OPTHALMOLOGY

SUMMARY

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Ophthalmology

Red Eye

• ***Painless/not sight threatening***: Conjunctivitis, degenerative conjunctival conditions, subconjunctival hemorrhage, lid diseases

Painless Conditions

• Part 1: Conjunctivitis:

• ***Conjunctival anatomy***:
- mucous membrane terminating at the limbus
- ***divided into***:
a. *palpebral* (posterior aspect of eyelid, firmly adhere),
b. *forniceal* (loose, redundant, cover upper outer fornix),
c. *bulbar* (intermediate, anterior part of sclera)
- richly vascular, supplied by anterior ciliary and palpebral arteries
- it mediates both passive and active immunity
- ***Histology***: epithelium, stroma (has mucin secreting goblet cells, accessory lacrimal glands of krause and wolfring), CALT (conjunctival associated lymphoid tissue)
- ***Lymphatic drainage***: periauricular, submandibular

• ***Discharge***:
- *Watery*: acute viral or acute allergic
- *Mucoid*: Chronic allergic, dry eye
- *Mucopurulent*: Chlamydial or acute bacterial
- *Moderately purulent*: Bacterial
- *Severe purulent*: Gonococcal

• ***Two type of reactions***:

	Follicles	Papillae
Site	translucent grains of rice, mostly in the fornices	in palpebral conjunctiva and limbal of bulbar conjunctiva
Vascular core	Around it (not in it)	present
Histology	- subepithelial lymphoid germinal center - central immature lymphocyte - mature cells peripherally	- folds of hyper plastic epithelium - fibrovascular core - subepithelial stromal infiltration - inflammatory cells
Causes	viral, chlamydial	bacterial, allergic, chronic blepharitis, contact lens

• ***Acute bacterial Conjunctivitis***:
- common, self-limiting, mostly bilateral (starts as uni)
- ***caused by***: direct contact
- ***organisms***: staph (MC), strep, H.influenza, N. gonorrhoeae, meningococcal (severe)
- discharge on waking, no lymphadenopathy, corneal involvement, edema (severe)
- ***discharge***: hyperacute purulent: gonoccal, meningococcal
- ***systemic symptoms***: gonococcus, meningococcal, chlamydia, H.infleunza
- ***investigations***: not necessary, culture, stain, PCR
- ***Treatment***: topical antibiotics, systemic (if systemic disease)

• Part 2: Chlamydia & Ophthalmia Neonatorum:

• Chlamydia has 2 types LGV, TRIC, we will focus on TRIC (trachoma-inclusion conjunctivitis) agents: serotypes A, B, Ba, C cause trachoma, while D to K cause genital, systemic, ocular diseases

• C. Trachomatis:

• depend on host cells (cannot replicate extracellularly), 2-20% in sexually active
• ***transmission by***: autoinculation from genital secretions, 10% by eye to eye spread
• ***incubation***: 1 week
• ***males***: non-gonococcal urethritis (NGU/NSU), might be asymptomatic, epididymitis, trigger Reiter (reactive arthritis)
• ***females***: dysuria, discharge, may lead to pelvic inflammatory disease, or even infertility
• ***S/Sx***: subacute onset, might become chronic, ask about sexual exposure, with lymphadenopathy, large follicles in the inferior fornix and may also involve the upper tarsal conjunctiva
• ***other***: superficial punctate keratitis, perilimbal corneal infiltrates (in 2-3w), mild conjunctival scarring, superior corneal pannus (superficial blood vessel invading cornea)
• ***Investigations***: conjunctival scraping, giemsa stain (inclusion bodies), immunogluoresence (chlamydial antigens), cell culture, swabs, PCR, ELISA
• ***Treatment***:
- *systemic antibiotics*: Azithromycin (TOC), doxycyclin, erythromycin, amoxicillin
- *Topical*: might be used: ointment

• Trachoma:

- in poverty, overcrowding, poor hygiene, recurrent infections
- cell mediated delayed hypersensitivity (type 4)

Active Trachoma	Cicatricial Trachoma
- pre-schoolers - **mucopurulent** - **mixed** follicular/papillary - in <2y papillary predominate - Keratitis and **pannus** formation	- middle age - Scars (**Arlt line**) – linear, stellate, broad - **Triachiasis**: misdirected lashes - **Distriachiasis**: new line of eye lashes that originate from Meibomian glands due to metaplasia - **Entropion**: inward rotation of eyelid margin - **Corneal Opacification** - **Dry eye**: goblet cells, destruction of lacrimal gland - Superior limbal follicles resolve – **Herbert pits**

• ***WHO Trachoma Staging***: **FISTCO**

T**F**: **F**ollicular inflammation T**I**: **I**ntense inflammation T**S**: **S**carring

T**T**: **T**richiasis C**O**: Corneal **O**bacification

• ***Management of Trachoma***: **SAFE**:

- **S**urgery: for triachiasis, entropion (late stage)
- **A**ntibiotics: Systemic: azithromycin, Topical: ointment
- **F**acial hygiene
- **E**nvironmental improvement

• **Opthalmia Neonatrum** (**Neonatal Conjuctivitis**):

- in 10%, both ocular and systemic

• ***Transmission***:

- *From the mother* (birth canal – C.tachomatis, N. gonorrhoeae, HSV)
- *Other organisms*: Staph, strep, H.influenza
- Staph MC mild / C. Trachomatis: MC overall / Gonorrhea (most serious)
- *Other causes*: topical preparation as prophylaxis to infection (silver nitrate – chemical), also congenital nasolacrimal obstruction (uncanalized tear duct: watery, mild)

• ***Timing***: Chemical: few days, Gonococcal: 1st w, Staph, bacteria: end of 1st w, HSV: 1-2w, Chlamydia: 1-3 w

• ***Discharge***: depend on the cause, eyelid periocular vesicles in HSV

• ***Eye exam***:

- corneal exam is a must! (Progressive ulcer is common – fluorescein helpful)
- pseudomembrane in chlamydia
- congenital glaucoma might appear as neonatal conjunctivitis

• ***Treatment***:

- if chlamydial then treat by oral erythromycin
- HSV is always considered systemic

• Part 3: Viral and Allergic Conjuctivitis:

• Viral:

- 90% due to adenovirus (non enveloped DsDNA, mostly in epidemics, might be sporadic)

• **Presentation**:

A. Non-specific acute follicular:

- *MC type*, *unilateral* (other eye affected in 1-2 days)
- mild photophobia, *mild systemic symptoms* (sore throat, cold)

B. Pharyngoconjuctival fever (PCF):

- Serotypes 3,4,7,, families with *URTI*
- *mild Keratitis* 30%, *prominent sore throat*

C. Epidemic keratoconjuctivitis (EKC):

- Serotypes 8,19,37,, *most severe* type
- *Keraititis* 80%, *photophobia*

D. Other types:

- *Acute hemmoraghic*: rapid onset, *enterovirus*, *Coxsackie*, resolve in 1-2w
- *HSV*: follicular type, unilateral + skin vesicles
- *Systemic viral*: *varicella* (opthalmis shingles), *measles*, *mumps*: follicular
- *Molluscum Contagiosum*: by *Pox virus* (in healthy children, peak 2-4 yr)

• ***Clinical Picture***: hyperemia, eyelid edema (nothing to severe), peri-auricular lymphadenopathy, follicles, papillae (maybe), in severe cases (hemorrhage, chemosis, pseudomembrane, keratitis (adeno), anterior uveitis)

• ***membranes***:

- *pseudo* (adherent to inflamed area) *true* (adherent to epithelium) both leave scarring
- ***causes***: adenoviral, gonococcal, bacterial (chlamydia)

• ***Investigations***: (not done)

- Giemsa stain, nucleic acid amplification, viral culture, point of care immunochromatography, serology, other causes investigation

• ***Treatment***:

- *adeno*: resolve spontaneously in 2-3 weeks
- *topical weak steroids*
- *other*: cold (or warm) compresses for symptomatic relief, topical antihistamines, vasoconstrictors, nonsteroidal anti-inflammatory, topical antibiotics (if bacteria 2ry)

• **Allergic**:
- type 1 hypersensitivity reaction, IgE action

• **3 types**:
A. Acute Allergic Conjunctivitis:
- Hx is typical (farm, pollens,..), acute itching and watering of the eye
- Chemosis (severe conjunctival edema)
- ttt. Adrenaline 0.1%, + cold compress to avoid allergen

B. Seasonal and perennial:
- seasonal (hay fever eye): more common, worse in spring and summer, mostly due to grass, tree's, pollens
- ***perennial***: worse in autumn (milder), dust mites, animal dander, fungal
- ***symptoms***: acute, subacute redness, watering, itching, sneezing, nasal discharge
- ***investigations***: not done
- ***signs***: hyperemia, papillary (mild), chemosis, lid edema (variable)
- ***treatment***: mast cell stabilizers, antihistamines, (both: ketotifen), antihis +vasoconstrictor
, topical steroids, oral antihis (if systemic symptoms present)

C. Vernal Keratoconjuctivitis:
- recurrent bilateral disorder
- both IgE and cell mediated
- 5 yr and older, remission in teens
- Hx. of atopy, family hx
- peak: late spring, summer, or perineal
- dx. Clinical (intense itching, photophobia, foreign body sensation, thick mucoid discharge, more blinking, and lacrimation)
- ***Classification***:
a. palpebral: upper lid.. affects the cornea (rubbing action)
b. limbal: more in Asian and black
c. mixed
• ***Papillae***: hyperemia, if >1mm (giant), mucus deposition between papillae

• ***Keratopathy***:
- more in palpebral disease
- punctate epithelial erosion

- plaques and shield ulcers in palpebral or mixed then inadequate wetting and delayed re-epithelization, leading to 2ry bacterial infection
- subepithelial scars
- treatment: same as allergic seasonal

• Part 4: Degenerative & Subconjunctival Hg:

• Degenerative Diseases:

• ***Pinguecula***:
- *elastotic* degeneration of stroma
- yellow-white elevation of bulbar (*does not cross the limbus*)
- cause: actinic damage (UV, dry)
- *calcification* present mostly (might be prominent lesion)
- might be *inflamed* (pingueculitis)

• ***Pterygium***:
- triangular *fibrovascular* ingrowth *over the limbus* into the cornea
- histology similar to pinguecula
- cause: actinic damage (UV, dry)

• ***Pseudopterygium***:
- caused by a *band of conjunctiva adhering to an area of compromised cornea*
- response to *acute inflammation* (ulcer, chemical burn, trauma, cicatrizing conjunctivitis)

• ***Concretions***:
- associated with aging, multiple tiny cysts (yellow-white deposits), they can be calcified

• ***Conjuctival retention cysts***:
- thin wall lesions on bulbar conjunctiva (consist of clear, turbid fluid)

• Subconjunctival Hemorrhage:

- disappear in 10-14 days, painless, frightening appearance
- DDx. Vit C deficiency, straining and many others..
- might be associated with base fracture: if we can't see the posterior extend of the Hg, if associated with head trauma, if associated with bilateral orbital ecchymosis (Raccoon)
- Treatment: if not spontaneous: topical antibiotics

• Part 5: Lid Disorders:

• **Abnormal palpebral fissure**:
- palpebral fissure: 27-30mm horizontally, 8-11mm vertically
- if <8mm then drooping of eyelid, if >11 then lid retraction

• **Abnormal lid margin position**:
- **Entropion**: eyelid margin inward rotated
- **Ectropion**: eyelid margin outward rotated

• **Abnormal eyelashes**:
- **misdirected**: inward rotated
- **Trichomegaly**: congenital condition, long eyelashes (>12mm central & 8mm peripheral)
- **Madarosis**: loss of eye lashes (madao in greek: to fall)
- **Poliosis**: patch of white hair, due to lack of melanin pigment

• **Eyelid inflammation**:
- the eyelid is very thin and subcutaneous tissue has little fat, the upper eyelid in particular is susceptible to rapid fluid accumulation leading to severe swelling

• **Blepharitis**:
- chronic inflammation of eyelid margin, very common
- might be associated with systemic conditions as acne vulgaris
- can cause secondary ocular diseases like dryness, triachiasis

• **it has 2 forms**:
1. *Anterior*: subdivided into

a. *Seborrhoeic*:
- *greasy oily secretions* of lid margin with scale which are adhered to lashes and the lashes are adherent to each other, also oily skin
- erythema and telangiectasia of eyelid margin

b. *Staphylococcal*:
- more severe, *hard scale crusting* mainly located in the base of eye lashes called collarettes, it may lead to loss of eyelashes, trichosis, poliosis if severe enough
- more associated with blindness, also in long cases: scarring and notching of eyelids

2. *Posterior*:
- *Meibomian gland dysfunction*
- bilateral lipase lead to FFA which increase melting point of the Meibomian leading to prevention of its secretion
- it is highly associated with acne rosacea
- *on examination*: excessive secretion, tooth paste like, oily foamy tear film accumulated at the medial and lateral canthus
- *systemic associations*: seborrheic dermatitis, atopic, acne

3. ***Mixed***

• ***Treatment of blepharitis***:
- *warm compresses* (to liquefy secretions, and remove discharge)
- *topical antibiotics*: chloramphenicol, fusidic acid
- *oral antibiotics* especially in post: tetracyclin, erythromycin
- *topical steroids* if severe
- *hygiene*, *lubricants*, *mixture* (of all of these)

• **Lid Lumps (Tumors)**:

• might be:

1. Non-neoplastic: chalacion, cyst of zeis, cyst of moll, sebaceous cyst, comdeons, epidermoid and dermoid cyst, lipid deposition, epidermal inclusion cyst, millia, chalacion, endocrine cyst of hydrocystoma

2. Neoplasia (abnormal tissue growth):
- Benign: capillary hemangioma
- Malignant: BCC, SCC, Sebaceous gland carcinoma, malignant melanoma

• ***Stye*** (***external hordeolum***):
- acute staph abscess of eyelash follicle and its associated gland of zeis
- red tender nodule at the lid margin
- if it rupture it will heal spontaneously and will have discharge from the external skin, if it doesn't we might need to do incision and drainage and even remove the eyelash with its follicles, also it may extend to the adjacent eyelashes or cause preseptal cellulitis
- treatment: hot/warm compresses with topical antibiotics

• ***Internal hordeolum***:
- Meibomian gland abscess, staph infection
- more painful, tender, it might open and discharge either into the skin or into the conjunctival aspect of the eyelid, if not spontaneous: also incise and curettage
- treatment: topical, systemic antibiotics

• ***Chalazion***:
- Meibomian gland cyst, leads to occlusion of secretion and strangulation, leading to chronic inflammation and granulation tissue formation
- lipogranulomatous inflammation, it is sterile unless there is a 2ry infection
- might be associated with poor hygiene, sebborhea, blephritis
- painless, and the dx. Clinically
- surgery is done form the inner side of eyelid by incision and curettage

• ***Sebaceous gland carcinoma***:
- rare, more serious (BCC: delayed)
- recurrent chalazion in same place, may present with localized blepharitis
- if we incise and drain the Meibomian cyst we should take a biopsy for histopathology

• ***Basal cell carcinoma*** (***BCC***):
- most common malignant (90% of all lid malignancies), also count for 10% of all BCC
- painless, slowly growing, locally invasive, with no mets
- nodular, nodulo-ulcerative, ulcerative rodent ulcer, sclerosing (superficial spread)
- treatment: if small do incisional biopsy to confirm the excision
- prognosis is very good unless deep invasion or incomplete excision

• ***Squamous cell carcinoma*** (***SCC***):
- less common, more malignant, hard to differentiate between BCC, SCC clinically (biopsy)
- can mets to LN, present with nodule, scaly patch, localized blepharitis
- UV is a risk factor

Painful Conditions

• Part 6: Keratitis:

- normally bacteria can only invade when the defenses are compromised
- some bacteria able to penetrate healthy epithelium: N. gonorrhea, N. meningitides, Corynebacterium diphtheria, and H. influenza

• ***Risk factors***:
- *contact lens wear* (hypoxia, trauma, mainly soft lens)
- *Trauma*: refractive surgery (LASIK), agricultural injury
- *ocular surface disease*: trichiasis, allergy, entroption, blepharitis..
- *others*: immunosuppression: DM, vit.A deficiency

• ***Common Pathogens***:
- *Pseudomonas aeruginosa*: gram (-), 60% of contact lens keratitis
- *Staph*. A: gram (+), focal keratitis
- *Streptococci*: gram (+), aggressive (e.g. pyogenes, pneumonaie)

• ***Presentation***:
- severe pain, redness (ciliary injection), discharge (mucopurulent, purulent), photophobia, blurred vision
- ***signs***: circumcorneal injection (ciliary), epithelial defect, stromal edema, dols in Descemet membrane, if severe (chemosis, eyelid swelling, anterior uveitis, keratic precipates, hypopyon (fluid level), posterior synechiae)
- ***note***: anterior synechiae is between iris and cornea, while the posterior is between the iris and the lens
- severe ulceration may lead to perforation (specially pseudomonas), endophthalmitis if with perforation

• ***Treatment***:
- Duo therapy: fortified: cefuroxime (gram +), Gentamicin (gram -)
- Mono: fluoroquinolone (moxifloxacin, gatifloxacin, besifloxacin)
- Mydriatics (cycloplegic: to reduce posterior synechiae)
- Subconjuctival antibiotics
- Steroids (after healing, sterilization of ulcer to reduce scarring)
- Systemic antibiotics: Augmentin (for H.influenza), Ciprofloxacin (antibacterial, can reach periumbilical area), Doxycycline (for anti-collagenase effect to reduce scarring)
- post treatment: improvement is noticed by reduction in edema, chemosis

• ***Herpes Simplex Virus*** (***HSV***):
- DsDNA enveloped virus
- ***2 types***: HSV1 (above waist), HSV2 (below waist), either primary or recurrent
- ***primary infection***: acute follicular, vesicles, young age, maybe as hemorrhagic rash, then it hides in the trigeminal ganglion waiting for recurrent infection
- ***risk factors for severe disease***: atopic eye, childhood, measles and malaria,

inappropriate topical steroids (develop geographic ulceration)
- ***might be as***: epithelial or stromal keratitis, neurotrophic keratopathy, anterior uveitis
- ***DDx of epithelial keratitis*** (***dendritic***): Herpes zoster healing corneal abrasion (pseudodendrite), acanthamoeba, soft contact lens
- ***Dendritic ulceration***: virus in the trigeminal bulbs and enlarges on steroids
- ***Treatment***: self-limiting, acyclovir (for 7-10 days), topical lubricant
- ***Stromal keratitis***: either
a. ***disciform***: immune reaction to virus, epithelium is intact, epithelial and stromal edema with keratopathy, and wessely ring due to Ag-Ab deposit in stroma, treated by topical steroids and anti-viral
b. ***Necrotizing stromal keratitis***: severe form of active viral invasion and destruction of corneal stroma, treated by high dose of topical antiviral and sometimes systemic + lubricants + topical A.B
- ***Neurotrophic keratopathy***: reduced corneal innervation in HSV, HZV
- ***Iridocyclitis***: iris atrophy, defect, wedge shaped due to vasculitis which involve one sector of the iris

• ***Herpes Zoster Opthalmicus***:
- ***Varcilla-zoster*** (morphologically identical but antigenically distinct from HSV)
- ***Hutchinson sign***: involvement of skin supplied by external nasal nerve (branch of nasociliary nerve), note (lower lid is rarely involved), Unlike HSV, there is usually a prodromal period with the patient systemically unwell. Ocular manifestations are usually preceded by pain and the appearance of vesicles in the distribution of the ophthalmic division of the trigeminal nerve. Ocular problems are more likely if the *nasociliary branch* of the nerve is involved, starts by pain in the distribution of ophthalmic nerve then maculopapular rash then small vesicles the pustules and crusting ulcers it stops at the midline and doesn't exceed it
- ***Involvement of eye in acute eye disease*** (***HZV***): might be follicular, papillary, cause uveitis (both A, P) & cause keratopathy (acute, stromal (interstitial) keratitis, nummular

• Part 7: Iritis, Episcleritis, Scleritis:

- painful but without discharge
- ***there are 3 pre-equatorial vascular layers***:
a. ***conjunctival vessels***: most superficial with tortuous vessels
b. ***superficial episcleral plexus***: maximal congestion In episcleritis
c. ***deep vascular plexus***: maximal congestion in scleritis
- constriction to phenylephrine (alpha1 agonist): a (2.5%), b (10%), c (no constriction)

• **Episcleritis**:
- inflam. of superficial layer of sclera, it is recurrent, typically self-limiting (days-3w)
- associated with ocular disease more than systemic: dry eye, lens wear, roaseca (skin)
- in middle aged
• ***Types***:
A. ***Simple*** (75%):
- recur in 60%, >50% bilateral, visual acuity (normal), photophobia may occur
- might be sectoral or diffused, it has inter-palpebral distribution (triangle, base at limbus)
- Treatment: cool compresses, weak topical steroid, topical NSAID (alt), oral NSAID (ibuprofen, or indomethacin – occasionally required)
B. ***Nodular***:
- tender, almost always within the interpalpebral fissure
- flat anterior scleral surface (because sclera is not edematous)
- ttt. Same as simple

Note: differentiate btw scleritis and episcleritis , HOW ? (1) phenylephrine will cause blanching in superficial and middle layers (conjunctivitis and episcleritis) but in scleriris no blanching (2)can be moved over sclera in epi (3) sclera it self is not thick in epi

• **Scleritis**:
- unlike episcleritis (more serious (vision loss risk), needs systemic ttt (systemic vasculitis))
- eye complications: choroidal effusions, macular edema, optic neuritis
- systemic associations (imp!): RA, relapsing polychondritis, PAN, Wegner granulomatosis
• ***Types***:
I. ***Immune mediated***:
- edema and cellular infiltration of the entire thickness of the sclera
A. ***Anterior***:
- anterior to recti muscle origin, discovered earlier than posterior. Because it causes redness and discomfort (unlike the posterior which just causes reduce vision (vision loss)
- Complications: keratitis, uveitis, glaucoma (MCC of loss of vision), hypotony (low IOP), perforation
- subdivided into:
a) ***non necrotizing*** (***simple scleritis***): diffused, nodular (after HZV mostly):
- redness, morning pain, vascular congestion, edema (resolves and the affected area takes on a slight grey/blue appearance due to increased scleral translucency), chemosis, increased ICP
b) ***Necrotizing with inflammation*** (severe form):
- sight threatening, more dangerous: vaso-occlusive (associated with RA), granulomatous, surgically induced (might be infectious)
- later onset age, gradual onset (progressive)

c) ***Necrotizing without inflammation*** (***scleromalacia perforans***):
- no vascular thinning, progressive, 5% of scleritis
- mostly in elderly females with longstanding RA
- S/Sx: mild irritation, absent pain, unaffected vision, keratoconjuctivitis sicca (suspected), no vascular congestion
- treatment: by treating the primary disease

B. ***Posterior***
C. ***Mixed***
II. ***Infectious***: HZV, TB, leprosy, syphilis..

• Part 8: Uveitis:

- inflammation if uveal tract, causes photophobia
- uvea is the middle core of the eye expanding from the CHOROID posteriorly to the CILIARY BODY in its 2 divisions (pars plicata & pars plana) and IRIS
** Pars plicata --> anterior part of ciliary body (secrets aqueous humor).. attachment of zonule , ciliary epithelium , ciliary body
** Pars plana --> posterior part of ciliary body , avascular , doesn't have any important structures so we enter it in surgeries
** the choroid: is formed by arterioles, venules and dense fenestrated capillary network, it's the basement membrane together with the RPE forms bruch's membrane, it has a high blood flow (nourishes the deep, outer retina layers + temp homeostasis)

• ***Classification of Uveitis***:
• ***Anatomical***:
a. Anterior: 50% idiopathic, anterior chamber
- causes iridis or iridocyclitis -->manifest as red eye
- anterior -> iris, plicata /intermediate -> vitreous , plana

b. Intermediate:
- primary vitreous inflammation, includes pars planitis
- manifests as opacities, visual problems

c. Posterior: retina, choroid
- manifest as visual problems, might not be painful
- doesn't cause red eye , most common causative agent is: toxoplasmosis

d. Panuveitis
• ***Etiological***: infectious (toxoplasmosis, syphilis, herpes, CMV, fungal), noninfectious (with/out systemic), masquerade (non/neoplastic)
• ***Onset***: sudden, insidious
• ***Duration***: limited (<3m), persistent
• ***Course***: acute, recurrent, chronic (no 3 m free)

• ***Presentation***:
- ***Eye***: redness, ocular pain, blurred vision
- ***RS***: cough, SOB, sputum / TB, sarcoidosis
- ***Skin***: Ulcers (oral, genital,, Behcet (painful), Reactive (Reiter - Painless)), skin and nail lesions, Psoriasis, Erythema nodosum (Behcet, sarcoidosis), thrombophlebitis (Behcet)
- ***Joints***: Ankylosing spondylitis (HLA-B27), Juvenile idiopathic arthritis (seronegative RF female , ANA+ complications : cataract , glaucoma , corneal opacity , NO PAINFUL uveitis it's pauci-arthritis pausi means few)
- ***Bowel***: UC, CD, Whipple's disease
- ***Renal***: TIN, IgA GN

CAUSES OF UVEITIS

Infectious	Associated with systemic disease	Ocular disease
Toxoplasmosis	Ankylosing spondylosis	Advanced cataract
Postoperative infection	Sarcoidosis	Sympathetic ophthalmitis
Fungal	Reiter's disease	Retinal detachment
CMV	Behçet's disease	Angle closure glaucoma
Herpetic	Psoriatic arthritis	Intraocular tumours
Tuberculosis	Juvenile chronic arthritis	
Syphilis	Inflammatory bowel disease	
Metastatic infection		
Toxocara		

• ***Symptoms in Acute anterior uveitis*** (***AAU***):
- rapid onset of redness, pain, photophobia, blurring of vision, hx of similar episodes
- ciliary injection (perilimbal), conjuctival hyperaemia with a violaceous (purplish) hue due to deeper vessels involvement
- ***Miosis***: sphincter pupillae muscles are stronger than dilator pupillae muscles in anterior uveitis, pupil is miotic and irregular
- ***Active inflammation***: Inflammatory cells in the anterior chamber, aqueous flare (due to protein leakage)
- ***Hypopyon***: sterile exudate composed of inflammatory cells in the inferior part of AC
- ***Keratic Precipitates*** (***KP***): cells and flare in AC after entering of inflammatory components will be called keratic precipitate (Arlt triangle – inflammatory cells), KPs are usually in the lower part of the cornea according to the current of aqeous and thermal distribution , KPs can be small (as in uveitis) or large -called mutton fat KPs (as in granulomatous disease)
- ***Posterior Synechiae*** (***PS***): inflammatory adhesions between pupil and lens

• ***Iris***:
- ***Atrophy***: as in HSV, HZV
- ***Nodules***: signify the inflammatory process .. could be in the middle of iris ,(called busacca nodules) or at periphery of the iris
- ***Crystals***
- ***Heterochromia iridis***: decreased pigmentation of the affected eye due to inflammation
- IOP maybe elevated , decreased or normal if it's elevated , it's mostly due to : - steroids tx, closure of trabeculae meshwork, In acute phase --> ciliary body is inflamed --> decreased production of aqueous humor -->decreased IOP * when pt gets better --->increased production of aqueous humor --> increased IOP
- Posterior segment exam (important for toxoplasmosis

• ***Treatment***:
- Anti-microbial, anti-viral
- Steroids: topical, systemic, ocular and periocular, implants
- Immunomodulators, biologics
- Dilating eye drops

• Part 9: Trauma And Chemical Injuries:

• Chemical Injuries:
- alkali is x2 more common than acids, also more dangerous (penetrate more unlike the acid which tend to coagulate forming layer that protects cornea from further penetration)
- the severity is based on the properties, area, duration, related affects
- most common alkalis: ammonia, sodium hydroxide, lime
- some bad acids like: hydrofluoric acid (in cleaning – penetrate more rapidly), sulphyric acid (car battery explosion – complicated by thermal effects)

• ***Management***:
A. ***Copious Irrigation***: (even before Hx!)
- the most important step in management is Copious irrigation (you use any fluid you have to do irrigation such as: balance salt solution (BSS), ringer, saline, tap water) topical anesthesia, eye speculum, evert eyelid, remove particles
- we evert lower eyelid by pulling it downward, while the upper eyelid is harder (we pull eye lashes downward by hand, put your finger or any device on the upper tarsus then pull upward), or by using the lid retractor (Desmarres)
- we maintain irrigation till the pH is neutral (measured by dipstick)
B. ***Debridement of necrotic areas*** by cotton to promote re-epithelization
C. ***Admission*** to hospital for monitoring

D. ***Grading of severity***:
- on basis of corneal clarity
- severity of limbal ischemia (roper-hall system)
- Other: extent of epithelial loss, iris changed, lens status, IOP
Grade 1: no limbal ischemia, clear cornea – excellent prognosis
Grade 2: hazy cornea (but visible iris), less than 1/3 limbus ischemia – good
Grade 3: total epithelial loss, haze obscuring iris detail, 1/3-1/2 limbal ischemia – guarded
Grade 4: opaque cornea, >50% ischemia – poor

E. ***Medical Treatment***:
- ***mild*** (***1,2***): topical steroids + cycloplegics + topical antibiotic ointment
- preservative free drops should be used
- ***Ascorbic acid*** (***vit.C***): improve wound healing
- ***Citric Acid***: reduce the intensity of inflammatory response
- ***Tetracylines*** (topically tetracycline, systemically doxycycline): collagenase inhibitors, inhibit neutrophils, reduce ulceration
- ***Symblepharon*** formation should be prevented by lysing it with a sterile glass or cotton (adhesion between bulbar (fornix) conjuctiva and palpebral (eyelids) conjunctiva, so it leads to reduction of the size of conjuctival sac causing epiphora + less lubrication of eye)
- ***IOP monitoring***: oral acetazolamide to avoid adding further to ocular surface burden

F. ***Surgery***:
a. ***Early***:
- to revascularize the limbus, restore limbal cell, re-establish fornices
- surgeries: advancement of tendon capsule, limbal stem cell transplant, amniotic membrane grafts, gluing or keratoplasty
b. ***Late***:
- conjuctival bans and symblephara
- conjuctival, or other mucus membrane grafting
- correction of eyelid deformities
- keratoplasty (for corneal scarring – should be delayed for atleast 6 m for maximal inflammation resolution)
- Leratoprosthesis (if severely damaged)

• Foreign Bodies and Corneal Abrasions:
- either external (on cornea, conjuctiva), or intraocular, superficial
- the presence of vasculization indicated that the FB is here from several days
- Rust ring around the Fb indicates that it goes deeper
- if the FB is stuck to cornea give topical anesthesia and do irrigation > cotton > if still there remove it > topical antibiotics + mydriatics
- Intraocular FB needs axial and coronal CT scan

• **Hyphema**:
- collection of blood in the anterior chamber (bleeding from the iris root or ciliary body face), might produce D-shaped pupil (iris torn from insertion – iris dialysis)
- mild-severe ocular damage
- associations: iris dialysis, AC angle damage (increased IOP), traumatic cataract, lens displacement, posterior segment injury
- Treatment: conservative, hospitalize (if severe), steroid eye drop with mydriasis, vit C, monitor IOP
- Surgery: if elevated IOP (persistent high IOP >35 for 7d), Corneal blood staining, Secondary Hg, Persistent clot >10d, Sickle cell disease

• **Blunt Trauma**:
- direct damage to globe could occur by transmission of pressure which may cause sudden anterio-posterior compression of the globe and widening of the globe horizontally then it returns back to normal position this sudden rapid movement causes damage to the eye as: hyphema, Vossius ring (due to compression of the iris against the lens), rupture of sphincter pupillae (semi dilated pupil, also called traumatic iridoplegia or traumatic mydriasis), iridodyalisis, traumatic cataract, dislocated/subluxated lens, rupture (limbal opening)

• **Periocular Hematoma**:
- black eye, rule out other causes (basal skull, orbital wall fracture, trauma to globe, orbit)

• **Blow-out fracture (floor fracture)**:
- object with diameter > globe diameter (>5cm) .. increase globe pressure, globe cant absorb this pressure, instead it will transmit to orbital wall causing orbital wall fracture
- thinnest bone: ethmoidal bone, weakest bone: floor of orbit

• ***Presentation***:
- periocular: ecchymosis, edema (exophthalmos), emphysema (due ethmoidal sinus)
- enopthalmos
- infraorbital nerve anesthesia
- diplopia (binocular): entrapment, direct injury to extraocular muscle
- damage to inferior rectus (upgaze)
- restriction of movement
- ocular damage

• ***treatment***:
- if mild no ttt, if severe (muscle entrapement, enopthalmous >2mm, severe diplopa) needs ttt and maybe surgery

• **Orbital (retrobulbar) hemorrhage**:

- associated risk of acute orbital compartment syndrome with compressive optic neuropathy, can lead to irreversible blindness, it can occur without or in association with orbital bony injury
- ***surgery***: lateral canthotomy and cantholysis to spread the pressure and decrease it

• **Lacerations**:

- ***Lid lacerations***: risk of levator damage, ptosis,, clinically imp. If deep, if involving eyelid margins, if the medial aspect (lacrimal drainage – involving of lower canaliculus system in the medial aspect requires special attention, management and surgery)
- ***Penetrating corneal laceration***: the pupil won't be rounder and regular as before, if there was an irregular pupil (investigate immediately)
- ***Penetrating scleral laceration***

Refractive Errors

• ***Biometry***: test used to calculate power of IOL by measuring size, shape (inputs: axial length, corneal power)
• visible EM spectrum is between 390-760nm
• ***focusing components of the eye are***: 2/3 cornea, 1/3 lens, axial length (23-24mm)
• the power of refraction is measured by diopters (D)
• ***Emetropia***: normal, Ametropia: abnormal (myopia, hypermetropia, astigmatism)
• Concave lens (negative – divergent, concave), Convex (positive – converging, convex)

• Myopia:

• ***Types***:
1. ***Axial***: big eye >24mm (1mm > 3Ds)
2. ***Curvature***: steeper more convex (1mm > 6Ds)
3. ***Index***: higher diopters, seen in nuclear sclerosis cataract (normal is 1.42)
4. ***Abnormal position of the lens***: the more anterior the stronger lens

• ***Complications***: (mostly axial):
- retinal tear, PVD, RD (due to big and thin eye), open angle glaucoma, cataract, staphyloma (thinning of sclera and expansion)
• ***Treatment***: negative concave lens
** Child with myopia: check IOP if increase it will increase the eye size and cause myopia
** Normal IOP is 16 +- 5

• Hypermetropia:

• ***Types***:
1. ***Axial***: small eye (<23mm)
2. ***Curvature***: flat
3. ***Index***
4. ***Abnormal position of the lens***: the more posterior the less power of the lens

• ***Complications***: Angle closure glaucoma (crowded eye), pseudopapilloedema, amblyopia
• ***Treatment***: by positive convex lens

** After birth the eyes are small and start the process of emmetropization until reach the normal axial length (AL) at the age of 10-12 years

** Visual Acuity: we examine the best corrected distant visual acuity, by: looking at distant objects sitting on >6m wearing the glasses if present (no accommodation)

** Decreased VA is either from refractive error or non-refractive error (cataract, neuritis), to differentiate we use Pinhole test if the acuity is corrective then its refractive (regardless which type)

• Astigmatism:

- non-spherical lens or cornea, more than one focal point
- like the rugby ball not spherical we have vertical line and horizontal line (one is steep)
- Regular astigmatism: two lines are 90 degree to each other if not irregular
- regular is treated by glasses or soft contact lens, while irregular by rigid
- Regular: with the rule (the vertical is steeper)
- Sub-clinical: vertically steeper than horizontally by 0.5 diopter this is normal

• ***Subtypes***:

- ***Simple***: focus point on retina, another either posterior or anterior
- ***Compound***: two point either anterior or posterior
- ***Mixed***: one is anterior other is posterior

• ***Treatment if mixed***: we start by one either hypmetropic, myopic with angle (cylindrical)

• Presbyopia:

- physiologic aging process, lens rigid more concave (hyperopia), inability to see near, lens capsule less elastic, weakening of ciliary muscle

• Aphakia:

- absence of the lens
- pt is hypermetropic due to absence of lens (1/3 of refractive power)
- ***Causes***: congenital, surgery (MC is cataract surgery), trauma
- ***Diagnosis***: Iridonesis, Dark-black pupil, one reflex (corneal light reflex), hypermetropia

• ***Pseudophakia***: after cataract surgery (3 light reflexes)

• ***Treatment***: IOL, contact lens, glasses

• Keratoconus:

- it will develop myopia (more convex cornea), progressive disorder (myopia > myopia + astigmatism > conical shape)
- ***Causes***: genetic (down, collagen disease, atonic allergy), environmental, mechanical (rubbing eyes)
- ***Treatment***: stage dependent.. we use collagen cross linking to stop the progression and eventually we might need keratoplasty

• Anisometropia:

- different refractive error in each eye and the difference is more than 2 Diopters
- might lead to amblyopia or unilateral aphakia

• **Refractive Error Correction**:

- ***Contact lenses***: soft, rigid, hard, toric (spherical & cylindrical)
- ***Glasses***
- ***Surgery***: IOL, Laser, complicated surgeries

• ***Refractive Surgeries***:

- PRK (photo-refractive keratectomy)

Chronic Visual Loss

• *Gradual decrease in vision painless causes*: cataract, open angle glaucoma, ARMD..
• ***Vision loss can be***: (Acute/Chronic) (Sudden/Gradual) (Painful/Painless)
→ ***sudden painless*** ➔ mostly vascular cause (CRAO, CRVO, RD, ischemic
→ ***sudden painful*** ➔ (optic neuritis, closed angle glaucoma)
→ ***Gradual painless*** ➔ (cataract, open angle glaucoma, DM retinopathy, corneal opacification , degeneration & dystrophy)

• Cataract:

- opacification of the lens, MCC of treatable blindness
• ***Causes are categorized into***: age related (senile – environmental: smoking, UV,..) and pre-senile: associated with ocular, systemic conditions

- ***ocular***: trauma, uveitis, *high myopia*, IO tumors
- ***systemic***: DM, Down, congenital cataract, metabolic, infection (TORCH – rubella)

• ***Senile cataract***: main types:
1. ***Nuclear***:
- MC, might cause angle closure glaucoma (because the lens become so big)
- second sight phenomena – round cataract lens is more powerful and offsets the co-existing presbyopia so the elderly can read better
- Exaggeration of normal nuclear ageing change, causes increasing myopia
- Increasing nuclear opacification, initially yellow then brown

2. ***Cortical***:
- opacification when changes in the water content of the periphery causing fissures (white spokes of wheel pointing inwards)
- initially vacuoles and clefts, then progressive spoke like opacities
- Sx: problems with glare and light scatter at night

3. ***Posterior subcapsular***:
- forms at back of the lens, they occur in pt on steroids, diabetes, those with hx of ocular inflammation
- opacities looks like breadcrumbs or sand sprinkled (they create vision difficulty although appearing not harmful on slit lamp exam)

• ***Sx of Cataract***: painless progressive loss of vision, glare, refractive error, amblyopia

• ***Signs of Cataract***: less acuity, lens opacity, black spot against red reflex, location of C

• ***Investigations***: acuity, fundoscopy (to rule or retinal problems), further (if systemic/congenital/early age)

• ***Treatment***: Surgical
1. ***Phacoemulsification***: Steps:
- Capsulorhexos
- Hydrodissection
- Sculpting, Cracking of nucleus
- Emulsification of each quadrant
- Cortical cleanup and insertion of IOL
** in phaco and ECCE we remove lenticular material leaving part of the lens to give support of IOL implanted, while in ICCE we remove the whole lens
2. ***Extracapsular cataract extraction*** (***ECCE***)
3. ***Intracapsular cataract extraction*** (***ICCE***)

• ***Post-op care***: steroids, antibiotics, near vision adds
• ***The differences are***:
1-Sutureless Phaco.
2-Small wound in Phaco.
3-Fast visual recovery in Phaco.
4-Foldable lens in Phaco, hard lens in Extra.
5-Less complications in Phaco

• ***Complications of Surgery***:
- ***Pre-op***:
a. ***Ocular***: conjunctivitis, Glaucoma, improper calculation of the IOL
b. ***Systemic***: Uncontrolled (hypertension, diabetes, IHD… etc.)
- ***Intra-op***: **anesthetic complications**: depend on anesthesia type (Local, Topical, General)
- ***Post-op***:
a. ***Early***: Iris prolapse, Corneal edema, Endophthalmitis
b. ***Late***: refractive errors (1.wrong power of IOL. 2. Tight sutures: they induce astigmatism), RD, Glaucoma, Posterior capsule opacification (Poor visual acuity=> treatment…. Using laser), cystoid macular edema

• ***Congenital Cataract***:
- 1/3 hereditary AD, 1/3 syndrome or metabolic, 1/3 unknown
- one of the most common causes of blindness in children
- risk of amplyopia [lazy eye] at age of 5-6 years (unilateral cataract is worse cause bigger difference between eyes)

- the best age to do surgery is before 2-3 months
- the surgery: lensectomy and vitrectomy (will result in aphakia, which is as amblyopiogenic as the cataract itself, so we treat aphakia as well)
- IOL after the age of 2 years (not before)

• Glaucoma:

- mostly due to increased IOP (normal: 16 ± 5) It's a heterogeneous group of diseases all of which are characterized by progressive characteristic optic nerve damage with progressive characteristic visual field changes with or without raised IOP

• ***Aqueous Humour***:
- ***Production***: by ciliary processes
- ***Excretion***:
Conventional (95% - meshwork, schlemms canal),
Uveoscleral (5% - iris, ciliary body > supra choroidal space, venous system > sclera)

• ***Optic nerve damage***: by raised IOP (indirect effect) and ischemia of nerve fibers

• ***Cup to Disc Ratio*** (***CDR***):
- measure to assess the progression of glaucoma
- normal CDR is 0.1-0.4
- The cup area doesn't include nerve tissue or dural tissue, the surrounding area is orange pink contains nerve fibers and dural tissue, in glaucoma we have atrophy of neuroretinal rim, cup becomes larger, CDR increases to 0.5-0.7, ending with optic atrophy.
If we find one eye 0.1 and the other is 0.4, This means that glaucoma is advancing in the eye because it's bilateral asymmetric disease. 0.4 eye was 0.1 and increased!
If both eyes were 0.7 it's considered normal, we care about symmetry.
It's called physiological cupping. so with disease progression we have increase in CDR

• ***Classification of primary glaucoma*** is based on whether the or the iris is clear of meshwork then ***open*** angle (***POAG***), if covered then ***closed*** (***PCAG***)

• ***Angle is examined by***: Geniolenses (Genioscopy)

• Glaucoma is familial disease of elderly.

• ***End stage***: *Tunnel vision* [wide area is lost], it is usually asymptomatic (present late, so screening for every 40y every 2 years is required)

• ***Treatment***: (aim to lower IOP – cause it's the only modifiable factor)

1. ***Medical therapy***:

Topical Agents			
Agent	**Examples**	**Action**	**Side effects**
β blockers	- Timolol - Betaxolol	↓ secretion	Exacerbate asthma & COPD - Timolol (cardiac non-selective) - Betaxolol (cardiac selective)
Parasympat-mimetic	- Pilocarpine	↑ outflow	Induce myopia (blurring)
Sympatho-mimetic	- Adrenaline	↑ outflow & ↓ secretion	Redness
α-2-agonist	- Apraclonidine - Brimonidine	↑ uveoscleral pathway & ↓ secretion	Redness, fatigue (sleepiness)
Carbonic Anhydrase Inhibitors (CAI)	- Dorzolamide - Brinozolamide	↓ secretion	Stinging, bad taste
Postaglandin analoges	- Latanoprost - Travaprost - Bimatoprost	↑ uveoscleral pathway	↑ Pigmentation of iris & skin (↑ melanocyts – bad if unilateral, and we should avoid it in patients with colored iris), lengthening & darkening of lashes, conjunc. hyperemia
Systemic Agents			
Agent	**Examples**	**Action**	**Side effects**
CAI	Acetazolamide	↓ secretion	tingling & numbness in 100%!, might lead to SJS (acidosis), renal stones

** ***Note***: if IOP is still high then change/add another medication or go for laser, surgery

** ***How can topical B-blockers have systemic effects***? Because topically used drugs come in very high concentrations so they are absorbed by the mucosa in larger amounts, How to overcome these effects? By pressing over the lacrimal duct for 2 min to prevent leakage of tears to the nasolacrimal duct > nasal mucosa > systemic effect.

2. Laser therapy: *laser Trabeculoplasty* (burn in trabecular meshwork), it improve the aqueous flow, it is a temporary solution for 4-5 years

3. Surgical therapy: *Trabeculectomy bleb*;

we create fistula between posterior chamber & subconjunctival space bypassing the angle Open sclera and Iris > fluid go from sclera / *Peripheral iridectomy*: we create a 1 way valve

• Age Related Macular Degeneration (ARMD):

- commonest cause of irreversible visual loss in developed world (used to be 2nd to DM)
** to see in light cons work and rods off, and vice versa

** Normally RPE metabolizes the outer segment of photoreceptors and get rid of lipofucin and other metabolites, with aging this function decreases and metabolites accumulate in Bruch's membrane, these metabolites can be seen with ophthalmoscope as yellow subretinal lesions called ***Drusens***

• *We have 2 types*:

A. Dry/Non-exudative ARMD:

- Dry [no blood vessels] hallmark: *Drusens and choroid retinal atrophy*.

B. Wet/Exudative ARMD:

- Wet; hallmark is *choroidal neovascularization* (*CNV*) [result from VEGF] that builds collaterals, but here the process of new blood vessels building occur *haphazardly* in uncontrolled pattern > neo vessels [neoplastic abnormal, uncontrolled mechanism], These vessels are abnormal, fragile, lack important structures in their walls so they leak easily, rupture easily causing hemorrhage > plasma and cholesterol deposition > edema and exudation, eventually scaring and foveal detachment

Dry	Wet
1- Tissue is atrophic 2- no evidence of scarring 3- slow vision loss and painlessly worsens. 4-Both eyes may be affected simultaneously.	1- Abnormal blood vessels (CNV) under macula. 2-These vessels may leak fluid and blood under the retina in that area. Eventually scar develops 3- Initially in 1 eye but eventually affects both 4- Loss of vision tends to progress quickly and may be particularly sudden if one of the abnormal blood vessels bleed.
Management	
- No ttt available for atrophic retina - reassure that total blindness won't occur - ARED studies: Vit.A (Bictins) + Anti-oxidants delay progressions	- we use ***Anti-VEGF*** (for neovascularization – Anti-angiogenics): Bevacizumab (***Avastin***), Ranibizumab (Lucentis), Aflibercept (Eylea) ** Costly and should be repeated monthly ** given intra-vitreous ** Lucentis, Avastin has same efficacy

Acute Vision Loss

Retinal Detachment

• ***Definition***: separation of neurosensory retina from the RPL

** the most superficial layer in retina is nerve fiber layer (the axons), ischemia in this layer shows Cotton-wool spots (irregular margins, we can't see the blood vessel – superficial spots), and Exudates (we can see the BV – Deep)

• ***3 Types***:
1. Retraction retinal detachment (Tractional): neovascularization > fibrous component (no tear) > no sub-retinal fluid, commonly seen in Diabetic Retinopathy, CRVO (ischemic)
2. Exudative/Serous retinal detachment: any pathology that affects choroidal vascular permeability, such as: choroiditis, choroidal tumors, systemic steroids
3. Rhegmatogenous RD (with tear): result from dynamic vitreoretinal tractions (Posterior Vitreous Detachment *PVD*, fluid accumulate in *subhyaloid space*) & peripheral retinal degeneration
• ***RF of RD***:
- *Aging (v.imp)*: degeneration to collagen of the vitreous occurs with aging > Liquaefaction > vacuoles in posterior vitreous > it becomes heavy and detaches
- *myopia* (macular holes), FHx, trauma, cataract
- Retinal surgery: as in cataract surgery (phacoemulsification – you may dmg the posterior capsule and vitreous leak will be at higher risk, if so.. we stop the surgery cause of RD risk)
• ***Complications of PVD***: *no (90%),* Tears (10%, U shaped), avulsion without tear is rare

• ***Myopia and RD***:
- *40% of all RDs occur in myopic eyes*
- *predisposing factors*: *lattice* (peripheral retina becomes atrophic in a **lattice** pattern and may develop tears, breaks, which may further progress to RD) *and snail track degeneration*, chorioretinal atrophy, vitreous degeneration, vitreous loss during cataract surgery, posterior capsulotomy

• ***Clinical Features***:
- Rhegmatogenous RD:
- *Sx*: flashes (photopsia), floaters, peripheral visual loss (relative scotoma)

- *Signs*: RD leads to sub-retinal fluid (lower IOP), curtain like vision (peripheral vision loss), vitreous hemorrhage, *RAPD*, vitreous opacity, retinal breaks, retinal appearance (*convex* - due to sub-retinal fluids)

** the tear is mostly in the periphery, if it's in the central (metamorphosia, blurred vision without floaters and photopsia)

** Tobacco dust eye is another sign of cells within the retina
** RD with funnel shape means the retina is separated from the disc

• ***Examination techniques***: *indirect ophthalmoscopy* (fundoscope – you have to dilate the pupil by: cyclopentolate, phenylephrine, tropicamide) and *slit lamp*, *indirect briomicroscopy*, *B-scan US*

** Note: *MC site of detachment: superior temporal*
** *Upper > More serious* (because against gravity – so pushing)

• ***ER situation depend on***: state of *macula*, (ON/OFF), *duration* of detachment

• ***Treatment of RD***:
** we treat retinal tear to prevent it from extending to RD by ***Argon Laser Retinopexy*** (around the tear not on it because of sub-retinal fluids – also used in mild RD)
** *we have to advice the patient*: based on the site of the tear: if left eye temporal (left cheek to the pillow), right eye nasal (right cheek to pillow), inferior tear (semi-sitting) to prevent it from progressing
** ***treatment is based on the Macula***: if ***ON*** (emergency, we do surgery within 24 hr, the visual acuity is preserved), if ***OFF*** (the pt can wait but the acuity is decreased), the Macula can be protected by positional advice
• ***Specific techniques for treating RD***:
A. Pars Plana Vitrectomy with Tamponade (for retinal support)
- most common , note: if there is vitreous loss you should stop
- Tamponade types:
a. ***Gas***: be careful of IOP by positional advice, and it will be absorbed after 6 weeks
b. ***Silicon oil***: you have to remove it after 6 w (not absorbable), it is used in PVR (proliferative vitreoretinopathy)
B. Scleral Buckling: if you can't do vitrectomy
C. Pneumatic Retiopexy (***Bubble***):
- only for superior RD, and mild RD

• **Imp:**
- patient with loss superior visual field >> inferior retinal detachment
- patient with loss inferior visual field >> superior retinal detachment
** ***who comes first***? superior visual field defect is neglectable by the patient more ,so inferior retinal detachment is chronic (2-3 months) so patient with superior retinal detachment comes first (we need our inferior visual field more ,and losing it will affect the whole life style) >>and this is a bless ^_^ because which detachment can progress to macula more ?? the superior ,it takes hours but inferior retinal detachment days-weeks and sometimes it is never reach the macula .

Retinal Vascular Occlusions

• ***Retinal Vein Occlusion***:
- *Risk factors*:
A. *Systemic* (DM, HTN, Age, Atherosclerosis)
B. *Ocular* (glaucoma, hypermetropia (crowded eye), periphlebitis)
- *Venous stasis conditions* (hypercoagulability state, autoimmune diseases)
** the central retinal artery and vein enter the eye through the optic nerve and they share the same sheath (limited adventitial sheath), once the artery becomes atherosclerotic it will compress the vein and cause venous occlusion

** we divide the fundus at the optic disc to 4 quadrants (Superior temporal and nasal, and Inferior temporal and nasal), each 2 branches originate from same trunk (Superior trunk: superior nasal, temporal / Inferior trunk: inferior nasal, temporal), CRVO is occlusion in one of the branches not the major trunk

• ***Vein Occlusion Types***: Central Retinal Vein, Branch Retinal Vein:
A. Branch Retinal Vein Occlusion (BRVO):
- *Sx*: Sudden loss of visual acuity and/or relative (partial) visual field loss, RAPD
- *Signs*: hemorrhages, dilatation, tortuosity
- *complications* (*causes of blindness*): Chronic macular edema, neovasculization (retinal, ttt. Laser photocoagulation)

** ***Inferior Retinal Vein Occlusion***: signs: cotton wool (ischemia), indistinct disc margin, flamed shaped hemorrhage (leak of RBC's), decreased acuity, visual field might be affected (the superior field), central vision is affected (cause of edema), and hard exudates (indicates leakage of lipids and lipoproteins)

** *Differentiation between soft and hard exudates*: soft (pale, white, ill demarcated), hard (yellow, multiple sizes, regular, well demarcated)

B. Central Retinal Vein Occlusion (CRVO):
** CRV: is the main drainage of the eye
** Definitive Diagnosis by: Fluorescein Angiography (FFA)

• ***Non-Ischemic***: 75% of cases
- *Sx*: sudden painless loss of visual acuity (due to cystoid macular edema - Note: *NO RAPD*)
- *Signs*: tortuosity of all veins, mild-moderate (retinal hemorrhages, optic disc swelling)
- *Prognosis*: 15% to ischemic in 4 m, 34% in 3 y, *acuity to normal in 50%*
- *treatment*: anti-VEGF (because the macular edema won't respond to laser)

• ***Ischemic CRVO***:
- *Sx*: more profound, severe sudden loss of visual acuity (counting finger - there is RAPD)
- *Signs*: afferent papillary defect, more hemorrhages, exudates & severe optic swelling
** on fluorescein – no perfusion area unlike the non-ischemic
** deeper than non-ischemic so more darker
- *prognosis*: acuity loss is permanent (due to macular ischemia), *Robeosis iridis (Neovascularization on the Iris and retina)*, 2ry angle closure glaucoma (after 3 months - 100-day glaucoma – so there is screening by geniosopy – ttt. PRP – Pan retinal photo-coagulation)
- *Management*: macular edema (Anti-VEGF, Argon laser (to stimulate RPL to absorb fluids before Anti-VEGF), steroids, & vitrectomy), neovascularization (PRP, vitrectomy if severe)

Ischemic	Non-Ischemic
Cotton wool	No Cotton wool
RAPD	No RAPD
Blot-dark-deep Hemorrhages	Flamed Hemorrhages & congested BV
Vision is affected more, & has worse prognosis	Less damaging

• ***Arterial Occlusions***:
- sudden dramatic visual loss for more than 24 hr (if less: amurosis fugax)
- *causes*: *embolization* (e.g. cholesterol, thrombus, fibrinic platelet, vegerations – calcific), *vaso-obliteration* (atherosclerosis, hematological disorders, DM, HTN, smoking)

• ***Central retinal artery occlusion (CRAO)***:
- acute total visual loss, most common cause is atherosclerosis
- *Signs*: RAPD, on fundus examination: pale retina (cloudy edematous), cherry red spot of the macula, narrowing and segmentation of arteries
** 10-20% of population have cilioretinal artery which supply parts of the retina (Macula) even in CRAO (extra-blood supply)
- *Treatment*: interfere immediately, reduce IOP (to dislodge the emboli, by: pressure and massage, CAI (acetazolamide), re-breathing to increase CO2, and AC paracentesis (as a last chance))
- *Prognosis*: usually disappointing, if more than 12 hr (ischemia)

** ***Laser Types***:
1. YAG Laser: used in peripheral iridotomy (PI)
2. Argon Laser
3. Femto-Laser
4. Excimer Laser (used in refractive error surgeries like hypo-metropia LASIK)

Neuro-ophthalmology

• Pupillary Disorders:

• ***Sphincter papillae*** (***CNIII***): supplied by parasympathetic fibers: meiosis
• ***Dilator papillae***: supplied by sympathetic fibers: mydriasis

• ***Afferent and Efferent Pathways***:
- Retina > CNII > Lateral Geniculate Body > Pretectal Nucleus > Bilateral synapse in Edinger Westphal > CNIII > ciliary ganglion > short ciliary nerves

** Any retinal will cause afferent pupillary problem
** there is no condition in which the light reflex is intact but the near reflex is defective
** the ciliary body receives 9x more nerves than iris sphincter
** near triad is accommodation, convergence, constriction
** normally both pupils are equal in size, only 20% has anisocoria without disease

• ***Causes of pupil disorders***:
A. Ocular causes:
- ***inflammation*** (irregular pupil, due to posterior synechiae)
- post-ocular ***surgery*** (physical dilation, iris injury)
- ***trauma***: blunt, penetrating: mydriasis
- severe, acute rise ***IOP*** (acute glaucoma – fixed mid dilated pupil due to ischemia, even if you decrease in IOP it remain dilated)

B. Neurological:
- ***sympathetic interruption***: syringiomyelia (upper limb wasting), small cell carcinoma (pancost syndrome), neck injuries, cavernous sinus disease, congenital Horner (associated with heterochromia)
- ***parasympathetic interruption***

- ***Horner's syndrome***: interruption of sympathetic, traid: miosis (even in dark room the pupils remain constricted), partial ptosis (levator palpebrae), ipsilateral anhidrosis, maybe enophthalmus (sunken eyes), for Dx. Alpha agonist (apraclonidine), cocaine, hydrocyamphetamine

- ***Argyll Robertson pupils***: bilateral small assymetrical, irregular pupils (constrict to near but not to light – light-near dissociation), difficult to dilate, specific for neurosyphilis (other: DM, obese, dorsal brain lesion), suggested that the cause is a peri-aqueductal lesion on the dorsal aspect of edinger westphal nucleus

- ***Adie's tonic pupil*** (***myotonic pupil***): unequal pupil size (tonically dilated pupil that reacts more to accommodation than light – light near dissociation), in young adults, not serious, females are more common, it is due to a ciliary ganglionitis (infection to ciliary ganglion) which denervates the parasympathetic to the iris, ciliary body, the pupil is enlarged and poorly reactive to light pupil movement (slow, worm like (vermiform contraction in response to light)), might also show slow, sustained meiosis on accommodation (due to supersensitivity), the supersensitive sphincter constricts to dilute pilocarpine (0.05%, while normal 1-2%, diagnostic test), we examine it by taking the diameter in light and in dark and compare

- ***Midbrain pupil*** (***midbrain lesion***): affect the pretectal nuclear complex (disrupting the retinorectal fibers but preserve the supranuclear accommodative pathway), causes are demyelination, infarction, 3rd ventricle enlargement, tumors (pinealoma – parinaus syndrome (can't look at the vertical axis)

- ***RAPD*** (***marcus gunn pupil***): pupils are equal but light reflex in the affected side is reduced, while the accommodation is intact, tested by swinging light, causes are optic nerve damage, total retinal damage, while media opacity (cataract, hemorrhage, scar wont cause RAPD)

C. Drugs:
- ***muscarinic blockers*** (cyclopentolate(24 hr ½ life), atropine(2w), tropicamide(6hr))- dilate
- ***alpha agonist*** (phenyphrine, adrenaline) – dilate
- ***Muscarinic agonist*** (pilocarpine) – constrict
- ***systemic***: atropine, adrenaline (dilate), morphine (constrict)

• Swollen Optic Disc:

- the optic disc is the blind spot, normally is temporal in visual field
- ischemic optic neuropathy, papilledema, optic neuritis (all will end by pale optic disk)
- ***defined by***: hyperemia, loss of cupping, indistinct margin, loss of SVP (spontaneous venous pulsation – seen normally in 80%, absent as an early sign of swelling)
- ***3 C's of disc***: contour (margin), cupping, color (normally pinkish rim to orange)
- normal CDR (cup to disc ratio: 0.2-0.4
- ***cupping***: central white depressed area, devoid of nerve fibers
- the veins are wider and darker than arteries

• ***Causes***: central or localized / bilateral, unilateral:
- ***Bilateral***: high ICP (papilledema – vision intact but if severe they vision might get affected in positions – obscurations of vision), malignant HTN (vision reduced)
- ***Unilateral***: space occupying lesions (vision reduced), papillitis (inflammation of the optic disc – vision is reduced), ischemic optic neuropathy (sudden visual loss), central retinal vein occlusion (CRVO - sudden visual loss)

• **Papilloedema due to raised ICP**:
- exclude space occupying lesions, if there was no lesions then the MCC is idiopathic intracranial HTN (mostly young, obese females, might lead to optic atrophy)
• ***high ICP presentation***: obscurations of vision, headache, nausea, vomiting, diplopia (6th nerve), focal neurological, hx. Of head trauma
• ***Raised ICP signs***: swollen optic disc (edges blurred, capillaries dilated, no spontaneous venous pulsation (5-20% normal), large blind spot, visual field constriction, focal neurological signs
• ***Investigations***: BP, CT, MRI, MRA, LP
• ***Treatment***: reduce ICP by: medications (acetazolamide), VP shunt, optic nerve decompression, neurosurgery (if space lesions found)

• **Optic Neuritis**:
- inflammation or demylination of optic nerve
• ***can be in form of***:
a. ***Papilitis*** (nerve head involved – disc swollen)
b. ***Retrobulbar neuritis*** (optic nerve involvement is posterior – without disc swelling)
• ***Causes***:
- Demyelination: MS
- Compression
- Inflammation: Sarcoidosis
- Malignant lymphoma, parainfectious in children
• ***Presentation***:
- Acute loss of vision that may progress over few days then slowly improves but it will never get better than before the attack (high recurrence rate 80! – cumulative dmg)
-Pain upon eye movement due to stretching of optic nerve sheet.
- Focal neurological defects 30-70% as in MS
• ***how will the patient come***?
- ***Gender***: more common in females.
- ***Age***: young 20's-30's.
-Complaining of blurred vision in one eye.
*if the pt has one attack with peri-ventricular plaques (more than 2,) then this increase the possibility of having MS. *the more episodes the pt has, the worst his vision become.
- on examination nearly everything is affected (color, vision, blind spot..)

• ***Management***:
- test 7 things for the functions of the optic nerve
- MRI, ESR, CRP as investigations
- Systemic IV steroids then oral to accelerate the recovery of vision (never use oral alone)
• ***Prognosis***: variable, high recurrence rate

• **Ischemic Optic Neuropathy**:
• ***Two types***: ***arteric*** (inflammatory 25%), ***non arteric*** (non-inflammatory, not painful 75%)
- non arteric is mostly due to atherosclerosis, thrombosis, elderly, HTN, DM, hyperlipid
- arteric: worse visual loss, jaw claudication, headache, scalp tenderness, constitutional Sx
- in arteritic: ESR is very high and treatment is systemic steroids, it is an ER
- Non-arteritic is more associated with myopic eye or hypermetropic eye? In hypermetropic eye cause it is SMALL CROWDED eye so this increase the chance of vascular occlusion, and if you examine the other eye you will find that its optic disk is small and we call it = disk at risk (risk of vascular occlusion and ischemic disk neuropathy)
- in non-arteric the rate of other eye involvement 50% (rapid in arteric)

• ***Presentation***: sudden loss of vision, can cause blindness, painful/not

• ***Giant cell arteritis*** (***GCA***):
• ***Presentation***:
1. Old patients – autoimmune – mainly medium sized arteries affected
2. Sudden loss of vision "permanent not temporary "
3. Headache, malaise, shoulder pain
4. Jaw claudication "very pathognomonic "= you ask the patient if he has jaw pain at the middle of his meal, he has to stop eating for 2 minutes then he can resume his
5. Scalp tenderness
6. Proximal muscle weakness and pain (in shoulder as example)
7. Night sweat

• ***Signs***: decreased VA, VF defect, swollen (or Hg) disc (chalky white), tender, pulseless, cord like temporal artery, marked RAPD
• ***Investigations***:
- ESR (>100 – normal value age/2 (+5 for females)), CRP – they decrease after treatment
- *Definitive Dx is temporal artery biopsy* (needs 3-4 days) – skip areas
- Steroids
- in non arteric: CBC, blood sugar and pressure, autoimmune workup
• ***Treatment*** (***for arteric***):
- *immediate treatment is IV with oral steroids* (until biopsy show up)
- steroids will relive headache, but will not improve vision (unlike neuritis – permanent) but will prevent the other eye from being affected

• **Optic Atrophy**:
- pale featureless swollen optic disc (loss of nerve fibers at head), vision/color affected
- vascularity of the disc is lost, extensive cupping
- RAPD present
- ***Causes***: nerve compression, neuropathy, glaucoma, neuritis, rise ICP.. etc

• Cranial Nerve Palsies:

- all superior are intorters, and all oblique's are abductors
- ***SR***: 1 elevation 2 adduction 3 intorsion
- ***SO***: 1 depression 2 abduction 3 intorsion
- Unilateral diplopia as in cataract, macular edema, corneal scarring, vitreous opacity
- when testing diplopia we care for: is mono-ocular, or bi-ocular, horizontal or vertical, and what is the worst gaze

Medial rectus	CN III	Adduction	--	--
Superior rectus	CN III	Elevation	Intortion	Adduction
Inferior rectus	CN III	Depression	Extortion	Adduction
Inferior oblique	CN III	Extorsion	Elevation	Abduction
Superior oblique	CN IV	Intorsion	Depression	Abduction
Lateral rectus	CN VI	Abduction	--	--

• **3rd Nerve Palsy**:
• ***Presentation***:
a. *ptosis* (partial, complete – levator palpebrae)
b. Eye is *out and down* (exotropia + hypotropia)
c. *Mydriasis*
• ***Types***:
A. ***Surgical*** (***pupillary involvement***): painful (throbbing), pupil is affected dilated, there is compression on the parasympathetic fibers
- *Causes*: aneurysm (PCA MC), trauma, IC mass lesion, transtentorial herniation (ER!)
B. ***Medical*** (***pupillary sparing***): painless, pupils are not affected
- *Causes*: diabetic, ischemia
** so we differentiate between the two by presence of pain, pupillary involvement
** Patients with PARTIAL ptosis sometime come to you complaining of DIPLOPIA, while patients with total ptosis don't because the eye is already closed

• ***Investigations***: CT (aneurysm), MRI, LP (SAH)

• **4th Nerve Palsy**:
** only nerve that escapes from dorsal aspect of brainstem
- binocular vertical diplopia (trouble down stairs, reading)
- ***WOOG***: *worse on opposite gaze* // ***BOOT***: *better on opposite head tilt*
- ***Causes***: idiopathic, closed head trauma (without fracture), aneurysm, tumor, MS

• **6th Nerve Palsy**:
- eye is esotropia, inward, convergent squint, bi-ocular horizontal diplopia (looking to affected side)
- ***causes***: idiopathic, diabetic (diabetic infarction – v.common), elderly, compression of the 6th nerve in the cavernous sinus by tumor, trauma, Wernicke's encephalopathy, MS
- ***Investigations***: if ICP (fundoscopy), MRI, CT, LP, collagen vascular screen (vasculopathic process)

** treat the primary cause and it will be treated probably
** 6th nerve has the longest intracranial course so it doesn't tell you the site of the lesion leading to palsy which leads to something called false localizing sign.
Patient might have problem in the right side and it is caused by as mass in the left hemisphere, so there might be 6th nerve palsy on the right side regardless of the site of the lesion (in which hemisphere, might be at the opposite side, and in neurology you need to know the site of the lesion and the type of the lesion

Systemic Diseases

DM

- the 1st diabetic complication is DR
- might affect anterior, posterior segment, periocular, optic nerve & neurological changes
- the diabetic renal disease is a marker of severe retinopathy progression

• ***RF***: duration (most important – non-modifiable), pregnancy, HTN, renal disease, good metabolic control (delay but don't prevent - modifiable)

• ***Pathogenesis of DR***:
- ***Microangiopathy***:
a. ***Microvascular leakage***: pericytes injury which results in leakage.
Consequences: formation of microaneurysms, retinal edema and hemorrhage
b. ***Microvascular occlusion***: In the capillary wall this damage causes thickening in the basement membrane, damage of RBCs or/and increase in coagulation status which will result in microvascular occlusions (occlusions at the level of capillaries).
Consequences: Shunts and neovascularization on retina and iris capillary changes
- ***Neuropathy***
- ***Other processes***

• ***Pathologic Findings***:
1. ***Microaneurysms***: earliest manifestation – red tiny dots on fundoscopy
2. ***Hemorrhages***: due to leakage of blood into extracellular space at different levels: deep (dark within the retina – dark dots – plot hemorrhages), superficial (in nerve fiber layer – flame shaped hemorrhages)
3. ***Exudates***: deposits of lipoproteins (hard exudates) in the outer plexiform layer

• ***Manifestations of Ischemia***:
1. ***Cotton wool spots***: pale, white ill demarcated, very superficial
2. ***Venous Changes***: 2ry to ischemia effect on the venous wall "venous beading – loop"
3. ***IRMAs*** (intra-retinal microangiopathies): shunting of blood from arterial to venous tree
4. ***Neovascularization***: NVD (Disc) NVE (elsewhere) NVI (Iris – rebeosis iridis) NVA (angle)

• ***Stages***:

1) Nonproliferative DR (NPDR):

- include all manifestations except neovascularization, it is classified according severity:

a) Very Mild: single separated micro-aneurysm not more than 2-3

b) Mild: up to 20 micro-aneurysm or hemorrhages in less than 1 quadrant

***c) Moderate NPDR*:** microaneurysms, hemorrhages, cotton wool spots, limited venous changes and Tiny IRMAs in 2-3 quadrants.

***d) Severe NPDR*:** very important to be diagnosed.. you should NOT miss severe NPDR and usually you have to treat immediately.

** ***Criteria***: the rule of 4/2/1 (one criteria is sufficient for diagnosis)

4: Hemorrhages and microaneurysms in 4 quadrants> red dots of different sizes all over

2: venous changes (beading, looping and edema) in 2 quadrants.

1: IRMAs and shunting in 1 quadrant.

***e) Very Severe NPDR*:** more than one of the above criteria.

*Cotton wool spot is found in moderate & severe NPDR but don't alter the classification.

2) Proliferative DR (PDR):

- ***Hallmark***: Neovascularization NVD, NVI, NVE

- ***Sub-classified into***: less than high risk, high risk, and advanced Diabetic Eye Disease, if the pt has only neovascularization and no edema or ischemia in the macula then he didn't reach advanced DR yet and he has a normal vision

• ***Complications*** (***Advanced diabetic eye disease***):

- *Hemorrhage*: due to fragility of new vessels
- *Vitreous hemorrhage*: occur in NVD, NVE
- *Tractional macular detachment* due to fibrosis
- *Neovascular glaucoma*
- *Painful red eye or phthisis bulbi* (wasting away or atrophy of the eye, needs enucleation)
- *Posterior segment complication* of DM is vein occlusion

Retinal		Non-retinal
PDR	**NPDR**	**Related Ocular Conditions**
Vitreous hemorrhage Retinal detachment Fibrovascular proliferation in front of the macula Macular edema or ischemia (specially the center of macula)	Macular edema or ischemia (specially the center of macula	Glaucoma Cataract Corneal problems Extraocular muscle palsy due to diabetic neuropathy

Retinopathy stage	Findings on ophthalmoscopy	Management and review/referral timeframe
No apparent retinopathy	No abnormalities	In line with AOA, AAO, and JDC, OA recommends annual review
Minimal NPDR	Microaneurysms (MA) only	Review 6-12 months taking into consideration proximity of MA to fovea
Mild to moderate NPDR	More than just MA but less than severe NPDR. This may include: • dot haemorrhages • blot haemorrhages • cotton wool spots • intraretinal microvascular anomalies (eg venous beading)	Refer or closely monitor Depending on level of DR present, 3-6 monthly or annually
Severe NPDR	Any of the following: • more than 20 intraretinal haemorrhages in each of 4 quadrants • definite venous beading in 2+ quadrants • prominent IRMA in 1+ quadrant AND no signs of proliferative retinopathy	Ophthalmology referral
PDR	One of the following (or unexplained fall in VA) • neovascularisation • vitreous/pre-retinal haemorrhage	Urgent ophthalmology referral (days – week)

• ***Maculopathy***:
- ***Definition***: presence of hard exudates or edema or ischemia in the macular region ```
** The macula is between the superior temporal arcade and inferior temporal arcade temporal to the disk (*approximately it is 4 x disc size = 4 x 1.5 = 6*)
** might be present in proliferative and non-proliferative
** if diagnosis is in doubt we confirm by ***FFA*** (Fluorescence angiography) or ***OCT*** (Optical coherence tomography - gives us quantitative & qualitative assessment of ME degree)

• ***Classification of Diabetic Macular Edema*** (***DME***):
- *ETDRS* (early treatment DR study) group classification
- Clinically significant (*CSME*) X *Non-clinically* significant
- *Centrally* involved X *Non-centrally*
- *Focal* X *Diffused*
- *Ischemic* X *Non-ischemic*

• ***Treatment***:
- *Conservative measures* (lifestyle)
- *Prophylaxis*: prophylactic laser for pregnant women with severe NPDR
A. *Treatment of NPDR* – *without ME*:
- conservative and frequent follow ups (based on the severity)

B. *Treatment of PDR*:
- Argon laser photocoagulation (*PRP*) or *scatter laser*
- if advanced macular detachment, vitreous hemorrhage we need *Pars plana vitrectomy*

C. *Macular Edema treatment*:
- *Anti-VEGF*: injected into the intravitreal space
- *Drugs*: Avastin, lucentis (equal effect, lucentis is approved by FDA, patients need 3-4 injections (intravitral space injections)

• ***Safety concerns in Rx***:
- *Laser*: retinal damage
- *Anti-VEGF*: ocular complications, and systemic risk of thromboembolic phenomena
- *Steroids*: ocular problems, cataract, glaucoma
- *Vitrectomy*: bleeding, cataract, glaucoma, loss of vision

• ***Screening*** is done by ophthalmoscopy (50% sensitive for early retinal changes) and fundoscopy (100% sensitive, specific, but less used for ME) based on the case and HBA1c

Thyroid Eye Disease

• ***Graves Disease*** (***Toxic diffuse goiter***):
- autoimmune disorder (IgG bind to TSH receptors and stimulate it)
- MCC of thyrotocoicosis

• ***Presentation***:
- 4^{th} to 5^{th} decade, females (x5! Than males)
- Thyroid acropachy (clubbing), pretibial myxedema, enlarged thyroid gland
- hyperthyroidism Sx

• ***Treatment***: medical (carbimazole, propylthiouracil), thyroid ablation with radioactive iodine, Surgical (partial thyroidectomy)

• ***Thyroid Opthalmopathy***:

• ***Risk factors***: smoking, females, radioactive iodine (might worsen thyroid eye disease although it treats hyperthyroidism)

• ***Stages***:
1) ***Congestive*** (***inflammatory***): eyes are red, painful, remits within 1-3y, 10% (serious)
2) ***Fibrotic*** (***Quiescent***): eyes are white, painless motility defect is present

• ***Clinical Presentation***:
a) ***Soft tissue involvement***:
- periorbital and lid swelling and chemosis, conjunctival hyperemia (Conjunctival injection: Blood vessels are apparent with the naked eye), superior limbic keratoconjuctivitis, keratoconjuctivitis sicca, corneal signs exacerbated by lid retraction
b) ***Lid retraction***:
- both lids in 50%, scleral area visible
- ***Dalrymple sign***: abnormal wideness of the palpebral fissure (in thyrotoxoicosis)
- ***Kocher sign***: Staring and frighten appearance of the eye (especially on attentive fixation)
- ***Von Graefe sign***: retarded descent of the upper lid on down gaze (lid lag)
c) ***Proptosis***:
- Exophthalmos, protrusion the eyeball anteriorly out
- Test: ***Hertel exopthalmometer*** (binocular)
d) ***Optic neuropathy***:
- by direct compression of the optic nerve or its nerve supply
- severe but preventable visual impairment
e) ***Restrictive myopathy***:
- ***Hering's Law***: There is reciprocal signals for two antagonistic muscles. There will be positive and negative impulse for opposite muscles. If this doesn't happen, the muscle will move halfway and then the action will be opposed by the contract of the other muscle. A negative impulse on that other muscle will result in full movement. In restrictive myopathy, the nerve and muscles' impulses are intact, but the muscle's skeletal fibers become fibrotic fibers à restrictive movement. This fibrosis is irreversible.

• ***Severity of TED***: ***European Group on Graves Orbitopathy*** (***EUGOGO***):
a) ***Mild***: only minor impact on life
b) ***Moderate-severe*** (one of the following):
- moderate to severe soft tissue involvement
- lid retraction of 2 mm or more
- diplopia
- proptosis of 3 mm or more
c) ***Sight-threatening***: optic neuropathy (ttt. Pulsed IV methylprednisolone, orbital wall decompression and or orbital apex decompression, corneal breakdown

• ***Management***:
- *Conservative*: lifestyle: lubricants, head elevation, eyelid taping
- *Medical*: *Topical*: anti-inflammatory, *Systemic*: steroids (oral: prednisolone, IV: methylprednisolone – pulsed IVE methylprednisolone in acute compressive optic neuropathy, *Orbital* steroids: less effective and less SE), immune modulators
- *Surgery*: orbital decompression, extraocular muscle surgery, eyelid surgery
- Stabilize the thyroid state of the patient

- Lid retraction and proptosis may result in dryness of the cornea, we will have to do eyelid surgery to narrow the fissure and decrease the dryness.
- Steroids, radiotherapy or surgical decompression for the optic nerve
- Surgical decompression – orbit has four walls. I can break one or all four of the walls to separate the orbit and allow the pressure to be released
- Diplopia
- Surgery is one of the options; use other muscles to heal the fibrotic fibers of the restrictive myopathy-affected muscles.

• ***Prognostic factors***:
- Level of thyroid hormones, pretibial myxedema, etc.
- One of the most important factors is smoking. It will change the course of the treatment.

Pediatric Ophthalmology

• visual acuity in newborns are reduced because the fovea is covered by a thick layer of ganglion cells (still immature), sharp vision around 5th year of age

• **Tests done for acuity depending on age**:
- *newborn to 1 month*: blink to bright light
- *6 week to 3 month*: fix and follow (start at 6 w. fully mature at 3 m)
- **1 year – 3 year**: Cardiff acuity card (**CAC**) / **Kay pictures** (matching)
- *3-5 years*: Shreidan Gardiner (letters with no confusion)
- *>5 years*: Snellen Chart: E (*Tambling*), C (*Landort*), Pictures

• **0 – 4 weeks**: blinking to bright light (bilateral blinking if exposed to one eye), response to hand motion threat, binocular fixation pattern (gross objection and crying to contralateral occlusion of the eye)

• **Newborn – 2 months**:
- *fix and follow*,
- *spinning* (normally nystagmus develop and stops once you stop),
- *forced choice prefential looking* (FPL/PL – heterogeneous – children prefer to look at complex rather than plain structures),
- *Electrophysiologic* testing with visual evoked potentials (VEFs – it measure the function of the optic nerve, which determines the acuity), and visual evoked responses/ electroretinogram (ERG – it measures photoreceptors of the retina only cones/rodes)

• ***poor visual behavior*** (***blindness***): is categorized to blind with/out nystagmus

• **Blindness with Nystagmus**:
- *Characteristic*: *bilateral*, *significant*, *anterior visual pathway problem* (cornea to chiasm), *appeared and not corrected during first 3 months of life*
- nystagmus only appears after 3 months of life, it indicate severity, irreversibility
- *Nystagmus blindness has two types*: one affecting *anatomical* integrity (abnormal eye exam), and one the *function* (normal eye exam but blind, here we do the electrophysiological testing)

- **Nystagmus**:
- it is involuntary, oscillatory eye movement, it has two types: ***Jerk*** (slow drift in one eye, one direction with the other rapid recovery movement in other direction), ***Pendular*** (slow eye movement in equal velocity)
- ***Congenital*** nystagmus: patient feel the world is stable, ***Acquired***: the world is moving

• **Diseases for anatomical integrity of the eye**:
- *Corneal* (opacity, dystrophy, cloudiness)
- *Congenital* (glaucoma, cataract)
- *Anridia*, *vitreous hemorrhage*, *foveal hypoplasia*, *albinism*, *retinal detachment*
- *Optic nerve* (hypoplasia, coloboma, atrophy)

• **Congenital Glaucoma**:
- ***Triad***: *photophobia* (with blepharospasm), *hazy cloudy cornea*, *hyperlacrimation*
- bad prognosis
- ***Treatment***: always surgical (geniotomy, trabeculotomy (in adults trabeculectomy)
- ***DDx***. Foreign bodies, congenital nasolacrimal duct obstruction (both cause lacrimation)

• **Congenital Cataract**:
- bilateral cataract causes nystagmus (unilateral don't), bilateral can cause severe irreversible blindness, while unilateral leads to blind eye and not blind infant
- ***causes***: 1/3 heriditary (AD), 1/3 syndrome/metabolic/torch, 1/3 idiopathic
- ***Treatment***: Lensectomy + posterior capsulotomy + anterior vitrectomy + IOL (if older than 2 years and if its his primary surgery)

• **Aniridia**:
- absence of iris tissue
- associated with foveal hypoplasia (causes blindness and nystagmus)
- AD, sporadic (deletetion of shot arm of chromosome 11, may be a part of WAGR)
- **WAGR**: **W**ilms tumor (nephroblastoma – needs US), **A**niridia, **G**U defects, **R**etardation

• **Vitreous Hemorrhage** (**Non-accidental trauma**):
- shaken baby syndrome (child abuse)
- ***RF***: 1st child, single dad, daycare, chronic medical illness/syndromes
- mechanism: acceleration-deceleration

• **Foveal Hypoplasia**:
- *Causes*: Aniridia, Albinism
- *Albinism types*: *Oculo-cutaneous* (AR, due to tyrosine enzyme deficiency), *Ocular* (X-R)

- Albinism are photophobic because of missing filter function of pigmented layer

• **Retinopathy of prematurity** (**ROP**):
- *the 2 main RFs* (*needs screening*): gestational age <32w (>32w risk is ZERO), BW <1.5kg

- ROP occur in 10% of those at risk and 10% of those with ROP will require treatment
- *other RFs*: NICU hyper-oxygenation, intraventricular hemorrhage, neonatal sepsis, necrotizing enterocolitis

- ***Stages***:
I. Demarcation line
II. Ridge
III. Extra-retinal neovascularization
IV. Partial retinal detachment
V. Total retinal detachment

- ***Screening***: 1st screening is at 4 weeks, or at corrected GA of 32 weeks, when to stop: full maturation of retinal vasculature (peripheral vasculature), baby reached corrected GA 44 weeks without ROP |
- ***Treatment***:
early: argon laser to ischemic retina
complicated (4,5 bad prognosis): complex vitreoretinal surgeries

• ***Optic nerve diseases***:
- Optic nerve *coloboma*: incomplete growth of optic nerve, if bilateral (blind, nystagmus)
- Optic nerve *hypoplasia* (ONH): if bilateral (blind, nystagmus)
- *De Morsier Syndrome* (Septo-opic dysplasia): bilateral ONH + atrophy of anterior pituitary gland + absent corpus callosum and septum pellucidum

• ***Diseases affecting the function***:
• *Lebers congenital amaurosis* (*LCA*):
- AR, very poor vision at birth, total damage to both rods, cones
- ***ERG***: extinguished ERG (flat line, no cones/rods), ERG is diagnostic

• *Other diseases*: ERG is diagnostic in all
- *Con's dystrophy*: cons
- *Achromatopsia*: cons
- *Congenital stationary night blindness* (*CSNB*): rods
- *Retinitis pigmentosa*: rods

• ***Blindness without nystagmus***:
- the problem is posterior to the chiasm (in the brain)
- *divided into* 2 categories based on CNS abnormalities present or not:
- very high refractive errors may mimic blindness, so we always exclude high refractive errors by: dilated fundus exam, cycloplegic refraction

A. ***CVI*** (*Central Vision Impairment*):
- Hx. Of asphyxia, obstructed labor, seizures
- helpful signs. Preference of bright colored objects, staring at bright light, turning head when they look at an object of interest
- CT, MRI, neurologist are needed for Dx
B. ***DVM*** (*delayed visual maturation*):
- diagnosis of exclusion and retrospection (unlike CVI, normal MRI, no Hx)
- visual impaired with normal everything, idiopathic
- prognosis is excellent, visual function improves to normal or near normal by the end of 1st year of life

• ***WHO definitions***:
- *Blindness*: acuity <3/60 in best eye with best correction or visual field <10 degree
- *Low vision*: acuity <6/12 with best eye and correction
- *Amblyopia*: difference in best visual acuity of 2 snellen lines or more between the two eyes in the absence of organic lesion

• ***Amblyopia***:
- *Classification*:
A. *Sensory deprivation amblyopia*:
- a disease causes obstruction of entry of light and pictures into the eye in the early vision development (e.g. congenital cataract, corneal opacity)
- can be treated until age of 5 years

B. *Strabismus amblyopia*:
- the brain suppress the deviated eye which with time becomes amblyopic (in adults diplopia)
- can be treated until age of 8 years

C. *Refractive errors* (*anisometropic*) *amblyopia*:
- different refractive errors between 2 eyes and suppression of the worse image and eye
- can be treated until age of 11 years

- *Treatment*:
- *patch* good eye for 2-6 hrs
- *Atropine drops* for good eye
- When to stop treatment: equal vision in both eyes, no improvement after 6 m (persist)

• ***Strabismus***:
- malalignment of the yes
- *types*: *comitant* (equal angle of deviation in different positions) & *Incomitant* (unequal)
- incomitant has 2 types: *restrictive* (thyroid opthalmopathy, and myasthenia gravis), *paralytic* (6^{th}, 3^{rd})
- *Hirshberg light test*: temporal, nasal, superior, inferior/ used for tropia not phoria
- *Pseudostabismus*: false squint in absence of deviation, due other abnormalities (e.g. wide nasal bridge)
- *Alternate cover test*: it measures total angle of deviation (phoria, tropia) combined with prisms (to know exactly the angle of deviation), Corrective movement
- *clinical exam for strabismus*: acuity, Hirschberg, alternate cover test, cycloplegic refraction test, dilated fundus exam
- *Treatment*: surgically treat the residual angle of strabismus: *resection* (strengthen), or *recession* (weaken) the muscle

BEST WISHES

DONE BY:

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