OPHTHALMOLOGY REVISION

OPHTHALMOLOGY

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Embryology

- Ectoderm
  - Surface
  - Neuroectoderm
- Mesoderm
- Neural crest

Surface Ectoderm (LEVL)

- Lens
- Epithelium (except iris and ciliary body)
- Vitreous
- Lacrimal apparatus

Neuroectoderm (ROSE)

- Retina
- Optic nerve
- Sphincter and dilator pupillae
- Epithelium of iris and ciliary body

Mesoderm (MESO)

Muscles (Extraocular)

- Endothelium of all ocular and orbital blood vessels
- Sclera and Schlemm's canal
- Vitreous

Neural Crest

- Corneal stroma and endothelium
- Trabecular meshwork
- Ciliary muscle

Anatomy Orbit

- Orbit – Rule of 7- 7 bones, 7 muscles, 7 nerves
- Volume of orbit - 30 ml
- Volume of eyeball – 6 ml
- Thinnest wall – medial wall, m/c fractured inferior wall

Anatomy of Globe

- 3 concentric layers – outer cornea /sclera
- middle uvea = iris , ciliary body , choroid
- inner retina
- 2 avascular structures – cornea, lens
- Total refractive power – + 60 Dioptres
- Cornea - 43 D, lens – 19D

Basic Ophthalmic measurements

- Orbital volume 30 ml
- Eyeball volume 6 ml
- Axial length 24 mm
- AC depth 3 mm
- Critical angle total internal reflection air tears interface 46 degree
- Average corneal thickness 540 microns
- Ascorbate 30 times higher in vitreous
- Aqueous humour formation 2.5 µL/minute
- Fovea = Optic disc = 1.5 mm
- Monocular visual field S -60 N-60 I – 70 T -100

Basic ophthalmic measurements …

- High myopia > -6.0 D
- IOP 10 -21 mm of Hg
- Site of intravitreal injection – 3.5 to 4 mm from limbus

Orbit and Adnexa

- M/c proptosis in adults → Thyroid eye disease
- M/c presentation Thyroid eye disease → Lid retraction
- M/c muscle to get restricted in TED -inferior rectus, IMSL rule
- Fusiform dilation of muscle belly with sparing of tendons is THE sign of TED, Coke bottle sign

Orbit

- M/c fracture of orbit – Blow out fracture
- M/c wall to fracture – Inferior wall
- Triad – Diplopia , infra orbital anaesthesia, enophthalmos (DIE)
- Tear drop sign

Orbital Tumours

- M/c primary tumour children – Dermoid cyst
- M/c secondary tumours – Metastases from Neuroblastoma
- M/c primary tumours adults – Cavernous haemangioma
- M/c secondary tumours – metastases from breast ca, lung ca
- M/c orbital tumour → enophthalmos → scirrhous breast carcinoma

Ptosis

- Drooping of upper lid
- Congenital – maldevelopment of LPS
- Acquired –
- Aponeurotic / Involutional ptosis – senile , post contact lens wear
- Myogenic – Myasthenia, myotonic dystrophy

Ptosis

- Neurogenic – 3rd nerve palsy, Horner’s syndrome
- Mechanical – Lid tumours, chalazion
- LPS function – determines surgery of choice
- Normal – 15mm, good – 12, poor <4
- M/C LPS resection – good LPS function
- Frontalis suspension – poor LPS function <4mm

Lacrimal drainage apparatus

Congenital NLDO

- Congenital nasolacrimal duct obstruction – NLDO - 6 %
  - Persistent valve of Hasner
  - Epiphora, discharge, crusting
- D/D – Congenital glaucoma
- 90% improve spontaneously
- Digital lacrimal sac massage – 6 months to 1 year
- NLD probing after 1 year
- DCR after 3 years

Cataracts

- M/c cause of blindness worldwide
- Blindness BCVA <3/60 better eye → NPCB, WHO
- M/C cataract→ Age related cataract
- 3 types of cataracts – Nuclear/cortical/posterior subcapsular cataract (PSC)
- Nuclear cataract → hemeralopia, second sight, index myopia
- Cortical cataracts are cuneiform
- PSC → max visual handicap, located closest to nodal point of eye, max glare

Types of cataracts

- M/c congenital cataract → Zonular (Lamellar)
- Critical period of fixation – 3 months
- Rosette cataract → trauma
- Complicated cataracts are PSC, polychromatic, breadcrumb appearance
- Snowflake → Diabetes
- Oil droplet → Galactosemia

Types of cataracts

- Sunflower cataract → Wilson's disease
- Christmas tree cataract → Myotonic dystrophy
- Glassblower's cataract → Heat(True exfoliation)
- Amiodarone, Busulphan, Chlorpromazine/Chloroquine, Dexamethasone (systemic steroids) ABCD → drug induced cataract
- Fluctuating vision → Diabetic cataract

Cataract

- 4 stages of cataract → Incipient(early)/immature (partially opaque)/ mature (totally opaque) /hypermature (shrunken)
- Symptoms – ↓VA, diplopia/polyopia, coloured halos, glare, ↓contrast sensitivity
- Treatment – Early → glasses, late → surgery
- ICCE – removal of lens/capsule → aphakia → corrected by glasses
- Diplopia
- Jack in the box scotoma
- Pin cushion distortion
- ECCE – removal cataract → post capsule intact → PMMA PCIOL implanted

Cataract surgeries

- Phacoemulsification – Scleral/corneal tunnel incision < 2.8 mm → cataract emulsified → foldable IOL implanted → sutureless
- SICS – 6mm incision → manually cataract extracted → non foldable IOL → no stitches → economically viable
- FLACS – Femto laser assisted cataract surgery – advantage of perfect capsulorhexis/tight incision

Types of IOLS

- IOL power calculated with Biometry –
- Axial length / Keratometry (corneal curvature)
- Foldable IOL’s made of Silicone/ Acrylic
- Non foldable IOL’s made of PMMA
- Lowest incidence of PCO’s with Hydrophobic acrylic IOL’s
- Multifocal IOL’s both near and distance vision

Complications

- M /c → PCO/After Cataract- Elschnig’s pearls, Soemmerring's rings
- Treated by Nd YAG Laser capsulotomy, 1064 nm
- Irvine Gass syndrome – post cataract surgery CME
- Early onset endophthalmitis m/c by Staph epidermidis
- Late onset endophthalmitis by Propionibacterium acnes

Amblyopia

- ↓ VA(2 lines or more) in children not attributable to any structural defect
- Causes –Strabismus / Refractive error / Cataracts/ptosis
- Critical period development amblyopia – 8 years
- No improvement VA after correcting disorder
- Crowding phenomena- VA ↑ single optotype
- Occlusion of good eye -6 hours a day
- Penalization – weekend atropine in good eye

UVEITIS

- Inflammation of iris / ciliary body / choroid
- Anterior/ Intermediate /Posterior /Pan
- M/c – Anterior – 70% → Idiopathic
- HLA B 27 → Ankylosing spondylitis/IBD – Crohn's/Ulcerative Colitis/ Psoriatic arthritis / Reactive arthritis
- M/c AU in children – JRA / JIA
- Circumciliary congestion
- Marker of activity → Cells
- Earliest sign – Flare
- KP's → Arlt's triangle
- Mutton fat KP's/ Koeppe's/ Busaca nodule: Granulomatous uveitis
- Festooned pupil – sign of posterior synechiae

Uveitis

- Intermediate uveitis – M/c – idiopathic (Pars planitis) /Sarcoidosis
- M/C of loss of vision – CME
- Snowballs and snow banks
- M/c/c posterior uveitis – Toxoplasmosis/Tuberculosis
- ‘Headlight in fog appearance’

Treatment of uveitis

- Anterior uveitis – topical steroids – m/c SE – glaucoma
- Cycloplegics – atropine / homatropine
-
- Intermediate uveitis – Injection steroids –Triamcinolone

- Posterior uveitis - Systemic steroids and antimicrobials

Sympathetic ophthalmitis

- B/L granulomatous uveitis following trauma to one eye
- Penetrating/ Perforating injuries to ciliary body
- M/c seen within 14 days to 3 months after injury
- Dalen Fuchs nodules, mutton fat KP’s / retrolental flare
- Enucleation within 14 days

Ocular manifestations of HIV

- M/C – Microangiopathy/ Cotton wool spots
- M/C ocular infection – CMV retinitis(Pizza pie)
- M/C systemic infection – Tuberculosis
- M/C ocular malignancy – Kaposi’s sarcoma
- M/c ocular side effect of HAART therapy – Immune recovery uveitis

Glaucoma

- Triad – ↑ IOP, optic disc damage, visual field defects –any two
- Destruction of retinal ganglion cells
- Buphthalmos / Primary congenital glaucoma – Photophobia, blepharospasm, lacrimation
- Cause – Barkan's membrane blocks angle , raises IOP , enlarged eyeball
- Axial myopia , corneal oedema , Haab's striae

Open Angle Glaucoma (OAG)

- Risk factors – Family history , thin CCT, coloured races, myopia
- Mechanism – TM blockage
- Optic disc signs – ↑CDR, disc pallor, splinter haemorrhages
- Visual field – Paracentral scotoma, Bjerrum's scotoma, nasal step, arcuate scotoma
- Only symptom – frequent change of presbyopic glasses

Angle Closure Glaucoma (ACG)

- Risk factors – middle aged females, hypermetropes, Asians
- Mechanism – Pupillary block
- Stages – Latent, subacute, acute, chronic, absolute
- Acute ACG – sudden , severe pain , redness , blurring , coloured haloes
- Pupil – vertically oval , mid dilated , not reacting to light
- Chronic ACG to be differentiated from OAG
- Fincham's test – differentiates coloured haloes AACG vs cataract by using stenopic slit – no splitting of halos in glaucoma

Treatment of Glaucoma

- Decrease IOP – only treatment
- Increase aqueous outflow / decrease aqueous production
- Only two classes of drugs ↑ outflow , rest all decrease production
- Cholinergic agonists – Pilocarpine/ Carbachol → TM
- PGA – Latanoprost/ Bimatoprost → Uveo – scleral pathway
- DOC of OAG → PGA
- DOC normal tension glaucoma → PGA
- Most powerful IOP lowering drug – PGA → Bimatoprost

Anti Glaucoma Drugs

- Children – DOC topical CAI's- Dorzolamide, Brinzolamide
- Contraindicated – Brimonidine
- Pregnancy – PGA C/I
- DOC in pregnancy – Brimonidine
- Asthma – Beta blockers C/I
- Diabetes – Glycerol and Acetazolamide with caution
- Depression – Brimonidine and Timolol C/I

Anti Glaucoma drugs

- Apraclonidine highest allergic reaction/ brimonidine
- Brimonidine and Pilocarpine cause follicles
- Fastest AG drug IV Mannitol and Acetazolamide

Lens induced glaucoma

- Phacomorphic – mature cataract → intumescence → secondary ACG
- Phacolytic – hypermature cataract →HMW lens protein leaks out through microscopic leaks → TM block → ↑IOP

Neovascular Glaucoma

- M/c/c Diabetes
- CRVO (90 day glaucoma)
- Retina → hypoxia → VEGF →NV
- Red painful eye, ↑IOP, corneal oedema, rubeosis iridis
- Early – Anti VEGF drugs – Bevacizumab/Ranibizumab/Aflibercept
- Pan Retinal Photocoagulation
- ↓ IOP with anti glaucoma drugs except PGA / Cholinergic agonists
- Trabeculectomy / Aqueous drainage implants

Neovascular glaucoma

- Late
- Cyclodestruction
- Diode Laser Cyclophotocoagulation
- Cyclocryopexy

Refraction and Optics

- Emmetropia - condition when rays focus on the retina
- Ammetropia – when rays do NOT focus on retina
- Refractive status depends upon corneal curvature, AL, AC depth and lens thickness
- Most important factor corneal curvature
- 3 types of ammetropia – myopia, hypermetropia, astigmatism

Myopia

- Myopia – Light rays focus in front of retina
- Uncorrected myopes → eyes half closed → pin hole effect
- M/C Axial myopia, AL each 1mm extra → 3 dioptres
- Curvature myopia → Keratoconus
- Distance vision blurred , near vision can manage

Correction of Myopia

- Concave lenses prescribed, worn close to eyes
- Concave lenses → 1 D → 2% minification
- Myopes are under corrected
- Over correction checked by duochrome test

Hypermetropia /Hyperopia

- Rays of light focus behind retina
- Complaints of asthenopia, eye strain induced by excessive accommodation
- Excessive accommodation leads to convergence squint
- Corrected by convex lenses, each 1 D causes 2% magnification
- Uncorrected hyperopia (4 D)→ convergent squint
- Cycloplegic refraction for hypermetropia/ Children – Atropine → children (5 years) Homatropine (5-10 years) Cyclopentolate (10-15 years)
- Wear glasses on tip of nose

Astigmatism

- M/c refractive error
- Condition when image formed by two different foci, curvature ammetropia
- Conoid of Sturm is the cone like space in between two meridians
- Circle of least confusion
- Maximum asthenopia
- Corrected by cylindrical/ toric lenses
- 3 numbers → -3.0DS/ -2.0DC X90°
- Power of sphere, power of cylinder, axis

Astigmatism

- Simple myopic astigmatism -2.0 DCx 90
- Simple hypermetropic astigmatism +3.0DCx180
- Compound myopic astigmatism
  - - 2.0DS/ -3.0DCx 25
- Compound hypermetropic astigmatism
  - +3.0DS/+2.0DCx 170
- Mixed Astigmatism
  - +4.0DS/-5.0DCx90

Astigmatism

- Regular – principal meridians at 90°/180°
- Irregular – not correctible by spherocylindrical lens ,only by contact lenses – keratoconus , pterygium , Salzmann ‘s nodular degeneration

Presbyopia

- Loss of accommodation with age
- Starts at age 40, amplitude of accommodation is 4 D
- Blurred vision at normal reading distance
- Corrected with convex lenses
- 45 years + 1.5 DS
- 50 years + 2.0 DS
- 55 years +2.5 D S
- 60 years + 3.0 D S

Presbyopia

- Corrected by convex lenses
- Bifocals for people with refractive error
- Progressive lenses most recent

Dark Room Procedures

- Retinoscopy
- Distant Direct Ophthalmoscopy DDO
- Direct Ophthalmoscopy DO
- Indirect Ophthalmoscopy IO

Retinoscopy/ Skiascopy

- Technique → objective measurement of refractive error eye
- Performed → 1 metre distance, subtract 1 D from retinoscopic reading, subtract for cycloplegia
- Retinoscope streak → move it side to side , then up and down
- Watch light reflex in pupil – with/against movement of retinoscope
- SPAM – Same Plus Against Minus

SPAM

Distant Direct Ophthalmoscopy

- Direct ophthalmoscope used
- DDO done at 22- 25 cms
- Real, inverted, same size image
- Red glow - normal healthy fundus
- Grey glow - Retinal Detachment
- No glow - Vitreous haemorrhage

DDO

- Dislocated lens – dark crescent
- Keratoconus – Charleaux oil droplet sign

Direct Ophthalmoscopy

- Distance close to face
- Virtual, erect, magnified image
- 15 X magnification
- Field of view – 5 to 10 degrees
- Optic disc, fovea, macula

Indirect ophthalmoscopy

- IO head mounted, binocular lens with mirrors, condensing lens (20D)
- Real, inverted, magnified image
- 3-5 X magnification
- Structures visible till ora serrata

Retina / Vitreous

- Retina - 10 layers -9 NSL / 1 RPE
- Blood supply
  - Inner 2/3 – Central retinal artery
  - Outer 1/3 – Posterior ciliary artery
- Watershed layer –Outer plexiform layer

Retinal detachment

- Separation of NSL from RPE
- Separation within NSL – retinoschisis
- Symptoms - Floaters, flashes, curtain falling down
- Sudden painless loss of vision
- Grey glow visible
- Classification – rhegmatogenous / tractional / exudative

Rhegmatogenous Retinal Detachment

- Causes – Myopia, cataract surgery, trauma
- Characteristic – break in retina – holes/lattices/ tears/horse shoe tear
- Sudden painless loss of vision
- M/c site – superotemporal quadrant
- Shaffer's sign – tobacco dusting of vitreous

Exudative Retinal Detachment

- Separation of NSL / RPE by fluid, no breaks
- Causes – malignant melanoma of choroid, PIH, choroiditis
- Characteristic – shifting fluid

Tractional Retinal Detachments

- → membranes on surface of retina/vitreous
- Diabetes, Sickle cell anaemia, ROP
- Slow painless loss of vision

Management

- Aim – Reattach NSL with RPE
- Rhegmatogenous – Laser photocoagulation, buckling
- Exudative – treat underlying condition
- Tractional – Vitrectomy

Diabetic retinopathy

- M/c vascular disorder of retina
- Retinopathy depends upon glycemic control, duration of DM, associated hypertension, hyperlipidemia, pregnancy
- Blurring of vision, fluctuating vision
- Screening – Type 1 DM – 5 years
- Type 2 DM – At diagnosis DM
- Earliest ocular manifestation → Microaneurysms

Diabetic retinopathy

- DR – 2 stages
- NPDR – MA, cotton wool spots, hard exudates, IRMA
- Mild/moderate/severe NPDR
- Severe NPDR – 4-2-1 rule
- m/c/c loss of vision –macular oedema
- PDR – NVD /NVE
- Loss of vision → Vitreous haemorrhage/ Neovascular glaucoma/ Tractional RD

Diabetic retinopathy

- Treatment
- Control of DM/systemic conditions
- NPDR- macular oedema
  - IV Anti VEGF drugs-Bevacizumab/Ranibizumab/ Aflibercept
- PDR – Pan retinal photocoagulation PRP

Vitreous haemorrhage

- M/c /c PDR , trauma, Eales disease
- Sudden painless loss of vision , floaters , red tint to vision
- Bed rest
- Treat underlying cause
- Vitrectomy

CRVO Central Retinal Venous Occlusion

- Commonest site of occlusion → CRV behind lamina cribrosa of optic nerve
- Risk – Age, DM, HT, hyperlipidemia , glaucoma
- Two types of CRVO – Ischemic / Non ischemic
- Non – ischemic CRVO – 75% – moderate ↓vision, dilated tortuous veins, retinal haemorrhages 4 quadrants, mild disc oedema
- M/c complication – macular oedema
- Ischemic – 25 %(>10DD of capillary non perfusion)

Ischemic CRVO

- Transient visual obscuration (TVO)severe, painless sudden loss of vision
- O/E – dilated tortuous veins, widespread cotton wool spots, RAPD, severe disc oedema, extensive haemorrhages – ‘blood and thunder fundus ‘/ splash tomato fundus
- Complications – NVG – ‘90 Day glaucoma‘
- Macular oedema

CRVO

- M/c/c of visual loss → macular oedema
- Treatment- Intravitreal Anti – VEGF drugs
- Ranibizumab / Bevacizumab / Aflibercept

Central Retinal Artery Occlusion CRAO

- Major ocular emergency → irreversible ↓ vision
- Causes – DM, HT, hyperlipidaemia , GCA
- M/C embolus – Cholesterol (Hollenhorst plaque)
- M/C site – narrowest part of CRA → enters the dural sheath of ON
- Cherry red spot diagnostic
- Cattle truck / Box car appearance
- Retinal ischemic time 90 minutes, irreversible after 4 hours

Treatment Of CRAO

- Ocular massage
- ↓IOP with IV Mannitol/ Acetazolamide
- Paracentesis → highest success rates
- Carbogen inhalation
- Hyperbaric oxygen
- Protect other eye – Systemic steroids if GCA

CYSTOID MACULAR EDEMA

- Fluid in outer plexiform layer NSL (Henle's layer)
- DM / -post cataract surgery – Irvine Gass syndrome
- Painless loss of vision, metamorphopsia, loss of contrast sensitivity
- O/E – Loss of foveal contour, yellow spot

Management of CME

- FA – Flower petal / petalloid appearance
- Topical steroids / Topical NSAIDS / Inj Triamcinolone sub – Tenon ‘s
- CME in DM – Anti – VEGF drugs → Bevacizumab / Ranibizumab
- CME in Retinitis pigmentosa – Acetazolamide

Central serous retinopathy – CSR

- Fluid between NSL and RPE, under macula
- Causes → Type A personality, stress, steroid usage
- Painless loss of vision, metamorphopsia, central scotoma, hyperopic shift
- O/ E -Halo light reflex

CSR

- FA- Ink blots and Smoke stacks
- Reassurance, rest, anxiolytics
- Spontaneous reabsorption of fluid

Age related macular degeneration ARMD

- Degenerative condition macula with age
- Risks – Age > 50, smoking, hyperlipidemia, hypertension, white race
- Pathology – damage to Bruch ‘s membrane
- Dry -90 % Wet ARMD – 10%
- Gradual loss of vision, scotoma , metamorphopsia
- Dry AMD – hallmark drusen, geographic atrophy
- Wet AMD – CNVM/ SRNVM

Management of AMD

- Treatment – Dry ARMD
- Quit smoking, green leafy vegetables, foods ↑ omega – 3-fatty acids, anti – oxidants(AREDS 2)
- Wet ARMD –
- Intravitreal Anti VEGF drugs
- Bevacizumab / Ranibizumab / Aflibercept

Retinitis pigmentosa

- M/c INHERITED disorder of retina
- Apoptosis of rods , M/c AR
- Earliest symptom nyctalopia
- Ring scotoma progresses to tunnel vision
- Triad – Pale waxy disc, arteriolar attenuation, bone corpuscular pigmentation
- Ocular associations –posterior subcapsular cataract

Management

- Confirmed by flattening of ERG
- No proven therapy
- Anecdotal – 15000 IU of Vitamin A in the palmitate form every day for life
- DHA 1200mg /day and lutein 12mg /day
- Macular oedema – Carbonic anhydrase

Retinoblastoma

- M/c ocular tumour in children (< 5 years)
- Sporadic/Familial : 90%/ 10%
- U/L / B/L : 70 % / 30
- Rb 1 gene →13 q14, mutation of both alleles (Knudson's 2 hit hypothesis)
- M/C → leukocoria, second → squint, ↓vision
- D/d leukocoria → Congenital cataract/ Coats disease, Toxocariasis , ROP

Retinoblastoma

- M/c /c of intraocular calcification in children
- Hallmark → Flexner Wintersteiner rosettes
- MRI – investigation of choice
- Latest → International Classification of Retinoblastoma
- Laser photocoagulation/Transpupillary Thermotherapy → small tumours
- EBRT→ recurrent disease not responding to any treatment
- Chemotherapy – Intravenous, intravitreal, intra arterial
- Intravenous – Vincristine, Etoposide, Carboplatin

Retinoblastoma

- Enucleation – advanced RB occupying > 50% volume
- At least 15 mm of optic nerve sacrificed
- 3 causes of death- metastases /intracranial tumours / secondary tumours
- M/c metastases through optic nerve
- Bilateral retinoblastomas with pinealoblastoma called TRILATERAL RB
- Osteosarcoma femur m/c secondary tumour

Neuroophthalmology

Optic neuritis/ Retrobulbar neuritis

- M/c/c multiple sclerosis, 20-45 year old women
- Sudden painful ↓vision, worsens on ocular movements
- Colour vision desaturation, Marcus Gunn pupil
- Hallmark disc oedema, none in retrobulbar neuritis
- M/c field defect Central /Centrocecal scotoma
- IV methyl prednisolone ↑visual recovery, no long term impact, oral steroids C/ I

Pupillary light reflex

- Constriction of pupil in response to increased illumination of retina
- Direct –response in same eye and consensual – opposite eye
- Normal response – balance between sympathetic and parasympathetic nerves

Argyll Robertson pupil

- Argyll Robertson pupil – Neurosyphilis
- Bilateral, constricted, irregular pupils
- Does not constrict to light, but constricts to near vision- Light near dissociation
- ARP mnemonic -Accommodation Reflex Present
- Prostitute’s pupil

Adie ‘s pupil

- Adie’s pupil- U/L pupil dilation, young ladies- idiopathic , post viral
- No reaction to light, reacts to near vision- light near dissociation
  - Pilocarpine (.125%) test confirmatory- normal pupil does not constrict but Adie’s pupil does

Marcus Gunn pupil/RAPD

- Marcus Gunn pupil –asymmetric defect of optic nerve or retina → optic neuritis, AION , ON gliomas
- Tested by Swinging torchlight test
- Normal pupil constricts on light stimulation, diseased pupil dilates abnormally in light

Horner's Syndrome

- Oculosympathetic paralysis
- Ptosis, miosis, anhidrosis – classic triad
- Congenital HS – heterochromia, due to birth trauma
- Acquired- Pancoast tumour, internal carotid artery dissection
- Confirmatory test –Cocaine test – normal pupil dilates , HS doesn't

Papilledema

Disc oedema with ↑ICT (normal 50-180 mm H2O)

- M/c/c –ICSOL, traumatic brain injuries , subarachnoid haemorrhage
- B/L Disc oedema, 6th CN palsy
- Earliest sign- loss of spontaneous venous pulsation /blurring nasal disc margin
- Enlargement of blind spot – m/c visual field defect

Visual Pathway

Visual Field defects

• Lesion	Field defect
• Pre chiasmal	Monocular blindness
• Optic chiasma	Bitemporal hemianopia
• Optic tract	Homonymous hemianopia
• Occipital lobe	Macular sparing

Strabismus / Squint

Extraocular muscles

SINRAD mnemonic

S rectus	Inferior R	Superior O	Inf Oblique
Intorsion	Extorsion	Intorsion	Extorsion
Adduction	Adduction	Abduction	Abduction
Elevation	Depression	Depression	Elevation

Primary actions

- SR – elevation
- IR - Depression
- SO -Intorsion
- IO - Extorsion

Terminology

- Tropia – manifest squint
- Phoria – latent squint
- Orthophoria – straight eyes
- Heterophoria – Squinting eye
- Exo – Outward deviation
- Eso – inward deviation
- Hyper – Upward deviation
- Hypo - Downward deviation

Phorias

- Latent squint visible during exhaustion → fusion disrupted
- Symptoms of phorias – eyestrain / headache/asthenopia
- Test for detection of Phorias -Cover uncover test

Tropia

- Tropia /manifest squint → Hirschberg test
- 1 mm displacement= 7 degrees of squint
- 1 degree = 2 prism dioptres
- Divided into
  - Comitant
  - Paralytic

Tropias – Comitant

- The angle of deviation b/w both eyes remains constant
- No double vision
- Primary deviation = Secondary deviation
- Two types -Accommodative squint
- Non accommodative squint

Accommodative squint

- Due to uncorrected refractive error
- Uncorrected hypermetropia – Convergent squint
- Uncorrected myopia – Divergent squint
- Treated by prescribing glasses

Non accommodative squint

- No refractive error
- Treatment – Surgery
  - Recession – weakens muscle
  - Resection – strengthens muscle

Tropias – Paralytic squint

- Paralysis of muscle/ NMJ
- M/c causes – vasculopathic causes – DM /BP
- Angle b/w visual axes changes in every gaze , depends upon direction of gaze
- Secondary deviation > primary deviation
- Symptoms-Diplopia/Abnormal head posture /vertigo/disorientation
- Binocular diplopia - double with both eyes open
- AHP depends upon the plane of paralysed muscle –
- Face turn – horizontal rectus muscles paralysed
- Chin lift – vertical muscle palsy
- Head tilt – Oblique muscle palsy

Management of paralytic squint

- Control underlying disorder – vasculopathic causes –DM /BP
- Wait and watch for 6 months
- Manage diplopia

Principles of diplopia management

- Patching
- Prisms
- Botulinum toxin

Paralytic squint-3rd CN palsy

- Diplopia – presenting complaint
- Down and out and ptosis
- Pupil sparing – Medical cause – DM, Hypertension
- Pupil involving – Surgical cause – Aneurysms (PCA/ICA junction) Tumours
- Treat underlying cause
- Correct diplopia
- Watch for 6 month
- Full recovery

4th nerve palsy

- Longest, thinnest CN, first to be affected in trauma, only one to cross over
- Vertical diplopia in downward / inward movement – reading /walking down stairs
- Eye upwards and head tilt on opposite shoulder
- M/c/c children → congenital ,adults – traumatic
- Treat underlying cause
- Treat diplopia
- Recovers by 6 months

6th nerve palsy

- M/c CN palsy, longest subarachnoid course
- Horizontal diplopia, esotropia, face out
- Children – pontine glioma, adults – vasculopathy ,lumbar puncture
- Treat underlying cause
- Wait and watch for 6 months
- Correct diplopia

Myasthenia gravis – the great mimic

- M/c disorder of neuromuscular junction
- Antibodies to Acetylcholine receptors at NMJ
- LPS first muscle to be affected
- Fluctuating ptosis and diplopia worsening in evening
- Pupils and accommodation spared → distinguishes from 3rd nerve palsy
- Tensilon test positive
- Investigation of choice – Acetylcholine receptor antibodies
- DOC Steroids / Pyridostigmine

Conjunctiva

- Conjunctiva – Semi transparent membrane on sclera, reflects on back surface of eyelids
- Function – Lubrication, protection
- Goblet cells produce mucin, important for tear film stabilization

Dry eyes

- 3 layers of tear film –
- outer lipid layer → Meibomian glands
- middle aqueous layer → lacrimal glands,
- inner mucin layer → goblet cells
- Common causes – Advanced age
- Sjogren's syndrome -Dry eyes + dry mouth + RA
- prolonged CL wear
- drug induced
- trachoma /Vitamin A ↓/ MGD
- M/c signs – FB sensation, burning worsening in evening, red eyes, blurring of vision, ocular fatigue, tear meniscus height ↓

Keratoconjunctivitis sicca

- Dyes -Rose Bengal/ Lissamine green – conjunctival dryness
- Fluorescein stain positive for corneal dryness
- TBUT< 10 sec/ Schirmer's< 10 mm 5 minutes
- Tear film supplements→ methylcellulose
- Cyclosporine eye drops

Xerophthalmia

World health organization re-classification of xerophthalmia signs

Classification	Ocular Signs
XN	Night blindness
X1A	Conjunctival xerosis
X1B	Bitot's spots
X2	Corneal xerosis
X3A	Corneal ulceration-keratomalacia involving one-third or less of the cornea
X3B	Corneal ulceration-keratomalacia involving one-half or more of the cornea
XS	Corneal scar
XF	Xerophthalmic fundus

Management of xerophthalmia

Serum levels > 0.7µmoles / L(>20 micrograms per decilitre) →3 doses

Age	Dosage of VIT A	Frequency
>12 months	200,000 IU	0, 1 ,14

Conjunctival degenerations

- Pterygium–wing–like conjunctival overgrowth over cornea
- Stocker’s line – iron line at the leading edge
- Treatment – Excision with conjunctival autograft has least recurrence

Conjunctivitis

inflammation of conjunctiva – bright red congestion, painless, discharge

- M/c viral conjunctivitis – Adenovirus
- Epidemic Keratoconjunctivitis (EKC) – Adenovirus→ Pink eye/Madras eye
- Acute haemorrhagic conjunctivitis (AHC) – Enterovirus 70 → Apollo 11 virus

Trachoma

- Caused by Chlamydia trachomatis A /B/Ba/C
- M/c infectious cause of blindness
- Risks – Endemic areas, lack of hygiene, overcrowding, poor water supply, poverty
- P/c Redness, photophobia, watering
- Children and women affected most
- Hallmark ‘ sago grain follicles ‘
- Herbert’s pits
- Arlt’s line

Trachoma

- Late sequelae – Trichiasis
- Tylosis
- Madarosis
- Entropion
- Corneal opacity – nebula / macula / leucoma
- Dry Eyes
- WHO classification FISTO – Follicles >5 upper tarsus, Intense inflammation sufficient to obscure 50% tarsal vessels, Scarring, Trichiasis, corneal Opacity involving pupillary margin

Egyptian ophthalmia – Trachoma

- Active infection DOC – 1 gram oral Azithromycin single dose
- Topical – 1% Tetracycline ointment bd X 6 weeks
- SAFE strategy – Surgery –Trichiasis
- Antibiotics – DOC Azithromycin
- Facial cleanliness
- Environmental improvement
- Blanket therapy – 1% Tetracycline ointment bid for 5 days for 10 days in a month for 6 months

Spring catarrh/vernal catarrh

- Allergic conjunctivitis- seasonal, recurrent
- Hot summers- Indian subcontinent, Africa
- Itching, redness, tearing, photophobia
- Cobblestone papillae – hallmark
- Horner Trantas spots/ shield ulcers
- Treatment – Mast cell stabilizers – Na cromoglycate/topical steroids

Vision 2020

- Cataract
- Trachoma – GET 2020
- Childhood blindness
- Refractive error
- Onchocerciasis – River Blindness
- Indian scenario
- Corneal blindness
- Glaucoma
- Diabetic retinopathy

Cornea

- Cornea –principal refractive surface/ protective barrier
- Thinnest centrally CCT → 540 microns , thickest periphery
- Power 43 Dioptres -main optical element
- 5 layers
- Epithelium – regenerated by stem cells → limbus(palisades of Vogt)
- Bowman's Membrane – cannot repair itself → leads to scar/opacity
- Stroma – 90% corneal thickness
- Descemet's M – strongest layer – only fungi penetrates intact DM

Cornea

- Endothelium – most imp-maintains transparency
- Endothelium pumps – Na K ATPase pumps
- Cannot regenerate → corneal oedema
- New 6th layer of cornea, between stroma and Descemet's M- PDL/Dua's layer
- 2 irreparable layers of cornea
- Bowman's M → scar, Endothelium → oedema
- Endothelial count- 3000 cells/mm2
- Critical density - 500 cells/mm2
- Corneal donation > 2000 cells/mm2

Keratoplasty

- Types – Penetrating(PK) / Lamellar (LK)
- Maximum rejections against endothelium → LK more successful
- M/C indication Pseudophakic bullous keratopathy/corneal scars /non healing ulcers
- Therapeutic K- to eradicate active infection / repair structural defect-
- M/c indication- microbial keratitis

Corneal donation

- No age limit for donation, but corneas <75 years best
- Within 6 hours of death
- Endothelial count > 2000cells / mm 2
- Preservative media – MK medium -4 days
- Optisol GS / Cornisol -7-14 days
- C/I – HIV, Hepatitis B, Rabies, Septicaemia, Creutzfeld Jacob disease, Retinoblastoma

Bacterial keratitis

- Risks- Trauma, CL, loose sutures, dry eyes, exposure keratopathy
- M/c bacterial keratitis→ Staph aureus/ Pseudomonas
- Ulcus serpens/hypopyon corneal ulcer → streptococcus pneumoniae
- M/c CL induced ulcer – Pseudomonas
- Bacteria penetrating intact epithelium – Corynebacterium, Neisseria
- DOC – Moxifloxacin, Gatifloxacin

Acanthamoeba keratitis

- H/o CL wear → rinsing in tap water, corneal trauma → exposure contaminated water, h/o suspected HSV keratitis not responding
- ALL dendritic ulcers in CL wearers suspected
- ‘ Pain out of proportion ‘
- Ring shaped ulcer, radial keratoneuritis, epithelial stippling
- Pseudodendrites visible
- DOC PHMB, Propamidine, Chlorhexidine

Fungal keratitis

- M/c/c -Fusarium /Aspergillus- filamentous fungi
- Predisposing causes -Injury with organic matter, topical steroids
- “Signs more than symptoms “
- Finger like projections, feathery margins, satellite lesions
- DOC Natamicin, discontinue ALL steroids

Viral keratitis

- HSV –Type 1 m/c → primary /reactivation → stress, CL, trauma, sun exposure
- Epithelial / Stromal / Endothelial
- Epithelial keratitis – dendritic ulcers caused by HSV → true dendrites
- Loss of corneal sensation
- Treatment – Topical Acyclovir

Herpes zoster ophthalmicus (HZO)

- Caused → Varicella zoster → chicken pox
- Reactivated in immunocompromised patients, age >60
- HZO in young patients → HIV
- U/L painful vesicles along dermatome of V CN
- Hutchinson's sign → tip of nose affected → eye involved
- Risk post herpetic neuralgia ↑, unless treatment starts within 72 hours
- Acyclovir 800mg 5 times a day X 7 -10 days

Keratoconus

- Non – inflammatory, B/L, progressive corneal ectasia with central thinning
- Main risk – Rubbing of eye
- Onset early adolescence → blurring of vision → frequent change of glasses
- High irregular / asymmetric astigmatism with scissoring of reflexes on retinoscopy
- Munson's sign – notching of lower lid on looking down
- Vogt's striae – vertical folds on corneal stroma
- Fleischer's ring – Epithelial iron ring on base of cone

Management

- Glasses
- RGP lenses
- Penetrating keratoplasty
- Corneal Collagen Crosslinking with Riboflavin – C 3R / CXL

THANK YOU