Eyelids

What is a chalazion and how does it form?

-chronic sterile lipogranuloma of the meibomian gland> obstruction of outflow of gland, accumulation of sebaceous secretion in acini.
-chronic infla reaction with epithelioid histiocytes and giant cells, plasma cells, lymphocytes.

What is the presentation and exam findings of a chalazion?

-MC in upper eyelid
-painless increasing swelling without any erythema on eyelid
-palpable small nodule with red/purple conjunctiva below
-if v big then blurred vision b/c of astigmatism induced by corneal compression
-can also cause mechanical ptosis

What is the fate of a chalazion?

1.Spontaneous resolution
2.liquefy to become meibomian cyst
3.fibrosed and calcify into hard nodule
4.infected and pus
5.burst and present as fleshy mass in conjunctiva

What is the TX for a chalazion?

-Warm compress
-topical steroids if bad> if worse then corticosteroid injection + antibiotics
-Surgical excision if permanent

What is a hordeolum?

Infectious nodule that forms in the eyelid due to staph A. infection

Hordeolum Internum vs externum?

-Externum= ant part of eyelid in gland of zeis in lash follicle -> stye
-Internum= post eyelid in meibomian gland

What is the presentation of a hordeolum?

-Pain+ tender eyelid
-red and oedema in eyelid
-pus coming out on margin or internally can burst through conjunctiva

What are the complications of a hordeolum?

1. Preseptal cellulitis
2. orbital cellulitis
3. cavernous sinus thrombosis

What is the Tx for a hordeolum?

-analgesic, systemic antibiotic
-warm compress
-pus evacuation
-clean lash line regularly

What is blepharitis?

Chronic infla of the eyelid margins

Anterior vs posterior blepharitis

-Ant= Base of eyelashes due to squamous or ulcerative causes
-Post= meibomitis

What is ulcerative blepharitis?

-Acute and chronic infection of lash follicles and associated glands of zeis and moll
-s. aureus and epidermitis

What is the clinical presentation of ulcerative blepharitis?

Red eyelid margins + oedema
yellow pus and crust
matted eyelash in severe cases

What are the eyelid, conjunctival and corneal complications of ulcerative blepharitis?

1. Eyelid: Trichiasis, madarosis (no eyelash), Poliosis (white eyelash), stye + chalazion, ptosis
2. conjunctiva: recurrent bacterial conjunctivitis
3. cornea: (due to hypers to staph exotoxin) keratitis, ulcer, tear film instability

What is the Tx for ulcerative blepharitis?

-Eyelid hygiene
-antibiotic ointment
-topical steroids
-artificial tears

What is squamous blepharitis?

Disorder of the glands of zeis and moll causing eyelid infla

What are the clinical features of squamous blepharitis?

-White dandruff on margins
-shiny waxy eyelids
-greasy eyelashes, red eyelid with oedema in -
-severe cases
-tx is selenium sulphide shampoo

What are causes of pseudoptosis?

-endophthalmitis
-microphthalmia

What are the 3 causes of congenital ptosis?

1. Simple congenital: usually unilateral, absent eyelid crease, reduced levator function
2. Jaw-winking ptosis: retraction of ptotic eyelid with jaw movement due to messed up innervation between oculomotor and V3 of trigeminal. synkinetic syndrome
3. blepharophimosis syndrome

What are the causes of neurogenic ptosis?

-CN3 palsy
-Horner's syndrome

What are the myogenic causes of ptosis?

-myasthenia gravis
-thyroid ophthalmopathy
-myotonic dystrophy

What are the aponeurotic causes of ptosis?

-Damage to levator aponeurosis
-Trauma or post op

What causes mechanical ptosis?

-Due to increased weight of upper eyelid caused by weight of a lesion
-eyelid tumour or heavy chalazion

What are the clinical features of ptosis?

-cosmetic disfigurement
-amblyopia
-backward tilt of head
-wrinkling of upper forehead to try to bring lid up

Lacrimal System

What causes congenital nasolacrimal duct obs?

-non-canalization of mem at the end of the nasolacrimal duct
-maldevelopment of punctum and canaliculi

What is the presentation of a child with congenital nasolac duct obs?

-Bilateral mostly, epiphora
-come by 3-4 weeks of age
-watery eyes, sticky mucoid discharge that accumulates in medial canthus
-positive regurgitation test

What are the complications of a congenital nasolac duct obs?

mucocele, conjunctivitis, acute/chronic dacryocystitis

What is the conservative and surgical tx for the above?

-Conservative: spontaneous patency by 6-9m, massage lacrimal sac to rupture mem with antibiotics to control infection
-Surgical: 1) probing under general anaesthesia with irrigation. 2) balloon catheter dilation of nasolacrimal duct. 3) Dacryocystorhinostomy if persists for too long

What is an amniotocele?

blue gray swelling of lacrimal sac due to collection of amniotic fluid

What are the causes of acquired nasolact obs?

idiopathic involution; stenosis in women
trauma- nasal fracture
infla disease eg sarcoidosis
tumour

What is the tx of acquired nasolac obs?

DCR or treat underlying condition

What is acute dacryocystistis?

infla of lacrimal sac by staph or strep after obs or just like that

What is the presentation of acute dacryocystitis?

-sub-acute onset redness/swelling at medial canthus, epiphora, tenderness, mucopurulent discharge may be present
-Untreated -> abscess formation

What are the complications of acute dacryocystitis?

preseptal cellulitis, external fistula formation

What is the tx for acute dacryocystitis?

antibiotics (systemic/topical), analgesics/anti-inflammatory, hot fomentation
Incision/draining of abscess
DO NOT do probing/irrigation

What is acute dacryoadenitis?

infla of the lacrimal gland usually due to EBV, CMV, mumps, staph, strep, gonococcus

What is the presentation of acute dacryoadenitis?

-pain in upper outer portion of eyelid
-red swollen tender eyelid, s shaped curve or outer eyelid
-downward displacement of eye
-pre auricular Ln swelling

What is the tx for acute dacryoadenitis?

Hot compress
analgesic
topical antibiotics
incision and drain if abscess

What is keratoconjunctivitis Sicca?

dec tear film production or increased evaporation leading to ocular surface disease

What are the causes of KCS?

1.Pure KCS: only lacrimal glands are involved
- congenital alacrima
- denervation hypo secretion (CNV ganglion surgery)
- idiopathic hypo secretion
2.Primary Sjogren syndrome: KCS + dry mouth
3.Secondary Sjogren syndrome: KSC + systemic disease; may be due to:
- trauma, infection (trachoma), inflammation (sarcoidosis, thyroid ophthalmopathy), hypersensitivity (SJS), autoimmune (SLE/RA), tumours of lacrimal gland, leukaemia/lymphoma

What is the presentation of KCS?

-irritation, burning, FB sensation which increases in wind/sun;
-Mucus discharge/transient blurring of vision; pain worse on blinking (corneal epithelial filaments)
-Thinning of tear film, mucus strands/debris in conjunctiva, punctate epithelial erosion, corneal
filaments

What are the diagnostic tests for KCS?

Tests:
- tear film break up time after blink <10 seconds. normal = 15-20s
- Rose-Bengal staining/fluorescein dye
- Schirmer test: filter paper on conjunctiva -> wetting <6mm

What is the Tx for KCS?

Treatment is tear substitutes (artificial tears)
- mucolytic agents
- topical retinoids
- reduction of tear drainage by occlusion of puncta
- surgery: transplant parotid -> conjunctiva

What are the causes of lacrimation?

Lacrimation= excess tear production
Psychic stimulation
FB
Inflammation
Acute rise in IOP
Abnormal regeneration after CNVII palsy - croc tear while eating
Pseudo bulbar palsy
Hyperthyroidism
Cholinergic stimulation

What is epiphora?

Watering of eye due to tear drainage obstruction

What are the causes of epiphora?

Obstructive epiphora: mechanical obstruction
- trauma
- punctal stenosis/canalicular atresia
- involutional stenosis of NL duct in elderly
- infection dacryocystitis
- chronic sinus disease
- tumour in NL sac/nose
- nasal polyp
Lacrimal pump failure: inability of pumping mechanism to drain tears
- lower lid laxity, CNVII palsy
- lower lid ectropion

How to diagnose epiphora?

Diagnosis: lacrimal system patency tests
- dye disappearance test of fluorescein dye after 5 min. retention in marginal eye strip is due to obs
- diagnostic probing/syringing
- Jones dye test (suspected partial obstruction)
- dacryocystography
- lacrimal scintillography

What is the jones dye test?

Jones dye test:
- primary: fluorescein dye -> eye -> recovery from nose
- secondary:
no recovery from nose -> irrigation w/ normal saline -> recovery of dye = partial obstruction
-no recovery of dye even after irrigation = canalicular obstruction/pump failure

Conjunctiva

What are the different parts of the conjunctiva?

Thin transparent membrane covering inner eyelid -> limbus
-Palpebral part: attached to inner eyelid
-Forniceal part: upper/lower fornix, freely moveable sac b/w eyelid and eyeball
-Bulbar part: covers eyeball -> limbus, freely moveable

What are the different layers of the conjunctiva?

1.Epithelium: non-keratinised squamous; 2-5 cells thick- goblet cells
2.Adenoid: fine connective tissue reticulum
3.Fibrous: collagenous meshwork; blood vessels, nerves, accessory lacrimal glands, Muller's muscle

What is the classification of conjunctivitis?

Inflammation of the conjunctiva

Discharge:

- serous: viral infection/toxic inflammation

- mucoid: vernal conjunctivitis/keratoconjunctivitis sicca

- mucopurulent: mild bacterial infection, chlamydia

- purulent: severe bacterial infection, gonococcal

Conjunctival reaction

Follicular

- viral: adenovirus, herpes simplex, picornavirus

- chlamydial infection

- medication: eserine, epinephrine

Papillary

- allergic: vernal, hay fever, atopic

- autoimmune: cicatricial pemphigoid, SJS

- chronic irritation: contacts, prosthesis, nylon sutures

- chronic squamous blepharitis

- medication: atropine

What is mucopurulent conjunctivitis (pink eye)?

Acute bacterial inflammation w/ mucopurulent discharge and red eye

What organisms cause pink eye?

staph/strep, Neisseria gonorrhea, Moraxella lacunata, H. influenzae, Proteus, Klebsiella

What is the clinical presentation of pink eye?

PC: acute onset redness, FB sensation, burning, discharge, eyelids glued together in morning

Signs:

- redness: mild fornix/palpebral portions -> severe whole conjunctiva

- mucopurulent exudates

- mild papillary reaction

- normal visual acuity

What is the tx for pink eye?

Treatment: irrigate conjunctiva, lid hygiene, topical antibiotics

- 60% resolve within 1 week w/o treatment

What is purulent (Gonococcal) conjunctivitis?

Purulent discharge and red eye caused by N.

gonorrhea

- infants infected during delivery, adult's genital infection -> eye

What is the clinical presentation of purulent conjunctivitis?

PC: acute onset, profuse/thick creamy purulent discharge, severe discomfort, pain w/ corneal involvement

Signs:

- oedematous eyelids

- periocular oedema/tenderness

- purulent discharge

- red/velvety conjunctiva

- chemosis of conjunctiva

- pre-auricular lymphadenopathy

- corneal ulcers may develop

What are the complications of purulent conjunctivitis?

corneal ulcers, iritis, iridocyclitis

What is the tx for purulent conjunctivitis?

hospitalize, conjunctival smear, irrigate eye, topical/systemic antibiotics, topical atropine w/ corneal involvement.

What is membranous conjunctivitis?

Membrane forms on the conjunctiva due to infection by Corynebacterium diphtheria

- inflammation of conjunctiva w/ fibrinous exudate -> membrane formation

- removal of membrane -> tearing of epithelium/bleeding

What is the presentation of membranous conjunctivitis?

- swelling of eyelids

- serous discharge

- whitish membrane on palpebral/forniceal conjunctiva

- enlarged pre-auricular lymph nodes

What are the complications of membranous conjunctivitis?

Corneal ulceration, paralysis of ciliary muscle, wound healing (scar formation) -> xerophthalmia, symblepharon, ankyloblepharon, entropion, trichiasis

What is the tx for membranous conjunctivitis?

irrigate, try peel of membrane, anti-toxin 4 x 10,000 units, topical/systemic antibiotic

What is epidemic keratoconjunctivitis?

Acute follicular conjunctivitis associated w/ superficial punctate keratitis

Caused by adenovirus 8, 19 -> highly infectious (epidemics)

Incubation = 8 days

What is the clinical presentation of epidemic KCS?

PC: acute onset watering, redness, discomfort, photophobia; B/L 60%

Signs:

- may have edematous lids

- watery discharge

- hyperemia of whole conjunctiva

- severe: chemosis, subconjunctival hemorrhage, pseudo-membrane

- follicles after few days in tarsal/forniceal conjunctiva

- keratitis

- lymphadenopathy: pre auricular/submandibular

Keratitis in 80% of cases 7-10 days after infection

- diffuse epithelial keratitis -> resolve in 2 weeks or -> discrete epithelial keratitis

- sub epithelial opacities due to immune response to virus -> persist months-years

How is epidemic KCS diagnosed?

clinical, immunofluorescence test (detect viral Ag), immunoglobulin

What is the Tx for epidemic KCS?

Symptomatic/supportive treatment: cold water

- topical antibiotics: reduce risk secondary infection

- topical steroids: uncomfortable eye/subepithelial corneal opacities

What is HSV conjunctivitis?

Follicular conjunctivitis following herpetic infection of eyelid/face skin

Usually w/ ocular involvement

What is the clinical presentation of HSV conjunctivitis?

- vesicles on eyelids

- watery discharge

- hyperaemia of whole conjunctiva

- chemosis/pseudo-membrane in small kids

- follicles in palpebral/forniceal conjunctiva

- punctate epithelial keratitis w/o corneal opacities-> resolve or -> superficial stroll opacities or multiple dendritic figures

Clinical, decreased corneal sensation, fluorescein Ab test, serum antibody titers

How can HSV conjunctivitis be diagnosed?

Clinical, decreased corneal sensation, fluorescein Ab test, serum antibody titers

How can HSV conjunctivitis be treated?

same as EKC except steroid contraindicated

What is vernal KCS?

Recurrent, B/L allergic inflammation of conjunctiva

HSR types I & IV

Seen in children 5-15 years, males > females, spring/summer, 2/3 patients have FH

Exogenous antigens -> IgE formation -> degranulation of mast cells -> inflammation

What is the clinical presentation of vernal KCS?

PC: itching, lacrimation, photophobia, redness, burning, FB sensation

- B/L and recurrent

Palpebral type:

- hyperaemia of conjunctiva

- diffuse papillary hypertrophy on upper tarsal conjunctiva

- cobble stone papillae

- giant papillae (cauliflower like)

- mucoid/sticky exudate: ropy secretion

- ptosis

Limbal form:

- hyperaemia/edema of limbal conjunctiva

- gelatinous papillae on limbal conjunctiva

- trantas dots: white superficial spots made of eosinophils around limbus

Mixed form: features of both

Corneal changes: punctate epithelial erosion

(Earliest finding)

- epithelial micro erosion/ulceration

- shield ulcers/plaque

- ring sub epithelial scarring

- peripheral superficial vascularization

What is the tx for vernal KCS?

- topical: antihistamines, NSAIDs, mast cell stabilizers, corticosteroids, acetylcysteine, cyclosporine

- supra tarsal injection: dexamethasone/ triamcinolone

- systemic: immunosuppressive agents, oral

antihistamines

What is atopic KCS?

Rare chronic B/L inflammation of conjunctiva associated w/ atopic dermatitis. HSR type I

Young males w/ atopic dermatitis, asthma

What is the presentation of atopic kcs?

Symptoms same as VKC except:

- minimal seasonal exacerbation

- micro-papillae over tarsal/inferior forniceal conjunctiva

- pale tarsal conjunctiva

- increased corneal vascularisation/opacification

- conjunctival scarring

How is atopic KCS treated?

same as VKC

What is a pterygium?

Degeneration of conjunctiva, triangular fibrovascular connective t covers cornea.

What are the causes of a pterygium?

-idiopathic

-Dryness of conjunctiva/UV damage of conjunctiva can result in disordered growth.

-Degeneration of corneal epi to stroma by invading pterygium.

What are the different types of pterygiums?

1. Progressive-> Thick fleshy vascular cap

2. stationary

3. Regressive-> Thin, atrophic with minimal invasion.

What are the parts and stages of a pterygium?

Parts = Cap, head body

Stage 1 = 2mm over cornea, asymptomatic

Stage 2 = 4 mm over cornea, astigmatism + messed up precorneal film

Stage 3 = More than 4 mm, obstructs vision

What is the clinical presentation of a pterygium?

-Ppl living in dry climates

-more on nasal side

-early is asymptomatic except looks bad to patient

-astigmatism + blindness later on, with diplopia

What is the tx for a pterygium?

Non-surgical= Artificial tears, topical steroids, sunglasses

Surgical= Simple excision (high rate of recurrence) or Simple conjunctival flap excision with covering of bare conjunctiva.

What are the post op complications of a pterygium?

1.recurrence

2.pyogenic granuloma

How can recurrence of a pterygiums be prevented?

-conjunctival autograft or amniotic mem transplant

-mitomycin c after surgery

-radiation or Argon laser

-peripheral lamellar keratoplasty for deep lesions

What is a Pinguecula?

Yellow white mass on bulbar conjunctiva near limbus

-dryness or UV damage of subepithelial conjunctiva.

-B/L, more common in old ppl, non-vascular, can transform into pterygium

What is the tx for a pinguecula?

No tx really required. Surgical excision if cosmetic issues.

What are the causes of a subconjunctival hemm?

1.idiopathic

2.post-op

3.eye or head trauma

4.blood clot disorders/aspirin + warfarin use

5.severe HTN

6. scurvy

What is the presentation of a subconjunctival hemm?

Benign condition, blood shot eye

How can a subconjunctival hemm be treated?

Resolves in 1-2 weeks by itself, artificial tears

Cornea

What is the anterior and posterior diameter of the cornea?

11.5mm vertical 12 mm horizontal
11.7 mm posterior

What is the shape of the cornea?

Central = spherical, peripheral = flat

What is the thickness of the cornea?

Central = 0.50-0.54mm
peripheral = 0.65-1.00mm

What is the refractive index and power of the cornea?

1.376, +43 diopters

What are the 5 layers of the cornea?

Epithelium, Bowmans, stroma, Descemet's, endothelium

What are the functions of the 5 layers?

-Epi- Basal, wing, surface, limbal cells > help replace corneal epi
-Bowman- Condensed collagen fibrils for strength
-Stroma- Collagen, fibroblasts, keratocyte's> maintain thickness
-Descemet's- basement mem of endo cells
-Endothelium- Single layer of cells that maintains dehydration of cornea
only epi can regenerate

Where does the cornea get its nutrition from?

Anterior - tears
posterior- aq humor

What are the most common causes of bacterial corneal ulcers?

S. aureus, S. pnuemo, moraxella, pseudo, klebby, proteus

What are the 4 pathogenic stages of bacterial corneal ulcers?

1. infiltrative: Injury to epi> neutrophils and m0 enter, whitish yellow opacity
2. Active stage: necrosis and sloughing of epi> ulcer with stromal oedema, grey-white swollen hazy cornea, hypopyon
3. Regressive stage: line of wbc surrounding the ulcer with rest of cornea clear
4. cicatrisation: healing with progressive epithelialization of ulcer with scarring of cornea

What are the symptoms of a bacterial corneal ulcer?

pain, blurred vision, lacrimation, photophobia, redness, halos

What is seen on examination of a bacterial corneal ulcer?

-purulent discharge, reduced vision

-Hazy cornea with ulcerated part taking up fluorescein

-hypopyon, iridocyclitis, raised IOP

How can the microorganism from a corneal ulcer be identified?

Corneal scraping under anesthesia for culture and gram stain

What is the treatment for an early bacterial corneal ulcer?

1. Control infection via topical antibiotics, ointment, subconjunctival injection
2. manage pain with nsaids, cycloplegic drugs to relive ciliary spasm and prevent post synechiae, antiglaucoma drugs

What is a descemetocele and how do you treat it?

-complete stromal erosion to expose Descemet's mem allowing it to bulge out b/c of ulcer
-Tx: Lower iop by anti- glauc drugs, pressure bandage or contact lens for support, conjunctival flap, amniotic mem, corneal graft

How do you treat a perforated ulcer?

intense topical antibiotics, anti-collagenase, pressure pad, conjunctival flap, amniotic mem, graft keratoplasty

Why are steroids not used to treat cornea ulcers?

it reduces infla but also inhibits healing by inhibiting fibroblasts and may actually prolong infection

What are the complications of a perforated ulcer?

Iris prolapse
subluxation of lens
cataract formation
endophthalmitis
IO hemm
retinal detachment
anterior synechiae
2* glaucoma
anterior uveitis

What are the different types of corneal scars that can form after an ulcer?

Nebula: thin insignificant corneal scar
macula: semi dense corneal opacity
leucoma: dense white corneal opacity
leucoma adherent
ectatic cornea: thin healed criticized cornea
pseudocornea

What are the risk factors for acanthamoeba keratitis?

contact lens, swim, ocular trauma

What is the presentation of acanthamoeba keratitis?

-blurred vision, lacrimation, pain out of prop from clinical findings
-Punctate epi keratitis
-Limbitis
-Ring abscess
-perineural infiltrates
-possible corneal melting

What is the Tx for Acanth keratitis?

debridement, topical amebicides or therapeutic keratoplasty

What is the most common cause of unilateral corneal scarring?

HSV keratitis

What is acute epithelial keratitis?

Corneal ulcer due to HSV with dendritic or ameboid/geographic pattern

What is the pathogenesis of acute epi keratitis?

Trigeminal reactivation> spreads down axons, damages corneal epi cells to cause early punctate lesions then dendritic pattern then geographic.

What are the clinical features of acute epi keratitis?

-FBS, Lacrimation, photophobia, pain, dec vision, reduced corneal sensitivity
ciliary congestion,

-Branched ulcer under fluorescein

What is the Tx for acute epi keratitis?

Topical acyclovir, debridement, topical antibiotic for secondary infection, atropine to relive pain b/c of ciliary spasm, oral antiviral for immunocompromised

What is herpes zoster ophthalmicus (HZO)?

Viral infection of trigeminal ophthalmic dermatome by HHV3 e.g., VZV or shingles

What is the clinical progression of HZO?

-Prodrome: facial pain in ophthalmic dermatome
-Skin Lesions Maculopapular rash> vesicles that crust over> Periorbital oedema and tenderness
-ocular lesions: Acute epithelial keratitis, micro dendritic ulcers, follicular conjunctivitis, episcleritis, sec glauc and ant uveitis
-Can eventually progress to optic neuritis

What is exposure keratopathy?

Keratopathy caused by incomplete closure of lids causing damage to cornea via desiccation and erosions that may become ulcers

What are the causes of exposure keratopathy?

bells palsy, thyroid eye, ectropion, blepharoplasty

What is the presentation of exp keratopathy?

-Dry eye
-punctate epithelial keratopathy lower 1/3 of cornea
-erosion and ulcer
-red eyes

How is exp keratopathy treated?

Artificial tears, Taping, correct underlying condition

What is keratoconus?

progressive disorder with central or paracentral corneal thinning causing apical protrusion of cornea and associated astigmatism

What is the pathogenesis of keratoconus?

defective synthesis of mucopolysachs and collagen> fragmentation of Bowmans mem> degeneration of stromal collagen> thinning> myopia and astigmatism

What are the symptoms of keratoconus?

-progressive near and far vision loss
-photophobia
-monocular diplopia
-hydrops in corneal stroma

What is seen on examination of keratoconus?

-Oil droplet on DD ophthalmoscopy
-Munson sign lower eyelid
-Scissor reflex on retinoscopy
-rizzuti sign
-prominent corneal nerves
-do corneal topography

What is the treatment for keratoconus?

-Cylindrical glasses
-Intacs: helps shape cornea back to normal
-collagen crosslinking (CXL): strengthen stroma
-Deep anterior lamellar keratoplasty: DALK for ppl who can't wear lens
-Keratoplasty: when there is significant corneal scarring

What are corneal dystrophies?

Slowly progressive non infla bilateral opacifications of cornea that are usually congenital or happen in early life

What is Fuchs endothelial dystrophy?

Hereditary congenital dystrophy more common in women with bilateral endothelial loss

What is the presentation of Fuchs endothelial dystrophy?

corneal oedema, corneal guttata, bullous keratopathy, corneal scar with blurred vision esp in the morning

Lens & Cataract

What is the average refractive index of the lens?

1.39 20-23 diopters

What are the 4 structures of the lens and their functions?

1.Capsule: Basement mem that holds the lens. is water permeable
2.Epithelium: Cuboidal cells on anterior surface that produces fibers throughout life
3.Lens Fiber: Cortex + nucleus
4.Zonules: Hold the lens in place for accommodation
4mm thick

What is a congenital cataract?

Present at birth or early childhood

What are the different types of congenital cataracts?

Capsular
polar
sutural
nuclear
lamellar
central oil droplet>galactosemia
coronary
blue-dot
membranous
complete

What are the maternal, foetal, and other causes of congenital cataract?

-Maternal: Rubella, intrauterine hypoxia, radiation, corticosteroids, gdm
-foetal: hereditary, downs, metabolic disorders (galactosemia, hyper, hypo, Lowe syndrome), forceps
-idiopathic

Presentation of congenital cataract?

dec vision, white reflex, squint, nystagmus, LEUKOCORIA

DDx of leukocoria

Congenital cat
RB
ROP
anterior persistent hyperplastic vitreous
coats disease
endophthalmitis
toxicarial granuloma

Tx of congenital cataract?

Remove lens, put new lens after 2 yrs of age. Phaco not lensectomy. Can now put lens even before 2 yrs.

What are the causes of acquired cataracts?

Senile
Radiation
metabolic disorders
Ocular disease complication
Drugs e.g., corticosteroids
atopic dermatitis

What are the 3 types of senile cataracts?

1. Post Subcapsular>happens to young ppl
2. cortical>permeability of lens capsule changes, Lamellar sep, incipient cataract (cortical spokes), immature cataract, hypermetropic shift, intumescent cataract with shallow ant chamber, mature cataract
3. nuclear>myopic shift

What are the 3 possible fates of a mature cataract?

1.Hyper mature = phacomorhic/lytic glaucoma
2.morganian= cortex liquid, nucleus sinks to bottom
3.subluxation/luxation into ant chamber or vitreous

Signs and symptoms of acquired cataract?

painless gradual loss of vision, photophobia, coloured halos, change in number, bad daytime vision, possible lens induced glaucoma

Complications of Cataract?

-Phacomorphic/lytic glaucoma
-phacoantigenic Uveitis
-Luxation/subluxation of lens
-Glaucoma

Types of cataract extraction?

A) Extracapsular cataract extraction: Phaco, Femtosecond laser, manual surgical, conventional extracapsular
B) intracapsular extraction
C) pars plana lensectomy

What are the post op complications of cataract surgery?

Early
Acute endophthalmitis
corneal oedema
wound leakage
iris prolapse
flat ant chamber
secondary glaucoma
choroidal detachment
iridocyclitis
hyphaemia
Late
chronic endophthalmitis
phacoantigenic uveitis
corneal decompensation
cystoid macular oedema
RD
opacification of post capsule
subluxation/opacification of IOL

Glaucoma

What are the 4 visible structures on gonioscopy for angle closure?

1.Schwalbe's line
2.trabecular meshwork
3.scleral spur
4.ciliary body ant surface

What are the 3 components of the trabecular meshwork?

Uveal
Scleral
juxtacanalicular/cribriform

How does Aq humour drain beyond the trabecular meshwork?

T mesh> schlems canal> Aq veins/Collector channels> Episcleral veins

What is the total volume of aq humour and what is the avg secretion rate?

0.30ml
2-2.5ml/min

What are the 3 routes of Aq humour drainage?

Trabecular mesh>85%
uveoscleral route
iris route

What is the normal IOP?

10-21 mmHg

What is glaucoma?

Inc IOP that is sufficient to bring about damage to optic N fibers which results in characteristic visual field defects

What is the pathogenesis of glaucoma?

1. Raised iop causes mechanical stretch which disrupts axonal flow so neuro trophins can't reach ganglion cells and they die
2. High iop compresses caps and hence ischemic injury > loss of retinal ganglion cells and nerve fibers

What techniques are used to diagnose glaucoma?

1. tonometry> provocative tests
2. gonioscopy
3. fundoscopy
4. perimetry
5. OCT, Scanning laser polarimetry, stereo disc photography, pachymetry

What are the 4 angle grades for angle closure on gonioscopy?

4) schwalbes line, Trab m, Scleral spur, ciliary body
3) schwalbes line, Trab m, Scleral spur
2) schwalbes line, Trab m
1) schwalbes line
0) None

What are the optic disc changes in glaucoma? (Optic neuropathy)

Nerve fiber layer defect
neuroretinal rim
disc cupping
nasalization of blood vessels
splinter hemm
peripapillary atrophy

What are the early, moderate, and advanced field defects in glaucoma?

Early: paracentral scotoma, roennes nasal step, enlargement of blind spot, seidels(crescent) scotoma
Mod: arcuate scotoma, ring scotoma. peripheral breakthrough
Adv: central and/or temporal islands of vision

What is the use of OCT in glaucoma?

3d analysis of optic nerve head and retinal layers

What is primary open angle glaucoma (POAG)?

Bilateral optic neuropathy, asymetrical with IOP>21, Optic disc changes, visual field defect

What are the risk factors for POAG?

Hereditary, age, Blacks, myopes, DM, Retinal disease, Steroid usage

Why is there elevated IOP in POAG?

ECM deposition with loss of trabeculocytes in trabecular mesh that occludes channels and causes inc resistance to aq outflow

What are the symptoms of POAG?

Painless slow bilateral loss of vision, usually asymptomatic
Night blindness
eye ache, headache

What are the signs of POAG?

Inc IOP
Optic disc changes
open angle
>8mmhg diurnal variation

What is the medical Tx for POAG?

Single or combo drugs
1. PG analogues: Inc outflow from uveoscleral route, Xalatan, Travatan
2. B-blockers: Dec aq production by ciliary epi eg timolol
3. Alpha2 agonist: Dec aq prod by ciliary epi + inc outflow from uveoscleral
4. CAI: Dec prod
5. Miotics: Parasympathomimetic

What are the laser and surgical techniques for POAG?

Argon laser/ Selective laser trabeculoplasty: Enhance outflow
Trabeculectomy: Fistula b/w scleral flap then subconjunctival space to sub tenon space

What is PACG?

Angle closure causes elevated IOP

What are the risk factors for PACG?

hyperopia
small corneal diameter
iris close to lens
shallow ant chamber
dim light, emotional stress, mydriatic drugs

What are the 5 stages of PACG?

1.Latent/suspect
2.sub-acute
3.acute angle closure
4.chronic angle closure
5.absolute glaucoma

What is a primary angle closure suspect?

Asymptomatic
normal iop
shallow ant chamber
grade 1 or 2 on gonioscopy
positive provocative tests
give prophylactic iridotomy/dectomy

What is subacute ACG?

Mid dilation of pupil> relative pupillary block> Iris bombe
complete angle closure> Rise in IOP to 40-45> pain
episodic pain in eye + blurry vision, halos, IOP normal b/w episodes,

physiological miosis fixes everything

What is acute PACG?

Sudden severe rise in iop that does not go down by physiological miosis, resulting in ant chamber infla
Pain, halos, dec vision, **nausea, vomit**, red eye
up to 70 iop,
circumcorneal congestion,
hazy cornea,
semi dilated vertically oval pupil,
swollen optic disc

What is the Tx for acute PACG?

IV acetazolamide
IV mannitol
analgesics+ antiemetics
topical pilocarpine after IOP below 40
iridotomy to prevent further attacks

What is chronic ACG?

-IOP keeps rising slowly and angle function gets completely compromised
-Meds as well as iridotomy or eventually trabeculectomy

What is absolute glaucoma?

painful/painless hard blind eye
no light perception
raised IOP
atrophic optic disc
eventual enucleation

What are the corneal causes of 2 glaucoma?

1. Non perforated ulcer: exudate blocks trab mesh
2. perforated ulcer: peripheral ant synechiae formation

What are the iris related causes of 2 glaucoma?

1. iridocyclitis: Inflammatory cells + fibrin block trab mesh, post synechiae
2. pigmentary g: Open angle, trab obs b/c of iris pig obs
3. Neovascular G: ICRVO, DM ret, BRVO, tumours> cause angle closure + proteins in trab

What are the lens induced causes of 2 Glaucoma?

1. Phacomorphic
2. Phacolytic
3. Lens sub/luxation

What are the blood induced forms of 2 glaucoma?

1. Red cell glaucoma: fresh rbc block trab
2. Haemolytic: Rbc debris blocks trab
3. ghost cells from vit hemm block trab mesh, empty spherical cells

What tumours can induce glaucoma?

Rb, malignant melanoma

How do steroids induce 2 glaucoma?

Steroids inhibit endothelial cells in trab mesh > debris from aq humour deposits in mesh> also prevents lysozymes in trab mesh, too many undigested glycosaminoglycans> dec aq flow

What is the classification of congenital glaucoma?

1. True CG: Raised iop at birth
2. primary infantile: Raised iop within 3 yrs of life
3. Juvenile: raised iop within 3-16 yrs

What is primary infantile glaucoma?

impaired aq flow by maldevelopment of ant chamber angle

What are the causes of primary infantile glaucoma (PIG)?

barkans mem on trab meshwork
dysgenesis of trab mesh
thickening of juxtacanalicular trab
maldevelopment of schlems canal

What are the symptoms of PIG?

Lacrimation, photophobia, blepharospasm, corneal haze, buphthalmos

What are the signs of PIG?

raised IOP
corneal enlargement, oedema
Breaks in Descemet's mem that become haab striae
deep ant chamber
lens sublux
optic disc cupping

What is the TX for PIG?

-Drugs but eventual surgery
-Goniotomy, trabeculotomy, trabeculectomy, artificial drainage shunt, laser ablation of ciliary body

Uveal Tract

What are the 3 parts of the uveal tract?

Iris, choroid, ciliary body

What are the 3 layers of the choroid?

Suprachoroidal lamina, Stroma, Bruch's mem

What are the infectious, non-infectious and AI causes of uveitis?

Infectious: TB, syphilis, hsv, vzv, HIV, candida, toxoplasmosis, parasites
Non: corneal ulcer, scleritis, retinitis
AI: Phacoantigenic, ankylosing spondylitis, psoriatic arthritis, juvenile idiopathic arthritis, Bechet, sarcoidosis, IBD, FUCH's iridocyclitis

What is acute anterior uveitis?

infla of ciliary body and iris for 6 wks to 3 months

What is the pathogenesis of acute iridocyclitis?

Acute infla> spasm of ciliary body>vascular dilation>inc cap permeability> Chemotaxis> Secondary glaucoma

What is the presentation of acute iridocyclitis?

Deep ocular pain
dec vision
photophobia
red eye, lacrimation
Circumcorneal congestion
Keratic precipitates
Aq flare plus cells, hypopyon
muddy iris
miotic pupil
post synechiae
tender eyeball

What is the TX for acute iridocyclitis?

Mydriasis: Atropine> relieve spasm+ remove the synechiae
Steroids: Topical or IVt pred to reduce infla
Systemic NSAIDS for pain
Immunosupps eg azathioprine or cyclosporine
Antibiotics: infections

What is chronic iridocyclitis?

more than 3 months

What is the presentation of chronic iridocyclitis?

Generally asymptomatic
White eye
flare + cells
post synechiae
iris nodules

What is the Tx for chronic iridocyclitis?

Treat the underlying cause

What is intermediate uveitis?

Ocular inflammation involving anterior vitreous/vitreous base overlying ciliary body and peripheral retina (pars plana complex)

- systemic associations: sarcoidosis, MS, peripheral toxoplasmosis, TB

Pars planitis: snowbank/snowball formation w/o systemic diseases

- most common type 85-90% cases

- seen in ages 5-40

- probably due to autoimmune reaction against vitreous, peripheral retina, ciliary body

What is the presentation of intermediate uveitis?

PC: floaters, reduced visual acuity, slow progression

Signs:

- small KPs, aqueous flare and cells

- vitreous: snowballs (aggregations of inflammatory cells in periphery), snow banking (Accumulation of inflammatory exudates usually in inferior quadrant)

- retina: peripheral phlebitis/venous sheathing

What are the complications of intermediate uveitis?

- cystoid macular edema -> visual loss

- retinal phlebitis -> ischemia -> neovascularization -> vitreous hemorrhage

- tractional retinal detachment

- complicated cataract

- secondary glaucoma

- band keratopathy

What is the Tx for intermediate uveitis?

- 10% spontaneously regress, 30% remission and exacerbation, 60% chronic exacerbation

- topical/periocular injection steroids, intra-vitreal triamcinolone, systemic steroids, systemic immunosuppressants

- ablate peripheral retina: cryotherapy, laser photocoagulation (neovascularization), vitrectomy (CME, inflammation)

What is posterior uveitis?

Inflammation of choroid, usually involves retina (chorioretinitis)

What is the clinical presentation of posterior uveitis?

PC: decreased vision (retinal involvement/vitreous haze), floaters, metamorphosis, micropsia (separation of visual receptors), macropsia (crowding of rods/cones), photopsia, positive scotoma.

What is the tx for posterior uveitis?

steroids, immunosuppressants; treat underlying cause (infective/non-infective)

What is endophthalmitis?

infla of intraocular structures

What are the causes of endophthalmitis?

Infection via injury or corneal ulcer, surgery or IVt (intravitreal) injection

What are the signs and symptoms of endophthalmitis?

Ocular pain
red eye, lacrimation
hazy cornea
congested conjunctiva
moderate vision loss
loss of red reflex
vitreous exudation
raised IOP

What is the DDX for endophthalmitis?

Lens material in ant chamber
post op uveitis
vitreous hemm

What is the Tx for endophthalmitis?

IVt antibiotics
Topical steroids
topical atropine
pars plana vitrectomy

What is pan ophthalmitis?

Infla of the whole eyeball

What are the signs and symptoms of pan ophthalmitis?

severe ocular pain and headache
marked vision loss
lid oedema
congestion of conjunctiva
cloudy cornea
pus in ant chamber
restricted extraocular movements>>>>> same tx except evisceration

What is Fuchs uveitis syndrome?

chronic low-grade non granulomatous iridocyclitis or cyclitis

What are the signs and symptoms of Fuchs?

mainly asymptomatic
cataract formation and blurry vision
stellate kp's
mild cells plus flare in ant chamber
diffuse iris atrophy
iris heterochromia
no post synechiae

What are the complications of Fuchs?

cataract, glaucoma

Vitreous

What is vitreous humour?

Inert transparent jelly-like structure that is about 4ml in vol, behind the lens

What is the structure of vit humor?

-Network of collagen fibrils interspersed with hyaluronic acid
-Cortical vit= Dense fibrils that form mems with the inner walls of the eye
-central vit= Low density with hyaloid/Cloquet's canal

What happens to vitreous is old age?

Liquefies (synchesis) and shrinks (syneresis)

What are the functions of the Vit?

Refraction
volume/shape
nutrients for retina + lens

What is post Vit detachment?

Sep of cortical vit from retina posterior to the ora serrata> normal in ppl over 60

What is clinical presentation of PVD?

Floaters, flashes, ring like opacity near optic disc showing separation is star feature

What are the complications of PVD?

Vit/retinal hemm
retinal tear

What is a vit hemm?

Blood accumulation in vit cavity that causes sudden painless vision loss

What causes vit hemm?

1. rupture of normal vessels because of RRD related to PVD
2. Neovascularization: DM, RVO, ROP
3. retinal vasculitis
4. tumors

What are the signs and symptoms of vit hemm?

Floaters, flashes, painless vision loss, dec visual acquity
black shadows on direct ophtalmo

What is the fate of a vit hemm?

1. complete absorption in 4-8 weeks
2. organization of hemm with yellow white debris
3. ghost cell glaucoma
4. hemosiderosis bulbi b/c of Fe2+
5. retinal detach

What is hemosiderosis bulbi?

Retention of iron containing foreign body in the eye that results in cataract, rust coloured anterior sub capsule deposit, iris heterochromia, Fleischer ring, and pupillary mydriasis

What is the Tx for vit hemm?

Rest eyes with patch
anti-VEGF injection
treat underlying cause
Vitrectomy in case nothing works

Retina

Thickness of the retina?

0.56-0.1mm

What is the arterial blood supply of the retina?

1. Central retinal artery: Inner half up to outer plexiform layer
2. Short Posterior Ciliary arteries: Outer half up to pigment layer via choriocapillaris
3. Cilioretinal Artery: 30% present, supplies macula

ICA > ophthalmic a. > branches

What is special about the Fovea and F. Centralis?

Thinnest part of retina and avascular zone with highest conc of cones. Supplied by underlying choriocapillaris.

What is the venous drainage of retina?

Central retinal vein + s. post ciliary veins + cilioretinal a.>>> Sup ophthalmic vein>>> cavernous sinus

What are the external and internal blood retinal barriers?

Internal: tight junctions in cap
External: tight junctions in retinal pigment layer
no blood clouding the retina to reduce its transparency

Classify retinal vascular disease

1. Cap occlusion: Cotton wool spots (soft spots of ischemia), irregular veins, new vessels
2. cap hemm: Hemm, edema, lipid exudates (well demarcated)

What are the 10 layers of the retina?

RPE
rods, cones
external limiting mem
outer nuclear layer
outer plexiform layer
inner nuclear layer
inner plexiform layer
ganglion cells
nerve fiber layer
internal limiting mem

What are the risk factors for dm retinopathy?

duration, control of dm, htn, obese, preg, anaemia, smoking, nephropathy, hyperlipidaemia

What are the 2 main pathogenic processes in dm retinopathy?

1. microvascular leakage: exudates, retinal edema, hemm
2. micro vascular occlusion: ischemia

Name 3 ischemia related changes in retina due to dm retinopathy

Cotton wool spots, neovascularization, IRMA (arteriovenous shunts)

What causes vision loss in dm retinopathy?

Macular edema or ischemia with exudates and hemm, possible retinal detachment, vitreous hemm, neovascular glaucoma

What is the classification of dm retinopathy?

TABLE I. CLINICAL CRITERIA FOR DR GRADING [12]

Level	Clinical Criteria
Normal	No abnormalities
Mild NPDR	Microaneurysms only
Moderate NDPR	Microaneurysms and one or more of exudates but less than the definition of sever
Severe NPDR	One of the following: 1. Microaneurysms and exudates in all four quadrants as fovea is a center of four quadrants, 2. Intra retinal micro vascular abnormalities in one or more quadrants 3. Venous beading in at least two quadrants
PDR	Any of the following: 1. New vessels elsewhere 2. New vessels on the (optic) disc 3. Neovascularisation

3 diagnostic tools for dm retinopathy?

Fundus exam, Fundus Fluorescence angiography, Optical coherence tomography

3 treatment options for dm retinopathy?

Medical: Anti-VEGF
Laser: Focal tx or pan retinal photocoagulation
Surgical: pars plana vitrectomy

What is the subretinal space?

Space between sensory and pigment layer of retina

3 types of retinal detachment?

Rhegmatogenous (Full thickness break), Traction, Exudative/serous

What are the risk factors for RRD?

-Posterior Vitreous detachment
-Peripheral retinal degenerations (Lattice + snail Track)
-Severe Myopia (excess elongation of eyeball)
Trauma
-Previous cataract Surgery (Ultrasound rays damage the retina)

4 different types of retinal breaks?

Horseshoe/flap tear > PVD
Retinal Hole> Chronic atrophy of sensory retina
Giant Tear> greater than 3 clock hours
Dialysis> gets separated from ora serrata

What are the classic signs and symptoms of RRD?

Flashing lights, floaters, painless loss of vision, visual field defects
Decreased visual acuity, RAPD, IOP<than normal eye, mild anterior uveitis, Tobacco dust (Pigment cells in anterior chamber), detached retina

What are the diagnostic techniques for Retinal detachment?

Indirect ophthalmoscope, Goldman three mirrors, B-scan(U/S)

What are the treatment options for RD?

Locate break> drain fluid from subretinal space, seal off break with laser/cryophotcoagulation to induce infla for repair> external scleral buckling or pars plana vitrectomy with silicon oil or expanding gas insertion

What is TRD?

Tractional RD happens because of damaged vessels causing vitreoretinal contraction that detaches sensory retina from retinal pig epi

What are the causes of TRD?

Proliferative DM retinopathy
ROP
posterior segment trauma
aphakia (vitreous displacement)

What are the microscopic features of TRD?

Vitreoretinal Band, decreased retinal mobility, elevation of retina at site of traction

What is ERD?

Exudative RD is due to damaged RPE allows choroid plexus to leak serous fluid into the subretinal space

What causes ERD?

Hypertension, Ocular tumors or infla

How does ERD appear on fundoscopy?

Smooth convex dome, retinal position changes with gravity

How do we treat ERD?

Treat the underlying cause and it resolves on its own

What are the common risk factors for retinal vein occlusion?

Age>55yrs, HTN, DM, increased IOP, Drugs eg OCP, Smoking

What causes CRVO?

atherosclerotic plaque at AV crossing compresses the vein and causes occlusion

What are the signs and symptoms of Non-ischemic CRVO?

Sudden unilateral painless loss of vision
Decreased visual acuity, raised IOP, Tortuous engorged retinal veins, dot blot + flame hemm in periphery, Cotton wool spots, edema

2 complications of NI CRVO?

Cystoid macular edema, convert to ischemic CRVO

What is the treatment for NI CRVO?

Control risk factors, Anti platelet therapy > vision returns to normal
In bad conditions uses Anti-VEGF and IO steroids with laser photocoagulation

What are the clinical features of ischemic CRVO?

Sudden + severe vision loss > only count fingers
Afferent pupillary defect
Tortuous retinal veins, Deep blot + flame hemm, Disc edema + hyperaemia, Cotton wool spots

FFA of NI vs ischemic CRVO?

FFA in non-ischemic has good capillary perfusion while non ischemic has no cap perfusion

How is the prognosis of NI different from ischemic CRVO?

You can recover from non-ischemic but not from ischemic

What are the complications of ischemic CRVO?

Macular ischemia, Chronic cystoid macular edema, Rubeosis Iridis

What is the difference between quadrant vs hemiretinal BRVO?

Branch vein occlusion away from disc vs right at disk margin

Clinical features + complications of BRVO?

Sudden blurred vision in macular involvement, decreased visual acuity, Edema + hemm in area of occlusion

How do we treat BRVO with decreased visual acuity?

Anti-VEGF, Laser photocoagulation, IO steroid (Triamcinolone)

What are the causes of CRAO?

Thrombosis or embolism
Carotid A. atheroma can give cholesterol or platelet fibrin emboli
Calcific emboli from cardiac valves
vasospasm

What is Amaurosis Fugax?

Transient vision loss, painless, because of platelet fibrin embolus

What are the signs of CRAO?

Profound vision loss
Dilated pupil
Afferent pupil defect
Pale white retina
Cherry red spot
narrow retinal arteries
Cattle tracking
atrophic changes

What is seen in the FFA of CRAO?

Delayed arterial filling and choroid masking due to retinal edema

What is the Tx of CRAO and what principles does it use?

1. Dislodge emboli: Reduce IOP, Ocular massage, Sublingual Isorbide nitrate, NdYAG laser to destroy visible embolus
2. Relieve vasospasm: Inhale CO2 in bag
3. Reduce retinal edema: Methylpred to reduce infla

What are the signs of BRAO?

Sudden painless sectoral vision loss, variable visual acuity, RAPD

What is Retinopathy of prematurity (ROP)?

Bilateral proliferative retinopathy due to risk factors

3 Risk factors for ROP?

Preterm less than 31 weeks
LBW - less than 1500g
high conc O2 supplement

What is the pathogenesis of ROP?

Nasal retina forms before temporal. Temporal continues to develop till a month after birth. High o2 conc destroys caps in temporal retina hence to replace them excess VEGF released. Compensatory aberrant neovascularization.

What are the 3 retinal zones of ROP?

Zone 1 = macula + optic disc
zone 2 = up to nasal ora serrata
zone 3 = crescent up to temporal ora serrata

What are the 5 stages of ROP?

S1 - Demarcation line b/w normal vascular and peripheral nonvascular retina
S2 - Elevated ridge due to vascular tufts infiltrating nonvascular retina
S3 - Extraretinal fibrovascular proliferation into vitreous cavity + vit hemm
S4 - Partial retinal detachment with or without macular involvement
S5 - Complete retinal detachment b/c of extensive fibrovascular proliferation > leukocoria

What is plus disease?

S3 plus Dilated/tortuous vessels or vitreous haze or failure of pupil to dilate

What are the Tx guidelines for ROP?

S1+ 2 = carefully observe but no intervention
S3 onwards is Laser ablation of peripheral retina, Anti- VEGF, Pars plana vitrectomy

What is age related macular degeneration (ARMD)?

Macular degeneration with irreversible vision loss

What age group is mostly affected by ARMD?

Age>50yrs

What are the risk factors for ARMD?

Age, drusen formation, white ppl, HTN, Family hx, smoking, hypercholesterolemia, obesity, high sunlight exposure, aspirin use

What are Drusen?

Yellow white ECM deposits b/w RPE and bruchs mem

What is pathogenesis of Dry/Atrophic/Non-neovascular ARMD?

Drusen deposits coalesce. RPE can't get blood from choroid. Photoreceptors in top atrophy. thickening of bruchs mem, overall atrophy.

What are the symptoms of dry ARMD?

Gradual central vision loss, distorted vison, bilateral but asymmetrical

What are the signs of the early, intermediate, and advanced stages of ARMD?

Early - Asymptomatic small drusen with focal hyperpig
Intermediate - Some loss of vision with large drusen and small areas of atrophy
Advanced - Significant visual loss with geographic atrophy

What is the Tx for dry ARMD?

No tx but give antioxidants in high-risk individuals + reduce risk factors. Give visual aids plus lifestyle modifications

What is the pathogenesis of Wet/Exudative/Neovascular ARMD?

Choroidal neovascularization which breaks bruchs mem to leak into sub retinal space. Exudative lesions form a scar eventually

What are the symptoms of Wet ARMD?

Sudden central vision loss with metamorphopsia, with scotoma

What is choroidal neovascularization (CNV)?

Choriocaps grow into sub retinal space through breaks in bruchs mem

What are the 3 subtypes of Sub-RPE CNV?

-Serous pigment epi detachment - orange dome elevation
-hemm PED - dark red dome with defined borders
-fibrovascular PED - Orange dome with irregular margins

What are the 3 possible Tx options for wet ARMD?

Anti - VEGF + IO steroids
Argon laser photocoagulation
Surgical macular removal

What is retinitis pigmentosa (RP)?

Group of hereditary progressive degenerative disorders of retina with atrophy of RPE esp rods with night blindness and constriction of visual field till blindness occurs

What is the pathogenesis of RP?

Peripheral destruction or rods till only central tubular vision remains with RPE atrophy due to which pigment piles up in vessels and causes pigmentation

What are the symptoms of RP?

Night blindness, visual field constriction, defective vision, blindness

What is seen on fundic exam of RP?

Attenuated blood vessels
Pigment bone corpuscles or bone spicules
Pale waxy optic disc
Maculopathy

What are the 5 ocular associations of RP?

Cataract
open angle glaucoma
keratoconus
high myopia
PVD

What are the Tx options for RP?

No tx. Wear sunglasses to avoid sunlight

What is a Retinoblastoma (RB)?

Congenital tumor from primitive retinal cells of sensory retina

At what age does RB present?

less than 3 yrs

What Chromosome s affected in RB?

13q14

How does RB develop?

malignant transformation of retinal cells before final differentiation

What is the difference between exophytic and endophytic RB?

Detaches the retina vs tumor protrudes into vitreous cavity w/o RD

What are the 4 stages of RB?

1.Quiescent - 6m to 1 yr Leukocoria, squint, decreased vision, mass on ophthalmoscope, Calcium deposits
2.Infla/Glaucomatous - Painful red eye or pseudo hypopyon
3.Extraocular - Proptosis
4.Metastasis - Optic nerve, preauricular LN, Bones, Liver

What investigations are done for RB diagnosis?

CT, MRI, B-scan, Bone marrow biopsy

What are the 2 Tx options for RB?

Small = Tumour destructive therapy with chemo and transpupillary thermotherapy
Large = enucleation

Optic Nerve

How many axons are there in the optic nerve?

1.2 million

What are the 4 anatomical parts of the optic nerve?

1.Intraocular: optic disc to a little beyond lamina cribrosa
2.Orbital: back of the eye to optic foramen
3.Intracanalicular: within optic canal
4.Intracranial: inside cranial cavity to optic chiasm

What is passive vs active optic disc edema?

Passive= non infla disc edema vs Active= infla of optic disc aka papillitis

What is papilledema?

Passive optic disc edema due to raised ICP, mostly bilateral

What are the causes of papilledema?

1. Intracranial tumor
2. cerebral hypertension> pseudotumor cerebiri, vit a
3. hydrocephalus
4. encephalitis, meningitis
5. sub-arachnoid hemm
6. head injury or excess vit A

What is the pathogenesis of raised ICP that leads to papilledema?

Raised ICP travels through shared meningeal sheaths, compresses, and stops axoplasmic flow, ischemia, and mechanical compression> venous stasis causes edema, exudates, hemm, cotton wool spots

What are the symptoms of papilledema?

Headache esp in the morning, nausea vomit, dec consciousness, vision blacks out, diplopia

Classify the 4 different stages of papilledema

1. Early: No symptoms, normal vision + pupil, Mild **disc hyperaemia**, lack of spontaneous venous pulse, **blurring of disc margins**
2. Fully developed: Normal pupil and vision, **Blurred margins**, hyperaemia, tortuous veins, no venous pulse, hemm, cotton wool, exudates, **macular star**, cup still present
3. Chronic: dec visual acuity, **champagne cork disc**, enlarged blind spot
4. Atrophic: after 6-9mo, severe dec visual acuity, **grey disc with blurred margins**

What should immediately be ruled out in papilledema?

space occupying lesion

How do you treat papilledema?

treat underlying cause
control raised icp by using diuretics, LP, Lumboperitoneal shunt, optic n sheath decompression.

What is optic neuritis?

Inflammation of the optic N

What are the causes of optic neuritis?

1. Demyelinating disorder e.g., MS
2. Infection e.g., VZV, Syphilis, Lyme, cat scratch, meningitis, orbital cellulitis
3. Intraocular infla e.g., uveitis or endophthalmitis
4. AI disease e.g., lupus, polyarthritis

What are the different ophthalmoscopic classes of optic neuritis?

1. Papillitis: Infla of optic nerve head with hyperaemia and edema
2. neuroretinitis: same as papillitis but with retinal involvement and macular star
3. retrobulbar: MS type with normal optic disc

What are the symptoms of optic neuritis?

Acute or subacute monocular vision loss, eye pain, phosphenes

What are the signs of optic neuritis?

dec visual acuity, loss of colour vision, RAPD, tender globe, and brow pain

What is seen on fundus exam for the 3 types of optic neuritis?

Papillitis: Swollen, hyperaemic, blurred margin, tortuous retinal veins, splinter hemm, exudates, inlfa cells in vitreous
Neuroretinitis: Same disc findings as above but with retinal involvement and macular star
Retrobulbar: normal disc

What is the Tx for optic neuritis?

Treat underlying cause
IV steroids, IFN-b injection, Vit b1,6,12 to help in healing

What is optic neuropathy?

Non-infla disease of optic nerve or ganglion cells resulting in vision loss

What are the common causes of optic neuropathy?

Glaucoma, giant cell arteritis, HTN, DM, Hyperlipidaemia, Compressive orbital lesions, Infiltrative tumours or disorders, Trauma

Pupil

What is the normal pupillary size?

2-3 mm

What is the parasympathetic pupillary pathway?

Light recepted by afferent CN2 nerve neurons at the retina> Info taken to pretectal nuclei in midbrain> **bilateral** innervation of Edinger Westphal nucleus> efferent signals via CN3 through ciliary ganglion to sphincter pupillae muscle via short ciliary nerves> miosis/constriction. 4 neurons

What is the sympathetic pupillary pathway?

Pain fear etc act as stimuli> first order neuron in hypothalamus exit **unilaterally** and synapse in paravertebral ganglia> second neuron goes to carotid plexus and synapses there> Third neuron aka postganglionic sympathetic exit from superior cervical ganglion and go to dilator pupillae + superior tarsal muscle via long ciliary branch of nasociliary nerve

Accommodation/Near reflex pathway?

Retina to parastriate cortex via CN2> Parastriate cortex to EW nucleus bilaterally> EW to sphincter pupillae via CN3 ciliary ganglion. Fibers innervating EW are sep from those for the light reflex hence not injured together.

What is a Relative afferent Pupillary defect/Marcus Gunn Pupil?

Injury to optic nerve or retinal damage, partial vision in affected eye
Light in normal eye = both constrict
Light in damaged eye = Both dilate

What is Total Afferent pupil defect?

Optic nerve damage, blind in affected eye
Light in normal eye = both constrict
light in damaged eye = neither constricts

Argyll Robertson pupil?

-Accommodates but does not react to light
-Fibers for light reflex injured b/w EW and pretectal nucleus but accommodation reflex fibers intact
-Small irregular pupils that do not react to light but do accommodate

-Causes: Syphilis, DM, MS, Alcohol, Trauma, Surgery

Adie's tonic Pupil?

Damage to ciliary ganglion> both light and accommodation reflex fibers damaged
Dilated pupil at rest - anisocoria

Orbit

What are the 7 bones that form the orbit?

Frontal, maxilla, zygomatic, sphenoid, palatine, ethmoid, lacrimal

What are the contents of the orbit?

Eye, muscles, lacrimal gland and sac, blood vessels, nerves, fascia, and fat

What are the 4 surgical spaces of the orbit? look at diagram

1. Central: retrobulbar space between EO muscles> axial proptosis or injection
2. Peripheral: B/w EOM and periosteum> peribulbar anaesthesia
3. Subperiosteal space: potential space b/w periosteum and bone
4. Sub tenon's space: around eyeball ender tenons capsule

What are the Infla, Infectious and Neoplastic causes of proptosis?

1. Infla: Orbital cellulitis, panophthalmitis, cavernous sinus t
2. Infectious: thyroid disease, Wegener's granulomatosis
3. Tumors: dermis cyst, haemangiomas, optic n glioma, RB, malignant melanoma

What are the causes of painful proptosis?

Orbital cellulitis, rhabdomyosarcoma, acute dacryoadenitis, ruptured dermoid cyst

What is the pathological value of proptosis?

21mm

What is preseptal cellulitis?

Acute infla of the tissue anterior to the orbital septum

What causes preseptal cellulitis?

S. aureus, strep pyogenes via
1. Spread of adj infection
2. Exogenous infection
3. Hematogenous spread

What are the clinical features of preseptal cellulitis?

Edema of eyelid, reds, swollen tender, no proptosis cause eye not involved

What is the Tx of preseptal cellulitis?

Systemic antibiotics
topical antibiotic
systemic analgesics

What is orbital cellulitis?

Acute infla of orbital T posterior to the orbital septum> medical emergency

What are the common infectious causes of orbital cellulitis?

Strep pneumonia, staphylococcus aureus, strep pyogenes, h.influenza

Which sinus particularly can spread to the orbit to cause orbital cellulitis?

ethmoid sinus

What are the distinguishing factors between orbital and preseptal cellulitis?

orbital= proptosis, ophthalmoplegia, papilledema, dec vision

What are the 5 modes of infection for orbital cellulitis?

1. trauma
2. extension from paranasal sinus or soft t
3. endogenous bacteraemia
4. post op
5. endophthalmitis

What are the signs and symptoms of orbital cellulitis?

Fever, lid edema, red eye,warm tender, proptosis, restricted EO movements, dec visual acuity, disc edema

What is the Tx for orbital cellulitis?

ADMIT
systemic antibiotics, analgesics, topical antibiotics, surgical abscess drain

What are the complications of orbital cellulitis?

Exposure keratopathy
optic neuropathy
raised iop
BLIND
meningitis
brain abscess

What is the most common cause of bilateral or unilateral proptosis?

Thyroid disease

What is the pathogenesis of thyroid eye disease?

TSH stimulating immunoglobulins react with fibroblasts and adipocytes to cause proliferation via glycosaminoglycans synthesis and water retention> inc in size of EOM and deposition of myxoedema> proptosis

What are the signs of thyroid eye disease?

1. lid retraction: Mullers muscle
2. Lid lag
3. infrequent blink
4. periorbital swelling
5. proptosis
6. Dry eye
7. optic neuropathy
8. restrictive myopathy

What is the Werner's classification of thyroid eye disease?

No signs
Only lid retraction

Soft t involved
Proptosis
EOM restriction
Corneal involvement
Sight loss

NO SPECS

What is the Tx of thyroid eye disease?

Treat hyperthyroid
Ocular discomfort: artificial tears, tape eyelids at night
Orbital decompression
Surgery