints of redness, photophobia, decrease in vision for the last 2 months. He gave the history of contact with cats. On examination, there was severe vitritis associated with a focal necrotizing Chorioretinitis on the posterior segment. Which of the following is the most likely cause for this condition?
- A. Toxocara
- B. Toxoplasma gondii

- C. Taenia solium
- D. Plasmodium falciparum

Answer: B

Solution

- Toxoplasmosis is the most common cause of posterior uveitis and accounts for approximately 90 % of focal necrotizing retinitis.
- Causative agent Toxoplasmosis gondii.
- H/O contact with cat faeces
- C/F Vitritis with underlying white-yellow choroiditis patch 'Headlight in Fog Appearance'

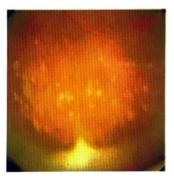
Other causes of Posterior Uveitis

- Infections
- Toxoplasmosis
- TB(2nd M/C/CAUSE)
- Toxocariasis
- CMV, HIV

- Immune Disorders
- Sarcoidosis
- PAN
- Scleroderma

Reference: A.K. Khurana ophthalmology 7th e/p -170

Q. A 30 years old lady, who has been diagnosed with Multiple sclerosis presented with redness, blurred vision associated with floaters, and also pain in both eyes. On fundus examination, the following finding was noted (shown in the image). Your probable treatment would be



- A. Cycloplegics
- **B.** Analgesics
- C. Steroids
- D. Antibiotics

Answer: C

Solution

The fundus finding shown in the image is **Snowball & snow banking appearance** which is a feature of **intermediate uveitis**

Intermediate uveitis

- Causes: Idiopathic (MC); Sarcoidosis (2nd MC); Multiple sclerosis
- Treatment
 - o Intravitreal or Posterior Subtenon's Steroid injections: Triamcinolone acetonide
 - Steroids may also be given by subconjunctival route or by Subtenon's route

Reference: A.K. Khurana ophthalmology 7th e/p -166



LEARNING OBJECTIVES

GLAUCOMA

- Triad of Glaucoma
- TONOMETRY
- PRIMARY CONGENITAL GLAUCOMA
- PRIMARY ADULT GLAUCOMA
 - Primary Open Angle Glaucoma
 - Primary Angle Closure Glaucoma (PACG)
 - POAG V/s ACUTE ACG
- VISUAL FIELD
- VISUAL FIELD DEFECTS IN GLAUCOMA
- OPTIC DISC
- OTPIC DISC CHANGES IN GLAUCOMA
- GONIOSCOPY
- MANAGEMENT OF OAG and PACG
 - ANTI GLAUCOMA DRUGS
 - SURGICAL MANAGEMENT
- SECONDARY GLAUCOMA
- GLAUCOMA DRAINAGE DEVICES / SETONS



7 GLAUCOMA

2nd MCC of blindness in the world

Classic Triad of Glaucoma (At least 2 out of 3 → © 00:05:04 Glaucoma)

- 1. ↑IOP (> 22 mm of Hg)
- 2. Visual field Defects
- 3. Optic Disc Changes



Important Information

- I. If only IOP is raised but visual field defects and optic disc changes are not seen it is called as ocular hypertension
- If visual field defects and optic disc changes are seen without increase in IOP it is called as Low tension glaucoma/Normaltension glaucoma



Previous Year's Questions

Q. A 50-year-old male with family history of glaucoma presents with headache. IOP is 22 and 24. angles open on gonioscopy, no field defects noted. True statement regarding management of this patient. (JIPMER MAY 2018)

- A. Normal tension glaucoma treat it
- B. POAG to be treated
- C. Ocular hypertension no intervention
- D. Ocular hypertension to be treated

Primary glaucoma	Secondary glaucoma
Cause unknownMore common	 Approx. 30-40 Known causes Uveitis (ant. Chronic uveitis causes max glaucoma)
	NeovascularLens inducedTraumaSteroid induced



Important Information

Causes of Glaucoma in Anterior uveitis

- Anterior synechiae blocking flow of aqueous humor
- · Steroids + 10P
- Trabecular meshwork block

TONOMETRY



Tonometer	Features
Goldman applanation	Gold standard, most accurate
Perkins's tonometer	• Tonometer of choice for children
Mackay – Marg	Used for scarred corneas
Tono pen	 Can be used over cataract lenses, portable, scarred corneas
 Dynamic contour tonometry (DCT) 	 least dependent on variables like CCT, Ac depth, AL
• i-care rebound	For self-tonometry



TONOMETRY

PRIMARY GLALICOMA

00:13:40

- Childhood
 - Congenital (Buphthalmas): From birth till 3 months
 - o Infantile: 3 months to 3 years
 - Juvenile: > 3yrs of Age to 45 years
- Adult > 45 vrs
 - o Open Angle Glaucoma (DAG)
 - Angle closure Glaucoma (ACG)

2.differentiated by Gonioscope (measure the angle)



Gonioscopy

PRIMARY CONGENITAL GLAUCOMA & 00:17:49



- M/c of all childhood glaucoma's
 - o From birth till 3 months
 - Buphthalmas (Large eye)
 - Barkan's Membrane: Blocks aqueous flow
 - AR (autosomal recessive)
 - o A/w consanguineous marriage

Classical Triad

- 1. Lacrimation
- 2. Photophobia
- 3. Blepharospasm
- 4. Decrease vision, nystagmus, myopia, strabismus

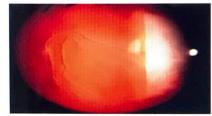
Sians

- Large eye (>11 mm in newborn, >12 mm in children, under 1 year of age, >13mm at any age)
- Hazy cornea d/t corneal edema
 - o †IOC overcomes the endothelial pump resistance



Hazy cornea

HAAB'S Striae



HAAB'S Striae

- Perkins's tonometry: Gold standard for IOP
- CDR > 0.5 in any age is suspicious

Previous Year's Questions

Q. A one month comes with watering eye and cloudy cornea. Identify the diagnosis? (NEFT 2021)

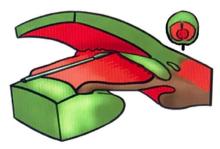


- A. Cataract
- B. Congenital glaucoma
- C. Ophthalmia neonatorum
- D. Mucopolysaccharidosis

Treatment

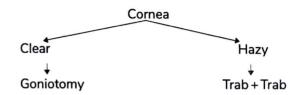


- 1. Medical management
 - ß- blockers first line
- 2. Brimonidine is contraindicated
- 3. Gonjotomy (Surgery of choice)
 - Safer but rare
 - Gonioscopy is difficult d/t Hazy cornea



Goniotomy

- 4. Trabeculotomy
- 5. Trabeculotomy + Trabeculectomy (TRAB + TRAB)



Previous Year's Questions

Q. A 2 year old child with watering of eyes with bilateral proptosis and photophobia, what may be the diagnosis?

(AIIMS NOV 2018)

- A. Congenital glaucoma
- B. Retinoblastoma
- C. Congenital endothelial dystrophy
- D. Megalocornea



Previous Year's Questions

Q. A 50-year-old male with family history of glaucoma presents with headache. IOP is 22 and 24, angles open on gonioscopy. no field defects noted. True statement regarding management of this patient.

(JIPMER MAY 2018)

A. Normal tension glaucoma-treat it

B. POAG - to be treated

C. Ocular hypertension - no intervention

D. Ocular hypertension - to be treated

PRIMARY ADULT GLAUCOMA



Open Angle Glaucoma

- 3-4 time more common than PACG
- Glaucoma ≈ OAG
- More dangerous
- Highest in Africans

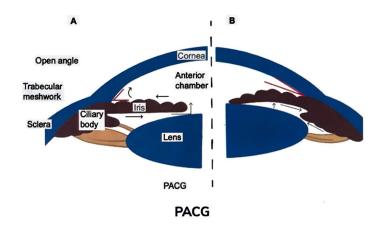
Primary Angle Closure Glaucoma (PACG)

- More painful
- More common in Asians
- Highest in Inuit
- Has 3 times more risk for developing B/L blindness
 - PACG occurs when iris mechanically blocks TM and CB which leads to †IOP
 - Iris pushed from behind (more common) or pulled from in front

Definition

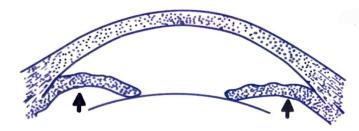


 A condition with > 180 degrees of iriodotrabecular contact (ITC), with PAS, with raised IOP, with optic neuropathy



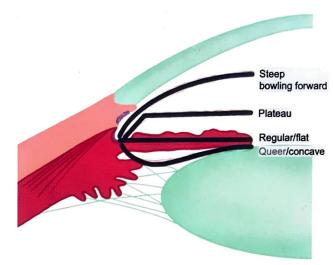
Mechanism

 Relative pupillary block: Apposition of iris to lens impedes aqueous flow from PC to AC leading to PCG



Relative pupillary block

- Non pupillary block: Occurs When patient has plateau iris
 - Configuration: Iris displaced anteriorly by abnormal ciliary processes, AC depth normal centrally, Shallow peripherally
 - Syndrome: Peripheral iris bunches up, blocks TM persisting narrow angle despite a patent peripheral iridotomy



Types of Iris Insertion

Clinical classification

- 1. Subacute/intermittent
 - Abrupt †IOP, mild symptoms, self- limiting and recurrent
- 2. Acute
 - Abrupt onset of †IOP (> 40 mmHg) due to total closure of angle, not self limiting
- 3. Chronic
 - †IOP from angle closure glaucoma, asymptomatic, angle closes slowly, insidious †IOP, mistaken for POAG

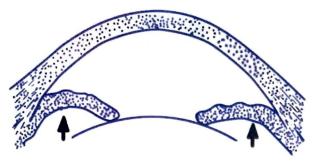
ACUTE ANGLE CLOSURE GLAUCOMA



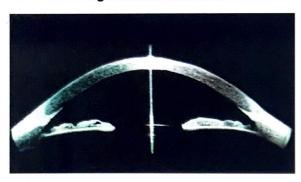
Risk factors

- M/c seen in Middle Ages and women because of growing lens and shallow A.C respectively
- 2. Asian origin
- 3. Hypermetropia
- 4. Fellow eye having ACG

5. Emotionally unstable women



Angle closure Glaucoma



Normal Angle



Angle closure

- Late night attack
- Mid-dilated pupil
- Pupillary Block d/t mid dilated pupil as max contact between iris and lens occurs in mid dilatation



Important Information

Diameter of pupil

Normal

-3-4 mm

Fully dilated

-9-10 mm

Mid dilated

-6-8 mm

- In 30 min, IOP changes from 15 mm \rightarrow 60 mm (4 times the Normal pressure)
 - Severe pain
 - Loss of vision

Acute Angle closure glaucoma

Vomiting

Acute congestive glaucoma

· Relative seal traps aqueous

- Iris TM block
- Sudden rise in IOP

Symptoms



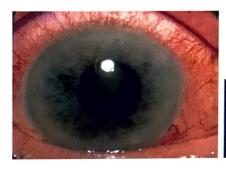
d/t corneal edema

00:51:00

- Severe pain
- Colored halos
- Sudden drop in vision
- Frontal headache
- Nausea/vomiting

Signs

- Closure of angle
- Conjunctival injection
- ↑IOP: stony hard eye
- Corneal edema: "Steamy cornea"
- Vertically oval, mid-dilated, non-reacting pupil



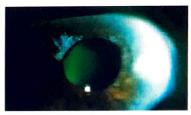


VOGT's triad: Tells us about the past attack

00:57:30

- 1. Iris atrophy
- 2. Pigment dispersion
- 3. Glaucomaflecken





Glaucomaflecken

Iris atrophy

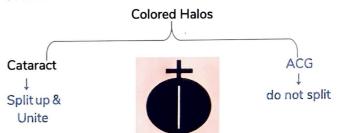
AC Angle Assessment

1. Van Herick technique



- Peripheral temporal depth of AC <1/4 normal corneal thickness is suspicious
- 2. Gonioscopy: Gold standard
- 3. UBM (Ultrasound bio microscopy)

Fincham's Test



PRIMARY OPEN ANGEL GLAUCOMA / SILENT THIEF OF SIGHT 00:00:13

Risk Factors

- 1. Coloured Races
- 2. Thin corneas
- 3. Myopia
- 4. Increasing age
- 5. Trabecular meshwork fibrosis
- Myocilin gene (MYOC)
- 7. Optineurin gene (OPTN)

Pathology

- Fibrosis of Trabecular Meshwork
- Trabecular outflow blocked
- Max site of resistance
- Juxta canalicular meshwork
- Very slow painless loss
 20-30 yrs of vision
- N aqueous production
- 2.5 µL/min¹
- C' value N value
- 0.2 µL/min/mm of Hg
- Above 2 values will

 d/t

 trabecular blockade



Fibrosis of Trabecular Meshwork

Symptoms

- · Painless d/t very slow onset
- · No corneal Edema as it is a slow process and endothelium develops new pumps to pump out the aqueous humor
- No Colored Halos
- Initially no loss of vision

Can be diagnosed earlier by

- Progressive risk of IOP +nt (may get unnoticed) >40 yrs, measurement of IOP to be done every year
- Perimetry should be done

By the Time of Presentation

Tunnel Vision +nt (End Stage Glaucoma)

Aka Silent Thief of sight (slowly progressive in nature)



- Only Symptom
 - Frequent Change of Presbyoptic Glasses
 - → Every 6-8 months
 - → Normal Frequency: 3 yrs
 - o ↑ Dark Adaptation Time





Important Information

- Frequent Change of Presbyopic Glasses: OAG
- Frequent change of Distant Vision Glasses: Cataract
- Frequent change of Glasses in Young: Keratoconus> Pathological Myopia



Keratoconus



Previous Year's Questions

Q. All are drugs given in primary open angle glaucoma except. (INICET Nov 2020)

- A. Latanoprost
- B. Pilocarpine
- C. Physostigmine
- D. Phenylephrine



Previous Year's Questions

Q. A patient of Primary open angle glaucoma with a known case of bronchial asthma. What is the Drug of choice? (NEET Sep 2021)

- A. Latanoprost
- B. Carboprost
- C. Alprostadil
- D. Gemeprost

POAG V/s ACUTE ACG



Ö 00:17:08

Acute ACG	POAG
 Female predominance 45 years (middle aged) Hypermetropic Sudden, painful Coloured halos Pupillary block 	 No gender predisposition Elderly Myopic Slow, painless No symptoms Trapecular fibrosis / TM blockage

VISUAL FIELD



00:18:27

Superior & Nasal \rightarrow 60°; inferior \rightarrow 70°; temporal \rightarrow 100°

- Area of space visible to human eye without moving the eye from a central target.
- · Measured by Perimeter [Humphrey's Perimeter (Gold Standard)]



Humphrey's perimeter

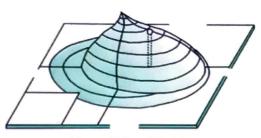
Humphrey's Field Testing

- Rays from Inferior Field focus on Superior Retina
- Rays from Superior Field focus on Inferior Retina
- Rays from Temporal Field focus on Nasal Retina
- Rays from Nasal Field focus on Temporal Retina



Important Information

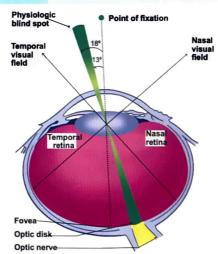
 Physiological Blind Sport is located in Temporal Visual Field as optic disc lies in Nasal retina



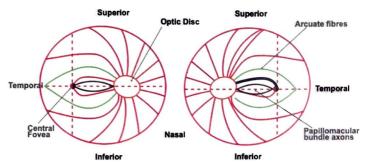
Normal Visual Field

Retina and Visual Filed Papulomacular bundle

Papulomacular bundle	 Information from macula to optic disc 	 Central scotoma and paracentral scotoma and centrocecal scotoma
Arcuate fibres	 Information from the temporal half of retina to superior and inferior Pole of optic disc 	Bjerrum's ScotomaSeidel's scotomaArcuate scotoma
Nasal fibre	 Directly into optic disc called as sup/inf. Nasal radiating fibres. 	Temporal wedge defects



Retina and Visual Filed

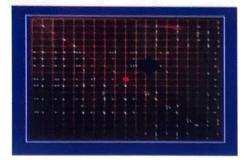


Retinal nerve fibre layer

VISUAL FIELD DEFECTS IN GLAUCOMA

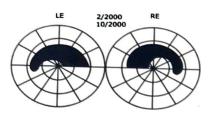
00:30:20

- 1. Para Central Scotoma
- · Earliest Visual field defect
- Localized loss of vision surrounded by normal vision



Para Central Scotoma

- 2. Bjerrum's Scotoma: Scotoma in Bjerrum's area
- Bjerrum's Area
 - An extension of blind spot in the shape of an arc in the central 30°
 - Occupied by the arcuate nerve fibres
 - o These are the 1st attacked nerve fibres in glaucoma
- Characteristic field defect of glaucoma



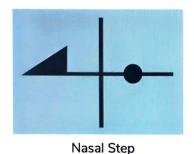


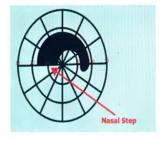
Bjerrum Scotoma



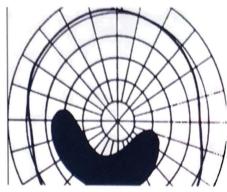


- 3. Generalized Constriction of Field / Concentric Contraction of Isopters
 - · Constriction visual field in all directions
- 4. Nasal Step: Characteristic field defect of glaucoma



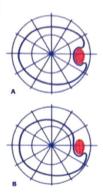


Siedel' Scotoma: Sickle shaped extension of the blind spot



Seidel' Scotoma

6. Baring of Bling Spot



7. Arcuate Scotoma: Scotoma in shape of an arc



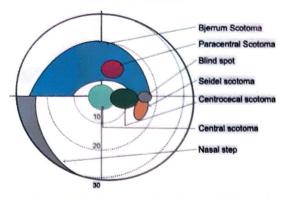
Arcuate Scotoma

8. **Temporal wedge defect**: Wedge shape field defect in the temporal area of retina

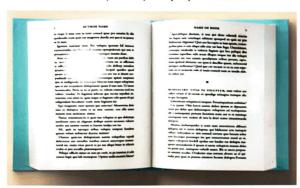


Temporal wedge defect

GLAUCOMA FIELD DEFECTS



- No particular order in formation of visual field defects
 - Mostly the earliest is paracentral scotoma
 - Rule of Thumb: Earliest VFD in glaucoma is always begin from close to centre (paracentral > Nasal step > Bjerrum > Generalised constriction of field)
- Characteristics
 - Supero-Nasal Fields are first destroyed & Temporal vision is last to be destroyed
 - Most scotomas are Arc shaped
 - o Follow horizontal meridian
- Negative Scotomas
 - Seen in optic nerve disorders
 - o Glaucoma scotomas are negative scotomas
 - → Can't observed d/t cortical filling in
 - → Example: Blind spot
 - → Measured by Humphrey's perimeter



Negative Scotomas

- o Positive scotomas are seen in Retinal disorders
 - → Patient can see scotoma



Positive scotomas

OPTIC DISC





Optic cup

 Allow blood vessels to enter & exit the eye, No function perse occupies 30% of the area of OD

NRR

- Contains neurons of optic Nerve
- Each optic nerve contains 1.2 M neurons
- In Glaucoma, NRR surface area gradually decreased
- occupies 70% of the area of OD

Cup Disc Ratio (CDR)

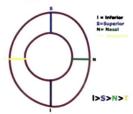
- CDR = (Area of Cup)/(Area of Disc)
- Normal CDR = 0.3 (Range: 0.3-0.6)
- CDR: > 0.7 → Glaucoma
- CDR = 1 → NRR is completely lost → Glaucomatous optic atrophy
- CDR indicates the NRR damage

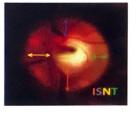


Normal Optic disc

Examination of Optic disc

- Evaluate with Slit lamp & 90 D lens
- Sequence of Width decrease
 - o ISNT Rule (Inf. Sup. Nasal & Temporal)
 - 1. Inferior: widest
 - 2. Superior
 - 3. Nasal
 - 4. Temporal: Narrowest
- ISNT Rule broken in glaucoma (d/t vertical ovalization of the cup)





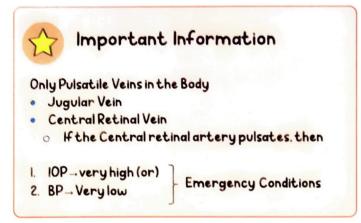
Normal Optic Disc

Normal Optic Disc

O 01:18:14

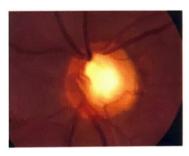
O 01:19:40

- Clear margin
- Colour of NRR: Reddish pink colour
- OD: 0.3 to 0.6
- · Distribution of vessels
 - 2 Central Retinal Artery: Relatively narrow
 - o 2 Central Retinal Vein
 - → More dilated
 - -> Pulsates
 - o 1 artery & 1 vein: Nasal
 - o 1 artery & 1 vein: Temporal

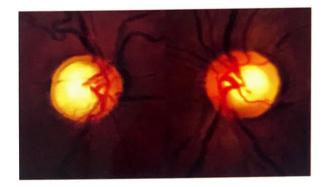


OTPIC DISC CHANGES IN GLAUCOMA

1. ↑CDR > 0.7



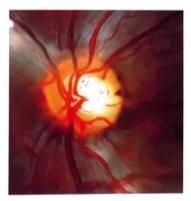
- 2. Asymmetry in CDR > 0.2
- 3. OD Pallor



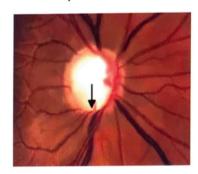
4. Splinter Haemorrhage / Drance haemorrhage



- 5. Focal notching
- 6. Nasalization of OD (apparent shift)
- Laminar DOT Sign:
 † no. of dots d/t more exposed lamina cribrosa



- 8. Bayonetting
- Apparent discontinuity of blood vessel

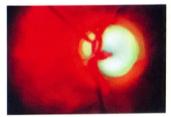


- 9. Peripapillary atrophy
- Presence of β zone

10. Glaucomatous Optic Atrophy







Advanced cupping

Ö 01:32:02

GONIOSCOPY

- Examines angle of eye
- Critical angle: 46 degrees
- Direct lenses: Koeppe, swan Jacob
- · Indirect lens: Goldmann 3 mirror

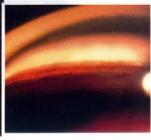




Lens

Gonioscopy





Grading

Normal eye gonioscopy

Structures Lying in the Normal Angle

- From cornea to iris
 - **1.** S Schwalbe's Line (Represents the termination of Descemet's membrane on to cornea)
 - 2. T-Trabecular Meshwork (Band)
 - S Scleral Spur (Line)
 - 4. C Ciliary Body Band
 - 5. I-Iris root



How to remember

Structures lie in the normal angle from iris to cornea ICan See the Stuff

Grading

- Grade IV
 - o Angle is 40°
 - o All 4 structures can be seen
 - Usually do not close (no ACG)
- Grade III
 - o Angle is 30°
 - o Ciliary band cannot be seen, other 3 structures seen
 - Usually do not close (NO ACG)
- Grade II
 - o Angle is 20°
 - Schwalbe's line & Trabecular meshwork seen
 - Scleral spur & ciliary band not seen
 - Can progress to ACG
- Grade I
 - Only structure seen is → Schwalbe's line
 - High risk for ACG
- Grade 0
 - ACG occur right now

 Schwalbe's line also not seen (not even a single structure is seen)

MANAGEMENT OF OAG

Ö 01:47:52

Medical Management

↑ Outflow	↓ Production
Pilocarpine	β blockers
PG Analogues	Carbonic anhydrase inhibitors

- Surgical Management
 - ALT (Argon Laser Trabeculoplasty)
 - 2. Trabeculectomy

ANTI GLAUCOMA DRUGS

Cholinergic Agonist



- Examples
 - Pilocarpine/Carbachol/Echothiophate
- Mechanism
 - Contracts the ciliary muscle pulls the scleral spur away

↑ Trabecular outflow

- But ↓ uveo-scleral outflow
- Side effect of Pilocarpine
 - o Uveitis
 - Ciliary spasm
 - → Ocular pain Present
 - → Loss of distant vision
 - Pseudomyopia
 - o Punctal stenosis
 - Retinal detachment (Therefore, avoided in young myopes)
 - o Cataract



Important Information

Pilocarpine decreases uveoscleral outflow

Blockers



- † Production by 20-30% during daytime
- Non-Selective
 - o Timolol
 - Levobunolol
 - Carteolol
- Selective
 - o Betaxolol
- Side effect

- C/I in Bronchial Asthma, COPD
- C/I in Arrythmias
- Dry eyes
- Depression
- Nasolacrimal Duct Block



Important Information

A patient presents to ER with bronchial asthma. Patient gives history of taking anti-glaucoma drugs. Drug responsible for patient's' condition is Timolol



Previous Year's Questions

Q. Beta blockers should be avoided in all the conditions except. (FMGE June 2021)

A. Glaucoma

B. Peripheral vascular disease

C. Diabetes

D.COPD

Adrenergic agonists

- Epinephrine/Dipivefrin
- ↑Trabecular outflow
- J Production
- o S/E

02:04:54

Dual Mechanism

Systemic **Ocular**

- Sweating
- **Palpitation**
- **Tachycardia**
- HTN
- Nervousness
- **Tremors**
 - C/I in ACG, HTN

- CME in aphakia
- **Pupil dilatation**
- Stinging
- Blepharoconjunctivitis
- Adenochrome deposits

- Dipivefrine
 - Prodrug of epinephrine
 - Only intraocular S/E
 - C/I in ACG

α2 agonist

Brimonidine

02:08:20

- ↓ production
- † Uveo sclera outflow
- Side effect
 - C/I children (d/t apnoea & death)
 - **Drowsiness & depression**
 - Neuro Protection
 - Contraindicated in MAO inhibitors & TCA
- Apraclonidine
 - Side effect
 - Tachyphylaxis
 - Follicular conjunctivitis
 - Blepharoconjuctivitis (maximum)
 - → Maximum allergy causing drugs (40%)
 - → Short term, JIOP, posterior LI, Capsulotomy
 - Less selective α2 agonist
 - α1 effects like pupil dilatation, lid retraction, vasoconstriction



Important Information

Drug causing maximum blepharoconjuctivitis is Apraclonidine > Brimonidine

Carbonic Anhydrase Inhibitors

- 1 Production
- Systemic: Acetazolamide/methazolamide
- Topical: Dorzolamide & Brinzolamide
- Acetazolamide
 - Side effect
 - → C/I in Sulfa Allergy (contains sulfa group)
 - → Hypokalaemia
 - → Acidosis
 - → Chronic Renal Failure and kidney stones
 - → Hepatic Failure (C/I)



Important Information

- A chronic liver failure patient presents with drowsiness, disorientation, and coma on treatment with antiglaucoma drugs. Drug responsible for patient's present condition is Acetazolamide.
- Acetazolamide causes hypokalaemia leading to hepatic encephalopathy in patient with liver failure as already has electrolyte disturbances.
- Dorazolamide & Brinzolamide
 - Safest
 - DOC in children

- Side effect
 - → Corneal Decompensation († corneal edema)
 - Punctate keratopathy



Important Information

- Brinzolamide is a DOC in children for glaucoma
- · Brimonidine is C/lin children



Previous Year's Questions

- Q. The ocular hypotensive agent causing apnoea in infants is. (NEETJAN 2019)
- A. Latanoprost
- B. Timolol
- C. Retaxolol
- D Brimonidine

Prostaglandin (PGF2α) Analogues



02:23:26

- †uveo scleral out flow
- Commonly used PGF2 Analogues
 - Latanoprost
 - Bimatoprost (most powerful antiglaucoma effect)
 - Travoprost
 - Tafluprost
- DOC for OAG & Normal Tension Glaucoma
- Most Potent antiglaucoma drugs
- Side effect
 - Uveitis, CME.
 - o Iris Hyperchromia (irreversible)
 - o Blepharoconjunctivitis
 - o Trichomegaly (common with Brematoprost)







Trichomegaly

Iris Hyperchromia

Previous Year's Questions

Q. what is the DOC for decreasing IOP by increased uveoscleral outflow in a patient with increased IOP and optic disc changes with ciliary congestion? (NEET Jan 2020)

A. Latanoprost

- B. Dorzolamide
- C. Pilocarpine
- D. Timolol



Previous Year's Questions

- Q. Which of the following are true? (CIOS VON 2MILA)
- A. Latanoprost is used with caution in patients of bronchial asthma
- B. Topiramate can cause bilateral angle closure alaucoma
- C. Methazolamide causes decrease in ocular blood flow
- D. central scotoma is seen in open angle glaucoma

Hyperosmotics



O 02:30:14

- Indicated for treatment of ↑IOP
- S/E and limited duration of LIOP: Not for chronic use



Mannitol

- Mechanism
 - Vitreous humor contains: 98% of water
 - Increase the osmolarity of blood causing extraction of water from vitreous humor causing shrinkage of vitreous volume decreasing IOP
- Mannitol
 - o Given IV
 - Fastest acting antiglaucoma drug (within 20 min)
 - DOC for Acute ACG
 - Side effect: Decompensation in case of CHF
 - → Glycerol/Isosorbide
 - Oral Syrup
 - o Glycerol: Hyperglycaemia, nausea and vomiting
 - o C/lin DM
- Isosorbide is oral hyperosmotic of choice

Previous Year's Questions

Q. All are drugs which lower IOP except?

(CIOS VON 2019)

- A. Clonidine
- B. Mannitol
- C. Dexamethasone
- D. Acetazolamide

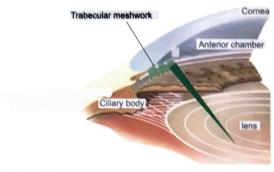


Previous Year's Questions

- Q. Intravenous mannitol is indicated in? (NEET JAN 2019)
- A. Primary open angle glaucoma
- B. Acute angle closure attack
- C. Normal tension glaucoma
- D. Sympathetic ophthalmitis

SURGICAL MANAGEMENT

ALT (Argon Laser Trabeculoplasty)



Trabeculectomy

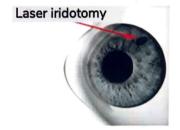


MANAGEMENT OF PACG



00:03:45

Peripheral Iridectomy (PI) / Laser iridotomy (LI)



ND-YAG Laser Iridotomy (Better)



Important Information

Other use of PI/LI

- Rx of Secondary cataract
- PI/LI are done in opposite eye in case of ACG (prophylactic)



ND-YAG Laser Iridotomy

ACG MX Algorithm

- 1. JIOP
 - Mannitol (DOC)
 - Acetazolamide
 - Glycerol

Systemic Drugs



How to remember

- MAG
- 2. Prophylactic PI/LI of Second Eye
- 3. LI of Attacked EYE
 - Drugs
 - o B-blockers
 - o α2 agonist
 - CAI inhibitors
 - Trabeculectomy

SECONDARY GLAUCOMA



00:15:29

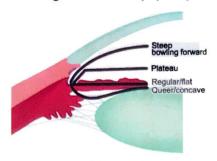
1. PIGMENTARY GLAUCOMA

- Secondary OAG in pigment dispersion syndrome
- Risk factors
 - Young age
 - White
 - Males
 - Myopics



Pathology

- Rubbing of iris against zonules leads to pigment deposits on TM
- Concave Iris → touches zonules → Dispersion & Settlement of pigments on Iris
- Posterior bowing iris → reverse pupillary block



Posterior bowed iris

Clinical Features

- Krukenberg spindle
- Radial transillumination iris defects
- Pigmentation of trabecular meshwork
- Scheieligeuripe
- Sampaolesis line: Anterior to schwalbe's line





Krukenberg spindle

Radial transillumination iris defects

- †IOP: Coloured halos, blurring of vision, pain
- Exercise induced glaucoma
- DOC
 - Miotics (pilocarpine)
 - Argon laser Trabeculoplasty
 - o Trabeculectomy

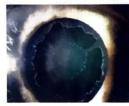
2. PSEUDO EXFOLITATION GLAUCOMA



- MC Secondary glaucoma worldwide
- MC cause of U/L glaucoma

Pathology

- Secondary OAG leads to deposition of fibrillar, protein material in TM: ↑↑IOP
- Elderly Females, Scandinavian





3 Ring / Target Sign

Moth Eaten Defect & Pseudo exfoliation flakes

Signs

- 3 Ring/Target Sign on anterior lens capsule
- Pseudo exfoliation flakes on papillary margin
- Moth Eaten Transillumination Defects, close to papillary margin
- Sampadlesi's Line (line of pigment anterior to schwalbe's line)

Unique points

- PXF glaucoma much more aggressive than OAG
- IOP varies widely
- More asymmetric
- Less responses to topical anti glaucoma drugs, more surgical intervention
- Topical βblockers, PGA, pilocarpine at night ↓lens iris interaction
- ALT/SLT
- Cataract surgery with trabeculectomy



3 Ring / Target Sign

3. MALIGNANT GLAUCOMA/POSTERIOR AQUEOUS MISDIRECTION SYNDROME/ CILIARY BLOCK GLAUCOMA © 00:32:00

- 2° glaucoma occurring post intraocular surgery characterised by
- 1. Shallow AC
- 2. Elevated IOP
- 3. Absence of papillary block



Ciliary Block Glaucoma

- Expansion of vitreous volume d/t sequestration of aqueous posteriorly with resulting anterior shift in Lens Iris diaphragm
- M/c: H/o ACG undergoing glaucoma filtering surgery

Symptoms

- Pain
- Blurred vision
- Colored halos

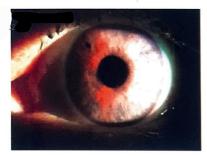
Pathology



- For certain reasons the Aqueous humor goes posteriorly to vitreous humor, instead of going anteriorly → Posterior Aqueous Misdirection Syndrome
- Creates Pockets of Aqueous Humor inside vitreous
- Expanded vitreous volume → Iris & Lens pushed upwards
- AC becomes completely flat or shallow & the angle closes

Diagnosis

 Flat AC, ↑IOP, absence papillary block (Patent PI), absence of suprachoroidal effusion



Posterior Aqueous Misdirection Syndrome

MC in eyes with H/O angle closure undergoing glaucoma Filtering Surgery

Treatment

- Medical management
 - o Effective in 50%
 - o Triad
- 1. Cycloplegics (Atropine)

Paralyse The ciliary muscles

Zonular Contraction

Pulls the lens backwards

Deepening the AC

Aqueous moves forward

TIOP

- 2. Aqueous suppression with βblockers, α2 agonist, CAI inhibitors
- 3. Hyperosmotic drugs: Mannitol
- Surgical management

 Vitrectomy (Definitive treatment) with anterior hyaloid disruption



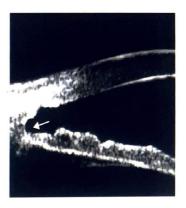
Important Information

10P decreases on Pl in pupillary block glaucoma but remains high in malignant glavcoma

1. TRAUMATIC GLAUCOMA



- Angle recession glaucoma
 - M/c glaucoma associated with trauma
 - Separation of longitudinal and circular fibres of ciliary body



Angle recession glaucoma

Pathology

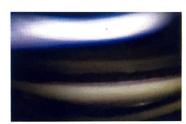
- Blunt trauma → creates shock waves → causes TM scarring \rightarrow leads to obstruction of aqueous \rightarrow OAG
- Angle recession > 180 degree leads to glaucoma
- · Onset of glaucoma is from 6 months onwards may take years, life long, screening
- IOP levels can be dramatic > 70 mmHg possible
- Asymptomatic, history of blunt trauma

Hyphema

o Deep AC, iris sphincter tears, iridoschisis (splitting of iris), phacodonesis, subluxation

Diagnosis

- Gonioscopy
 - Widening of ciliary body band
 - Pigmentation of TM
 - Whitening of scleral spur



Gonioscopy findings: Angle recession glaucoma

Treatment

- β-blocker (timolol)
- α2 agonist
- CAl inhibitors
- Prostaglandin analogues effective

5. HYPERTENSIVE UVEITIS



- Uveitis leading secondary glaucoma (not d/t HTN)
- M/c cause chronic, anterior uveitis
- TM mechanical obstruction by cells, debris, fibrin
- 360-degree posterior synechiae blocks pupil

↓ leads to iris bombe

AČG

- Peripheral anterior synechiae
- Steroid induced glaucoma
- In Secondary Glaucoma, We Treat the Primary Cause
- DOC: Atropine > steroid
- Rx: First with cycloplegics then topical steroids, aqueous suppressants
- Pilocarpine/PGA avoided.



Hypertensive Uveitis



Important Information

Drug of choice of Hypertensive Uveitis

- A. Pilocarpine
- B. Atropine
- C. Latanoprost
- D. Timolol

Answer: Atropine

Pilocarpine and latanoprost both cause uveitis so not given to this patient. DOC is cycloplegics and timolol is an adjuvant in treatment.

6. LENS INDUCED GLAUCOMA



Types

- 1. Phacomoprhic glaucoma
- 2. Phacolytic glaucoma
- 3. Lens particle glaucoma
- 4. Phacoantigenic

1. Phacomorphic glaucoma

 Angle closure glaucoma caused by mature cataract which becomes intumescent



Leading to pupillary block

- †IOP because angle is close
- Symptoms
 - o Pain, blurred vision, coloured halos
- On examination
 - Congestion, corneal edema, shallow AC, mid-dilated pupil, intumescent cataract



Phacomorphic glaucoma

- Treatment
 - Aqueous suppressants, topical steroids, cataract extraction is definitive treatment

2. Phacolytic glaucoma



- Open angle glaucoma caused by hypermature cataract / morgagnian cataract
- Lens matter leaks out (high molecular lens proteins) → which blocks TM → leading to ↑IOP
- Symptoms
 - o Pain, redness, blurred vision
- On examination
 - o †IOP, corneal edema, deep AC, cells in AC



Phacolytic glaucoma

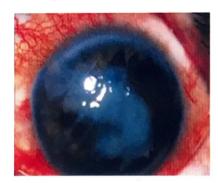
- Treatment
 - Topical steroids, cycloplegics, cataract extraction definitive

Phacomorphic	Phacolytic
ACGMature cataractShallow AC	Open angle glaucomaHypermature cataractDeep AC

3. Lens particle glaucoma

- ↑IOP due to blockage of aqueous by lens particle → leading to OAG
- Disrupted lens capsule releases lens matter in AC
- H/O of cataract surgery, lens trauma, capsulotomy
- Congestion. †IOP with corneal edema, lens fragments in AC
- Similar to phacolytic, but

 inflammation, pupillary membranes, synechiae



Lens particle glaucoma

- Treatment: Antiglaucoma drugs, steroids, removal of lens
- 4. Phacoantigenic/Phacoanaphylactic glaucoma
- **Ö** 01:08:52
- Rarest type of lens induced glaucoma
- Granulomatous inflammatory reaction against own lens protein, normally immune privileged with lens capsule → leading to TM block

↓ ↑IOP

Due to complicated cataract surgery

Mixture of lens matter and vitreous

Release of lens proteins

- Usually occurs within 2 weeks
- Congestion, corneal edema, intense AC reaction, mutton fat KP's
- Treatment: Removal of lens matter

7. NEOVASCULAR GLAUCOMA



- Severe secondary ACG with poor prognosis
- Highest O2 consumption in eye
 Retina

↓ Hypoxia

1

VEGF, insulin, GF-1, IL6 [Vascular Endothelial Growth Factor]

Neovascularization

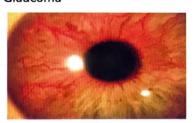
Neovascularization

↓ Retina ↓ Vitreous

Iris (Rubeosis iridis) [Neovascular iris (NVI)]

 \downarrow

Neovascular Glaucoma



Neovascular Glaucoma

Causes

- Proliferative diabetic retinopathy
 - Most common cause
 - Responsible for 1/3rd of cases of neovascular glaucoma
- Ischemic CRVO: "90-day glaucoma"
- Ocular Ischemic Disease
- CRAO: "30-day glaucoma"
- Sickle cell anaemia
- Retinoblastoma
 - Present with chronic red painful eye, ↑↑ IOP, corneal edema, VA <6/60 anterior segment inflammation, visible NVIand NVA, ectropion uveae
 - On gonioscopy we find near total angle closure with microhyphema
 - Secondary angle closure glaucoma: ↑IOP
 - o Zipper-like closure

Management

- Early NVG
 - o Stop neovascularization
 - a. Anti-VEGF Drugs
 - → Bevacizumab (Avastin)
 - → Ranibizumab (Lucentis)
 - → Afibercept
 - b. Pan Retinal Photocoagulation (except macula) for hypoxia



- o JIOP
 - → Latanoprost & pilocarpine are C/I
 - → Trabeculectomy/Aqueous drainage devices
- Late Neovascular Glaucoma (Absolute Glaucoma)
 - Patient start losing vision
 - o IOP: 70-80 mm Hg
 - Can lead to glaucomatous optic atrophy
 - o Pain+nt
 - Cyclodestruction
 - → Diode laser Cyclophotocoagulation (DLCP)
 - → Cyclocryopexy (Temp. of probe → 80°C)





Cyclocryopexy

Absolute Glaucoma

GLAUCOMA DRAINAGE DEVICES / SETONS



- Designed to direct aqueous from AC to an external reservoir
- Done in refractory glaucoma: Uveitis glaucoma, NVG, paediatric, aphakia, glaucoma
- Design: silicone tube, with valve mechanism draining aqueous to end plate on equatorial sclera, m/c in superotemporal quadrant
- Indications
 - o Previous failed Trabeculectomy
 - o Trabeculectomy likely to fail or, hazardous: E.g.
 - → NVG
 - → ICE syndrome
 - → Aphakic glaucoma
 - → Uveitis glaucoma
 - → Severe Conjunctival Scarring
 - o Congenital, traumatic, post keratoplasty glaucoma

2 types

- Valved
 - o pressure sensitive valves
 - o IOP control
 - Predictable
 - Prevents hypotony
 - First valve: Molteno
 - Most popular: Ahmed glaucoma valve (AGV) and Baerveldt
- Non valved
 - Ex-press mini shunt is a stainless steel, MRI compatible stent

Complications

- Hypotony
- Flat AC
- Strabismus
 - Generally, Trabeculectomy

 IOP to greater degree, but-greater risks







Glaucoma Drainage devices

?

Previous Year's Questions

- Q. which one of the procedures involves using glaucoma drainage device? (NEETJAN 2019)
- A. Seton operation
- B. Deep sclerectomy
- C. Viscocanalostomy
- D. Trabeculectomy



Previous Year's Questions

- Q Mast common eye manifestation in Sturge weber syndrome? (AIIMS NOV 2018)
- A. uveitis
- **B.** Keratitis
- C. Glaucoma
- D. Retinitis pigmentosa



Previous Year's Questions

- Q. Which of the following drugs are used to differentiate fixed dilated pupil? (INICETNOV 2020)
- A.4% cocaine
- B. Pilocarpine 1/
- C. Phenylephrine
- D. Adrenaline





Q. A 43-year-old patient who was on topical antiglaucoma drug therapy for several months presented with the features shown in the given image. The likely antiglaucoma drug responsible for this adverse effect is:



A. Timolol

B. Olopatadine

C. Latanoprost

D. Brimonidine

Answer: C

Solution

Side effects of Prostaglandin Analogues (Latanoprost, Travoprost, Bimatoprost, Tafluprost)

- Ocular
 - Conjunctival hyperemia
 - Eyelash lengthening
 - Peri-ocular skin hyperpigmentation may be reversible
 - Irreversible iris hyperpigmentation due to increase in number of pigment granules in the superficial stroma of the iris
 (not the pigment cells) more common in patients with light coloured iris, hence can produce heterochromia iridis
 - Peri-ocular atrophy
 - Cystoid macular edema especially when used in post-operative patients hence avoided in patients with postoperative glaucoma/IOP spikes
 - Anterior uveitis hence avoided in patients with inflammatory glaucoma
 - Promotion of herpetic keratitis
- Systemic
 - o Headache, precipitation of migraine
 - Malaise, Myalgia, Skin Rash, mild upper respiratory tract symptoms

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, 2020, Chapter 11 - Glaucoma, Page 404

- Q. A patient has primary open-angle glaucoma in his right eye. His visual acuity was 10/90 and despite being on timolol, pilocarpine, and acetazolamide, the IOP remains 20 to 24mmhg. A subsequent visual field test showed evidence of further progression of field loss in this eye. Which one of the following is the best treatment option for this patient?
- A. Seton Placement
- B. Trabeculectomy
- C. Argon laser trabeculoplasty
- D. Addition of apraclonidine

Answer: C

Solution

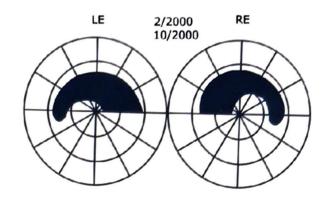
This is a case of medically refractive open angle glaucoma. In this case Surgery is commonly undertaken to arrest visual field loss.

Reference: Ophthalmology, Yanoff & Duker, Saunders Elsevier, 4th Edition, 2014, Part 10 - Glaucoma, Chapter 10.5 - Visual Field Testing in Glaucoma, page 1029-1035.

Q. A 50 year old women presented with below visual field defect. It has been diagnosed as a case of primary open angle glaucoma. Which part of visual field will be the most resistant to glaucomatous visual field loss?

- A. Central vision
- B. Nasal peripheral vision
- C. Superior vision
- D. Temporal peripheral vision

Answer: D



Solution

Visual field abnormalities in glaucoma:

- Paracentral scotoma Earliest reliable field defect in glaucoma
- Siedel glaucoma
- Arcuate or Bjerrum scotoma
- Ring scotoma
- End stage or near total field defect- Only residual temporal island of vision, occurs at the last stage

Reference: Parson's Diseases of the Eye, 22nd Edition, 2015, Section I - Anatomy and Physiology, Chapter 2 - Physiology of the Eye, Intraocular pressure, page 17.

- Q. A 70 year old patient presents with progressive deterioration of vision. On examination, the pupillary reaction is sluggish and IOP is normal. Fundoscopy shows a large and deep cup. Visual field reveals paracentral scotoma, What is the probable diagnosis?
- A. Neovascular glaucoma
- B. Primary Open angle glaucoma
- C. Normal tension glaucoma
- D. Absolute glaucoma

Answer: C

Solution

The question describes a patient with normal IOP but optic disc changes and visual field changes suggestive of glaucoma. Hence the answer is normal tension glaucoma

Reference: Parson's Diseases of the Eye, 22nd Edition, 2015, Section IV - Diseases of the Eye, Chapter 19 - The Glaucomas, Congenital Glaucoma, page 304-305.



LEARNING OBJECTIVES

REFRACTION

- MYOPIA/ SHORT SIGHTEDNESS
 - Axial, Curvature and Index Myopia
- HYPERMETROPIA/ HYPEROPIA/ LONG SIGHTEDNESS
 - Axial, Curvature and Index Hypermetropia
- ASTIGMATISM
 - Types of Astigmatism
 - With the rule and against the rule astigmatism
- STURM'S CONOID
- PRESBYOPIA
 - AGE EXPECTED PRESBYOPIA CORRECTION
- DARK ROOM PROCEDURES
 - Retinoscopy
 - Distant Direct Ophthalmoscopy
 - o Direct Ophthalmoscopy
 - Indirect Ophthalmoscopy



8

REFRACTION

DEFINATIONS

Emmetropia

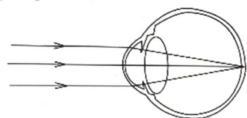
尚 00:01:09

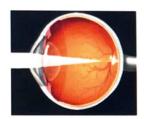
Condition where the rays of light focus on the retina

Ametropia

O 00:01:34

- Condition where the rays of light do not focus on the retina
- Myopia
 - Rays of light focus in front of retina
- Hypermetropia/Hyperopia
 - Ravs of light focus behind the retina
- Astigmatism
 - o Rays of light have 2 different foci





Ametropia



Previous Year's Questions

Q. In myopia image is formed in?

(FMGE Dec 2019)

- A. Anterior to retina
- B. Posterior to retina
- C. On retina
- D. None of the above

MYOPIA/SHORT SIGHTEDNESS



- Condition where the rays of light focus in front of Retina
- Eyes half close → Myopic look
- Causes
- 1. Axial Myopia
- 2. Curvature myopia
- 3. Index myopia



Myopia

Axial Myopia



- MC cause of myopia
- · Axial length is longer than normal
 - o Normal: 24 mm
 - o Eye ball is longer than normal
- For each 1 mm of extra length of eye ball, 3 D myopia occurs.

Myopic look



Axial myopia

\Diamond

Important Information

- If Axial Length of a patient is 31 mm. The correction needed in this patient will be: 31 mm - 24 mm = 7mm. (7 x3 = 21D)
- · Thus 21D concave lens is needed.
- Myopics wear the glasses close to the eyes



 Hypermetropes wear convex lenses lower down on the nose





Important Information

Q. Hypermetropes wearing glasses & reading newspaper. The glasses pushed down the nose will the vision improve or worsen

Ans. Vision Improves

Curvature Myopia



- Curvature of cornea is more
- Example: Keratoconus
- Each 1 mm for extra curvature of eyeball, 6 D myopia in occurs



Curvature myopia

Index Myopia



- Refractive index increases
- Example: Nuclear cataract



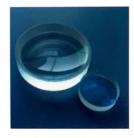
Important Information

Myopes are short sigted people (can't see long distance objects)

- Long distance rays are parallel & Focus Infront of retina
- Short distance rays are divergent rays, will focus on retina

Treatment

- By Concave/ Divergent/ Minus Lens
 - Limitation: Minification of Images
 - → Each 1D, minifies image by 2%
 - o Under correction is done for myopia
 - o Uncorrected myopes develop Divergent Squint



Concave lens





Myopia

Corrected myopia



Previous Year's Questions

Q. Tigroid pattern sign in eye is seen in?

(JIPMER May 2019)

- A. Shagreen degeneration
- B. Degenerative myopia
- C. Pantothenate kinase deficiency
- D. Retinitis pigmentosa



Previous Year's Questions

Q. The following features are seen in high myopia except (JIPMER Nov 2017)

- A. Tilted optic disc
- B. Anterior staphyloma
- C. Vitreous syneresis
- D. Macular chorioretinal atrophy

REDUCED SCHEMATIC EYE

22.6 mm
60
17 m
5.6 mm

HYPERMETROPIA/HYPEROPIA/LONG SIGHTEDNESS



1.0

1.33

O 00:31:57

Rays of light focus behind the retina

Causes

1 Axial hypermetropia (m/c)

Refractive index of air

Refractive index of eye

- 2. Curvature hypermetropia
- 3. Index hypermetropia

Axial Hypermetropia

- **Ö** 00:36:26
- M/C cause of hypermetropia
- Axial length is smaller than the normal
- For each 1 mm of shortening, 3D of hypermetropia occurs



Important Information

 All newborn are hypermetropic (2.5-3.0 D) as axial length of eye is less than normal at birth

Curvature Hypermetropia

- 00:39:10
- Cornea is comparatively flat
- Example: Congenital causes (rare)
 - Cornea Plana
 - ScleroCornea
- For each 1 mm of flattening, cause 6 D of hypermetropia

Index Hypermetropia

- **Ö** 00:41:10
- Refractive index decreases
- Example: cortical cataracts



Important Information

- Cortical cataracts cause index hypermetropia
- Nuclear cataracts cause index myopia

Treatment

- By convex/ Convergent/ Plus lens
 - o Limitation: Magnification of image
 - o Every 1D, magnifies image by 2%



Convex lens

Symptoms of Hypermetropia



- Long sightedness
 - Neither rays from long distance nor the rays from short distance will focus on retina
- But one can accommodates upto 16 D
 - By this mechanism, rays from long distance will focus on retina
 - o So, hypermetropes do not require convex lenses upto

- certain limit without any visual disturbances
- But d/t excessive Accommodation it leads to asthenopia (eve strain)
- Uncorrected hypermetropes develop convergent squint



Important Information

- · Uncorrected myopes develop Divergent Squint
- Uncorrected hypermetropes develop Convergent Squint
- Blurred vision for both, more for near
- Pseudo myopia: Prolonged accommodation → accommodation spasm → sudden blurring of vision, often seen in hyperopic teenagers
- Asthenopia: tiredness of eyes, headache
- Early onset presbyopia
- Amblyopia
- Frequent styes: rubbing eyes to clear vision
- Convergent squint





Asthenopia

Convergent squint



Previous Year's Questions

Q. Esotropia is commonly seen in which type of refractive error?

(NEET Jan 2020)

- A. Myopia
- B. Hypermetropia
- C. Astigmatism
- D. Presbyopia

Types of Hypermetropia



- Total = Manifest + latent
- Latent: hyperopia overcome by ciliary muscle, Uncovered by cycloplegia
- Manifest: remaining part, maximum power of convex lens that still gives clear vision
- Manifest = Absolute + Facultative
- Facultative: that can be overcome by accommodation
- Absolute: cannot be corrected by accommodation

Prescribing glasses in hyperopia



- Total hyperopia revealed by cycloplegic
- Symptomatic patients and children to be treated
- Maximum accepted plus power with clear vision (6/6) should be prescribed
- Children prescribed full hyperopic correction
- If associated with convergent squint full correction to be given

EMMETROPIZATION



- The development of an eye from ametropia towards emmetropia
- During infancy there is 1 in corneal and lens power
- Refractive errors minimized in children by age 6, average +0.75D.

ASTIGMATISM



01:07:52

- Condition where there are 2 different foci are present
- M/ C refractive error: 40%
- Image formed by two different foci
- Principal reason: corneal curvature
- · Periphery of cornea is steeper than centre
 - o Rays from periphery focus inform of the retina
 - o Rays from centre focus behind the retina



Astigmatism

Original Compromise

Clr Clr

Horizontal Focus

Clr Clr

Problem with Astigmates

Classification of Astigmatism



 Total astigmatism = Corneal + lenticular (- 0.5D X 90°) + Retinal



Important Information

Javal 's rule: predicts total astigmatism → corneal astigmatism

- Regular: two principal meridians separated by 90°, correctible by sphero-cylindrical lens
- Irregular principal meridians not symmetric, do not lie 90° apart, not correctible by Sphero – cylindrical lens

Signs & Symptoms of Astigmatism

Distortion and blurring of images



- Asthenopia, headaches, squinting, diplopia
- Reading small print is difficult

VERTICAL

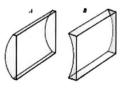
HORIZONTAL

Treatment of Astigmatism

- Cylindrical lenses / Toric lenses
- · Prescription should be
 - ±2DCX90 degree

C = Cylindrical

90 degree = Indicates axis (1-180 degree)



Cylindrical lenses

Types of Astigmatism

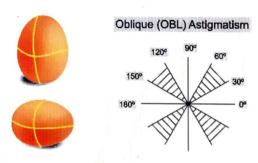
- Simple myopic astigmatism
 - 2 focal points, one on retina, other in front of retina
 - o Corrected with concave cylindrical lens
 - E.g. –2DC X 10°
- Simple hyperopic astigmatism
 - o One focal point on retina, one behind retina
 - Corrected with concave cylindrical lens
 - E.g. +10 DC X 180°
- Compound myopic astigmatism
 - Both focal points in front of retina
 - Corrected with combination of concave spherical and concave cylindrical lens
 - E.g.-3DS/-2DC X 140°
- Compound hypermetropic astigmatism
 - Both focal points behind the retina
 - Corrected with combination of convex spherical and convex cylindrical lens
 - Eg +5DS/+1DCX70°

- Mixed astigmatism
 - One focal point in front of retina, one behind retina
 - Corrected with lens of opposite signs with power of cylindrical lens more than spherical lens
 - E.g. +3DS/-10DC X 180°

Regular astigmatism



- · With the rule astigmatism
 - Common; seen in young < 50 years
 - Steepest meridian is vertical or within 30 °of 90° meridian
- · Against the rule ATR: Steepest
- Meridian horizontal or within 30 of 180 or
- Oblique: Steepest meridian is not within 30° of vertical or horizontal meridians



Axis of Astigmatism: (1-180)

- Oblique Astigmatism: 30-60 or 120-150
- Regular Astigmatism: Outside the limits of oblique astigmatism

With the rule (WTR) / Against the rule (ATR)



WTR + cylindrical lens X 90° (+2.0D C X 90°)
- Cylindrical lens X 180° (-2.0DS / -3.0DC X 180°)

ATR - cylindrical lens X 90° (-3.0D C X 90°)
+ cylindrical lens X 180° (4.0D C X 180°)



Previous Year's Questions

Q. Which one is against the rule astigmatism?

(NEET Jan 2019)

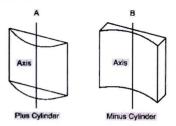
A. +2.2 D C x 90°

B. - 2.0 DC X90°

C. -1.5 DCX 180°

D. None of the above

Types of Toric lenses



 Minus cylindrical lens are more comfortable and preferred than the plus cylinder forms

Transpositions and conversions



- Reverse the sign of the cylinder
- Change the axis of the cylinder by 90
- Algebraic addition of sphere and cylinder
- -3DS/-2DC X 90° can be converted to plus form as -5DS/+2DC X 180°Both are same prescriptions
- Spherical Equivalent: spherical lens which places an astigmatic eye in meridional balance
- Formula: Power of Cylindrical lens/2+power of sphere (algebraic)



Previous Year's Questions

Q. Spherical Equivalent of +10DS/+2DC X 90°is?

Ans: Applying Formula: Power of Cylindrical lens/2+power of sphere to this case, 2/2+10 =+11. So SE is +11DS

Q. Spherical Equivalent of -6DS/-4DC X 180°is?

Ans: Applying Formula: Power of Cylindrical lens/2*power of sphere to this case, -4/2 + (-6) = -8. So SE is -8DS

?

Previous Year's Questions

Q. A 15-year-old female with myopic astigmatism refuses to wear glasses, what would be ideal management?

(NEET 2021)

A. LASIK

B. Femto LASIK

C. ICL

D. Spherical equivalent glasses

STURM'S CONOID



 Configuration of rays formed by an astigmatic optical system consisting of a primary focal line, a circle of least confusion and a secondary focal line

perpendicular to primary

- Circle of Least Confusion
 - Smallest diameter of blur circle between the two focal lines, corresponding to dioptric midpoint of the two lines
 - o Should focus on retina for providing clearest vision

Referimage 8.1

PRESBYOPIA



- Loss of accommodation with age [40-45 years]
- Only ocular condition with prevalence of 100% in patients older than 50



- · Physiological phenomenon
 - Normally, Lens power is 19 D Lens can be able to accommodate additional 16 D of power
 - With age the accommodation power (ability to become spherical) decreases d/t
- 1. Hardening of lens d/t calcification
- 2. Capsule becomes more fibrous
- 3. Ciliary muscle becomes weaker

AMPLITUDE OF ACCOMMODATION © 00:04:54 WITH AGE

Age in years	Amplitude of Accommodation in Dioptres
10	14
25	8.5
35	5.5
45	3.5
60	1.0

Difficulty in seeing near object



Corrected by Convex lens

AGE EXPECTED PRESBYOPIA CORRECTION



Age in years	Addition
40	+ 1.00 D
45	+ 1.50 D
50	+ 2.00 D
55	+ 2.50 D
60	+ 3.00 D

DIFFERENTIATING FEATURES FROM HYPEROPIA

Presbyopia

- 40-45 yrs
- Difficulty for near vision (wears glasses)
- Distant vision is normal (no need of glasses)

Hyperopia

· Wears glasses all the time

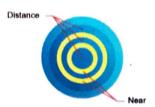


Bifocal vs Progressive lenses

Treatment of Presbyopia



- 1. Glasses
- Bifocals/trifocals
- Progressives (lens blend into each other without dividing line, technically more advanced)
- Not ideal for high prescriptions



- 2. Contact lenses
 - Monovision
 - Multifocal contact lenses
- 3. Surgery
 - Refractive surgery
 - Corneal inlays

DARK ROOM PROCEDURES



- 1. Retinoscopy
- 2. Distant Direct Ophthalmoscopy
- 3. Direct Ophthalmoscopy
- 4. Indirect Ophthalmoscopy



Important Information

In Dark rooms, there is

- 1. Less reflection from cornea
- 2. Pupil dilates more

RETINOSCOPY

- Patient with 6/60 vision
- Check for
 - 1. Optical Error
 - a. Myopia
 - b. Hypermetropia
 - c. Astigmatism
 - 2. Organic Error
 - a. Glaucoma
 - b. Cataract
 - c. Retinal detachment etc.

PIN HOLE TEST



- A completely opaque plastic disc with a central hole [pin size is 0.5mm to 1.5mm diameter]
- On applying pin hole test
 - o If vision improves: Optical Error
 - o If vison do not improve: Organic Error
- Pin Hole allows only single Ray of Light. Single ray of light passes through nodal point of eye (No distraction)
- Pin hole test differentiates the optical & organic error but it doesn't differentiate the type of optical Error



RETINOSCOPY/SKIASCOPY



00:32:05

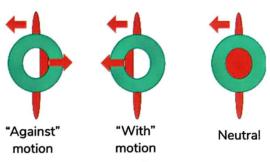


Trial box

Retinoscopy

Retinoscopy streak

- Technique to objectively measure the refraction
- Done with Retinoscope
- Dilate / Fixate on distant target
- Distance 1 metre away from patient
- Retinoscopy streak moved from side to side; check the movement of light reflex in patient's pupil
- Differentiate b/w the types of refractive Errors



Retinoscopy streak movement



How to remember

SPAM: Same Plus Against Minus

CALCULATION OF POWER OF LENS

Example

- On Retinoscopy if Retinal reflex has opposite movement with vertical movement of retinoscope
 - It indicates power > 1
 - o Adjustment with trial set done until the light fills the entire pupil without any further movement: Neutral
 - Assume at -5D, neutral movement achieved
- Same procedure done with Retinoscope with horizontal movement
 - Assume at -5D, neutral movement achieved
- Both values are plotted on 'power cross'
- It indicates -5 DS at 1 meter
- To calculate the power at infinity → Subtract the power that have been introduced into the eye by standing at 1 m
 - P = 1/ (Focal length)
 - \circ P=1/1=1,so-5DS-1=-6DS
- So, power of lens used in this case is -6DS
- Astigmatism- on rotation of streak if consistently same width and brightness: no astigmatism

DISTANT DIRECT OPHTHALMOSCOPY (DDO)





Distant direct ophthalmoscopy

- Done with direct ophthalmoscope
- Distance approx. 22 cm
- Healthy red glow

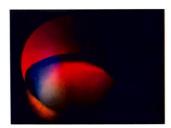


- Real inverted not magnified
- Grey glow in retinal detachment





- No glow in Vitreous haemorrhage
- · Depth of opacity estimated by parallactic displacement
 - No movement in pupillary plane
- Oil droplet reflex seen in Keratoconus
- Subluxation of lens



Subluxation of lens

METHOD OF PARALLAX

- It Differentiates cataract & corneal opacity
- Ask the patient to look up
 - o In case of corneal opacity, blackspot moves up
 - In case of cataract, black spot moves down

- Ask the patient to look down
 - In case of corneal opacity, blackspot moves down
 - In case of cataract, block spot moves down



Important Information

- Same Sided Movement: Corneal Opacity/Scar
- Opposite Sided Movement: Cataract/ Lenticular Opacity

DIRECT OPHTHALMOSCOPY (DO)



- Done with direct ophthalmoscope
- · Close to patient: within anterior principle focus: 15 cm roughly



DIRECT OPHTHALMOSCOPY

- Aim is to see the basic structures of Retina
 - o Disc
 - Fovea
 - o Macula
 - Venous pulsations
- Field of view: 5-10°
- Optical quality
 - Virtual, erect, magnified
- Magnification is 15 times



INDIRECT OPHTHALMOSCOPY (IO) 6 00:58:50



Done with indirect ophthalmoscope

INDIRECT OPHTHALMOSCOPY

- Lying down position
- Approximately arm length distance
- Optical quality
 - Real, inverted, magnified image
- Magnification is 3-5 times
- Structures visualized
 - Wide field of view: 30° 45°
 - Central retina

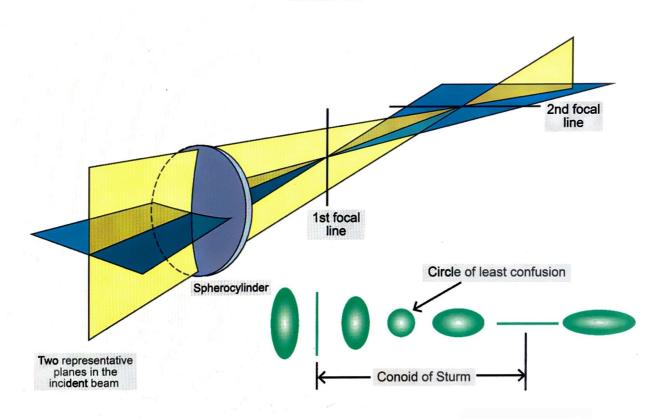
- o Peripheral retina
- Vitreous
- o Ora serrata upon indentation
- Stereopsis: 3 D view possible



Structures visualized in IO

	DDO	DO	10
Distance from patient	22-25 cm	15 cm	Arm length of doctor
Findings	Red glow	Central retina	Entire retina
Image	Real and inverted	Virtual, erect and magnifie d	Real, inverted and magnified
Magnification	No magnification	15X	3-5X
Field of view	Na	5-10°	30-45°

Image 8.1



STURM'S CONOID





- Q. 42 year-old male presents with dimness of near vision and can not read the newspaper print clearly. On examination, the media was clear in both the eyes and no fundus abnormality was seen. What would be the next step?
- A. Refraction with near addition
- B. Refraction under atropine
- C. Radial keratotomy
- D. Cataract surgery

Answer: A

Solution

PRESBYOPIA

There is a decline in the accommodative power of the crystalline lens with age. Infants have a 14D accommodation and falls to 4D at 45 years of age and 1D at 60 years of age. This decline in the accommodative power is called **PRESBYOPIA** for which **near add (convex glasses) is prescribed to individual.**

Surgical Modalities to correct Presbyopia

- 1. Multifocal IOL
- 2. Crystalens
- 3. Presbyopic LASIK

Reference: Clinical Optics, Elkington, 3rd Edition, 1999, Chapter 11 - Presbyopia, page 141.

- Q. On performing refraction using plane mirror on a patient who's has a refractive error of -3 D sphere with -2 D cylinder at 90 degree from a distance 1 metre under no cyclopgia, the reflex would be seen to move
- A. With the movement in the horizontal axis and against the movement in the vertical axis
- B. With the movement in both the axis
- C. Against the movement in both the axis
- D. With the movement in the vertical axis and against the movement in horizontal axis

Answer: C

Solution

Error of -3 D sphere that is person using concave sphere(diverging) advised in cases of myopia.

Reflection of ligth with Retinoscope

1. Against / opposite movement :

Myopia > -1

2. With / same movement

Myopia < -1 or

Emmetropia = 1 or

Hypermetropia = + value

3. No movement

Myopia = -1

This patient is having a -3DS with -2DC x 90 - compound myopic astigmatism, so the reflex would move in opposite Join the Telegram Channel - https://t.me/prepladderlatestnotes

direction to the beam irrespective of the axis

Reference: Comprehensive Ophthalmology, 6th edition, A K Khurana, Pg 570,576

Q. A person came to eye OPD for routine eye check. On snellen's chart he was found to read 6/6 in both eyes. What is the largest approximate distance at which he would be able to read the first topmost letter

A. 36 m

B. 24 m

C. 60 m

D. 1 m

Answer: C

Solution

What does 6/6 mean in Snellen's chart

 It means that the patient with or without glasses can see the letter at a distance of 6 m which a person normally should have been seeing at 6 m

Now you would understand it by next example

- 6/12 means a letter which a normal patient should be seeing at 12 metre, this patient can see only when he is nearer, that means 6 metre from Target
- 6/60 means that a normal patient should be seeing at 60 metre, but this patient has to be much closer that means 6
 metre
- This also means that the letter written at 6/60 is big enough that 6/6 vision person can read it at even 60 metre

Reference: Optics and refraction A K Khurana 2nd edition

- Q. A 7 year old boy came to ophthalmologist for correction of his eyesight. After evaluation of his eyesight the doctor found that one of his eyes was myopic and other was hypermetropic. The doctor advised immediate correction of the defects either surgically or non surgically. Which of the following is the most Preferred Non-surgical Management of Anisometropia?
- A. Spectacle Correction
- B. Contact lenses
- C. Prisms
- D. No managment

Answer: B

Solution

The optical state with equal refraction in the two eyes is termed isometropia.

When the total refraction of the two eyes is unequal the condition is called anisometropia.

Anisometropia can lead to amblyopia hence management is very important

Treatment

- 1. Spectacles correction can take care of anisometropia up to 4D (some patients may not tolerate as high)
- 2. Contact lenses for higher degrees of anisometropia.
- 3. Intraocular lens-implantation for uniocular aphakia.
- 4. Refractive corneal Surgery
- 5. Phakic Refractive Lenses (PRL) and Refractive Lens Exchange (RLE)

Reference: Comprehensive ophthalmology A K Khurana 6th edition Pg 44



LEARNING OBJECTIVES

RETINA & VITREOUS

- Layers of Retina
- RETINAL DETACHMENT
 - RHEGMATOGENOUS DETACHMENT
 - EXUDATIVE RETINAL DETACHMENT
 - VOGT KOYANAGI HARADA SYNDROME
 - TRACTIONAL RETINAL DETACHMENT
- DIABETIC RETINOPATHY (DR)
- VITREOUS HEMORRHAGE
- CENTRAL RETINAL VEIN OCCLUSION (CRVO)
 - Ischemic vs Non-Ischemic
- CENTRAL RETINAL ARTERY OCCLUSION (CRAO)
- CYSTOID MACULAR EDEMA
- CENTRAL SEROUS RETINOPATHY (CSR)
- AGE RELATED MACULAR DEGENERATION ARMD/AMD
- MACULAR HOLE
- RETINOPATHY OF PREMATURITY (ROP)
- RETINITIS PIGMENTOSA
- Best's disease and Stargardt's disease
- RETINOBLASTOMA



RETINA & VITREOUS





ANATOMY

- Thin transparent membrane
- Choroidal glow (Reddish-pink) is seen through the retina
- Painless structure

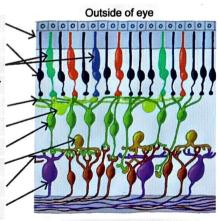
Blood supply of retina

- 00:01:25

- Dual Blood Supply
 - Inner 2/3rd: CRA [central Retinal Artery]
 - o Outer 1/3rd: PCA [Posterior ciliary artery] (also supplies choroid)
- Whatershed Layer (Junction of 2 blood supplies): OPL [outer plexiform layer)

Ten layers of Retina

- 1st 9 layers Neuro sensory layers (NSL)
- 10th layer Retinal pigment epithelium (RPE)
- Exception: FOVEA, MACULA do not have 10 layers
- · Pigment epithelium
- · Rods
- Cones
- Outer plexiform layer
- Horizontal cells
- Bipolar cells
- Amecrine cells
- Inner plexiform layer
- Ganglion cells
- Nerve fiber layer



Vitreous(intside of eye)

layers of Retina



Previous Year's Questions

Q. Most radioresistant layer of retina?

(JIPMER Nov 2019)

- A. Layer of rods and cones
- B. Outer plexiform layer
- C. Retinal pigment epithelium
- D. Ganglion cell layer



Previous Year's Questions

Q. Blood retinal barrier is formed by?

(JIPMER Nov 2018)

- A. Muller cells
- B. Amacrine cell
- C. Bipolar cell
- D. Horizontal cell layer

VITREOUS

- Occupies 70-80% by volume
- Single largest structure of the eye
- Composition
 - 98% H₂O
 - Hyaluronic acid
 - Type II collagen
- Clear gel like consistency
- Vitreous substitutes
 - o SF
 - o C₃F₈
 - o Silicone oil

RETINAL DETACHMENT



Separation of Neurosensory layer (all 9 layer) from Retinal pigment epithelium



Retinal Detachment

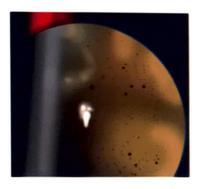
Retinoschisis: Separation of Neurosensory layers from each other

Symptoms and signs

- Floaters (Muscae Volitantes)
 - Sign of vitro-retinal pathologies
- Flashes of lightening (Photosiae)
- Curtain like sensation falling in front of eye

On examination

- Schaffer's sign (Tobacco dusting of vitreous)
 - Seen on Slit lamp examination



Schaffer's sign

Grey glow of retina



Greyish yellow glow of retina

Classification of retinal detachment

- Rhegmatogenous (m/c)
- Exudative
- Tractional



Important Information

 Majority of Retinal detachments are Rhegmatogenous so word retinal detachment means Rhegmatogenous unless specified otherwise.

RHEGMATOGENOUS DETACHMENT © 00:14:55 (BREAK IN THE RETINA)

Risk factors

Myopia (M/C cause)



High Myopia

- Post cataract surgery
- Trauma

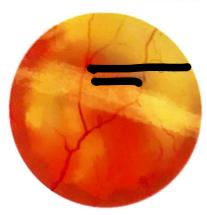
Myopic lesions predisposing to retinal detachment

- Holes
 - o Liquified vitreous flows through the holes
 - Gets accumulated between neurosensory and RPE layers leading to retinal detachment



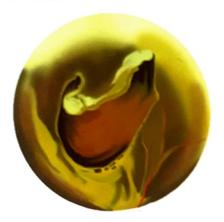
Holes

- Lattices
 - Higher chance of detachment than holes



Lattices

- Tears
 - Due to VR traction
 - Horseshoe tears



Tears

- o Giant retinal tears
 - → Spread in > 3 clock hours
 - → Run parallel to limbus)

Post cataract surgery

Aphakia: Tamponade effect of lens is lost



Aphakia

Trauma

- Leads to dialysis
 - separation from root
 - o Retinal dialysis: Retina separates from ora serrate



Important Information

- Most common dialysis is in inferotemporal quadrant of the eye
- Maximum number of retinal detachments occurs in superotemporal quadrant of the eye



Retinal dialysis

Pathology of Rhegmatogenous detachment

Break in the retina

†

Liquefies vitreous goes through the break (rhegma)

1

Separates the two layers of retina (i.e. retinal detachment occurs)

Symptoms of Rhegmatogenous detachments

Sudden painless loss of vision

Watermarks in chronic Retinal detachment



Watermarks (chronic Retinal detachment)

- Hyperpigmented lines in retina caused by deposition of pigmented epithelium in chronic retinal detachment.
- It is a sign of long-standing retinal detachment.



Previous Year's Questions

- Q. Rhegmatogenous retinal detachment is seen in which of the following conditions?
 - (JIPMER DEC 2019)

- A. Diabetes
- B. Myopia
- C. Accelerated hypertension
- D. Complicated cataract

EXUDATIVE RETINAL DETACHMENT © 00:27:37



- No hole or break, or lattice in retina
- Separation occurs due to exudation of fluid
- Characterized by shifting fluid
- Sudden painless loss of vision



Exudative Retinal Detachment

Causes

- Malignant melanoma of choroid
 - o M/C cause of exudative retinal detachment



Malignant melanoma of choroid



Important Information

- Commonest tumor of eye in children: Retinoblastoma
- · Commonest tumor of eye in adults: Malignant melanoma of choroid

- Pregnancy induced HTN/Gestational HTN
 - o BP > 140/90 after 20 weeks of POG



Exudative retinal detachment



Important Information

Q. A lady in a third trimester complains of a sudden painless loss of vision. Most probable cause of detachment is?

Ans. Exudative retinal detachment

- Choroiditis
- Malignant HTN (HTN + Papilledema)
- VKH syndrome (Vogt Koyanagi harada Syndrome)
 - Seen commonly in Japan
 - Bilateral granulomatous Pan uveitis



Previous Year's Questions

Q. Shifting fluid sign is seen in?

(NEET Jan 2020)

- A. Exudative Retinal detachment
- B. Tractional retinal detachment
- C. Rhegmatogenous retinal detachment
- D. Retinal hole



Previous Year's Questions

Q. Enlargement of the blind spot occurs in which of the following?

(AIIMS Nov 2019)

- A. Primary open angle glaucoma
- B. Diabetic macular edema
- C. Optic nerve hypoplasia
- D. Papilledema

VOGT KOYANAGI HARADA SYNDROME

Seen commonly in Japan

Bilateral granulomatous Pan uveitis

Symptoms

- Young lady with viral Fever without ocular trauma or surgery
- Neurological features
 - Headache
 - Neck Stiffness
 - Auditory symptoms [mc]
 - Tinnitus
 - Vertigo
 - Deafness
- Ophthalmic Syndrome
 - o B/L Granulomatous panuveitis
 - o B/L Exudative Retinal detachment
 - o "Orange sunset glow fundus"
- **D**ermatological Symptoms
 - Poliosis (Whitening of eye lashes)
 - Vitiligo
 - Alopecia



How to remember

NOD



Important Information

 A young lady presents with B/L exudative retinal detachment. Most probable diagnosis is VKH syndrome

Signs

- Sugiura's sign
 - o Perilimbal vitiligo
 - o Earliest depigmentation



Sugiura's sign

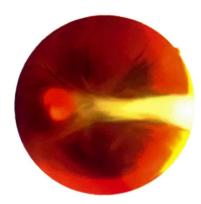
- Sunset glow fundus
 - o Also, k/a orange sunset fundus because of depigmentation of choroid



Sunset glow fundus

TRACTIONAL RETINAL DETACHMENT





Tractional Retinal Detachment

Slow painless loss of vision

Causes

- Diabetes Mellitus (m/c)
- Proliferative vitreoretinopathy (PVR)
- Sickle cell anemia
- Retinopathy of prematurity
- Retinal venous occlusion



Diabetes (Tractional Retinal Detachment)

Investigations



00:37:39

- Fundus examination: Indirect ophthalmoscopy
- Optical coherence Tomography (OCT)

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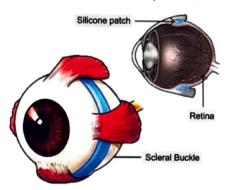
- For examining foveal conditions
- B scan ultrasonography
 - For vitreous haemorrage

TREATMENT OF RETINAL DETACHMENT

- 00:38:43
- · Principle to attach the retina back again
- RPE provides nourishment for NSL
- In 48-72 hours, photoreceptors [rods & cones] starts dying

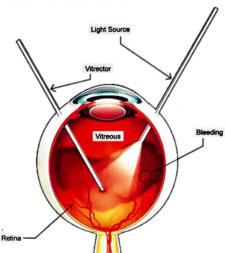
Close the holes, breaks, Lattices, tears

- Cryopexy
 - o Small, peripheral breaks
 - o Freezing causes retinal adhesions after 3 weeks
- Laser photocoagulation
 - Causes chorioretinal adhesion
 - o Counters VR (vitreoretinal) traction & does not allow the passage of liquefied vitreous
- Buckling
 - o Particularly used in phakic eyes
 - o Silicone buckle on the sclera
 - o Reduces VR traction and displaces subretinal fluid



Buckling

- Pars plana vitrectomy
 - o Though the pars plana a needle is introduced
 - Slices of vitreous are removed



- Pneumatic retinopexy
 - o Gas bubble tamponades the break
 - E.g. (SF₆₉ C₃ F₆)



Pneumatic retinopexy

Time frame for ED management

- Depend on status of the macula
 - o "Macula on": Within 24 hours
 - "Macula off": Within 7 to 10 days

DIABETIC RETINOPATHY (DR)



- According to WHO diabetic capital of the world is India
- 40% diabetics develop DR,
- More common in type 1 DM

Risk factors

- Duration
 - Most important risk factor
 - 90% after 30 years develop DR
- Poor control blood glucose
- Hypertension
- Pregnancy
- Nephropathy
- Hyperlipidemia
- Smoking
- Obesity

Clinical symptoms

- **Blurred vision**
- Fluctuating vision
- Impaired colour vision
- **Floaters**
- Redness/ocular pain

Screening guidelines

- In type 1 diabetes: Examine after 5 years
- In type 2 diabetes: Examine at the time of diagnosis
- Subsequently annual exam once every year
- Gestational diabetes screening is not required

 Diabetics who become pregnant to be screened at the end of first trimester

Pathology

1. Occlusion & neovascularization

Hyperglycemia causes

Basement membrane thickening, platelet aggregation, leucocyte adherence

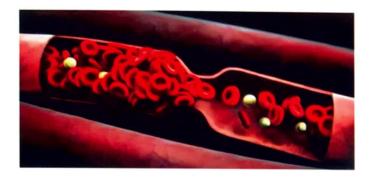
Thrombus formation

Cocclusion of vessels

Retinal hypoxia

VEGF formed

Neovascularization



Occlusion of vessel

- 2. Pericyte necrosis
- Pericytes located in blood vessel wall
- · Functions of pericytes
 - o Regulation of blood flow
 - o Stabilization of structure
- Normal pericytes to endothelial cells ratio is 1:1
 - o In pericyte necrosis P: E ratio is 1:4

Due to pressure walls weaken

↓

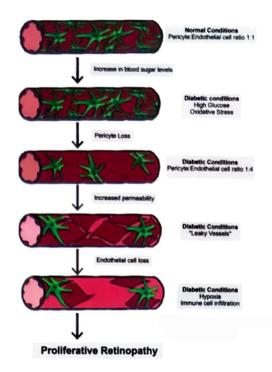
Leads to ballooning out

↓

Microaneurysms

↓

Leakage







Important Information

Earliest ocular manifestation of diabetic retinopathy is microaneurysm

Classification

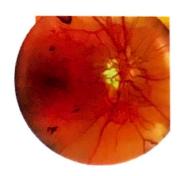


- Non- proliferative diabetic retinopathy (NPDR)
 - o Mild
 - → Only microaneurysm are seen
 - Moderate
 - → microaneurysm + dot / blot hemorrhages / beading blood vessels
 - o Severe (any one)
 - → 4-2-1 rule

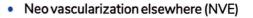


Non- proliferative diabetic retinopathy (NPDR)

- → 4 quadrants of dot & blot retinal hemorrhages or
- → 2 quadrants of dot & blot retinal hemorrhages or
- → 1 quadrant of intra-retinal microvascular abnormalities
- Very severe (any two)
 - → Proliferative diabetic retinopathy (PDR)
- o Neovascularization/pre retinal hemorrhage



Proliferative diabetic retinopathy (PDR)





2

Previous Year's Questions

Q. ETDRS is done for?

(AIIMS Nov 2019)

- A. Endothelial count
- B. Cornealtopography
- C. Primary open angle glaucoma
- D. Classification of diabetic retinopathy

Diagnosis of NPDR

- 1. Microaneurysms
- 2. Dot and blot haemorrhages

Superficial haemorrhages	Deep haemorrhages
Common in hypertensive retinopathy	Common in diabetic retinopathy
Flames shaped haemorrhages	Dot & blot haemorrhages

- 3. Hard exudates
- Deposits of serum cholesterol in retina due to leakage of blood from blood vessel into retina
- 4. Soft exudates / cotton wool spots
- Collection of axoplasmic debris due to occlusion
- 5. Intra retinal microvascular abnormalities
- Shunt vessels in the retina

Diagnosis of PDR

Neo vascularization of disc (NVD)





- NPDR
 - o Macular edema



- PDR
 - o Vitreous haemorrhage
 - Tractiona8fc`wfyi
 IRD
 - o Neovascular glaucoma

Lesions on layers



Finding	Location
Hard exudates	OPL (plexiform layer)
Soft exudates	Nerve fibre layer
Dot / blot hemorrhage	Inner nuclear layer / OPL
Microaneurysms	Inner nuclear layer
Flame shaped hemorrhage	Nerve fibre layer
Macular edema	OPL

Investigations

Ö 01:13:01

- Colour and red-free fundus photography
- Fluorescein angiography

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- OCT: optical coherence tomography
- Ultrasonography

Diabetic macular edema (DME)

Retinal thickening within 2 DD's of fovea



Clinically significant macular edema (CSME)

- Thickening of retina < 500µ from centre of macula
- Hard exudates <500µ from centre of macula
- Thickening > 1 DD in size, any portion of which is <1 DD from centre

Centre involving macular edema

Edema right at the centre

Non centre involving Macular edema

· Edema not exactly in the centre



Previous Year's Questions

- Q. A patient with hypertension and diabetes presents with blurred vision. Fluorescein angiography shows?
 - (AIIMS Nov 2019)

- A. Macular edema
- B. Sub macular edema
- C. Papilledema
- D. Pre macular hemorrhage

MANAGEMENT OF DIABETIC RETINOPATHY



- Healthy lifestyle
- Diet
- Exercise (30 mins of brisk walking for 5 days a week)
- Weight control
- HbA1c < 7% recommended
- Reduce blood pressure < 140/80 mmHg and
- serum lipids <150 mg/dl

NPDR with macular edema

- 1. Focal Laser photocoagulation
- Double frequency ND YAG laser
 - Wavelength: 532 m
 - o Aka green laser

o 60-80 C heat used which absorbs edema





NPDR with macular edema

Retinal Photocoagulation

- 2. Anti VEGF drugs
- Ranibizumab
- Bevacizumab
- Afibercept

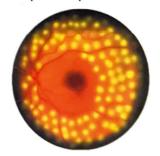


- 3. Intravitreal Steroids
- Triamcinolone
- Dexamethasone (Ozurdex)
- Fluoccinolone



PDR treatment

- Panretinal photocoagulation / Scatter photocoagulation
 - Entire retina except macula
 - Double frequency Nd YAG laser, wavelength -532nm
 - Energy absorbed by RPE/ choroid which denatures proteins and leads to coagulative necrosis
 - o 2000-3000 Spots are placed



Panretinal photocoagulation

- Side Effect
 - → Peripheral vision loss

→ Night vision loss

VITREOUS HEMORRHAGE

- O 00:00:13
- Sudden bleeding inside the vitreous cavity
- Anatomy of vitreous humor
 - o Optically clear jelly
 - Occupies 80% of volume of eye
 - Vitreous is firmly attached to retina at 3 places
 - → Vitreous base
 - → Optic disc margin
 - → Retinal vessels
 - With age, vitreous liquefies and shrinks

Causes of vitreous hemorrhage

- PDR: M/c
- Posterior Vitreous Detachment (PVD)
- Trauma: Commonest in young patients
- Retinal tears
- Retinal vein occlusions (RVO)
- Eales disease (Periphlebitis retinae)

Symptoms of vitreous hemorrhage

- · Sudden painless loss of vision
- Floaters/shadows/cobwebs
- Red tint to vision

Eales disease

- Also known as Periphlebitis retinae
- Common in India
- Occurs in young males
- · Spontaneous vitreous hemorrhage
- Recurrent hemorrhage
- Associated with tuberculosis
- Idiopathic inflammatory venous occlusive disease
 - o Leads to non-perfusion and neovascularization



Important Information

Recurrent spontaneous vitreous hemorrhage is the hallmark of Eales disease

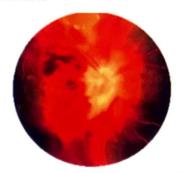




Eales disease

- Stages of Eales disease
 - o Ischemia

- Neovascularization
- Treatment of Eales disease
 - o ATT for 9 months
 - Systemic corticosteroids
 - Retinal photocoagulation to treat retinal hypoxia due to occluded veins



Management of vitreous hemorrhage



VH clears slowly about 1% per day

Conservative management

- Ask the patient to sleep with elevated head position for around 3 months
- · Worst prognosis of VH is seen with PDR and ARMD
- Intravitreal anti VEGF drugs
- PRP in PDR to reduce angiogenesis
- Pars Plana Vitrectomy

Urgent vitrectomy is done in

- Retinal detachment
- NVI/NVA
- Type 1 DM (within One month)





Before vitrectomy

After vitrectomy

RETINAL VEIN OCCLUSION (RVO)



Second most common retinal vascular disorder



Retinal Vein Occlusion (RVO)

3 Major types

- 1. CRVO
 - Occlusion at the level / posterior to lamina cribrosa
- 2. HRVO
 - Occlusion at disc, superior or inferior retina involved
- 3. BRVO
 - Occlusion of a tributary vein, compression by overlying arteriole

CENTRAL RETINAL VEIN OCCLUSION (CRVO)

 Occlusion of central retinal vein posterior to lamina cribrosa of optic nerve

Pathology

- Central retinal artery & vein share common sheath
- Thickening of overlying central artery compresses retinal vein close to lamina cribrosa
- Thickening of artery occurs due to atherosclerosis



Compression of central retinal vein

Two types

- 1. Ischemic
 - If >10 Disc diameters of retinal capillary non perfusion
- 2. Non-ischemic

Risk factors

- Age (>60 years)
- Uncontrolled Diabetes
- Uncontrolled Hypertension
- Hyperlipidemia
- †IOP
- · Oral contraceptives in young women

Ischemic vs Non-Ischemic



Ischemic	Non-ischemic
• 25%	• 75%
• Vision < 6/120	 Vision > 6/18
• RAPD	• None
Dilated veinsDisc swellingflame shaped hemorrhagesCotton wool spots	Much less

- Blood and thunder / splash tomato appearance
- 90-day glaucoma (neovascular glaucoma)



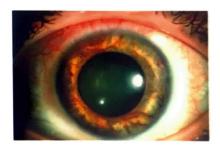


Blood & Thunder

Splash tomato fundus

CRVO ↓ Hypoxia ↓ VEGF ↓ Neovascularization

Neovascular glaucoma (90/100 day glaucoma)



90 day glaucoma

Investigations of CRVO

- OCT
- Macular edema
 - Single m/c cause of loss of vision in CRVO
- Fluorescein Angiography
- Extent of capillary non perfusion

Join the Telegram Channel - https://t.me/prepladderlatestnotes

Treatment of Ischemic CRVO



- 1. Intravitreal anti VEGF drugs (For macular edema)
- Ranibizumab
- Bevacizumab
- Afibercept



- 2. Intravitreal steroids
- Triamcinolone
- Ozurdex



- 3. Pan retinal photocoagulation
- NVE
- NVI
- NVA

Treatment of Non-ischemic CRVO

- If visual acuity >6/12
 - o Observation and follow up
- If visual acuity <6/12
 - o Causes macular edema → Therefore, treatment of macular edema by



- Intravitreal Anti VEGF drugs
 - → E.g., Bevacizumab/Ranibizumab/Afibercept
- o Intravitreal steroids
 - → E.g., Triamcinolone

CENTRAL RETINAL ARTERY OCCLUSION (CRAO)





- M/c cause is embolus
- It blocks the narrowest site of CRA piercing dural sheath of optic nerve just behind the lamina cribrosa



Central retinal artery

 Embolism most commonly is cholesterol plague known as Hollenhorst plaque



Hollenhorst Plaque

Risk factors of CRAO

- Age (>65 yrs)
- Uncontrolled Hypertension
- Uncontrolled diabetes
- Hyperlipidemia
- Smoking
- Family history of cerebrovascular disease

Signs & Symptoms of CRAO

- Sudden painless loss of vision
- Cherry Red Spot (diagnostic finding of CRAO)
- Cattle Truck/Box car appearance



Important Information

Cherry Red Spots are also seen in

Cherry: CRAO

Acute U/L

Trees: Trauma

Never: Niemann pick disease

Grow: Gaucher's disease

Chronic B/L

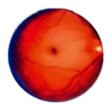
Tall in: Tay Sachs disease

Sand: Sandoff's disease



How to remember

Cherry Trees Never Grow Tall in Sand



Cherry red spot

Cattle - Truck / Box Car Apperance

- Discontinuity in blood column
- Slow & Jerky movement of blood column



Cattle - Truck / Box Car Appearance

- Retinal Ischemic Time: 90 minutes (complete recover can be done)
- Partial recovery: Up to 240 minutes
- Irreversible damage: After 4 hours

Treatment of CRAO

- - 00:41:47

- Ocular massage
- Immediate \(\) of IOP by IV mannitol/Acetazolamide



Important Information

- Ocular perfusion pressure = Mean arterial blood pressure-IOP
- Paracentesis
 - Aspiration of aqueous from anterior chamber



Paracentesis

- Carbogen inhalation
 - Mixture of CO₂ (5%) +O₂ (95%)
 - o CO, prevents vasoconstriction caused due to O,
 - 10 minutes per hour every hour for 48-72 hours
- Hyperbaric oxygen
 - 2.5 atm x 90 minutes within 8 hours
- IV heparin or Tissue plasminogen activator

CYSTOID MACULAR EDEMA



00:49:32

 Fluid causes edema of the macula in the outer plexiform layer

Causes

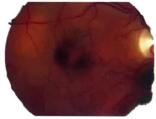
- D Diabetes
- E Epinephrine
- P Pars planitis
- R Retinitis pigmentosa
- I Irvine Gass syndrome
- V Vascular path, CRVO, BRVO
- E E2 prostaglandins
- N Nicotinic acid Niacin > 1500 mg/day
- S Post Surgical
 - Refractive surgery
 - Retinal detachment surgery
 - Glaucoma surgery
 - Keratoplasty



How to remember

DEPRIVENS





Cystoid Macular Edema

Sign & symptoms

- Slow, painless loss of vision
- Metamorphopsia
 - Distortion of shape
 - o Occurs in all macular disorders
 - Two forms
 - Micropsia: objects are small and distorted
 - Macropsia: objects are large and distorted

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Amsler's grid

- Diagnosed by Amsler's grid
- Best instrument to examine CME is + 90 D lens





Slit lamp examination with + 90D lens

Investigations of CME

- 1. Fluorescein Angiography
- 2. Optical Coherence Tomography (OCT)

1. Fluorescein Angiography (FA)

- Dye used is Fluorescein Sodium
- Dye is injected in Ante cubital vein
- Arm retina circulation time 12 seconds
- In case of no pathology
 - o Dye is excreted through urine
- In case of pathology
 - Dye leaks into surrounding retina
 - Leak is recorded by fundus camera
 - o Pattern of leak tells us the pathology
 - → Petalloid appearance (confirms cme)
- Limitations: invasive & may cause allergy



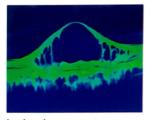




nal FA

2. OCT (Optical coherence tomography)

- Gold standard investigation for all macular disorders
- Non invasive
- Cross section of retina can be studied



Optical coherence tomography

Treatment of CME

- Topical NSAIDS
 - o Bromfenac
 - Nepafenac
- Topical Steroids
 - Prednisolone
 - Dexamethasone
- Sub-Tenon's Injection
 - Triamcinolone



Sub-Tenon's Injection

- Carbonic Anhydrase Inhibitors
 - Acetazolamide
- Anti VEGF agents
 - Ranibizumab
 - Bevacizumab



Previous Year's Questions

Q. Administration of which causes vitamin macular edema and cysts?

(NEET Jan 2020)

01:04:03

- A. Vitamin A
- B. Vitamin D
- C. Vitamin E
- D. Niacin

Berlin's Edema



- Post traumatic macular edema
- Caused due to high impact sprots activities, motor vehicle accidents
- Commotio Retinae
 - Concussive injury of retina
 - o Leading to edema and swelling of retina
- Disruption of photoreceptor outer segments
- Symptoms
 - Sudden painless loss of vision following and injury to the eye
- On examination
 - Grey: White opacification of retina
 - o Pseudo Cherry red spot seen



Pseudo Cherry red spot

Treatment

- Systemic steroids are used
- Clears in 3-4 weeks

CENTRAL SEROUS RETINOPATHY (CSR)

- Idiopathic disorder due to serous fluid between NSL and RPE
- Occurs in young male executive (Type A personality)



Important Information

- CSR: Edema present b/w neurosensory layer & RPE
- CME: Edema present in outer plexiform layer



Central Serous Retinopathy (CSR)

Sings & symptoms of CSR

- Sudden painless loss of vision
- Metamorphopsia



Metamorphopsia

- Lolour perception
- Central scotoma



Central scotoma

- Hyperopic shift
- Halo light reflex

Causes of CSR

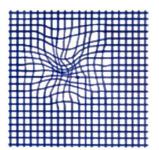
Stress

01:08:09

- Type A personality
- Steroid intake
 - o Inhalers
 - o Intraarticular injections
- Helicobacter pylori infection
- Hypertension
- Obstructive sleep apnea
- Pregnancy
- Drugs
 - o Taken for erectile dysfunction
 - Sympathomimetic agents

Investigations of CSR

Amsler's grid



Amsler's grid

- Fluorescein Angiography
 - o Ink blots and Smoke stacks

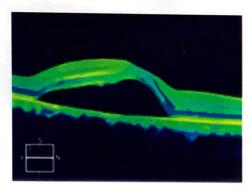




Smoke stacks appearance

Ink blots appearance

OCT



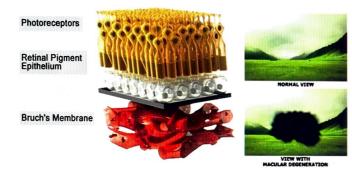
Treatment of CSR

- **Ö** 01:15:58
- Spontaneous recovery in 3 months
- Reassure
- Stop steroids
- For Recurrent CSR
 - o Focal laser photocoagulation
- For chronic CSR
 - o Photo Dynamic Therapy PDT with Verteporfin

AGE RELATED MACULAR DEGENERATION ARMD/AMD



- Degenerative condition of macula with age
- RPE, Bruch's membrane and choricocapillaries all
- Maintain the health of photoreceptors



- Lesions in ARMD are principally found on Bruch's membrane
- Macula affected specifically, sparing peripheral retina

Risk factors of ARMD

- Age > 50: strongest risk factor
- Family history
- Smoking: Strongest modifiable risk factor
- Hypercholesterolemia
- Hypertension
- Obesity
- White race

Sings & Symptoms of ARMD

- Delayed dark adaptation (earliest symptom)
- Slow, painless loss of central vision



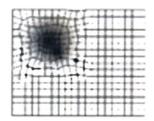


central loss of vision

- Metamorphopsia
- Decreased contrast

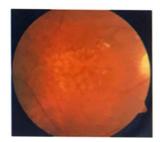


Amsler's grid



Varieties of ARMD

- 1. Dry
- 90%, benign variety
- Self-limiting variety
- Drusen
 - o Lipid deposits on Bruch's membrane of choroid
 - o hallmark
- Geographic atrophy

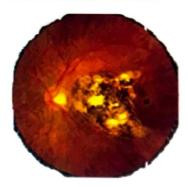


Drusen

- 2. Wet or exudative
- 10%
- Dangerous variety
- Choroidal neovascular membrane (hallmark)



Last stage of wet ARMD is disciform scar



Classification of ARMD

- 1. Early AMD
 - Several small drusen < 63
 - One Medium drusen (> 63 and <125)
- 2. Intermediate AMD
 - Many medium drusen
 - One large drusen > 125
- 3. Late AMD
 - Geographic Atrophy
 - Exudative ARMD



Important Information

 Any drusen greater than diameter of central retinal vein is termed as large drusen

Investigation of ARMD

- Fluorescein Angiography
 - o For classic CNVMs
- · Indocyanine Green Angiography
 - For occult CNVMs
- Fundus autofluorescence (FAF)
 - †in background FAF
- OCT
 - o Fluid marks active Wet AMD
- OCT Angiography
 - Blood flow of retinal and choroidal vasculature can be visualized without injections

Treatment of ARMD

1. For Dry ARMD



- Quitsmoking
 Diet and lifestyle green, leafy vegetables, fish,
- Antioxidants (AREDS 2)
 - o Lutein
 - o Zeaxanthin

antioxidants

- Vit C
- Vit E
- o Zn
- o Cu



2. For Wet ARMD

- Intravitreal Anti VEGF drugs
 - Bevacizumab
 - o Ranibizumab
 - Afibercept



Intravitreal Anti VEGF drugs



Important Information

Location of pars plana is 3.5-4 mm form temporal limbus

Summary of ARMD



- B Bevacizumab
- C CNVM
- D Drusen
- E-RPE
- F Fluorescein Angiography
- G Geographic atrophy
- H Complement factor H



01:33:01

MACULAR HOLE

- A macular hole is a retinal break commonly involving the fovea
- M/cidiopathic

Risk factors

- Old age
- Intraocular surgery
- Myopia
- Trauma
- Chronic CME



Macular Hole



Important Information

M/c presentation of macular hole

 67-year-old lady presents with gradually progressing loss of vision.

Pathology

Vitreo-macular traction at fovea

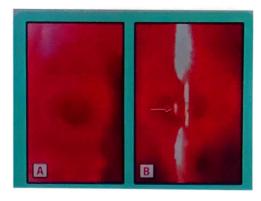
Clinical features

- Painless, gradual loss of vision
- Metamorphopsia
- Central scotoma



Macular hole

- Watzke Allen test
 - o Thin beam of light is projected over fovea
 - o Patient perceives a break in continuity of beam of light



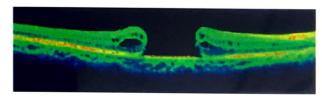
Watzke - Allen test

- Fellow eye at risk: 10% develop macular holes
- Distinguish between
 - Full thickness macular hole.
 - Lamellar hole
 - Pseudohole
 - → No true absence of retinal tissue



Pseudohole

- OCT: used in classification
 - o Small: <250μ
 - Medium: 250-400μ
 - o Large: > 400μ



OCT

Management

- Conservative management
- Spontaneous closure: 5%

Surgery

 Pars plana Vitrectomy with ILM peeling with gas tamponade (most cost-effective treatment)



- Ocriplasmin injection in vitreous (new treatment)
 - Causes vitreolysis
 - relieves posterior vitreous adhesion from retina

RETINOPATHY OF PREMATURITY (ROP)



00:06:10

- · Occurs in premature children
- ROP is a retinal vascular proliferative disorder affecting premature infants undergoing oxygen therapy
- Approximately 60% of high risk babies develop ROP

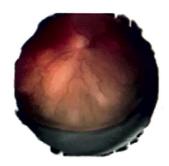


Important Information

- ROP is 2[™] m/c cause of loss of vision in children
- 1st m/c cause of loss of vision in children is Vit A deficiency

Definitions

- Full term baby
 - Between 39 weeks and 40 weeks 6 days
- Pre-Term baby
 - <37 weeks of pregnancy
- Gestational age (GA)
 - Time elapsed between the first day of LMP and the day of delivery, conventionally expressed as completed weeks
- Chronological age (CA)
 - o Time elapsed after birth
- Post menstrual age (PMA)
 - Gestational age + Chronological age



ROP

Risk factors of ROP

- Baby <30 weeks GA
- Birth weight < 1500 gms
- O₂ supplementation



Important Information

 Smallest, sickest, most premature baby has highest risk of developing ROP

The Oxygen Dilemma

Classification	Target SpO ₂
Full term healthy baby	95-100%
Preterm baby	90-94%

Pathogenesis

- Retina of full-term baby is completely vascular
- In Preterm infant vascularity of retina is not complete, extends only up to Ora serrata
- After birth baby is exposed to Hyperoxia
 - 1. High constituent atmospheric O₂
 - 2. Supplemental O₂

Prematurity

Incomplete retinal vascularization

Supplement O₂ (Hyperoxia)

Vasoconstriction

Vaso-obliteration

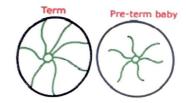
Hypoxia (paradox)

VEGF

Neovascularization

Fibrovascular proliferation

Tractional retinal detachment



Causes of loss of vision

- Tractional Retinal detachment
- Vitreous hemorrhage
- Macular dragging

Screening guidelines

American Academy of Ophthalmology	Indian guidelines
< 30 weeks GA	< 34 weeks GA
< 1500 grams	< 2000 grams
	after delivery or 31 weeks e whichever is later

Stages of ROP

- Stage 1
 - Demarcation line (line where the normal and abnormal vessels meet)



- Stage 2
 - Intraretinal ridge (line with elevation as a result of the growth of the abnormal vessels)



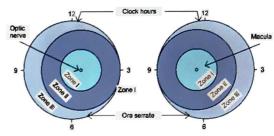
- Stage 3
 - Ridge with fibrovascular proliferation (the ridge grows from the spread of the abnormal vessels and extends into the vitreous)
- Stage 4
 - Subtotal retinal detachment (the partial detachment of the retina)

- Stage 5
 - o Total retinal detachment



ROP - A Primer

- Retina is divided into 3 zones, centred on optic disc
- Zone 1
 - o Small circle of retina around optic disc
- Zone 2
 - Ring of retina around zone 1 extending to ora serrata on nasal side
- Zone 3
 - Crescent shaped temporal retina



PLUS Disease

 Venous dilation and arterial tortuosity present in at least 2 quadrants



Pre-PLUS Disease

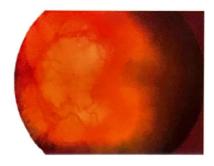
 Venous dilation and arterial tortuosity more than normal but less than plus disease

Aggressive Posterior ROP (APROP)

- Uncommon, rapidly progressing, server form of ROP
- RUSH Disease
- Posterior location (zone 1 or 2)
- Increased dilation and tortuosity of vessels in all

quadrants out of proportion with an incidence rate of 2.5%

- May skip traditional ROP stages
- Requires immediate treatment



APROP

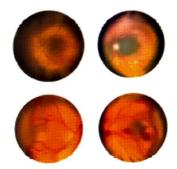
Natural History

- Spontaneous regression n 85% of cases
- In 90% of babies who develop ROP, disease gradually disappears within 15 weeks not requiring treatment and vision develops normally, as long as no other eye problems occurs
- 10% have severe ROP

Treatment

Threshold ROP

- Must be treated
- Threshold ROP
 - ROP if not treated at this level
 - o 50% of children develop blindness
- Four criteria for threshold ROP
 - Zone 1 & zone 2 should be involved
 - 5 continuous clock hours or 8 non-continuous clock hours of fibro vascular proliferation
 - o Stage 3 or beyond
 - o Plus disease



Examples of ROP (crossed threshold)

Type 1 ROP of pre – threshold stage must also be treated

Pre-Threshold ROP

Type 1

- High risk pre threshold ROP which requires laser photocoagulation within 72 hours
- Defined as
 - → Zone 1, any stage with Plus disease
 - → Zone 1, stage 3 without Plus disease
 - → Zone 2, Stage 2 or 3 with Plus disease
- Type 2
 - Low risk pre threshold ROP which requires observation and follow up
 - o Defined as
 - → Zone 1, stage 1 or 2 without Plus disease
 - → Zone 2, stage 3 ROP without disease

Treatment

- Laser photocoagulation
- · Anti-VEGE drugs: Bevacizumab/Ranibizumab used in
 - Zone 1 ROP
 - Zone 2 ROP
 - AROP: Immediate treatment required because it progresses rapidly to detachment





Photocoagulation

Anti-VEGF drugs

RETINITIS PIGMENTOSA



Most Common inherited disorder of retina



Important Information

- Most Common inherited disorder of retina: retinitis pigmentosa
- Most common vascular disorder of retina: Diabetic retinopathy

Mechanisms

 Due to Apoptosis which first damages rod outer segment

Genetics involved

 Autosomal recessive (m/c) Autosomal dominant / Xlinked (worst prognosis)/sporadic

Clinical features

Nyctalopia (earliest)



Ring scotoma



Progressive loss of visual fields leading to tunnel vision



Clinical features

- Triad
- 1. Pale waxy disc
- 2. Arteriolar attenuation
- 3. Bone corpuscular pigmentation

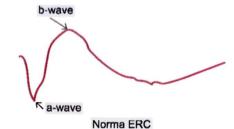


- Associated with
 - Posterior subcapsular cataract
 - o Macular edema
 - Keratoconus

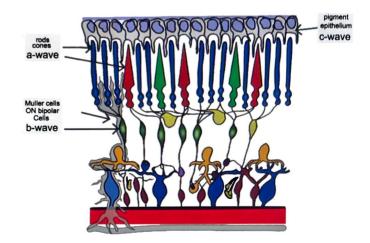
Investigations

- Dark Adaptometry ↑ (Normal DAT: 20 30 minutes)
- · Visual Fields analysis shows Ring scotoma

- · ERG (Gold standard)
 - Measures electrical activity of retina
 - o Flat ERG confirms retinitis is pigmentosa
 - Normal ERG has 3 waves
 - → a Wave (Negative)
 - → b Wave (Positive)
 - → c Wave



Origin of ERG waves



Wave	Origin
a-wave	R-Rods & Cones (photoreceptors)
b-wave	M-Muller cells > Bipolar cells
c-wave	P- Retinal pigment epithelium



How to remember

RMP

Flat ERG Confirms retinitis pigmentosa

Refer Image 9.1



Previous Year's Questions

Q. "a 'wave in ERG is seen in? (JIPMER Dec 2019)

A. Muller cells

B. Rods and cones

C. Bipolar cells

D. RPE activity

.........

- No proven therapy
- 15,000 IU/day of Vit A in Palmitate form every day for life
 - Watch out for hip fractures because excessive vit A causes osteoporosis
- · Weekly consumption of at least 2 servings of oily fish in which DHA (Docosahexaenoic Acid) Component present
- Daily 200 mg DHA
- Daily 12 mg Lutein
- ARUGS II implant: FDA approved/Bionic eye



ARUGS II implant (Bionic eye)



Important Information

Retinitis Pigmentosa

- · X-linked recessive
 - · Has the earliest onset
 - Has worst prognosis
- Patient becomes blind by 30 years
- Ultraviolet absorbing sunglasses recommended
 - Amberfilter is placed
- CME of RP is treated by Acetazolamide
- RPInversa
- Cone rod Dystrophy (CORD) cause daytime blindness



Retinitis Pigmentosa Syndromes



00:44:55

- Bassen Kornzwig Syndrome
- Laurence Moon Syndrome
- Bardet-Beidl Syndrome
- Usher Syndrome (M/C)
- Refsum disease
- Wardenburg syndrome
- Alport syndrome



Previous Year's Questions

Q. Which of the parameters is decreased in Retinitis pigmentosa?

(NEET Jan 2019)

- A. Arachidonic acid
- B. Docosahexanoic acid
- C. Thromboxane
- D. Trielonic acid



Previous Year's Questions

Q. Oquchi's disease is?

(JIPMER May 2019)

- A. Retinitis pigmentosa
- B. Fundus albipunctatus
- C. Congenital stationary night blindness
- D. Cone dystrophy



Previous Year's Questions

Q. Which of the following is seen in retinitis pigmentosa?

(FMGE June 2019)

- A. Arteriolar attenuation
- **B.** Neovascularisation
- C. Retinal artery thrombosis
- D. Papilledema

JUVENILE MACULAR **DEGENERATIONS**



00:45:45

- Groups of inherited macular disorders
- · Affects children and young adults
- Stargardt's Disease (M/c)
 - o Profound central loss of vision



Ultraviolet absorbing sunglasses Join the Telegram Channel - https://t.me/prepladderlatestnotes

- Best's Vitelliform Macular Degeneration (2nd commonest)
- X-linked Juvenile Retinoschisis

Best's disease	Stargardt's disease (m/c)
Autosomal dominant	Autosomal Recessive
 Moderate loss of Vision 	 Severe loss of Vision
Hypermetropia is associated	Myopia is associated

- O/E
 - Scrambled egg appearance (egg yolk appearance)
- O/E
 - Beaten bronze sheen with yellow pisciform flecks





ERG: Normal

EOG (Electrooculogram)

Diagnostic (measures potential difference b/w cornea & retina)

ERG: Normal

EOG: Normal

Confirmed by

Arden ratio < 1.5

Confirmed by

 Fluorescein Angiography (confirmatory)

Dark choroid





Previous Year's Questions

Q. Dark choroid in FFA is seen in?

(JIPMER May 2019)

- A. Retinitis pigmentosa
- B. Age related macular degeneration
- C. Leber's congenital dystrophy
- D. Stargardt's disease

Treatment

- No treatment
- Low visual aids



Low visual aids

- Genetic counselling
- Avoid high doses of Vitamin A
- Good sun protection
- Stop smoking



Previous Year's Questions

Q. Mizuo Nakamura phenomenon in fundus is seen in?

(JIPMER Nov 2019)

- A. Congenital rod absence
- B. Best Disease
- C. Congenital stationary night blindness
- D. Congenital cone defect

RETINOBLASTOMA



- M/c primary tumor of eye in children
- Commonest age of presentation 18 Months, usually within 5 years



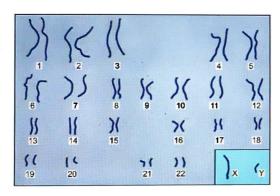
Retinoblastoma

Classification

1	Sporadic	90%
	Familial	10%
2	Unilateral	70%
	Bilateral	30%
3	Germline (Heritable)	40%
	Somatic (Non- Heritable)	60%

Genetics

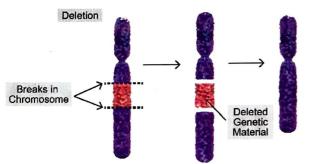
- RB gene (RB₁)
 - o Code instructions of PRB (protein retinoblastoma)
- RB/Gene is the first tumor suppressor gene
- It is located on 13q14
 - o 13-Chromosome
 - o q Long arm
 - o 14-Band



Karyotyping

- Knudson's 2 hit hypothesis
- 2 mutations one to each allele of RB₁ which inactivates the suppressor gene

Retinoblastoma is not suppressed anymore





Important Information

- 90/RB patients have mutations
- 107 RB occurs due to chromosomal 13 long arm deletion
 - o Absence of RBI gene
 - o Called as 13q minus syndrome

Clinical features

Leucocoria (Amaurotic cat's eye pupil) (m/c)



Leucocoria (Amaurotic cat's eye pupil)

- Strabismus (2nd m/c)
- Neovascular glaucoma (3rd m/c)
- Red painful eye
- Pseudohypopyon



Pseudohypopyon

- Loss of vision
- Heterochromia
- Hyphema
- Uniocular mydriasis

Differential diagnosis of Leucocoria



- Retinoblastoma
- Persistence of Fetal Vasculature (PHPV)
 - Differentiated by presence of
 - → Microphthalmos
 - → U/L Congenital cataract



Persistence of Fetal Vasculature

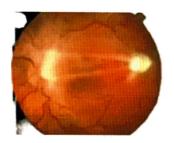


Congenital cataract

Coat's Disease (only seen in boys)



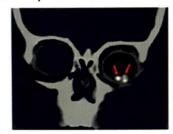
Toxocariasis



- ROP
- Endophthalmitis
- Retinal detachment
- Choroidal coloboma

Diagnosis

- Retinoblastomas are mostly diagnosed Clinically
- Ultrasonography
- CT: Intraocular calcification (avoid, radiation induces second cancers)



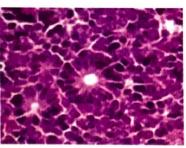
Intraocular calcification



Important Information

M/c cause of intraocular calcification is retinoblastoma

- MRI (Investigation of choice)
- Flexner Winter Steiner rosettes (Hallmark)



Flexner Winter Steiner rosettes

Management

- 1. Primary goal Save life
- 2. Secondary goal Save eye
- 3. Tertiary goal Save vision



International Classification of Retinoblastoma 00

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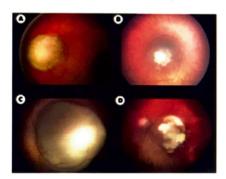
Stage A	• ≤3 mm
Stage B	 >3 mm Macular location Minor sub – retina fluid
Stage C	Localized seeds within 3 mm, sub retinal or vitreous
Stage D	Diffuse seeds more than 3mm
Stage E	 Massive Retinoblastoma more than 50% of eyeball, with secondary glaucoma, hemorrhage

Treatment

- Focal
 - Cryotherapy
 - ightarrow Transscleral cryotherapy freezes tumor with ice ball
- Laser photocoagulation
 - o Argon green laser
 - Used indirectly on tumour
 - o Coagulates blood vessels feeding tumour

tps://t.me/prepladderlatestnotes

- Transpupillary Thermo Therapy (TTT)
 - Hyperthermia by infrared radiation
 - Temperature of 40 to 600 C using a semiconductor diode laser
- Chemotherapy: Four types
- 1. Intravenous (IVC)
 - VEC (Vincristine, Etoposide Carboplatin) regimen
 - o for 6 cycles
- 2. Intravitreal (IVit)
 - o Melphalan (m/c) Topotecan
 - Used for Recurrent vitreous seeds
 - Risk of extraocular extension
- 3. Intraarterial (IAC)
 - Melphalan (m/c), Topotecan, Carboplatin injected into ophthalmic artery
- 4. Periocular (POC)
 - Posterior sub Tenon's injection of Carboplatin or Topotecan in the quadrant closest to location of vitreous seeds
 - Advanced tumour group D or E with diffuse vitreous seeds
 - o Risk of eyelid edema and ecchymosis



Chemotherapy results

- Radiotherapy
 - o lodine 125, Cobalt-60, Ruthenium-160
 - o Placed on sclera corresponding to base of tumour



Plague radiotherapy

- Used for Recurrent/residual tumour
- Radiation retinopathy

- External Beam Radiotherapy (EBRT)
 - Orbital RB/Vitreous seeding
 - Complications of EBRT
 - → Midface hypoplasia
 - → Radiation cataracts (m/c)
 - → Second cancers develop
- Enucleation
 - o Indications for Enucleation
 - → Advanced intraocular retinoblastoma with NVI
 - → Secondary glaucoma
 - → AC invasion
 - → >75% of vitreous volume
 - → Hyphema
 - → Necrotic tumors with orbital inflammation
 - o 15 mm optic nerve is also taken out along with eye ball
 - o Implant porous, (hydroxyapatite)
- Role of implants
 - o It replaces volume lost
 - Provides better prosthetic
 - o Better cosmetic improvement



Hydroxyapatite implants

- Decision making in Management
 - Unilateral less advanced (A, B, C) Focal therapy, IVC IAC
 - Unilateral advanced (D, E) IVC, IAC, Enucleation
 - Bilateral less advanced (A, B, C) IVC
 - Bilateral advanced (D, E) IVC + POC

Causes of death in Retinoblastoma

- Metastases: M/c optic nerve within one year
- Intracranial tumours associated with Rb
- Pinealoblastoma/PNET
 - Trilateral retinoblastoma
 - → Bilateral RB with central pinealoblastoma
- Secondary tumours Osteosarcoma of the femur



Important Information

- Most common route of metastasis of RB is via Optic nerve
- Most common site of metastasis in RB is CNS



Important Information

Q. A 7-year-old boy is brought with complaints of defective vision in his right eye for 3 months. ON, yellow reflex is seen in the pupillary region of the right eye. On fundus examination, Large, raised yellowish areas of exudation is seen in the posterior Role of the fundus. Most probable diagnosis?

(JIPMER May 2019)

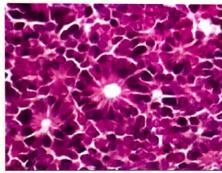
- A. Toxocariasis
- **B.** Toxoplasmosis
- C. Coats disease
- D. Incontinentia pigmenti



Important Information

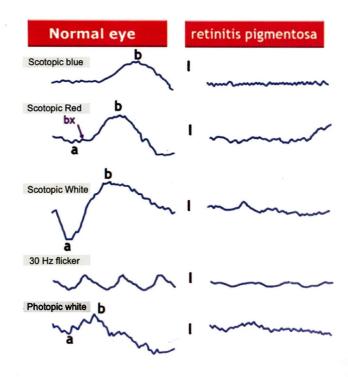
Q. A child with whitish pupillary reflex has undergone enucleation and shows bright round blue cells with Flexner wintersteiner rosettes. What is the diagnosis?

(NEET Sep 2021)



- A. Retinoblastoma
- B. Rhabdomyosarcoma
- C. Medulloblastoma
- D. Astrocytoma

Image 9.1



ERG





Q. A 35-year-old male with a history of non-insulin-dependent DM x 3 years presents to the Eye Clinic with c/o progressive blurring of vision since his last visit 3 years ago. Thispatient should have been screened for Diabetic Retinopathy at what duration from the time of diagnosis of Diabetes?

A. Type 2 DM Immediately

B. Type 2 DM Within 6 months

C. Type 2 DM Within 1 year

D. Type 1 DM Within one week

Answer: A

Solution

Diabetic Retinopathy Screening

The most important risk factor for the development of Diabetic Retinopathy is the duration of Diabetes mellitus

- In patients with diabetes before 30 years age (most likely Type 1 DM), incidence of DR after 10 years if 50% and 90% after 30 years
- In patients with Type 2 DM, 5% of patients have DR at the time of presentation/diagnosis
 Hence suggestions for DR Screening are:
- Type-1 diabetes: Within 5 years of diagnosis.
- Type-2 diabetes- Immediate fundus examination
- MODY & GDM: Immediate

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, Chapter 13 - Retinal Vascular Diseases, page 497

Q. A 30 year old women presented with history of nyctalopia and constriction of visual field of the left eye. On fundus examination, a pale optic disc, narrowed arterioles and extensive proliferations of the pigment epithelium in form of bone spicules in the periphery could be seen. The doctor made the diagnosis of retinitis pigmentosa. Which of the following is not associated with Retinitis pigmentosa?

A. Refsum's disease

B. Hallervorden-Spatz disease

C. NARP

D. Abetalipoproteinemia

Answer: B

Solution

Remember RP associations by mnemonic LUCHR.

- Lawrence-Moon-Beidel Syndrome
- Usher Syndrome
- Cockayne Syndrome
- Hallgren Syndrome
- Refsum Syndrome

Other associations: NARP, Abetalipoproteinemia

Retinitis Pigmentosa is not associated with Hallervorden Spatz Syndrome

Reference: Comprehensive Ophthalmology 6th Edition AK Khurana Page no.302

- Q. A 25 year old patient presented to the ophthalmology OPD with complains of floaters and painless loss of vision in the left eye. The patient stated that nine days prior he was struck in the face by shrapnel from an explosion at his workplace, for which he did not seek medical attention at that time as there was no hospitals nearby. After detailed evaluation by the ophthalmologist he was found to have rhegmatogenous Retinal Detachment. All of the following are risk factors for Rhegmatogenous Retinal Detachment except.
- A. Pilocarpine
- B. Aphakia
- C. Lattice degeneration
- D. Hypermetropia

Answer: D

Solution

Hypermetropia is not a risk factor for Rhegmatous retinal detachment. Risk factors for RRD

- Myopia (especially high myopia)
- Previous intra-ocular surgery
- Aphakia, Psuedophakia
- · Family History of Retinal Detachment
- Trauma
- Inflammation
- Retinal Necrosis Acute Retinal Necrosis, CMV Retinitis
- Drugs Pilocarpine

Reference: Parsons' Diseases of the Eye, 22nd Edition, 2015, Section IV - Diseases of the Eye, Chapter 20 - Diseases of the Retina, page 331.

- Q. A 65 year old woman admitted in the oncology department for management of breast cancer which metastases to brain. A week after her admission she started experiencing sudden painless loss of vision. After evaluation by the doctors she is found to have exudative retinal detachment. Which of the following is not a cause of exudative retinal detachment?
- A. Toxemia of pregnancy
- B. Malignant hypertension
- C. Posterior scleritis
- D. Diabetic retinopathy

Answer: D

Solution

EXUDATIVE RETINAL DETACHMENT

Serous fluid accumulates between RPE & Neurosensory retina due to leakage from vasculature. There is no hole or tear in retina

Causes:

- Choroidal Tumours
 - o Choroidal Melanoma
 - o Haemangioma including Von Hippel Lindau Disease

- Metastasis
- Inflammation
 - Vogt Koyanagi Harada Disease
 - Sympathetic Ophthalmia
 - Retinal vasculitis
 - Posterior Scleritis
- Choroidal Neovascularization
- Hypertensive choroidopathy
 - Toxemia of Pregnancy
 - Malignant Hypertension

Diabetic retinopathy can cause tractional retinal detachment or combined rhegmatogenous-tractional retinal detachment but not exudative retinal detachment

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, Chapter 16 - Retinal Detachment, page 681

- Q. A 33-year-old woman presents with a 3-day history of seeing "spots" floating around in her eye. Today, she notes streaks of light in the same eye accompanied by a "shadow" in her peripheral vision that moves when looking up and down. Her medical history is significant for migraine headaches; however, she has never had such visual symptoms with her headaches. What is the most likely diagnosis?
- A. Ophthalmic migraine
- B. Malingering
- C. Retinal detachment
- D. Amaurosis fugax

Answer: C

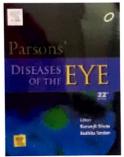
Solution

Retinal detachment:

- The history suggests retinal detachment, the vitreous detach from the retina and causes the perception of "floaters"
- Vitreous detaching from the retina may cause a peripheral "horseshoe" tear in susceptible areas of the retina. The tear allows fluid within the vitreous to accumulate under the retina and cause a detachment.
- When the retina is partially detached and the eye moves, the patient may notice a "shadow" that corresponds to the torn retina
- Retinal detachment is ophthalmic emergency because the detachment can progress to the fovea and threaten central vision.

Opthalmic migraine: An eye condition that causes brief attacks of blindness or visual problems like flashing lights in 1 eye. **Amaurosis fugax**: Condition in which a person cannot see from one or both eyes due to transient failure of blood supply to retina.

Reference:







THYPERTENSIVE RETINOPATHY

- Arterial Venous Crossings of Retina
- Retinal Changes in Hypertensive Retinopathy
- Acute Choroidopathy
- Acute Optic Neuropathy



10

HYPERTENSIVE RETINOPATHY

- Changes occurring in retina due to uncontrolled elevated Blood Pressure.
- WHO defines HTN AS> ¹⁴⁰/₉₀
- Raised BP causes
 - Retinopathy (M/C)
 - Choroidopathy
 - Neuropathy



Retinopathy

Phases in Hypertensive retinopathy

- Acute phase: Due to Vasospasm
- 2. Chronic phase: Due to Arteriosclerosis

PATHOPHYSIOLOGY



- Retinal blood vessels have 3 distinct features
 - Absence of sympathetic nerve supply
 - Autoregulation of blood flow
 - Presence of blood retinal barrier

CLASSIFICATION

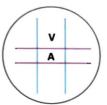


Modified Scheie's classification

Grade 0	No changes
Grade 1	Barely detectable arterial narrowing
Grade 2	Obvious arterial narrowing with focal irregularities
Grade 3	- • Grade 2 + retinal hemorrhages/ exudates
Grade 4	• Grade 3 + disc swelling

ARTERIAL VENOUS CROSSINGS OF © 00:03:56 RETINA

 Sites in retina where veins & arteries cross over each other.



Arterial Venous Crossings

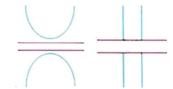
- A: Arteries (narrower)
- V: Veins (dilated)
- Normally A:V ratio = 2:3
 - Means if Artery diameter is 2x vein diameter is 3x.
- In Sustained HTN A:V ratio becomes 1:3
- In 70% cases: Arteries cross over veins In 30% cases: Veins cross over arteries
- Because of compensated hypertrophy

Arteries become thick walled

Start compressing thin-walled veins (Mostly, arteries lie over the veins)

Site of max AV crossings → Supero-temporal quadrant

Has max Branched retinal venous occlusions (BRVO)



- Normal arterial wall is transparent
- Blood column seen in Normal Arterial light reflex (ALR) is narrow: About – ¼ diameter of blood column
- But due to uncontrolled HTN

Wall thickens & becomes less transparent

So, blood column is not clearly visible anymore

Signs seen

Copper wiring: ALR widens and coppery colour

- Silver wiring
 - Wall becomes opaque & no blood visible
 - More advanced stage than copper wiring



Silver wiring

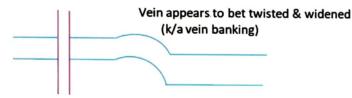
- AV nicking/AV nipping
 - o Nicking means to cut
 - Vein appears to stop abruptly
 - As artery thickens & becomes less transparent

Veins running under artery appears to be cut this is K/a AV nicking/AV nipping

- Banking
 - Vein twisted on distal side and widens
 - Because of the thickened artery present on the delicate vein
 - o As artery thickens

It compresses the vein

Vein appears twisted & widened



Banking

SIMPLIFIED CLASSIFICATION: BY WONG AND MITCHELL

o 00:11:03

Divides retinopathy in to 3 stages

Mild	 1 or more of the following signs Generalized arteriolar narrowing Focal arteriolar narrowing AV nicking
Moderate	 1 or more of the following signs Retinal hemorrhages (dot, blot, flame shaped) Cotton wool exudates Hard exudates Micro aneurysms



RETINAL CHANGES IN HYPERTENSIVE RETINOPATHY



Hemorrhages

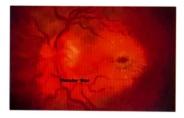
- Dot & blot hemorrhages
 - o Bleeding in inner retinal layers
- Flame shaped hemorrhages
 - Bleeding in superficial retinal layers

Exudates

- Hard exudates
 - o Lipid deposits on the retina
- Soft exudates
 - Cotton wool spots
 - o Axoplasmic debris on the NFL

Macular changes

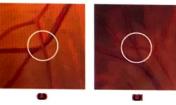
Macular star



Focal signs

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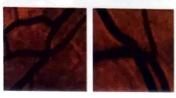
- Gunn's sign
 - Tapering of veins on either side of crossing

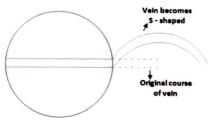




Gunn's sign

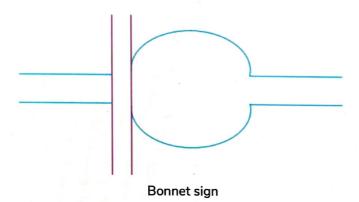
- Salus sign
 - S Shaped deflection of vein after AV crossing





Salus sign

- Bonnet sign
 - Banking of vein distal to AV cross



ACUTE CHOROIDOPATHY



Because of sudden rise of BP, the Choroid gets damaged

Signs of choroidopathy

- Elsching's spots
 - Hyperpigmented spots surrounded by hypopigmentation



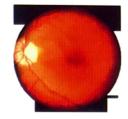
- Siegrist streaks
 - Linear hyperpigmentation's following choroidal arteries



ACUTE OPTIC NEUROPATHY



- Optic disc pallor
- Optic disc edema
- Papilledema



Acute Optic Neuropathy

TREATMENT OF HYPERTENSIVE RETINOPATHY

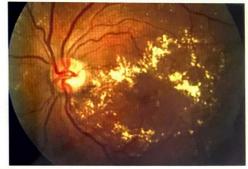


- Control BP
- · Watch out for
 - CRAO & BRAO
 - o CRVO&BRVO
 - o ARMD
 - o CSR

?

Previous Year's Questions

Q. A elderly female presents with gradual painless vision loss, the Fundus image given below. What is the diagnosis? (NEET Sep 2021)



A. Hard exudates in DM

- B. Flame shaped haemorrhages in HTN
- C. Soft exudates in HTN
- D. CRVO





- Q. A 27 year old pregnant woman comes to your ophthalmology clinic accompanied by her husband. She is a G2P1, she came as her gynecologist recommended her a to visit the ophthalmologist for evaluation of her vision as she was having hypertension. Since she is having a risk factor, which of the following would be the earliest ocular manifestation of pregnancy induced hypertension?
- A. Soft Exudates
- B. Flame shaped hemorrhages
- C. Constriction of nasal blood vessels
- D. Papilledema

Answer: C

Solution

Fundus changes in Pregnancy Induced Hypertension (Clinical Manifestations in PIH) The clinical course of fundus changes in PIH may be divided into three stages:

- (i) Spastic stage: Characterised by spasm of retinal arterioles.
- (ii) The stage of sclerosis: When pregnancy induced hypertensive changes are superimposed on pre-existing organic sclerotic changes in the vessels.
- (iii) The stage of retinopathy: Characterised by cotton wool spots, micro aneurysms, flame shaped and splinter haemorrhages, hard exudates, disc edema etc.
 - The first change observed in normal retinal arterioles is constriction of the lumen of superior nasal arterioles.

Reference: Comprehensive ophthalmology A K Khurana 6th edition Pg276

- Q. 65 year old man came to eye OPD with chief complaints of metamorphosia. On examination he has yellow deposits subretinal near macula in both eyes. Rest of the fundus is normal. What is the most probable diagnosis?
- A. Hypertensive retinopathy
- B. Age related macular degeneration
- C. Eales disease
- D. Diabetic retinopathy

Answer: B

Solution

Metamorphosia , age of the patient and the yellowish exudates beneath the retina pointing towards age related macular degeneration

Age related macular degenerations (ARMD

It is a degenerative disease of persons above the age of 50 years that is characterized by the following abnormalities in the macula:

- Soft drusen
- Hyperpigmentation and/or hypopigmentation of the RPE
- Peri retinal haemorrhages or Geographic atrophy of RPE or retinal fibrous scarring in the absence of other retinal vascular disorders

Types of ARMD:

- Dry ARMD
- Wet ARMD

Reference: Comprehensive ophthalmology A K KhuranaPg 295





NEURO OPHTHALMOLOGY

- CLINICAL ANATOMY AND PHYSIOLOGY OF OPTIC NERVE
- OPTIC NEURITIS
- TOXIC AMBLYOPIA / TOXIC OPTIC NEUROPATHY
- ISCHEMIC OPTIC NEUROPATHY
 - o Anterior ischemic Optic Neuropathy (AION)
 - → ArteriticAION (AAION)
 - → Non arteriticAION (NAION)
 - o Posterior ischemic Optic Neuropathy (PION)
- ABNORMALATIES OF PUPIL
- PAPILLOEDEMA
 - o Pseudo-papilledema
 - o Papilloedema Vs Optic Neuritis
- VISUAL PATHWAY and LESSIONS
- GAZE CENTRES AND LESSIONS
- OCULAR MYASTHENIA GRAVIS (OMG)
- NYSTAGMUS



11

NEURO OPHTHALMOLOGY

CLINICAL ANATOMY AND PHYSIOLOGY OF OPTIC NERVE



OPTIC NERVE



- It is 2nd Cranial nerve which transmits impulses for vision
- It is Derived from Optic Vesicle, not a true nerve, but an extension of the brain, myelinated by oligodendrocytes and not Schwann cells

Three order neurons

- Light → photoreceptors (First order) → Bipolar cells (2nd order) → Ganglion cells (3rd order)
- On → axons of the retinal Ganglion cells while exiting the retina, turns sharply and enters the optic disc and becomes myelinated after leaving the scleral canal
- 1.2 million axons in each optic nerve
 - o ON is 50 mm long, S shaped in orbit

Parts of optic nerve

1. Intraocular

- 1mm

2. Intraorbital

- 30 mm

3. Intracanalicular

-5-10mm

4. Intracranial

- 10 mm



Parts of optic nerve

Optic nerve head (ONH)

- Also called as Optic disc
- Has 4 parts
 - o Superficial nerve fibre layer,
 - o Prelaminar.
 - o Lamina cribrosa,
 - Retrolaminar
- Central retinal artery enters optic nerve 1 cm behind globe

?

Previous Year's Questions

- Q. First order neuron in visual pathway?
- A. Bipolar cells

(AIIMS May 2019)

- B. Ganglion cells
- C. Photoreceptors
- D. Lateral geniculate body

OPTIC NEURITIS



- Acute inflammatory demyelinating disorder of the optic nerve
- Typically, in young ladies between 20-45 years
- M/c cause of optic neuritis multiple sclerosis
- Neuromyelitis Optica / NMO / Device's Disease: Bilateral optic neuritis

Types

- ON/Papillitis
 - 0 40%
 - o Inflammation of ON anteriorly close to optic disc
- Retrobulbar neuritis
 - 0 60%
 - o Inflammation of ON posteriorly away from optic disc



Important Information

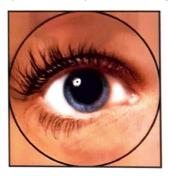
- Bilateral optic neuritis: Neuromyelitis Optica
- Unilateral optic neuritis: Multiple sclerosis
- A young lady come with recurrent optic neuritis.
 most probable diagnosis is multiple sclerosis
- M/c ocular manifestation of multiple sclerosis is Optic neuritis.

Clinical Features

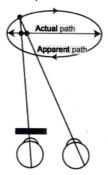
- Sudden painful loss of vision
- Pain with ocular movement
- Colour vision desaturation



- Worsening of symptoms with ↑ body Temperature (Utthoff phenomenon)
- Abnormal pupil reactions (Marcus Gunn pupil/RAPD)



 Course of pendulum perceived as elliptical movement (Pulfrich phenomenon)



Visual field defect: M/c - Central scotoma



- There should be spontaneous improvement in optic neuritis starting within 3 weeks
- Hallmark: Disc edema



Retrobulbar neuritis; absence of disc edema



?

Previous Year's Questions

- Q. When a small target is oscillated in front of a patient with binocular vision, patient sees movement of the object in elliptical orbit rather than to and fro path. What is this phenomenon known as?

 (JIPMER May 2018)
- A. Oppenheim phenomenon
- B. Pulfrich phenomenon
- C. Uthoff phenomenon
- D. Paroxysmal convergence spasm

Investigations

- MRI with contrast
- Visual evoke potential
- Lumbar puncture: CSF Oligoclonal bands confirms Multiple sclerosis
- Neuromyelitis Optica (NMO/ Devic's Disease): Aquaporin 4 antibody - AQ4 Ab

Treatment

- 1 gram IV methylprednisolone x 3 days
- Oral prednisolone contraindicated
- IV steroids may accelerate visual recovery but no effect on ultimate visual gain
- Treatment with immunomodulators (Interferon beta 1a and 1b) may be considered for patients whose MRI findings suggest high risk of developing Multiple sclerosis

TOXIC AMBLYOPIA / TOXIC OPTIC NEUROPATHY



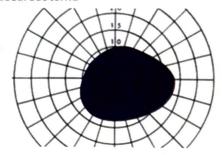
- Painless, progressive, bilaterally symmetric loss of vision due to consumption of certain drugs like
 - Tobacco
 - Alcohol
 - Chloramphenicol
 - Chloroquine
 - Ethambutol/Isoniazid
 - Amiodarone/Digitalis

Clinical Features

- Loss of vision
- Poor colour perception,
- No RAPD if bilateral
- Pale atrophic discs



Centrocecal scotoma



Affects papillomacular bundle

Treatment

- Withdrawal of Drugs
- Well balanced high protein diet with B complex vitamins

ISCHEMIC OPTIC NEUROPATHY



00:22:29

Types 1. Anterior ischemic Optic Neuropathy (AION)

- M/C
- 2 types
 - o Arteritic AION (AAION): Giant cell arteritis
 - o Non arteritic AION (NAION): More common
- 2. Posterior ischemic Optic Neuropathy (PION)
- AION involves the ONH, an acute ischemic event of PCA
- PION involves rest of ON

NON ARTERITIC ANTERIOR © 00:24:13 ISCHEMIC OPTIC NEUROPATHY (NAION)

- More common
- Infarction of anterior part of optic nerve with ischemia of short posterior ciliary artery (SPCA)

Causes

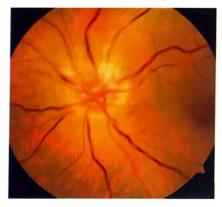
- Idiopathic (M/c)
- Sleep Apnoea syndromes
- Medications: Anti-hypertensive, Sildenafil
- Nocturnal hypotension

Clinical findings

- Sudden painless loss of vision in middle aged/elderly patients on getting up in morning
- Loss of colour vision
- Inferior altitudinal hemianopia: most characteristic field defect
- RAPD
- Disc edema, fellow eye small, crowded disc 'disc at risk'



Altitudinal hemianopia



Disc edema (NAION)

Investigations

- ESR/CRP
- Visual field analysis
- OCT: disc edema

Treatment

- Spontaneous partial recovery: 40 %
- Systemic steroids
- Intravitreal triamcinolone
- Optic nerve decompression



Important Information

Main task is to exclude AAION

ARTERITIC ANTERIOR ISCHEMIC OPTIC NEUROPATHY (AAION)



- Caused by Giant cell arteritis
- Seen in White, elderly females > 60

Symptoms

- Fatique
- Weight loss
- Fever
- Temple pain
- Jaw claudication (most specific)
- Scalp tenderness
- Massive headaches
- Temporal artery pulsation

Ocular symptoms

- Amaurosis fugax
- Transient diplopia
- Sudden severe (<6/60) painless loss of vision
 - o Altitudinal field defect, RAPD
 - Pale disc edema (Hallmark)



Investigations

Gold standard: Temporal artery biopsy

Treatment of AAION

- Systemic corticosteroids / IV, if loss of vision
- GCA on systemic steroids have dramatic relief in headache and malaise in 24 hours, but only 10% have improvement in visual loss
- If left untreated, visual loss may be bilateral within weeks in 50%



Previous Year's Questions

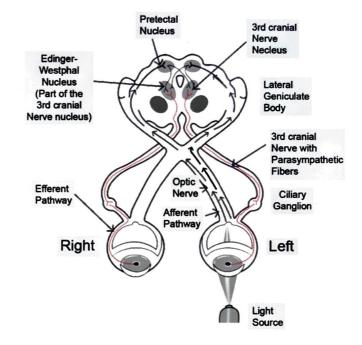
- Q. Photo stress test is used to differentiate visual loss between, (NEET Jan 2020).
- A. Cataract and glavcoma
- B. Cornea and lens disease
- C. Macular and optic nerve disease
- D. vitreous and retina

PUPILLOMOTOR PATHWAYS



- Rays of light from pupil focus on Retina
- From retina and fovea impulse travels to Optic nerve
- Afferent fibres start from ON travel to Optic chiasma
- At optic chiasma some of fibres cross to opposite optic

- tract (First Crossing) while majority passes to same sided optic tract
- Just before reaching LGB Pupillary fibres come out of optic tract and anastomose with pretectal nucleus of midbrain
- From pretectal nucleus of midbrain half fibres pass to opposite side Edinger-Westphal nucleus (2nd Crossing) while other half passes to same side EWN
- Efferent fibre starts from EWN travel along with third cranial nerve and join with ciliary ganglion
- Post ganglion fibres come from ciliary ganglion and innervate lris sphincter
- 97% of Efferent fibre innervate ciliary body while only 3% goes to iris sphincter



Pupillomotor Pathways

Double hemi-decussation (two crossing)

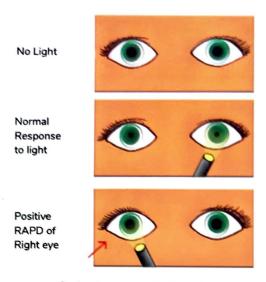
- 1. Chiasma
- 2. Between pretectal nuclei and Edinger: Westphal nuclei which allows direct and consensual reactions to be equal



Light reflex

ABNORMAL PUPIL

1. MARCUS GUNN / RELATIVE AFFERENT PUPILLARY DEFECT 65 00:40:10



Swinging torch light test

- Pupil instead of constricting in response to light, dilates paradoxically and opposite pupil is also dilated.
- Occurs when there is unilateral / Asymmetric damage to ON
- Conditions leading to Marcus Gunn Pupil
 - o Optic neuritis
 - o AION/PION
 - o Traumatic optic neuropathy
 - o Optic nerve glioma
 - Significant retinal detachment
 - o Large macular lesion
 - o CRAO
 - Ischemic CRVO



Important Information

- Any unilateral/Asymmetric damage from ON to LGB can lead to RAPD.
- Damage beyond LGB do not leads to RAPD

2. ARGYLL ROBERTSON PUPIL



- Three features
 - Bilateral
 - Constricted
 - Irregular pupils
- Seen in Neurosyphilis
- Obeys Light negative and accommodation positive



Argyll Robertson pupil





 Aka "Prostitute 's Pupil": behaves like a prostitute (accommodates but does not react)

3. ADIE'S TONIC PUPIL





Adie's Tonic pupil

- Unilateral dilated pupil usually seen in young ladies 70%
- Seen after viral fever
 - o 1 pupil dilates and other remains normal
 - o Virus attacks ciliary ganglion → III CN affected
 - → Parasympathetic constrictor fibres of pupil damaged
 - → Unopposed sympathetic dilation of pupil occurs.
- · 97% fibres go to ciliary body, 3% to iris sphincter

Pathology

- Aberrant regeneration of the damaged parasympathetic fibres going from the ciliary ganglion, CB fibres are misdirected towards the pupil
- Obeys Light negative, accommodation positive





Adie's Tonic pupil

Argyll Robertson Pupil	Adie's Pupil
Constricted pupilB/L	Dilated pupilU/L

4. HOLMES ADIE'S PUPIL

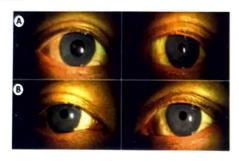
Sectorial iris paralysis





Hallmark

 Strong, tonic response to near and slow and sustained relaxation



Confirmatory test

- Pilocarpine 0.125% solution is used
- Normal pupil does not constrict
- Adie's pupil will constrict due to denervation super sensitivity

Treatment

Spontaneous recovery

5. HORNER'S SYNDROME



There is Oculo sympathetic palsy (OSP)



Important Information

- Ist order: Hypothalamus: Ciliospinal centre of Budge (C8-T2)
- 2nd order: Ciliospinal centre of Budge: Superior cervical ganglion
- 3rd order: superior cervical ganglion: Dilator pupillae

Functions of sympathetic system in the eye

- Sympathetic innervation of eye
- Dilator pupillae, Muller's/inferior tarsal muscle
- Sweat glands secretion
- · IOP: Regulation of secretion of aqueous

Clinical Features

- Triad
 - Mild Ptosis
 - Miosis
 - Anhydrosis
- Enophthalmos
 - Not a part of Horner's syndrome
 - Not a true Enophthalmos
- Dilation lag
 - Slow dilation of pupil in darkness
 - o Characteristic features of Horner's Syndrome



Important Information

- · Normally.
 - Lower eye lid present at limbus (d/t inferior tarsalmuscle)
 - o Upper eye lid covers 2 mm (d/t muller's muscle)
 - Both muscles are supplied by sympathetic system
- In Horner's syndrome.
 - o Muller's muscle affected: ptosis (mild)
 - Inferior tarsal muscle affected: lower lid rises (inverse ptosis)
 - Appears to Enophthalmos (pseudo Enophthalmos)

Congenital

Acquired

Within first two years of life .

Ptosis

Ptosis

Miosis

. . .

. . .

Miosis

- Anhydrosis
- Anhidrosis
- Enophthalmos
- Enophthalmos
- Heterochromia (different coloured Iris)



- Mcc: Birth trauma
- Mcc: Pancost tumor

Causes of Horner's Syndrome

- Internal Carotid Artery dissection: painful Horner's syndrome
- Lateral Medullary syndrome (Wallenberg's Syndrome)

- Brainstem stroke
- Pancoast Tumour
- Cluster or migraine headaches
- Birth trauma
- Neuroblastoma



Pancoast tumor

Confirmatory tests

- 1. Cocaine test
 - Normal pupil dilates
 - Horner's pupil no dilatation
- 2. Apraclonidine test
 - Normal pupil no dilatation
 - Affected pupil dilates





Apraclonidine test for Horner's Syndrome

6. HUTCHINSON'S PUPILS



Occur after cerebral compression: Post trauma

3 Stages

- 1. Pupil on traumatised side constricts, opposite pupil normal
- 2. Trauma side pupil dilates, opposite side pupil constricts

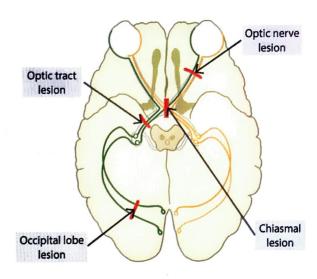


Hutchinson's pupil

3. Both side pupils fixed and dilated, not reacting to light: an ominous sign

7. WERNICKE'S HEMIANOPIC PUPIL

Seen in hemianopia caused by optic tract lesion



- Pupil reacts when seeing area of retina (nasal) stimulated, no reaction when blind (temporal) retinal side stimulated
- · Difficult to elicit due to diffusion of light



Important Information

- Wernicke's Hemianopic pupil are not seen in Wernicke's Syndrome
- Wernicke's Syndrome occurs due to deficiency of thiamine (vitamin BI) and is characterised with
 - o Ataxia
 - o Ophthalmoplegia
 - o Mental confusion



Previous Year's Questions

Q. In a patient of head injury who presents with headache and increased intracranial pressure. the. Effect on pupill is?

(FMGE June 2019)

A. Ipsilateral mydriasis

- B. Contralateral mydriasis
- C. Ipsilateral miosis
- D. contralateral miosis



Previous Year's Questions

Q. All of the following conditions cause miosis EXCEPT?

(FMGE June 2019)

- A. Bright light
- B. Horner's syndrome
- C. Oculomotor paralysis
- D. Iridocyclitis



Previous Year's Questions

Q. A slightly dilated pupil remains as it is, even in a dark room. What is the diagnosis?

(AIIMS JUNE 2020)

- A. Argyll Robertson pupil
- B. Holmes Adie 's pupil
- C. Blindeye
- D. Horner's syndrome

PAPILLOEDEMA





Papilledema

- Disc oedema with raised ICT
- Normal ICT: 50-180 mm of water
- In Papilloedema
 - o ICT in Adults: >250 mm of H₂O (20mm of Hg)
 - o ICT in Children: >200 mm of water
- Brain basics
 - Volume of average skull: 1500 ml of which 85% brain, 10% blood, 5% CSF
 - o M/c cause of ↑ ICT: Traumatic brain injuries
 - o Untreated ↑ ICT leads to
 - → Loss of vision
 - → Severe headache lasting for 48 hours
 - o ICT measurement
 - → Intraventricular catheter
 - → Lumbar puncture: In absence of an obstruction ICT measured corresponds closely to ventricular pressure

Causes of Papilledema

Traumatic brain injuries



Intracranial space occupying lesions (ICSOLS): 60 - 80% infratentorial



- Cerebral haemorrhage: Sub arachnoid, intra parenchymal
- Meningitis: cerebral edema
- Obstructive hydrocephalus
- Cerebral venous sinus thrombosis
- Idiopathic Intracranial Hypertension

Clinical features of papilledema

- Headache
 - Occipital headaches
 - Throbbing, pulsating
 - Changes with changing posture
 - Worsens with straining, coughing, sneezing, Valsalva manoeuvre
- Projectile vomiting: no prior nausea
- Amaurosis fugax: Transient visual obscuration (TVO)
- Pulsatile tinnitus
- Disc edema: earliest sign nasal blurring of disc margins
- 6th nerve palsy (false localizing sign)



Friesen classification of Papilledema

Investigations of papilledema

- MRI with contrast: CNS mass lesions
- MRV: cerebral venous sinus thrombosis

- Lumbar puncture: CSF opening pressure >250 mm of H2O, glucose, proteins, cell count
- · Perimetry: enlargement of blind spot
- Blood pressure to rule out malignant hypertension
- Fluorescein Angiography: leakage of dye
- No cause found, diagnosis of Idiopathic Intracranial Hypertension

Pseudopapilledema

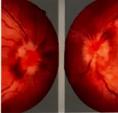
(1) 01:43:05

- Mimics papilledema
- Causes
 - Optic disc drusen
 - Hypoplastic discs
 - Congenital anomalies
 - High hypermetropia
- Spontaneous venous pulsations present
- Fluorescein Angiography
 - o Gold standard for diagnosis
 - o True papilledema shows leakage of dye



Papilloedema Vs Optic Neuritis









Paton's line

Papilledema	Optic Neuritis
• B/L	Usually U/L
Excessive disc oedema	Less disc oedema
Paton's lines o Circumferential lines around optic disc margin	Not seen as volume off disc edema is less
 Loss of venous pulsations 	 No Loss of venous pulsations
No Loss of vision	Sudden painful loss of vision

Important Question of Papilledema

No pain on ocular

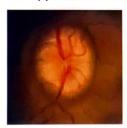
Colour vision normal

movements

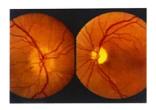
1. 'Champagne cork' appearance: chronic papilledema

Pain on ocular movements

Colour vision desaturation



- 2. Foster Kennedy Syndrome (FKS)
- Triad
 - o Ipsilateral optic atrophy
 - o contralateral papilledema
 - o Anosmia
- Cause of FKS is subfrontal meningioma
- 3. Pseudo Foster Kennedy syndrome
- Seen in bilateral sequential AION



Treatment of papilledema

- Primary cause to be addressed: resection of intracranial tumour
- CSF diversion procedures for VP shunts in hydrocephalus
- Anticoagulation for Dural venous thrombosis
- Weight loss and carbonic anhydrase inhibitors in IIH
- Optic nerve sheath fenestration where the vision threatened

IDIOPATHIC INTRA CRANIAL HYPERTENSION (IIH)



- Idiopathic disorder of young, fat, females of child bearing age
- Chronic elevation of ICT due to unknown causes which leads to Papilledema

Drugs Causing IIH

Vit A

- Tetracycline
- Oral contraceptive pills
- Amiodarone
- Lithium

Symptoms

- Headaches (m/c)
 - o Severe
 - Wakes patient from sleep (LP as last resort)
- Transient Visual Obscuration (TVO)
 - Temporary transient loss of vision
- Visual field loss
- Pulsatile tinnitus
- Diplopia

Diagnosis

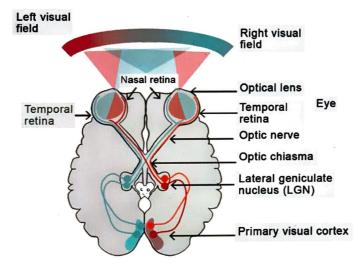
- Exclusion of all other causes of raised ICT
- CSF opening pressure > 250 mm H2O normal CSF

Treatment

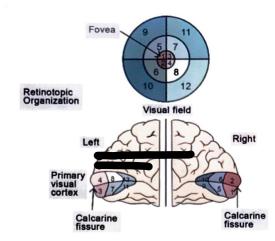
- Lose weight: Only disease modifying therapy
- DOC Acetazolamide, Topiramate may help
- Stop drugs causing IIH
- Serial lumbar punctures not recommended
- Optic nerve sheath fenestration
- Ventriculo-Peritoneal shunts for imminent loss of vision

VISUAL PATHWAY



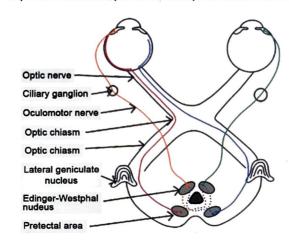


- Image is transduced within photo receptors (1st order neuron) and encoded within ganglion cells (3rd order neuron)
- Information from each retinal area is kept separate from information coming from other areas, each portion of visual cortex has corresponding region on retina is called as Retinotopic mapping

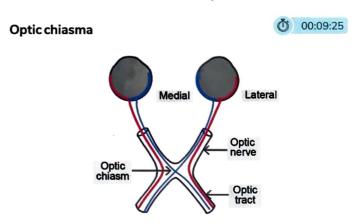


Retinotopic mapping

 ON - 90% fibres terminate in LGB, 10% project to superior colliculus, pretectum, suprachiasmatic nucleus



Visual Pathway



- X shaped space where 2.4 million axons cross
- 53% fibres cross over, 47% don't
- Enables stimulation of corresponding points in field → signals to same half of visual cortex. Seen in species with front facing eyes, allowing binocular vision with stereopsis

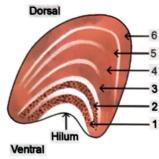
Functional anatomy of Visual Pathway

- Ö 00:12:46
- Almost 50% cortex process visual information
- 4 neurons in afferent visual pathway
- First order neuron-Photoreceptors
 - Second order
- Bipolar cells
- Third order
- Dipolar cells
- o Inira oraer
- Ganglion cells
- Fourth order
- Geniculocalcarine

Key points LGB and OR



- LGB is a 6 layered onion peel structure which is pyramid shape
- Magnocellular neurons: Sensitive to motion are present in 1, 2 layers



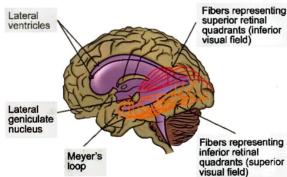
- Parvocellular neurons sensitive to colour and shape are present in 3,4,5,6 layers.
- From LGB Optic radiations come out & these radiations split into two fibres

Inferior fibres

Meyer's loop: through temporal lobe to occipital lobe

Superior fibres

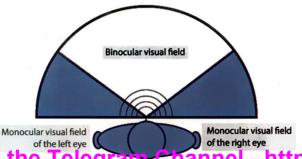
Baum's loop: Pass through parietal lobe to reach occipital lobe



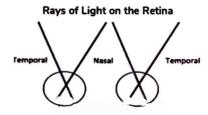
VISUAL FIELD INTERPRETATION

00:17:38

Binocular visual fields: 120 degrees



- Visual fields are opposite to retina
 - o Rays from Superior fields focus on inferior retina



o Rays from Nasal fields focus on temporal retina

PRE-CHIASMAL LESIONS

- Monocular field defects and ipsilateral
- **Ö** 00:19:43

- Reduced visual acuity
- Relative afferent pupillary defect

Examples of pre-chiasmal lesions

- Optic neuritis
- · Optic nerve gliomas
- Ischemic infarction of optic nerve

JUNCTIONAL SCOTOMA

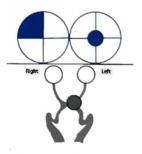


- Lesion at junction of optic nerve and chiasma
- Characteristic Ipsilateral central scotoma with contralateral superior temporal quadrantanopia



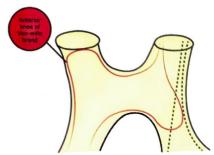
Important Information

 Unilateral optic nerve lesion involving Von-Willebrand's knee causes bilateral visual loss



Junctional Scotoma

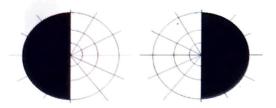
 Inferonasal fibres loop anteriorly for a short distance in the optic nerve before they go into the optic tract



OPTIC CHIASMA LESIONS



Bitemporal hemianopia



- Temporal crescents lost, central 110- 120 degrees remains, problems in driving, navigation
- · Hemi field slide phenomenon

Causes

- M/c Pituitary adenoma
- Craniopharyngioma
- Meningioma



Previous Year's Questions

Q.A tumour in the anterior pituitary causing pressure over optic chiasma will present as?

(FMGE June 2021)

- A. Homonymous hemianopia
- B. Bitemporal hemianopia
- C. Monocular vision loss
- D. Heteronymous hemianopia with central sparing

POST-CHIASMAL LESIONS



May be hemianopia, quadrantanopia or scotoma

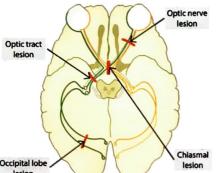
Characteristics

- Bilateral
- Homonymous
- Respect midline, do not cross over

OPTIC TRACT LESIONS

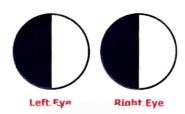


- 20 to 30mm in length, well vascularised
- Unusual for field defects to be caused by lesion
- Contralateral RAPD
- OT close to internal capsule, frequent hemisensory loss

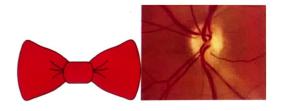


Characteristics

Contralateral homonymous hemianopia



- Abnormal pupillary reaction: Contralateral RAPD without loss of vision
- Wernicke's pupil
- Contralateral bow tie optic atrophy



Bow tie optic Atrophy



HOMONYMOUS HEMIANOPIA

 Contralateral retrochiasmal structures like optic tract, LGB, radiations visual cortex damaged

Symptom

Difficulty with reading and visual scanning

Causes

 M/c strokes, traumatic brain injury and tumours in children



Previous Year's Questions

Q. Lesion producing incongruous homonymous hemianopia with Wernicke's pupil?

(NEET Jan 2020)

- A. Optic radiations
- B. Optic nerve
- C. Optic tract
- D. Visual cortex



Previous Year's Questions

Q.30-year-old with complaints of diminishing vision on right halves of both eyes. What is the probable diagnosis? (NEET Sep 2021)

A. Left optic tract

- B. Right occipital lobe
- C. Optic chiasma
- D. Right optic nerve

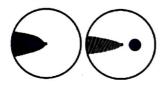
LGB LESIONS



LGB supplied by anterior and posterior choroidal arteries: unlikely to be wiped out by single large infarct

Characteristic

Wedge shaped defects: Sectoranopias



- "Keyhole" field defects
- Normal pupillary reflexes

POST LGB LESIONS

- Defects may show increasing congruity
- Normal pupil reactions
- No optic Atrophy

OPTIC RADIATIONS LESIONS



- · Radiations Project from Lateral Geniculate Nucleus to primary visual cortex V1
- 2 major bundles
 - o Meyer's loop
 - → Temporal lobe
 - → More susceptible to damage
 - o Baum's loop
 - → Parietal lobe
- Quadrantanopia seen in damage to radiation
- Homonymous superior quadrantanopia: Pie in the Sky



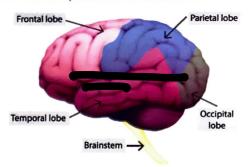
Pie in the Sky

Homonymous inferior quadrantanopia: Pie on the Floor

PIE IN THE SKY/ HOMONYMOUS SUPERIOR QUADRANTANOPIA



· Lesion of the optic radiations passing through the subcortical temporal lobe white matter



Seen in Ischemic stroke (m/c), resection of temporal lobe

Characteristics

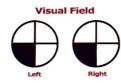
- Slow development: Compressive lesion
- Tumours produce sloping field defects, vascular sharp edges

PIE ON THE FLOOR/HOMONYMOUS © 00:47:25 INFERIOR QUADRANTANOPIA

- Optic radiations in sub cortical parietal lobe white matter
- Uncommon

VISUAL CORTEX

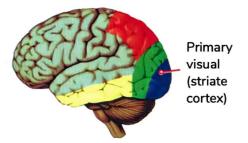






Parietal lobe

- Visual cortex is the largest continuous sector of brain's surface devoted to a single sensory function
- 50% cortex devoted to central 5% visual field
- 75% lesions are vascular



Human Visual Cortex

OCCIPITAL LOBE/VISUAL CORTEX 0 00:50:27 **LESIONS**



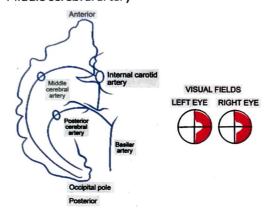
Characteristic

Congruity

Macular sparing (5 degrees around macula is spared)



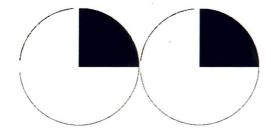
- Dual blood supply of the occipital pole (watershed zone)
 - Posterior cerebral artery
 - Middle cerebral artery



RULE OF CONGRUITY



Congruous: Identical and similar field defects



Incongruous: not identical and symmetrical



Rule of Congruity states

- Congruous
 - More posteriorly located lesions
 - Occipital lobe/Optic radiations
- Incongruous
 - More anteriorly located lesions
 - o Optic tract/LGB lesions

SUMMARY

- Optic disc/ nerve
- Same sided monocular blind
- Optic chiasma
- Bitemporal hemianopia
- Optic tract
- C/L homonymous hemianopia
- Lateral Geniculate Body
- Sectoranopia
- Keyhole field defect
- Optic radiations
- Meyer's loop: Pie in the sky
- Parietal lobe: Pie on the floor
- Occipital lobe
- Macular sparing



Previous Year's Questions

Q. Which lesion of the visual pathways produces a macular sparing field defect?

(FMGE June 2021)

- A. Optic chiasma
- B. occipital lobe
- C. Optic nerve
- D. Optic tract

GAZE CENTRES



 Centre Horizontal gaze: PPRF (Paramedian Pontine Reticular Formation)



Final common pathway: 6th nerve nucleus

Centre for vertical gaze

- Rostral interstitial nucleus of MLF
- Interstitial nucleus of Cajal
- Posterior commissure





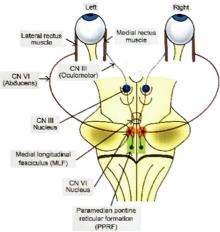
Important Information

- Vertical gaze centre: Midbrain
- Horizontal gaze centre: Pons
- Signal from PPRF passes to VI Cranial nerve
- VI CN innervates same sided lateral rectus muscle while some fibres pass through same sided MLF to stimulate opp. sided III CN
- III CN innervates Medial rectus muscle along with other EOM except LR and SO

HORIZONTAL GAZE PALSY



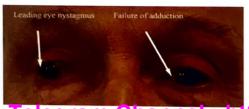
 Lesion is at PPRF causes same sided horizontal gaze palsy



Centre for Horizontal Gaze

INTERNUCLEAR OPHTHALMOPLEGIA (INO)

- Lesion is at Medial longitudinal fasciculus (MLF)
- Ipsilateral adduction deficit with contralateral abduction nystagmus



- Accommodative convergence retained
- Hallmark: impaired adduction
- Abducting nystagmus is due to compensatory response to overcome Medial rectus → Hering's law ↑ innervation to Lateral rectus

Causes

- Infarction
- Multiple Sclerosis

Unilateral INO

- Seen in elderly
- Caused by systemic vascular disease

Bilateral INO / WEBINO (Wall Eyed Bilateral INO)

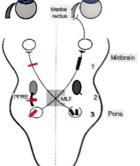
- Seen in younger patients
- Caused by Multiple Sclerosis



ONE AND A HALF SYNDROME



- Loss of all horizontal movements except contralateral abduction, which shows abduction nystagmus
- Lesion is due to combination of MLF with PPRF of the same side



EIGHT AND A HALF SYNDROMES

Ŏ 01:18:13

 7th nerve palsy with one and a half syndrome of the same side

Summary

Lesion	Palsy's
1. PPRF	Horizontal gaze palsy
2. MLF	INO
3. PPRF + MLF	1 ⅓ syndrome

4. PPRF + MLF + 7 nerve 8 ½ syndrome

Join the Telegram Channel - https://t.me/prepladderlatestnotes

OCULAR MYASTHENIA GRAVIS (OMG)

- 01:20:41
- M/C disorder affecting neuromuscular junction
- In OMG: skeletal muscles are affected, visceral muscles (pupil and ciliary muscles) are unaffected
- LPS is the first ocular muscle affected
- MR/SR are the extra ocular muscles affected

Pathology

· Antibodies to ACh receptors are formed this leads to destruction of ACh receptors

Symptoms

 Fluctuating ptosis and diplopia worsening in evening, variability in muscle function



Ptosis

Signs

- Lid retraction: one lid ptotic, other retracted
- Lid fatigue: sustained up gaze, ptosis worsens
- Cogan's lid twitch sign: looks down for 15 seconds, rapidly refixates to primary, leads to upward overshoot of eyelid then falls back to ptotic position



Cogan's lid twitch sign

- Tensilon test: positive test diagnoses MG
- Ice test: Resolution of ptosis after 2 minutes ice pack to evelid

Drugs worsening MG

- Lithium
- Propranolol
- Quinidine

Treatment

- Pyridostigmine (DOC)
- Systemic steroids

NYSTAGMUS



· Involuntary, rhythmic, oscillatory to and fro movement of

the eyes

Types

- Pendular: Phases of equal velocity
- Jerk: phases of unequal velocity
- Direction
 - o Direction of fast component
 - Pathological movement is the slow one
- Trajectory: Horizontal, vertical, rotatory
- Conjugate: both eyes same movement
- Null zone gaze in which intensity is minimal

Steady gaze Mechanisms



- 1. Fixation: detect retinal image drift and initiate corrective eye movements
- 2. Vestibulo-ocular reflex (VOR): eye movements compensate for head rotations ensuring clear vision during locomotion
- 3. Oculomotor Neural integrators: muscle activity to counteract pull of extraocular muscles

Classification of Nystagmus



- 1. Congenital/Infantile nystagmus: < 3 months of life
- Acquired: Adult ages, usually neurological cause
- 3. Physiological: Optokinetic vestibular, end point
- 4. Spasmus nutans
- Benian
- Triad
 - Head nodding
 - Torticollis
 - Nystagmus
- 5. Generally pathological nystagmus: diseases affecting vestibular system, brainstem, cerebellum, less commonly anterior visual pathways

Symptoms of Nystagmus



01:41:49

- 1. Blurred vision: retinal image slip > 5 degrees per second degrade vision
- 2. Oscillopsia: illusion that the stationary world is moving
- 3. Vertigo, dizziness, loss of balance (vestibular)
- 4. M/ c form of nystagmus: Gaze evoked nystagmus (only when eyes moved into eccentric gaze)
- 5. Drugs causing nystagmus
- Benzodiazepines
- Barbiturates
- Phenytoin

Congenital / Infantile nystagmus



- Congenital: at birth, or appear later shortly
- Due to ocular defects that are congenital/acquired in first month of life
- Usually horizontal, pendular or jerk
- Abolished in sleep

- Uniplanar: plane remains unchanged in all gazes
- Not suppressed by fixation

Defects causing congenital nystagmus

- Congenital cataract
- Albinism
- Optic nerve hypoplasia

Acquired Nystagmus

Ö 01:45:23

- Definition
 - o After 6 months of age
- Symptoms
 - Blurred vision
 - o oscillopsia
- Causes
 - MS
 - Strokes
 - Tumours
 - Trauma
 - o Drug
- Neurological causes
 - Associated signs nausea
 - Vomiting
 - Tinnitus
 - Vertigo

Latent Nystagmus



- · Latent: seen only when one eye is covered
- Reversal of nystagmus direction on alternate fixation of eyes is defining characteristic
- Poor vision with nystagmus eye, improves when both eyes open

Downbeat Nystagmus



- Downbeat Fast phase down, while eyes in primary position of rest
- Pathology in Cervico medullary junction
- Causes
 - Arnold Chiari malformation
 - o Platybasia

Upbeat Nystagmus

- Fast phase up, eyes in primary position of rest,
- Pathology in Vermis
- Causes
 - Cerebellar degeneration.
 - o MS
 - o Brainstem

Refer Image 11.1

Physiological nystagmus



- Some physiological nystagmus used every day
- Optokinetic, caloric, end gaze
- Typically, low amplitude, not sustained, symmetric, horizontel

Caloric Nystagmus



- Is a type of VOR elicited by stimulating horizontal semicircular canal with warm or cold water in ear canal to create convection currents in endolymph?
- Cold water produces horizontal nystagmus with fast phase AWAY from tested ear
- Used to differentiate central vs peripheral vestibular lesions, absent response means peripheral vestibular dysfunction

Refer Image 11.2



How to remember

COWS: Cold Opposite Warm Same Side

Optokinetic Nystagmus (OKN)



Image stabilization when viewing a constantly moving visual field



- Initial smooth pursuit followed by saccade (rapid)
- Asymmetric OKN is abnormal, greater OKN when target is moving in one direction compared to opposite
- Cogan's dictum
 - Homonymous hemianopia + asymmetric OKN-Parietal lobe lesion, probably mass lesion
 - Homonymous hemianopia + symmetric OKN-Occipital lobe lesion, vascular lesion



Previous Year's Questions

Q.Slow eye movement in the direction of a moving object and a rapid return of eye position in the opposite direction is known as?

A. End gaze nystagmus B. vestibular nystagmus

(JIPMER May 2019)

C. Optokinetic nystagmus

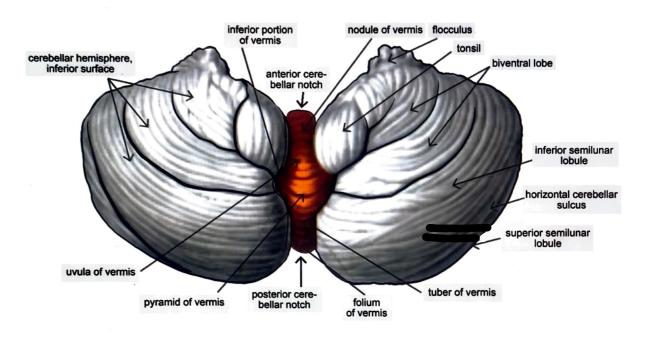
D. Bell's phenomena

Treatment of nystagmus



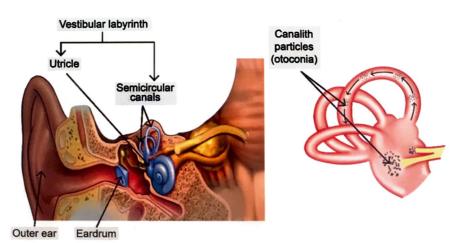
- Baclofen (DOC) for Periodic Alternate Nystagmus (PAN)
 - PAN is a spontaneous horizontal nystagmus reverses direction every 2 minutes
- Botulinum toxin: may cause diplopia and ptosis
- Chlorzoxazone for Downbeat nystagmus
- Gabapentin, Memantine, clonazepam, carbamazepine
- Optical: Glasses, contact lenses, prisms
- Surgical Rx: Anderson Kestenbaum

Image 11.1



Upbeat Nystagmus: Pathology in Vermis

Image 11.2







Q. A 35-year-old female presented with a sudden loss of vision of the right eye for 3 daysassociated with pain in eye movement. She had a history of being diagnosed with acute disseminated encephalomyelitis and treated with oral prednisolone. Ocular examination revealed visual acuity of the right eye was counting finger with a positive afferent pupillary defect. The ophthalmologist suspected optic neuritis. Which of the following is not necessary for investigating optic neuritis?

A. MRI head and orbit

B. ESR

C. USG B scan

D. Visual Fields

Answer: C

Solution

USG B scan is not useful for evaluating optic neuritis.

- MRI head and orbit is needed for evaluation of optic neuritis to rule out MS.
- ESR- †sed in infectious causes of optic neuritis.
- · Visual fields are used for follow up and extent of vision loss.
- OCT (Optical coherence tomography) used to see amount of optic nerve edema.

Reference: Comprehensive ophthalmology, AKKhurana 6th edition Pg 317

- Q. A 30-year-old lady presents with sudden severe bilateral loss of vision more so on the right side with no perception of light.

 The rest of the examination including pupillary reflex, fundus and optokinetic nystagmus is normal. She was able to touch the tips of her finger with the right eye closed but not with the left eye closed. The most likely diagnosis is?
- A. Optic neuritis
- B. Anterior ischemic optic neuropathy
- C. CMV retinitis
- D. Functional visual loss

Answer: D

Solution

The above symptoms are more in favour of functional visual loss.

Options A and B: Ruled out because pupillary reactions will not be normal in optic neuritis and anterior ischemic neuropathy and fundus shows blurred disc margins

Option C: In CMV retinitis, fundus shows sauce and cheese retinopathy

Reference: Comprehensive ophthalmology, AKKhurana 6th edition Pg 317

- Q. A 33-year-old male patient presented to your hospital with complaints of diplopia. On examination, pupils were noted to be dilated. Both direct and consensual light reflexes were lost. The probable diagnosis is:
- A. 2nd nerve palsy
- B. 3rd nerve palsy

C. 5th nerve palsy

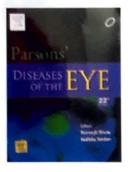
D. 7th nerve palsy

Answer: B

Solution

- PUPILLARY LIGHT REFLEX:
 - If light enters an eye, the pupil of the same eye contracts (direct light reflex), activity shared equally by the pupil of the other eyes (consensual light reflex)
- Afferent limb of light reflex is the Optic nerve (CN2) and the efferent limb is the Oculomotor nerve (CN3)
- Both direct and consensual light reflexes are lost in third nerve palsy.

Reference:



- Q. A 36-year-old female patient complains of recurrent episodes of diminution of vision in both eyes. She was treated with steroids after which her symptoms improved. On examination, vision in RE -6/60 and LE 6/18 and colour vision is defective in both eyes. She also develops spastic paraplegia. What is the diagnosis?
- A. Multiple sclerosis
- B. Syringomyelia
- C. Carotid artery dissection
- D. Neuromyelitis optical

Answer: D

Solution

Neuromyelitis Optica - it is characterized by a recurrent attack of bilateral optic neuritis and the subsequent development of transverse myelitis within days or weeks.

Drugs used in acute attacks: high dose glucocorticoids or plasma exchange

Note:

Early in the course of the disease, it may be difficult to distinguish neuromyelitis optica and multiple sclerosis because both may cause optic neuritis and myelitis as symptoms. However, optic neuritis and myelitis tend to be more severe in Neuromyelitis Optica. The brain MRI is more commonly normal, and the spinal fluid analysis does not usually show oligoclonal bands in Neuromyelitis Optica, which are features that help distinguish it from MS.

Reference: AK khuran 7th edition pg369



LEARNING OBJECTIVES

SQUINT and Strabismus

- Anatomy & Physiology of Extraocular Muscles
- Laws of Ocular Motility
- Types of Squint
 - o Tropia: Manifest Squint (Visible)
 - → Esotropia and Exotropia
 - o Phoria: Latent squint (Not visible)
- Classification and Measurement of Tropias
- Test for Phorias
- Tests for Restriction v/s Paralysis
- Exotropia
- 3rd Nerve palsy
- 4th Nerve palsy
- 6th Cranial Nerve Palsy



12

SQUINT/STRABISMUS

- Ideally two eyes of humans must be parallel
- But when they are not parallel

It is called as squint



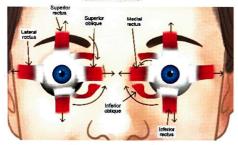
ANATOMY & PHYSIOLOGY OF EXTRAOCULAR MUSCLES



7 extra ocular muscles [EOM]

- 1 muscle belong to eyelid
 - Levator palpebrae superioris [LPS]
- 6 muscles belong to eyeball

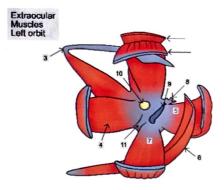
4 Recti	2 Oblique
 Superior rectus [SR] Inferior rectus [IR] Medial rectus [MR] Lateral rectus [LR] 	Superior oblique [SO] Inferior oblique [IO]
Superior rectu (upward moveme	Superior oblique (downwerd and outwerd movement)
Lateral rectus (outward incoment)	Medial rectus



Extra Ocular Muscles

Movements of eyeball

- 1. Elevation moving up
- 2. Depression moving down
- 3. Abduction moving outwards
- 4. Adduction moving inwards
- 5. Intorsion —rotating inwards
- 6. Extorsion rotating outwards



EOM & movement of eyeball



Actions of Muscles

- Medial rectus: adduction
- · Lateral rectus: abduction



How to remember

SIN RAD

- Superior muscles cause intorsion
- Inferior muscles cause extorsion
- Rectus muscles cause adduction
- Oblique muscles cause abduction

Superior rectus	Inferior Rectus	Superior oblique	Inferior oblique
Intorsion	Extorsion	Intorsion	Extortion
Adduction	Adduction	Abduction	Abduction
Elevation	Depression	Depression	Elevation



Previous Year's Questions

Q. Levator palpebrae superioris is supplied by?

(JIPMER DEC 2019)

- A. 2nd cranial nerve
- B. 3rd CN
- C. 4th CN
- D. 6th CN

9 GAZES	00:14:33
• 1 Primary	• Straight
4 Secondary	UpDownLeftRight
• 4 Tertiary	Up & rightUp & leftDown & rightDown & left
3	E



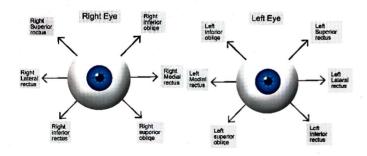
9 GAZES

PRIMARY ACTIONS

Muscle	Primary action
Superior rectus	Elevation
Inferior rectus	Depression
Superior oblique	Intorsion
Inferior oblique	Extorsion

Rule

- In abduction Recti work best
- In adduction oblique's work best



Actions of Muscles

In abducted gaze (abduction)	In adducted gaze (adduction)
Elevation → SR Best elevator in abduction	Elevation → IO Best elevator in adduction
Depression → IR Best depressor in abduction	Depression → SO Best depressor in adduction

TERMINOLOGY

(1) 00:23:43

Ductions

- Used for monocular movements
- Movements involving one eye only

Versions

- o Both eyes in same direction
- o Both versions and vergence are binocular movements

Vergence

 Both eyes in opposite direction (e.g., Divergence, Convergence)

Agonist

 Primary muscle which moves eye in a given direction e.g., Abduction of Right eye → Right LR is the agonist for this movement

Synergist

 Muscle of the same eye which helps the agonist in pushing the eye in the same direction e.g., Right LR (abductor)

↓ Synergists

Right IO, Right SO (abductors)

Antagonist

 Muscle of the same eye which pulls the eye in the opposite direction to that taken by the agonists e.g. Right LR (abduction)

J Antagonists

Right MR (adduction)

Yoke muscles

 Pair of muscles in opposite eyes pulling the eyes in the same direction

e.g. Right LR (abduction)

↓Yoke muscles

Left MR

TWO LAWS OF OCULAR MOTILITY © 00:28:50

- Herring's law of equal innervations (Involves both eyes)
 - Equal and simultaneous innervations flow through paired yoke muscles of each eye during any conjugate eye movement.
 - Conjugate means both eyes directed to same direction

- Sherrington's law of reciprocal innervation (Involves one eye)
 - Increased innervation to a muscle is accompanied by a ↓sed innervation to its antagonists
 - Example
 - →Right eye Abduction

Right LR contracts (i.e., †sed innervations to agonists) and

Right MR relaxes (Lsed innervations to its antagonist)

TYPES OF SQUINT

Ö 00:31:56

- 1. Tropia: Manifest squint (Visible)
- 2. Phoria: Latent squint (Not visible)





Tropia

Phoria



Important Information

- Fusion mechanisms of the eye prevents latent squint from becoming manifest
- Disruption of fusion mechanisms converts latent squint to manifest squint
- Mostly all humans have phorias but are asymptomatic

SUBTYPES OF TROPIAS

1. Exotropia: one or both eyes are diverted outwards



Right Exotropia

2. Esotropia: one or both eyes are inwards



3. Hypertropia: one eye is above the other eye



Right Hypertropia

4. Hypotropia: one eye is below the other eye



Right Hypotropia

SUBTYPES OF PHORIAS

- 1. Exophoria
- 2. Esophoria
- no right & left
- 3. Hyperphoria
- demarcation
- 4. Hypophoria

MEASUREMENT OF TROPIAS



Hirschberg test

- Flash a torch in the center of the patient's forehead (glabella), ask the patient to look at the torch light
- Reflection of light called as corneal reflex is seen on the center of the cornea
 - Orthophoric: reflection of light is exactly at the center of the pupil
 - Heterophoric: reflection of light is not at the center of pupil



Orthophoric

- Calculation of squint
 - Measure the distance between center of pupil & where reflection is present
 - Each 1 mm decentration = 7 ° Squint = 14 prism diopters (as 1° = 2 prism diopters)
 - o Reflex at pupil margin = 15 degrees
 - Reflex at limbus = 45 degrees



Calculation of squint



Understand with an example

Q. Left eye decentration of 3 mm to temporal Ans. 3 X7 = 21º

21 X 2 = 42 prism Diopter * 42 prism Diopter ESOTROPIA

reflection is outwards as eye is moved inwards





Important Information

Squint is always opposite to the direction of reflection

- Exotropia: Reflection of light is inwards
- Esotropia: Reflection of light is outwards
- Hypertropia: Reflection of light is downwards
- Hypotropia: Reflection of light is upwards

TEST FOR PHORIAS



The 3 cover tests

- 1. Cover Test
- · Detects manifest squint
- One eye is covered for 2 seconds

Watch for movement in the uncovered eye

- If there is movement detected → squint in the eye
- If there is no movement detected → No squint in the eye
- Most widely used test



Cover Test

2. Coveruncovertest

- Detects latent squint
- Cover & uncover one eye & look for movement in the same eye
- Resting eye (under the cover) always goes to its normal position
- Remove the cover and ask patient to see the light again



- o In Exophoria: Eye ball moves from out to In
- o In Esophoria: Eye ball moves from in to out
- o In Orthophoria: No movement of eyeball



Esophoria

3. Alternate cover tests



- It detects total squint (i.e., latent squint & manifest squint) but does not measure exact amount of each squint
- Alternately occlude each eye, holding occluder for several seconds to suspend fusion, looking for movementineacheye.

[For measuring exact amount of both squints we use prismbar]

4. Simultaneous Prism Cover Test





Simultaneous:

- Prism over deviating (squinting) eye
- Cover over fixating (normal) eye
- Measures total amount of tropia only (Unlike alternate cover test)

Maddox rod test

- **Ö** 01:00:18
- Maddox rod detects phorias at distance
- It is Series of fused glass cylindrical rods
- Put over one eye (rods can be oriented vertically/ horizontally)
- The rods convert white spot of light (which the patient is asked to look at/into a red streak
- This red streak is at 90° to the orientation of rods



Maddox rod test

Suppose Rods are placed vertically over the Right eye Patient is asked to look at the white spot of light with normal Left eye Rods (placed vertically) convert the white spot of light into the horizontal red streak With Right Eye With Left eye Horizontal red beam of White spot of light is seen

- In orthophoric: Horizontal red beam & white spot of light must lie on the same line
- In phoria: White spot of light is either above or below the red beam of light

Maddox wing test

Testing phorias for NEAR

light is seen



Bifocals

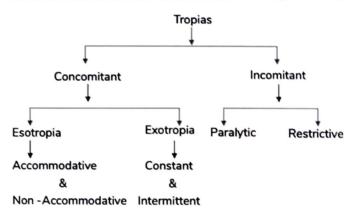
Treatment of Phorias

O 01:04:07

- Optical correction
 - Glasses
 - Bifocals
- Miotics
 - In esophoria due to †AC/ A ratio
 - o AC/A ratio
 - → Accommodative conversion/accommodation
 - → Normally AC/A ratio = 3-5 Prism dioptres
 - → Miotic DOC for children's with ↑AC /A ratio is Echothiophate
- For Asthenopic symptoms Prisms are prescribed
 - o Exophoria: Base in
 - Esophoria: Base out
- Orthoptic exercises convergence exercises

CLASSIFICATION OF TROPIAS





CONCOMITANT SQUINT



Angle between two eyes remains same in ALL 9 gazes

Features

- Usually, congenital
- No diplopia
- Both eyes have FULL movement
- Primary deviation = secondary deviation

Causes

- Uncorrected refractive error
 - Suppression and amblyopia may develop



Concomitant squint

INCOMITANT SQUINT





Incomitant squint

Angle changes in every gaze

Features

- Usually acquired
- Secondary deviation > Primary deviation

Symptoms

- Diplopia
- Abnormal head posture
- Vertigo
- Disorientation

Two types

- Paralytic (Neurogenic)
- Restrictive (Mechanical)

PRIMARY VS SECONDARY DEVIATION

- Primary: Angle between the eyes with normal eye fixing
- Secondary: Angle between the eyes with squinting eye fixing



Important Information

- Secondary > primary seen in incomitant squint
- Primary = Secondary seen in comitant squint



Primary Vs Secondary Deviation

PARALYTIC SQUINT

- Paralysis of cranial nerves (3,4,6)
- Paralysis of neuromuscular junction leading to myasthenia gravis



Paralytic Squint

RESTRICTIVE SQUINT

- Restriction of muscle movement due to fibrosis or scaring
- Seen in Thyroid Related Ophthalmopathy (TRO) / Thyroid Eye disease (TED)
- 1st muscle to get restricted in TED is inferior rectus

Other causes of restrictive squint

- Orbital fractures
- Duane's syndrome



Restrictive squint

Order of muscle Restriction

- I Inferior Rectus
- M Medial Rectus
- So Superior Rectus
- Lucky Lateral Rectus



How to remember

IM So Lucky

PARALYTIC SQUINT VS RESTRICTIVE SQUINT

Case study

Possibilities

- Right Lateral Rectus palsy (paralytic Squint)
- Right medial Rectus Restriction (Restrictive Squint)

TESTS FOR RESTRICTION V/s PARALYSIS



- 1. Forced duction tests
- 2. Forced generation test
- 3. † IOP as eye rotates against restriction
- 4. Saccadic velocity generation normal in restricted

Forced duction test (FDT)

- 1. Local anaesthetic drops used to paralyse the sensations
- 2. With 2 forceps, superiorly & inferiorly examiner pull &







Forced duction test (FDT)

In paralytic squint

- Full passive movement (Eyeball can be moved)
- Reduced saccadic velocity
- IOP constant

In Restrictive squint

- In FDT eye cannot be rotated into position of limitation
- †IOP as eye attempts to move in direction of limitation
- Normal saccadic eye movements: 'Dog on a leash movement'



Forced duction test (FDT)

SYMPTOMS OF INCOMITANT SQUINT

00:23:44

- Diplopia
- Abnormal head posture
- Vertigo
- Disorientation

Diplopia

- · Diplopia is greatest in the field of action of the underacting muscle
- Plane of diplopia

Horizontal	Vertical	Oblique
1 1	1	
 One of the horizontal muscles paralysed 	 One of the vertical muscles paralysed 	 One of the oblique muscles paralysed



E.g., SR, IR

E.g., SO, 10

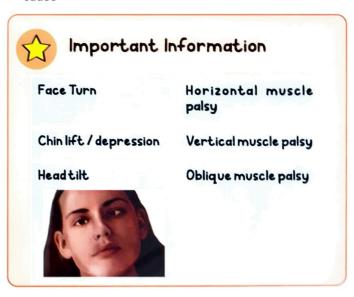


Diplopia worsens in near vision means medial rectus is affected

Monocular Diplopia	Binocular Diplopia
Seen by one eye	 Seen by two eyes together
Causes Astigmatism Keratoconus Cataract Subluxated lens Dry Eyes	Causes Squint Myasthenia gravis Intranuclear ophthalmoplegia

Abnormal Head Posture (AHP)

- Compensatory posture to reduce diplopia
- Head is deviated out of normal straight head position
- If AHP persists with eye closed, then it is not a visual cause



SYMPTOMS OF CONCOMITANT **SQUINT**

00:35:19

- Esotropia
 - Accommodative

- Non accommodative
- Exotropia
 - Constant
 - Intermittent

ACCOMMODATIVE ESOTROPIA

- Esotropia due to accommodation problems
- Presents between 1 and 5 years age

Two types

- 1. Refractive
- 2. Non refractive

Refractive Accommodative Esotropia

Pathology

Uncorrected hypermetropic refractive error

Leads to blurred retinal image

Stimulate accommodation

Accommodative convergence

Accommodative esotropia

- Uncorrected hypermetropia average 5.0 D
- Normal AC ratio
- Intermittent at first, then constant
- Often preceded by illness or trauma
- Average Angle of deviation around 20-40 PD



Uncorrected Hypermetropia

- Treatment
 - Full hyperopic correction based on cycloplegic refraction



After Correction



Previous Year's Questions

Q. A man with convergent squint in one eye has vision has 6/60, and another eye has no squint with vision 6/60. What is the next step?

(FMGE Dec 2019)

A. MRI

B. Squint surgery

C. Botulinum toxin

D. Refraction

Non-Refractive Accommodative Esotropia

Esotropia for near: high AC/A ratio





Near: Squint

Distant: No squint

- Usually no refractive error
- Associated hypermetropia has to be corrected
- Treatment
 - Bifocal executives' glasses



- o In small children give Miotics, e.g., Echothiophate
- Surgery: Bilateral MR recession



Previous Year's Questions

Q. Miotics are useful in which type of squint?

(JIPMER NOV 2018)

- A. Paralytic squint
- B. Accommodative squint
- C. Divergent squint
- D. Congenital squintn ointment after sometime

NON-ACCOMMODATIVE ESOTROPIA

Esotropia not due to accommodation problems

Two types

- 1. Infantile esotropia (m/c)
 - < 6 months of age</p>
- 2. Acquired
 - · Develops after 6 months of age

Causes

- Unknown etiology
- Low hyperopia < 3 D
- Often associated with neurologic / néoplastic disorder

Diagnosis

If Residual esotropia > 10 PD persists for > 4 weeks



Treatment

Surgery: Recession / resection

EXOTROPIA





Intermittent Exotropia

Two types

- 1. Constant Exotropia (Infantile)
- Large angle exotropia
- Presents at 2-6 months age
- Surgical treatment
- 2. Intermittent Exotropia (m/c)
- Presents in childhood
- May vary throughout the day
- Often causes patient to close either eye in bright light
- Difficulty in eye contact and social interactions

Causes

- Uncorrected myopia
- Divergence

Treatment

- Correct myopia by over minussing which leads to convergence
- Surgery

SQUINT SURGERY



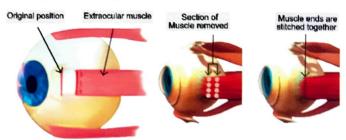


Principle of Squint surgery

- One set of Surgery: makes muscles strong
- Other set of Surgery: Weakens the muscle

Example

- Esotropia in Right eye
 - Right LR → Made stronger (eye drifts out)
 - o Right MR → Made weaker (eye drifts out)
- Exotropia in Right Eye
 - Right Medial Rectus → Made stronger (Eye drifts in)
 - Right lateral Rectus → Made weaker (eye drifts in)
- · Surgery to weaken the muscle: Recession
- Surgery to strength the muscle: Resection



Recession

Resection

INCOMITANT SQUIT

- Paralytic (Neurogenic) squint
 - Nerve Palsy
 - → 3rd Nerve Palsy
 - → 4th nerve palsy
 - → 6th Cranial Nerve Palsy
 - NMJ disorders
- Restrictive (Mechanical) squint

3RD NERVE PALSY



Two components

- Outer parasympathetic fibres
 - Sphincter pupillae
 - Ciliary muscle
- Inner Somatic
 - o LPS
 - o Four extraocular muscle

Causes

- M/c Micro vascular ischemia due to DM, Hypertension
- Trauma

- Intracranial neoplasm
- Haemorrhage
- Aneurysms at junction of PCA/ICA



Important Information

- 3rdNerve palsy is the first sign/last sign of aneurysm
- In Children
 - Congenital
 - Traumatic forceps delivery



Down and out eye, Ptosis

Presentation

- Down & Out eye, ptosis
- Fluctuating Diplopia
- The typical feature of 3rd nerve palsy

Patient complains of diplopia (Fluctuating diplopia)

To diagnose 3rd nerve palsy: check whether pupil is dilated or normal

When pupil is normal

When pupil dilated

K/a pupil sparing 3rd nerve Pupil involving 3rd nerve

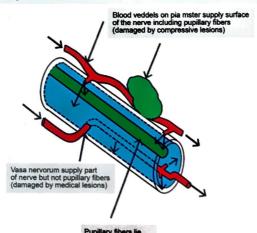
(medical 3rd nerve palsy)

palsy (surgical 3rd nerve palsy

M/c cause

M/c cause of 3rd nerve palsy with pupil sparing is

- Diabetes mellitus
- **Tumours**
- **Blood pressure**
- **Aneurysms**



4. Syndromes of 3rd nerve Palsy

- 1. Benedikt
 - Ipsilateral 3rd Nerve palsy +C/L tremors
- 2. Weber's
 - Ipsilateral 3rd Nerve palsy+ C/L hemiplegia
- 3. Nothnagel
 - Ipsilateral 3rd Nerve palsy + cerebellar ataxia
- 4. Claude
 - Benedikt + Nothnagel

4[™] NERVE PALSY



Important points about 4th cranial nerve

- Longest intracranial nerve
- Thinnest intracranial nerve
- First nerve to be damaged in closed head injury
- Only cranial nerve to cross over
- · Only cranial nerve to come out of dorsal surface of brainstem

Causes

- M/c cause of isolated 4th nerve palsy is Congenital
- M/c cause of acquired 4th nerve palsy is Trauma > Micro vascular ischemia (due to DM, HTN)

Symptoms

· Acute onset vertical diplopia on looking downwards and inwards which worsens on reading and walking downstairs

On examination

- Head tilt on opposite shoulder
- Eye position upward worsens on adduction



Diagnosis

Parks Bielschowsky 3 step test

6[™] CRANIAL NERVE PALSY



- M/C isolated ocular cranial nerve palsy
- Longest subarachnoid course

Causes

- In Children
 - Pontine gliomas
 - o Trauma
- In Adults

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- Microvascular ischemia (due to Diabetes, Hypertension)
- o In ↑ ICT 6th cranial nerve is most vulnerable to damage

Presentation

- Bilateral horizontal diplopia which ↑ on gaze towards affected side
- Esotropia with face turn to affected side



Esotropia

Syndromes of 6th Nerve palsy

- Raymond's
 - Ipsilateral 6th nerve palsy +c/L hemiparesis
- Millard Gubler
 - o Ipsilateral 6th +7th nerve palsy + c/L hemi paresis
- Foville
 - Ipsilateral 5th, 6th, 7th, 8th nerve palsy+ Horner's syndrome
- Gradenigo's
 - o 6th nerve palsy + otitis media + facial pain

PARKS BIELCHOWSKY'S THREE STEP



- Diagnostic test for identifying underacting muscle in hypertropia
- M/c cause of hypertropia is superior oblique palsy

3 Cardinal questions

1. Which eye is hypertropic in primary gaze?

	Under acting Muscles
Right eye	Rt Inferior rectusRt Superior oblique
Left eye	Left Superior rectusLeft Inferior oblique

2. Does the hypertropia get worse in Right or Left gaze?

	Under acting Muscles
Right eye	Rt Superior oblique
Left eye	Left Superior rectus

3. Does the hypertropia get worse in Right or Left head tilt?

Right Eye

Superior oblique



?

Previous Year's Questions

Q. A kid comes in with left sided head tilt. when doctor corrects that, he notices right hypotropia which increases on dextroversion and right tilt head which muscle is paralyzed?

(AIIMS JUNE 2020)

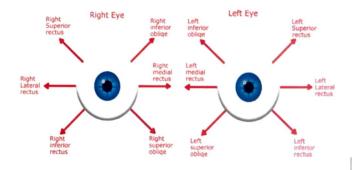
A. Right superior oblique

B. Right superior rectus

C. Left inferior oblique

D. Left superior oblique

Muscles Action



	Best Elevator	Best depressor
Abducted gaze	superior rectus	Inferior rectus
Adducted gaze	Inferior oblique	Superior oblique



CLINICAL QUESTIONS



- Q. An 18-year-old male football player was struck on the head just above his right ear by an opponent's elbow during a game Initially, the athlete was normal but later complained of blurry vision. He was able to see clearly through, each individual eye with the other eye closed but experienced a type of double vision with both eyes open. Cranial nerve screening revealed 4th nerve palsy. All of the following can be seen in this patient, except:
 - A. Diplopia on downward and inward gaze
 - B. Right Head tilt
 - C. Extorsion
 - D. Hypertropia

Answer: B

Solution

Actions of Superior Oblique include Intorsion, Depression, Abduction which would be lost in the case of Superior Oblique Palsy.

Patients complain of diplopia – vertical, diagonal, or torsional. If the principal complaint is torsion - B\L palsy should be suspected

RIGHT SUPERIOR OBLIQUE PALSY





- Now in Fig a, you can see that on the right head tilt there is elevation and slight esotropia, which is not seen on the left head tilt. So the patient would like to have a left head tilt. So remember mnemonic BOOT- Better on opposite tilt
- Similarly it would be worse on the opposite gaze, which means more diplopia in levoversion. So remember mnemonic-WOOG

EXTRA EDGE:

• Brown syndrome/ superior oblique tendon sheath syndrome is caused by malfunction of superior oblique muscle, causing eye to have difficulty moving up, particularly during adduction (when eye turns towards the nose).

Reference: Clinical Ophthalmology A systematic approach 7th edition: Kanski & Bowling Pg 834-835

Q. A 2 month-old girl baby was referred for evaluation of esotropia. Her parents noted that the baby began crossing her eyes shortly after birth and feel that it is worsening. They also noted that the crossing worsens when she is tired and alternates between the right eye and the left eye. She was diagnosed with a case of infantile essential esotropia. All are features of this condition, except:

- A. Amblyopia
- B. Large angle squint
- C. Surgery should be done as soon as possible
- D. Low Accommodative convergence / Accommodation ratio

Answer: D

Esotropia



Solution

Infantile Essential Esotropia (Congenital Esotropia)

- · Onset usually after birth
- Amblyopia
- · Large angle squint
- From Birth
- Surgery should be done as soon as possible
- High Accommodative Convergence / Accommodation ratio
- DVD is a common association.

Reference: Comprehensive ophthalmology A K Khurana 6th Edition Pg 348



LEARNING OBJECTIVES

CONJUNCTIVA

- STRUCTURE OF TEAR FILM
- KERATOCONJUNCTIVITIS SICCA
- EYE SIGNS OF VITAMIN A DEFICIENCY (VAD)
- PINGUECULA
- PHLYCTEN
- PTERYGIUM
- FOLLICLES
- PAPILLAE
- CONJUNCTIVITIS
 - o BACTERIAL CONJUNCTIVITIS
 - EPIDEMIC KERATOCONJUNCTIVITIS
 - ACUTE HAEMORRHAGIC CONJUNCTIVITIS
 - ANGULAR CONJUNCTIVITIS
 - OPHTHALMIA NEONATORUM / NEONATAL CONJUNCTIVITIS
- TRACHOMA
- VISION 2020
- VERNAL CATARRH/ SPRING CATARRH / VERNALKERATOCONJUNCTIVITIS



13 MALNUTRITION

CONJUNCTIVA

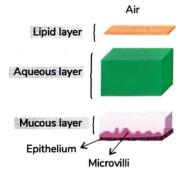
- **(5)** 00:01:09
- It is a Semi-transparent membrane which runs on the posterior surface of eyelids and reflects back on the eyeball
- Functions
 - o Lubrication and protection of eye
 - Prevents microbial entry has a role in immune surveillance



Conjunctiva

TEAR FILM STRUCTURE

(1) 00:03:33



Tear Film Structure

Precorneal tear film-functions

- 00:04:29
- · Lubricates surface, facilitating eyelid movements
- Smooth refracting surface
- Provides cornea with nutrients and oxygen
- Enzymes and antibodies to destroy bacteria
- Removes waste products from cornea

The 3 layers of tear film

- le 3 layers of tear film
- Lipid layer
 Formed from trials
- Formed from triglycerides, free fatty acids, cholesterol esters

- Secreted by Meibomian glands
- Function
 - o Prevent evaporation of tear
- 2. Aqueous layer
- Formed by 98% H₂O, O₂, CO₂, electrolytes, glucose, urea, PG, thyroid hormone, lysozyme
- Function
 - Provide nutrition



Important Information

Two types of tears

- A. Basal tears: secreted by accessory lacrimal
- B. Reflex tears: secreted by Main lacrimal glands

3. Mucous layer

- · Formed by MPS, glycoprotein, sialic acid
- Secreted by goblet cells, conjunctival epithelial
- Function
 - ⊥ surface tension
 - stabilise the tear film



Normal Caorne



Dry Eyes/ Keratoconjunctivitis sicca

CAUSES DRY EYE SYNDROME / KERATOCONJUNCTIVITIS SICCA



- Age
- Vitamin A deficiency
- Meibomian gland dysfunction (MGD)
- Sjogren's Syndrome / Rheumatoid A/Thyroid Eye
- Prolonged contact lens wear
- Post refractive surgery
- Prolonged computer usage
- Drug induced
 - Anti-cholinergic
 - o Anti-histaminic

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00:05:54



Important Information

Sjogren's syndrome consists of classic triad

- 1. Dryeyes
- 2. Drymouth
- 3. Rheumatoid arthritis

Signs & Symptoms of Dry Eyes



- Scratchy, FB sensation
- Burning/Stinging/Itching
- Frequent blinking
- Redness



Dry Eye

- Blurring of vision
- Ocular fatigue / tired eyes / Pressure / pain on eyes
- Paradoxically in some cases Wet eyes and tears running into cheeks
- Evening worsening



Previous Year's Questions

Q. A patient presenting with diminishing vision in dim light with dry eyes and roughening of corneal surface. Which of the deficiency can be associated with it? (NEET SEP 2021)

A. Iron

B. Protein

C. Retinoic acid

D. Niacin



Investigations of Dry Eyes

- Fluorescein sodium staining
- Rose Bengal



Rose Bengal

- Lissamine green stain (Newer and preferred)
- Schirmer's test



Schirmer's test

- Two filter paper strips entrapped in eyelid margin at Junction of lateral 1/3rd and medial 2/3rd.
- Cornea and limbus are avoided to prevent the reflex tears.
- Normal: 10mm of wetting
- Tear Break up Time: 10 seconds cut off
- Tear Osmolarity ↑ tear osmolarity: 308 mOsm/L
- MMP 9 marker of inflammation > 40 mg/ml

Management of Dry Eyes



- Tear film supplements
 - Preservative (BAK) free preferred
 - o lubricants like Methyl cellulose



Tear film supplements

- Cyclosporin drops
- Topical steroids in short bursts
- Punctal plugs
 - o For severe dry eyes
 - Plugs punctum with silicone plugs
 - Increase retention time of tears



Punctal plug

- Autologous serum
- Bandage contact lenses: silicone hydrogel

EYE SIGNS OF VITAMIN A DEFICIENCY (VAD)



00:17:37

- Xerophthalmia: spectrum of ocular disease due to Vitamin A deficiency
- · Xerophthalmia is the m/c cause of childhood blindness
- VAD defined as serum retinol level < 20 µg/dL
- Function of vitamin A
 - Normal functioning of vision
 - o Maintains epithelial cellular integrity
- Some signs reflect chronic VAD, others reflect sudden, severe VAD
- Children with any signs at high risk of dying

World Health Organization Re-classification of Xerophthalmia Signs ClassificationOcular signs

Classification		Ocular signs
XN	•	Night blindness
X1A	•	Conjunctival xerosis
X1B	•	Bitot's spots
X2	•	Corneal xerosis
ХЗА	•	Corneal ulceration: keratomalacia involving one - third or less of the cornea
ХЗВ	•	Corneal ulceration: keratomalacia involving one-half or more of the cornea
XS	•	Corneal scar
XF	•	Xerophthalmic fundus

Night blindness (XN)

- **o** 00:32:13
- Earliest clinical manifestation of VAD
- Also called "chicken eyes": chickens don't have rods, are night blind
- Early stages precipitated by photic stress like playing outdoors in bright sunlight
- Night blindness responds rapidly to Vitamin A therapy, within 24-48 hours

Conjunctival xerosis (X1A)



 Epithelium is transformed from normal columnar to stratified squamous epithelium with loss of goblet cell and keratinisation



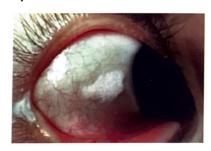
Conjunctival xerosis

- Expressed as marked dryness or unwettability and appears roughened
- "sandbanks after receding tides"
- Starts first in temporal quadrant

Bitot's spot (X1B)



- · Characteristic of VAD, not caused by any other condition
- · Elevated foamy white lesion of keratin



Bitot's spot

Corneal Xerosis(X2)



- Begins with superficial punctate lesions in infero nasal quadrant of cornea
- Cornea becomes hazy and lustreless, starting from inferior limbus



Corneal Xerosis

Ulceration/Keratomalacia (3 A, B)



- Pathology
 - Liquefactive necrosis of corneal stroma
- Ulcers are typically round or oval 'punched out' defects surrounding the clear cornea, no infiltration as in bacterial ulcers



Ulceration /Keratomalacia

- Shallow ulcers heal well with vitamin A therapy, deep ulcers perforate and lead to adherent leucoma
- 3B keratomalacia usually perforates, with loss of globe
- Prompt therapy may save other eve

Corneal Scars (XS)



Corneal opacities of all kinds: Nebula, macula, leucoma. adherent leucoma



Corneal Scar

- May lead to staphyloma which is
 - o Thinning and outpouching of sclera with iris incarceration
- Descemetocele
- Phthisis bulbi

Xerophthalmic fundus (XF)



- White, yellow punctate dots on the retinal periphery
- Causes Constriction of visual fields
- Disappears within 2-4 months of Vitamin A therapy

Management of Xerophthalmia



- Serum retinol levels > 0.70 µmoles / Litre
- 3 doses required to restore serum levels and boost liver stores

Age	Dose of Vitamin A	Frequency
<6 months	50,000 lµ	Day 1, 2, 14
6-12 months	100,000 Ιμ	Day 1, 2, 14
>12 months	200,000 Ιμ	Day 1, 2, 14



Previous Year's Questions

Q. The dosage of Vitamin A in keratomalacia in a 2year-old boy who is 12 kg weight is?

(NEET JAN 2019)

- A. Vitamin A: 2 lakh in oral, 1st. 2nd, 14th day
- B. Vitamin A: I lakh in oral 1st. 2nd 14th
- C. Vitamin A: 2 lakhs in oral, 1st, 2nd, 3rd
- D. Vitamin A: I lakh in oral, 1st. 2nd. 3rd



Previous Year's Questions

Q. which is the most sensitive screening test for vitamin A deficiency?

(FMGE DEC 2019)

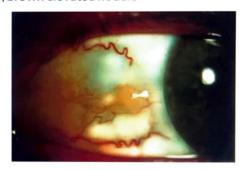
- A. Serum retinol < 10 ug /decilitre
- B. Beta carotene < 50 ug / decilitre
- C. Bitot's spots
- D. Night blindness

PINGUECULA



00:41:33

- Benign degeneration of conjunctiva
- Exposure to dust, wind, sand, radiation, CL wear
- · Grey, brown elevated nodule



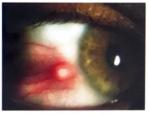
Pinguecula

- It is Asymptomatic
- Sometimes get Inflamed called as pingueculitis
- No treatment required

PHLYCTEN

尚 00:43:08

Pinkish white nodule surrounded by ring of vessels



Phlycten

It is Hypersensitivity reaction commonly to:

- o Tuberculin (India),
- Staph aureus (Western countries)

Risk factors

- Blepharitis
- Keratitis

Clinical features

- Seen along the limbal region,
- Presents with
 - o Pain.
 - o Photophobia,
 - Congestion
- Characteristically stains with fluorescein

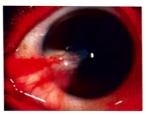
Treatment

- Topical steroids (DOC)
- For recurrent phlycten: Cyclosporine A
- Lid hygiene with warm compresses

PTERYGIUM



- Triangular overgrowth of subconjunctival tissue
- Wedge shaped, pink-white translucent membrane with apex extending into cornea



Pterygium

Stocker's line: iron line at leading edge

Risk factors

Exposure to UV rays, hot, dry weather, wind, dust

Symptoms

Irritation, lacrimation, FB sensation, vision \(\psi\), difficulty in contact lens wear

Treatment

- Excision with conjunctival autograft,
- Amniotic membrane transplant with fibrin glue / sutures

?

Previous Year's Questions

- Q. What is the treatment of choice for recurrent pterygium? (FMGE JUNE 2021)
- A. Simple excision
- B. Observation
- C. Excision with conjunctival auto graft
- D. Treatment with Mitomycin C

FOLLICLES



- Follicles are collection of lymphocytes,
- · Grey-white, rounded elevations,
- Pale surface with a red base
- Mostly found in inferior fornix and superior tarsal conjunctiva with overlying blood vessels



Follicles

Causes

- Chlamydia (m/c)
 - o Trachoma (Children)
 - o Inclusion conjunctivitis (Adults, milder)
- Viral
- Toxic: Brimonidine/Pilocarpine
- Molluscum contagiosum

PAPILLAE



- Collection of flattened nodules with central vascular core
- · Red Surface with pale base



Papillae

M/c causes

- Allergic immune response (VKC)
- FB as in Contact lens
- ocular prosthesis

Rule

- Bacterial conjunctivitis shows papillae
- Viral conjunctivitis shows follicles
- Chlamydia shows both



Important Information

- Follicles have Pale surface with a red base
- Papillae have Red Surface with pale base

CONJUNCTIVITIS



· Bright red congestion of conjunctiva



Conjunctivitis

Painless: May have FB sensation, itching, photophobia



Important Information

- Purulent discharge is seen in bacterial conjunctivitis
- Serous discharge is seen in Viral conjunctivitis
- Mucoid discharge is seen in Allergic conjunctivitis

BACTERIAL CONJUNCTIVITIS

Three types

- 1. Acute bacterial conjunctivitis
 - Cause
 - o In adults: Staph aureus (m/c)
 - o In children: Haemophilus influenzae (m/c) and strep pneumoniae
- 2. Hyperacute bacterial conjunctivitis
 - Caused by Neisseria gonorrhoea
- 3. Chronic bacterial conjunctivitis
 - Cause by Chlamydia

Clinical features

- Congested red eye,
- Yellow green purulent discharge,
- Photophobia,
- Matting of eyelashes



Bacterial Conjunctivitis

Treatment

- Fluroquinolones 4-6 hourly X7 days
- Hyperacute: Injection Ceftriaxone 1 gm IM single dose

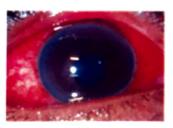
EPIDEMIC KERATOCONJUNCTIVITIS © 01:00:40



- Viral conjunctivitis caused by adenovirus 8,19,37
- 4 syndromes
 - EKC
 - 2. PCF (3,7)
 - 3. Acute nonspecific follicular conjunctivitis (NFC)
 - 4. Chronic keratoconjunctivitis
- Characteristic features are subepithelial infiltrates(7-10days)

Clinical Features

- Congestion
- Chemosis
- Photophobia
- **Epiphora**
- FB sensation
- **Pseudomembranes**
- Superficial punctate keratitis (SPKs)



Epidemic Keratoconjunctivitis

Also known as Pink eye, Madras eye, Shipyard eye

Treatment

- Lubricants
- Antihistamines
- Cool compresses

ACUTE HAEMORRHAGIC CONJUNCTIVITIS



- Caused by Enterovirus 70 and Coxsackie A24
- Also known as Apollo Disease
- · Rare compared to adenoviral conjunctivitis
- Usually seen in younger patients 11-15 years.
- More in developing nations
- Highly contagious, hand to hand spread

Symptoms

- Eyelid edema,
- FB, tearing,
- Discharge,
- Photophobia
- Conjunctival haemorrhage



Acute Haemorrhagic Conjunctivitis

Treatment

 Self-limited, disappears in 1-2 weeks, supportive care like cool compresses and antihistamines

ANGULAR CONJUNCTIVITIS

- **Ö** 01:07:45
- Subacute bilateral conjunctivitis of lateral canthus of evelid
- Caused by Morax Axenfeld bacillus

Clinical features

Congestion, scaling, fissuring, maceration



Angular Conjunctivitis

Seen in Chronic alcoholics, nutrition deficient

Treatment

DOC: Erythromycin, Bacitracin, zinc oxide

OPHTHALMIA NEONATORUM / NEONATAL CONJUNCTIVITIS © 01:09:20

Acute, mucopurulent infection of conjunctiva within 4 weeks of birth



Ophthalmia Neonatorum

Reduced tear secretion, Limmunity



Important Information

- Crede's method: Instillation of 1/. AgNO₃ (Silver nitrate) at birth to prevent infection from Neisseria gonorrhoea
- Silver nitrate prevented the gonococcal conjunctivitis but causes chemical conjunctivitis

Causes

- First day of life: Chemical conjunctivitis (Silver nitrate prophylaxis)
- 2-3-day: Gonococcal conjunctivitis
- 5-14 days: Chlamydia trachomatis (m/c)
- Herpes simplex virus (HSV)
- Pseudomonas, though rare, may perforate cornea

TRACHOMA



Chronic follicular conjunctivitis caused by Chlamydia



Trachoma

- Also known as Egyptian ophthalmia seen in Africa, Middle East, Indian subcontinent Southeast Asia South America
- Types of Chlamydia trachomatis
 - o Chlamydia serovars A, B, Ba, C causes Trachoma
 - Chlamydia serovars D, E, F, G, H, I, K causes Inclusion conjunctivitis
 - Chlamydia serovars L1, L2, L3 causes Lymphogranulomavenereum (LGV)
- · Commonest infective cause of blindness
- 2 diseases: Infection mostly seen in children (3-5-year old's), blinding sequelae in adults 40 years later

Risk Factors of Trachoma

- Endemic areas
- Crowded living conditions
- Poor water supply
- Poverty
- Domestic animals in close proximity

Transmission of Trachoma

- Flies
- Fingers
- Fomites

Clinical features of Trachoma

- Acute stage
 - o Hyperemia
 - o Chemosis
 - Watery discharge
 - o Photophobia
- Late stages
 - o FB sensation
 - Tearing

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- Photophobia
- o Ivision
- "Sago grain follicles"
 - o Grey, white oval elevations
 - o 0.5-1.5 mm diameter



Sago grain follicles

Herbert's pits



Herbert's pits

- · Arlt's line
 - o White line in upper palpebral conjunctiva



Arlt's line



Important Information

Six states having maximum Trachoma

- I. Punjab
- 2. Haryana
- 3. Uttar Pradesh
- 4. Uttaranchal
- 5. Rajasthan
- 6. Gujarat



Previous Year's Questions

Q. Herbert's pits are seen in?

(JIPMER NOV 2018)

- A. vernal conjunctivitis
- B. Atopic conjunctivitis
- C. Gonococcal conjunctivitis
- D. Chlamydial conjunctivitis

Late Sequelae

Trichiasis (Posterior misdirection of eyelashes)



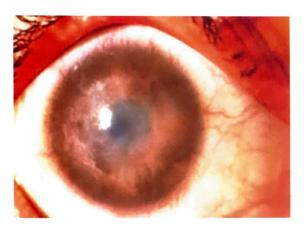
Trichiasis

- o Tylosis (Thickening of the eyelid margin)
- o Madarosis (Loss of eyelashes)



Madarosis

- Entropion (Inward rolling of lid margin)
- Dry Eyes
- Corneal opacities
 - → Nebula (Superficial, maximum discomfort)
 - → Macula



Corneal opacity

→ Leucoma (maximum loss of vision)



Leucoma



Previous Year's Questions

Q. what is the term given to a condition with an extra Layer of cilia posterior to grey line?

(NEET JAN 2020)

- A. Tylosis
- B. Madarosis
- C. Distichiasis
- D. Trichiasis

WHO GRADING OF TRACHOMA



I. F - (TF): > 5 follicles, 0.5 mm in diameter

II. I - (TI): obscuring > 50% tarsal blood vessels



III. S - (TS): Scarring of tarsal conjunctiva



IV. T - (TT): One trichiatic cilia rubbing on cornea V. O - (CO): Opacity obscuring a part of pupil



How to remember

FISTO

Management of Trachoma



- SAFE strategy
 - Surgery
 - Antibiotics
 - Facial Hygiene
 - Environmental improvement
- Blankettherapy
- 1% Tetracycline ointment bd X 5 days in a month X 6
- Active infection
 - DOC Azithromycin 1gram single dose (20 mg/kg)
- Doxycycline 100mg twice a day for 7 days
- Topical therapy
 - Tetracycline 1% or Erythromycin ointment 2 times a day X 6 weeks

Management of Late stage

- For Entropion correction: Bilamellar tarsal rotation (BLTR)
- For Trichiasis: Epilation / Electrolysis
- For Corneal opacity: Keratoplasty / Optical iridectomy

VISION 2020



01:36:50

Aim

To obtain 6 / 6 vision by 2020 for everyone

By elimination of

- Cataract
 - o CSR: number of cataract surgeries per Million population in a year
- Trachoma
- o GET 2020 (Global eradication of Trachoma)
- Childhood blindness
- SAFE strategy
- Refractive error
- Onchocerciasis
- o Treated by Ivermectin
- India: Diabetic retinopathy
- Corneal opacities
- Glaucoma

VERNAL CATARRH/SPRING CATARRH/ VERNAL KERATOCONJUNCTIVITIS (§ 01:40:29)

- It is Seasonal, recurrent, bilateral, hypersensitivity reaction in childhood, resolves after puberty
- Type I hypersensitivity allergic reaction
- Allergic condition: 'Morning misery'
- · Occurs in Summers: Indian sub-West Africa, Mediterranean

3 subtypes

Palpebral

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- Limbal
- Mixed

Symptoms

- Itching
- Tearing
- Irritation
- Redness
- Photophobia
- May be severe enough to interrupt a child's social / educational development

Signs of Spring Catarrh

- Cobblestone papillae
 - Red center
 - Flat surface
 - Giant papillae



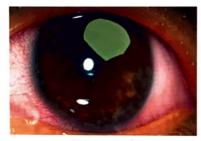
Cobblestone papillae

- Ropy discharge
- Horner Tranta's spots



Horner Tranta's spots

o Shield ulcer (very severe)



Shield ulcer

- Maxwell Lyon's sign
 - Fibrin enhanced by heat accumulates on papillae



Important Information

Any papillae greater than Imm in diameter are termed as Giant papillae

Management of Spring Catarrh



- Step ladder pattern
 - o Mild forms
 - →Mast cell stabilizers: Sodium Cromoglycate / Lodoxamide
 - → Antihistamines: Levocabastine
 - → Dual acting: Alcaftadine / Azelastine / Olopatadiene
 - Moderate to severe forms
 - → Topical steroids: Loteprednol/ Flurometholone /Prednisolone
 - Severe forms
 - → Immunomodulators: Cyclosporine A / Tacrolimus



Previous Year's Questions

Q. Phlyctenular conjunctivitis is seen due to:

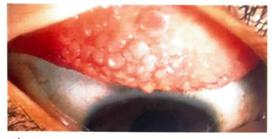
(FMGE DEC 2019)

- A. Post fungal infection
- **B.** Allergic reaction
- C. Post protozoal
- D. Post tuberculoid



Previous Year's Questions

Q. A female comes with 2-year history of contact lens use presents with eye pain, irritation and foreign body sensation. Identify the diagnosis? (NEET SEP 2021)



- A. Trachoma
- B. Giant papillary conjunctivitis
- C. Spring Catarrh
- D. Acute follicular conjunctivitis



LEARNING OBJECTIVES

CORNEA

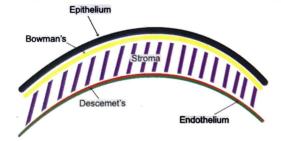
- Layers of Cornea
- Keratoplasty
 - Penetrating Keratoplasty (PK)
 - Lamellar Keratoplasty
- Graft Rejection and Donation of Cornea
- Corneal Ulcers / Keratitis
 - Acanthamoeba Ulcer
- Various Types of Keratitis
- Herpes Zoster Ophthalmicus (HZO) (SHINGLES)
- Treatment of Corneal Ulcers
- Corneal Dystrophies
- Fuchs' Endothelial Dystrophy
- Various Types Of Refractive Surgery
 - LASIK (laser Assisted in Situ Keratomileusis)
 - LASIK V/s SMILE
- Keratoconus
- INTACS



14 CORNEA

LAYERS [OUTER TO INNER]





Layers of Cornea

1. Epithelium

- Stratified squamous non keratinizing
- 50 µ in thickness
- 3 layers
 - o Squamous
 - o Wing
 - o Basal

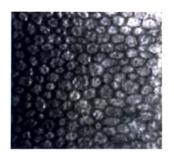
2. Bowman's membrane

- Strong, thin, avascular layer, 10 μ
- Corneal scar/ opacity results from damage to Bowman's membrane
- Bowman's membrane can't regenerate
- 3. Stroma
- Thickest layer (90%)
- 75% water
- Contains Keratocytes & Collagen lamellae in
- Ground substance
- 4. Pre descemet's layer / Dua's layer
- 15 µ in thickness
- Immensely Strong
- Cannot regenerate
- 5. Descemet's membrane
- 10µ, protects against microbes



Important Information

• Only fungus can penetrate intact Descemet's membrane



Endothelium

- Single layer of cells (Monolayer cells)
- Irreparable: leads to irreversible corneal edema
- Active endothelial Na⁺-k⁺ ATPase pumps pumps out aqueous humor
- Most important layer responsible for corneal transparency
- · Two main functions of cornea
 - Protect intraocular contents
 - Serve as principal optical element

Factors maintaining corneal transparency

- Corneal avascularity
- Epithelium inhibits diffusion of water and ions
- Corneal lamellae are arranged orderly in lattice
- Spacing between collagen fibrils > half of wavelength of light
- Endothelial pumps: Na⁺ k⁺ ATPase pumps (1.5 million pumps per cell)

Specular microscope is used to count endothelium cells

- Average/endothelial/specular count: 3000 cells/mm²
- Every year loss of 0.5% cells
- Critical density: <500 cells /mm²
- For corneal donation: >2000 cells /mm²

<50 cells /mm3

Irreversible corneal edema

⊥ vision

Treatment by corneal transplantation [keratoplasty]

6. Endothelium

Join the Telegram Channel - https://t.me/prepladderlatestnotes



Important Information

- Damage to Bowman's membrane: Corneal scar
- Damage to Endothelium: Corneal edema



Previous Year's Questions

- Q. Which layer of cornea helps in maintaining hydration of stroma of cornea? (NEETJAN 2020)
- A. Descemet's membrane
- B. Endothelium
- C. Epithelium
- D. Stroma



Previous Year's Questions

- Q. All of the following are features of corneal epithelium except? (AIIMS JUNE 2019)
- A. Lined by stratified squamous epithelium
- B. Bowman's membrane regenerates
- C. Apical cells have microvilli
- D. Mitosis is limited to limbus

KERATOPLASTY



00:15:36

Penetrating Keratoplasty (PK) • Full thickness keratoplasty • Partial thickness keratoplasty

- All 5 layers taken from donor
- Only outer layers taken from donor recipient retains his own
 - o Endothelium
 - Descemet's membrane
 - o Stroma ±
- Donor provides
 - Epithelium
 - o Bowman's membrane
 - o Stroma ±

- Relatively easy
- LK is technically more demanding
- Less successful than LK
 More successful







Important Information

- Corneal transplant is the most successful organ transplant in the body [cornea is avascular]
- Max. rejection is from endothelium

Indications of PK (Penetrating keratoplasty



- PK [penetrating Keratoplasty]
- Pseudophakic bullous keratopathy [PBK] [mc-worldwide]
- 2. Fuchs Endothelial Dystrophy (FED)
- 3. Non healing ulcer
- 4. Corneal scar [mc indication in India]
- 5. Corneal dystrophy
- 6. Keratoconus [mc indication in America]
- 7. Chemical injuries



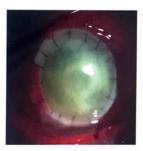
Pseudophakic bullous keratopathy

Fuchs Endothelial Dystrophy

Therapeutic Keratoplasty

Ö 00:20:54

 Transplant to eradicate active infectious disease, or to repair a structural defect of cornea



Therapeutic keratoplasty

- Indication
 - Microbial keratitis
 - Persistent epithelial defect: Sterile melt

PK Complications



- · Persistent epithelial defect
- · Suture related complications
- Microbial keratitis
- Glaucoma
- Wound leak
- Primary endothelial failure
- Graft rejection



Previous Year's Questions

Q. A patient presents to OPD with decreased vision in left eye and opacification of cornea. He has a history of cataract surgery years bacle. what is the line of management in this case? (FMGE JUNE 2021)

- A. Kertotomy
- B. Keratoplasty
- C. LASIK
- D. DCR



Previous Year's Questions

Q. Whorled keratopathy is seen with use of (JIPMER NOV 2018)

- A. Digoxin
- B. Amiodarone
- C. Ethambutol
- D. Steroids

GRAFT REJECTION



 Immunologic response of host to donor cornea → remains clear for 2 weeks, then develops edema



Graft Rejection

Types

- Epithelial rejection
- Sub epithelial rejection
- Endothelial rejection
- M/c rejections are against Endothelial layer -50%

Symptoms

- ţvision, pain, redness, photophobia
- Corneal edema, KP's on graft, stromal infiltrates



Important Information

 Khodadoust line on endothelium is typical sign of graft rejection



Graft Rejection

Treatment

- Topical/Systemic steroids
- Cyclosporine A
- Tacrolimus

LAMELLAR KERATOPLASTY



Types

- 1. Anterior Lamellar Keratoplasty (ALK)
- Replacement of anterior part of cornea (Epithelium and stroma)
- Most famous type of ALK is Deep Anterior Lamellar Keratoplasty (DALK)
- Posterior Lamellar keratoplasty (PLK) / Endothelial Keratoplasty (EK)
- Replacement of DM and endothelium keeping anterior layers of cornea intact
- Descemet's Stripping Automated Endothelial Keratoplasty (DSAEK)
- Descemet's Membrane Endothelial Keratoplasty (DMEK)

DALK (Deep Anterior Lamellar Keratoplasty) 0 00:31:09

Selective transplantation of stroma, leaving DM and endothelium intact

- Indications
 - o Keratoconus
 - Stromal dystrophy
 - Corneal scars



DALK

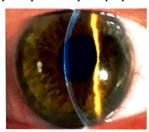
- Stronger post op wound
- Shorter healing time: Because we use 1 steroids
- Sutures can be removed in 4-6 months
- ↓ rejection chances because endothelium is intact

Endothelial Keratoplasty (Posterior Lamellar © 00:34:10 Keratoplasty)

- Replacement of DM and endothelium keeping intact anterior layers of cornea (Epithelium, Bowman's membrane, and stroma)
- Indications
 - o Fuchs' Endothelial dystrophy,
 - Posterior polymorphous dystrophy
 - o ICE: endothelial dysfunction syndromes
- Benefits
 - o Maintains structure integrity
 - o Sutures are not used
 - o Superior VA
 - o Astigmatic neutral
 - o 1 chances of rejection

DSAEK

- **Ö** 00:36:16
- Replacement of diseased tissue with DM, endothelium and 100 µ posterior stroma
- Indications
 - Fuchs' endothelial dystrophy
 - o Pseudophakic corneal Edema
 - o Posterior polymorphous dystrophy



DSAEK

- Advantages
 - Rapid healing time
 - o No sutures (So no associated complications)
 - Jgraft rejection
 - No epithelium related complications
- Complications
 - Hyperopic shift of 1 1.5 OD

DMEK



- Offers the fastest visual rehabilitation of any keratoplasty technique
- Final VA can be exceptional due to minimal optical interface defects

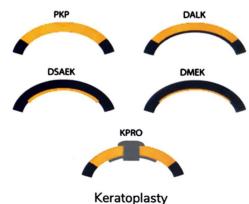


DMEK

- Replaces diseased tissue with DM & Endothelium
- tchances of rejection because minimal tissue transplanted
- Lower reliance on steroids
- Graft tissue 10-15 µ, fragility and thinness may be difficult to handle
- Technically more demanding

Evolution of Keratoplasty





CORNEAL DONATIONS

(D) 00:40:44

- HLA/ABO matching not required
- No age limit for donations but best results from corneas of donor age < 75 years
- · Within 6 hours of death
- Cornea is preserved in MK (McCarey Kaufmann) media: 96 hours
- Other media

o Optisol GS: Preserve cornea for 7 days - 14 days



Corneal preservative solution

Contraindications

- o HIV
- o Hepatitis B
- o Septicemia
- o Rabies
- o Prions
- o Retinoblastoma
- o Metastatic brain tumor
- o Leukemias
- o Lymphomas
- Head & neck cancers
- Only 65,000 corneas were collected in India last year
- India requires 1.5 lakh corneas per year
- Tamilnadu & Gujrat are the two states which have highest number of cornea donors
- The country which donates eyes: Srilanka

CORNEAL ULCERS/KERATITIS



00:42:14

- · Defect in epithelium with underlying necrosis of stroma
- Hypopyon formed
 - d/t accumulation of white blood cells (1st line of defence)
 - Steroids C/I: causes perforation of cornea

\Diamond

Important Information

Hypopyon is seen in both Anterior uveitis and Corneal ulcer

Stages

- Infiltration
- Active ulceration
- Regression
- Cicatrisation

Risk factors

Ocular trauma

- Contact lens wear
- Agricultural workers exposed to organic matter
- Immunosuppression
- Exposure to hot tubs or swimming pools

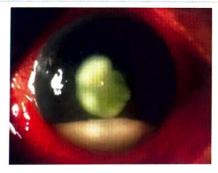
Potentially Sigh Threatening (PST) ulcers: 1, 2, 3 rule

- > Cells 1+ in Anterior Chamber
- Dense infiltrates > 2 mm in greatest linear dimension
- Edge of infiltrate less than 3 mm from centre of cornea



How to remember

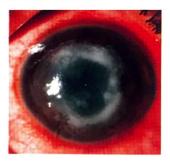
• 1, 2, 3 rule



Corneal Ulcers

ACANTHAMOEBA ULCER





Predisposing Factor

Contact lens Wearer

Risk factor

- 1. Contact lens cleansed with water
- 2. Corneal trauma: exposure to soil or contaminated H₂O



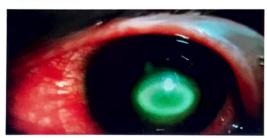
Important Information

- MC organism attacking contact lens wearer:
 Pseudomonas > Acanthamoeba
- Pseudomonas can attack any contact lens wearer whereas Acanthamoebas preferably attacks people that wash there contact lens with water

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Clinical diagnosis

- Cystic epitheliopathy without infiltrate, confused with herpes
- Ring shaped ulcer 50% cases
- Radial keratoneuritis
 - Pain out of proportion
 - Forms Pseudodendrites
 - →H/o suspected HSV keratitis not responding to treatment



Diagnosis

- Immediate: Double walled cysts in smears stained with calcoflor white
- Gold standard: Cultured on Non nutrient agar with E coli lawn overlay
- PCR is used when contact lenses are involved
- Confocal microscopy (latest instrument)

Treatment

- DOC: PHMB (Poly hexa methylene biguanide)
- Chlorhexidine
- Propamidine



Previous Year's Questions

- Q. Drug of choice for acanthamoeba keratitis. (JIPMER MAY 2019)
- A. Dilaxanide furoate
- B. Paromomycin eye ointment
- C. Chlorhexidine
- D. Azithromycin I gm single dose

BACTERIAL KERATITIS



M/c cause of microbial keratitis

Risk Factors

- Trauma
- Contact Lens wear
- Loose sutures
- Dry eyes
- Exposure Keratopathy
- Bullous keratopathy



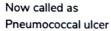
Important Information

 M/C cause of bacterial keratitis worldwide: Staph aureus/Pseudomonas
 M/C cause of bacterial keratitis in India:

Pneumococcus

Symptoms

- Pain
- Redness
- Photophobia
- Discharge
- Vision loss
- Illaus serpens
- Hypopyon corneal ulcer





Bacterial keratitis

Bacterial keratitis

LIST OF BACTERIA PENETRATING INTACT CORNEAL EPITHELIUM (5) 00:59:47

- Corvnebacterium
- Gonococcus
- Meningococcus
- Hemophilus
- Listeria
- Shigella

Diagnosis

- All bacterial ulcers are scrapped and cultured
 - o Gram and Giemsa stains of corneal smears
 - o Culture on blood and chocolate agar

Treatment

- · For periphery ulcers
 - o Topical fluoroquinolones
- For central ulcers
 - o PST ulcers: Fortified Cefazolin and Tobramycin
- For Severe pain
 - o Cycloplegics: Atropine, Homatropine, Cyclopentolate



Important Information

 Systemic antibiotics are not used in corneal ulcers as cornea is avascular. They are only used in corneal ulcers in case of scleral extension and perforation.

HIGHLIGHTS



- Topical antibiotics to prevent acute bacterial keratitis in contact lens related corneal abrasions
- NEVER patch the eye in bacterial keratitis
- Steroids may be prescribed after 24-48 hours organism identified/responding to therapy
- Avoid in Fungus, Nocardia, Acanthamoeba
- Beware of ↑ resistance to MRSA and Pseudomonas to topical fluoroquinolones
- Smears / Cultures: In large (>2 mm), central, deep stromal infiltrate, not responding, atypical features

?

Previous Year's Questions

Q. A patient wearing contact lenses since two years develops decrease in vision and redness of eyes. what could be the cause? (FMGE JUNE 2021)

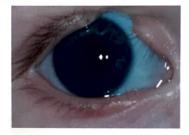
A. Bacterial corneal ulcer

- B. Fungal keratitis
- C. Giant papillary conjunctivitis
- D. HSV keratitis



Previous Year's Questions

Q. Patient presenting with following finding what is the most likely complication associated with it?
(NEET 2021)



- A. Cataract
- B. Exposure keratitis
- C. Difficulty in ocular movements
- D. Glaucoma

FUNGAL KERATITIS / KERATOMYCOSIS

- Slow, relentless, potentially catastrophic ulcers
- M/c cause of fungal ulcers: Filamentous fungi (Fusarium and Aspergillus)
- Signs are more than symptoms

Risk Factors

- Topical steroid usage
- Injury with vegetable matter
- Warm tropical climates
- Exposure to soil

Examination

- Finger like projections
- Feathery margins
- Satellite lesions
- Hypopyon
 - o Non-sterile and infectious (Contains fungal hyphae)
 - Non-mobile



Finger like projections

Fungal Keratitis

Management

- KOH wet mount of Corneal scrapings (best)
- Culture media: blood agar and Saboraud's agar
- Long, protracted course, difficult to treat

Treatment

- DOC 5% Natamycin for filamentous keratitis
- Topical voriconazole, Amphotericin B
- Cycloplegics
- Taper all topical steroids IOP checked frequently → inflammatory glaucoma
- Systemic anti fungals for deep ulcers, endophthalmitis and perforations



Important Information

Steroids worsen the fungal corneal ulcers



Previous Year's Questions

- Q. Characteristic finding of fungal ulcer (NEET JAN 2020)
- A. Satellite lesions
- B. Dendritic ulcer
- C. Ring abscesses
- D. Feathery ulcer with white hypopyon



Previous Year's Questions

Q. A patient comes to AIMS OPD, with acute pain and watering from eye for 3 days. There was 3x2 mm ulcer on the cornea with ROLLED OUT margins and feathery and finger like projections with minimal hypopyon.

(AIMS NOV 2018)

- A. Acanthameba
- B. Aspergillus
- C. HSV2
- D. Pseuomonas

VIRAL KERATITIS



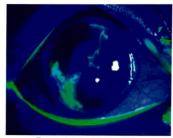
- M/C cause is HSV Type I
- HSV keratitis: Other body part affected
- Reactivated by: Stress, fever, trauma, sun exposure, Contact Lens

3 types

- 1. Epithelial
- 2. Stromal
- 3. Endothelial

1. Epithelial

- · Dendritic/Geographiculcer
- Cause Photophobia/redness/Blurring of vision
- Loss of corneal sensation in HSV keratitis
- Footprints on cornea: Anterior stromal opacities



Dendritic Keratitis

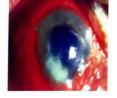
Important Information

- Pseudo dendrites are present in
 - Acanthamoeba
 - Herpes zoster
 - Contact lens wearers
- But only true dendrites are present in HSV

2. Stromal Keratitis



- Highest and most severe ocular morbidity
- 2 Types
- 1. Immune Stromal keratitis (ISK)
 - o Mid stromal infiltrates with intact epithelium
- 2. Necrotising stromal keratitis (NSK)
 - Epithelial defect, stromal infiltrates → leading to perforation



HSV Stromal keratitis

- Treatment
 - Topical steroid therapy under cover of oral antivirals

3. Disciform Keratitis (Endothelitis)

- **(1)** 01:19:02
- Cell mediated immune reaction leads to diffuse stromal edema
- Acute onset, U/L central corneal disc shaped stromal edema with KP's
- DOC: Topical steroids



Disciform Keratitis (Endothelitis)

HERPES ZOSTER OPHTHALMICUS (01:20:24 (HZO) (SHINGLES)

- Caused by Varicella Zoster Virus (VSIV): Chicken pox
- Reactivated from dorsal ganglion in immuno compromised patients as herpes zoster

Examination

- U/L painful skin rash along the dermatome distribution of trigeminal nerve (V)
- · Painful vesicles along Vth nerve associated with prodrome of fever, malaise, headache and pain



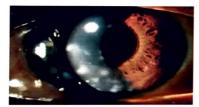
Important Information

- HZO is Usually seen in patients above 60 years
- · If seen in Young patient, it is sign of HIV



Hutchinson Rule

- Ocular signs: Severe eyelid edema
- Hutchinson sign: If Tip of nose affected → eye gets involved
- · Punctate keratitis, pseudodendrites: No terminal bulbs, nummular keratitis
- Treatment is started within 72 hrs: Post herpetic neuralgia (PHN)



HZO (Shingles)

Management of Herpes Zoster

- 800 mg Acyclovir 5 times X day for 7 10 days
- Valacyclovir/Famcyclovir
- Systemic corticosteroids controversial
- Topical steroids for keratouveitis
- Lubricants, Punctal occlusion, Bandage Contact Lens dry eyes

PHN (Post Herpetic Neurolgia)

- Spontaneous resolution of pain
- Treatment can \(\pain \) but not eliminate
- Gabapentin, Pregabalin, Amitriptyline, Nortriptyline

TREATMENT OF CORNEAL ULCERS © 01:28:19



Bacterial ulcer Fungal ulcer	 4th gen fluoroquinolones Moxiflaxacin Gatifloxacin Natamicin Voriconazole
Viral ulcers	Acyclovir Famcyclovir Valacyclovir
Acanthamoeba	PHMB (Polyhexamethylene biguanide)Chlorhexidine/Propamidine
Pain	Cycloplegics
Non healing ulcer	Uncontrolled DMwrong diagnosisForeign Body
Perforated corneal ulcer	Keratoplasty





Perforated corneal ulcer

Tissue glue

- 1. Fibrin
- 2. Cyanoacrylate glue

For ulcers sizes <2 mm



Previous Year's Questions

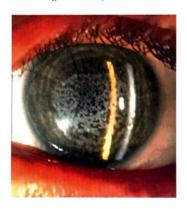
Q. Drug contraindicated in keratitis.

(FMGE JUNE 2019)

- A. Tear drops
- B. Systemic steroids
- C. Cycloplegics
- D. Timolol

CORNEAL DYSTROPHIES

- **Ö** 00:00:13
- It is inherited bilateral, symmetrical, slowly progressive, non-inflammatory, corneal opacities without any systemic involvement
- Congenitally derived mutations which causes deposition of insoluble matter (proteins) on cornea



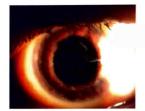
Corneal dystrophies

Types

- 1. Anterior corneal dystrophies deposits seen in Epithelium /Basement membrane/Bowman's membrane
- 2. Stromal Dystrophies deposits seen in Corneal stroma
- 3. Posterior corneal dystrophies deposits seen in Descemet's and endothelium

ANTERIOR CORNEAL DYSTROPHIES

- 1. Map dot fingerprint dystrophy (Epithelia Basement membrane Dystrophy) 00:02:41
- Most common dystrophy
- Abnormality in production of BM → multiple BM layers in epithelium
- Mostly Asymptomatic (90%)
- In 10% painful recurrent corneal erosions with loss of vision
- Looks like continents on map, dots (microcysts), fingerprint like lesions, RCE: areas of loose epithelium, epithelial defect



Map dot fingerprint dystrophy

Treatment

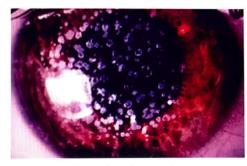
 Lubricants, hypertonic (5% NaCl) drops, bandage contact lenses, Excimer laser PTK

STROMAL DYSTROPHIES

1. Granular dystrophy



- Classic granular dystrophy recurrent painful erosions in young adults
- Hyalin deposits stain with Masson trichrome
- Symptoms
 - Mostly asymptomatic, \u00edvision, recurrent painful erosions, glare, photophobia
- Examination
 - Small, discrete, white granules ('crushed breadcrumbs') in the stroma with clear intervening spaces → may cover pupillary axis



Granular dystrophy

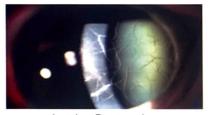
- Treatment
 - Lubrication, bandage contact lenses, excimer laser photo therapeutic keratectomy (PTK)

2. Lattice Dystrophy



00:09:29

- M/c stromal dystrophy
- Amyloid deposits stain with Congo red dye
- Symptoms
 - Painful, recurrent erosions with loss of vision
- Examination
 - Central, branching, refractile lines, glass like opacities, white dots, diffuse stromal haze



Lattice Dystrophy

- Treatment
 - Lubrication, bandage contact lenses, Excimer laser PTK

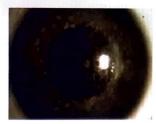
3. Macular Dystrophy



00:11:18

- Autosomal recessive
- Mucopolysaccharides deposits stain with Alcian blue
- Symptoms
 - Glare, \u00edvision, painful recurrent erosions in young adults
- Examination

- Central grey-white opacities with diffuse cloudiness of intervening stroma
- Cornea thinner



Macular Dystrophy

- Treatment
 - Excimer PTK, may need keratoplasty

Compared to other stromal dystrophies, macular dystrophy is



- Is least common
- Reduced vision at early age
- Has thinner central corneal thickness
- No clear areas in intervening stroma
- Extends to peripheral cornea
- Requires keratoplasty at an earlier age

Summary of stromal dystrophies

Granular	Lattice	Macular	
AD	AD	AR	
Breadcrumbs	Filaments/Lines	Diffuse	
Clear interval	Clear interval	Hazy interval	
Limbal sparing	Limbal sparing	Limbus to	
Good vision	Good vision	Limbus	
		Poor vision	

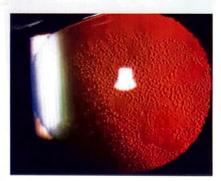
FUCHS' ENDOTHELIAL DYSTROPHY © 00:16:00



- Common, B/L progressive dystrophy affecting DM/ Endothelium, in 5th - 6th decade of life
- More common in females than males

Pathology

- Cornea guttata (excrescences of thickened DM), pigment dusting on endothelium leading to corneal edema
- · Stromal edema giving a blue -grey haze (ground glass appearance) with Slow, painless loss vision which worse on getting up in morning
- Epithelial edema → 'bedewing' → significant loss of vision
- Bullae ruptures → severe pain



Fuch's Endothelia Dystrophy

Treatment

- Hypertonic saline, JIOP
- Blowing Warm air into using hair dryer for 10 minutes
- Keratoplasty



Important Information

- Most common gene associated with corneal dystrophies is $TGF\beta I$ - transforming growth factor β induced
- All stromal dystrophies are AD except Macular → AR
- Most manifest during childhood except Map Dot Fingerprint and Fuchs' which manifest later in life

How to Remember Dystrophies

- M Macular dystrophy
- M Mucopolysaccharide deposits
- A Alcian blue stain
- G Granular dystrophy
- H Hyaline deposits
- M Masson trichrome stain
- L Lattice dystrophy
- A Amyloid deposits
- C Congo red stain



How to remember

Marilyn Monroe Always Gets Her Man in LA City

REFRACTIVE SURGERY



Principle	Corneal curvature changed
In myopia	Cornea is flattened
In hypermetropia	Cornea is steepened
In Astigmatism	Both done

Types of Surgery

- 1. Incisional surgery
 - Radial keratotomy (RK)
 - Astigmatic keratotomy (AK)

2. Laser

- Photo Refractive Keratectomy (PRK)
- Laser assisted in situ keratomileusis (LASIK)
- Small incision lenticule extraction (SMILE)

3. Implant

Phakic IOL

RADIAL KERATOTOMY (RK)



- Radial incisions of 90 % depth: Guarded diamond knife in paracentral and peripheral cornea
- It Flattens central cornea: Correct myopia
- Corrects myopia: <5 Dioptres



Radial Keratotomy (RK)

PHOTO REFRACTIVE KERATOTOMY © 00:34:24 (PRK)

- Uses excimer laser having Argon Fluoride gas which has wavelength of 193 nm
- It Flattens central cornea in myopia
- It Steepens central cornea in hypermetropia, removal of tissue paracentrally
- Corrects Myopia: 8-10 Dioptres
- Corrects hyperopia: 3-4 D
- Corrects Astigmatism: 3-4 Dioptres

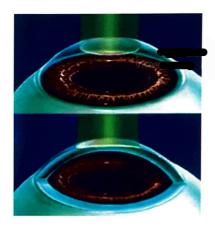


Photo Refractive Keratetomy (PRK)

Indications of PRK

- In cases where LASIK cannot be done
- Dry eyes
- Thin corneas
- Highly active lifestyle: No flap dislocation

Complications

Corneal scarring

LASIK (LASER ASSISTED IN SITU KERATOMILEUSIS)



- M/c Laser surgery for refractive correction
- Uses Excimer laser
- Flap is made under Bowman's membrane, No risk of scarring
- Average flap 120 μ , Residual Stromal Tissue >250 μ , 14 μ ablation \rightarrow 1 D
- Corrects Myopia < 9 Dioptres
- Corrects Hypermetropia: + 4 D
- Corrects Astigmatism: 5 D

Absolute C/I for LASIK

- Keratoconus
- Thin corneas
- Unstable refractive error (if power changes more than 0.5D in last 1 year)

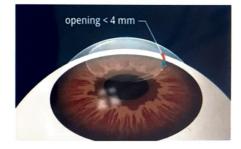
Relative C/I

- Glaucoma
- Corneal dystrophy
- HSV

SMALL INCISION LENTICULE EXTRACTION (SMILE)



- Femtosecond Laser: create a corneal lenticule which is extracted whole through a small incision
- With lenticule removed the cornea flattens correcting myopia
- Corrects Myopia: 10 dioptres
- Corrects Astigmatism: 3 D



Small Incision Lenticule Extraction

Advantages

Preservation corneal nerves

- Jdry eyes
- † Biomechanical stability
- More technically demanding

LASIK V/s SMILE



LASIK	SMILE
• Excimerlaser	• Femtolaser
 Corrects Myopia, hyperopia, astigmatism 	• Correct myopia & astigmatismonly
Flap required	 No flap required
• 2 – corneal flaps & then Laser ablation	• 2 Femtolaser cuts & lenticule dissected
• Healing is Faster	Healing is Slower
• ↓Biomechanical stability	• ↑ biomech stability
• Dry Eyes	• ↓Dry eyes

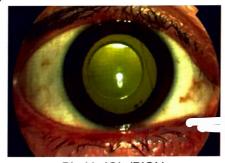
PHAKIC IOL (PIOL)



- High refractive errors unsuitable for laser surgery
- IOL inserted into AC / PC through corneal incision, without removing crystalline lens

Indications

 Thin corneas, high refractive errors, (IOL approved upto -20.0 D) Keratoconus



Phakic IOL (PIOL)

Ideal candidate

- Not suitable for laser, poor tolerance of glasses / Contact Lens
- Stable refraction
- Irido-corneal angle > 30°
- Endothelial counts > 2300/sq mm
- Pupil < 5-6 mm

Complications

- Cataract
- Endothelial cell loss

KERATOCONUS



- It is non-inflammatory, bilateral, progressive corneal ectasia with central thinning
- It is cone like protrusion of cornea
- Onset typically in early adolescence: Mid 30's



Keratoconus

Risk factors

- Eye rubbing
- Associated with atopy
- Sleep apnoea
- Floppy eyelid syndrome
- Associated with Down's Syndrome, Leber's Congenital Amaurosis, Retinitis pigmentosa



Important Information

 M/C presentation of Keratoconus is Blurring of vision with frequent change of glasses in young patient due to curvature myopia

Symptoms

Early signs

- High irregular / asymmetric astigmatism
- Scissoring of reflex on retinoscopy
- Fleischer's ring
- Vogt's striae



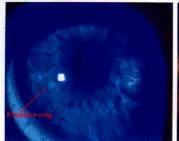
Important Information

- Fleischer's ring is deposition of Iron on epithelium around base of cone in keratoconnus
- KF ring is deposition of copper on Descemet's membrane in Wilson's disease



Important Information

 Vogt's striae are vertical folds in corneal stroma seen in keratoconus whereas Haab's striae are horizontal striae seen in buphthalmos





Fleischer's ring

Voqt's striae

Later Signs

Munson's sign: Lower lid notching on down gaze



Munson's sign

- Acute hydrops: Corneal edema → break in DM → aqueous entry → pain, ↓vision & photophobia
 - Treatment
 - → Antibiotics, cycloplegic, hypertonic saline, patching
 - → Stromal scarring after resolution of hydrops

Diagnosis

 Slit lamp Examination, refraction, Keratometry, Pachymetry, Topography

Management

- Functional vision and arrest progression
- Glasses and Rigid gas permeable Contact Lenses
- Special Contact Lenses: Rose K, piggyback, scleral lenses
- Corneal Collagen Crosslinking with Riboflavin: C3R
- Surgery
 - o INTACS
 - o DALK
 - o PK



Previous Year's Questions

Q. A 20 year old male complains of repeated changes in glasses, may result in, (FMGE DEC 2019)

- A. Keratoconus
- B. Pathological myopia
- C. Glaucoma
- D. Cataract



Previous Year's Questions

Q. A 20 year old boy complains of sudden painful loss of vision .0/E. LE was hazy and there was bulging of left lower lid on looking down Retinoscopy shows scissoring: oil drop sign was positive. Diagnosis? (JIPMER May 2018)

- A. Corneal dystrophy
- B. Keratoconus
- C. Pathological myopia
- D. Keratoglobus

CORNEAL COLLAGEN CROSSLINKING WITH RIBOFLAVIN (C3R) /CXL



- Increases corneal stiffness by induction of cross links within the matrix
- By Ultraviolet radiation after saturation with riboflavin (photosensitizer)
- Minimum 400 microns recommended
- Riboflavin shields endothelium, lens, retina



Corneal collagen Crosslinking with Riboflavin (C3R) /CXL

INTACS



- Intrastromal corneal ring segments
- 2 PMMA ring segments
- Flatten cornea by shortening arc length: VA ↑



INTACS

Indications

- Progressive deterioration of vision
- Clear central cornea
- Corneal thickness > 450 microns at site of entry
- Delay/eliminate keratoplasty





- Q. A young female patient presented with the complains of blurring of vision, diplopia and photophobia for last three months. She told to the doctor that she changed the glasses ten times last year. On further examination, the doctor noticed that there is conical protrusion of the cornea with central thinning and the apex of the cone is usually directed inferionasally. Which of the following is **incorrect** about this condition?
- A. Earliest clinical sign is Scissor reflex on retinoscopy
- B. Bow tie pattern on corneal topography
- C. Usually has regular astigmatism
- D. Associated with Marfan's syndrome

Answer: C

Solution

Keratoconus usually have irregular astigmatism KERATOCONUS

- Non -Inflammatory corneal ectasia characterized by central/paracentral corneal thinning
- Young male with constantly changing power of spectacles with myopia & high cylinder

SIGNS IN KERATOCONUS

- Scissor reflex on retinoscopy (Earliest Clinical Sign)
- Munson sign notching of lower lid due to the corneal bulge noticed in downgaze
- Enlarged corneal nerves
- Oil droplet reflex central area of corneal bulge visible in retro-illumination as an oil droplet against an illuminated retinal glow during slit lamp examination or on retinoscopy
- Rizutti sign light is shown from the temporal side (as in examining the anterior chamber depth), due to the conical shape of the cornea the light is focused at a point on the nasal limbus giving the light an arrow shaped reflex
- Acute Hydrops aqueous accumulated in stroma due to micro breaks in the corneal endothelium due to progressive corneal thinning. Cause of sudden painful defective vision with keratoconus
- Fleischer ring Partial or complete iron deposition ring in deep epithelium encircling the base of the cone in keratoconus. Characteristic in eyes with keratoconus.
- Vogt's Striae vertically oriented deep stromal lines (pre-Descemet's level) indicating corneal thinning and weakening which disappear on corneal pressure

Keratometry

- Steep cornea
- Irregular corneal astigmatism
- Bow tie pattern

Corneal Pachymetry

Paracentral thinning of cornea

Investigation of Choice

Pentacam (automated corneal topography)

Important Associations of Keratoconus

- Marfans
- Leber congenital amaurosis
- Vernal kerato conjunctivitis
- Down's syndrome
- Atopy
- Floppy eyelid syndrome
- Ehler Danlos
- Achondroplasia
- Alagille syndrome
- Apert syndrome

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, 2020, Chapter 7 - Cornea, Page 248

- Q. A patient came to OPD with intense watering, pain and photophobia. She was a regular contact lens user and she forgot to remove contact lens in the night. On examination there was an epithelial defect. What is first line of treatment?
- A. Discontinue contact lens use immediately
- B. Start fortified antibiotic steroids treatment
- C. Normal saline irrigation
- D. Wait and watch

Answer: A

Solution

Patient presents with overnight use of contact lenses not intended for such use and symptoms suggestive of corneal involvement. Commonest complication with such use is development of corneal abrasion (epithelial defect) which can be potentially complicated by secondary corneal infections.

Corneal abrasions are potentially serious complications and must not be ignored. Such cases need to be carefully evaluated to rule out any corneal infiltrate and ensure no contact lens induced keratitis has set in.

Contact Lens Over-wear Syndrome:

Treatment

- 1. Immediately Discontinue contact lens use
- 2. Frequent antibiotic eye drops or ointment use (preservative free) 2 hourly commonly used are tobramycin, Bacitracin/Polymxyin or newer Fluoroquinolones (Moxifloxacin)
- 3. Follow up within 1 day or sooner if symptoms worsen
- 4. Topical cycloplegics and Oral NSAID to relieve pain
- 5. Patching is usually avoided so as to prevent Serious Pseudomonas infections which can develop overnight chances of which increase with patching
- 6. Steroid eye drops are avoided as they can inhibit corneal epithelial repair mechanisms and thereby lead to non healing abrasions.

High Yield fact Infection

 Most common Organism in Contact Lens induced Keratitis: Pseudomonas Most specific Organism for Contact Lens associated Keratitis: Acanthamoeba

Reference: Cornea, Krachmer & Mannis, Elsevier, 3rdedition, 2011, Part VII - Diseases of the Cornea, Section 9 - Contact Lenses, Chapter 102 - Complications of Contact Lens Wear, page no.1232



LEARNING OBJECTIVES

TEPISCLERITIS AND DEVASTATING SCLERITIS

- Simple Episcleritis
- Nodular Episcleritis
- Anterior Scleritis
 - Diffuse scleritis
 - Nodular scleritis
 - Necrotising Scleritis
 - Necrotizing without inflammation
- Posterior Scleritis



15 SCLERITIS **EPISCLERITIS & DEVASTING**

SCLERA

- 00:00:20
- It is a tough, opaque outer layer, which extends from limbus to optic disc, and merges with dura of optic nerve
- Function
 - Maintains shape of eyeball,
 - Protection of eyeball and
 - Attachment of extra ocular muscles
- Thinnest part of sclera
 - Lies just behind the insertion of rectus muscles
- Thickest part of sclera
 - o Posterior pole
- There are 3 vascular plexi
 - Outermost (conjunctival plexus)
 - → Within conjunctiva
 - → No pattern
 - 2. Superficial episcleral
 - → Radial pattern
 - → Gives salmon pink appearance when inflamed
 - Deep Scleral plexus
 - → Criss cross pattern
 - → Violet appearance when inflamed

EPISCLERITIS

- 00:03:31
- Benign, Self-limited inflammation of episclera
- Types
- 1. Simple
- 2. Nodular
- 1. Simple Episcleritis
- Sectorial in distribution



Episcleritis (Simple)

- 2. Nodular Episcleritis
- Nodule present
- Elevated
- discrete area inflamed

Cause

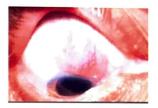
Idiopathic

Treatment

- Reassurance
- Topical/ oral NSAIDs

SCLERITIS





Scleritis

 Painful inflammation of Sclera often associated with systemic disorders

Types

- Anterior
 - o Inflammation present anterior to insertion of rectus muscles
- Posterior
 - Inflammation present posterior to insertion of rectus muscles

ANTERIOR SCLERITIS

00:06:35

- More common
- Types
 - Diffuse
 - Nodular
 - Necrotizing with inflammation
 - Necrotizing without inflammation (scleromalacia perforans)
- Clinical Features

- 00:07:02
- Systemic Autoimmune disorders
- MC association is Rheumatoid arthritis
- o More in women in their 50's & 60's
- o Pain
 - → Severe boring pain(characteristic)
 - → Exacerbated by movements
 - → Worse at night
- o Redness & globe tenderness
- Violet blue colour (Characteristic) with sclera edema & dilated vessels
- o Phenylephrine drops cannot blanch vessels
- Treatment
 - Topical steroids

- Oral NSAIDS
- Oral Steroids

Diffuse scleritis

- **Ö** 00:09:45
- · M/c and most benign form of anterior scleritis
- Widespread inflammation of anterior sclera
- Management
 - Topical steroids
 - Oral NSAIDS
 - Oral Steroids (1mg/kg body wt/day)



Diffuse scleritis

Nodular scleritis

- 2nd MC form of scleritis
- Nodules on sclera
 - One or more
 - Tender
 - o Inflamed
 - o Immobile
 - Erythematous
- Management
 - Topical steroids
 - Oral NSAIDS
 - Oral Steroids (1mg/kg body wt/day)

Necrotising Scleritis

O 00:11:37

00:10:14

- More severe & destructive form
- Least common
- May lead to loss of eye d/t perforation, may even cause loss of life
- Intense pain, out of proportion
- M/c rheumatoid arthritis, Wegner's granuloma
- White avascular areas surrounded by inflamed sclera
- Management
 - Systemic steroids
 - o Immunotherapy
 - Sclera grafting



Necrotising Scleritis

Necrotizing without inflammation



- Also known as scleromalacia perforans
- Rare, severe disease in elderly women with long staining rheumatoid arthritis
- Clinical signs
 - Inflammation
 - No Pain
 - No redness
 - Vision not affected
- Sclera thins, dark uveal tissue visible
- Staphyloma develops if IOP elevated, eyes rupture easily
- Management
 - Oral steroids
 - Cyclophosphamide
 - Methotrexate



Necrotizing without inflammation

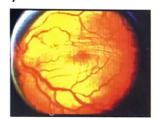
POSTERIOR SCLERITIS



- Difficult to diagnosis
- Rare, vision threatening scleral inflammation behind ora serrata
- Seen in elderly women
- M/c association with Rheumatoid arthritis, SLE
 Wegner's (granulomatosis with Polyangitis)

Clinical Features

- Pain
 - Moderate to deep
 - Boring pain
 - Wakes up the patient from sleep
- Tenderness
- Proptosis
- Visual loss &
- Restrictive motility



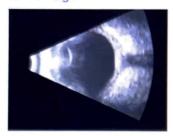
Posterior scleritis

Examination

- Choroidal folds
- Exudative retinal detachment
- Papilledema
- Angle closure glaucoma (ACG) without pupillary block

Diagnosis

B scan USG →T – sign



B. Scan showing T-sign

· OCT: thickening of choroid

Treatment

- Oral NSAID
- Systemic steroids (high dose)
- Immunosuppressants: Methotrexate/Mycophenolate
- Biologics: TNF Infliximab / Adalimumab

Episcleritis	Scleritis
No pain	 Painful
Bright red in colour	Violet blue in colour
No sclera edema	Scleral edema
Not a/w systemic disorders	• a/w RA, SLE etc
 On 10% phenylephrine drops vessels undergo blanching 	 On 10% phenylephrine drops vessels do not blanch



CLINICAL QUESTIONS



Q. A 45-year-old woman wakes up at 2 AM with deep boring pain. Past history reveals she has been suffering from SLE for the last year. B scan ultrasonography is shown below. Most likely diagnosis?



- A. Retinal detachment
- B. Posterior scleritis
- C. Staphyloma
- D. Anterior uveitis

Answer: B

Solution

B- scan shows T sign which is suggestive of posterior scleritis

Posterior scleritis:

- Inflammation with thickening of the posterior sclera may start primarily posteriorly or maybe an extension of anterior scleritis
- Clinical features include moderate to deep boring pain waking up the patient, tenderness, proptosis, visual loss and restricted motility.

Reference: Comprehensive ophthalmology 6th edition A K Khurana Pg 141

Q. A 25-year-old patient presents with localized redness in the right eye as shown below. On instillation of 10% phenylephrine, there is quick blanching of the vessels. What is the diagnosis?



- A. Angular conjunctivitis
- B. Nodular scleritis
- C. Nodular episcleritis
- D. Scleromalacia perforans

Answer: C

Solution

The image shows a purplish nodule on the medial epicanthus with surrounding injected blood vessels. They are not reaching the limbus though. This is a presentation of nodular episcleritis.

- Nodular episcleritis also tends to affect females but has a less acute onset and a more prolonged course than the simple variant.
- Symptoms: A red eye is typically first noted on waking. Over the next 2–3 days the area of redness enlarges and becomes more uncomfortable.
- Signs: Attacks usually clear without treatment, but tend to last longer than simple episcleritis.

Reference: ak khurana 7th edition pg 149



LEARNING OBJECTIVES

OCULAR TRAUMA

- Seven Rings Of Trauma
- Orbital Fracture
- Subconjunctival Haemorrhage
- Scleral Rupture
- Corneal Injuries
- Traumatic Hyphema
- Angle Recession Glaucoma
- Uveal and Lens Trauma
- Vitreous Trauma
 - Vitreous haemorrhage
- Retinal Trauma
 - Commotio retinae
 - Retinal dialysis
 - Retinal detachment
- Optic Nerve Injury
 - Traumatic optic neuropathy
 - o Traumatic ON avulsion

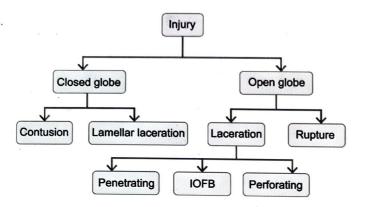


16

OCULAR TRAUMA

BIRMINGHAM EYE TRAUMA TERMINOLOGY SYSTEM





- Contusion
 - o Partial thickness injury
 - o Caused by blunt trauma
- Lamellar Laceration
 - o Partial thickness injury
 - Caused by a sharp object
- Laceration
 - Full thickness injury
 - o Caused by a sharp object
- Rupture
 - o Full thickness injury
 - o Caused by a blunt trauma

SEVEN RINGS OF TRAUMA



- Classically following a closed globe injury with blunt force
- Pathology
 - Intraocular fluids cannot compress → forcibly expand
 → disrupt normal architecture



Seven Rings of Trauma

Seven Rings

- In centre of iris /Sphincter pupillae → radial tears
- In periphery of Iris → Iris base tear → Iridodialysis → D-shape pupil
- Anterior CB trauma → Angle recession
- Longitudinal fibres of CB separate from scleral spur → Cyclodialysis cleft
- 5. Trabecular meshwork → TM tear
- 6. Zonules \rightarrow zonular dialysis \rightarrow subluxation / dislocation
- Retinal dialysis → disinsertion of retina from Ora serrata: superonasal (pathognomonic)

ORBITAL FRACTURE



- Orbital rim fractures
 - Due to direct impact to face
 - M/c cause steering wheel in car crash
- Great force required to damage thickened orbital rims, often extensive injuries to other facial bones
- Blowout fracture
 - M/c floor fracture is inferior wall
 - As sitting on hollow maxillary sinus
 - o Triad
 - → Diplopia
 - → Enophthalmos
 - → infraorbital anaesthesia
 - o Injury with size of impact > than orbital opening



Orbital Fracture (Inferior wall)



Previous Year's Questions

Q. A boy gets punched in the eye. Which of the following is the most likely occurrence? (FMGE June 2021)

- A. Subluxation of lens
- B. Inferior orbital wall fracture
- C. Medial wall fracture
- D. Lateral wall fracture

Join the Telegram Channel - https://t.me/prepladderlatestnotes

SUBCONJUNCTIVAL HAEMORRHAGE @ 00:08:27

Bleeding under conjunctiva



Causes

- Hypertension
- Trauma
- Valsalva manoeuvre
- Anti-coagulants
- Bleeding diathesis

Symptoms

- · Painless redness of eye
- Blood under conjunctiva of which posterior border can be seen

Treatment

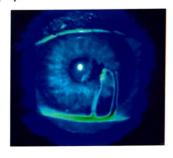
- Reassure patient
- Cold compress to reduce discomfort
- Clears within 5-10 days

SCLERA

(1) 00:10:18

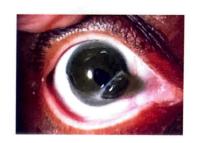
Scleral rupture

- Break in sclera due to internal pressure caused by blunt
 trauma
- Occur at weakest spots of sclera: Limbus, close to insertion of rectus muscles
- Often hidden by unbroken conjunctiva
- 360°subconjunctival haemorrhage/chemosis
- Flat/ 'Deflated 'looking AC with hypotony
- Teardrop pupil



Diagnosis

- Seidel's test
 - o Fluorescein dye applied to ocular surface
 - Any aqueous leak dilutes the dye allowing the leak to become obvious



CORNEAL INJURIES



- Partial thickness
 - Lamellar lacerations
- Full thickness
 - Penetrating injuries
 - o Perforating injuries: Positive Seidel's test
- Healing occurs by cell migration, proliferation, and differentiation
 - Leads to extracellular matrix remodelling
- Epithelial healing depends upon limbal stem cells

TRAUMATIC HYPHEMA

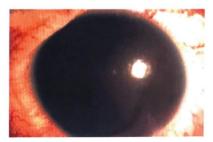


Accumulation of RBC's within AC



Traumatic Hyphema

- M/c cause is blunt trauma
- Stage IV hyphema
 - Total hyphema
 - Bright red blood
- · 8 Ball/Black ball hyphema
 - o Dark red-black blood
 - o Due to impaired aqueous circulation



Black ball hyphema

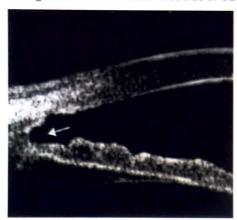
Treatment

- Topical steroids
- Cycloplegics
- Topical aqueous suppressants
- Tranexamic acid for preventing secondary haemorrhages

ANGLE RECESSION GLAUCOMA



 Blunt trauma forces aqueous posteriorly → causes tear between longitudinal and circular muscles of CB



Angle Recession Glaucoma

- Trabecular Meshwork damaged
 - Leads to slow obstruction of aqueous outflow
- Recession > 180° leads to glaucoma
- Traumatic hyphema ↑ risk of ARG
- Asymptomatic
 - As may take years †IOP
 - o At time of presentation IOP may be dramatically high
 - Presents with visual loss
 - Severe field loss

Diagnosis

- · Gonioscopy (key to diagnosis)
 - o Separation of fibres is seen

Treatment

- · Topical aqueous suppressants
 - o Topical beta blockers
 - Topical Alpha-2 agonists (Brimonidine)
 - o Topical Carbonic anhydrase inhibitors
- Prostaglandin analogues (Latanoprost)
- Miotics: Not useful

UVEAL TRAUMA

O00:20:50

· Iridodialyis: D shaped pupil



D shaped pupil

Sphincteric tears: Traumatic mydriasis



- Cyclodialysis cleft: CB separate from scleral spur creating direct communication between AC/ suprachoroidal space
- · High chances of developing Sympathetic ophthalmitis
- Choroid
 - Choroidal rupture
 - Break in choroid, Bruch's membrane, RPE
 - Yellowish white crescentic streak is seen concentric to optic disc

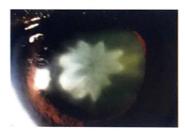


Yellowish white crescentic streak

LENS TRAUMA

Ö 00:23:06

- · Vossius ring: Imprint of iris pigment on anterior capsule
- Traumatic cataract: Rosette cataract



Subluxation / Dislocation of lens

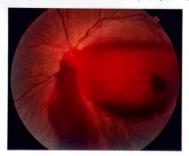


VITREOUS TRAUMA

Ö 00:24:23

- Vitreous haemorrhage
 - M/c cause of VH in young patients is Trauma

- o M/c cause of VH Overall is Diabetes mellitus
- Sudden painless loss of vision with floaters
- Vitreous base avulsion pathognomonic of blunt ocular trauma
- · Visible as bucket handle in retinal periphery



Vitreous haemorrhage

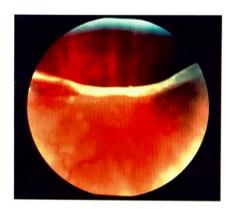
RETINAL TRAUMA

00:26:15

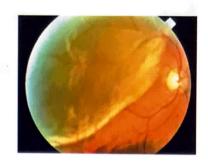
- Commotio retinae
 - o Concussive injury to retina
 - o If involves Fovea
 - → Bright red glow called cherry red spot is seen
 - → Edema around Fovea is called Berlin's edema



- Retinal dialysis
 - Separation of retina from its root
 - o Circumferential break at Ora serrata
 - → M/c site in traumatic cases is superonasal
 - → M/c site overall is Inferonasal



Retinal detachment



Macular holes

OPTIC NERVE INJURY

Ö 00:27:50

Traumatic optic neuropathy

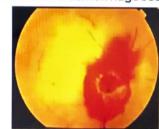
- M/c cause: Indirect injury
- M/c Part damaged: Intracanalicular ON
- Due to transmitted shock from orbital impact
- Max chances of injury due to impact at lateral 1/3rd and medial 2/3rd of eyebrow
- Clinical features
 - ↓ Vision
 - ↓ Colour vision
 - Field defects
 - o RAPD
- Initially Asymptomatic, 6-8 weeks Later optic disc atrophy occurs



Optic disc atrophy

Traumatic ON avulsion

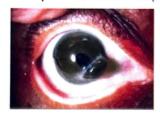
- Sudden trauma with rotation of globe
- Blows optic nerve off the sclera into dural sheath
- Sudden Loss of vision
 - No light perception (NLP)
- Diagnosis
 - o Clearly visible defect at site of optic disc
 - o Confirmed by B-scan USG
 - → Dense Vitreous haemorrhage seen





Previous Year's Questions

Q. A patient presents to Emergency with History of trauma to eyes while working with chisel and hammer. Which of the following investigation will be detrimental to the patient? (NEET Sep 2021)



- A. MRI orbit
- B. X-ray orbit
- C. CT- orbit
- D. B scan



Previous Year's Questions

- Q. Not true about epiblepharon is? (JIPMER Nov 2018)
- A. Seen in hypertelorism
- B. Congenital epicanthus
- C. Trauma to epicanthus can cause
- D. Fold of skin and conjunctiva turned inwards





- Q. A 19-year-old young boy was presented to the emergency department with bleeding eyes following a motor vehicle accident, where he lost control of the vehicle and crashed into a building under construction. The hospital nurse called the ophthalmologist immediately to look into the case. The ophthalmologist could diagnose this as a case of blunt trauma to the eye with all of the following signs except?
- A. Sphincter tear
- B. Angle recession
- C. Corneal perforation
- D. Retinal dialysis

Answer: C

Solution

BLUNT TRAUMA OF EYE

- Most common cause of blunt trauma sports injuries assault
- Causes anteroposterior compression of the globe and simultaneous expansion along the equatorial plane.
- Extent of ocular damage depends on the severity of trauma
- Cornea
 - o Corneal abrasion
 - o Acute corneal edema focal or diffuse dysfunction of the endothelium
 - o Descemet's membrane tears
- Hyphaema hemorrhage in the anterior chamber typically from iris root or ciliary body face, may be associated with raised IOP, corneal staining, ischemic optic neuropathy
- Anterior uvea
 - Pupil Transient miosis, Vossius Ring (an imprint of pigment from the pupillary border on Anterior lens surface),
 Traumatic mydriasis (secondary to sphincter tears), D shaped pupil
 - o Iris Sphincter tear, Iridodialysis
 - o Ciliary body Angle Recession, Cyclodialysis (separation of the ciliary body from scleral spur)
 - o Trabecular meshwork tear
 - o Traumatic Uveitis
- Intraocular Pressure
 - o Transient IOP rise due to injury per se, sustained IOP rise due to hyphaema, inflammation
 - Hypotony due to Ciliary body injury and shut down
- Lens
 - Cataract Rosette Cataract
 - o Subluxation Zonular dehiscence when partial causes subluxation
 - Dislocation Complete zonular dehiscence
- Globe Rupture in severe blunt trauma, commonly anterior (in the vicinity of Schlemm's canal) with prolapse of intraocular structures
- Vitreous hemorrhage associated posterior vitreous detachment, retinal breaks
- Commotio retinae concussion of neurosensory retina, Berlin's edema (if macular is involved)
- Choroidal rupture
- Retinal breaks and retinal detachment
 - o Retinal dialysis retinal break at ora serrata most commonly in superonasal > inferotemporal quadrants

- o Equatorial breaks direct retinal disruption at the point of scleral impact
- Giant Retinal Tear
- Retinal Detachment
- Macular hole
- Optic Nerve Injury
 - Traumatic Optic Neuropathy
 - o Optic Nerve Avulsion

Reference: Kanski's Clinical Ophthalmology - A Systematic Approach, 9th Edition, Chapter 22 - Trauma, page 899-907.

Q. A 39-year-old man was brought to the hospital after an alleged workplace accident. Initial assessment revealed only mild chest injury and mild confusion, with no other injury. His vision was unaffected with no RAPD. A CT brain was performed to rule out brain injury but revealed an incidental finding of a foreign body in the left intravitreal cavity. This foreign body can be left in the eye without any intervention if it is:

A. Iron

B. Copper

C. Lead

D. Nickel

Answer: C

Solution

Lead is an inert material that can be left alone in the eye without any intervention.

Else all intraocular foreign bodies should ideally be removed surgically as the injury itself may induce damage as well as long term chemical toxicity

INTRAOCULAR FOREIGN BODIES

They can be classified into the following types.

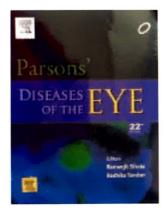
1. Frequently produce severe inflammatory reactions

- · Magnetic: Iron, steel, tin
- Non magnetic: Copper and vegetable matter
- Typically produce mild inflammatory reactions
- Magnetic: Nickel
- Nonmagnetic: Aluminium, mercury, zinc, vegetable matter.

2. Inert foreign bodies

· Carbon, coal, glass, gold, lead, stone

Reference:







CHEMICAL INJURIES

- · Acid burns
- Alkali burns
- Management of chemical injuries
- · Mechanism of development of glaucoma after chemical injury



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CHEMICAL INJURIES

CHEMICAL INJURIES

(3) 00:04:47

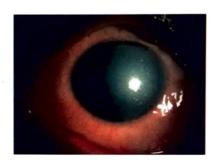
- True ocular emergency
- Two types
- Acid
- Alkali
- Acid to alkali burns ranges from 1:1 to 1:4
- Alkali burns are four times more common than Acid burns.



Chemical Injury

ACID BURNS

- 00:01:46
- · Less destructive than alkali
- Acids coagulates Proteins → Precipitation of proteins → forms barrier preventing penetration
 - o Exception: Hydro fluoric acid,
 - o Fluoride ion penetrates
- Damaged by acids pH < 4
- Sources
 - Car batteries, toilet bowl cleaners, swimming pool additives
- Ground Glass appearance



Acid Burn

Mp

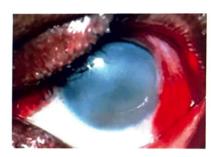
Important Information

- M/c acid injury are Sulphuric acid injury followed by hydrochloric acid
- Most severe injury is by Hydrofluoric acid

ALKALI BURNS



- Alkali is lipophilic and penetrates membranes through saponification of membrane lipids
- Hydroxyl ions denature collagen matrix of cornea
- Tissue undergoes liquefactive necrosis, triggers release of proteolytic enzymes, Cascading damage
- · Alkalis can reach AC in 15 seconds



Alkali Burn

Examples

- Detergents
- Ammonia
- Bleach
- lime



DUA CLASSIFICATION

Grad e	Prognosis	Clock hours of limbal involve ment	Conjun ctival involve ment	Analog scale
1.	Very good	0	0%	0/0%
II	Good	3	< 30%	0.1 – 3/1 – 29.9%
III	Good	> 3 - 6	> 30 – 50%	3.1 – 6%/31 – 50%
IV	Good to guarded	>6-9	> 50 – 75%	6.1 – 9/51- 75%
٧	Guarded to poor`	>9 - < 12	75 - < 100%	9.1 – 11.9/ 75.7 – 99.9%
VI	Very poor	12	100%	12/100%



Limbal Ischaemia

MANAGEMENT OF CHEMICAL INJURIES



Emergency treatment

- Copious Irrigation, 1-2 L over 30-40 minutes delivered through IV tube
- Stop when pH 7.0
- Examine sac up to fornices by double eversion
- Removal of residual chemical debris,
- Excise devitalized tissue

Recommended treatment

- Based grade of injury
 - o Topical antibiotic Fluoroguinolones
 - Cycloplegic Homatropine/Atropine
 - o Topical steroids: prednisolone
 - → Given for first 10 days
 - → Tapered by day 14, to minimise corneal melting
 - Doxycycline inhibits MMP 100 mg a day
 - o Anti-glaucoma drugs
 - o Topical 10% ascorbate and oral 2 grams/day
 - o Tear supplements: Preservative free
- Sodium citrate
 - o 10% drops 4-6 times a day
 - o Inhibits polymorphonuclear proteases

Additional treatment

- · Punctal plugs if tear film inadequate
- Lid taping to reduce exposure
- Ring conformer to prevent symble pharon formation
- Acetylcysteine
- Autologous serum drops 20% 6 times a day

MECHANISM OF GLAUCOMA



- Most important preventable complication (almost 75%)
- Most important for visual outcomes
- Acute rise
 - o Collagen shrinkage and contraction
 - o Increase in uveal and episcleral flow
- Long term
 - Trabeculitis
 - PAS
 - Steroid induced



CLINICAL QUESTIONS



- Q. A 19-year-old girl who was a victim of an acid attack is brought to the ER for management. After initial stabilization, the damages were evaluated and there was extensive ocular damage. Which of the following facts is false about acid injury to the eye?
- A. Makes a barrier and prevent deeper penetration
- B. More destructive than alkali injuries
- C. Steroids are used to control inflammation
- D. Glaucoma is the most preventable complication following the acid injury

Answer: B

Solution

Chemical injuries of the eye may be divided into 2 types

- Acid injuries (Burns)- less destructive → coagulate and precipitate proteins → create a barrier and prevent further penetration (Except- Hydrofluoric acid → fluoride ions penetrate into deep tissues → more damage)
- Alkali injuries- severe, they are lipophilic → saponification of membrane lipids → tissues undergo liquefactive necrosis +
 release of proteolytic enzymes → damage

Mx

- Immediate Irrigation with normal saline (even water if normal saline is not available) for a minimum of 30 mins, pH
 of conjunctiva must be 7-7.2.
- Chemical debris can be removed by double eversion

Rx

- Topical antibiotics (Grade I-III injuries- erythromycin/Grade IV- Moxi/Gatifloxacin)
- Cycloplegics (atropine/homatropine)
- Topical steroids (prednisolone acetate 10days, tapered by 14 days based on clinical response)
- Doxycycline (-) MMP
- Antiglaucoma drugs to avoid secondary glaucoma
- Vitamin C
- Tear supplements

Other Mx options - Autologous serum, Amniotic membrane transplantation (AMT), mucous membrane graft, Conjunctival flaps

Reference: Parson 22nd edition page 383





TIMAGING IN OPHTHALMOLOGY

- Fluorescein Angiography (FA)
- Indocyanine Green Angiography (ICG)
- Optical Coherence Tomography (OCT)
- Optical Coherence Tomography Angiography
- Anterior Segment Optical Coherence Tomography (ASOCT)
- Ultrasound Biomicoscopy (UBM)
- As-Oct Vs UBM
- Pentacam
- Confocal Microscopy



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IMAGING IN OPHTHALMOLOGY





FLUORESCEIN ANGIOGRAPHY (FA) & 00:00:40

- Used for study of circulation of retina and choroid in normal and diseased states
- Black and White photographs taken after IV injection of 10% sodium fluorescein



Fluorescein Angiography

 80% fluorescein is albumin bound ,20% is unbound and circulates in vasculature of retina and choroid, can be visualised

Procedure

- 10% Na Fluorescein dye is injected
- Through antecubital vein
- Dye reaches to eye via ophthalmic artery to short posterior ciliary arteries in 8-10second (arm-retinal circulation time)
- Dye fills up retinal capillaries and vessels
- Photographs are taken by fundus camera



Fluorescein Angiography: Procedure



Important Information

Arm-Retinal circulation time is 8 - 10 seconds

Phases

- Pre retinal
- Retinal
- Arteriovenous
- Venous
- Late recirculation

Pathology

- First choroidal vessels fill, then retinal vessels
- Dye leaks out of capillaries into retina when endothelium is damaged
- Dye leaks from choriocapillaries into interstitium when RPE is damaged
- In normal retina dye does not leak out
- Dark Choroid: Hallmark of "Stargardt's Disease"



Petalloid / Flower petal Appearance seen in cystoid macular edema





Previous Year's Questions

Q. A patient with hypertension and diabetes presents with blurred vision. Fluorescein angiography shows.

(AIIMS Nov 2019)

- A. Macular edema
- B. Sub macular edema
- C. Papilledema
- D. Pre macular hemorrhage

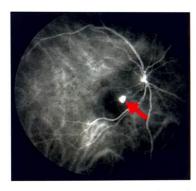
INDOCYANINE GREEN ANGIOGRAPHY (ICG)



- ICG is 98% protein bound
- Diffusion through fenestrations of choriocapillaries is limited
- Retention of ICG makes it ideal for imaging choroidal circulation
- Longer wavelength, fluoresces better through pigment, fluid, lipid and haemorrhage
- Detects abnormalities such as Choroid neovascular membranes (CNVM's) obscured by overlying haemorrhage, melanin, xanthophyll
- Occult CNVM s

Indications of ICG

- Occult CNVM's
- Polypoidal choroidal vasculopathy



Polypoidal choroidal vasculopathy

- Pigment Epithelial Detachments
- Serpiginous Choroidopathy
- Birdshot retinochoroidopathy
- Multiple evanescent white dot syndromes (MEWDS)

OPTICAL COHERENCE TOMOGRAPHY (OCT)

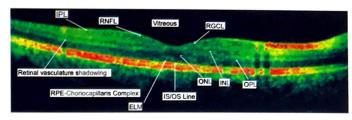


A non-invasive technique reveals cross sectional area



Optical Coherence Tomography

 Interferometry to create a cross sectional map of retina each layer of retina can be seen, and their thickness measured



Interferometry

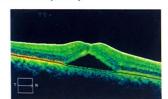
Accurate to 10–15 microns

Advantages

- Cross–sectional imaging
- Quantification in the form of thickness maps

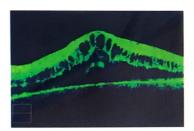
Indications of OCT

- Cystoid macular edema
- Macular pucker
- · Central serous retinopathy



Central serous retinopathy

- Vitreo macular traction
- Macular holes



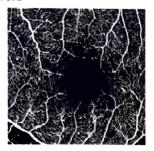
Cystoid macular edema

Glaucoma

OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY

Ö 00:10:35

 Non-invasive technique imaging microvasculature of retina and choroid



OCT Angiography

 Laser light reflectance from surface of moving RBC's to accurately depict vessels, eliminating intravascular dyes

Advantages of OCTA

- Non-invasive
- Image acquisition speed faster
- Image detail and resolution much better

Indications of optical coherence tomography angiography

- Diabetic Retinopathy
- Dry ARMD
- Wet ARMD
- CSR
- Vascular occlusions

ANTERIOR SEGMENT OPTICAL O0:12:06 COHERENCE TOMOGRAPHY (ASOCT)

- ASOCT uses higher wavelength of light than posterior segment OCT
- · Greater absorption and less penetration
- Anterior segment structures (cornea, AC, iris, angle, lens)
 can be seen



Anterior Segment OCT

Applications of ASOCT

- Angle anatomy, particularly angle occludability and closure
- Plateau iris

- Ciliary body tumours and cysts
- Corneal thickness measurements
- Keratoconus

ULTRASOUND BIOMICOSCOPY (UBM)

Ö 00:13:13

- Non-invasive technique for imaging anterior segment using high frequency ,50 MHZ
- Depth of tissue structures determined by measuring time delay of returning ultrasound signal
- Requires contact with eye and a coupling media necessary
- scanning performed through immersion bath



Ultrasound Biomicoscopy

 Tissue depth penetration approximately 5 mm, can view through opaque media, unlike OCT

Clinical applications of UBM

- All structures up to lens can be seen
- AC anatomy and pathology
- Angle closure glaucoma



Angle closure glaucoma

- Corneal pathology
 - o Keratoconus
 - Dystrophies
 - o Scars

AS-OCT VS UBM



Anterior Segment Optical Coherence Tomography (AS-OCT)

Ultrasound Biomicoscopy (UBM)

- Non-contact
- Requires contact and a liquid coupling medium
- Does not require a skilled operator
- Requires skilled operator
- Higher axial resolution
- Lower axial resolution
- Limited ability to visualize structures posterior to the iris pigment epithelium
- Can visualize structures posterior to the iris pigment epithelium
- Faster acquisition time
- Slower acquisition time
- Wider field of view
- Smaller filled of view
- Seated upright position
- Seated upright or supine positions
- Use for clear corneas
- Can image through opaque corneas

PENTACAM



- Rotating Schiempflug camera takes 2 seconds to generate complete image of anterior segment
- Second camera detects eye movements and corrects it → 3 D model generated using 25000 elevation points



Pentacam

Clinical applications of Pentacam

- Screening for corneal ectatic disorders
- Refractive surgery screening
- Phakic IOL implantation
- Lens densitometry
- Improved IOL calculations

CONFOCAL MICROSCOPY



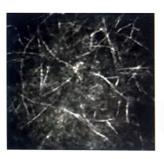
 Non-invasive technique allowing in vivo visualization of entire corneal thickness



Confocal Microscopy

Indications

- Diagnosis and treatment of Microbial keratitis
- Diagnosis of
 - Hyphae in Fungal keratitis



Hyphae in Fungal keratitis

- Cysts/Trophozoites in Acanthamoeba
- o Langerhans cells activation in viral
- o Endothelial disorders
- Fuchs Endothelial Dystrophy
- Neurotrophic keratopathy



Important Information

Confocal Microscopy is only used for cornea





Q. A 27-year-old male carpenter presented to ER with complaints of redness in the left eye after being hit by a flying iron chip while working on an iron statue. On examination, there was a puncture in the anterior capsule with focal anterior subcapsular opacity and a temporal intralenticular metallic foreign body found. Which of the following investigation is contraindicated in this case?

A. CT Scan

B. ERG

C. B mode ultrasound

D. MRI

Answer: D

Solution

INVESTIGATIONS IN CASE OF INTRAOCULAR FOREIGN BODY (IOFB):

Clinical Examination

 The most important part of examination in a suspected case of IOFB is direct visualization of the IOFB using a slit lamp, gonioscopy and indirect ophthalmoscopy

Radiological Examination

- X-ray orbit can help to confirm the presence of IOFB
- Ultrasound gives a general idea of the presence and relative position of IOFB especially in patients with hazy media
 - → B mode Ultrasonography
 - → UBM (Ultrasound Biomicroscopy) for obscured IOFB hidden in the anterior chamber or angle
- o Metal locators replaced largely due to advances in CT Scan
- CT Scan can help in accurate localization of the IOFB (radio-opaque). Details of radio-lucent IOFB may not be very clearly made out
- MRI Scan -MRI is contraindicated in Metallic intraocular foreign body due to its magnetic field
 - → The strong static magnetic field (B0) of MRI scanners can attract and accelerate ferromagnetic objects toward the centre of the machine and turn them into dangerous projectiles.
 - → This magnetic field can also displace implants or affect the function of devices (pacemakers and pumps), Metallic Implants
 - → It May be useful once a metallic FB has been ruled out i.e. Nonmetallic IOFB which give a low signal on T1 and T2 weighted imaging
- · Electrophysiology in patients with an unexplained visual loss with a history of trauma
 - Electroretinogram (ERG) very sensitive in the diagnosis of metallosis bulbi which may be characterized by a decrease in b wave amplitude and complete flattening of ERG in advanced cases

Reference: Eye Trauma, Shingleton, Mosby, 1991, Part 3 - Posterior Segment Trauma, Chapter 21 - Posterior Segment Intraocular Foreign Bodies, page 227.



LEARNING OBJECTIVES

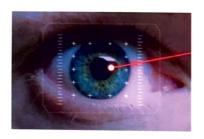
LASERS IN OPHTHALMOLOGY

- Diagnostic Applications of Laser
- Therapeutic Applications of Laser
- Types of Interactions of Laser with Tissue
 - Photocoagulation
 - Photochemical
 - Photovaporization
 - Photodisruption



LASERS IN OPHTHALMOLOGY

- Light Amplification by Stimulated Emission of Radiation
- Ophthalmology is first medical speciality to utilize laser energy in patient treatment
- Uses
 - Diagnostic applications
 - Therapeutic applications



DIAGNOSTIC APPLICATIONS

- 00:01:13
- Scanning laser ophthalmoscopy
- Fundus camera
- Angiography (FA and ICG)
- Scanning laser tomography
- Corneal topography
- Autofluorescence
- OCT
- Anterior segment
- Posterior segment

THERAPEUTIC APPLICATIONS

00:01:48

- Cornea
 - Refractive
 - o Non refractive PTK
- Lens
 - Capsulotomy
 - Femto laser assisted cataract surgery
- Glaucoma
 - Laser iridotomy
 - o Trabeculoplasty (ALT, SLT)
 - Iridoplasty
 - Cyclophotocoagulation
- Retina
 - Photocoagulation
 - o Photo dynamic therapy
- Oculoplasty
 - Aesthetic laser treatment

LASER TISSUE INTERACTIONS **Photocoagulation**



- Light absorption → tissue → ↑ temperature → denaturation of proteins
- Absorbed by melanin in RPE and choroid
- DF Nd YAG (532 nm):
 - Pan retinal photocoagulation
 - Used in
 - → PDR
 - → NVG
- Argon laser for Laser Trabeculoplasty
 - POAG
 - PXF glaucoma
 - Pigment dispersion
- Selective laser trabeculoplasty (SLT)
 - More targeted
 - Less energy

Photochemical



- 1. Photoablation
- High energy laser wavelengths
- Break long chain tissue polymers into smaller ones
- Excimer refractive surgery: PRK, LASIK
- Argon Fluoride, 193 nm
- 2. Photoradiation

IV photosensitizing agent taken up by target tissue

sensitization of target tissue,

exposure of sensitized tissue to laser light

cytotoxic free radicals - Photodynamic therapy

Photovaporization



- Laser light absorbed by tissue → vaporization of intracellular and extracellular water
- Advantage:
 - Seals adjacent blood vessels

bloodless surgical field

- CO2 laser vaporizes lymphangiomas and capillary hemangiomas
- o Haemostasis in bleeding disorders

Photodisruption

High energy laser strips electrons from molecules of tissue →expanding rapidly causing acoustic shock wave disrupts tissue

Join the Telegram Channel - https://t.me/prepladderlatestnotes

- Nd YAG laser, 1064 nm
 - Nd Neodymium
 - Y-Yitrium
 - A Aluminium
 - G-Garnet
- Uses
 - Laser capsulotomy for PCO
 - o Laser iridotomy for angle closure glaucoma



Important Information

 Femtosecond Laser 1053 nm (Photo disrupter) is used for Femto laser assisted cataract surgery (FLACS)

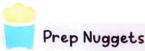


Previous Year's Questions

- Q. A 55-year-old man has undergone LASIK for myopia. What is the best method to check for IOL power calculation? (AIIMS June 2020)
- A. Hoffer
- B. Haigis
- C SRKI
- D. SRK 2

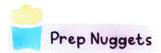


PREP NUGGETS



Abirtmass tree contract

Prep Nuggets		
Argyll Robertson pupil	Adie's pupil	
Prep Nuggets		
Lesion	Field defect	
Optic chiasma		
	No ocular blindness	
Optic tract		
LGB		
ACT TO SERVICE AND ACT OF THE SERVICE AND ACT	Mocularspaning	
Prep Nuggets		
Snow Hake contract		
	Galactosemia	
	Wilson's disease	



Granular	Lattice	Macular
	AD	
Crumbs		
Spares limbus		
	Good	