CHAPTER

6

Iris and Anterior Chamber

COMPETENCY

OP6.1 DESCRIBE CLINICAL SIGNS OF INTRAOCULAR INFLAMMATION AND ENUMERATE THE FEATURES THAT DISTINGUISH GRANULOMATOUS FROM NONGRANULOMATOUS INFLAMMATION. IDENTIFY ACUTE IRIDOCYCLITIS FROM CHRONIC CONDITION

LONG ESSAYS

1. A 24-year-old woman presents with gradual onset of pain and redness in her right eye. on examination, there is ciliary congestion, corneal endothelium is studded with greasy mutton fat keratic precipitates in the inferior part. The pupil is sluggish, and the anterior chamber shows 2+ cells and 2+ flare.

A. What is the most probable diagnosis and what is its type? (2 marks)
B. Name three causes for this condition. (3 marks)
C. Explain the influence of this condition on intraocular pressure. (3 marks)
D. Name two posterior segment signs of this condition. (2 marks)

A. Most Probable Diagnosis

- Iridocyclitis (anterior uveitis)
- It is of 'granulomatous' type

B. Causes for Granulomatous Iridocyclitis

1. Tuberculosis
2. Sarcoidosis
3. Vogt–Koyanagi–Harada syndrome

C. Influence of Iridocyclitis on Intraocular Pressure

Normal IOP	Low IOP	High IOP
Iridocyclitis can be associated with normal intraocular pressure	More often, iridocyclitis causes low IOP due to ciliary shock, which recovers with the initiation of treatment	High IOP is seen in the following conditions • Trabeculitis • Annular synechiae causing the pupillary block and angle-closure • Peripheral anterior synechiae • Steroid-induced

D. Posterior Segment Signs of Iridocyclitis

1. Cystoid macular edema
2. Epiretinal membrane

2. A 44-year-old man with HIV presents with floaters and diminution of vision in his right eye. His CD4+ count is 120/mm^3.

A. Enumerate three causes for floaters. (3 marks)

B. Name three organisms that can cause posterior uveitis in a patient with HIV. (3 marks)

C. Explain the importance of knowing the CD4+ count. (3 marks)

D. Name the typical description for active ocular toxoplasmosis. (1 mark)

A. Causes of Floaters

1. Muscae voltantes (physiological)
2. Vitreous hemorrhage
3. Intermediate uveitis

B. Organisms Causing Posterior Uveitis in HIV Infected Patients

1. *Mycobacterium tuberculosis*
2. *Toxoplasma gondii*
3. Cytomegalovirus

C. Importance of knowing the CD4+ count

- The ocular manifestations are due to the infection *per se* or opportunistic infections or due to the drugs used against HIV/AIDS
- The opportunistic infections depend on the CD4+ cell count. Knowing the CD4+ count helps in expecting the organism and diagnosis

Manifestations	CD4+ cell count 0–199	CD4+ cell count 200-499	CD4+ cell count >500
Adnexal lesions	—	Blepharitis	—
Anterior segment	Ocular surface squamous neoplasia	Dry eye Kaposi sarcoma Molluscum contagiosum Herpes zoster ophthalmicus	Allergic conjunctivitis
Posterior segment	CMV retinitis Progressive outer retinal necrosis Candidal retinochoroiditis Endophthalmitis	Toxoplasmosis Intermediate uveitis Retinal vasculitis Tuberculous uveitis	HIV retinopathy Optic neuropathy

D. Typical description for active ocular toxoplasmosis

'Headlight in the fog appearance'.

3. A 24-year-old man had trauma to his right eye with a stone while using an electric motor. After two days he developed severe pain,loss of vision, and redness in the same eye

A. State the most probable diagnosis. (1 mark)
B. Explain the etiology and clinical features of this condition. (4 marks)
C. Explain the investigations and treatment of this condition. (5 marks)

A. Most Probable Diagnosis

Post-traumatic endophthalmitis.

B. Etiology and Clinical Features

Etiology

- Infective endophthalmitis
  - Bacteria (most commonly in post-operative is Staphylococcus, Strepcocci)
  - Fungi: More in vegetative injury
- Sterile endophthalmitis
  - Post-operative sterile endophthalmitis (TASS)
  - Pure copper foreign body
  - Tumor necrosis

Clinical Features

Symptoms

Pain, loss of vision.

Signs

- Lid edema
- Hazy cornea, anterior chamber reaction, hypopyon
- Edges of the wound may be swollen and gaping
- Yellow reflex in the pupil
- Exudation in the vitreous—fundus may not be visible

C. Investigations and Treatment

Investigations

- Aqueous and vitreous tap for culture and sensitivity with Gram's stain will help understand the organism
- Diagnosis is confirmed by B-scan. It shows multiple moderately echogenic opacities within the vitreous. Any associated retinal detachment may be seen

Treatment

Intravitreal Injection

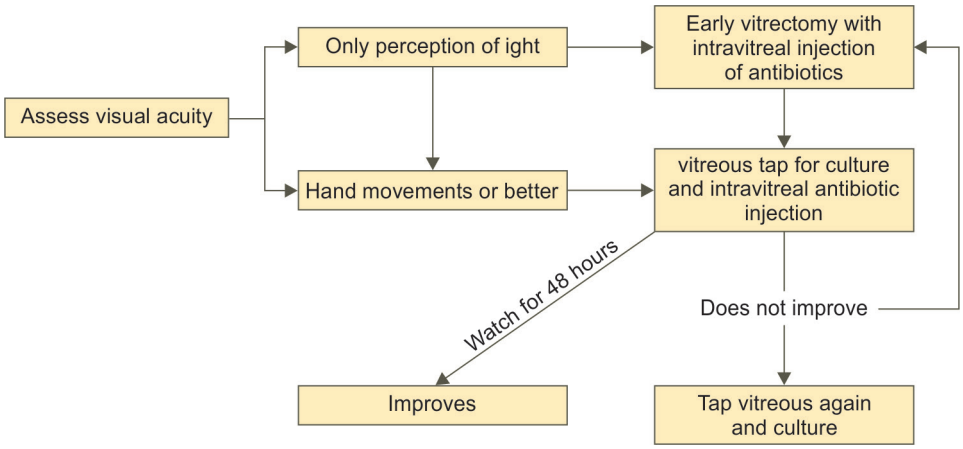
- Broad-spectrum antibiotics is given in the pars plana region (3 mm behind limbus in pseudophakic eyes, 3.5 mm behind limbus in phakic eyes)
- The usual antibiotics preferred are vancomycin (0.1 mg/0.1 ml) and ceftazidime (2.25 mg/0.1 ml)
- Other options are vancomycin + amikacin (0.4 mg/0.1 ml) and vancomycin + gentamicin (0.2 mg/0.1 ml)

Subconjunctival Injection

- Combination of antibiotics with steroids is a form of adjunct therapy
- It is given for a week to maintain intraocular concentration
  - Vancomycin 25 mg/0.5 ml + ceftazidime 100 mg/0.5 ml
  - Vancomycin + cefuroxime 125 mg/0.5 ml
- Subconjunctival dexamethasone (4 mg/ml) is given to decrease the inflammation under the cover of antibiotics

Topical Antibiotics

- Vancomycin 50 mg/ml or cefazoline (50 mg/ml) + amikacin (20 mg/ml) or tobramycin (15 mg%)
- Topical steroids like prednisolone and dexamethasone are frequently instilled
- Supportive therapy in the form of cycloplegics and antiglaucoma medications has to be started
- A study-endophthalmitis vitrectomy study (EVS) outlines the treatment protocol for postoperative endophthalmitis



SHORT ESSAYS

1. Give the SUN classification of uveitis. (5 marks)

SUN Classification of Uveitis

1. **Anterior uveitis**
   - i. Iritis (iris inflammation)
   - ii. Anterior cyclitis (ciliary body inflammation)
   - iii. Iridocyclitis
2. **Intermediate uveitis**
   - i. Pars planitis
   - ii. Posterior cyclitis
   - iii. Hyalitis
3. **Posterior uveitis**
   - i. Choroiditis (focal or diffuse)
   - ii. Retinitis

iii. Retinochoroiditis
iv. Chorioretinitis
v. Retinal vasculitis
vi. Neuroretinitis

4. **Pan uveitis**
   i. Inflammation in the anterior chamber, vitreous, and retina/choroid

2. Explain the formation of keratic precipitates (KPs). Explain the classification based on appearance. (2+3 marks)

Formation of KPs

- In iridocyclitis, the corneal endothelium becomes sticky
- There are inflammatory cells in the aqueous that keep floating with the convection currents of the aqueous. They stick to the lower part of the cornea and are termed 'keratic precipitates'

Classification of KPs

Fine KPs (small, non-pigmented)	They are associated with acute nongranulomatous inflammation
Large, mutton fat, greasy KPs	They are associated with granulomatous inflammation
Pigmented, crenated KPs	They are a sign of chronic or healed anterior uveitis
Diffuse, stellate KPs	Occurrence of KPs above the horizontal midline Viral uveitis, Fuch's heterochromic iridocyclitis

3. Classify uveitis based on etiology. (5 marks)

Etiological Classifcation of Uveitis

1. **Infective uveitis**
   - It can be due to exogenous (entry of organism from the environment), secondary (spread from other ocular structures), or endogenous (through blood from distant sources)
     i. *Bacteria:* Mycobacteria, *Brucella*, *Treponemes*, streptococci, staphylococci
     ii. *Viruses:* Herpes simplex and zoster, cytomegalovirus, human immunodeficiency virus
     iii. *Parasites: Toxoplasma*, *Toxocara*, onchocerciasis
     iv. *Fungi: Candida*, *Aspergillus*, cryptococci
2. **Allergic uveitis**
   i. Microbial allergy
   ii. HLA associated: B27: ankylosing spondylosis, Reiter's; B5: Behcet's, DR4 and DW15: VKH syndrome
   iii. Autoimmune: Associated with Still's disease, rheumatoid arthritis, Wegener's granulomatosis
3. **Toxic uveitis**
   i. Endotoxins: Uveitis associated with acute pneumococcal or gonococcal conjunctivitis, keratomycosis
   ii. Endocular toxins: Uveitis associated with long-standing retinal detachment, tumors (masquerade syndromes), and phacotoxic uveitis
   iii. Exogenous toxins: Drugs, chemical irritants

4. **Traumatic uveitis**
   - It is due to:
     i. Mechanical injury
     ii. Entry of foreign body or organism
     iii. Blood
     iv. Sympathetic ophthalmia in the other eye
5. **Uveitis associated with noninfective systemic diseases**
   - Polyarteritis nodosa, SLE, diabetes, gout, etc.
6. **Idiopathic uveitis**
   - Pars planitis, Fuch's heterochromic iridocyclitis, other nonspecific idiopathic uveitis

4. Describe the anterior segment signs of iridocyclitis. (5 marks)

Cornea

- Circum corneal congestion or ciliary congestion is congestion of the anterior ciliary vessels
- Ciliary tenderness is elicited by gentle pressure over the ciliary region
- Keratic precipitates (Fine KP, mutton fat KPs)
- Band shaped keratopathy (late stages)

Iris

- Muddy iris
- Koeppe nodules: Nodules at pupillary border
- Busacca nodules: Nodules on the iris surface
- Berlin nodules: Nodule in angle
- Festooned pupil: The pupil is irregular due to patchy posterior synechiae at various positions. It is discovered on pupillary dilatation
- Ectropion pupillae: eversion of the iris surface leading to the visibility of the posterior surface of the iris. It happens due to the contraction of fibrinous exudate on the anterior surface of the iris
- Seclusio pupillae formation of posterior synechiae in an annular fashion that blocks the outflow of aqueous from the posterior chamber to anterior chamber
- Occlusion pupillae: Blocking of the pupil by exudates

Anterior Chamber

- Cells
  - A sign of activity, are seen as particles in the aqueous
  - It is graded as follows:

Grade	Cells in field
0	<1
0.5+	1–5
1+	6–15
2+	16–25
3+	26–50
4+	>50

- Flare
  - A measure of turbidity of the aqueous
  - Its grading is shown below

Grade	Description
0	None
1+	Faint
2+	Moderate (iris and lens details clear)
3+	Marked (iris and lens details lazy)
4+	Intense (fibrin or plastic aqueos)

- Plasmoid aqueous
  - Turbid aqueous when the protein concentration of aqueous is as high as plasma
- Hypopyon
  - It is formed due to collection of exudates in the anterior chamber
- Depth of the anterior chamber varies with the clinical situation

Seclusio pupillae	Shallow at the periphery, deep at center
Occlusio pupillae	Diffusely shallow
Peripheral anterior synechiae	Irregularly shallow

- Lens
  - Pigments on the anterior lens capsule
  - Posterior synechiae
  - An exudative membrane on the lens capsule
  - Complicated cataract

5. Compare and contrast granulomatous and nongranulomatous uveitis with examples. (5 marks)

Or

Explain the pathological classification of uveitis. (5 marks)

Feature	Nongranulomatous uveitis (plastic)	Granulomatous uveitis
Cause	Due to toxins, antigen, and antibody reactions. (Types II and III)	Usually due to invasion of an organism or due to type IV hypersensitivity
Pathology	Polymorphonuclear cells, lymphocytes	Giant cells, epithelioid cells, macrophages
Onset	Acute	The usually slow, indolent course
Clinical course	Short	Chronic, remissions exacerbations
Grade of inflammation	High	Low
Keratic precipitates	Many, small	Less, large mutton fat KPs
Aqueous cells and flare	Marked, numerous cells, fibrinous exudates commonly seen	Less, a few cells
Iris nodules	Absent	Present

Contd.

Feature	Nongranulomatous uveitis (plastic)	Granulomatous uveitis
Posterior synechiae	Thin and tenuous	Thick and broad-based
Posterior segment involvement	Not commonly involved	Commonly involved
Examples	Viral, HLA B27 associated uveitis, Behcet syndrome, JRA	Tuberculosis, VKH, sympathetic ophthalmia, sarcoidosis Tuberculosis can cause both granulomatous and nongranulomatous types of uveitis

6. Explain the clinical features and management of intermediate uveitis.

(5 marks)

Intermediate Uveitis

- An insidious, chronic, relapsing inflammation primarily of the vitreous
- They tend to be bilateral and more in females
- Its subset, pars planitis is the term used for idiopathic inflammation
- Intermediate uveitis can be associated with multiple sclerosis (HLA DR15), Lyme's disease, and sarcoidosis. An idiopathic variant is more commonly seen in children and tends to be aggressive
- IU associated with systemic disorders occurs in the third and fourth decades

Clinical Features

Symptoms

- The most common presenting complaint is unilateral or bilateral floaters associated with diminished vision
- Metamorphopsia

Signs

- On examination, the eye may show signs of anterior uveitis, especially in those with systemic associations
- Vitreous cells and haze, more so in the anterior part, are pathognomonic for IU
- Vitreous may also be studded with exudates, called 'snowballs' appearance
- The peripheral retinal examination is mandatory in these cases, which reveal snow banking, i.e. whitish-gray exudative membrane is seen circumferentially; more so inferiorly
- Neovascularization may develop on the snowbanks
- Perivascular sheathing and periphlebitis can also be seen
- Cystoid macular edema can also be present in many cases as a result of inflammation

Management

Investigations

- FFA helps to detect peripheral neovascularization
- OCT helps to detect cystoid macular edema
- Following a thorough ophthalmic examination, systemic associations of IU as mentioned before have to rule out

Treatment

- 80% of cases resolve spontaneously, hence may not require treatment
- In severe non-resolving conditions, steroids are given in the form of subTenon injections or oral administration
- If associated with multiple sclerosis, interferon-beta may be of help
- In presence of neovascularization in the peripheral retina, laser photocoagulation is indicated
- Vitrectomy: It is indicated when there is no response to steroids, vitreous opacities significantly decreasing vision, unresolving cystoid macular edema, vitreous hemorrhage, tractional retinal detachment, and epiretinal membrane
- Accompanying endo-LASER can be done if associated neovascularization is present

7. Explain the clinical features of posterior uveitis. (5 marks)

Posterior Uveitis

- Inflammation of the choroid and/or retina
- It can be infectious or noninfectious

Clinical Features

Symptoms

- The patient usually presents with visual symptoms predominantly associated with floaters
- There is no pain or redness of the eye as such
- In cases of peripheral lesions, no symptoms may be reported and hence tend to be accidentally found on routine examination
- The other symptoms are metamorphopsia (distorted images), macropsia (due to increase in distance between photoreceptors), micropsia (due to crowding of photoreceptors), photopsia (sensation of flashes of light), and positive scotoma

Signs

- One may find a few KPs and the anterior segment is usually quiet
- Fundus examination may reveal vitreous cells and flare
- Vitritis is more commonly seen with retinitis lesions
- Fundus examination shows retinitis/choroiditis/retinal vasculitis/a combination of these in the form of focal, multifocal, or diffuse lesions.
  - The retinitis patch looks yellowish bright spot with fuzzy margins, obscuring the blood vessels
  - Choroiditis is seen as pale yellow subretinal lesion with normal overlying blood vessels
  - Retinal vasculitis is seen sheathing of veins or arteries or both. There may be associated hemorrhages or capillary occlusion
- After resolution, either due to therapy or remission
  - Retinitis lesion becomes atrophic, thinned out and translucent
  - Choroiditis lesion heal by pigmentation. The vessels which run through that segment appear thin and sclerotic. Sclera may be visible below and may simulate a coloboma
- In cases of severe posterior uveitis, complications like complicated cataracts, choroidal neovascularization, secondary glaucoma, and rhegmatogenous retinal detachment can occur

- Some of the important patterns of posterior uveitis are listed below:

Congenital toxoplasmosis	Usually bilateral punched out lesions simulating macular coloboma
Acquired toxoplasmosis	Headlight in the fog appearance: intense vitreous haze with creamy yellow retinitis patch Arising from the edge an old healed lesion
CMV retinitis	A brush-fire pattern of full-thickness retinal necrosis with hemorrhages and exudation
Tuberculosis	Choroid tubercles (yellowish subretinal lesions of varying size) usually associated with meningitis, tubercular conglomerate granuloma (solitary) Serpiginous-like choroiditis: centrifugal spread of choroiditis with healing seen in the other end
Sarcoidosis	Choroidal and retinal granulomas, periphlebitis retinae: sheathing around veins resembling 'candle wax drippings
Toxocariasis	Chronic endophthalmitis: Presents with leukocoria due to marked vitreous clouding, in children of the age of 2–10 years, poor prognosis Posterior pole granuloma: A yellow-white, round, solitary, raised nodule, mild inflammatory response, about 1–2 disc diameter in size, located either at the macula or in the centrocaecal area, in children of age 5–15 years, presents with decreased vision Peripheral granuloma: Can have vitreous bands and develop retinal detachment

8. Describe the etiology, clinical features, and management of sympathetic ophthalmia. (1+2+2 marks)

Or

A 56-year-old woman presents with left-sided ocular pain and redness. Granulomatous panuveitis is diagnosed in the left eye. She gives a history of penetrating injury and loss of vision in the right eye. State the diagnosis. Explain the signs and management of this condition. (1+2+2 marks)

Sympathetic Ophthalmia

A bilateral serious, chronic granulomatous, panuveitis due to sensitization to choroidal antigen due to either injury or surgery in the fellow eye.

Predisposing Factors and Etiology

1. Injury in the ciliary region
2. Penetrating injury/vitreoretinal surgery
3. Incarcerated ciliary body/lens capsule or iris
4. Can occur in any age group
   - It is thought to be due to a T cell-mediated response driven against sensitized uveal antigens (Fig. 6.1.1)
   - Viral infection may be initiating factor
   - The eye which is traumatized is called the exciting eye
   - The other eye is called the sensitizing eye

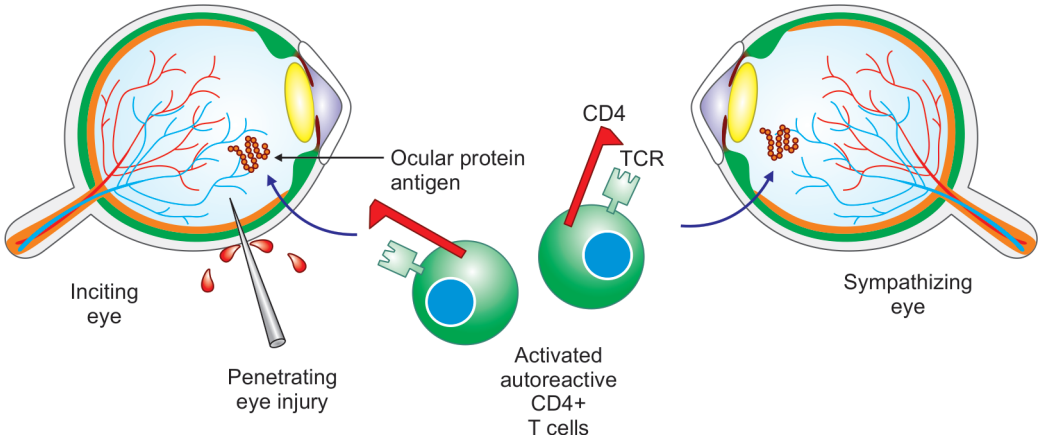


Fig. 6.1.1: Pathogenesis of sympathetic ophthalmia

Clinical Features

Prodromal Stage

- When the sympathetic ophthalmia begins, there is a return of ciliary congestion, tenderness, and lacrimation in the shrunken eye
- In the sympathizing eye, the first symptom to begin is photophobia and defective near vision as a result of the weakness of accommodation
- A careful examination may reveal ciliary tenderness, KPs, vitreous cells, and flare
- Fundus examination reveals Dalen–Fuch nodules (yellow subretinal lesions)

Established Stage

- Full-fledged granulomatous uveitis features may be seen: Iridocyclitis, peripheral retinal exudation, Dalen–Fuch nodules, and papillitis
- It tends to be chronic and may last many months
- Inflammation may be severe enough to cause complications like phthisis

Prophylaxis

- Meticulous repair of any penetrating injury to take care of the uveal tissue not to be incarcerated in the wound
- Uncontrolled uveitis in the severely damaged eye for more than 2 weeks should be enucleated

Treatment

- Early recognition of signs of inflammation and starting steroid therapy in the form of intravenous methylprednisolone 1000 mg followed by oral prednisolone 1 mg/kg and topical steroids and cycloplegics
- After the eye quietens down, a maintenance dose of 15–20 mg is administered for many months and the eye has to be monitored
- Enucleation of the exciting blind eye is proposed by some but must be done before the initiation of inflammation

9. Explain various complications of uveitis. (5 marks)

Complications of uveitis can be due to inflammation per se and due to the treatment of uveitis.

Complications due to Inflammation

1. **Cornea**
   - Band keratopathy: It usually occurs in chronic inflammation
2. **Angle**
   - Peripheral anterior synechiae form between the peripheral iris and the cornea that bridge the angle and cause a block of aqueous outflow
3. **IOP**
   - Hypotony and hypotonous maculopathy occur as a result of ciliary shock and ciliary shutdown
   - Raised intraocular pressure can occur as a result of trabeculitis and scarring of the trabecular meshwork
4. **Iris**
   - Posterior synechiae form between the lens and posterior surface of the iris
   - Iris bombe forms due to annular synechia that causes forward displacement of the peripheral iris causing secondary angle-closure glaucoma
5. **Lens**
   - Complicated cataract, usually seen as posterior subcapsular cataract with bread-crumb appearance
   - Poor dilatation during cataract surgery occurs due to thinning of the iris and formation of posterior synechiae
   - Cyclitic or retrolental membrane forms due to the organization of exudates behind the lens
6. **Retina**
   - Cystoid macular edema is the most common retinal complication associated with uveitis. It is a frequent cause of diminished vision
   - Epiretinal membrane
   - Retinal detachment (exudative)
   - Subretinal fibrosis
7. **Phthisis bulbi**
   - Occurs as a result of chronic inflammation leading to ciliary body shut down leading to low IOP and complete loss of vision

Complications due to Treatment

1. **Steroids**
   - Steroid-induced glaucoma
   - Steroid-induced cataract

10. Describe the etiology, clinical features, and management of Pan ophthalmitis. (5 marks)

Pan ophthalmitis

Inflammation of all the coats of the eyeball along with orbital tissues.

Etiology

- Exogenous entry of organisms can follow penetrating injury or postoperatively. The usual organisms implicated are gram-positive cocci
- Endogenous infection is seen in severely immunocompromised individuals with indwelling catheters and prostheses

Clinical Features

Symptoms

- Severe eye pain
- Complete loss of vision

Signs

- On examination, visual acuity is negative to the perception of light
- The lids are edematous, the eyeball is proptosed and extraocular movements are restricted due to orbital edema
- Conjunctival chemosis and congestion are present
- The cornea is usually sloughing off with an anterior chamber full of purulent material
- Fundus cannot be visualized. Intraocular pressure is raised

Management

- Evisceration is the treatment of choice. Supportive systemic antibiotics and anti-inflammatory drugs are given
- Evisceration is the removal of the entire contents of the eyeball leaving behind the sclera
- Evisceration can be done in two methods: Simple and frill evisceration
- In the former, the whole sclera is left behind

SHORT ANSWERS

1. Explain the clinical features of plastic iridocyclitis. (3 marks)

Plastic Iridocyclitis

- Another name for nongranulomatous iridocyclitis
- It is usually acute and is associated with intense inflammation

Clinical Features

Symptoms

- Patients present with acute onset of redness, periocular pain, and photophobia
- Visual acuity depends on the severity of the inflammation

Signs

- The eye is congested, with dilated ciliary vessels. Ciliary tenderness is usually present
- The cornea shows fine, nonpigmented KPs in the inferior aspect of the cornea. Corneal edema may be present
- In the anterior chamber, there is a reaction in the form of cells and flare. In non-granulomatous uveitis, the reaction tends to be intense with 'plasmoid aqueous'. The protein content of the aqueous may be as that of plasma
- The iris is muddy and engorged. The pupil is usually miotic
- Hypopyon is usually present

2. Explain the terms **(3 marks)**

A. Occlusio pupillae

B. Seclusio pupillae

C. Iris Bombe

A. Occlusio Pupillae

Occlusion of the pupillary aperture due to exudates.

B. Seclusion Pupillae

Occlusion of the aqueous outflow through the pupil due to formation of annular or ring synechiae circumferentially.

C. Iris Bombe

- When annular synechiae forms, the aqueous cannot escape through the pupil.
- As a result, it builds up in the posterior chamber and pushes the peripheral iris towards the angle.
- This causes bulging of the peripheral iris forward to cause 'iris bombe' (Fig. 6.1.2)

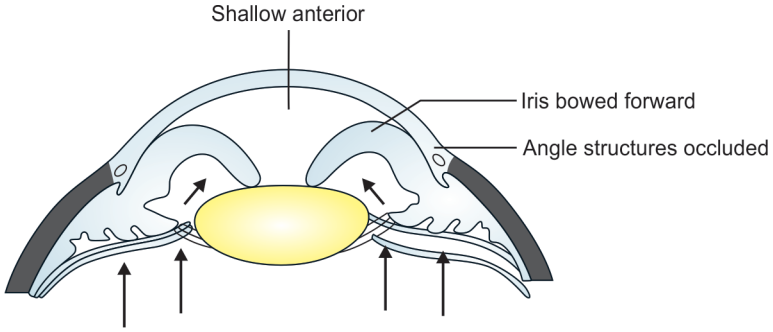


Fig. 6.1.2: Iris bombe

3. Explain the pupillary changes that can occur in uveitis. **(3 marks)**

- The pupil becomes small and the reaction becomes sluggish in acute iritis
- Iris nodules
  - It may be studded with an inflammatory nodule at the border (Koeppe's nodules) or the collarette (Busacca's nodules) as seen in granulomatous inflammation.
  - The Koeppe's nodules form posterior synechiae
- The pupil may be irregular due to patchy posterior synechiae at various positions (Festooned pupil, Fig. 6.1.3)
  - It is discovered after instilling mydriatics.
  - The adhered part of the iris does not dilate whereas the free iris moves behind
- Ectropion pupillae
  - Eversion of the iris surface leading to the visibility of the posterior surface of the iris
  - It happens due to the contraction of fibrinous exudate on the anterior surface of the iris

Fig. 6.1.3: Festooned pupil

4. Justify the reason for keratic precipitates being present in the inferior half of the cornea. Name one condition where this rule is not followed. (2+1 marks)

KPs are seen on the inferior half of the cornea

- Keratic precipitates are a sign of uveitis. In uveitis, the endothelium of the cornea becomes sticky
- As a result, polymorphonuclear cells that are released into the aqueous get attached to the endothelium
- The anterior of the eye is relatively cooler than the posterior due to exposure to air and evaporation of the tear film. As a result convection currents are set up in the aqueous. The aqueous that is closer to the cornea, sinks and the aqueous that is near the iris rises (Fig. 6.1.4)
- During this process, the keratic precipitates settle down in the form of an 'Artl's triangle'

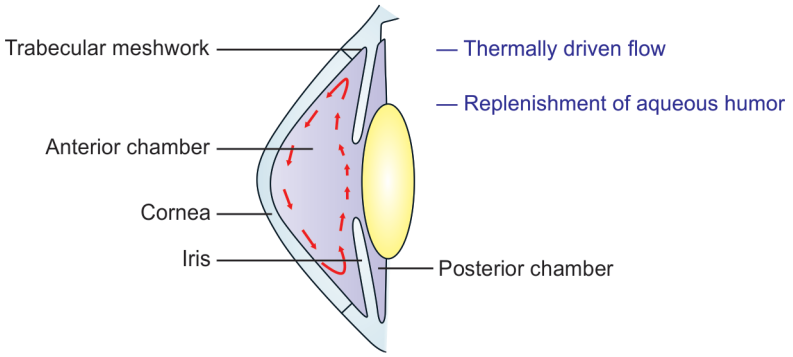


Fig. 6.1.4: Convection currents

Condition in which this Rule is not Followed

Stellate diffuse KPs are characteristically seen in herpetic iridocyclitis and Fuch's heterochromic cyclitis

5. Compare and contrast atrophic bulbi and phthisis bulbi. (3 marks)

- Atrophic bulbi is the initial stage of phthisis bulbi
- Phthisis bulbi is also known as atrophic bulbi with disorganization.

Similarities	Differences
• Both have no visual acuity • The lens is cataractous • The retina is atrophic and detached • The eye is smaller than usual and IOP is low	• Phthisis bulbi is a shapeless eye and does not have a structure, whereas atrophic bubli still maintains the original eyeball shape • Phthisis bulbi is associated with calcifications in all parts of the eye evident on CT scan or B scan. There is bone metaplasia evident in RPE • The cornea becomes sclera-like and the sclera becomes markedly thickened in phthisis bulbi. The parts of the eyeball cannot be differentiated from one another

6. Enumerate three indications of evisceration. (3 marks)

1. Expulsive choroidal hemorrhage
2. Panophthalmitis
3. Bleeding anterior staphyloma

COMPETENCY

OP6.2 IDENTIFY AND DISTINGUISH ACUTE IRIDOCYCLITIS FROM CHRONIC IRIDOCYCLITIS

SHORT ESSAY

1. Differentiate between acute and chronic iridocyclitis. (5 marks)

Differentiating Features

Parameter	Acute Iridocyclitis	Chronic Iridocyclitis
Symptoms	• Acute onset of pain, brow ache, and hazy vision • Lasts for about 6 weeks to 3 months	• Chronic indolent course, with acute exacerbations • Diminished vision is a more common presentation • Duration >3 months
Signs	• Ciliary congestion and tenderness • Corneal edema • Fresh KPs • Anterior chamber cells and flare, • Exudative membrane, hypopyon may or may not be there	• No congestion • Old crenated KPs • Anterior chamber flare is more in comparison with cells • Presence of posterior synechiae • A complicated cataract may be present • Peripheral anterior synechiae and glaucoma may be present
Complications	• Hypertensive uveitis • Ciliary shock and hypotony • Disc edema and hypotonus maculopathy • Choroidal detachment	• Complicated cataract • Annular synechiae and pupillary block glaucoma • Retinal detachment due to contraction of fibrovascular membrane • Neovascular glaucoma • Phthisis bulbi • Band-shaped keratopathy
Pathology	• Usually nongranulomatous response	• May be granulomatous or nongranulomatous
Examples	• Idiopathic anterior uveitis • Traumatic uveitis • Bacterial endophthalmitis • HLA B27 associated iridocyclitis	• Cyclitis • Intermediate uveitis • Sympathetic ophthalmia • VKH syndrome • Tuberculous uveitis • Syphilitic uveitis • Stills disease or juvenile idiopathic arthritis-associated uveitis

SHORT ANSWERS

1. Define (1 mark each)

A. Acute uveitis
B. Chronic uveitis
C. Recurrence of uveitis

A. Acute Uveitis

Onset is sudden and duration of symptoms and signs of uveitis last not more than 3 months.

B. Chronic Uveitis

Insidious onset, duration of disease lasting for more than 3 months.

C. Recurrent Uveitis

Repeated attacks of uveitis separated by periods of inactivity of more than 3 months without treatment.

2. Enumerate three causes for chronic uveitis. (3 marks)

1. Fuch's heterochromic cyclitis
2. JIA associated uveitis
3. Syphilitic uveitis

3. Describe three signs of chronic uveitis. (3 marks)

Signs of Chronic Uveitis

1. **Visual acuity**
   - Maybe subnormal owing to sequelae of uveitis
2. **Cornea**
   - Old crenated KPs
   - Band keratopathy
3. **Anterior chamber**
   - Flare is more common than cells and is out of proportion to cells
   - Depth may be uneven due to the formation of anterior synechiae
4. **Iris**
   - Areas of iris atrophy may be present
5. **Lens**
   - Complicated cataract
   - Pigments on the lens
6. **Retina**
   - Subretinal fibrosis
   - An epiretinal membrane may be present
   - Chronic macular edema

COMPETENCY

OP6.3 ENUMERATE SYSTEMIC CONDITIONS THAT CAN PRESENT AS IRIDOCYCLITIS AND DESCRIBE THEIR OCULAR MANIFESTATIONS

SHORT ESSAYS

1. A 36-year-old man with back pain and stiffness presented with recurrent episodes of iridocyclitis. On further investigating, an X-ray of the lumbar spine shows a "bamboo-spine" appearance.

A. Which systemic condition is being described here and what is the HLA type expected? (2 marks)

B. Enumerate other two systemic manifestations of the same HLA type with ocular manifestations. (1 marks)

C. Annotate the management of this condition. 2 marks)

A. The Systemic Condition Described Here is

- Ankylosing spondylosis
- It is associated with HLA B27

B. Other Conditions Associated with HLA B27

1. Reiter's disease
2. Psoriatic arthritis

C. Management

- Uveitis associated with HLA B27 is usually unilateral, acute, and anterior
- It is treated with topical steroids and cycloplegics
- The cycloplegics have to be continued for three weeks after control of inflammation to decrease recurrence
- In case of recurrent disease, systemic NSAIDs and monoclonal antibodies should be used

2. Enumerate four systemic conditions that are associated with anterior uveitis. Describe the features of Heerfordt syndrome. (4+1 marks)

Systemic Conditions That are Associated with Anterior Uveitis

1. Ankylosing spondylosis
2. Reiter's disease
3. Inflammatory bowel disease
4. Tubulointerstitial nephritis

Heerfordt's Syndrome

- It is also known as uveoparotid fever and is characterized by bilateral granulomatous panuveitis + painful enlargement of parotid glands + cranial nerve palsies + skin rashes + fever
- It is seen as an acute manifestation of Sarcoidosis

3. Name the systemic autoimmune disorder in which the eye develops a recurrent "cold hypopyon"? Describe other ocular manifestations of that disease. (1+4 marks)

Systemic Autoimmune Disorder is

- Cold hypopyon is seen characteristically in Behcet's syndrome
- It is characterized by recurrent episodes of anterior uveitis with hypopyon with minimal signs of inflammation like cells and flare. So, it is described as a 'cold hypopyon'

Ocular manifestations of Behcet's Syndrome

- Behcet's disease is an idiopathic multisystem disorder characterized by obliterative arteritis affecting vessels of all sizes
- The uveitis is typically bilateral and anterior. Iridocyclitis is recurrent and associated with hypopyon
- There is minimal inflammation in the form of cells and flare
- Posterior uveitis is characterized by vitritis, periphlebitis retinae, and retinitis that can present as white creamy exudates. It can also cause panuveitis

4. Describe the manifestations of tuberculosis in the eye. (5 marks)

- Ocular manifestations of tuberculosis are due to hematogenous spread of the organism or due to a hypersensitivity triggered by tubercular protein.
- It has varied manifestations from the ocular surface to the retina

Lid	• Lupus pernio, cold abscess (rare)
Conjunctiva	• Granuloma can occur in the conjunctiva • Phlyctenular conjunctivitis is a type IV hypersensitivity reaction to tubercular protein and is commonly seen in children. As a result, there is a formation of a pinkish nodule with feeding blood vessels. They respond well to topical steroids
Sclera	• Scleritis can occur as a hypersensitivity reaction • A scleral ulcer is an infectious process
Cornea	• Interstitial keratitis is a result of a hypersensitivity reaction
Uvea	• Anterior uveitis: It is usually granulomatous with mutton-fat KPs and iris nodules • Intermediate uveitis is characterized by vitritis and snow banking • Choroid tubercles, granuloma, serpiginous-like choroiditis are posterior segment manifestations of TB • Choroid tubercles are subretinal lesions of smaller dimensions usually seen in the posterior pole. They are seen in association with military tuberculosis and CNS tuberculosis
Retina	• Infectious retinitis • Eales' disease is a hypersensitivity reaction characterized by recurrent vitreous hemorrhages and perivenous exudates (retinal vasculitis)
Optic nerve	• Papilledema due to arachnoiditis and noncommunicating hydrocephalus

5. Annotate the ocular manifestations caused by this organism. (5 marks)

Or

Enumerate the manifestations of syphilis in the eye. (5 marks)

- Syphilis has many forms of ocular presentation and thus is known as the great masquerader"

Stages of Syphilis and Ocular Manifestations

Primary	• Chancres in eyelid and conjunctiva (rare)
Secondary	• Interstitial keratitis (with ghost vessels – salmon patch) • Scleritis • Cataract • Acute non granulomatous iritis (may be as a part of Jarisch–Herxheimer reaction) • Chronic granulomatous uveitis • Acute syphilitic placoid posterior chorioretinitis (ASPCC) • Optic neuritis, papilledema
Latent	• None
Tertiary	• Iris roseola (dilated blood vessels in the iris due to microembolism of vessels due to organisms) • Gumma of the uveal tract • Argyll Robertson pupil
Congenital syphilis	• Hutchison's triad: Interstitial keratitis + Hutchison's teeth + sensorineural deafness • Developmental cataract

6. Describe the clinical features of congenital and acquired ocular toxoplasmosis. (5 marks)

- Ocular toxoplasmosis is caused by *Toxoplasma gondii*
- It can be congenital or acquired

Congenital Toxoplasmosis

- Congenital toxoplasmosis is acquired by the fetus when the expectant mother suffers an episode of toxoplasmosis which may not be very apparent clinically
- When the infection happens in the first trimester, the fetus usually does not survive
- Congenital toxoplasmosis is characterized by convulsions, calcifications, and choroiditis
- The choroiditis is typically at the macula owing to the pattern of the ocular blood supply in the fetus. When discovered, the lesions are usually healed, bilateral, and appear as punched-out lesions with pigmentation. The children usually present with nystagmus and decreased central vision

Acquired Toxoplasmosis

- It may be due to *de novo* acquisition of infection due to consumption of undercooked meat or reactivation of old scars where the worms remain as bradyzoites
- They are usually seen in moderately immunosuppressed conditions
- Commonly, a retinitis lesion is seen to begin from the edge of old scars (recurrent infection). The parasites replicate and cause an intense vitreous reaction
- The lesion is yellowish raised plaque and appears as a "headlight in the fog". In extreme immunocompromise, vitritis may be less

7. Enumerate four opportunistic infections that cause posterior uveitis in immuno-compromised individuals. Discuss the clinical features of CMV retinitis. (2+3 marks)

Opportunistic Infections That Cause Posterior Uveitis

1. Toxoplasmosis
2. CMV retinitis
3. Posteriori uveitis caused by tuberculosis
4. Candidal endophthalmitis

CMV Retinitis

- Most frequent opportunistic infection in immunocompromised individuals
- Incidence has decreased after the initiation of HAART
- Most commonly seen when CD4+ counts <50/mm^3

Symptoms

- The patients present with floaters and diminution of vision depending on the amount of retinal involvement
- Many-a-times, they can be asymptomatic when the lesions are in the extreme periphery and not associated with significant vitritis

Signs

- The anterior segment is usually quiet
- A careful fundus examination of both eyes by mydriasis is necessary

Clinical Forms

1. **Fulminant form:** Brush-fire pattern, seen as hemorrhagic vasculitis and necrosis of retina along the major arterial arcade. This is a more fulminant variant and has more impact on visual acuity
2. **Granular form or indolent form:** Seen as granular lesions in the periphery. Sometimes, there is severe perivascular exudation leading to a 'frosted branch' angiitis-like appearance

SHORT ANSWERS

1. Describe the features of Vogt–Koyanagi–Harada syndrome. (3 marks)

- Vogt–Koyanagi–Harada syndrome is an idiopathic multisystem disorder
- Associated with HLA DR4 and DW15
- Ocular + cutaneous + CNS manifestations
- The disease occurs in many phases

Prodromal Phase

- Flu-like symptoms: fever, headache
- Tinnitus, hearing loss

Uveitic Phase

- Bilateral anterior granulomatous uveitis characterized by the formation of iris nodules and posterior synechiae
- Posterior uveitis with multifocal chorioretinitis lesions with bilateral exudative detachment
- Increased choroidal thickness

Chronic Recurrent Phase

- Chronic unteated posterior uveitis
- Depigmentation of the RPE (sunset glow fundus)
- Depigmentation of the limbus (Siguara sign)

Cutaneous Phase

- Alopecia, poliosis and vitiligo.

2. Describe the posterior segment manifestations of tuberculous uveitis. (3 marks)

- Tuberculosis can affect both the anterior and posterior segments
- The posterior segment manifestations of tuberculosis include

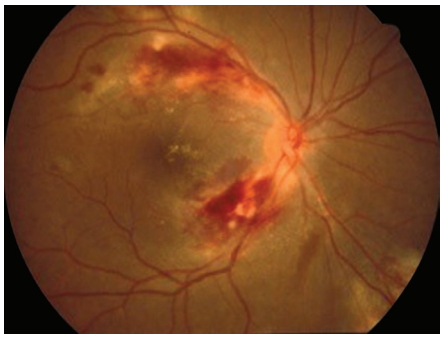
1. **Choroid tubercles**
   - These are usually multiple, subretinal, yellow flat lesions that are dispersed in the post-equatorial region and are associated with meningeal tuberculosis and disseminated tuberculosis
   - They are 1/6th to half the disc diameter size
2. **Solitary granuloma/choroidal granuloma**
   - Occurs as a focal lesion in any part of the retina. It appears yellow and well-demarcated and a size of more than 2 disc diameters
3. **Multifocal choroiditis**
   - Can also be seen in tuberculosis

4. **Serpiginous-like choroiditis**
   - There is centripetally advancing choroiditis that affects the posterior pole mainly
   - The lagging edge shows features of healing in the form of pigmentation
   - If not treated, there is diffuse chorioretinal atrophy

3. Summarize the treatment of CMV retinitis.

Or

A 32-year-old man with HIV presents with blurred vision. His CD4 count is 4/mm³. Fundus shows the following features. (3 marks)



Summarize the Treatment of this Condition

Treatment of CMV Retinitis

- In the pre-HAART era, the only treatment was lifelong antivirals. But the quality of life has improved significantly after the introduction of HAART
- Prompt initiation of HAART is quintessential for treating CMV retinitis
- Antivirals that are recommended are ganciclovir (first line), valganciclovir, foscarnet, and cidofovir; and they are continued till there is a doubling of CD4+ counts or if CD4+ counts cross 100/mm³ and maintained for 6 months

Drug	Induction dose	Maintenance dose
Injection ganciclovir	5 mg/kg twice daily intravenous for 3 weeks	5 mg/kg once daily IV
Oral valganciclovir	900 mg twice daily for 3 weeks	9000 mg once daily
Oral foscarnet	90 mg/kg BD for 14 days	90–120 mg/kg OD

- Other options include intravitreal injections and vitreal implants that release ganciclovir into the vitreous. They are helpful in unilateral cases
- The systemic side effects of these drugs include myelosuppression, renal dysfunction

4. Describe the management of tuberculous uveitis. (3 marks)

Management of Tuberculous Uveitis

Investigations

- In presence of features of tuberculous uveitis and a history suggestive of exposure to a patient with active tuberculosis, the following tests help to support the diagnosis of tuberculous uveitis
  - Positive tuberculin test (>10 mm induration) or positive interferon-gamma release assay
  - Chest X-ray suggestive of active or healed tuberculous lesions
  - Intraocular fluid (aqueous) PCR for tuberculous DNA
- IGRA or interferon-gamma release assay (commonly known as quantiferon) is a quantitative assay for measuring the amount of gamma interform released by the patient's WBC in response to exposure to tubercular proteins and peptides. It cannot differentiate between latent infection and active infection
- Other possible sites of tuberculosis should be investigated by relevant imaging techniques like MRI brain, CT chest, MRI spine, abdominal and pelvic ultrasound

Treatment

Topical

- Steroids (in anterior uveitis)
- Cycloplegics
- Periocular steroids in intermediate and posterior uveitis

Systemic

- ATT for 6 months followed by isoniazid and rifampicin for another 6 months
- Systemic steroids are indicated in cases with severe inflammation

5. Enlist the clinical features of syphilitic uveitis. (3 marks)

Treponemes can affect all the coats of the eyeball.

Anterior Uveitis

- Characterized by the formation of iris roseola (dilated vessels) caused by treponemes-causing emboli of the microvasculature of the iris
- Acute nongranulomatous iritis (may be as a part of Jarisch–Herxheimer reaction)
- Chronic granulomatous uveitis is characterized by mutton fat KPs and iris nodules

Intermediate uveitis

Seen as dense vitritis.

Posterior uveitis

- Deep chorioretinitis may be focal/solitary or multifocal or diffuse. It is seen as a yellow gray subretinal lesion with surrounding shallow retinal detachment and vitrits
- Acute syphilitic placoid posterior chorioretinitis (ASPPC) with typical leopard skin pigmentation of the RPE seen during fluorescein angiography
- Necrotizing retinitis (mimics acute retinal necrosis)

Panuveitis

Inflammation of all parts of uvea

COMPETENCY

OP6.4 DESCRIBE AND DISTINGUISH HYPHEMA AND HYPOPYON

SHORT ESSAYS

1. Define hyphema. Enumerate four causes for the same. (1+4 marks)

Definition of Hyphema

Blood in the anterior chamber

Causes

1. Blunt ocular trauma
2. Hemophilia
3. UGH syndrome due to ACIOL implantation
4. Fuch's heterochromic iritis

2. Explain the clinical features of hyphema. (5 marks)

Clinical Features of Hyphema

Symptoms

- Usually hyphema follows blunt trauma

- Blurred vision
- Ocular pain
- Photophobia

Signs

- There is blood in the anterior chamber and depending on its severity, it is graded as follows
  1. Microscopic hyphema: The absence of gross collection of blood, RBCs are seen floating in the aqueous
  2. Grade 1: <1/3 of the anterior chamber filled with blood
  3. Grade 2: 1/3–1/2 of the anterior chamber filled with blood
  4. Grade 3: 1/2 to a near-total anterior chamber filled with blood
  5. Grade 4: total hyphaema or 8-ball hyphema
- IOP is usually raised
- Other features of trauma like mydriasis, subluxation of the lens, sphincter tears, and iridodialysis may be seen

SHORT ANSWERS

1. Explain the management of hyphema. (3 marks)

- The patient is advised bed rest in a propped-up position so that the cells settle down and vision clears
- Topical steroids and cycloplegics are given to decrease intraocular inflammation
- Antiglaucoma medications are given to decrease intraocular pressure as hyphema is usually associated with raised IOP. Drugs like beta-blockers, alpha agonists, carbonic anhydrase inhibitors are useful. Cholinergic and prostaglandins should not be used
- Antifibrinolytics like e-amino caproic acid and tranexamic acid can be given to prevent rebleed
- Surgical treatment is required only if IOP is very high or the hyphema does not dissolve even after 7 days. The clot is evacuated by paracentesis

2. Enumerate three causes of hypopyon. (3 marks)

1. Corneal ulcer
2. Iridocyclitis
3. Endophthalmitis

3. Define a pseudohypopyon. Differentiate it from a true hypopyon. (3 marks)

Pseudohypopyon

A noninflammatory collection of cells in the anterior chamber that resembles a hypopyon.

Differentiation from True Hypopyon

- The most common cause of a pseudohypopyon is intraocular malignancies like retinoblastoma, intraocular lymphoma, and leukemias. It may also follow an intravitreal injection of triamcinolone
- It is more convex in configuration cf hypopyon. It may not be associated with corcumcorneal congestion, anterior chamber reaction, and flare
- Also, it persists despite the use of topical steroids and does not regress

4. Differentiate between hypopyon seen in bacterial and fungal keratitis. (3 marks)

- Hypopyon is a feature of both bacterial and fungal keratitis
- Hypopyon occurs more commonly in fungal keratitis than bacterial keratitis
- However, in fungal keratitis, the hypopyon is not sterile as the fungal hyphae penetrate the cornea
- Also, its size is out of proportion to the size of the ulcer in fungal keratitis and is relatively immobile

COMPETENCY

OP6.5 DESCRIBE AND DISCUSS THE ANGLE OF THE ANTERIOR CHAMBER AND ITS CLINICAL CORRELATES

LONG ESSAYS

1. A. Define the angle of the anterior chamber. (1 mark)
B. Explain the measurement of the angle of the anterior chamber. (3 marks)
C. Describe the formation of aqueous. (3 marks)
D. Describe the outflow of aqueous. (3 marks)

A. Angle of the Anterior Chamber

- The space between the peripheral part of the cornea and the base of the iris
- It is the site of aqueous drainage

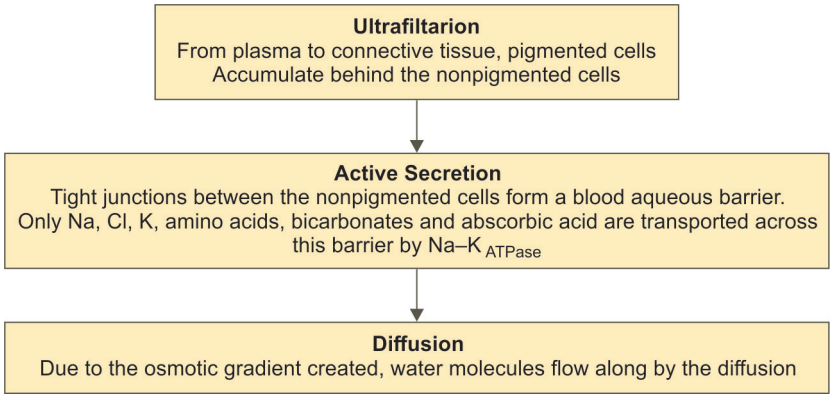
B. Measurement of the Angle of the Anterior Chamber

- The depth of the anterior chamber can be measured by gonioscopy
- It is measured based on the structures seen on gonioscopy

Grade 0	No angle structures visible	Closed	0°
Grade I	Schwalbe's line	High chance of angle-closure-very narrow-angle	10°
Grade II	Schwalbe's line, trabecular meshwork	Angle-closure possible-narrow-angle	20°
Grade III	Schwalbe's line, trabecular meshwork, scleral spur	Open-angle-no chance of angle-closure	20–35°
Grade IV	Schwalbe's line, trabecular meshwork, scleral spur, ciliary body band	Wide-open angle-no chance of angle-closure	35–45°

C. Formation of Aqueous

- It is secreted from the pars plicata of the ciliary body



- The following mechanisms are involved in aqueous production. Each villus has a vascular core with a fibrous core
- Aqueous is formed by diffusion, ultrafiltration, and active secretion
- The rate of formation is 2.3 µl/min

D. Outflow of Aqueous

- The aqueous flow out into the venous system of the eye through the angle of the anterior chamber
- The angle is formed at the intersection of the cornea and the root of the iris
- The aqueous is drained by two methods (Fig. 6.5.1)

1. **Conventional pathway**
   - Trabecular meshwork → Schlemm's canal → Collector channels → Aqueous veins → Episcleral veins (90% drainage)
   - Trabecular meshwork (Fig. 6.5.2)
   - It is a sieve-like structure situated at the angle of the anterior chamber
   - It consists of three parts:
     i. Uveal meshwork
        - Anterior-most with a diameter of 25–75 µm
        - Extends from the root of the iris to Schlemm's canal
        - Consists of large pores
     ii. Corneoscleral meshwork
        - Extends from the scleral spur to Schwalbe's line
        - Consists of pores a little smaller (5–50 µm)
     iii. Juxtacanalicular meshwork
        - A continuation of the corneal endothelium
        - Offers the highest amount of resistance

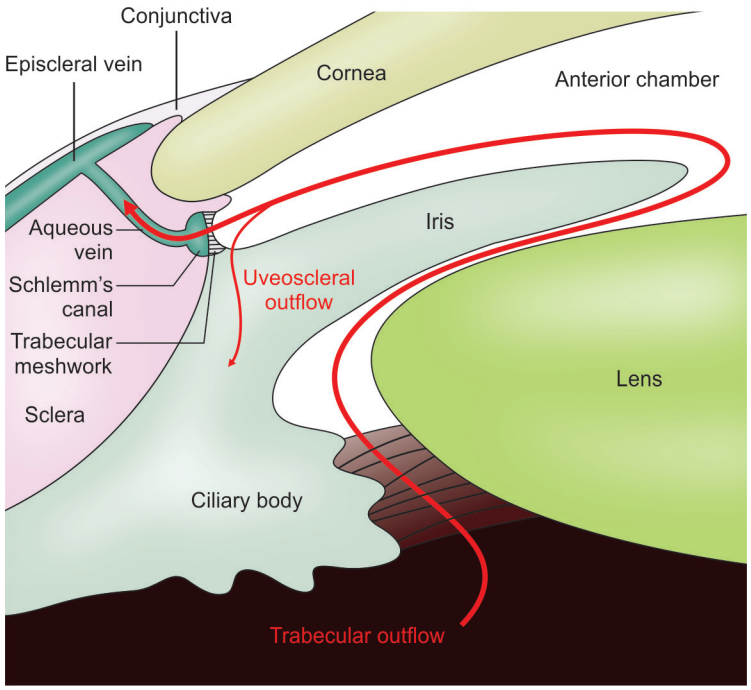


Fig. 6.5.1: Outflow of aqueous by conventional and uveoscleral pathway

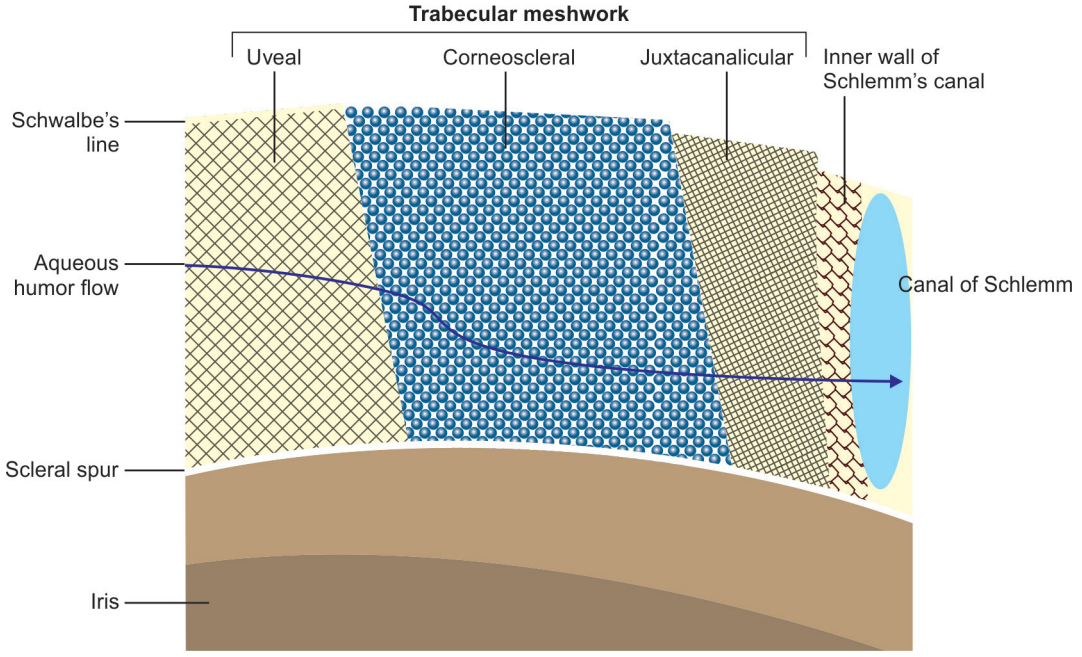
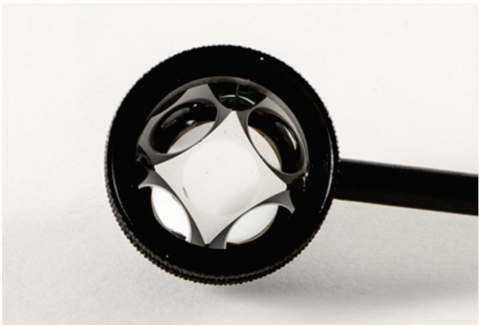


Fig. 6.5.2: Structure of trabecular meshwork

- Schlemm's canal
  - It is a circular canal that is present underneath the blue zone of the limbus circumferentially
  - On the cross-section, it is oval and is lined by endothelium
  - The aqueous is thought to enter the canal by vacuolation into the endothelial cells
- Collector channels
  - About 25–35 in number
  - Larger vessels run a short intrascleral course and empty into episcleral veins (direct system)
  - Smaller collector channels form an intrascleral plexus and drain into the episcleral veins (indirect system)

2. **Uveosceleral Pathway**
   - Anterior chamber → Suprachoroidal space → Veins of choroid and sclera (10% drainage)

2. A. Identify the instrument and state its use. (2 marks)
B. Explain Shaffer's grading with suitable drawings. (5 marks)
C. Describe three abnormalities that can be detected during this procedure. (3 marks)



A. The Instrument Shown is

- Gonio mirror
- It is used to measure the angle of the anterior chamber

B. Shaffer's Grading (Fig. 6.5.3)

Grade 0	No angle structures visible	Closed	0°
Grade I	Schwalbe's line	High chance of angle-closure- very narrow-angle	10°
Grade II	Schwalbe's line, trabecular meshwork	Angle-closure possible- narrow-angle	20°
Grade III	Schwalbe's line, trabecular meshwork, scleral spur	Open-angle-no chance of angle-closure	20–35°
Grade IV	Schwalbe's line, trabecular meshwork, scleral spur, ciliary body band	Wide-open angle-no chance of angle-closure	35° –45°

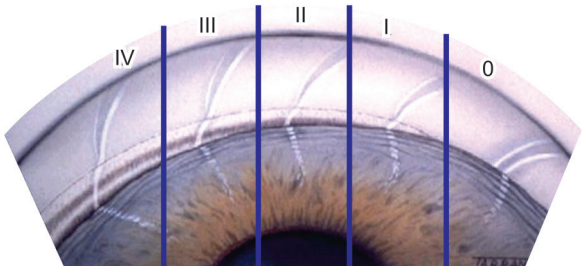


Fig 6.5.3: Grading of the angle

C. Abnormalities Detected on Gonioscopy vis-a-vis Causes/Associations

Abnormalities on Gonioscopy	Causes
• Peripheral anterior synechiae: ▪ Adhesions between iris and cornea seen as broad patchy adhesions bridging the angle between iris and Schwalbe's line	• Primary angle-closure glaucoma • Iridocyclitis • Iridocorneal endothelial syndrome
• Increased pigmentation along Schwalbe's line	• Pigmentary glaucoma • Pseudoexfoliation syndrome • Aphakic glaucoma • Blunt trauma • Anterior uveitis • Iris melanoma • Following Nd YAG iridotomy • Nevus of Ota
• Widened ciliary body band	• Angle recession glaucoma
• Neovascularization in the angle	• Neovascular glaucoma • Fuch's heterochromic iridocyclitis
• Blood in Schlemm's canal	• Increased episcleral venous pressure • Trauma • Caroticocavernous fistula

3. A. Identify the instrument and procedure. (2 marks)

B. Define intraocular pressure and give the normal range. (2 marks)

C. Describe diurnal variation of intraocular pressure and its implications. (3 marks)

D. Enumerate the factors that influence IOP dynamics. (3 marks)

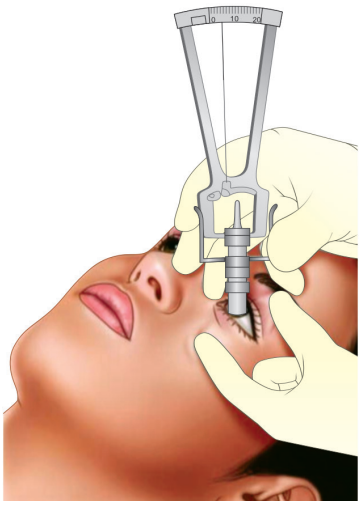
A. Identify the Instrument

The Instrument Shown Is

Schiotz tonometer

Procedure is Called

Tonometry (the measurement of intraocular pressure)



B. Intraocular Pressure

Definition

The pressure exerted by the contents of the eye on the coats of the eyeball

Normal Range

10.5–20.5 mm Hg

C. Diurnal Variation of IOP

- IOP fluctuates cyclically throughout the day (Fig. 6.5.4)
- The intraocular pressure is highest in the early morning and lowest in the evening. It follows a biphasic curve
- The normal difference between the highest and lowest values should be always less than 4 mm Hg
- If the difference is more than 4 mm Hg, it is suspicious of glaucoma and >8 mm Hg is pathognomonic of POAG

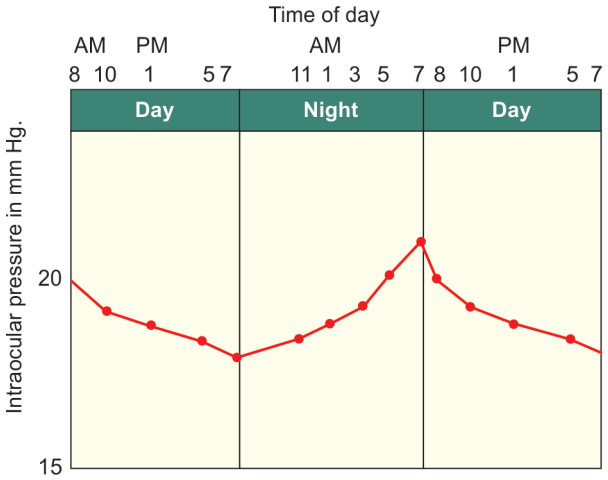


Fig. 6.5.4: Diurnal variation of IOP

Implications

1. Charting of IOP every 2–4 hours for 24 hours is needed to know the diurnal variation of IOP. It is known as phasing
2. Phasing is an important requirement for the diagnosis of POAG when the initial IOP may not be higher than 21 mm Hg
3. Phasing helps to know the highest pressure, so the target pressure can be set accordingly

D. Factors Affecting IOP Dynamics

1. **Age**
   - IOP increases with age, probably due to decreased drainage
2. **Heredity**
   - Many genes have been linked to causing different types of glaucoma
   - They interfere with the drainage of the aqueous
3. **Sleep**
   - IOP decreases during sleep

4. **Diurnal Variation**
   - IOP tends to peak early in the morning
   - This is the basis for phasing of IOP (every 4 hours) in a glaucoma suspect patient to find the peak
   - Normal eyes have a smaller fluctuation (<5 mm of Hg) than glaucomatous eyes (> 8 mm of Hg)
   - A biphasic curve, with two peaks, can be seen in primary open-angle glaucoma patients
5. **Postural variations**
   - IOP increases when changing from the sitting to the supine position
6. **Blood pressure**
   - It is not directly proportional to IOP, but some hypertensives who consume antihypertensive medications at night are found to develop normal-tension glaucoma
7. **Osmotic pressure of blood**
   - Increase in plasma osmolarity (like intravenous mannitol, oral glycerol, or in patients with uremia) can lead to a fall in IOP; whereas a reduction in plasma osmolality (as occurs with water drinking provocative tests) is associated with a rise in IOP
8. **General anesthetics and other drugs**
   - Ketamine is known to increase IOP

SHORT ESSAYS

1. Explain the structure of the angle of the anterior chamber. (5 marks)

- The angle of the anterior chamber is the space between the peripheral cornea and the root of the iris
- It is the site of aqueous drainage
- It consists of the following structures from posterior to anterior

Structure	Description
Root of the iris	• It is the posterior-most structure seen on gonioscopy • It is formed by the base of the iris and appears dark brown
Ciliary body band	• It is formed by the anterior-most part of the ciliary body • Its width depends on the iris insertion • It is wider in myopes and narrower in hypermetropes
Scleral spur	• It appears as a prominent white line • The longitudinal muscle of the ciliary muscle is attached to the posterior aspect of the scleral spur • Anteriorly, it is attached to the trabecular meshwork
Trabecular meshwork	• It is seen as a greyish-black band of varied pigmentation • It has three microscopic parts: Uveoscleral, corneoscleral, and juxtacanalicular • It is made of pores of decreasing sizes that act as a filter and help in the conventional aqueous outflow
Schwalbe's line	• It is a fine glistening ridge seen as the anterior-most structure of the angle of the anterior chamber • It is the prominent end of the Descemet's membrane of the cornea

2. Describe the structure and functions of the blood-aqueous barrier. (5 marks)

Blood-aqueous Barrier

Structure

- A blood-aqueous barrier is formed by the tight junctions between the cells of the inner nonpigmented epithelium of the ciliary body
- The tight junctions are zona occludens and zona adherens (Fig. 6.5.5)
- It is also formed by the nonfenestrated endothelium of the iris capillaries
- These are not joined by tight junctions and so, they lead to formations of cells and flare during inflammation

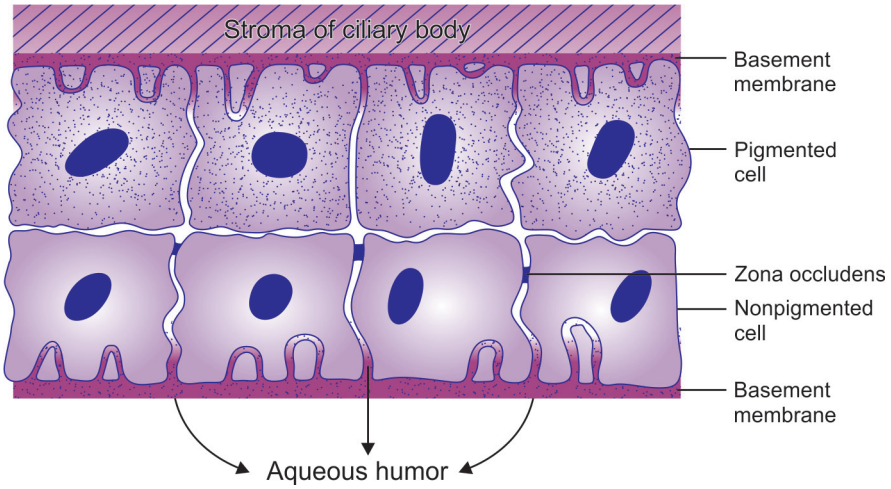


Fig. 6.5.5: Structure of blood-aqueous barrier

Functions

1. The blood-aqueous barrier prevents the entry of molecules into the aqueous and maintains its transparency and osmolarity
2. It is responsible for maintaining the difference in the chemical composition of the aqueous and plasma. An increase in contents of the aqueous (as in uveitis) causes the aqueous to become 'plasmoid' and thus makes the aqueous turbid
3. Low molecular weight molecules like chloramphenicol, cephalothin can penetrate the blood-aqueous barrier. Lipid soluble molecules pass easily. Thus, it helps in providing medications into the aqueous

SHORT ANSWERS

1. Explain the functions of aqueous humor. (3 marks)

1. Aqueous humor helps to maintain the IOP even though it occupies a smaller volume compared to the vitreous. This is because of its dynamic nature. IOP helps in maintaining the structure of the eyeball and helps in axoplasmic transport across the optic nerve head
2. It plays role in nourishing endothelium and lens
3. Helps to provide substrates and removing metabolic end products
4. Aqueous also provides an optically clear medium for vision
5. It has high ascorbate levels that prevent UV-induced oxidative damage
6. Facilitates cellular and humoral immunity responses

2. Contrast trabecular and uveoscleral outflow of aqueous. (3 marks)

Trabecular and Uveoscleral Outflow of Aqueous

Feature	Trabecular/conventional pathway	Uveoscleral pathway
Percentage of aqueous drainage	90%	10%
Pathway	Trabecular meshwork → Schlemm's canal → collector channels → aqueous veins → episcleral veins	anterior chamber → suprachoroidal space → veins of choroid and sclera
Factors affecting outflow	Dependent on IOP	Independent of IOP (not below 4 mm Hg) Age-dependent decrease in outflow Cyclodialysis causes an increased outflow
Process of aqueous outflow	Contraction of the ciliary body causes a pull on the scleral spur that pulls the TM and increases the size of the pores.	Relaxation of ciliary muscle causes an increase in outflow
Effect of antiglaucoma drugs that act	Beta-blockers Cholinergic Carbonic anhydrase inhibitors	Prostaglandins Alpha agonists Cholinergic drugs decrease aqueous outflow

COMPETENCY

OP6.6 IDENTIFY AND DEMONSTRATE THE CLINICAL FEATURES AND DISTINGUISH AND DIAGNOSE COMMON CLINICAL CONDITIONS AFFECTING THE ANTERIOR CHAMBER

SHORT ANSWER

1. Demonstrate the understanding of the procedure of gonioscopy and structures seen during this procedure. (3 marks)

Procedure of Gonioscopy

- The patient is seated at the slit-lamp. Topical anesthesia is instilled in both eyes
- A drop of methylcellulose is placed in the concavity of the lens
- The beam height is decreased to 3 mm so that it does not cross the pupil
- The gonio-lens is placed on the cornea and the slit beam is focused on the inferior mirror. This shows the angle structures of the superior angle
- This is followed by an examination of each quadrant of the angle

Structures Seen in Gonioscopy

Root of the iris	• It is the posterior-most structure seen on the gonioscopy • It is formed by the base of the iris and appears dark brown
Ciliary body band	• It is formed by the anterior-most part of the ciliary body • Its width depends on the iris insertion • It is wider in myopes and narrower in hypermetropes
Scleral spur	• It appears as a prominent white line • The longitudinal muscle of the ciliary muscle is attached to the posterior aspect of the scleral spur • Anteriorly, it is attached to the trabecular meshwork

Contd.

Trabecular meshwork	• It is seen as a grayish-black band of varied pigmentation • It has three microscopic parts: Uveoscleral, corneoscleral, and juxtacanalicular • It is made of pores of decreasing sizes that act as a filter and help in the conventional aqueous outflow
Schwalbe's line	• It is a fine glistening ridge seen as the anterior-most structure of the angle of the anterior chamber • It is the prominent end of the descemet membrane of the cornea

COMPETENCY

OP6.7 ENUMERATE AND DISCUSS THE AETIOLOGY, THE CLINICAL DISTINGUISHING FEATURES OF VARIOUS GLAUCOMAS ASSOCIATED WITH SHALLOW AND DEEP ANTERIOR CHAMBER. CHOOSE APPROPRIATE INVESTIGATIONS AND TREATMENT FOR PATIENTS WITH ABOVE CONDITIONS

LONG ESSAY

1. A 45-year-old man came for a regular eye check-up. His visual acuity was 6/6, N6 in both eyes after correcting presbyopia. The intraocular pressure was 30 mm Hg and 28 mm Hg in both eyes, and the discs showed an increase in the size of cupping.

A. What is the most probable diagnosis? (1 mark)
B. How is it confirmed? (1 mark)
C. Explain the disc changes associated with this condition. (4 marks)
D. Explain any 4 visual field changes associated with this condition. (4 marks)
E. Enumerate the classes of drugs useful in treating this condition with one example for each. (3 marks)
F. Describe ocular side effects of latanoprost. (2 marks)

A. Most Probable Diagnosis

Primary open-angle glaucoma

B. Diagnosis is Confirmed by

An automated visual field analysis

C. Disc Changes Associated with Glaucoma

Early Glaucomatous Changes

1. **Notching of neuroretinal rim (Fig. 6.7.1)**
   - Vertically oval cup due to selective loss of neural rim tissue in the inferior and superior poles. (ISNT rule → order of loss of neuroretinal rim: inferior > superior > nasal > temporal
2. **Asymmetry of the cups (Fig. 6.7.2)**
   - Adifference of more than 0.2 between two eyes is significant
   - Large cup i.e., 0.6 or more
   - Even if symmetrical, a cup more than 0.6 is suspicious

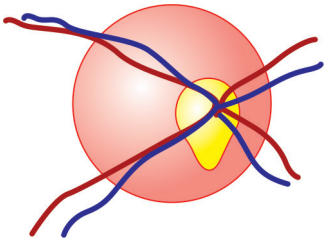


Fig. 6.7.1: Notching of neuroretinal rim

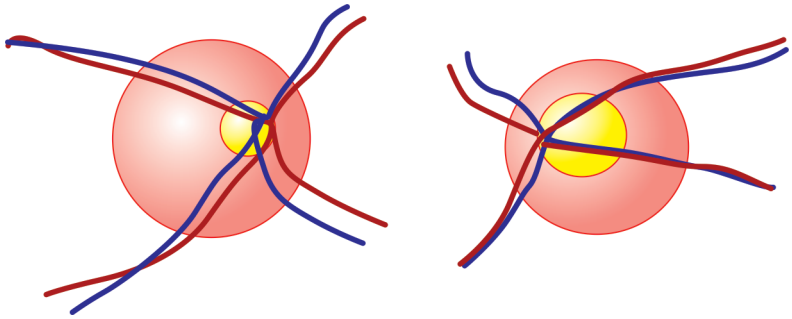


Fig. 6.7.2: Asymmetric cup

3. **Baring of circumlinear vessel (Fig. 6.7.3)**
   - A circumlinear vessel traverses along the rim of the cup
   - When the cup recedes, the vessels seem 'bared' or left afloat
4. **Splinter hemorrhages (Drance hemorrhages) (Fig. 6.7.4)**
   - Present on or near the optic disc margin, usually represent a sudden increase in glaucomatous damage
5. **Thinning of retinal nerve fiber layer (RNFL loss)**
   - May be seen as fan-shaped areas of thinning starting from the disc, especially in the inferonasal part
   - It is appreciated better with red-free light

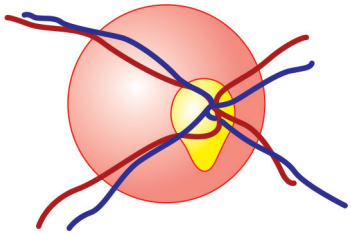


Fig. 6.7.3: Baring of circumlinear vessel

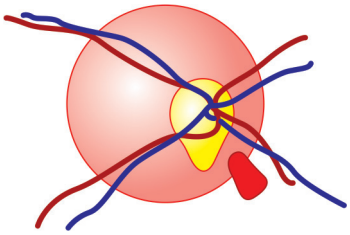


Fig. 6.7.4: Disc hemorrhage

Advanced Glaucomatous Changes

1. **Marked cupping**
   - (Cup size 0.7 to 0.9), excavation may even reach the disc margin (total loss of neuroretinal rim), the sides are steep (Fig. 6.7.5)
2. **Laminar dot sign (Fig. 6.7.6)**
   - Due to extreme thinning of the nerve layer, underlying lamina cribrosa is visible on ophthalmoscopy
   - Nasalization of vessels: The cup is usually a bit nasal. When there is progressive cupping, the cup extends temporally whereas the vessels remain. Hence, it appears like vessels are shifted nasally
3. **Bayoneting sign (Fig. 6.7.7)**
   - Double angling of vessels as they pass over the deep cup.

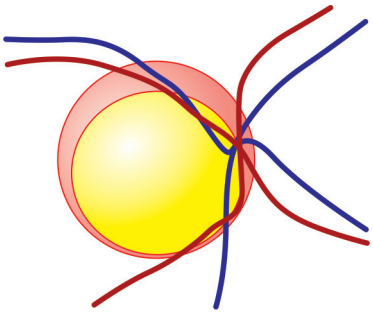


Fig. 6.7.5: Advanced cupping

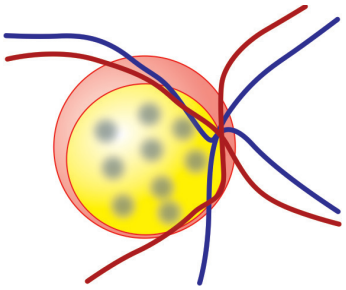


Fig. 6.7.6: Laminar dot sign

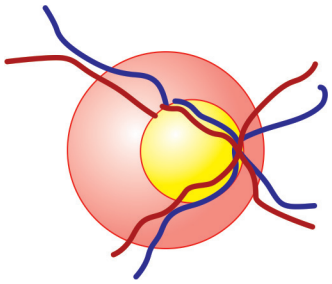
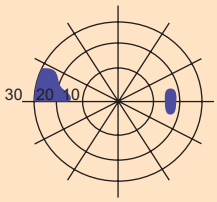
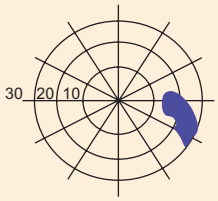
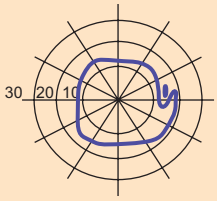


Fig. 6.7.7: Bayonetting sign

4. **Peripapillary atrophy**
   - Due to increased pressure, there is ischemia due to compression of short posterior choroidal vessels, which supply the peripapillary choroid
5. **Glaucomatous optic atrophy**
   - It is seen as a part of end-stage glaucoma, characterized by pale disc, advanced cupping, and other signs of advanced glaucoma

D. Visual Field Changes Associated with Glaucoma

Name of field defect	Description	Example
Nasal step (Fig. 6.7.8)	Can occur in the periphery (shown in picture), mid periphery (tends to perpendicular), or near the fixation (obtuse angle with raphe)	 **Fig. 6.7.8** Bayonetting sign
Temporal wedge (Fig 6.7.9)	Can be supero- or inferonasal; extension from the temporal edge of blind spot	 **Fig. 6.7.9** Temporal wedge
Baring of blind spot (Fig. 6.7.10)	It occurs as a result of an increased sensitivity of the retina below the blind spot compared to the same area above the blind spot	 **Fig. 6.7.10** Baring of blind spot

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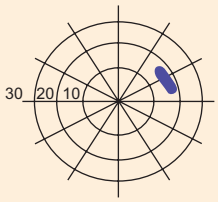
Name of field defect	Description	Example
Paracentral scotoma (Fig. 6.7.11)	Usually seen as a relative steep scotoma occurring in the superonasal quadrant	 **Fig. 6.7.11** Paracentral scotoma

E. Drugs Used in Open-angle Glaucoma

1. **Beta blockers:** Timolol
2. **Prostaglandin analogs:** Latanoprost
3. **Alpha agonists:** Brimonidine
4. **Carbonic anhydrase inhibitors:** Dorzolamide
5. **Cholinergic drugs:** Pilocarpine

F. Ocular Side Effects of Latanoprost

1. Conjunctival hyperemia, foreign body sensation, eye irritation and superficial punctuate keratopathy-temporary side effects. It is a quite common side effect seen with Latanoprost
2. Itching, burning sensation, and eye pain
3. Reduced corneal sensation, thickness
4. Periocular hyperpigmentation has been noted in most patients on long-standing treatment, which tends to disappear on discontinuation of the drug
5. Increase in length and number of lashes, long enough that can rub against the spectacles
6. Activation of latent Herpes and uveitis is also a complication
7. Cystoid macular edema along with uveitis which responds to NSAIDs
8. Darkening of iris
9. Choroidal effusion

SHORT ESSAYS

1. A 50-year-old woman presents with acute onset redness, pain, and diminution of vision in the right eye. She gave the history of the appearance of colored halos on and off for the last six months. Her anterior segment shows the following image.

A. What is the most probable diagnosis? (1 mark)

B. What is the probable finding if gonioscopy is performed in this stage? (1 mark)

C. Explain the management of this condition. (3 marks)

A. Most Probable Diagnosis

Right eye acute angle-closure glaucoma or congestive attack of angle-closure glaucoma.

B. If Gonioscopy is Performed

The angle is closed or grade 0. No structures are visible on the gonioscopy.

C. Management

1. **Immediate management**
   - Make the patient lie down supine (So that lens shift posteriorly)
   - Intravenous or oral acetazolamide 500 mg (To decrease aqueous production)
   - Topical steroid (dexamethasone or prednisolone) QID (To decrease the inflammation)
   - Topical timolol 0.5%
   - Analgesics and anti emetics
   - Draw blood for baseline sugars, electrolytes, and renal function tests
2. **After one hour**
   - Topical pilocarpine 2%, in both eyes, QID (It acts by straightening the iris and decreasing the contact between the anterior surface of the lens and the posterior surface of the iris.)
   - After 30 more minutes
   - Recheck IOP—if not fallen <35 mm Hg, oral or intravenous hyperosmotic drugs, IV 20% mannitol 1g/kg over 45–60 min (1 drop per second) or oral 50% glycerol 1g/kg
3. **Definitive management**
   - Perform gonioscopy to understand the synechiae status of the eye
   - Laser peripheral iridotomy in the affected eye, when IOP has dropped and is around 20–25 mm Hg. (The principle is to create a bypass for the flow of aqueous in the case of pupillary block)
   - Laser peripheral iridotomy in the fellow eye

2. Define glaucoma. Classify glaucoma based on gonioscopy. (2+3 marks)

Definition of Glaucoma

- A chronic, progressive optic neuropathy associated with damage of the optic nerve head associated with characteristic or typical disc and visual field changes, usually associated with an increase in IOP
- Based on gonioscopy, glaucoma can be broadly classified as open-angle and closed-angle glaucoma
- An angle is said to be open if trabecular meshwork is visualized during gonioscopy

Classiifcation of Glaucoma Based on Gonioscopy

I. Open-angle Glaucoma

1. **Primary open-angle glaucoma**
   i. Normal-tension glaucoma
   ii. Ocular hypertension
2. **Secondary open-angle glaucoma**
   i. Pseudo-exfoliation glaucoma
   ii. Pigmentary glaucoma
   iii. Aphakic glaucoma
   iv. Steroid-induced glaucoma
   v. Uveitic glaucoma

II. Angle-closure Glucoma

1. **Primary angle-closure glaucoma**
   - i. Primary angle-closure suspect
   - ii. Primary angle-closure
   - iii. Primary angle-closure glaucoma
2. **Secondary angle-closure glaucoma**
   - i. Neovascular glaucoma
   - ii. Lens induced glaucoma
   - iii. Uveitic glaucoma

3. Define the following terms (1 mark each)

A. Primary open-angle glaucoma
B. Ocular hypertension
C. Normal-tension glaucoma
D. Primary angle-closure glaucoma
E. Juvenile glaucoma

A. Primary Open-angle Glaucoma

When the intraocular pressure is more than 21 mm Hg, associated with glaucomatous disc changes and visual field changes, in the presence of an open angle and without any secondary ocular cause.

B. Ocular Hypertension

It is defined when the intraocular pressure is more than 21 mm Hg with no optic disc changes or visual field changes in the presence of an open-angle.

C. Normal-tension Glaucoma

It is defined when the intraocular pressure never exceeds 21 mm Hg, but the eyes show glaucomatous optic disc and visual field changes in the presence of an open-angle.

D. Primary Angle-closure Glaucoma

It is defined when the intraocular pressure is more than 21 mm Hg, associated with glaucomatous disc changes and visual field changes, in the presence of a narrow or closed-angle and without any secondary ocular cause.

E. Juvenile Glaucoma

Presence of raised intraocular pressure with characteristic disc and visual field changes in presence of an open angle and no other ocular associations seen in the age group of 10–35 years.

4. Define the visual field. Explain the normal visual field with the neat and labeled diagram and its values in four quadrants. (1+4 marks)

Definition of Visual Field

It is the area of space that is seen by an eye when fixated onto a point within that space.

Normal Visual Filed

- Normally, it is widest temporally (90–100°), followed by inferiorly (70–80°), superiorly (60°), and least nasally (55–60°) (Fig. 6.7.12).

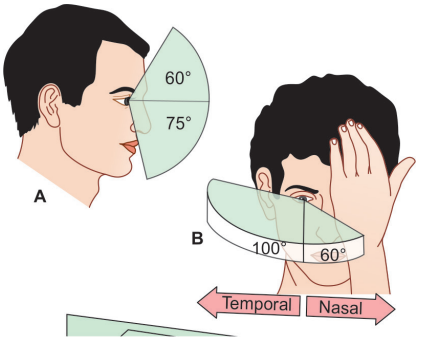


Fig. 6.7.12: Normal visual field

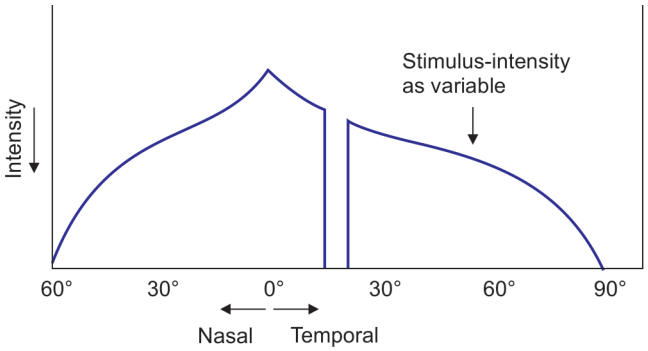
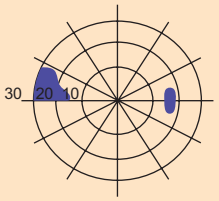
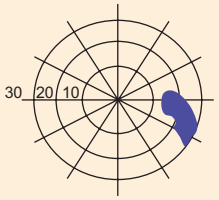
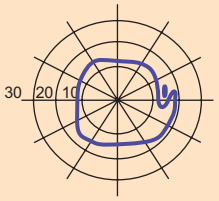


Fig. 6.7.13: Hill of vision

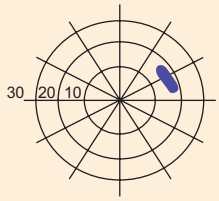
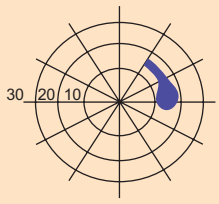
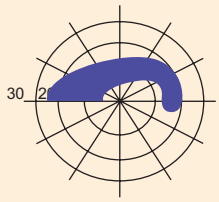
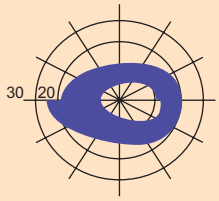
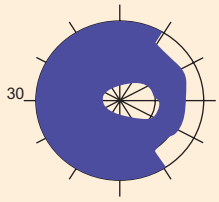
- The sensitivity of the retina peaks at the macula with a ''bottomless trough'' at the physiological blind spot (entrance of optic nerve) (Fig 6.7.13)
- The blind spot lies at the nasal side in the retina and hence is perceived temporal to fixation at approximately 12–15°
- The automated perimeter measures the sensitivity at all points on the retina in various quadrants by presenting to the patient, stimuli of varying size and brightness

5. Describe the visual field changes in glaucoma (5 marks)

Visual Field Changes Associated with Glaucoma

Name of field defect	Description	Example
Nasal step (Fig. 6.7.14)	Can occur in the periphery (shown in picture), mid periphery (tends to perpendicular), or near the fixation (obtuse angle with raphe)	 Fig. 6.7.14: Bayonetting sign
Temporal wedge (Fig. 6.7.15)	Can be supero- or inferonasal; extension from the temporal edge of blind spot	 Fig. 6.7.15: Temporal wedge
Baring of blind spot (Fig. 6.7.16)	It occurs as a result of an increased sensitivity of the retina below the blind spot compared to the same area above the blind spot	 Fig. 6.7.16 Baring of blind spot

Contd.

Name of field defect	Description	Example
Paracentral scotoma (Fig. 6.7.17)	Usually seen as a relative steep scotoma occurring in the superonasal quadrant	 **Fig. 6.7.17** Paracentral scotoma
Seidel's scotoma (Fig. 6.7.18)	Paracentral scotoma extends into the blind spot to form a Seidel scotoma	 **Fig. 6.7.18:** Seidel scotoma
Arcuate scotoma (Fig. 6.7.19)	Seidel scotoma extends into the temporal half to reach the horizontal raphe to form an arc Can be superior or inferior	 **Fig. 6.7.19:** Arcuate scotoma
Ring scotoma or double arcuate scotoma (Fig. 6.7.20)	When an inferior and a superior arcuate join at the horizontal raphe, a discrepancy is seen as a sharp perpendicular step. It is called Roenne's nasal step	 **Fig. 6.7.20:** Ring scotoma
Tunnel vision (Fig. 6.7.21)	A central tubular vision is remaining along with a peripheral temporal crescent.	 **Fig. 6.7.21:** Tubular vision
Isopteric contraction	Decrease in the central or peripheral viewing area	

6. Define perimetry. Describe the procedure of Static perimetry and its use in glaucoma. (1+2+2 marks)

Definition of Perimetry

The process by visual field is charted is called perimetry.

Procedure of Static Perimetry

- In static perimetry, stimuli of varying brightness are presented at various points in the visual field, and the patient is asked to press a switch upon seeing the stimulus
- Here, sensitivity is determined at each point on the retina and is measured in terms of apostilbs (abs)
- It is done using an automated perimeter called Humphrey's perimeter
- It has a dome-shaped bowl with central fixation and it has computerized software that calculates the sensitivity of the retina at each point
- The patient is seated at the mouth of the bowl with one eye patched and chin placed at the chin rest
- Appropriate refractive correction is placed
- The patient is given a button that is connected to the system
- The patient is instructed to click the button upon seeing a spot of light
- The computer offers many points in the visual field that is to be tested (entered into the computer by the operator). The field usually chosen for glaucoma is 30–2 or 24–2
- Depending on the strength of the stimulus presented at different positions in the bowl and the patient's response. A printout of the test result is provided after the test

Use in Glaucoma

1. The foremost use of perimetry in glaucoma is to establish a diagnosis of glaucoma
   - The common visual field changes seen in glaucoma are baring of the blind spot, siedel scotoma, Arcuate scotoma, and annular scotoma
2. Diagnosis of normal-tension glaucoma is made when there is a worsening of visual field defect
   - In presence of normal intraocular pressure and a stationary field defect, a diagnosis of normal-tension glaucoma cannot be made
3. The next use in understanding the progression of glaucoma and the efficacy of the treatment provided
   - The Humphrey perimeter house software helps to understand the progression of glaucoma based on three consecutive field tests
   - It can also be done manually by comparing the mean deviation, pattern standard deviation, and understanding the greyscale

7. Describe the etiology, clinical features, and management of primary congenital glaucoma. (1+2+2 marks)

Primary Congenital Glaucoma

A distinct entity, characterized by anatomical disturbances in the drainage of aqueous humor due to goniodysgenesis

Etiology

- It is sporadic in occurrence with a very high male preponderance
- It can be hereditary with polygenic inheritance

- The embryological basis for PCG is the cessation of the iris root and ciliary body recede posteriorly during the development of the angle
- As a result, the iris is inserted anteriorly into the angle
- The three major abnormalities seen are:
  1. Isolated trabeculodysgenesis
  2. Iridodysgenesis
  3. Corneodysgenesis

Clinical Features

- The presentation of childhood glaucomas is unique. Parents bring the child with excess tearing, child burying its head into pillow and blepharospasm. It is due to irritation of corneal nerves due to corneal edema
- The cornea appears hazy due to edema and is enlarged (buphthalmos—bull eye)
- Normally, the diameter of the cornea is 10.5 mm; more than 13 mm is considered pathognomonic
- Haab's striae: Tears in Descemet's membrane are seen as parallel peripheral concentric tears
- The sclera is thinned due to increased pressure and appears blue
- The anterior chamber is deep
- Iris appears thinned and the lens is stretched
- The optic disc shows cupping or atrophy due to increased pressure
- Axial myopia is seen

Management

- Definitive treatment is always surgical
- A short period of medical therapy with topical beta-blockers or carbonic anhydrase can be given to control IOP in case the child is unfit for surgery or before surgery
- The procedures that can be performed are:

1. **Goniotomy**
   - In this procedure, a Barkan's knife is introduced into the anterior chamber under gonioscopic control and advanced to the opposite angle and an incision is made in the midway of the angle and the knife is rotated for 75°
   - A similar procedure can be done on the other side at the next sitting
2. **Trabeculotomy**
   - It is the next step if goniotomy fails
   - It is also indicated when cloudy or scarred cornea permits the view of angle by gonioscopy
   - A small vertical incision is made on the sclera at the level of the Schlemm's canal at 12'o clock position
   - One prong of the trabeculotome is passed into the Schlemm's canal circumferentially for 90°
   - The upper prong acts as a guide
3. **Trabeculectomy**
   - It is a controlled aqueous filtration procedure in which a window is made in the trabecular meshwork and aqueous is made to filter out of the anterior chamber into the subconjunctival space
4. **Glaucoma drainage devices**
   - Synthetic devices are placed surgically in the subconjunctival space
   - It is indicated if the above procedures fail

5. **Cyclodestructive procedures**
   - It is indicated in intractable glaucoma with no visual potential; where a part or whole of the ciliary body is destroyed either by cryoapplication or LASER to decrease aqueous production

8. A 3-month-old child is brought with bluish appearing, enlarged corneas. The parents give a history of the child burying her head in pillows and the presence of watering.

A. Enumerate three causes of epiphora in a neonate and differentiate among the three. (3 marks)

B. What is the probable condition the child is suffering from? (1 mark)

C. Give one ocular association. 1 mark)

A. Causes of Epiphora in a Neonate

1. Congenital dacryocystitis
2. Ophthalmia neonatorum
3. Primary congenital glaucoma

Differentiation

Parameter	Congenital dacryocystitis	Ophthalmia neonatorum	Primary congenital glaucoma
History	Watering and occasional discharge Starts within the first month	Watering with or without discharge may start as early as the first two days of birth	Can manifest anytime before three years of age. Signs of photophobia (buries head in pillow)
Family history	Not significant	Genital herpes or gonorrhea in the mother may be important	Not relevant
Laterality	Usually unilateral	Unilateral or bilateral	Bilateral
Cornea	Normal	May be affected in cases of keratitis	Enlarged and hazy (buphthalmos)
Sclera	Normal	Normal	Thinned
Axial length	Normal	Normal	High
Intraocular pressure	Normal	Normal	High
Regurgitation test	Positive	Normal	Normal
Lacrimal syringing	Obstruction	Normal	Normal

B. Most Probable Diagnosis

Primary congenital glaucoma

C. Ocular Association

Axenfeld anomaly

9. Explain the risk factors for the development of primary open-angle glaucoma. Describe the role of steroid responsiveness in primary open-angle glaucoma. (3+2 marks)

- Primary open-angle glaucoma is a condition with no underlying ocular associations
- The genetic basis of POAG is linked to the genome GLC-1A. two main genes are involved—myocilin (chromosome 1q21-31) and optineurin (10p)

Risk Factors for the Development of Primary Open-angle Glaucoma

1. **Heredity:** Inheritance is polygenic, a familial tendency exists. If a person develops POAG before 35 years, there is a high chance his siblings will develop it too due to gene mutation
2. **Age:** Risk increases with an increase in age. It is seen in the 6th and 7th decade onwards
3. **Myopes:** They have more chances of developing POAG
4. Diabetics have a higher prevalence of glaucoma
5. Ocular risk factors include thin central cornea (due to associated thin lamina cribrosa), vertical cup and reduced diastolic perfusion pressure (diastolic blood pressure-intraocular pressure)

Steroid Responsiveness

- A large subset of POAG patients are high responders (develop a pressure of more than 30 mm Hg when treated with topical dexamethasone for 6 weeks)
- Steroids induced production of myocilin in the trabecular meshwork leading to decreased outflow
- It is therefore understood that steroid responsiveness is genetically inherited and very closely linked to POAG
- Hence, steroids have to be carefully used in patients with POAG

11. Describe the following drugs concerning concentration, dose, indications, and two side effects.

A. Pilocarpine (2.5 marks)

B. Timolol (2.5 marks)

Drug	Concentration	Dose	Indications	Side effects
Pilocarpine	1%, 2%, 5%, 10%	QID	• Primary angle closure glaucoma • Plateau iris syndrome • Before performing LASER peripheral iridotomy	• Conjunctival injection, ocular and periocular pain (due to ciliary spasm), twitching of eyelids • Fluctuating myopic shift of up to 12–15D
Timolol	0.25%, 0.5% (higher percentage used in darker irides)	BID	• Primary open-angle glaucoma • Steroid-induced glaucoma • Preoperatively to control IOP in congenital glaucoma. • Glaucoma is associated with uveitis. • Glaucoma associated with hyphema • Angle-closure attack: to decrease IOP so that pilocarpine can act	• Can induce bronchospasm: to be carefully used in asthmatic patients. • Can decrease heart rate and BP: Great caution in patients with heart failure, sinus bradycardia, hypotension, hypokalemia, and brittle diabetes.

11. Explain the ocular and systemic side effects of topical beta-blockers used for glaucoma. (2+3 marks)

Ocular Side Effects

1. Stinging, burning, or itching
2. Timolol can exacerbate keratoconjunctivitis sicca, can cause periocular dermatitis, allergic conjunctivitis, and punctate keratitis
3. Long-term drift-using for long-duration causes decreased sensitivity to the drug

Systemic Side Effects

1. Can induce bronchospasm: To be carefully used in asthmatic patients
2. Can decrease heart rate and BP: Great caution in patients with heart failure, sinus bradycardia, hypotension, hypokalemia, and brittle diabetes
3. Symptoms related to the central nervous system are common in patients receiving β blocker therapy. They are mood swings, fatigue, confusion, depression, and loss of concentrating ability
4. Timolol can cause slowing of fetal heart rate and ventricular arrhythmias. Hence β blockers have to be used with extreme caution in pregnant women
5. Long-term use of timolol has been found to cause an increase in triglycerides and low-density lipoproteins carteolol has a slightly better therapeutic index in such patients than does timolol

12. Describe the mechanism of action and adverse effects of prostaglandins as antiglaucoma medications. (2+3 Marks)

Prostaglandins as Antiglaucoma Medications

Mechanism of Action

- They have mixed pharmacological responses because of the diversity of receptors
- The prostaglandin receptors are widely distributed in the eye accounting for their diverse biological effects
- Prostaglandin analogs combine with FP receptors and cause
  1. Relaxation of the ciliary muscle increases the aqueous outflow by the uveoscleral pathway
  2. They bring about the remodeling of the extracellular matrix by induction of the nuclear transcription factor and c-FOS. This causes the release of matrix metalloproteinases and that brings about degradation of the collagen, fibronectin in the ciliary muscle, and trabecular meshwork
  3. Other mechanisms for prostaglandin are proposed: Increase in blood flow to the optic nerve head and interaction with adrenergic pathway and increase in the conventional pathway by relaxation of the contractile trabecular meshwork fibers

Adverse Effects

- Prostaglandins have more local side effects
  1. Conjunctival hyperemia, foreign body sensation, eye irritation and superficial punctuate keratopathy-temporary side effects
  2. Itching, burning sensation, and eye pain
  3. Reduced corneal sensation, thickness

4. Periocular hyperpigmentation has been noted in most patients on long-standing treatment, which tends to disappear on discontinuation of the drug
5. Increase in length and number of lashes, long enough that can rub against the spectacles
6. Activation of latent herpes and uveitis is also a complication
7. Cystoid macular edema along with uveitis which responds to NSAIDs
8. Darkening of iris
9. Choroidal effusion

- Systemic side effects are minimal, except for headache and muscle pains
- Prostaglandins are best avoided in pregnant women, especially in the last three trimesters for the fear of termination of pregnancy

13. Describe the mechanism of action and adverse effects of carbonic anhydrase inhibitors as antiglaucoma medications. (2+3 marks)

Carbonic Anhydrase Inhibitors as Antiglaucoma Medications

Mechanism of Action

- Carbonic anhydrase enzyme catalyzes the following reaction
  $CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow HCO_3 + H^+$
- This step is critical for the formation of aqueous. Carbonic anhydrase inhibitors block the transport of bicarbonate ions from inside to outside of the cell
- As a result, intracellular bicarbonate increases
- Chloride-bicarbonate channels are activated. As a result, the intracellular concentration of chloride increases causing an acidic environment
- This inhibits the net flux of chloride across the ciliary epithelium
- As a result, the net concentration of sodium, chloride, and bicarbonate decreases in the aqueous

Adverse Effects

- Ocular side effects
  1. Irritation
  2. Blurring of vision
  3. Hypersensitivity reactions
- Systemic side effects
  1. Paresthesia around mouth and fingertips
  2. Gastritis, nausea, diarrhea
  3. Hypokalemia
  4. Metabolic acidosis—avoid hepatic insufficiency, renal insufficiency, severe pulmonary obstruction, and hyperchloremic acidosis
  5. Stevens–Johnson syndrome
  6. Thrombocytopenia, agranulocytosis, aplastic anemia

14. Describe cyclodestructive procedures. Give one indication and one side effect. (3+1+1 marks)

Cyclodestructive Procedures

- These are glaucoma surgeries in which a part or whole of the secretory part of the ciliary body is destroyed by various mechanisms

- They are:
  1. **Cyclo-cryotherapy:** Cryotherapy is applied at the region of the ciliary body (3 mm from limbus) for a minute. This is done in an area of 180° circumferentially
  2. **Nd: YAG Laser cyclodestruction:** ND: YAG Laser is used to destroying the ciliary body
  3. **Endo-cyclophotocoagulation:** It uses Diode LASER, xenon, helium-neon aiming beam, and LASER is delivered through slit-lamp
  4. **Diode transscleral cyclophotocoagulation:** 810 nm LASER is used to destroy the ciliary epithelium under peribulbar anesthesia

Indication

Absolute glaucoma due to any cause

Side Effect

Hypotony

15. Describe the risk factors, clinical features, and management of normal-tension glaucoma. Give one differential diagnosis. (1+1+2+1 marks)

Normal-tension Glaucoma

A subset of POAG where the intraocular pressure is always lesser than 21 mm Hg but the eyes show characteristic optic disc and visual field changes in presence of an open-angle

Risk Factors

1. Old age
2. Use of antihypertensive drugs in the night (decreases the perfusion to the optic nerve head)
3. Vascular diseases like Raynaud's phenomenon and peripheral vascular disease, migraine

Clinical Features

- They are very similar to POAG, except that IOP is normal and always less than 21 mm Hg
- They too have a very large fluctuation of IOP
- Visual fields in the NTG subgroup tend to approach fixation more commonly than POAG

Management

- Reduction in IOP to low teens is desired to decrease progression
- Betaxolol is the drug of choice in NTG patients due to its beta1 specificity, and no associated vasoconstriction. It also has neuroprotective action
- Prostaglandin analogs are very effective in decreasing IOP
- Brimonidine is useful due to its neuroprotection
- Systemic calcium channel blockers in proven cases of vasospasm
- Trabeculectomy is considered in failure to medical treatment

Differential Diagnosis

Steroid induced glaucoma (after cessation of steroids).

16. Describe the arrangement of retinal nerve fiber at optic disc and explain the reason for polar notching as an early sign of glaucoma. (3+2 marks)

Arrangement of Retinal Nerve Fiber Set Optic Disc (Fig. 6.7.22)

1. **Nasal fibers (radially arranged)**
   - They are called superior radial and inferior radial fibers (SRF and IRF)
   - They represent the temporal field which is the largest of all. So, the nerve fibers are thicker
2. **Macular fibers (macula to optic disc-papillomacular bundle)**
   - These are the thinnest fibers and highly dependent on ATP and have more mitochondria
   - They are the last to swell during papilledema but the first to be affected in toxic amblyopia, Leber's hereditary optic neuropathy, and B12 deficiency, all of which leads to depletion of ATP
3. **Temporal fibers (arch around macular fibers)**
   - They are called superior and inferior arcuate fibers (SAF and IAF)
   - They represent the nasal visual field
   - So, when there is temporal retinal detachment, the nasal field is lost and vice versa

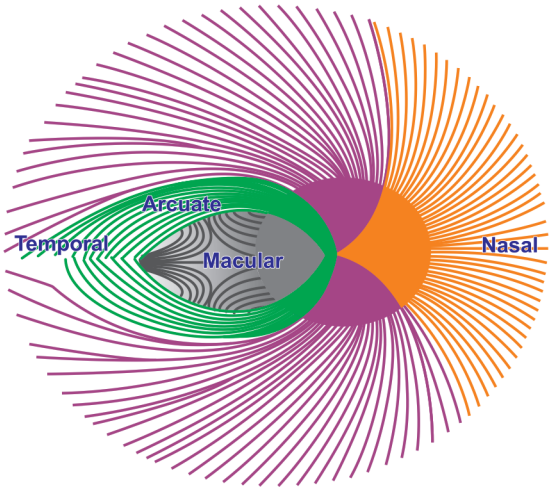


Fig. 6.7.22: Arrangement of nerve fibers

Polar Notching (Fig. 6.7.23)

- It is the phenomenon in which the neuroretinal rim in the upper and lower poles thin out as the earliest feature of glaucoma
- The cup starts to appear vertically oval
- This is because these fibers are more in number and get compressed easily with raised intraocular pressure or become vulnerable to hypoxia
- As a result, they are more sensitive to glaucomatous damage. So, polar notching is an early sign of glaucoma

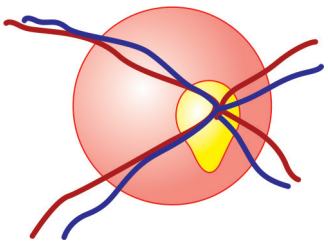


Fig. 6.7.23: Polar notching

17. Describe five investigations used in the diagnosis and follow-up of glaucoma. (5 marks)

Investigations Used In The Diagnosis and Follow-up of Glaucoma

1. **Tonometry and gonioscopy**
   - Tonometry is a measurement of IOP and is an important part of the examination for glaucoma diagnosis and monitoring treatment. It is best done with applanation tonometry or tonopen. A normal IOP is 10–21 mm Hg. Diurnal variation of more than 8 mm is diagnostic of glaucoma
   - Gonioscopy is required to classify glaucoma is open-angle or angle-closure. It is also useful to know other associated pathologies like neovascularization, synechiae, ICE syndromes, pigmentary glaucoma, and pseudoexfoliation syndrome

2. **Visual field analysis/perimetry**
   - Visual field analysis is usually performed with Humphrey 30-2, threshold program
   - It consists of a bowl perimeter where stimuli of varying strengths and sizes are presented to the patient's visual field
   - Various patterns of the visual field are discussed previously
   - A Humphrey printout shows a greyscale (which shows a pictorial representation of the field analysis) and various values like mean deviation, standard deviation, pattern standard deviation, and glaucoma hemifield test
   - These values should be compared every time to note progression and response to treatment. Also, various progression analyses are available
3. **Optical coherence tomography**
   - Optical coherence tomography is used to quantify retinal fiber loss
   - It gives an image of various layers of the retina in a transverse section and thickness in each quadrant
   - Usually, in glaucoma, superior and inferior RNFL thinning in the peripapillary area is seen
   - Various grades of thinning are color-coded and hence it is easy to interpret. It is useful to pick up pre perimetric glaucoma (very early stages of ganglion cell loss which do not show on visual field analysis)
   - Nowadays macular thickness profile is coming of use in detecting very early changes, which shoes nasal macular thinning
4. **Pachymetry**
   - It is a measurement of corneal thickness. It can be done by ultrasound or AS-OCT
   - It is necessary to get a corrected IOP when IOP is measured using applanation
5. **Scanning LASER polarimetry and confocal laser scanning tomography**
   - Other methods used similar to optical coherence tomography
   - Helps to measure peripapillary retinal nerve fiber thickness

18. Explain the classification of angle-closure glaucoma according to traditional and the AIGS method. (3+2 marks)

Classification of Angle-closure Glaucoma

Older Classification

Latent phase	Defined as a primary angle-closure suspect Posterior trabecular meshwork is not visible on glonioscopy >270° of iridotrabecular contact
Prodromal phase	Characterized by periods of headache and colored halos lasting for a few minutes to hours, associated with stress and dim illumination It terminates by self due to miosis or sleep
Acute congestive glaucoma	Severe headache, vomiting, and prostration associated with a drop in visual acuity Corneal edema, angle closed in all quadrants, and raised IOP around 40–50 mm Hg The disc is edematous
Post-congestive glaucoma	Vogt's triad 1. Glaucomflecken (anterior subcapsular lenticular opacity) 2. Patches of iris atrophy 3. Slightly dilated non-reacting pupil (due to sphincter atrophy)
Chronic congestive glaucoma	All features are the same as POAG (quiet eye, optic nerve head changes, and visual field changes) but gonioscopy shows synechiae angle closure

Contd.

Absolute glaucoma	The eye is painful and completely blind (no light perception) Mild ciliary congestion Caput medusae: a few radial and enlarged vessels are seen in long-standing cases Decreased corneal sensations may be the only early sign The cornea becomes hazy, bullous keratopathy

AIGS Classification

Feature	Definition	Clinical Signs
Primary angle-closure suspect	Contact between peripheral iris and posterior trabecular meshwork	The posterior trabecular meshwork is not visible on gonioscopy >270° of iridotrabecular contact
Primary angle-closure	Peripheral iris obstructs trabecular meshwork causing raised intraocular pressure	Peripheral anterior synechiae Raised IOP Glaucomflecken Excessive pigments on angle structures
Primary angle-closure glaucoma	All above features + glaucomatous optic neuropathy + visual field changes	

19. Describe the management of angle-closure glaucoma according to its stage. (5 marks)

Management of Angle-closure Glaucoma

1. **Primary angle-closure suspect**
   - Periodic follow-up
   - Prophylactic LASER iridotomy
   - Long-term pilocarpine
   - LASER iridoplasty
   - Cataract extraction in presence of cataractous lens
2. **Primary angle-closure**
   - Similar to primary angle-closure suspect
   - Peripheral laser iridotomy. This is done to create a bypass for aqueous outflow when a pupillary block occurs
3. **Acute congestive glaucoma**

Immediate	• Make the patient lie down supine ▪ Intravenous or oral Acetazolamide 500 mg ▪ Topical steroid (Dexamethasone or Prednisolone) QID ▪ Topical timolol 0.5% ▪ Analgesics and antiemetics
After one hour	• Topical pilocarpine 2%, in both eyes, 4 applications every 15 minutes
After 30 more minutes	• Recheck IOP—if not fallen <35 mm Hg, oral or intravenous hyperosmotic drugs ▪ IV 20% mannitol 1g/kg over 45-60 min (1 drop per second) ▪ Oral 50% glycerol 1g/kg
Later	• Perform gonioscopy to understand the synechiae status of the eye • LPI in the affected eye, when IOP has dropped and is around 20–25 mm Hg • LPI in the fellow eye after communicating with the patient

4. **Chronic congestive glaucoma**
   - Similar to POAG
   - Trabeculectomy at areas where peripheral synechiae

20. Describe the clinical features of lens-induced glaucoma. (5 marks)

Type	Clinical features
Phacomorphic glaucoma	Severe pain, low vision Irregularly/regular shallow anterior chamber Iridodonesis and phacodonesis may be present Vitreous in the anterior chamber in subluxation cases
Phacolytic glaucoma	Severe eye pain Corneal edema, ciliary congestion, a deep anterior chamber with 4+ cells and severe flare, hypopyon, and a Morgagnian cataract
Phacoanaphylactic glaucoma	History of cataract surgery in the fellow eye, following trauma or surgery in the present eye Uveitis, high IOP
Lens particle glaucoma	Features of uveitis, hypopyon Lens matter is seen in the anterior chamber Raised IOP

21. Explain the mechanism by which uveitis causes glaucoma. (5 marks)

Mechanism by which Uveitis Causes Glaucoma

- Glaucoma associated with or following uveitis is very common
- There are many mechanisms of causing high IOP in uveitis
- It can be associated with an open-angle orclosed angle

Open-angle	Angle-closure
Changes in anterior chamber • Increased inflammatory cells, osmolality, red blood cells, and lens particles (in lens-induced glaucoma) causes decreased aqueous outflow Changes in angle • Trabeculitis (inflammation of cells in TM) is seen as a part of certain infectious uveitis (herpes infection) • Due to scarring of the trabeculum (post-inflammatory) • Steroid-induced glaucoma	• Due to secclusio pupillae-pupillary block glaucoma • Organized inflammatory debris in angle • Formation of peripheral anterior synechiae causes decreased aqueous outflow and glaucoma
Examples 1. Acute herpetic iridocyclitis 2. Fuch's heterochromic cyclitis 3. Posner–Schlossman syndrome 4. Lens protein uveitis 5. Juvenile idiopathic arthritis	Examples 1. Pupillary block glaucoma

22. Describe the etiology, clinical features, and management of malignant glaucoma. (2+1+2 marks)

Malignant Glaucoma

- Also known as a ciliary block or aqueous misdirection glaucoma
- In this condition, aqueous gets collected in the vitreous instead of the anterior chamber

Etiology

- Risk factors include hypermetropic eyes, nanophthalmos, and plateau iris configuration
- Following intraocular surgery, the tips of ciliary processes are anteriorly rotated and press against the equator of the lens in phakic eyes (cilio-lenticular block) or the intraocular lens (cilio-IOL block) or against the anterior hyaloid phase of vitreous in aphakic eyes (ciliovitreal block)
- This leads to a block of the forward flow of aqueous humor, which is diverted posteriorly and collects as aqueous pockets in the vitreous. As a result, the iris lens diaphragm is pushed anteriorly leading to secondary angle closure

Clinical Features

- This condition is characterized by a shallow anterior chamber with a raised intraocular pressure
- As against pupillary block glaucoma, the anterior chamber is diffusely flat
- Visual acuity is reduced due to corneal edema

Management

- A patent Nd-YaG iridotomy is required to diagnose malignant glaucoma
- Atropine is the first line of treatment as it pushes the iris lens diaphragm posteriorly by posterior rotation of the ciliary body
- Aqueous suppressants like beta-blockers, carbonic anhydrase inhibitors are used to decrease aqueous formation
- Nd YAG hyaloidotmy (creating an opening with LASER to release aqueous from the vitreous) is indicated in pseudophakic and aphakic eyes
- Chandler's procedure is a traditional procedure in which aqueous is aspirated from the vitreous using a needle and a syringe
- Vitrectomy is done if all the above measures fail

23. Elaborate peripheral iridotomy concerning indications, procedure, and complications. (5 marks)

Peripheral Iridotomy

An outpatient procedure in which an opening is created in the peripheral aspect of the iris using Nd: Yag laser

Indications

1. Treatment of an acute attack of angle-closure glaucoma
2. Post-inflammatory pupillary block glaucoma
3. To prevent an attack of angle-closure on the fellow eye
4. Prophylactically in latent angle closure
5. Malignant glaucoma sometimes may respond

Procedure

- The location of 11 o'clock or 1 o'clock on the peripheral iris is preferred
- The 12 o'clock position is avoided as gas bubbles released during the procedure may block the iridotomy
- Lower quadrants may lead to diplopia and glare
- The pupil is constricted using pilocarpine
- Some prefer to use apraclonidine before LASER

- An Abraham lens is used to fix the eye and focus the LASER
- A spot is chosen 1 mm from the periphery (too much in the periphery may lead to anterior synechiae formation) in between the crypts, LASER shot of 5–7 mJ strength is applied
- Successful iridotomy is marked by aqueous flowing out

Complications

1. Hyphema
2. Raised IOP
3. Corneal endothelial burn

SHORT ANSWERS

1. Differentiate between Megalocornea and Buphthalmos. (3 marks)

Feature	Megalocornea	Buphthalmos
Presentation	At birth	First year of life
Genetic	X linked recessive	Sporadic
Natural history	Non progressive	Progressive
Symptoms	None	Photophobia, lacrimation
Cornea	Enlarged, >13 mm Cornea globose (globe-shaped) Clear cornea	Can vary, asymmetric Flat cornea due to stretching Hazy due to edema
Anterior chamber	Deep	Deep
Axial length	Normal	High
Optic disc	Normal	Cupping or atrophy
Intraocular pressure	Normal	High

2. Describe the clinical features of primary congenital glaucoma. 3 marks)

- The presentation of childhood glaucomas is unique. Parents bring the child with excess tearing, child burying its head into pillow and blepharospasm. It is due to irritation of corneal nerves due to corneal edema
- The cornea appears hazy due to edema and is enlarged. It is called buphthalmos—bull eye. Normally, the diameter of the cornea is 10.5 mm; more than 13 mm is considered pathognomonic
- Haab's striae: Tears in Descemet's membrane are seen as parallel peripheral concentric tears
- The sclera is thinned due to increased pressure and appears blue
- The anterior chamber is deep
- Iris appears thinned and the lens is stretched
- The optic disc shows cupping or atrophy due to increased pressure
- Axial myopia is seen

3. Describe the clinical features of Juvenile glaucoma. (3 marks)

- Juvenile glaucoma is an autosomal dominant condition that presents in the age group of 10–35 years. It is due to a mutation occurring in the myocilin gene
- The risk factors for juvenile glaucoma include male sex, myopia, and African ancestry
- It is characterized by raised intraocular pressure in presence of open angles

- The patients are usually asymptomatic similar to POAG and discovered accidentally. Some may present with mild ocular pain and diminution of vision
- On examination, there is corneal edema, increased IOP (in the range of 40–45 mm Hg), and open angles on gonioscopy
- Optic disc changes and field changes usually are similar to POAG and a large subset of these patients go on to develop POAG in their adulthood

4. Explain the implications of **(3 marks)**

A. Water drinking test

B. Prone test

C. IOP phasing

A. Water-drinking Test

- It is a provocative test of historical interest
- A decrease in plasma osmolality due to increased water intake causes a decrease in aqueous outflow. As a result, IOP rises, more so in eyes with POAG
- After an overnight fast, baseline IOP is checked. Later, the patient is asked to drink 1L of water, following which IOP is noted every 15 minutes for 1 hour. IOP peaks at 15–30 minutes and returns to baseline level after 60 minutes
- A rise of 8 mm of Hg or more is said to be diagnostic of POAG

B. Prone Test

- After noting baseline IOP, the patient is made to lie in a prone position in a darkroom
- Dim illumination leads to mydriasis and the prone position helps to move the iris-lens diaphragm forward
- An increase in IOP of more than 8 mm Hg at the end of one hour is diagnostic of compromised angle

C. IOP Phasing

- It is the charting of regular IOP over 24 hours
- Charting of IOP every 2–4 hours for 24 hours is needed to know the diurnal variation of IOP. It is known as phasing
- Phasing is an important requirement for the diagnosis of POAG when the initial IOP may not be higher than 21 mm Hg
- Phasing helps to know the highest pressure, so the target pressure can be set accordingly

5. Define the target pressure. Describe the concept of neuroprotection.
(1+2 marks)

Target Pressure

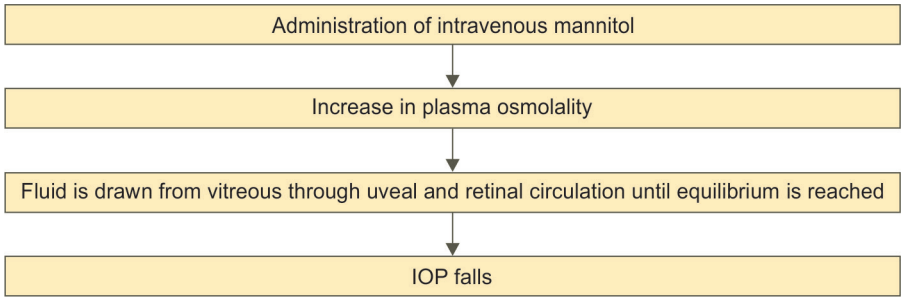
- Target pressure is an intraocular pressure at which progression of glaucoma is minimal
- It is based on factors like age of the patient, presence of risk factors, size of the cup, and family history of glaucoma

Neuroprotection

- It is a novel concept for treating glaucoma. It simply means protecting the remaining viable optic nerve head fibers
- It is an IOP-independent method of decreasing glaucoma progression

- Brimonidine is an alpha-agonist that has neuroprotective properties. It has been postulated to reduce the death of retinal ganglion cells from somatic, axonal, and dendritic degeneration. The dose is 0.2% BID and is indicated in low-tension glaucoma
- Another such drug is betaxolol. It is a selective beta 1 antagonist
- Mesenchymal stem cells are also found to be helpful in neuroprotection
- Various nerve growth factors like ciliary neurotrophic factor, brain-derived neurotrophic factor, and recombinant human nerve growth factor are being tried for neuroprotection in glaucoma
- Several drugs like ginkgo biloba, memantine, citicoline, calcium channel blockers, and nicotinamide are being tried for neuroprotection due to their effects on ocular blood flow

6. Explain the mechanism of action of mannitol. (3 marks)



7. What are glaucoma drainage devices? Name two commonly used devices. (2 + 1 marks)

Glaucoma Drainage Devices

- Glaucoma shunts are synthetic devices that are placed in the subconjunctival space connecting to the anterior chamber
- They have a plate that goes into the subTenon/subconjunctival space and a tube that enters the anterior chamber.

Commonly Used Devices

1. Beervedlt valve
2. Ahmed valve

8. Give three indications for the use of glaucoma drainage devices. (3 marks)

1. Neovascular glaucoma
2. Glaucoma with aniridia
3. Intractable cases of primary and secondary glaucoma

9. Explain Bjerrum scotoma. (3 marks)

- Bjerrum's area is that part of the visual field that lies between 10–20° from fixation
- It is seen as an extension of Seidel's scotoma (Fig. 6.7.24)
- Early glaucomatous changes are seen as decreased sensitivity in this area and are referred to as Bjerrum's scotoma
- It is a relatively steep scotoma and is more commonly seen in the superonasal quadrant

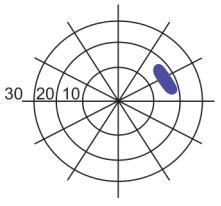


Fig. 6.7.24: Bjerrum's scotoma

10. Explain von Herick's grading of the angle of the anterior chamber. (3 marks)

- Depth is graded based on von Herick's grading. In this method, a narrow beam of light is focused on the peripheral cornea
- The distance between the beam reflecting off the cornea and the iris is considered as peripheral anterior chamber depth (PACD)

Grade 4 (Wide open angle)	PACD = ½ > CT
Grade 3 (Mild narrow angle)	PACD = ¼ to ½ CT
Grade 2 (Moderate narrow angle)	PACD = ¼ CT
Grade 1 (Extremely narrow angle)	PACD < ¼ CT
Grade 0 (Closed angle)	PACD = Nil

11. What is Fincham's test? Explain its use. (3 mark)

- Fincham test is a simple test using a stenopic slit and is performed in patients with colored halos
- It is a test with historical and academic interest
- In this test, a stenopic slit is placed in front of the eye with colored halos in different orientations (Fig. 6.7.25)
- If the halos break and reappear, then the colored halos of lenticular origin
- If they are constant, then it is corneal in origin as corneal edema is diffuse

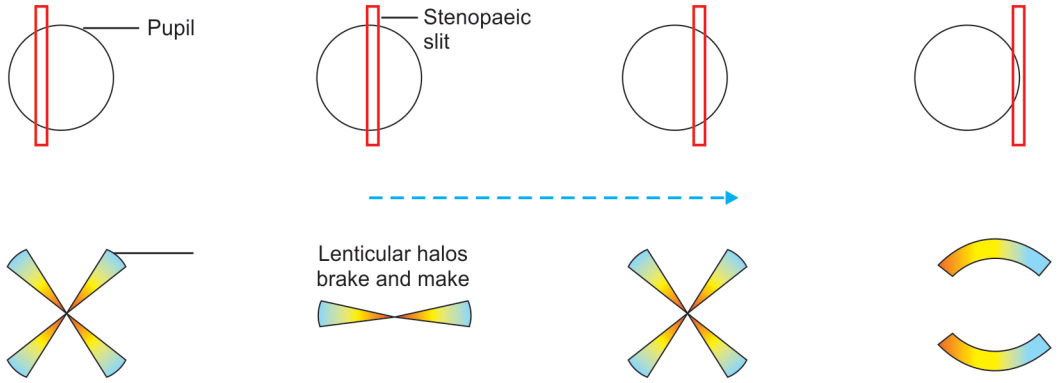


Fig. 6.7.25: Fincham test

12. Elucidate the features of Vogt's triad. (3 marks)

- Vogt's triad is seen as a consequence of acute congestive glaucoma and serves as a marker of a well-managed (IOP controlled) recent attack of angle closure
- It is characterized by:
  1. Glaucomflecken (anterior subcapsular lenticular opacity)
  2. Patches of iris atrophy
  3. Slightly dilated non-reacting pupil (due to sphincter atrophy)

13. Describe the anterior segment findings of pigmentary glaucoma. (3 marks)

Cornea	Krukenberg spindle: Pigments arranged in the form of a vertical spindle in the lower half of the cornea, on the endothelium
Anterior chamber	Deep
Iris	Mid peripheral transillumination defects Heterochromia in asymmetric cases
Angle	Wide-open angle, posterior insertion of the iris Sampaolesi's line: Deposition of pigments anterior to Schwalbe's line, more so in the inferior part.
Lens zonules	Eggert' line: pigments on the hyaloido-capsular ligament

14. Describe the clinical features of pseudoexfoliation glaucoma. (3 marks)

Symptoms

- The patients usually do not have any symptoms and are accidentally discovered
- A cataract is a common ocular association and thus may present with painless, progressive loss of vision

Signs

Cornea	Deposition of PEX and pigments on endothelium
Anterior chamber	Mild flare can be there
Iris and pupil	White powdery deposits on the pupillary border with a moth-eaten appearance at the sphincter The pupil does not dilate well
Lens	Due to the constant movement of the pupil, there is a preferential arrangement of this PEX material central material with mid-peripheral clearing and a peripheral ruff The higher tendency of nuclear cataract formation Zonules are weaker Cataract surgery is difficult in these cases- non dilating pupil, zonular weakness, and corneal decompensation
Gonioscopy	Pigment deposition may be confused with pigmentary glaucoma PEX material may be seen in angles

15. Explain the clinical features of various stages of neovascular glaucoma. (3 marks)

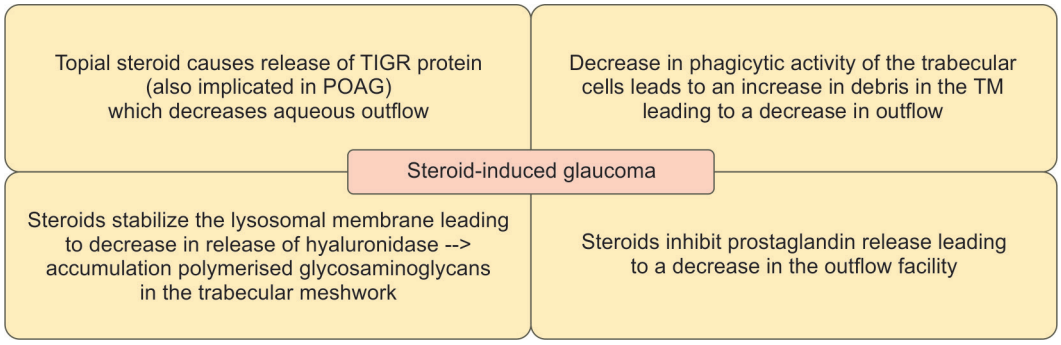
Preglaucomatous stage	It is an asymptomatic stage On examination, there is a presence of new vessels seen on the iris surface and in the angle. Fundus examination reveals the cause of neovascular glaucoma. (CRVO, PDR, etc)
Open-angle glaucoma stage	It is an asymptomatic condition characterized by raised intraocular pressure and the presence of new vessels in the iris and the angle There is progressive cupping
Secondary angle-closure stage	The patient presents with severe pain and diminished vision It is characterized by high IOP and corneal edema Gonioscopy reveals the presence of a neovascular membrane in the angle causing

16. Describe the principles of glaucoma shunt surgeries. (3 marks)

- Glaucoma shunts are synthetic devices that are placed in the subconjunctival space connecting to the anterior chamber
- They have a plate that goes into the sub-Tenon/subconjunctival space and a tube that enters the anterior chamber
- The aqueous is thus drained from the anterior chamber directly to the subconjunctival space and bypasses the natural course through the trabecular meshwork
- These are indicated when there are peripheral anterior synechiae that can interfere with a trabeculectomy. Also, they have better IOP control and are thus reserved for cases with intractable glaucoma
- Some types of glaucoma like ICE syndrome, neovascular glaucoma, and congenital glaucoma are well benefitted from glaucoma shunts
- Some shunts have valves (Ahmed glaucoma valve) which help in maintain IOP so that hypotony does not occur.
- They are indicated in cases of all types of glaucoma where trabeculectomy has failed

17. Explain the ways by which steroids can cause glaucoma. (3 marks)

The proposed mechanisms for steroid-induced glaucoma are:



18. Define ocular hypertension. Enumerate four risk factors (according to the ocular hypertension treatment trial) that have been found to increase the progression to primary open-angle glaucoma. (1+2 marks)

Definition of Ocular Hypertension

- It is a subtype of POAG characterized by raised intraocular pressure (>21 mm Hg) with no visual field changes of optic nerve head changes
- Usually, the IOP is in the range of the late twenties

Risk Factors

Conversion to POAG has some risk factors such as (according to ocular hypertension treatment trial)

1. Positive water drinking provocative test
2. IOP constantly more than 28 mm of Hg
3. Central corneal thickness <555 μm
4. Myopia

COMPETENCY

OP6.8 ENUMERATE AND CHOOSE THE APPROPRIATE INVESTIGATION FOR PATIENTS WITH CONDITIONS AFFECTING THE UVEA

SHORT ESSAYS

1. Describe the investigations that are used in various types of uveitis. (5 marks)

A thorough history regarding the onset of the disease must be elicited by questioning the patient regarding past episodes, recurrences and drugs that are being used.

Anterior Uveitis

- Investigations are not required in first episode of unilateral iridocyclitis
- They are indicated in bilateral, recurrent or if associated with posterior uveitis
- The investigations should be individualized based on clinical presentation and not done as a battery of tests

Investigations

Blood

- Complete hemogram
- ESR; inflammatory pathology
- Blood sugars, uric acid
- RA factor, antinuclear antibodies, ASO (to rule out JIA associated uveitis)
- HLA typing in relevant cases

Others

- Urine routine and culture (to rule out Reiter's)
- Chest X ray for ruling out sarcoid, TB
- X-ray of spine to look for ankylosing spondylosis
- Mantoux, toxoplasmin and Kveim's test
- Colonoscopy in relevant cases to rule out Inflammatory bowel disease

Based on the type of presentation, the investigations necessary in a case of anterior uveitis are:

Nongranulomatous anterior uveitis in an adult	HLA B27 if recurrent iridocyclitis Chest X-ray, VDRL, FTA Abs
Granulomatous anterior uveitis in an adult	Chest X-ray, Mantoux test Serum angiotensin converting enzyme (ACE) levels, VDRL and FTA-ABS tests
Anterior uveitis in a child	ANA, HLA B 27
Suspected sarcoidosis in an adult or a child with granulomatous uveitis	Chest X-ray, HRCT chest Serum CA, ACE levels Whole body gallium scan, bronchoscopy with biopsy

Intermediate Uveitis

- Following a thorough ophthalmic examination, systemic associations of IU
- Multiple sclerosis: MRI brain, CSF analysis
- Lyme disease: Serology ELISA for borrelia antibody
- VDRL, FTA Abs: To rule out syphilis
- Sarcoidosis: Serum calcium, serum angiotensin converting enzyme, chest X-ray
- To rule out tuberculosis: Chest X-ray and Mantoux test

Posterior Uveitis

Infective

- It is individualized based on the clinical appearance of the lesion
- For toxoplasmosis: serum IgG and IgM *Toxoplasma*
- For tuberculous uveitis: Mantoux test, X-ray chest
- For syphilitic uveitis: VDRL, RPR, treponema immobilization test, MRI brain
- For CMV retinitis: Aqueous and vitreous IgM
- PCR of aqueous

Noninfective

- Ocular investigations: FFA, B scan, ICG angiography
- HLA typing to rule out autoimmune vasculitis
- Sarcoidosis: Serum Ca, ACE levels, whole body Ga scan
- For disseminated choroiditis: MRI brain
- For periphlebitis: ANA, serum calcium and ACE (for sarcoid)

2. Enumerate the indications for investigations in uveitis. Describe the investigations for anterior granulomatous uveitis. (2+3 marks)

Indications for Investigations in Uveitis

- All cases of uveitis need to undergo a complete ophthalmic examination
- The first episode of anterior uveitis does not need to be investigated, especially if the fundus is normal
- Indications for investigations are:
  1. Recurrent iridocyclitis
  2. Bilateral iridocyclitis
  3. Any iridocyclitis in a child
  4. Any posterior uveitis
  5. Any intermediate uveitis
  6. Pan-uveitis need to be investigated

Investigations for Anterior Granulomatous Uveitis

Acute Granulomatous Uveitis in an Adult

- To rule out TB
  - Chest X-ray, Mantoux test
- To rule out sarcoidosis
  - Serum angiotensin-converting enzyme (ACE) levels
  - HRCT chest and whole-body Ga scan
- To rule out syphilis
  - VDRL and FTA-ABS tests

Acute Granulomatous Uveitis in a child

- To rule out JIA associated uveitis
  - HLA B27
  - ANA
- To rule out sarcoidosis
  - Chest X-ray, serum ACE levels

SHORT ANSWERS

1. Enlist the investigations of ocular sarcoidosis. (3 marks)

Supportive Diagnosis

- Cutaneous anergy
- Raised serum calcium, angiotensin-converting enzyme
- Whole-body gallium scan
- BAL (bronchoalveolar lavage) fluid for CD4: CD8

Confirmatory Diagnosis

Histopathology of a lymph node suggestive of noncaseating granuloma

2. Enumerate investigations for intermediate uveitis. (3 marks)

Following a thorough ophthalmic examination, systemic associations of IU as mentioned before have to be ruled out

1. Multiple sclerosis: MRI brain, CSF analysis
2. Syphilis: VDRL, FTA-ABs
3. Lyme disease: Serology ELISA for borrelia antibody
4. Sarcoidosis: Serum calcium, serum angiotensin-converting enzyme, chest X-ray

3. Enumerate the investigations that may help in diagnosing the cause for a solitary retinitis lesion. (3 marks)

Causes for a solitary retinitis patch include ocular toxoplasmosis, toxocariasis, syphilis, and tuberculosis

Toxoplasmosis	• Serum IgG and IgM toxoplasmosis • Aqueous IgG and IgM toxoplasmosis • HIV and CD4+ counts
Toxocariasis	• IgM toxocara
Syphilis	• Serum VDRL, FTA-Abs • Demonstration of live treponemes in aqueous from paracentesis
Tuberculosis	• Mantoux test, chest X-ray • Aqueous or vitreous tap positive for *Mycobacterium tuberculosis* by PCR • Histopathology of hilar or cervical lymph nodes • Sputum smear • Quantiferon

COMPETENCY

OP6.9 CHOOSE THE CORRECT LOCAL AND SYSTEMIC THERAPY FOR CONDITIONS OF THE ANTERIOR CHAMBER AND ENUMERATE THEIR INDICATIONS, ADVERSE EVENTS AND INTERACTIONS

SHORT ESSAYS

1. Describe the treatment of anterior uveitis. (5 marks)

Anterior uveitis or iridocyclitis is treated with:

A. Nonspecific therapy

B. Specific therapy

A. Nonspecific Therapy

1. **Cycloplegics**
   - These are the mainstay of treatment in anterior uveitis
   - Atropine is the best drug available
   - It is available as 1% atropine sulfate drops and ointment
   - It acts in the following ways
     - Relaxes the ciliary body and decreases pain
     - Dilates the pupil and thus decreases the contact between lens and pupil
     - Prevents the formation of synechiae and breaks the already formed ones
     - Reduces inflammation by decreasing vascularity and permeability
     - Increases absorption of other drugs
   - Other drugs that are used are 2% homatropine, 1% cyclopentolate, tropicamide-phenylephrine (mild cases)
   - Alternatively, subconjunctival injection of adrenaline, atropine, and procaine (mydricain) can be given in severe cases
   - They must be given for at least 2–3 weeks even after the inflammation subsides to prevent recurrences
2. **Corticosteroids**
   - The steroids that can be used may range from potent steroids like prednisolone and Dexamethasone to milder forms like loteprednol, fluorometholone, and medrysone
   - Depending on the severity, the steroids are instilled frequently and then tapered gradually
   - A few maintain a small dose of steroid in recurrent cases
   - It may also be given as subconjunctival or sub-Tenon injection in severe cases
   - Systemic steroids are only indicated in a few cases of bilateral iridocyclitis or resistance to topical steroids
3. **Other supportive drugs**
   - If associated with complications, their treatment is required as well

Cystoid macular edema	Topical NSAIDs
Complicated cataract	Cataract extraction
Glaucoma	Antiglaucoma medications like carbonic anhydrase inhibitors and beta-blockers can be used In cases of pupillary block, not resolving with mydriasis, peripheral iridotomy should be performed

B. Specific Therapy

- If caused by an infectious agent, then antimicrobial is used. For example, ATT for tuberculosis, acyclovir in herpes zoster uveitis, penicillin in syphilis, and so on
- NSAIDs (Sulfasalazine) are used in uveitis associated with ankylosing spondylosis

2. Describe the management of intermediate uveitis. (5 marks)

Intermediate Uveitis

Investigations

- Following a thorough ophthalmic examination, systemic associations of IU as mentioned before have to be ruled out
- Multiple sclerosis: MRI brain, CSF analysis

- Lyme's disease: Serology ELISA for *Borrelia* antibody
- Sarcoidosis: Serum calcium, serum angiotensin-converting enzyme, chest X-ray

Management

- 80% of cases resolve spontaneously, hence may not require treatment
- In severe non-resolving conditions, steroids are given in the form of subtenon injections or oral administration
- Side effects of steroids include steroid-induced glaucoma and cataract
- If associated with multiple sclerosis, interferon-beta may be of help
- In presence of neovascularization in the peripheral retina, laser photocoagulation is indicated
- Vitrectomy: Indicated when
  - There is no response to steroids
  - Vitreous opacities significantly decreasing vision
  - Unresolved cystoid macular edema
  - Vitreous hemorrhage
  - Tractional retinal detachment
  - Epiretinal membrane
- Accompanying endo-LASER can be done if there is associated neovascularization

3. Describe the treatment of noninfectious posterior uveitis. (5 marks)

Treatment of Noninfectious Posterior Uveitis

Noninfectious posterior uveitis is treated with corticosteroids and immunomodulators.

Corticosteroids

- Steroids can be given in the form of periocular steroids and systemic steroids
- Periocular steroids can be given as subtenon or orbital floor injections
- Systemic steroids are given in the inflammatory dose (1 mg/kg of prednisolone)
- Along with periocular steroids
  - Sarcoidosis: Systemic steroids
  - Behcet's disease: Systemic steroids, chlorambucil
  - Ankylosing spondylosis: Systemic indomethacin and Aspirin
  - VKH and sympathetic ophthalmia: Systemic steroids
  - When unresponsive to steroids, immunomodulators are to be used

Immunosuppressants

- Many immunosuppressants are useful in controlling inflammation in uveitis, especially if associated with a systemic component
- The immunosuppressives are of many types
- Antimetabolites (methotrexate, mycophenolate mofetil, azathioprine)
- T-cell inhibitors (tacrolimus and cyclosporine)
- Alkylating agents (cyclophosphamide and chlorambucil)

Biologicals

- Mainly reserved for chronic relapsing and nonresponsive, noninfectious uveitis
  1. **Daclizumab (IL 2 antagonist)**
     - It is a CD20 monoclonal antibody
     - Interleukin 2 is an important part of the lymphokine receptor system

- Blocking its action is brought about by daclizumab
- It is given as 1–2 mg/kg subcutaneously or intravenously every 2–4 weeks

2. **Infliximab (TNF alpha antagonist)**
   - It is approved for use in rheumatoid arthritis, ankylosing spondylitis, and Crohn's disease
   - It is used in uveitis refractory to conventional therapy and is useful in Behcet's syndrome, juvenile idiopathic arthritis, inflammatory bowel disease, VKH
   - It is given as an intravenous injection of 3–5 mg/kg dose, weekly

- Others are adalimumab (TNF alpha antagonist), etanercept (TNF alpha antagonist), rituximab (CD20 inhibitor), and anakinra (interleukin 1 receptor antagonist)

4. Describe the role of steroids in uveitis. Explain its side effects. (2+3 marks)

Uveitis

Role of Steroids

- Steroids act in uveitis by decreasing inflammation
- Steroids enter the anterior chamber through the conjunctival vessels and diffuse through the cornea
- Commonly used steroids are dexamethasone, prednisolone, loteprednol, fluorometholone, and medrysone
- Depending on the severity, the steroids are instilled frequently and then tapered gradually
- A few maintain small doses of steroids in recurrent cases. It may also be given as subconjunctival or subtenon injection in severe cases
- Systemic steroids are indicated in a few cases of bilateral iridocyclitis or resistance to topical steroids

Side Effects

Topical Steroids

- Steroid-induced glaucoma
- Steroid-induced cataract
- Increased propensity to fungal keratitis

Systemic

- Steroid-induced cataract
- Steroid-induced glaucoma
- Systemic hypertension and hyperglycemia
- Secondary Cushing's syndrome
- Osteoporosis
- Increased weight gain
- Easy bruising

5. Describe the procedure of evisceration. (5 marks)

Procedure of Evisceration

- Removing the contents of the eyeball is called evisceration
- It is indicated in panophthalmitis, bleeding anterior staphyloma, and expulsive choroidal hemorrhage

Simple Evisceration

- Conjunctival peritomy and tenonectomy are done circumferentially
- The eyeball is entered with a stab wound at the limbus
- The wound is extended on both sides to remove the cornea
- Intraocular contents are scooped out with an evisceration scoop
- Care must be taken to remove all choroid
- The remaining sclera is sutured in purse-string fashion with an implant or otherwise. The conjunctiva is closed over it
- A prosthesis is placed at a later stage

Frill Evisceration

- Conjunctival peritomy is done circumferentially
- The eyeball is entered with a stab wound at the limbus
- The wound is extended on both sides to remove the cornea
- Intraocular contents are scooped out with an evisceration scoop
- Care must be taken to remove all choroid
- The extraocular muscles are severed
- The remaining sclera is excised using scissors leaving a frill of 3 mm around the optic nerve
- Conjunctiva and Tenon's fascia are closed

6. Explain the methods of delivering periocular steroids. (5 marks)

Methods of Delivering Periocular Steroids

1. **Posterior sub-Tenon**
   - After instilling topical proparacaine, a cotton pledget soaked in paracaine is placed in the superior fornix
   - 40 mg/ml of triamcinolone is loaded in a 2 ml syringe and a 25 guage needle is inserted
   - The patient is advised to look inferonasally steadily
   - The needle is inserted in the superotemporal quadrant of the globe in the bulbar conjunctiva and the needle is advanced in a zig-zag manner along the contour of the globe
   - After withdrawing to check for vascular entry, the drug is delivered
2. **Inferior transseptal injection (orbital floor injection)**
   - The skin of the lower lid and maxillary area is cleaned with spirit
   - 40 mg/ml of triamcinolone is loaded in a 2 ml syringe and a 25 guage needle is inserted
   - The patient is advised to maintain a straight gaze
   - Through the skin of the lower lid, at the junction of the outer third and inner two-thirds, the needle is inserted along the orbital floor. The orbital septum is pierced and the needle is moved in a slight zig-zag manner in order to avoid perforation of the sclera
   - After withdrawing to check for vascular entry, the drug is delivered

SHORT ANSWERS

1. Outline the treatment of idiopathic anterior uveitis. (3 marks)

Anterior uveitis or iridocyclitis is treated with topical steroids and cycloplegics.

Topical Cycloplegics

- These are the mainstay of treatment in anterior uveitis.

- Atropine is the best drug available. It is available as 1% atropine sulfate drops and ointment
- Other drugs that are used are 2% homatropine, 1% cyclopentolate, tropicamide-phenylephrine (mild cases)
- Alternatively, subconjunctival injection of adrenaline, atropine, and procaine (mydricain) can be given in severe cases
- They must be given for at least 2–3 weeks even after the inflammation subsides to prevent recurrences

Corticosteroids

- The steroids that can be used may range from potent steroids like prednisolone and Dexamethasone to milder forms like loteprednol, fluorometholone, and medrysone
- Depending on the severity, the steroids are instilled frequently and then tapered gradually
- A few maintain a small dose of steroid in recurrent cases. It may also be given as subconjunctival or subtenon injection in severe cases
- Systemic steroids are only indicated in a few cases of bilateral iridocyclitis or resistance to topical steroids

2. Name three biologicals used in uveitis. (3 marks)

1. Daclizumab (IL-2 antagonist)
2. Infliximab (TNF alpha antagonist)
3. Adalimumab (TNF alpha antagonist)

3. Describe the treatment of ocular toxoplasmosis. (3 marks)

- Treatment is not mandated in all cases of ocular toxoplasmosis
- In the adult form, the infection usually arises from the proximity of a healed lesion
- Indications for therapy in toxoplasmosis
  1. Lesion close to the macula
  2. Lesion close to the papillomacular bundle
  3. Lesion close to the optic nerve
  4. Lesion close to a major arterial arcade
  5. Significant vitritis
  6. Immunocompromised individuals
- The treatment is continued till the resolution of the retinitis patch is seen
- Systemic corticosteroids are started under the cover of antimicrobials

Drug	Dose	Contraindications
Clindamycin	300 mg QID	Hepatotoxicity Renal toxicity Pseudomembranous colitis
Cotrimoxazole (not as efficacious as classical therapy)	800 mg/160 mg	G6PD deficiency Sulfa allergy Bone marrow suppression
Azithromycin	500 mg/day	GI disturbances Useful in pregnancy
Spiramycin	1.5 million IU	GI hypersensitivity Safest in pregnancy
Atovaquone	750 mg QID	Caution in liver failure

4. Enumerate three indications of vitrectomy. (3 marks)

1. Retinal detachment
2. Endophthalmitis
3. Vitreous hemorrhage

COMPETENCY

OP6.10 COUNSEL PATIENTS WITH CONDITIONS OF THE IRIS AND ANTERIOR CHAMBER ABOUT THEIR DIAGNOSIS, THERAPY AND PROGNOSIS IN AN EMPATHETIC MANNER IN A SIMULATED ENVIRONMENT

SHORT ANSWER

1. Justify the need for timely diagnosis and treatment of JIA-associated uveitis. (3 marks)

- JIA is associated with chronic, unilateral/bilateral, anterior uveitis
- It is also known as Still's disease and can present in the eye with complications of uveitis like cataracts, band-shaped keratopathy, and even phthisis bulbi
- The inflammation tends to be very mild and chronic and the children may not report the symptoms like pain, photophobia, blurred vision, and redness, which is usually seen in other causes of uveitis. This phenomenon is also called 'white-eye uveitis'
- On a careful slit-lamp examination, one can find evidence of uveitis in the form of cells, flare, and even posterior synechiae
- Children of the female gender, with ANA positivity, oligoarthritis, and age of onset of arthritis below 4 years have a higher risk of developing arthritis

Screening

- Meticulous screening with a slit-lamp examination and IOP measurement is required as these patients are also prone to hypertensive uveitis
- Diagnosis is based on the presence of cells and flare

Management

- They are treated with topical steroids and cycloplegics
- Atropine is best avoided in these children for the fear of developing amblyopia and posterior synechiae as the pupil remains dilated. Short-acting cycloplegic-mydriatics (cyclopentolate, tropicamide) are preferred
- Methotrexate is the gold-standard systemic treatment for JIA-associated anterior uveitis