IMPORTANT POINTS TO DIAGNOSE SCENARIOS OF OPHTHALMOLOGY

BY MARYAM MALIK –RMC
PUBLICATION DEPT- RIFAO

I have tried my best to right them accurately, kindly do point out if you find any mistake.

THE ORBIT

1. PRESEPTAL CELLULITIS
Edematous tender eyelids + purple red sharply demarcated swelling

2. ORBITAL CELLULITIS
Rapid onset of orbital swelling & pain associated with malaise & fever
Eye ball proptosed axially + restricted painful extraocular movements + decreased vision & pupillary abnormality + congestion of retinal vessels & disc edema.

SQUINT
1. AMBLYOPIA
Unilateral or bilateral Decrease in visual acuity for which no identifiable organic cause is there in eye or visual pathway

Sensitive period= amblyopia occurs below 8-9 yrs. Most sensitive period= first 6 months.

VISUAL ACUITY reduced to two or more than two lines of snellen's chart + no improvement with pin-hole phenomena + crowding phenomena

2. Latent (heterophoria)squint
Headache + eyeache + difficulty in changing focus from one distance to another + photophobia that is relieved on closing one eye + blurring/crowding of words while reading + intermittent diplopia + intermittent squint

3. Manifest (heterotropia) squint

NON-PARALYTIC (COMITANT) = amount of deviation in squinting eye remains same in all directions of gaze + no limitation of ocular movements

PARALYTIC (NON-COMITANT) = amount of deviation in squinting eye varies in different directions of gaze + limitation of ocular movements
4. **Non paralytic (comitant) squint**
   usually **Gradual/congenital** + usually childhood + infrequent history of head trauma + no difference in primary & secondary deviation + infrequent diplopia + no limitation of movement + rarely abnormal head posture + usually no neurological lesion + usually no systemic diseases

5. **Paralytic (non comitant) squint**
   Often **sudden** + **any age** + frequent history of head trauma + difference in primary & secondary deviation + frequent diplopia + limitation of movement + abnormal head posture + neurological lesion + systemic diseases

6. **PARTIAL 3\textsuperscript{rd} NERVE PALSY**
   Diplopia + variable limitation of adduction, upward & downward movement + **partial ptosis** + pupillary dysfunction

7. **COMPLETE 3\textsuperscript{rd} NERVE PALSY**
   Complete ptosis + eye positioned outward & downward + limited adduction, supraduction, infraduction + **sec deviation**
greater than primary deviation + pupil dilated with poor response to light.

8. **UNILATERAL Fourth nerve palsy**
   - Diplopia (vertical diplopia that occurs in down gaze) at near vision/reading + head tilt + increase in right hypertropia in left gaze + limitation of right depression on adduction + normal right abduction, depression & elevation + parks 3 step test & Double maddox rod test

9. **BILATERAL Fourth nerve palsy**
   Crossed hypertropia + excyclotropia of 10 degree or greater + v pattern esotropia + bilateral positive parks 3 step test

10. **SIXTH NERVE PALSY**
   - Horizontal diplopia that worsens on ipsilateral gaze esp viewing at a distance.
   If RIGHT eye 6th nerve palsy
   - Face turn on the affected side
   - Right esotropia
Marked limitation of right abduction
Diplopia that increases on right abduction

UVEAL TRACT
1. ANTERIOR UVEITIS

Inflammation of the iris and the ciliary body is known as anterior uveitis.

Anterior uveitis occurs in two forms namely,
1. Infective (granulomatous)—It is due to direct organismal infection. Inflammation is insidious in onset, chronic in nature with minimum clinical features. There is dense nodular infiltration of tissues by lymphocytes and plasma cells. It is characterized by presence of large greasy “mutton fat” keratic precipitates which are deposits of white blood cells (mainly lymphocytes), cluster of inflammatory cells on the pupillary border (Koepple’s nodules) or on the peripheral part of the anterior surface of iris (Busacca’s nodules).
2. Allergic (exudative or non-granulomatous)—It is of acute onset and short duration. It is diffuse in an extension, i.e. without focal lesion in the iris. It is characterized by the presence of fine keratic precipitates which are composed of lymphoid cells and polymorphs.

ACUTE IRIDOCYCLITIS
It is an acute inflammation of the iris (iritis) and the ciliary body (cyclitis).

Redness + Pain—It is worse at night. There is severe neuralgic pain referred to forehead, scalp, cheek, malar bone, nose and teeth (as the iris is richly supplied by sensory nerves from the ophthalmic division of 5th nerve). Lacrimation and photophobia may be present (without any mucopurulent discharge) + impaired vision + photophobia + decreased visual acuity + circum corneal congestion + keratic precipitates (pathognomonic of iridocyclitis) + aqueous flare + aqueous cells + fibrinous exudates (typically HLA-B27 associated uveitis) + hypopyon + muddy iris + miotic pupil that is sluggish in reaction + posterior synechae + inflammatory cells in anterior vitreous + poor red reflex + normal fundus + tenderness of eyeball may be present

2. CHRONIC IRIDO CYCLITIS
Persistent inflammation that relapses in less than 3 months + asymptomatic unless complications develop + eye is usually white + flare & cells + KP’s are large greasy (mutton occlusion) appearance + posterior synechae, occlusion-papillae, ring synechae + iris nodules (in granulomatous disease- koepepe, busacca)
3. INTERMEDIATE UVEITIS
PARS PLANITIS = snow bank or snow ball formation in absence of associated infection or systemic disease
Insidious onset + reduced VA + floaters + small KP, flare, cells in ant chamber + cells mainly in anterior part of vitreous + SNOW BALLS (aggregation of inflammatory cells) in anterior peripheral vitreous + SNOW BANKING (accumulation of inflammatory exudates) in all quadrants of vitreous esp inferior + retina shows peripheral phlebitis + retinal venous sheathing

4. POSTERIOR UVEITIS
PAINLESS + reduced vision + floaters + metamorphosis + micropsia + macroscopia + photopsia + positive scotoma

5. TOXOPLASMOSIS
• CONGENITAL = convulsion + intracranial calcifications + hydrocephalus + retinochoroiditis + active stage (thickened cream colored with overlying vitritis) + inactive stage (bilateral healed punched out heavily pigmented chorioretinal scar in macular area)
• AQUIRED = floaters + unilateral blurred vision + O/E white eye + mild- moderate GRANULOMATOUS anterior uveitis + vitritis + focal white retinitis lesion on retina
• **RECURRENT (RETINOCHOROIDITIS)** = after 10-40 yrs of active lesion (congenital toxoplasmosis) + floaters + unilateral blurred vision + O/E white eye + mild **NONGRANULOMATOUS** anterior uveitis + vitritis (head light in the fog appearance) + retinitis appearing as whitish yellow slightly raised area of infiltration with overlying vitreous inflammation adjacent to pigmented chorioretinal scar.

6. **ENDOPHTHALMITIS**
Ocular pain + redness + photophobia + Marked visual loss with defective projection of rays + lacrimation + congestion of conjunctiva + chemosis + hazy cornea + fibrinous exudates & hypopyon in anterior chamber + **LOSS OF RED REFLEX** + inability to visualize the fundus even with indirect ophthalmoscope. + vitreous exudation (yellowish white mass seen through pupil) + IOP initially raised then fall.
It occurs most commonly as an acute process 1-7 days following intraocular surgery such as cataract extraction and filtering operation.

7. **PANOPHTHALMITIS**
**Severe** ocular pain + headache + marked loss of vision + edema of lids + hyperemia of conjunctiva + cloudy cornea + puss in anterior chamber +
raised IOP + RESTRICTED PAINFUL EXTRAOCULAR MOVEMENTS (differentiates this from endophthalmitis)

8. SYMPATHETIC OPHTHALMITIS
Bilateral granulomatous disease
Occurs after penetrating ocular injury + traumatized eye (EXCITING EYE = red + irritable) & fellow eye which develops uveitis (SYMPATHIZING EYE = photophobic or irritable) + both eyes develop anterior uveitis, multifocal choroidal infiltrates develop in mid periphery + exudative RD in severe case + Dalen-Fuch’s nodules (granuloma) between bruch’s membrane & RPE

9. FUCH’S UVEITIS
Chronic non granulomatous uveitis
Usually unilateral + young adults + asymptomatic/vitreous floaters + gradual blurring of vision due to cataract formation + white eye + KP’s (small round or stellate, grey white scattered throughout the corneal endothelium) + mild flare & cells + diffuse iris atrophy (dull appearance of eye) + iris heterochromia (color difference between two eyes, affected eye becomes hypochromic) + posterior
synechae NEVER form + stringy opacities & cells in vitreous + goinoscopy angle neovascularization

10. **ACUTE POSTERIOR MULTIPLE PLACOID PIGMENT EPITHELIOPATHY** (APMPPE)
Idiopathic + bilateral + self limiting + follows flu-like illness in one third of patients + sudden onset of bilateral assymetrical visual loss + central & paracentral scotoma + multiple large flat yellow-white deep placoid lesions at the level of RPE which resolve over a period of 2-6 weeks leaving permanent alteration in RPE

**OPTIC NERVE**

1. **Optic neuritis**
   - Acute/subacute visual loss = unilateral or bilateral (viral)
   - Pain on extraocular movements
   - Reduced VA + impaired color vision (red, green)
   - RAPD + tenderness of globe + deep orbital pain/ brow pain
   - FUNDOSCOPY = swollen disc with inflammatory cells in viterious (papillitis)
• swollen disc with inflammatory cells in viterious + **macular star (neuroretinitis)**
• PERIMETRY= central/ centrocaecal **scotoma**
• **VISUAL EVOKE POTENTIAL= abnormal**

2. Papillitis
• Blurred disc margin
• Hyperaemic disc
• Engorged vessels

3. Papilloedema
Headache + nausea/vomiting + deterioration of consciousness + unilat/bilateral greying out/blacking out of vision many times a day for 10-15 sec + diplopia.
Normal VA + normal pupillary reactions + hyperemic disc with elevated blurry margins + obscurced blood vessels as they cross disc margin, tortuous veins + absent venous pulsation + hard exudates + cotton wool spots + hemorrhage + macular star + retained central cup + paton’s lines
• Champagne cork appearance in longstanding papilloedema, Dec VA & Blind spot enlarged

4. Optic atrophy
• VA reduced + altered color vision + reduced contrast sensitivity + Afferent pupillary defect + visual field defect
• PRIMARY OPTIC ATROPHY = Chalky white optic disc with clear margins + No sheathing of vessels
• SECONDARY OPTIC ATROPHY = Grey / dirty white optic disc with blurred margins + sheathing of vessels

**CORNEA**

1. **BACTERIAL CORNEAL ULCER (PYOGENIC KERATITIS)**

   Pain + Photophobia + Impairment of visual acuity occurs due to corneal opacity + . Lacrimation + blepharospasm + . Corneal opacification occurs due to infiltration and oedema + . Ciliary congestion with conjunctival hyperaemia + Hypopyon or pus in the anterior chamber may be present. fluorescein stain:—It stains the margin of the ulcer bright brilliant green.(superficial staining) It stains the stromal infiltration defect grass green and the endothelium yellow in colour respectively.(deep staining) + Slit-
lamp examination shows irregular margins of the ulcer and details of anterior segment of the eye.

2. **ANTERIOR SYNAECHAE**

Adhesion between iris and cornea

3. **LEUCOMA**

Dense white opacity of cornea

4. **ADHERENT LEUCOMA**

Adhesion between iris and leukoma

5. **ANTERIOR STAPHYLOMA**

Adhesion between iris and leukoma, There is ectasia due to secondary glaucoma usually.

5. **ULCUS SERPENS**

most common type of hypopyon ulcer. It occurs in adults due to *pneumococcus* bacteria usually + It has a tendency to creep over the cornea in a serpiginous fashion. marked pain in the eye and lacrimation + photophobia + Cornea is lustreless and hazy. A greyish white or yellow disc is seen in the centre. The opacity is greater at the advancing edge in one particular direction than centre.
The tissues breakdown on the side of the densest infiltration (yellow crescent) and ulcer spreads in size and depth. Often there is infiltration anterior to Descemet’s membrane at the floor of the ulcer while the intervening stroma is normal. Marked iritis with cloudy aqueous (hypopyon), conjunctival and ciliary congestion is usually present. The lids are red and swollen.

**MYCOTIC HYPOPYON ULCER (KERATOMYCOSIS, FUNGAL CORNEAL ULCER)**

It is commonly caused by *Candida albicans*, *Aspergillus fumigatus*, *Fusarium*, *Cephalosporium*, *Streptothrix actinomycosis*, etc. *Filamentous fungi*—e.g. *Aspergillus* species (most common) *Fusarium*. They are most prevalent in agricultural areas.

*Yeast*—e.g. *Candida albicans*. It frequently affects the immunocompromised host.

Mild pain, irritation, watering + yellow-white coloured ulcer with indistinct margin + dry in appearance with small satellite lesions around the ulcer due to the stromal infiltration with delicate feathery, finger-like hyphate edges protruding into adjacent stroma. Ulcer margin is often elevated above the surface + Massive
hypopyon is present commonly which is dense and organized.

Slit-lamp examination—Endothelial plaque and immune ring may be seen around the ulcer. Some degree of iridocyclitis is usually present

1. Staining with methamine silver, Gram and Giemsa stains.
2. Culture in Sabouraud’s medium, blood-agar plate or brain-heart infusion broth is essential.

MARGINAL ULCER caused by Morax-Axenfeld bacillus, Staphylococcus, H. aegyptius, etc. + often associated with chronic blepharoconjunctivitis + seen in old debilitated people usually + Deep marginal ulcer may occur rarely in cases of polyarteritis nodosa, systemic lupus erythematosus due to antigen-antibody complexes

There is neuralgic pain in the face and head. + Shallow, slightly infiltrated, multiple ulcers are seen near the limbus. The ulcers are often vascularised

CHRONIC SERPIGINOUS ULCER (RODENT OR MOOREN’S ULCER)

It is a rare superficial progressive marginal ulcer of a degenerative type.

occurs as a result of degenerative process due to ischaemia of cornea + common in elderly males +
severe persistent neuralgic pain with lacrimation + It starts as one or more grey infiltrations which breakdown to form small ulcers. Small ulcers spread centripetally and coalesce with each other. Characteristic white overhanging edges are seen as the ulcer spreads below the epithelium and superficial layers of stroma. This resembles the ears of the rabbit (rodent ulcer). There is vascularization at the base of the ulcer.

**EXPOSURE KERATITIS**

There is exposure of the cornea due to insufficient closure of the eye (Lagophthalmos). This condition is commonly seen,
- In eyes insufficiently covered by the lids due to paralysis of the orbicularis muscle.
- In extreme proptosis, e.g. exophthalmos, orbital tumour.
- There is absence of reflex blinking in extremely ill or comatose patients.

**Sign**
- There is corneal erosion with ulcer formation as the epithelium becomes desiccated.
- There is absence of reflex blinking and defective closure of lids during sleep.
NEUROPARALYTIC KERATITIS
There is loss of corneal sensation which results in the formation of corneal ulcer.
There is 5th nerve (trigeminal nerve) paralysis. It occurs typically as a result of injecting alcohol in gasserian ganglion in cases of trigeminal neuralgia + It is a painless condition due to corneal anaesthesia + Large corneal ulcers are seen due to peeling of the epithelium. The stroma is cloudy and yellow often associated with hypopyon + Ciliary congestion is marked.

HERPES SIMPLEX KERATITIS
caused by herpes simplex virus type I (HSV I) + It occurs in children or young adults usually.
• There is recurrence due to febrile cold, pneumonia, physical exhaustion or exposure to sunlight.
Types
1. Primary ocular herpes—There is acute follicular keratoconjunctivitis with regional lymphadenitis and skin involvement.
2. Recurrent herpes—It has following characteristic features:
   • Epithelial ulcers
   • Stromal interstitial keratitis
• Disciform keratitis
• Iridocyclitis.

Vesicles are seen on lips, nose, cornea (Herpes simplex virus type I / HSV I) and genitals (Herpes simplex virus type II / HSV II) + Great irritation, lacrimation and blepharospasm

1. Skin lesion—Initially vesicles with superficial crusts are formed. These vesicles heal without scar formation.

2. Severe follicular keratoconjunctivitis is present usually in children.

3. There may be regional lymphadenitis (preauricular lymph nodes).

4. Slit-lamp examination of the cornea shows:
   i. *Superficial punctate keratitis*
      • Numerous, white plaques of epithelial cells are present all over the corneal surface.
      • These are of minute pin—head size.
      • They are arranged in rows or groups.
      • There is absence of vascularization and corneal sensation
   
   ii. *Dendritic ulcer*—Erosions coalesce to form typical dendritic figure like liverwort. It is pathognomonic of herpes simplex.
iii. **Confluent ulcer**—Large geographical pattern type of ulcers are seen.

iv. **Disciform (deep) keratitis**—It involves the stroma forming disc-like opacity

**HERPES ZOSTER**

It is an acute infection of dorsal root ganglion by varicella—zoster virus. It is identical with chickenpox virus.

It is associated with the chickenpox infection in youth or childhood. It often occurs in elderly with depressed cellular immunity. It is unilateral always affecting the gasserian ganglion from where the virus travels down the branches of ophthalmic nerve.

It can be divided into three stages namely:

1. **Acute phase**—It occurs within the first 4 weeks which may totally resolve.
2. **Chronic phase**—It may persist for several years (10 yrs).
3. **Relapsing phase**—There is recurrence of the disease process even years later.

**Symptoms**

1. **Rows of vesicular eruption take place along the branches of the ophthalmic division of the 5th cranial nerve.** These suppurate, bleed and cause pitted scar.
   - Supraorbital nerve
• Supratrochlear nerve
• Infratrochlear nerve
• Nasociliary nerve
• Infraorbital nerve

2. **Severe neuralgic pain** along the course of the nerves is present due to neuritis.
3. Fever and malaise are present at the onset.
4. Skin of lid and face becomes red and oedematous.

**Signs**
1. **Hutchinson’s rule**—Ocular involvement is usually associated with eruption of vesicles on the skin of tip of the nose (nasociliary branch) during the acute stage.
2. **Corneal and skin anaesthesias** are characteristic and persist for a long-time.
3. Slit-lamp examination—
   • **Superficial punctate keratitis** is a most common feature. Numerous round white dots are seen in the epithelium which involve the stroma later.
   • **Micro dendritic epithelial ulcers**—Unlike herpes simplex, these ulcers are small, peripheral, stellate and with tapered ends, i.e. without rounded bulbs.
   • **Nummular keratitis**—Larger discoid lesions surrounded by stromal haze are seen.
• Disciform keratitis may be seen in few cases.
• Deep stromal involvement is often associated with iridocyclitis

ACANTHAMOEBA KERATITIS
Predisposing Factors
1. Keratitis may occur following a minor corneal abrasion.
2. Contact lens wearers who use distilled water and salt tablets instead of commercially prepared saline solutions for their lens care are at particular risk. Very severe pain (out of proportion to the degree of inflammation), watering, photophobia, blepharospasm and blurred vision. 
Acanthamoeba keratitis evolves over several months as a gradual worsening keratitis with periods of temporary remissions.
1. Initial lesions of Acanthamoeba keratitis are in the form of coarse and opaque streaks. Fine epithelial and subepithelial opacities are also seen.
2. Advanced cases show a central or paracentral ring-shaped lesion with stromal infiltrates. There is an overlying epithelial defect.
3. Severe cases show associated radial keratoneuritis, in the form of perineural infiltrates along corneal nerves.
Diagnosis
1. Clinical diagnosis—It is difficult and is usually made by exclusion and with strong clinical suspicion in non-responsive patients being treated for herpetic, bacterial or fungal keratitis.
2. Laboratory diagnosis—Corneal scrapings
   i. Potassium hydroxide mount - cysts.
   ii. Calcifluor white stain is stains the cysts of Acanthamoeba bright apple green.
   iii. Lactophenol cotton blue stained - cysts in corneal scrapings.
   iv. Culture on non-nutrient agar (E. coli enriched) may show trophozoites within 48 hours which gradually become cysts. E. coli prevents other organisms to grow whereas Acanthamoeba thrives on it.
3. Confocal microscopy—Acanthamoebae cysts can be demonstrated in optically cut parallel sections of cornea under confocal microscopy.

PHLYCTENULAR KERATITIS
Conjunctival phlycten may involve the corneal margin in later stages. It is an allergic reaction to an endogenous allergen, e.g. tuberculo-protein.
much pain and photophobia + grey nodule raised above the surface followed by formation of superficial yellow corneal ulcer + **Fascicular ulcer**—A leash of blood vessel may follow the corneal ulcer at times.

**Keratoglobus**
In this condition there is thinning and excessive protrusion of cornea which seems enlarged but its diameter is usually normal.

**Arcus Senilis**
There is bilateral annular lipoid infiltration of cornea in old persons + no symptoms. It does not require any treatment + it does not affect the vision or vitality of the cornea + concentric grey lines in the upper and lower part of the cornea.

- The lines join to form a ring 1 mm broad which is separated from the margin by a rim of clear cornea about 1.5 mm. It is also known as *lucid interval* of Vogt.

**White Limbal Girdle of Vogt**
chalky line in the nasal and temporal periphery of inter-palpebral area of cornea. The opacity is at the level of Bowman’s membrane

**Hudson-Stahli-line**—It is a horizontal line at the lower half of the cornea due to deposition of hemosiderin pigment

**Fleischer’s ring** is seen at the base of keratoconus

**Stocker-Busaca line**—It is seen in front of a Pterygium

**Band-shaped Keratopathy**
It is common in old, blind, shrunken eyes and in Still’s disease of children.
• It is associated with hyperthyroidism, vitamin D poisoning or sarcoidosis.
• It could be either primary or secondary to hypercalcaemia, chronic uveitis, chronic glaucoma, interstitial keratitis etc.
• A continuous band lies in the interpalpebral area starting in the inner and outer side

**Reis-Buckler’s Dystrophy**
There are subepithelial grey opacities arranged in a fish net pattern.

**Cogan’s Microcystic Dystrophy**
commonest epithelial dystrophy with dot, map or fingerprint opacities

**Messman’s Juvenile Epithelial Dystrophy (Recurrent Corneal Erosion Syndrome)**
appearance of small vesicles between epithelium and Bowman’s membrane

**Granular Dystrophy**
milky granular hyaline deposits in anterior stroma

**Macular Dystrophy**
deposition of mycopolysaccharides

**Lattice Dystrophy**
There are amyloid deposits in corneal stroma.
- Spider like opacities are seen in cornea.

**Fuch’s Endothelial Dystrophy**
Grey punctate opacities are seen in the stroma.
- The clinical features are divided into four stages
  a. Stage of cornea guttata
  b. Oedematous stage
  c. Stage of bullous keratopathy
  d. Stage of scarring

**Cornea Guttata**
bilateral symmetric lesions, which appear as golden hue on the posterior surface of cornea.

**KERATOCONUS [CONICAL CORNEA]**
bilateral condition occurring at puberty in girls usually.

**Classification**
1. **Keratoconus** can be classified by doing keratometry as follows:
   i. Mild < 48 D
   ii. Moderate 48-54 D
   iii. Severe > 54 D

**Morphological classification** is dependent on the shape.
   i. **Nipple cones**—These are characterised by their small size (5 mm) and steep curvature. The apical centre is often either central or paracentral and displaced inferonasally.
   ii. **Oval cones**—These are longer (5-6 mm), ellipsoid and commonly displaced inferotemporally.
   iii. **Globus cones**—These are largest (> 6 mm) and may involve 75% of the cornea

There is impaired vision due to progressive myopia. This cannot be corrected by ordinary glasses due to parabolic nature of the corneal curvature.

**Signs**
1. *Early signs*
   i. Ophthalmoscopy shows an ‘oil droplet’ reflex.
   ii. Retinoscopy shows an irregular ‘scissor reflex’.
   iii. Keratometry initially shows irregular astigmatism where the principal meridians are no longer 90° apart and the mires cannot be superimposed.

2. *Late signs*
   i. Conical shape of the cornea is characteristic. The apex of the cone is always situated below the centre of the cornea.
   ii. Placido disc shows distortion of corneal reflex.
   iii. Munson’s sign—There is indentation or acute bulge of the lower lid, when the patient looks down.
   iv. Slit-lamp examination
     a. Vogt’s lines—Fine, parallel lines are seen at the apex. These are vertical folds at the level of deep stroma and Descemet’s membrane.
     b. Fleischer ring—A brownish ring is seen at the base due to haemosiderin pigment.
     c. There is oedema and opacity of the stroma due to rupture in Descemet’s membrane.

**GLAUCOMA**

1. **PRIMARY OPEN ANGLE GLAUCOMA**
IOP > 21 mmHg + chronic slowly progressive with insidious onset + bilateral usually + There is painless, progressive loss of vision. Due to its insidious onset, it is usually noticed when vision is completely lost in one eye and the other eye is seriously impaired + Mild headache and eyeache + A defect in the visual field (noticed by an intelligent patient) + There is increasing difficulty in doing near work + frequent increase in the strength of presbyopic glasses + Light sense is defective. Light minimum is raised and dark adaptation is slowed + Visual acuity decreases gradually. However, it remains good till the late stage as the central field of vision persists. + Cornea is usually clear. + Anterior chamber depth and angle are normal. + Pupillary reactions remain normal until the late stage when they become sluggish + classical triad signs: Raised intraocular pressure + Cupping of the optic disc + Visual field defects

NORMAL TENSION GLAUCOMA (NTG)
The term normal tension glaucoma, also referred to as low tension glaucoma is characterized by typical glaucomatous disc changes with visual field defects, with an intraocular pressure remaining constantly below 21 mm of Hg. It is a variant of primary open angle glaucoma
2. CONGENITAL GLAUCOMA (BUPHTHALMOS, HYDROPHTHALMOS)

Glaucoma appearing between birth and the age of 3-4 years. There is raised intraocular tension present since birth.

Types
2. Infantile glaucoma—It presents between 1-3 years.
3. Juvenile glaucoma—It presents around puberty.

Lacrimation + photophobia + defective vision + enlargement of cornea and the eye as a whole, due to stretching of the sclera + corneal oedema and opacities due to endothelium damage and rupture of Descemet’s membrane (Haabs’ striae) + Deep anterior chamber with iridodonesis + Lens is flattened and displaced backwards + Sclera becomes thin and bluish as the uveal tissue shines through it + buphthalmos (Bull-like eyes), specially when the onset is before the age of 3 years. A normal infant’s cornea measures about 10.5 mm in diameter. A diameter of more than 13 mm confirms enlargement + cupping of optic disc is seen + Optic atrophy usually sets in after the third year.

3. PRIMARY ANGLE CLOSURE GLAUCOMA
Primary angle-closure glaucoma suspect (latent) =
Symptoms are absent + Slit lamp examination.
i. Axial anterior chamber depth is less than normal or decreased
ii. Iris-lens diaphragm is convex in shape
iii. Close proximity of the iris to cornea in the periphery.
Gonioscopy shows an ‘occludable’ angle (less than 20 degrees). The pigmented trabecular meshwork is not visible (Shaffer grade 1 or 0) without indentation or manipulation in at least three quadrants.

Sub-acute or intermittent primary angle-closure glaucoma = There are occasional attacks of raised intraocular pressure with unilateral blurring of vision, coloured halos, mild headache and browache. In between the recurrent attacks the eyes are free from symptoms.
occurs in an anatomically predisposed eye in which physiological factors such as reading in dim illumination or watching television in a dark room precipitates a pupillary block due to mydriasis.

Acute primary angle-closure glaucoma = Acute or congestive angle-closure glaucoma is always
associated with the sudden closure of the angle of anterior chamber with marked elevation of intraocular pressure and congestion (red eye). It is a sight-threatening emergency.

Severe unilateral headache, nausea, vomiting and prostration are often associated. It is often mistaken for an acute abdomen or appendicitis. Sudden onset of intense unbearable pain in the eye due to stretching of the sensory nerves. It radiates along the branches of the 5th nerve. There is marked dimness of vision. It may be reduced to only hand movement or perception of light. Redness, lacrimation and photophobia. Oedema of the lids and conjunctiva (chemosis). Marked conjunctival and ciliary congestion (red eye). Cornea is cloudy (oedematous) and insensitive. Anterior chamber is very shallow. Iris pattern is lost and may be discoloured. Atrophic patches (white or grey coloured) may be seen due to ischaemia. Pupil is moderately dilated and vertically oval. Light and accommodation reflexes are absent. Glaucoma fleckens are small greyish white anterior subcapsuler opacities seen in the lens in the pupillary area. They are diagnostic of previous attack of acute congestive glaucoma. There is markedly raised intraocular pressure (70mmHg or
There may be difficulty in visualizing the fundus due to hazy cornea. There is no cupping but the disc appears to be congested with small haemorrhages. Spontaneous pulsation of the central retinal artery may be seen. Gonioscopy—It reveals abnormally narrow angle of the anterior chamber with or without anterior synechiae. Peripheral anterior synechiae (organized exudates) occur as a result of prolonged and repeated acute congestive attack. Initially there is iridocorneal contact but later on it becomes iridotrabecular. The fellow eye usually has a shallow anterior chamber with a narrow angle.

**Chronic primary angle-closure glaucoma**

In this stage the angle of the anterior chamber becomes slowly and progressively closed resulting in diminution of vision. It is associated with eyeache and headache.

**Pathogenesis**

*Type 1 (Creeping)*—It is caused by gradual and progressive closure of the angle by synechiae over atleast 180 degrees. It always starts superiorly and progresses circumferentially.
Type 2 (Subacute)—It is caused by synechial angle closure as a result of subacute (intermittent) attacks secondary to the pupillary block.

Type 3 (Mixed)—It is caused by combination of primary open angle glaucoma (POAG) with narrow angles. It may be associated with long term use of miotics.

Clinical Features
1. There is diminution of vision associated with eyeache and headache.
2. The eye is irritable and the visual acuity is always impaired.
3. Circumcorneal ciliary congestion is present around the limbus as reddish blue zone.
4. Intraocular pressure is permanently raised when about two-third or more circumference of the angle is closed by peripheral anterior synechiae.
5. Typical scotomatous defects are seen in the visual field.
6. Cupping of the disc appears for the first-time. Thus, it simulates the clinical features of open angle glaucoma. Pallor and shallow temporal shelving may be also seen.

Diagnosis
1. Gonioscopy—It shows variable amount of angle closure.
2. Fundus examination—Cupping of the disc appears for the first-time. Thus, it simulates the clinical features of primary open angle glaucoma.

**Absolute primary angle-closure glaucoma**

The chronic phase if untreated, with or without the intermittent subacute attacks, gradually passes into the final stage of absolute glaucoma.

The eye is completely blind with markedly increased intraocular pressure.

*Painful blind eye* with no perception of light (no PL) + Ciliary congestion is present around the limbus.

+ Cornea is clear and insensitive with

i. Vesicles (bullous keratopathy) may be seen

ii. Filaments (filamentary keratopathy) may be present.

+ Anterior chamber is very shallow + The iris is atrophic (white patches) and may have a broad zone of pigment around the pupil (ectropion of the uveal pigment) due to fibrosis of the iris tissue.

The pupil is grey instead of jet black, dilated and vertically oval + The tension is usually very high and the eyeball is as hard as stone + There is deep cupping of the optic disc.

4. **PSEUDOEXFOLIATION GLAUCOMA (PXF)**

Abnormal basement membranes of aging epithelial cells in the trabeculum, lens capsule, iris, ciliary body
produce grey white fibrillary material that is deposited on anterior lens capsule, zonules, ciliary body, conjunctiva etc.

Usually unilateral chronic open angle glaucoma
CORNEA = Pigment deposition (diffuse/rarely krukenberg spindle) & PXF on endothelium.
Mild aqueous flare + PXF on pupillary margin of iris + spincher atrophy (MOTH-EATEN APPEARANCE AT PUPILLARY MARGIN), LENS shows a central disc & peripheral band of PXR with clear zone in between.
GONIOSCOPY = SAMPAOLESI LINE

5. PIGMENTARY GLAUCOMA
Bilateral + caused by liberation of pigmentary granules from iris pigment epithelium & their deposition throughout anterior segment. Myopia is risk factor, secondary open angle glaucoma, krukenberg spindle in cornea, very deep anterior chamber with floating melanin granules, pigmentary granules on surface of iris & partial loss of pupillary ruff. Radial slit like Transillumination defects, Scheie line on lens, corneal edema, haloes

6. NEOVASCULAR GLAUCOMA
History of ischemic central retinal vein occlusion, Diabetes mellitus, arterial retinal vascular diseases, intraocular tumors etc
Three stages
- **Rubeosis iridis** (tiny capillary tufts at pupillary margin)
- Secondary open angle glaucoma
- Secondary angle closure glaucoma

7. **PHACOLYTIC GLAUCOMA**
Open angle glaucoma, occurs in association with hypermature cataract, proteins leak through intact lens capsule into aqueous humor, pain, poor vision due to cataract, SLIT-LAMP shows corneal edema, hypermature cataract, deep anterior chamber, floating white particles which may form pseudohypopyon.

8. **PHACOMORPHIC GLAUCOMA**
Acute secondary angle closure glaucoma precipitated by intumescent cataract, similar presentation to PACG + shallow anterior chamber + dilated pupil but fellow eye shows deep anterior chamber & open angle

9. **HYPHAEMA**
Blood in anterior chamber, can lead to glaucoma

10. **IRIDOCORNEAL ENDOTHELIAL SYNDROME**
Middle aged woman + unilateral + corectopia (malposition of pupil) + pseudopolycoria
(supernumarary false pupils in a previously normal iris) + iris atrophy of varying severity

CATARACT

1) CONGENITAL CATARACT = unilat/bilateral +
  history (Maternal malnutrition, e.g. as in zonular cataract... Maternal infection by virus, e.g. rubella in the first trimester, Deficient oxygenation due to severe placental haemorrhage, e.g. placenta praevia,... Metabolic disorders of the foetus or infant like galactosaemia, galactokinase deficiency,

Chromosomal abnormalities, e.g. as in Down syndrome (trisomy 21)

Idiopathic

Visual impairment + leukoria (white pupillary reflex) + squint/nystagmus

Plane mirror examination—There is black opacity against a red background.

Ophthalmoscopic examination—There is black opacity against a red background.

+ CLINICAL TYPES =

- punctate/blue dot cataract (It does not interfere with vision usually.)
- **zonular cataract**: Malnutrition and lack of vitamin D may cause zonular cataract.
- **coronary cataract**: It commonly occurs at puberty.
- **anterior capsular** cataract: It is due to the delayed formation of the anterior chamber. It may occur following perforation of a corneal ulcer in ophthalmia neonatorum cases.
- **posterior capsular** cataract: It is often due to persistence of posterior part of vascular sheath. Persistence of hyaloid artery may eventually result in total cataract.

2) **SENILE CORTICAL CATARACT** = history of anticipation (more chances in subsequent generation), usually after 50 years, bilateral but develops in one eye earlier, frequent changes of glasses + glare + diminished visual acuity (It is gradual, painless and progressive) + colored halos + monocular diplopia or polyopia.

**IMMATURE CATARACT** = IRIS SHADOW PRESENT
slit lamp shows demarcation of cortical fibres due to separation by fluids + grey appearance of pupil. (stage of lamellar separation), lens striae + visual impairment + polyopia + colored halos that
make & break on **Fincham’s test** (incipient cataract)

Swollen lens + shallow anterior chamber that may lead to phacomorphic type of secondary glaucoma + colored halos. (intumescent cataract)

**MATURE CATARACT =**

Entire cortex opaque + no swelling + iris shadow absent

**HYPERMATURE CATARACT =**

Milky cortex + morgagnian cataract (brownish nucleus sinks in the bag of liquefied cortex, edge of nucleus seen as brown semi-circular line)

Thickened anterior capsule with calcium that later gets converted to membranous cataract with small nucleus + iridotondesis’ + deep anterior chamber + subluxation of lens due to degeneration of suspensory ligament + phacolytic glaucoma + phacoanalytic uveitis

**3) CUPULIFORM SENILE CATARACT**

dense aggregation of opacities just beneath the capsule usually in the posterior cortex

marked impairment of vision due to the opacity being near the nodal point of the eye + glare + There is loss of ability to see objects in bright sunlight or being blinded by light when driving at night.
Slit-lamp examination (A yellow layer is seen in the posterior cortex). Ophthalmoscopic examination—It is difficult to see the opacity clearly. A vaguely defined opacity is seen in the posterior cortex. It can be detected as a dark shadow on distant direct ophthalmoscopy.

4) **SENILE NUCLEAR CATARACT**
Black cataract (Cataracta brunescens)—The nucleus becomes diffusely cloudy and dark + The cloudiness gradually spreads towards the cortex + Mature cataract + progressive myopia. + Hypermaturity does not occur. Visual impairment + There may be ‘second sight’ or ‘myopic shift’. There is change in refractive index of the nucleus which causes index myopia, resulting in improvement of near vision. Colour shift—The blue end of the spectrum is absorbed more by the cataractous lens. It becomes more obvious after cataract surgery + Blackened pupillary reflex + The details of the fundus cannot be seen due to hazy media.

5) **COMPLICATED CATARACT**
markedly impaired vision + *Slit-lamp examination shows*
i. Irregular borders of opacity extend towards the equator and the nucleus.

ii. Breadcrumb’s appearance is seen.

iii. Polychromatic lustre, i.e. rainbow display of different colours is present.

6) DIABETIC CATARACT
Slit-lamp Examination

i. Immense number of fluid vacuoles appear under the anterior and posterior capsule. It is a reversible process.

ii. Numerous snow flakes are seen all over the cortex causing milky white appearance.

7) PARATHYROID TETANY
Slit-lamp Examination

i. Clouds of small discrete opacities appear in the cortex, separated from the capsule by a clear zone.

ii. They coalesce to form large crystalline flakes.

iii. Lens is opaque usually within 6 months

8) MYOTONIC DYSTROPHY
Clinical features are same as above, i.e. punctate subcapsular cataract is formed

9) MONGOLIAN IDIOCY (DOWN SYNDROME) & CRENISIM
Clinical features are same as above, i.e. punctate subcapsular cataract is formed
10) **GALACTOSEMIA**
bilateral cataract *(oil drop cataract)*

11) **CATARACT DUE TO RADIANT OR HEAT ENERGY**

*Heat (infrared)*— posterior cortical cataract

*Irradiation*— Irradiation by X-ray, ©-rays and neutrons= posterior cortical cataract near posterior pole.

*Electric*— punctate, subcapsular opacities which mature rapidly.

*Ultrasonic radiation*— Lens opacities are formed due to heat and concussion produced by ultrasonic radiation

12) **TRAUMATIC CATARACT**

Early or late *Rosette-shaped*’ cataract is formed usually in the posterior cortex or at times in the anterior cortex or both.

13) **Drug Induced**

Steroids, both systemic and topical are cataractogenic.

Chlorpromazine, Busulphan, Amiodarone,

Gold and Allopurinol are the other drugs associated with cataract.

**SCLERA**
1. **EPISCLERITIS**
Sudden onset + discomfort in eyes/pricking sensation + phenylephrine test positive + moveable swelling + no visual impairment + no complications + **Redness** (mild-fiery red flush) Salmon pink color of lesion + Lesion moves with cotton tip applicator over deeper tissue

2. **SCLERITIS**
Gradual + painful + visual impairment + swelling not moveable + no effect of phenyephrine + complication + frequent systemic association + requires treatment
   - **DIFFUSE SCLERITIS=** Redness + mild pain + distortion of normal radial vascular pattern + no visual loss
   - **NODULAR SCLERITIS=** Moderate pain + non moveable red nodule + visual impairment
   - Necrotizing scleritis (with inflammation)=Pain (mild -> severe) + deep vascular congestion + avascular patch + conjunctival ulceration over scleral necrosis + after resolution sclera appears bluish due to underlying uvea
   - Necrotizing scleritis (without inflammation)= Less pain + history of RA + yellow necrotic scleral patch + progressive exposure of underlying uvea
VITREOUS

1. VITREOUS LIQUEFACTION
Floaters (black moving spots) + loss of fibrillar structure + pockets of fluid with coarse aggregate materials that moves freely in vitreous

2. POSTERIOR VITREOUS DETACHMENT
Above 60 yr + flashes of light + floaters + a ring like opacity

3. MUSCAE VOLITANTES
Physiological opacities represent residue of hyaloids vasculature + black spots like small mosquitoes

4. ASTEROID HYALOSIS
Small white round bodies + Ca containing lipids + history of DM, Hypercholesteremia + unilateral + old age + asymptomatic

5. SYNCYSIS SCINTILLANS
Small white angular & refractile bodies + made of cholesterol + affects already damaged eyes + golden shower appearance + symptomatic but untreatable

6. **INFLAMMATORY OPACITIES**

Pouring of inflammatory exudates in vitreous sec to iridocyclitis, post uveitis, endophthalmitis.

7. **AMYLOID DEGENERATION**

Rare + bilateral + deposition of amyloid in vitreous

8. **PERSISTENT HYPERPLASTIC PRIMARY VITREOUS**

Microphthalmic eye + leucokoria seen shortly after birth + visible long ciliary processes in a dilated pupil + associated cataract & glaucoma

9. **Vitreous Hemorrhage**

Can change into

- Absorption
- Organization forming yellow white debris
- Retinal detachment
- Ghost cell glaucoma
Small hemorrhage

Sudden development of floaters + black shadow in red glow (distant direct ophthalmoscopy)

Large hemorrhage

Sudden painless loss of vision + no red glow (distant direct ophthalmoscopy)

RETINA

1. DIABETIC RETINOPATHY

Background Diabetic retinopathy

• Microaneurysms

• Haemorrhages (flame-shaped = superficial, dot:blot=deep)

• Hard exudates (plasma proteins + lipids + have yellow waxy appearance with relatively distinct margins)
• Retinal edema = gives cystoid appearance to macula

Preproliferative DR

• VASCULAR CHANGES (venous = beading, looping, sausage like segmentation) (arterioles = narrow)

• COTTON WOOL SPOTS = whitish grey areas with indistinct margins

• DARK: BLOT HEMORRHAGES (hemorrhagic renal infarcts)

• INTRARETINAL MICROVASCULAR ABNORMALITIES: arteriovenous shunts

Proliferative diabetic retinopathy

• NEOVASCULARIZATION (new vessel at disc (NVD), new vessels elsewhere (NVE) along course of internal temporal vascular arcades or elsewhere.

• VITEROUS DETACHMENT

• HEMORRHAGE
• PRERETINAL HEMORRHAGE (retrohyaloid space)

• VITREOUS HEMORRHAGE

ADVANCED/COMPLICATED STAGE

• Persistent viterous hemorrhage

• Retinal detachment

2. HYPERTENSIVE RETINOPATHY

GRADING IN OLD INDIVIDUALS by keith wegenar & barker

Grade 1

• Generalized arteriolar constriction + broadening of arteriolar light reflex + concealment of vein by arteriole

Grade 2

• Grade 1 + focal arteriolar constriction + deflection of vein at arteriovenous crossing (Salu’s sign)
Grade 3

- Grade 2 + retinal edema + hard exudates + cotton wool spots + hemorrhage
- Copper wire appearance of arterioles
- Banking of veins distal to AV crossing (bonnet’s sign)
- Tapering of veins on either side of crossing (Gunn’s sign) and right angled deflection of vein

Grade 4

- Grade 3 + disc edema + silver wire appearance of arterioles

MALIGNANT HTN = bilateral papilloedema

Toxemia of pregnancy
• Raised BP+ proteinuria + edema in pregnancy (usually last trimester)

• **STAGE OF ANGIOSPASM** (narrowing of nasal arteries followed by generalized narrowing)

• **STAGE OF HYPOXIC RETINOPATHY** (retinal edema + hard exudates + cotton wool spots + hemorrhage + macular star/ flat macular detachment + exudative retinal detachment)

• **COMPLICATION**= loss of life of mother/fetus, loss of vision

3. **CENTRAL RETINAL VEIN OCCLUSION**

**Ischemic CRVO**

• Sudden onset of severe visual loss

• VA= usually counting fingers or worse

• Marked Afferent pupillary defect

• Raised IOP

• **FUNDOSCOPY**= dilated engorged retinal veins + Dot:blot & flame shaped hemorrhages present in
all 4 quadrants and post pole (tomato splashed appearance)

• cotton wool spots + severe optic disc & macular edema

Non ischemic CRVO

• Most common

• Sudden onset of unilateral painless deterioration of vision

• VA= moderate to severe visual loss

• Mild/absent Afferent pupillary defect

• Raised IOP

• FUNDOSCOPY= absence of spontaneous venous pulsation + dilated engorged retinal veins + Dot:blot & flame shaped hemorrhages present in all 4 quadrants and most numerous in periphery + cotton wool spots + mild optic disc & macular edema

4. BRANCH RETINAL VEIN OCCLUSION
• Edema & hemorrhages limited to retinal area drained by affected vein
• Vision affected only when macular area involved
• Cotton wool spots
• Sec glaucoma rarely

5. CENTRAL RETINAL ATERY OCCLUSION
• Amaurosis fugax = monocular repeated transient episodes of decreased vision/blindness that may occur before visual loss
• Sudden painless severe loss of vision
• VA= profound loss of vision even upto no perception of light
• Pupil= direct pupillary light reflex is absent (total afferent pupillary conduction defect)
• FUNDUS= whitish (opaque) retina + extremely thin retinal arteries + almost normal veins + blood column in veins may be segmented (cattle tracking sign) + cloudy retina + cherry red spot + after few weeks edema subsides, arteries thread like, optic atrophy
6. **BRANCH RETINAL ARTERY OCCLUSION**
   - Retina distal to occlusion becomes edematous with narrow arterioles
   - Later involved area atrophied = permanent sectorial visual defect

7. **AGE-RELATED MACULAR DEGENERATION**

   **ATROPHIC ARMD** = gradual impairment of central vision (month – yrs) + affects both eyes asymmetrically + focal hyperpigmentation / atrophy of RPE & drusen at macula + sharply circumscribed areas of RPE atrophy + loss of choriocapillaries + geographical atrophy of RPE atrophic area increases and coalesce -> preexisting drusen disappears & choroidal vessels become visible

   **EXUDATIVE ARMD** = sudden deterioration of central vision + metamorphosia + sharply circumscribed dome-shaped elevation due to detachment of RPE by
fluid at posterior pole + exudates & hemorrhage in macular area leading to fibrous disciform scar at macula.

8. **CENTRAL SEROUS RETINOPATHY**

Usually unilateral + male (20-40 years) + sudden onset of unilateral painless blurred vision associated with positive scotoma + metamorphosia (wavy distortion of images) + micropsia + decreased visual acuity (correctable with weak + lens), hypermetropia on retinoscopy + oval/round elevation of sensory retina of macular area demarcated by circular ring reflex + absent/ distorted foveal reflex + amsler grid test to confirm metamorphosis + FFA (ink: spot/expansile dot pattern or smoke-stack pattern or mushroom/umbrella like pattern or multiple leak pattern)

9. **RETINOPATHY OF PREMATURITY**

I- Demarcation line between normally vascular & peripheral non-vascular area

II- elevated pink ridge representing mesenchymal shunt joining venules & arterioles
III. ridge + extra-retinal fibrovascular proliferation + neovessels leading to retinal hemorrhage

IV. regression, cicatrition

V. total retinal detachment

10. **RETINITIS PIGMENTOSA**
Hereditary + initially rods affected eventually all rods & cones destroyed + photoreceptor dystrophy occurs starts at equatorial region leading to ring scotoma + tubular vision ultimately leading to blindness + reduced VA + normal color vision + RETINA shows thread like vessels + pigmentary bone corpuscles/ bone spicules + pale wax appearance of optic disc + maculopathy + vit A & E can delay the onset of blindness

11. **RETINOBLASTOMA**
Most common intraocular tumor of childhood + gene on 13q14 + leukocoria + strabismus + defective vision + secondary glaucoma + pseudohypopyon + proptosis (advanced stage) + FUNDOSCOPY=
• ENDOPHYTIC TYPE = pale pink or white mass with newly formed vessels on its surface
• EXOPHYTIC TYPE = appearance of exudative retinal detachment + subretinal mutilobulated white mass
• Pearly white surface of tumor & chalky white inside

12. RETINAL DETACHMENT
• RHEGMETOGENOUS = floaters + flashing light (photopsia) + sudden painless fall of vision + field defects (dark curtain) + decreased V.A + RAPD in case of total detachment + decreased IOP by 5 mmHg + mild anterior uveitis + shaffer’s sign (anterior vitreous shows tobacco dust like opacities) + RETINA shows grey/translucent appearance + loss of normal choroidal pattern + bullous/corrugated appearance + tortuous dark blood vessels + arteries & veins appear to have blood of same color
• TRACTIONAL = viterioretinal band + decreased retinal mobility + increased elevation of retina at site of traction
• **EXUDATIVE/SEROUS** = smooth, convex appearance + change in position of detached area with gravity.