Ophthalmology Posters

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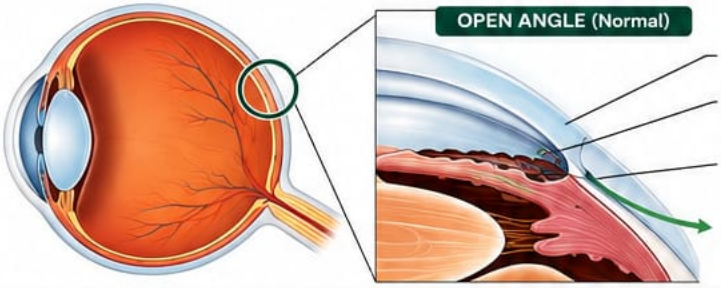
OPEN ANGLE GLAUCOMA

The Silent Thief of Sight

DEFINITION: A chronic progressive optic neuropathy characterized by retinal ganglion cell loss, optic disc cupping, and corresponding visual field defects, usually associated with elevated intraocular pressure.
(Can occur with normal IOP – Normal-Tension Glaucoma)



WHY IT MATTERS?

- One of the leading causes of irreversible blindness worldwide.
- Often asymptomatic in early stages – diagnosed late.
- Early detection and treatment can prevent vision loss.

PATHOPHYSIOLOGY

Degeneration and extracellular matrix accumulation in trabecular meshwork → increased resistance → Impaired aqueous outflow through trabecular meshwork (Schlemm's canal resistance) → ↑ Intraocular Pressure (IOP) → Mechanical stress + Ischemia at optic nerve head → Retinal ganglion cell death → Optic disc cupping & Retinal nerve fiber layer loss → Visual field defects (progressive)

TREATMENT GOAL: Achieve target IOP for each patient to prevent progression.

- IOP is the most important modifiable risk factor.
- Other factors (age, genetics, myopia, vascular dysregulation, low perfusion pressure, obstructive sleep apnea) also contribute.

CLINICAL FEATURES

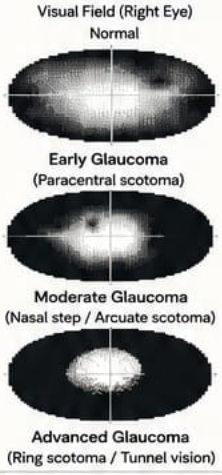
Early (Asymptomatic)

- Usually no pain or redness
- Normal vision
- Detected on routine eye examination

As Disease Progresses

- Peripheral (side) vision loss occurs first
- Difficulty in dim light
- Glare and haloes around lights
- Advance: Tunnel vision
- Late: Central vision may be affected

KEY POINT:
Peripheral visual field loss precedes central vision loss.



Optic Disc Changes (Cup-Disc Ratio)

Physiologic Cupping (C/D < 0.3) | Mild Glaucomatous Cupping (C/D 0.3 – 0.5) | Moderate Cupping (C/D 0.5 – 0.7) | Advanced Cupping (C/D > 0.7)

C/D = Cup to Disc Ratio

Other Important Clinical Points

- IOP may be high, normal, or rarely low.
- Examine optic nerve head, RNFL, and macula carefully.
- Check for relative afferent pupillary defect (RAPD) in advanced disease.

INVESTIGATIONS

	**Tonometry (Goldmann Applanation)**	Measures IOP (Normal: 10 – 21 mmHg; glaucoma can occur at any IOP)
	**Gonioscopy**	Open anterior chamber angle with visible trabecular meshwork (essential to confirm open angle)
	**Fundus Examination**	Increased cup-disc ratio, neuroretinal rim thinning, optic disc hemorrhage, RNFL defects
	**Perimetry (Visual Field Test)**	Detects functional loss – early changes in peripheral field (e.g. nasal step, arcuate scotoma)
	**OCT RNFL & Ganglion Cell Analysis**	Retinal nerve fiber layer thinning, ganglion cell complex loss
	**Optic Nerve Head Photography**	Baseline documentation and serial monitoring of optic disc
	**Pachymetry**	Central corneal thickness (affects IOP measurement interpretation)
	**Diurnal IOP Curve**	IOP measured at different times of the day (in selected cases)

TREATMENT

1. MEDICAL TREATMENT (First Line)

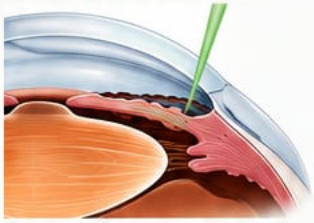
Prostaglandin analogues are FIRST LINE therapy
(Most effective IOP lowering)

Class	Examples	Mechanism	Frequency
Prostaglandin analogues	Latanoprost, Travoprost, Bimatoprost, Tafluprost	↑ Uveoscleral outflow	Once daily (at night)
Beta blockers	Timolol	↓ Aqueous production	1–2 times daily
Alpha-2 agonists	Brimonidine	↓ Production & ↑ Outflow	2–3 times daily
Carbonic anhydrase inhibitors	Dorzolamide (topical) Acetazolamide (oral)	↓ Aqueous production	2–3 times daily (oral as advised)
Rho kinase inhibitor	Netarsudil	↑ Trabecular outflow	Once daily

Adherence to medication and regular follow-up are crucial.

2. LASER TREATMENT

Selective Laser Trabeculoplasty (SLT)

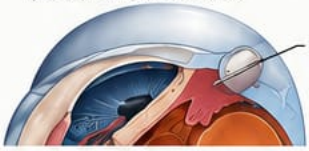


- Laser applied to trabecular meshwork to improve aqueous outflow and reduce IOP.
- Outpatient procedure. Repeatable.
- Consider as primary or adjunctive therapy.

3. SURGICAL TREATMENT

Indications: Uncontrolled IOP despite maximum medical/laser therapy

- **Trabeculectomy** (Gold standard filtering surgery)
- **Glaucoma drainage devices** (e.g. Baerveldt, Ahmed valve)
- **Minimally Invasive Glaucoma Surgery (MIGS)** – in selected cases



General Measures: Regular follow-up | Lifestyle modification | Control systemic risk factors

EARLY DETECTION • REGULAR FOLLOW-UP • IOP CONTROL = PREVENTION OF BLINDNESS

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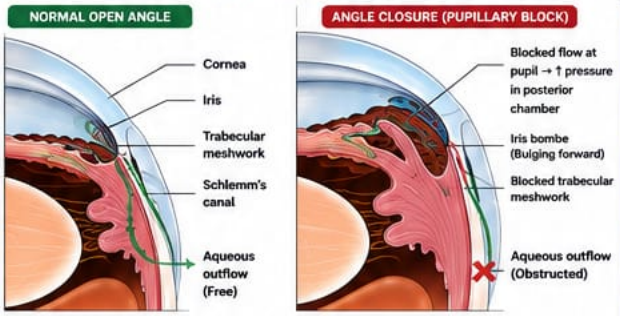
ANGLE-CLOSURE GLAUCOMA

An Ophthalmic Emergency – Early Detection, Prompt Treatment Saves Sight

DEFINITION

Angle-closure glaucoma is caused by closure or obstruction of the anterior chamber angle, leading to impaired aqueous outflow, sudden or progressive rise in intraocular pressure (IOP), optic nerve damage and visual field loss.

NORMAL vs ANGLE CLOSURE



PATHOPHYSIOLOGY: PUPILLARY BLOCK

1. Aqueous produced in posterior chamber
2. Resistance at pupil (prevents flow to anterior chamber)
3. Pressure builds up in posterior chamber
4. Peripheral iris bows forward (iris bombe)
5. Angle (trabecular meshwork) becomes obstructed
6. Sudden rise in IOP → Optic nerve ischemia & damage

RISK FACTORS

- Hypermetropia (short axial length)
- Elderly age (lens enlargement)
- Female sex
- Asian ethnicity
- Family history
- Shallow anterior chamber
- Cataract / Intumescent lens
- Dim illumination
- Anticholinergic / sympathomimetic drugs (cause mydriasis)

CLASSIFICATION

1. PRIMARY ANGLE CLOSURE DISEASE (Modern Classification)

Stage	Description
PACS	Primary Angle Closure Suspect Narrow occludable angle without raised IOP, PAS or optic nerve damage
PAC	Primary Angle Closure Narrow angle with raised IOP and/or PAS, but no glaucomatous optic neuropathy
PACG	Primary Angle-Closure Glaucoma Angle closure + glaucomatous optic disc and visual field damage

2. MECHANISM-BASED

- Pupillary block (most common)
- Plateau iris
- Lens-induced crowding

3. SECONDARY ANGLE CLOSURE GLAUCOMA

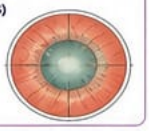
- Neovascular glaucoma
- Uveitis
- Lens subluxation
- Intumescent cataract
- Tumors
- Posterior synechiae
- Drug-induced angle closure

PRECIPITATING FACTORS (ACUTE ATTACK)

- Dark environment
- Emotional stress
- Reading in dim light
- Anticholinergic drugs
- Sympathomimetics / Mydriatic drops

CHRONIC ANGLE CLOSURE

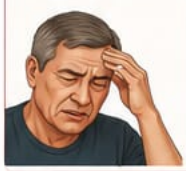
- Gradual peripheral anterior synechiae (PAS) formation → Permanent angle closure
- Often asymptomatic initially
- Gradual peripheral visual field loss
- Progressive optic disc cupping



ACUTE ANGLE-CLOSURE GLAUCOMA

SYMPTOMS

- Sudden severe ocular pain
- Blurring of vision
- Halos around lights
- Frontal headache
- Nausea & vomiting
- Photophobia
- Rapid diminution of vision

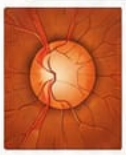


SIGNS

Conjunctiva	Circumcorneal congestion
Cornea	Steamy, edematous
Pupil	Mid-dilated, vertically oval, fixed
Anterior chamber	Shallow
IOP	Markedly elevated (40 – 70 mmHg)
Eyeball	Stony hard
Vision	Reduced
Gonioscopy	Closed / narrow angle

Closed angle on gonioscopy

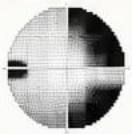
OPTIC DISC CHANGES



- Increased cup-disc ratio
- Vertical cupping
- Neuroretinal rim thinning
- Optic disc pallor in advanced disease

VISUAL FIELD DEFECTS

- Paracentral scotoma
- Nasal step
- Arcuate scotoma
- Ring scotoma
- Tunnel vision



INVESTIGATIONS

Tonometry	Raised IOP
Gonioscopy	Confirms narrow / closed angle
Slit lamp examination	Shallow anterior chamber
Van Herick grading	Peripheral anterior chamber depth assessment
Fundoscopy	Optic disc cupping
Perimetry	Visual field defects
OCT RNFL	Retinal nerve fiber layer thinning
Pachymetry	Central corneal thickness
Optic nerve photography	Serial documentation

MANAGEMENT

AACG vs ACUTE IRIDOCYCLITIS

Feature	AACG	Acute Iridocyclitis
Pain	Severe	Moderate
Pupil	Mid-dilated, fixed	Constricted
Cornea	Edematous	Usually clear
IOP	Raised	Low / Normal
Anterior chamber	Shallow	Normal
Conjunctival injection	Circumcorneal	Ciliary / Sectoral
Vision	Markedly reduced	Mild – Moderate

ACUTE ATTACK – OPHTHALMIC EMERGENCY

1. REDUCE AQUEOUS PRODUCTION

- Inj. Acetazolamide 500 mg IV / Oral
- Timolol eye drops
- Brimonidine eye drops

2. REDUCE VITREOUS VOLUME

- Inj. Mannitol 20% IV (1–2 g / kg)
- Oral Glycerol (1–1.5 g / kg)

3. MIOTIC THERAPY (After IOP begins to reduce)

- Pilocarpine 2% eye drops

Pilocarpine is initially ineffective in very high IOP due to ischemic paralysis of iris sphincter.

4. CONTROL INFLAMMATION AND PAIN

- Topical steroids
- Analgesics
- Antiemetics

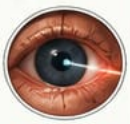
DEFINITIVE TREATMENT

LASER PERIPHERAL IRIDOTOMY (LPI)

Creates an alternative pathway for aqueous flow between posterior and anterior chambers.

INDICATIONS

- Affected eye
- Fellow eye prophylactically



SURGICAL TREATMENT (If laser fails or chronic damage exists)

Trabeculectomy

Glaucoma drainage devices (e.g., Ahmed / Baerveldt)

Lens extraction / Phacoemulsification

ROLE OF EARLY LENS EXTRACTION

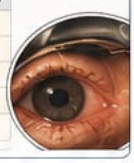
- Increasingly preferred in selected PAC / PACG patients
- Deepens anterior chamber
- Widens angle
- Reduces IOP & medication dependence

COMPLICATIONS

- Optic atrophy
- Permanent blindness
- Peripheral anterior synechiae
- Cataract
- Chronic glaucoma

GONIOSCOPY (SHAFFER GRADING)

Grade	Angle Width	Closure Risk
4	Wide open	None
3	Open	Low
2	Narrow	Possible
1	Extremely narrow	High
0	Closed	Closed



IMPORTANT POINTS (HIGH-YIELD)

- Most common mechanism: Pupillary block
- Hypermetropia predisposes to angle closure
- Definitive treatment: Laser peripheral iridotomy (LPI)
- Fellow eye must be treated prophylactically
- Halos occur due to corneal edema
- PAS causes permanent synechial closure
- Plateau iris can cause angle closure despite patent iridotomy
- Avoid mydriatic drugs in narrow-angle patients before angle assessment

OPEN-ANGLE vs ANGLE-CLOSURE (KEY DIFFERENCES)

Feature	Open-Angle Glaucoma	Angle-Closure Glaucoma
Onset	Gradual	Sudden
Pain	Absent	Severe
Redness	Minimal	Marked
Cornea	Clear	Edematous
Pupil	Normal	Mid-dilated, fixed
Angle	Open	Closed
IOP rise	Gradual	Sudden, marked

EARLY DETECTION • PROMPT TREATMENT • REGULAR FOLLOW-UP = PREVENTION OF BLINDNESS

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CATARACT

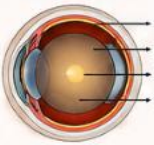
Essay Writing – Complete Guide

DEFINITION

Cataract is defined as any opacity of the crystalline lens or its capsule causing impairment of vision.

It is the commonest cause of reversible blindness worldwide and a major cause of avoidable blindness in India.

ANATOMY OF LENS



FUNCTIONS
- Refraction
- Accommodation
- Maintenance of optical clarity

ETIOLOGY / CAUSES

1. **Age-related (Senile)** – Most common
2. **Congenital** – TORCH infections, hereditary, metabolic diseases, chromosomal abnormalities
3. **Traumatic** – Blunt trauma, penetrating injury, radiation, electric shock
4. **Complicated** – Secondary to uveitis, glaucoma, high myopia, retinal detachment
5. **Metabolic** – Diabetes mellitus, galactosemia, hypocalcemia, Wilson disease
6. **Drug-induced** – Corticosteroids, chlorpromazine, miotics

PATHOGENESIS OF SENILE CATARACT

- Oxidative damage to lens proteins
- Protein denaturation and aggregation
- Hydration changes in lens fibers
- Loss of lens transparency
- Accumulation of chromophores

RISK FACTORS
- Increasing age
- Smoking
- Steroid therapy
- UV radiation exposure
- Diabetes mellitus
- Malnutrition

MORPHOLOGICAL CLASSIFICATION OF SENILE CATARACT

1. **Nuclear Cataract**
   Central nuclear sclerosis
   Yellow/brown discoloration
   Causes myopic shift ("second sight")

2. **Cortical Cataract**
   Peripheral spoke-like opacities
   Cuneiform pattern

3. **Posterior Subcapsular Cataract**
   Opacity beneath posterior capsule
   Causes marked glare and near vision difficulty

4. **Mixed Cataract**
   Combination of different types

STAGES OF SENILE CATARACT

1. Incipient Cataract

- Early lens opacity
- Minimal visual impairment

2. Immature Cataract

- Lens partially opaque
- Iris shadow present

3. Mature Cataract

- Entire cortex opaque
- Iris shadow absent

4. Hypermature Cataract

- Cortex liquefied
- Wrinkling of capsule
- Nucleus may sink (Morgagnian cataract)

SYMPTOMS

- Progressive painless diminution of vision
- Glare
- Halos around lights
- Monocular diplopia / polyopia
- Frequent change of glasses
- Difficulty in night driving

In Nuclear Cataract
Temporary improvement in near vision – "Second sight"

SIGNS

- Grey/white pupillary reflex
- Diminished visual acuity
- Dull red reflex
- Lens opacities seen on slit lamp

IRIS SHADOW TEST

Positive Iris Shadow → Immature cataract	Negative Iris Shadow → Mature cataract
	

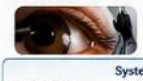
DIFFERENTIAL DIAGNOSIS OF LEUKOCORIA

In Children
- Congenital cataract
- Retinoblastoma
- Retinopathy of prematurity
- Persistent hyperplastic primary vitreous

In Adults
- Mature cataract
- Retinal detachment

INVESTIGATIONS

Ocular Evaluation
- Visual acuity
- Slit lamp examination
- Fundus examination



Preoperative Investigations
- Intraocular pressure
- Lacrimal sac syringing
- Keratometry
- A-scan biometry
- IOL power calculation

Systemic Evaluation
- Blood sugar
- Blood pressure
- ECG in elderly patients

COMPLICATIONS OF CATARACT

1. **Lens-induced Glaucoma**
   - **Phacomorphic glaucoma** – Intumescent swollen lens causing secondary angle-closure glaucoma
   - **Phacolytic glaucoma** – Leakage of lens proteins causing secondary open-angle glaucoma
2. **Lens-induced Uveitis** – Inflammatory reaction to lens proteins
3. **Subluxation / Dislocation of Lens**
4. **Blindness**

INDICATIONS FOR CATARACT SURGERY

- Visual disability affecting daily activities
- Lens-induced glaucoma
- Need for fundus visualization
- Traumatic cataract
- Cosmetic indications

CONTRAINDICATIONS / POOR PROGNOSIS
- Severe retinal disease
- Optic atrophy
- Advanced glaucoma with poor visual potential

TYPES OF CATARACT SURGERY

1. ICCE
(Intracapsular Cataract Extraction)

Entire lens with capsule removed.

Disadvantages
- Vitreous loss
- Retinal detachment
- Aphakia

2. ECCE
(Extracapsular Cataract Extraction)

Nucleus removed; posterior capsule retained.

3. SICS
(Small Incision Cataract Surgery)

Self-sealing scleral tunnel incision.

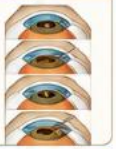
Widely practiced in India.

4. PHACOEMULSIFICATION
(Current Gold Standard)

Ultrasonic emulsification of nucleus followed by aspiration.

STEPS OF PHACOEMULSIFICATION

1. Clear corneal incision
2. Continuous curvilinear capsulorrhexis
3. Hydrodissection
4. Phacoemulsification of nucleus
5. Cortical aspiration
6. Intraocular lens implantation



INTRAOCULAR LENS (IOL)

Inserted after lens extraction.

TYPES
- PMMA rigid lens
- Foldable acrylic lens
- Multifocal lens
- Toric lens

APHAKIA

Definition: Absence of crystalline lens.

Causes: Postoperative / Congenital / Traumatic

Features:
- High hypermetropia
- Deep anterior chamber
- Iridodonesis

Correction: PCIOL / Contact lens / Aphakic spectacles

COMPLICATIONS OF CATARACT SURGERY

Intraoperative	Early Postoperative	Late Postoperative
• Posterior capsular rupture • Vitreous loss • Zonular dialysis	• Corneal edema • Raised IOP • Hyphema • Endophthalmitis	• Posterior capsular opacification (PCO) • Cystoid macular edema

ENDOPHTHALMITIS (Early Postoperative)

Features: Pain, redness, hypopyon, sudden severe visual loss

Treatment: Intravitreal antibiotics ± Vitrectomy

POSTERIOR CAPSULAR OPACIFICATION

Most common late complication.

Treatment:
Nd:YAG laser capsulotomy

CONGENITAL CATARACT

Types: Lamellar • Zonular • Polar • Sutural • Total

Causes: Rubella • Galactosemia • Down syndrome • Others

Importance: Early surgery is essential to prevent amblyopia.

MODERN ADVANCES

- Femtosecond laser-assisted cataract surgery (FLACS)
- Microincision cataract surgery (MICS)
- Premium IOLs – Multifocal, Toric, Accommodative lenses

KEY TAKE HOME MESSAGE

Cataract is the leading cause of reversible blindness. Senile cataract is the most common type and presents with progressive painless visual loss. Diagnosis is clinical. Surgical extraction with IOL implantation remains the definitive treatment, with phacoemulsification being the present gold standard.

VIVA QUESTIONS – QUICK REVISION

1. Commonest cause of cataract?	– Senile degeneration
2. Commonest type of senile cataract?	– Nuclear sclerosis
3. What is second sight?	– Temporary improvement of near vision due to myopia from nuclear sclerosis
4. Most common surgery today?	– Phacoemulsification with PCIOL implantation
5. Most common late postoperative complication?	– Posterior capsular opacification
6. What is Morgagnian cataract?	– Hypermature cataract with liquefied cortex and sunken nucleus.
7. What is phacomorphic glaucoma?	– Angle closure due to swollen lens.
8. What is phacolytic glaucoma?	– Open-angle glaucoma due to leakage of lens proteins.
9. Most important postoperative emergency?	– Endophthalmitis

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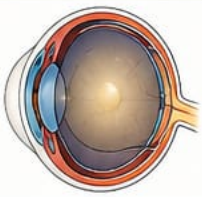
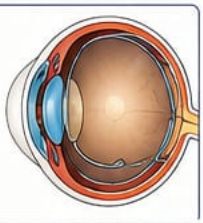


CATARACT SURGERY

Essay Writing – Complete Guide



DEFINITION : Cataract is any opacity of the crystalline lens or its capsule causing impairment of vision. Surgical removal of the opaque lens with implantation of intraocular lens (IOL) restores vision.

STEPS OF PHACOEMULSIFICATION CATARACT SURGERY (Gold Standard)

1 Anaesthesia
Topical (most common) / Peribulbar / Rarely retrobulbar. Eye is cleaned, painted and draped.

2 Main Corneal Incision
Small self-sealing clear corneal incision (2.2–3 mm) made, usually temporal.

3 Side Port Incision
Auxiliary small incision for instrument entry.

4 Entry into Anterior Chamber
Anterior chamber is carefully entered through main incision.

5 Injection of Viscoelastic
Maintains anterior chamber depth, protects corneal endothelium and facilitates intraocular maneuvers.

6 Capsulorrhexis (CCC)
Continuous curvilinear capsulorrhexis made in anterior capsule. Ideal size 5–5.5 mm and should overlap IOL optic all around. (One of the most critical steps)

7 Hydrodissection
Balanced salt solution injected between cortex and capsule to separate cortex from capsule and mobilize the nucleus.

8 Nucleus Rotation
Nucleus rotated within capsular bag. Confirms adequate hydrodissection.

9 Hydrodelineation
Fluid injected within lens substance to separate nucleus from epinucleus.

10 Phacoemulsification of Nucleus
Ultrasonic energy emulsifies the nucleus and the fragments are aspirated.
Techniques: Divide & conquer, Stop & chop, Phaco chop.

11 Cortical Aspiration
Residual cortical matter removed using Irrigation-Aspiration (I/A) cannula.

12 Capsular Bag Inflation
Viscoelastic reinjected into capsular bag to maintain space and facilitate IOL insertion.

13 PCIOL Implantation (Posterior Chamber IOL)
Foldable IOL inserted into capsular bag (In-the-bag implantation – standard). If posterior capsule ruptures, sulcus placement may be done.

14 Removal of Viscoelastic
Complete aspiration of viscoelastic performed to prevent postoperative rise in IOP.

15 Stromal Hydration of Wound
Corneal stromal hydration done to seal incision and maintain watertight closure. Usually no sutures needed.

16 Intracameral Antibiotic Injection
Moxifloxacin / Cefuroxime injected into anterior chamber to reduce risk of postoperative endophthalmitis.

17 Eye Patching / Shielding
Antibiotic–steroid drops instilled. Eye shield applied.

FLOW CHART – PHACOEMULSIFICATION

Anaesthesia
↓
Corneal incision
↓
Side port incision
↓
AC entry
↓
Viscoelastic injection
↓
Capsulorrhexis
↓
Hydrodissection
↓
Nucleus rotation
↓
Hydrodelineation
↓
Phacoemulsification
↓
Cortical aspiration
↓
PCIOL implantation
↓
Viscoelastic removal
↓
Stromal hydration
↓
Intracameral antibiotic
↓
Eye shield

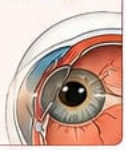
IMPORTANT INTRAOPERATIVE COMPLICATION

POSTERIOR CAPSULAR RUPTURE (PCR)

Most feared complication.

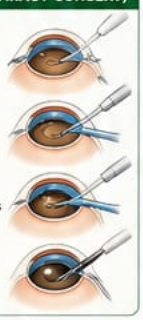
May lead to:

- Vitreous loss
- Dropped nucleus
- Need for anterior vitrectomy
- Sulcus IOL implantation



STEPS OF SICS (SMALL INCISION CATARACT SURGERY)

1. Peribulbar anaesthesia
2. Conjunctival peritomy
3. Scleral tunnel incision
4. Entry into anterior chamber
5. Capsulotomy / Capsulorrhexis
6. Hydrodissection
7. Nucleus prolapse into anterior chamber
8. Viscoexpression / Hydroexpression of nucleus
9. Cortical aspiration
10. PCIOL implantation
11. Wound closure



DIFFERENCE BETWEEN PHACO AND SICS

FEATURE	PHACO	SICS
Incision	Small (2.2–3 mm)	Large (6–7 mm scleral tunnel)
Nucleus removal	Ultrasonic emulsification (Phaco probe)	Manual delivery (Viscoexpression)
Astigmatism	Less	More
Recovery	Faster	Slightly slower
Equipment	Expensive	Less expensive
Cost	Higher	Lower
Wound	Self-sealing	May need sutures

MODERN ADVANCES

- Femtosecond Laser-Assisted Cataract Surgery (FLACS)
  – Laser for corneal incisions, capsulotomy, lens fragmentation
- Microincision Cataract Surgery (MICS)
- Premium IOLs
  – Multifocal IOLs
  – Toric IOLs (for astigmatism)
  – Accommodative IOLs
- Foldable IOLs – allow insertion through very small incision
- Improved viscoelastics, phaco machines and blades
- Intracameral antibiotics – reduce endophthalmitis

INDICATIONS FOR CATARACT SURGERY

- Visual disability affecting daily activities
- Lens-induced glaucoma
- Need for fundus visualization
- Traumatic cataract
- Cosmetic reasons

COMPLICATIONS OF CATARACT SURGERY

INTRAOPERATIVE	EARLY POSTOPERATIVE	LATE POSTOPERATIVE
• Posterior capsular rupture	• Corneal edema	• Posterior capsular opacification
• Vitreous loss	• Raised IOP	• Cystoid macular edema
• Zonular dialysis	• Hyphema	• IOL decentration
• Dropped nucleus	• Endophthalmitis	• Secondary glaucoma
• Suprachoroidal haemorrhage	• Wound leak	• Retinal detachment

ENDOPHTHALMITIS (POSTOPERATIVE)

Features:

- Pain, redness, photophobia
- Decreased vision
- Hypopyon

Treatment:

- Intravitreal antibiotics
- Pars plana vitrectomy (if severe)

APHAKIA

Definition: Absence of crystalline lens.

Causes: Postoperative / Congenital / Traumatic

Features: High hypermetropia, deep anterior chamber, iridodonesis, nystagmus

Correction: PCIOL / Contact lens / Aphakic spectacles

DIFFERENTIAL DIAGNOSIS OF LEUKOCORIA

In Children

- Congenital cataract
- Retinoblastoma
- Retinopathy of prematurity
- Persistent hyperplastic primary vitreous

In Adults

- Mature cataract
- Retinal detachment

KEY POINTS / VIVA PEARLS

- ✓ Most important step – Capsulorrhexis
- ✓ Hydrodissection separates cortex from capsule.
- ✓ Hydrodelineation separates nucleus from epinucleus.
- ✓ Standard IOL placement – In-the-bag PCIOL.
- ✓ Most common surgery today – Phacoemulsification with foldable PCIOL.
- ✓ Most feared intraoperative complication – Posterior capsular rupture.
- ✓ Most common late complication – Posterior capsular opacification.
- ✓ Most serious postoperative complication – Endophthalmitis.

WHO MESSAGE

Cataract is the leading cause of reversible blindness worldwide. Early diagnosis and timely surgery restore vision and improve quality of life.

SUMMARY

Cataract surgery is safe, effective and one of the most successful surgeries in medicine. Phacoemulsification with intraocular lens implantation is the present gold standard.

PREOPERATIVE EVALUATION (Essentials)

- Detailed ocular examination
- IOP measurement
- Keratometry & A-scan biometry
- IOL power calculation
- Systemic evaluation – BP, Blood sugar, ECG (elderly)

Dr Ashik Sureshkumar

STRABISMUS

ESSAY WRITING – COMPLETE GUIDE

DEFINITION

Strabismus (squint) is defined as a condition in which the visual axes of the two eyes are not aligned simultaneously toward the object of fixation.

ORTHOPHORIA

Normal alignment of both eyes maintained by fusion mechanisms.

NORMAL BINOCULAR SINGLE VISION

- Clear vision in both eyes
- Normal retinal correspondence
- Coordinated ocular movements
- Intact visual pathways and cortex

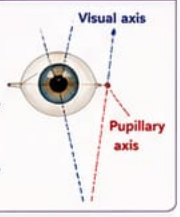
GRADES OF BINOCULAR SINGLE VISION

GRADE I	GRADE II	GRADE III
Simultaneous Perception	Fusion	Stereopsis
Ability to perceive two images simultaneously.	Ability to combine two images into one.	Depth perception due to binocular disparity. Highest grade of binocular vision.

ANGLE KAPPA

Angle between visual axis and pupillary axis.

- Positive angle kappa gives false appearance of exotropia.
- Negative angle kappa gives false appearance of esotropia.



CLASSIFICATION OF STRABISMUS

A. BASED ON MANIFESTATION

1. Heterophoria (Latent Squint)

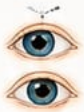
Deviation controlled by fusion. Appears only when binocular vision is interrupted.

- Esophoria
- Exophoria
- Hyperphoria

2. Heterotropia (Manifest Squint)

Deviation present even with both eyes open.

- Esotropia
- Exotropia
- Hypertropia
- Hypotropia



B. BASED ON DIRECTION

Horizontal	Vertical	Torsional
Esotropia	Hypertropia	Cyclotropia
Exotropia	Hypotropia	–

C. BASED ON OCULAR MOVEMENTS

1. Concomitant (Non-paralytic) Squint

- Angle of deviation same in all directions
- Ocular movements full
- Usually childhood onset

2. Incomitant (Paralytic) Squint

- Angle varies with direction of gaze
- Ocular movement restricted
- Diplopia common
- Usually acquired

DIFFERENCE BETWEEN CONCOMITANT AND PARALYTIC SQUINT

Feature	Concomitant (Non-paralytic)	Paralytic (Incomitant)
Age	Usually children	Usually adults
Diplopia	Usually absent	Present
Ocular movements	Full	Restricted
Angle of deviation	Constant in all directions	Variable in different gazes
Abnormal head posture	Rare	Common
Secondary deviation	Equal to primary	Greater than primary

PSEUDOSTRABISMUS

False appearance of squint without true ocular deviation.

Causes

- Epicanthal folds
- Flat nasal bridge

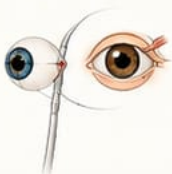
Features

- Symmetrical corneal reflexes
- Normal cover test

ETIOLOGY

A. Causes of Concomitant Squint

- Refractive errors
- Congenital causes
- Hereditary factors
- Poor vision in one eye
- Developmental defects



B. Causes of Paralytic Squint

- III, IV, VI nerve palsy
- Trauma
- Diabetes mellitus
- Hypertension
- Intracranial tumors
- Myasthenia gravis
- Thyroid eye disease

TYPES OF CONCOMITANT SQUINT

1. ESOTROPIA

(Most common in children)

Types

- Infantile esotropia
- Accommodative esotropia
- Non-accommodative esotropia

Accommodative Esotropia

Occurs due to hypermetropia with excessive accommodation and convergence.

AC:A RATIO

Amount of accommodative convergence produced per diopter of accommodation. Increased in accommodative esotropia.

Treatment

- Full cycloplegic correction
- Bifocal glasses if needed

2. EXOTROPIA

- Intermittent exotropia common
- Increases during fatigue or inattention

SPECIAL TYPES OF SQUINT

A. Convergence Insufficiency
Exodeviation greater at near fixation.

B. Divergence Excess
Exotropia greater for distance.

C. Microtropia
Very small-angle squint.

D. Restrictive Strabismus
Occurs due to mechanical restriction.
Causes: Thyroid eye disease, Blowout fracture.

SENSORY ADAPTATIONS (IN CHILDHOOD)

1. Suppression

Brain suppresses image from deviated eye to avoid diplopia.

2. Amblyopia

Reduced visual acuity without organic lesion due to abnormal visual development. Most important complication.

3. Abnormal Retinal Correspondence (ARC)

Adaptation where non-corresponding retinal points acquire sensory correspondence.

MOTOR ADAPTATION

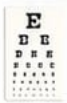
Abnormal Head Posture (AHP)

Patient adopts compensatory posture to maintain binocular single vision.

- Face turn
- Head tilt
- Chin elevation / depression

INVESTIGATIONS

1. Visual Acuity Assessment



2. Cycloplegic Refraction

3. Hirschberg Test

Corneal light reflex.

Normal: Light reflex slightly nasal.

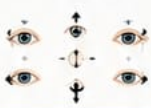
4. Cover Tests

- Cover-Uncover Test
  Detects manifest squint.
- Alternate Cover Test
  Detects latent squint.

5. Prism Bar Cover Test

Measures angle of deviation.

6. Ocular Movement Examination



7. Synoptophore

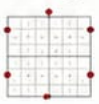
Assesses fusion, binocular vision and retinal correspondence.

8. Hess Chart

Used in paralytic squint to identify affected muscle.

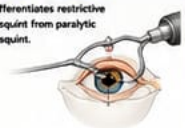
9. Diplopia Charting

Maps separation of double images.



10. Forced Duction Test

Differentiates restrictive squint from paralytic squint.



CRANIAL NERVE PALSY

1. THIRD NERVE PALSY

- Ptosis
- Dilated pupil
- "Down and out" eye

2. FOURTH NERVE PALSY

- Vertical diplopia
- Head tilt
- Superior oblique weakness

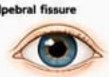
3. SIXTH NERVE PALSY

- Failure of abduction
- Horizontal diplopia
- Esotropia
  (Most common ocular motor nerve palsy)

ASSOCIATED SYNDROMES

A. Duane Retraction Syndrome

- Limited abduction
- Globe retraction on adduction
- Narrowing of palpebral fissure



B. Brown Syndrome

- Restriction of elevation in adduction due to superior oblique tendon abnormality.

MANAGEMENT

A. TREATMENT OF CONCOMITANT SQUINT

1. Correction of Refractive Errors

Glasses prescribed after cycloplegic refraction.

2. Amblyopia Therapy

Occlusion Therapy

Patching of normal eye to stimulate amblyopic eye.

Best results before 7–8 years.

4. Botulinum Toxin

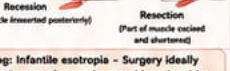
- Useful in sixth nerve palsy and small-angle deviations.

Tminlgce Exercant

5. Surgery

Principles: Weak muscle strengthened by resection; Strong muscle weakened by recession.

Weakening Procedures	Strengthening Procedures
• Recession	• Resection
• Tenotomy	• Advancement
• Myotomy	• Tucking



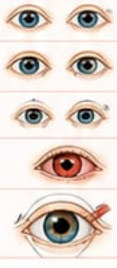
Timing: Infantile esotropia – Surgery ideally before 2 years of age to improve binocular vision.

B. TREATMENT OF PARALYTIC SQUINT

- Treat underlying cause
- Prism therapy
- Occlusion for diplopia
- Botulinum toxin
- Surgery after stabilization

COMPLICATIONS OF SQUINT SURGERY

- ✔ Overcorrection
- ✔ Undercorrection
- ✔ Diplopia
- ✔ Infection
- ✔ Slipped muscle



VIVA QUESTIONS

1. Most common childhood squint? Esotropia.
2. Most important complication? Amblyopia.
3. Best age for amblyopia treatment? Before 7 years.
4. Most common ocular motor nerve palsy? Sixth nerve palsy.
5. What is pseudostrabismus? False appearance of squint without true ocular deviation.
6. What is AC:A ratio? Amount of accommodative convergence per diopter.
7. What is suppression? Brain suppresses image from deviated eye.
8. What is ARC? False sensory correspondence between non-corresponding retinal points.

KEY TAKE HOME MESSAGE

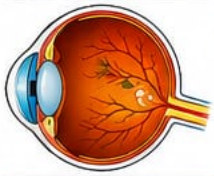
Strabismus is a disorder of ocular alignment that may lead to amblyopia, suppression and loss of binocular vision if untreated. Early diagnosis, correction of refractive errors, amblyopia therapy, orthoptic exercises and timely surgical intervention are essential for good visual and cosmetic outcome.

BINOCULAR VISION PYRAMID

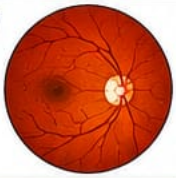
IMPORTANT POINTS

- ☑ Early detection is vital.
- ☑ Treat amblyopia before 7–8 years.
- ☑ Full cycloplegic refraction in all children.
- ☑ Surgery at appropriate time gives best result.
- ☑ Aim: Restore binocular single vision and good cosmesis.

Dr Ashik Suresh Kumar



HYPERTENSIVE RETINOPATHY



Retinal vascular changes secondary to systemic hypertension characterized by arteriolar narrowing, vascular sclerosis, hemorrhages, exudates and disc edema.

DEFINITION

Hypertensive retinopathy is a spectrum of retinal vascular and retinal parenchymal changes occurring due to systemic arterial hypertension.

ETIOPATHOGENESIS

- Vasospasm
- Endothelial damage
- Increased vascular permeability
- Arteriolar sclerosis
- Retinal ischemia

PATHOPHYSIOLOGY

A. ACUTE HYPERTENSION
- Vasoconstriction
- Breakdown of blood-retinal barrier
- Retinal edema

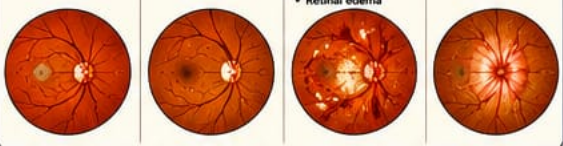
B. CHRONIC HYPERTENSION
- Intimal thickening
- Hyaline degeneration
- Medial hyperplasia
- Arteriolosclerosis → Narrowed lumen → Reduced retinal perfusion

C. MALIGNANT HYPERTENSION
- Fibrinoid necrosis
- Capillary leakage
- Retinal ischemia
- Optic disc edema (BP usually >180/120 mmHg)

CLASSIFICATION

1. KEITH–WAGENER–BARKER CLASSIFICATION

GRADE 1	GRADE 2	GRADE 3	GRADE 4
• Mild generalized arteriolar narrowing • Increased arteriolar light reflex	• More severe narrowing • AV crossing changes • AV nicking	• Features of grade 2 plus: • Flame hemorrhages • Cotton wool spots • Hard exudates • Retinal edema	• Features of grade 3 plus: • Papilledema • Optic disc edema (Malignant HTN)



2. SCHEIE CLASSIFICATION

Hypertensive Changes	H0 – H4
Arteriolosclerotic Changes	S0 – S4

3. WONG & MITCHELL CLASSIFICATION

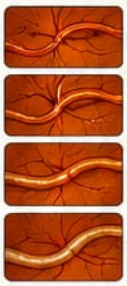
MILD	MODERATE	SEVERE
• Arteriolar narrowing • AV nicking	• Hemorrhages • Exudates • Cotton wool spots	• Optic disc swelling

ANGIOPATHY vs ARTERIOLOSCLEROSIS

FEATURE	HYPERTENSIVE ANGIOPATHY	HYPERTENSIVE ARTERIOLOSCLEROSIS
Cause	Acute rise in BP	Chronic hypertension
Main pathology	Vasospasm	Vessel wall thickening and sclerosis
Reversibility	Potentially reversible	Usually permanent
Signs	Narrowing, hemorrhage	Copper / Silver wiring

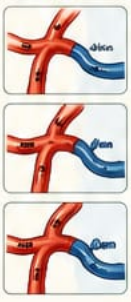
CLINICAL FEATURES / SIGNS

A. VASCULAR CHANGES



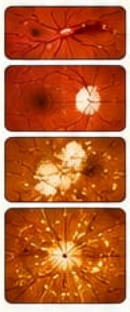
- **Generalized Arteriolar Narrowing**
  - Earliest sign
  - Due to vasospasm
- **Focal Arteriolar Narrowing**
  - Localized vasospasm
- **Copper Wiring**
  - Moderate sclerosis → broad light reflex
- **Silver Wiring**
  - Severe sclerosis → opaque vessel wall

B. ARTERIOVENOUS CROSSING CHANGES



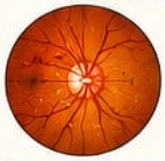
- **GUNN SIGN**
  - Tapering of vein on either side of AV crossing
- **SALUS SIGN**
  - Deflection of vein at AV crossing
- **BONNET SIGN**
  - Venous banking (distension) distal to crossing

C. RETINAL CHANGES



- **Flame-shaped Hemorrhages**
  - In nerve fiber layer
- **Cotton Wool Spots**
  - Nerve fiber layer infarcts
  - Due to retinal ischemia
- **Hard Exudates**
  - Lipid deposition from vascular leakage
- **Macular Star**
  - Radial arrangement of hard exudates around macula (seen in malignant HTN & neuroretinitis)

D. OPTIC DISC CHANGES

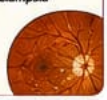


- Papilledema
- Disc edema
- Optic disc pallor in chronic disease

HYPERTENSIVE CHOROIDOPATHY

Seen in malignant hypertension, young patients, preeclampsia / eclampsia

- Elschnig spots
- Siegrist streaks
- Exudative retinal detachment



MALIGNANT HYPERTENSION

Severe hypertension associated with:
- Retinopathy
- Papilledema
- End-organ damage

(BP usually >180/120 mmHg)

PATHOGENESIS

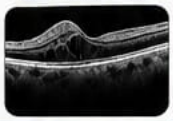
Severe hypertension
↓
Fibrinoid necrosis of arterioles
↓
Breakdown of blood-retinal barrier
↓
Retinal edema & ischemia
↓
Hemorrhages + exudates + disc edema

INVESTIGATIONS

- **Fundus Examination** – Main modality
- **Fundus Photography** – Documentation & follow-up
- **Fluorescein Angiography (FFA)**
  - Delayed retinal filling
  - Arteriolar leakage
  - Disc leakage
  - Choroidal non-perfusion
  - Capillary non-perfusion
- **Optical Coherence Tomography (OCT)**
  - Macular edema
  - Intraretinal fluid
  - Subretinal fluid
  - Serous retinal detachment

FFA – Disc Leakage

OCT – Macular Edema



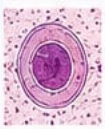
SYSTEMIC EVALUATION

- Blood pressure measurement
- Renal function tests
- Urine protein
- ECG / Echocardiography
- Lipid profile
- Blood sugar

DIFFERENTIAL DIAGNOSIS

- Diabetic retinopathy
- Retinal vein occlusion
- Radiation retinopathy
- Papilledema (raised ICP)
- Neuroretinitis
- Ocular ischemic syndrome

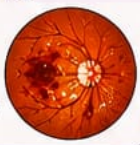
HISTOPATHOLOGY



- Hyaline arteriolosclerosis
- Endothelial damage
- Fibrinoid necrosis
- Narrowed vascular lumen

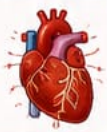
COMPLICATIONS

- Retinal vein occlusion
- Retinal artery occlusion
- Optic neuropathy
- Macular edema
- Exudative retinal detachment
- Visual loss



SYSTEMIC SIGNIFICANCE (PROGNOSTIC IMPORTANCE)

- Reflects increased cardiovascular mortality
- Indicates risk of cerebrovascular disease
- Reflects severity of renal disease
- Indicates systemic end-organ damage
- Grade 3 and 4 changes → Poor prognosis



PREGNANCY-RELATED HYPERTENSIVE RETINOPATHY

Seen in preeclampsia / eclampsia

- Arteriolar spasm
- Retinal edema
- Serous retinal detachment
- Choroidopathy

(Usually improves after delivery)

MANAGEMENT

GENERAL MEASURES
- Antihypertensive therapy
- Salt restriction
- Lifestyle modification
- Smoking cessation
- Diabetes control
- Lipid management

B. MALIGNANT HYPERTENSION

Medical emergency requiring:
- ICU monitoring
- Controlled BP reduction
- Prevention of end-organ ischemia

IMPORTANT PRINCIPLE:
BP should not be reduced too rapidly due to risk of cerebral and optic nerve hypoperfusion.

IV ANTIHYPERTENSIVE DRUGS
- Labetalol
- Nicardipine
- Sodium nitroprusside

C. OCULAR TREATMENT

Usually no specific ocular treatment required.

Treat complications if present:
- Macular edema
- Retinal vein occlusion
- Retinal detachment

IMPORTANT VIVA POINTS

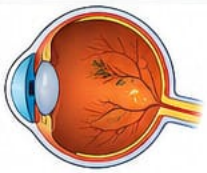
- ★ Earliest sign – Generalized arteriolar narrowing
- ★ Earliest acute change – Vasoconstriction
- ★ Most important sign of malignant HTN – Papilledema
- ★ Copper wiring – Moderate sclerosis
- ★ Silver wiring – Severe sclerosis
- ★ Cotton wool spots – Nerve fiber layer infarcts
- ★ Macular star seen in – Malignant HTN, Neuroretinitis
- ★ Hypertensive choroidopathy – Young pts, malignant HTN, preeclampsia

DIFFERENCE BETWEEN DIABETIC AND HYPERTENSIVE RETINOPATHY

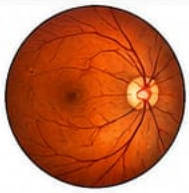
FEATURE	HYPERTENSIVE RETINOPATHY	DIABETIC RETINOPATHY
Primary pathology	Arteriolar sclerosis	Microangiopathy
Earliest sign	Arteriolar narrowing	Microaneurysm
Hemorrhage type	Flame-shaped	Dot-blot
Exudates	Macular star common	Circinate exudates
Neovascularization	Rare	Common
Papilledema	Seen in malignant HTN	Rare

FLOWCHART – PATHOGENESIS

Dr Ashik SureshKumar



DIABETIC RETINOPATHY



DEFINITION

Diabetic retinopathy (DR) is a progressive microangiopathy of the retinal vasculature occurring in patients with diabetes mellitus, characterized by increased vascular permeability, retinal ischemia, capillary occlusion, and retinal neovascularization, leading to visual impairment and blindness.

EPIDEMIOLOGY

One of the leading causes of preventable blindness worldwide.
Risk increases with:

- Duration of diabetes
- Poor glycemic control
- Hypertension
- Dyslipidemia

RISK FACTORS

A. Systemic

- Long duration of diabetes
- Poor glycemic control (high HbA1c)
- Hypertension
- Dyslipidemia
- Diabetic nephropathy
- Pregnancy
- Smoking
- Anemia
- Obesity

B. Ocular

- Cataract surgery
- Retinal vein occlusion

PATHOGENESIS

Chronic Hyperglycemia

- Polyol pathway activation
- Sorbitol accumulation
- Oxidative stress
- Protein kinase C activation
- Advanced glycation end product (AGE) formation

↓

Microvascular Damage

- Pericyte loss
- Basement membrane thickening
- Endothelial dysfunction
- Capillary leakage
- Microaneurysm formation

↓

Retinal Ischemia

- Capillary non-perfusion
- Cotton wool spots

↓

VEGF Release

- Neovascularization
- Increased vascular permeability
- Macular edema

↓

Complications
Vitreous hemorrhage / TRD / NVG / Blindness

DIFFERENTIAL DIAGNOSIS

- Hypertensive retinopathy
- Rotinal vein occlusion
- Radiation retinopathy
- Ocular ischemic syndrome

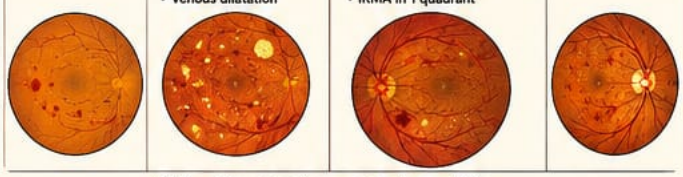
SCREENING RECOMMENDATIONS

- Type 1 DM
  - Screening 5 years after diagnosis
- Type 2 DM
  - Screening at diagnosis
- Pregnancy
  - Before conception or during first trimester

CLASSIFICATION (ETDRS)

1. NON-PROLIFERATIVE DIABETIC RETINOPATHY (NPDR)

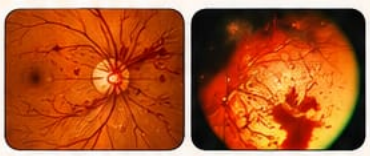
Mild NPDR	Moderate NPDR	Severe NPDR (4-2-1 Rule)	Very Severe NPDR
Microaneurysms only	More than microaneurysms but less than severe NPDR • Dot-blot hemorrhages • Hard exudates • Cotton wool spots • Venous dilatation	Presence of any one: • Hemorrhages/microaneurysms in 4 quadrants • Venous beading in 2 quadrants • IRMA in 1 quadrant	Any two features of severe NPDR



IRMA – Intraretinal Microvascular Abnormalities

2. PROLIFERATIVE DIABETIC RETINOPATHY (PDR)

- Neovascularization at disc (NVD)
- Neovascularization elsewhere (NVE)
- Preretinal hemorrhage
- Vitreous hemorrhage
- Fibrovascular proliferation
- Tractional retinal detachment



HIGH-RISK PDR CHARACTERISTICS

- NVD ≥ 1/3 disc area
- Any NVD with vitreous/preretinal hemorrhage
- NVE ≥ 1/2 disc area with hemorrhage

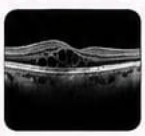
→ These require urgent panretinal photocoagulation.

DIABETIC MACULAR EDEMA (DME)

May occur at any stage of DR.

Types

- Focal
- Diffuse
- Ischemic
- Cystoid macular edema



CLINICALLY SIGNIFICANT MACULAR EDEMA (CSME) (ETDRS Criteria)

Presence of any one:

- Retinal thickening within 500 µm of fovea
- Hard exudates within 500 µm of fovea with adjacent retinal thickening
- Retinal thickening ≥ 1 disc area within 1 disc diameter of fovea

CLINICAL FEATURES

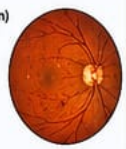
Symptoms

- Asymptomatic initially
- Blurring of vision
- Floaters
- Distorted vision (metamorphopsia)
- Sudden painless visual loss
- Difficulty in reading
- Loss of contrast sensitivity

Signs

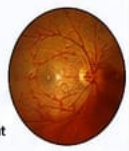
A. NPDR

- Microaneurysms (earliest sign)
- Dot-blot hemorrhages
- Flame-shaped hemorrhages
- Hard exudates
- Cotton wool spots
- Venous beading
- IRMA



B. PDR

- NVD / NVE
- Vitreous hemorrhage
- Preretinal hemorrhage
- Fibrovascular proliferation
- Tractional retinal detachment



C. Advanced Diabetic Eye Disease

- Rubeosis iridis
- Neovascular glaucoma
- Ghost cell glaucoma
- Combined tractional-rhegmatogenous retinal detachment

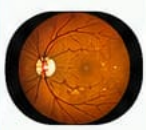
INVESTIGATIONS

Fundus Examination

- Direct ophthalmoscopy
- Indirect ophthalmoscopy
- Slit lamp biomicroscopy with 90D lens

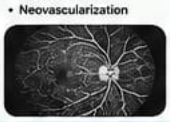
Fundus Photography

- Documentation and grading



Fundus Fluorescein Angiography (FFA)
Shows:

- Capillary non-perfusion
- Microaneurysms
- Leakage
- Macular edema
- Neovascularization

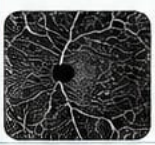


Optical Coherence Tomography (OCT)
Gold standard for macular edema assessment.
Shows:

- Retinal thickness
- Intraretinal cysts
- Subretinal fluid
- Vitreomacular traction

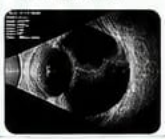
OCT Angiography

- Non-invasive retinal vascular imaging



B-Scan Ultrasonography
Useful in:

- Dense vitreous hemorrhage
- Retinal detachment



MANAGEMENT

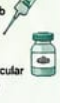
A. GENERAL MEASURES

- Strict glycemic control
- Control hypertension
- Lipid lowering therapy
- Treat nephropathy and anemia
- Smoking cessation
- Regular ophthalmic screening

C. TREATMENT OF DME

1. Anti-VEGF Therapy (First Line)
Drugs:

- Ranibizumab
- Aflibercept
- Bevacizumab
- Faricimab



Actions:

- Reduces vascular permeability
- Regresses neovascularization
- Improves visual acuity

2. Intravitreal Steroids
Used in refractory DME.
Drugs:

- Triamcinolone
- Dexamethasone implant

Complications:

- Cataract
- Raised intraocular pressure

3. Laser Photocoagulation

- Focal laser
- Grid laser

D. TREATMENT OF PDR

1. Panretinal Photocoagulation (PRP)
Treatment of choice.
Mechanism:

- Ablates ischemic retina
- Decreases VEGF production
- Causes regression of neovascularization

Complications:

- Peripheral field loss
- Night blindness
- Reduced color vision
- Exacerbation of macular edema

2. Anti-VEGF Therapy
Increasingly used for:

- PDR
- Vitreous hemorrhage
- Adjunct before vitrectomy

3. Pars Plana Vitrectomy
Indications:

- Non-clearing vitreous hemorrhage (>3 months)
- Tractional retinal detachment threatening macula
- Combined retinal detachment
- Dense premacular hemorrhage
- Refractory DME with traction

FOLLOW-UP SCHEDULE

Stage	Follow-up
Mild NPDR	12 months
Moderate NPDR	6 months
Severe NPDR	3–4 months
PDR	Immediate treatment

COMPLICATIONS

- Vitreous hemorrhage
- Tractional retinal detachment
- Neovascular glaucoma
- Macular edema
- Blindness

PREVENTION

- Tight glycemic control
- BP control
- Lipid management
- Annual retinal examination
- Early laser or anti-VEGF treatment

IMPORTANT VIVA POINTS

- Earliest clinical sign – Microaneurysm
- Earliest ophthalmoscopic finding – Microaneurysm
- Most common cause of visual loss – Diabetic macular edema
- Most common cause of sudden visual loss – Vitreous hemorrhage
- Treatment of choice for PDR – Panretinal photocoagulation
- Gold standard investigation for DME – OCT
- Major mediator – VEGF

PATHOGENESIS FLOWCHART

Diabetes Mellitus
↓
Chronic Hyperglycemia
↓
Pericyte loss + endothelial dysfunction
↓
Capillary leakage + microaneurysm formation
↓
Retinal ischemia
↓
VEGF release
↓
Neovascularization + macular edema
↓
Vitreous hemorrhage / TRD / NVG / Blindness

Dr Ashik SureshKumar

DIFFERENCES BETWEEN PARALYTIC SQUINT AND CONCOMITANT SQUINT

Concomitant squint	Paralytic squint
Mostly developmental, usually not associated with trauma/disease	Mostly acquired, usually associated with trauma/disease
Occurs in infants and children	Usually in adults
Diplopia absent	Diplopia present
No abnormal head posture	Abnormal head posture to avoid diplopia
Absence of false projection (i.e. false localization of object)	False projection is present
Angle of squint is usually constant in all directions of gaze	Angle of squint is variable in different directions of gaze
Ocular movements are normal and there is no restriction	Ocular movement is restricted in the direction of action of paralyzed muscle
Secondary angle of deviation is always equal to primary deviation	Secondary deviation is greater than primary deviation
Amblyopia is present	No amblyopia
Surgical result is usually good	Surgical result is not satisfactory, and at times, surgery may be contraindicated

LASER
IN OPHTHALMOLOGY

Lasers have revolutionized ophthalmology by providing precise, minimally invasive treatments for a variety of ocular diseases. They offer targeted therapy with minimal damage to surrounding tissues, promoting faster recovery and better outcomes.

WHAT ARE OPHTHALMIC LASERS?

Lasers emit focused light of specific wavelength that can be absorbed by ocular tissues to achieve precise cutting, coagulation, or tissue modification with minimal damage to surrounding structures.

APPLICATIONS OF LASER IN OPHTHALMOLOGY

RETINAL DISORDERS
Treatment of diabetic retinopathy, retinal tears, vein occlusions, and age-related macular edema.

GLAUCOMA
Laser peripheral iridotomy, trabeculoplasty, and laser iridoplasty to control intraocular pressure.

POSTERIOR CAPSULAR OPACIFICATION (PCO)
YAG laser capsulotomy to restore vision.

REFRACTIVE SURGERY
Excimer laser for correction of myopia, hyperopia, astigmatism.

CATARACT SURGERY
Femtosecond laser-assisted cataract surgery for precise incisions and reduced complications.

TYPES OF LASERS USED IN OPHTHALMOLOGY

- **Argon Laser (Blue-Green)**
  Used for retinal photocoagulation in diabetic retinopathy, retinal tears, and glaucoma.
- **YAG Laser (Nd:YAG)**
  Used for posterior capsulotomy (PCO), iridotomy in glaucoma, and laser iridoplasty.
- **Diode Laser**
  Used in pan-retinal photocoagulation and treatment of glaucoma.
- **Excimer Laser**
  Primarily used in refractive surgery (LASIK, PRK) to reshape the cornea.
- **Femtosecond Laser**
  Used in cataract surgery, corneal incisions, and refractive surgery with high precision.

ADVANTAGES

- ✓ High precision and accuracy
- ✓ Minimally invasive
- ✓ Reduced pain and inflammation
- ✓ Shorter recovery time
- ✓ Outpatient procedure
- ✓ Improved visual outcomes

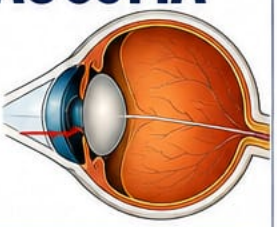
Laser technology continues to advance, offering safer, more effective, and targeted treatments that have transformed modern eye care.

Dr Ashik Suresh Kumar

LENS INDUCED (PHACOMORPHIC) GLAUCOMA

ESSAY ANSWER

60 years old patient presented with sudden onset of pain, redness in one eye and headache, with decreased vision since six months, with past history of cataract surgery in other eye.



ANSWER THE FOLLOWING

- What is your more probable diagnosis
- What are the differential diagnosis
- What are the clinical features of the condition
- How will you confirm the diagnosis
- How will you manage the case

1. MORE PROBABLE DIAGNOSIS

PHACOMORPHIC GLAUCOMA (LENS-INDUCED GLAUCOMA)

Secondary angle-closure glaucoma due to an intumescent cataractous lens.

REASONING:

- Decreased vision since 6 months suggests advanced cataract.
- Elderly patient with cataract in the affected eye.
- Sudden pain, redness, headache → acute rise in IOP.
- History of cataract surgery in other eye (strong clue).

2. DIFFERENTIAL DIAGNOSIS

1. Acute Angle-Closure Glaucoma (Primary)
2. Acute Iridocyclitis (Anterior Uveitis)
3. Acute Conjunctivitis
4. Corneal Ulcer / Keratitis
5. Endophthalmitis
6. Scleritis
7. Neovascular Glaucoma
8. Lens-induced Glaucoma (Phacomorphic)

PRIMARY AACG vs PHACOMORPHIC GLAUCOMA

Feature	Primary AACG	Phacomorphic Glaucoma
Cause	Pupillary block	Intumescent cataractous lens
Cataract history	May or may not be present	Usually present
Progressive visual loss before attack	Less prominent	Common
Definitive treatment	Laser iridotomy	Cataract extraction
Lens status	Often clear lens	Mature/intumescent cataract

3. CLINICAL FEATURES

A. Symptoms

Ocular Symptoms

- Sudden severe ocular pain
- Redness of eye
- Marked diminution of vision
- Colored halos around lights
- Lacrimation

Systemic Symptoms

- Headache
- Nausea
- Vomiting
- Sometimes abdominal pain

B. Signs

- Patient distressed, photophobic
- Eyelids – Mild edema
- **Conjunctiva** – Circumcorneal congestion (ciliary flush)
- **Cornea** – Hazy/steamy cornea (edema)
- **Anterior Chamber** – Shallow
- **Pupil** – Mid-dilated, vertically oval, fixed/sluggish
- **Intraocular Pressure** – Markedly elevated (40–70 mmHg)
- **Vision** – Reduced
- **Eyeball** – Feels stony hard on palpation
- **Fundus** – Often not visible: if visible → glaucomatous cupping

4. CONFIRMATION OF DIAGNOSIS

1. **Clinical Examination**
   - Typical triad: Painful red eye + Corneal edema + Mid-dilated fixed pupil with raised IOP
2. **Tonometry**
   - Shows markedly elevated intraocular pressure.
   - Normal IOP: 10–21 mmHg
   - In acute angle closure: Usually >40 mmHg

3. **Gonioscopy (Gold Standard)**
   - Shows closed / narrow anterior chamber angle

4. **Slit Lamp Examination**
   - Corneal edema
   - Shallow anterior chamber
   - Congested eye

5. **Optic Disc Evaluation**
   - To assess glaucomatous damage after acute attack subsides

6. **Visual Field Testing**
   - Done later after acute episode resolves
   - Shows peripheral visual field defects

DIFFERENCE BETWEEN AACG AND ACUTE IRIDOCYCLITIS

Feature	Acute Angle Closure Glaucoma	Acute Iridocyclitis
Pain	Severe	Moderate
Vision	Markedly reduced	Mild–moderate reduction
Cornea	Hazy	Usually clear
Pupil	Mid dilated	Constricted
IOP	Increased	Usually low/normal
Anterior chamber	Shallow	Normal
Congestion	Ciliary	Ciliary
Eye consistency	Stony hard	Normal

5. MANAGEMENT

AIMS OF TREATMENT

1. Rapid reduction of IOP
2. Relief of pain
3. Clearing corneal edema
4. Prevention of optic nerve damage
5. Definitive treatment of angle closure

A. IMMEDIATE MEDICAL MANAGEMENT

1. **ACETAZOLAMIDE**
   - 500 mg IV stat, then 250 mg orally 6 hourly
   - Decreases aqueous humor production

2. **TOPICAL BETA BLOCKER**
   - Example: Timolol 0.5%
   - Reduces aqueous production

3. **HYPEROSMOTIC AGENTS**
   - Mannitol 20% IV
   - 1–2 g/kg over 30–45 min (if IOP very high)

4. **PILOCARPINE EYE DROPS**
   - 2% Pilocarpine (after IOP begins to reduce)
   - Causes miosis & opens trabecular meshwork

5. **TOPICAL STEROIDS**
   - Example: Prednisolone acetate
   - Reduces inflammation

6. **ANALGESICS & ANTIEMETICS**
   - For symptomatic relief

B. DEFINITIVE TREATMENT

LASER PERIPHERAL IRIDOTOMY (Treatment of Choice)

Creates alternate pathway for aqueous flow from posterior to anterior chamber.
Usually performed in affected eye and prophylactically in fellow eye.

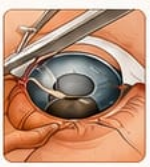
C. SURGICAL TREATMENT

If laser not possible or ineffective:

- **Trabeculectomy**

or

- **Lens Extraction** (if phacomorphic component present)



CONCLUSION

Phacomorphic glaucoma is a lens-induced secondary angle closure due to an intumescent cataract. It presents as a painful red eye with sudden rise in IOP. Early recognition and prompt medical management followed by definitive cataract extraction are essential to prevent irreversible blindness.

Dr. Ashik SureshKumar

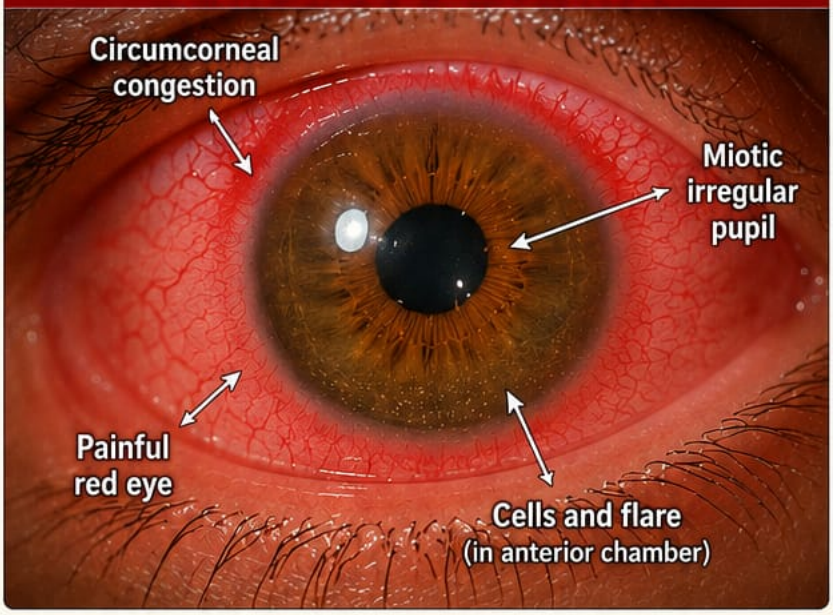
PAINFUL RED EYE – THINK UVEITIS!

"When in doubt, look for CELLS, FLARE & CILIARY CONGESTION"

CLINICAL CLUES

- **Painful** red eye
- **Photophobia**
- Circumcorneal **congestion**
- **Miotic** irregular pupil
- **Cells** and **flare** (in anterior chamber)

WHAT DO THESE FINDINGS MEAN?



ANSWER:

ACUTE IRIDOCYCLITIS

(ANTERIOR UVEITIS)

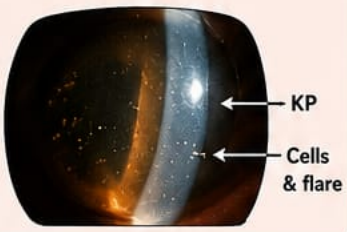
INFLAMMATION OF IRIS & CILIARY BODY

KEY DIFFERENTIATORS

FEATURE	ACUTE IRIDOCYCLITIS	ACUTE ANGLE CLOSURE GLAUCOMA
Pain	Moderate	Severe
Photophobia	Marked	Mild/Variable
Cornea	Clear	Hazy / Steamy
Pupil	Miotic, irregular	Mid-dilated, fixed
IOP	Low / Normal	Very High
Anterior Chamber	Normal depth, cells & flare	Shallow
Eye Consistency	Normal	Stony hard
Ciliary Tenderness	Present	Absent
Nausea / Vomiting	Rare	Common

SLIT LAMP FINDINGS

- Circumcorneal congestion (ciliary flush)
- Keratic precipitates
- Cells in anterior chamber
- Flare in anterior chamber
- Muddy iris
- Posterior synechiae
- Miotic irregular pupil



MANAGEMENT

Cycloplegics / Mydriatics
e.g., Atropine 1%
Relieve pain & prevent posterior synechiae

Topical Corticosteroids
e.g., Prednisolone acetate 1%
Reduce inflammation

NSAIDs
For pain relief

Treat underlying cause
If infectious / autoimmune cause identified

Monitor IOP
Secondary glaucoma may develop

INVESTIGATIONS (TO FIND ETIOLOGY)

- ESR, CRP
- HLA-B27
- VDRL
- Mantoux test
- Chest X-ray
- ANA, RF
- Serum ACE (if sarcoidosis suspected)
- Others as per clinical suspicion

POSSIBLE COMPLICATIONS

- Posterior synechiae
- Occlusio pupillae
- Seclusio pupillae
- Complicated cataract
- Secondary glaucoma
- Band keratopathy
- Hypotony
- Phthisis bulbi

HIGH YIELD POINT:
Painful red eye + Photophobia + Circumcorneal congestion + Miotic irregular pupil + Cells & flare = Think ACUTE IRIDOCYCLITIS

REMEMBER:
Check for CELLS, FLARE & KP – These are the keys!

Dr Ashik SureshKumar

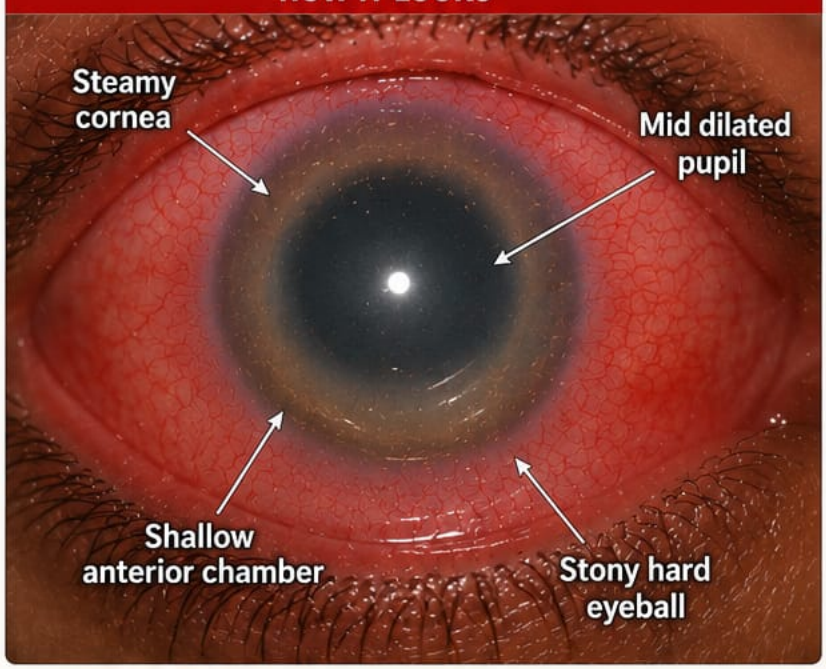
PAINFUL RED EYE – THINK GLAUCOMA!

"WHEN IN DOUBT, LOOK FOR RAISED IOP & A SHALLOW ANTERIOR CHAMBER"

CLINICAL CLUES

- Elderly female
- Sudden severe pain
- Halos around eye
- Vomiting
- Steamy cornea
- Shallow anterior chamber
- Mid dilated pupil
- Stony hard eyeball

HOW IT LOOKS



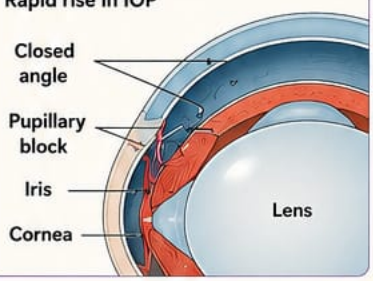
ANSWER:

ACUTE ANGLE CLOSURE GLAUCOMA

AN OPHTHALMIC EMERGENCY

PATHOPHYSIOLOGY

Pupillary block → Peripheral anterior synechiae → Angle closure → Aqueous outflow obstruction → Rapid rise in IOP



KEY DIFFERENTIATING FEATURES

FEATURE	ACUTE ANGLE CLOSURE GLAUCOMA	ACUTE IRIDOCYCLITIS
Onset	Sudden	Subacute
Pain	Severe	Moderate
Redness	Diffuse	Ciliary flush
Cornea	Hazy / Steamy	Clear
Anterior Chamber	Shallow	Normal depth
Pupil	Mid dilated, fixed	Miotic, irregular
IOP	Very High	Low / Normal
Eye Consistency	Stony hard	Normal
Nausea / Vomiting	Common	Rare
Halos around lights	Present	Absent

SIGNS (CLASSIC)

- Conjunctival congestion (diffuse)
- Steamy / hazy cornea
- Shallow anterior chamber
- Mid dilated, non-reacting pupil
- Raised intraocular pressure (40–70 mmHg)
- Stony hard eyeball
- C/O Halos, nausea, vomiting, severe headache

RISK FACTORS

- Age > 60 years
- Female
- Hypermetropia
- Shallow anterior chamber
- Family history

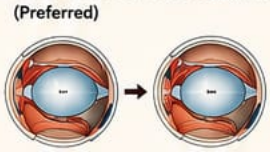
MANAGEMENT (EMERGENCY)

I. IMMEDIATE MEDICAL MANAGEMENT (TO LOWER IOP)

- Tab. Acetazolamide 500 mg IV stat, then 250 mg 6th hourly
- Inj. Mannitol 20% – 1 to 2 g/kg IV
- Topical Beta blocker (e.g., Timolol 0.5%)
- (e.g., Apraclonidine Agonist)
- Topical Miotic (e.g., Pilocarpine 2%) after IOP ↓
- Topical Steroid (short term)

II. DEFINITIVE MANAGEMENT

- Laser Peripheral Iridotomy (LPI) (Preferred)



- If laser not possible → Surgical Iridectomy

INVESTIGATIONS

- IOP measurement
- Gonioscopy (Angle closed)
- Ultrasound B-scan (if cornea hazy)
- Visual field (after recovery)
- Optic disc evaluation

COMPLICATIONS (IF UNTREATED)

- Corneal edema
- Peripheral anterior synechiae
- Chronic angle closure glaucoma
- Optic nerve damage
- Permanent vision loss

REMEMBER:
Painful red eye with halos, vomiting, steamy cornea, shallow AC, mid dilated pupil, raised IOP →
THINK ACUTE ANGLE CLOSURE GLAUCOMA!

TIME IS VISION – EARLY RECOGNITION PREVENTS BLINDNESS

"A small pupil in pain is uveitis, a big pupil in pain is glaucoma."
– Ophthalmology Pearl

Dr Ashik SureshKumar

DISEASE OF RETINA

Case: 70-year-old man with sudden painless loss of vision in left eye. Curtain coming down in front of the eye. Cherry red spot in the left eye.

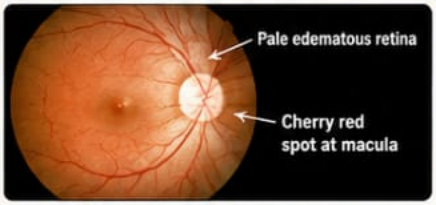
CASE SUMMARY

- 70-year-old hypertensive and diabetic male
- Sudden painless loss of vision in left eye
- Describes as "curtain coming down"
- Normal vision in right eye
- Cherry red spot in left eye

PROBABLE DIAGNOSIS

CENTRAL RETINAL ARTERY OCCLUSION (CRAO)

Sudden painless monocular visual loss with cherry-red spot at macula is classic of CRAO.



ETIOLOGY / RISK FACTORS

- Hypertension
- Diabetes mellitus
- Atherosclerosis
- Carotid artery disease
- Cardiac emboli
- Giant cell arteritis
- Hypercoagulable states

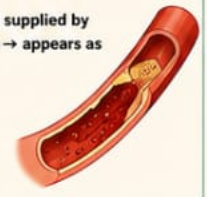
PATHOPHYSIOLOGY

Occlusion of central retinal artery

→ Acute retinal ischemia

→ Retinal edema and whitrening

The fovea is thin and supplied by choroidal circulation → appears as cherry-red spot.



CLINICAL FEATURES

Symptoms

- Sudden painless monocular visual loss
- Severe diminution of wision
- Amaurosis fugax may precede attack

Signs

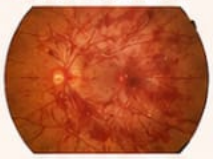
- Markedly reduced visual acuity
- Relative afferent pupillary defect (RAPD)
- Fundus: Pale edematous retina, cherry-red spot at macula, attenuated retinal arteries, box-carring of blood column

SIGNS TO LOOK FOR

1. Relative afferent pupillary defect
2. Retinal whitening / opacification
3. Narrow, attenuated retinal arteries
4. Box-carring / segmentation of blood column
5. Embolus in retinal artery
6. Neovascularization
7. Signs of hypertensive / diabetic retinopathy
8. Carotid bruit
9. Temporal artery tenderness (to rule out giant cell arteritis)

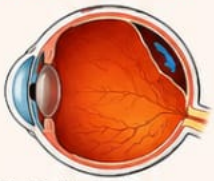
FOUR DIFFERENTIAL DIAGNOSES OF SUDDEN VISUAL LOSS IN ELDERLY

1. CENTRAL RETINAL VEIN OCCLUSION (CRVO)



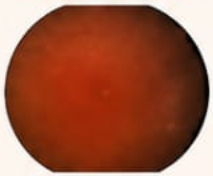
- "Blood and thunder" retina
- Dilated, tortuous veins
- Retinal hemorrhages

2. RETINAL DETACHMENT



- Curtain-like visual loss
- Flashes and floaters
- Shallow / total detachment on examination

3. VITREOUS HEMORRHAGE



- Sudden visual haze
- Floaters
- Common in diabetes

4. ISCHEMIC OPTIC NEUROPATHY

- Sudden visual loss
- Pain may be present
- Pale swollen optic disc

INVESTIGATIONS

Ocular

- Fundus examination
- Fundus fluorescein angiography (FFA)
- OCT retina
- Visual field testing

Systemic

- Blood sugar (FBS, PPBS, HbA1c)
- Blood pressure
- Lipid profile
- ESR / CRP (to rule out GCA)
- ECG, Echocardiography
- Carotid Doppler

MANAGEMENT – CRAO IS AN OPHTHALMIC EMERGENCY

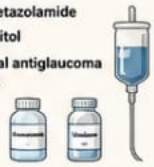
RETINAL SURVIVAL TIME: APPROXIMATELY 90–100 MINUTES

1. OCULAR MASSAGE

Attempt to dislodge the embolus.

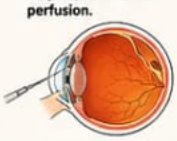
2. LOWER INTRAOCULAR PRESSURE

- IV Acetazolamide
- Mannitol
- Topical antiglaucoma drugs



3. ANTERIOR CHAMBER PARACENTESIS

May improve retinal perfusion.



4. CARBOGEN INHALATION

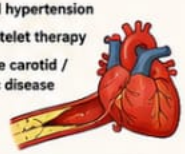
95% O_2 + 5% CO_2

5. HYPERBARIC OXYGEN THERAPY

Useful if initiated early.

6. TREAT UNDERLYING CAUSE

- Control diabetes
- Control hypertension
- Antiplatelet therapy
- Manage carotid / cardiac disease



COMPLICATIONS

- Permanent visual loss
- Optic atrophy
- Neovascular glaucoma
- Ocular ischemic syndrome
- Retinal neovascularization

PROGNOSIS

- Usually poor visual prognosis
- Better outcome if treatment is initiated early

KEY POINTS TO REMEMBER

- ✓ Sudden painless monocular visual loss → Think CRAO
- ✓ Cherry-red spot at macula is classic sign
- ✓ Time is retina – act within 90–100 minutes
- ✓ Evaluate for systemic vascular risk factors

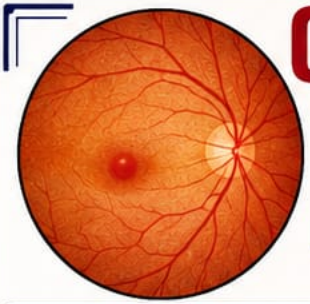
RED FLAGS

- ! Sudden painless visual loss
- ! Presence of vascular risk factors
- ! Cherry-red spot at macula
- ! Requires immediate management

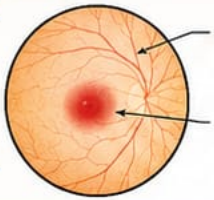
EARLY RECOGNITION + RAPID MANAGEMENT = BEST CHANCE TO SAVE VISION

Dr Ashik Sureshkumar

CHERRY RED SPOT – CAUSES



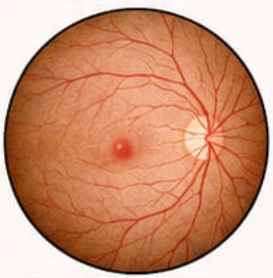
Definition: A cherry red spot is seen at the macula when the surrounding retina becomes pale or opaque while the fovea retains its normal reddish appearance due to intact choroidal circulation.



CAUSES OF CHERRY RED SPOT

1. VASCULAR CAUSES

1. **Central Retinal Artery Occlusion (CRAO)**
   - Most common acquired cause
   - Sudden painless loss of vision
   - Pale ischemic retina with cherry red macula



2. LYSOSOMAL STORAGE DISORDERS (INHERITED)

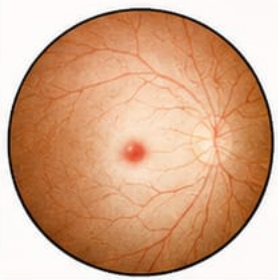
2. **Tay–Sachs Disease**
   - Hexosaminidase A deficiency
   - GM2 ganglioside accumulation
3. **Sandhoff Disease**
   - Hexosaminidase A and B deficiency
4. **Niemann–Pick Disease**
   - Sphingomyelin accumulation
5. **GM1 Gangliosidosis**
   - GM1 ganglioside accumulation
6. **Metachromatic Leukodystrophy**
   - Sulfatide accumulation
7. **Sialidosis**
   - Sialic acid accumulation
8. **Farber Disease**
   - Ceramide accumulation
9. **Gaucher Disease (occasionally)**
   - Glucocerebroside accumulation

3. TOXIC / METABOLIC CAUSES

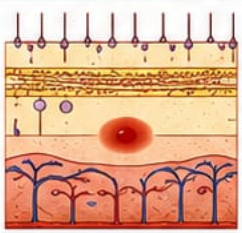
10. **Methanol Poisoning**
    - Formic acid toxicity
11. **Carbon Monoxide Poisoning (rare)**
    - Hypoxic retinal damage

4. TRAUMA

12. **Commotio Retinae (Berlin's Edema)**
    - Following blunt trauma
    - Transient retinal whitening with cherry red spot



MECHANISM OF CHERRY RED SPOT



- Retina becomes pale or opaque due to edema, ischemia or storage material.
- Fovea appears red because:
  - It is thin
  - Lacks ganglion cells
  - Choroidal vasculature is visible beneath

IMPORTANT VIVA POINT

Most common acquired cause

Central Retinal Artery Occlusion

Most common pediatric cause

Tay–Sachs Disease

OTHER POINTS TO REMEMBER

- ★ Cherry red spot is a sign, not a diagnosis.
- ★ Look for associated systemic history and signs.
- ★ Early diagnosis helps in management of treatable causes (e.g., CRAO, methanol poisoning).
- ★ In inherited disorders, cherry red spot is usually bilateral and seen in children.

MNEMONIC – "CRAZY STaNDS"

C	R	A	Z	Y	S	T	a	N	D	S
CRAO (Central Retinal Artery Occlusion)	Retinal Trauma (Berlin Edema / Commotio Retinae)	Amaurosis / Arterial Occlusion	(Storage Disorders Group)	Young Children → Tay–Sachs Disease	Sandhoff Disease	Tay–Sachs Disease	(Storage Disorders Group)	Niemann–Pick Disease	Drug / Toxic Causes (e.g., Methanol, CO poisoning)	Sialidosis (among other storage disorders)

★ *Dr Ashik Sureshkumar* ★

RETINOBLASTOMA

THE COMMONEST MALIGNANT INTRAOCULAR TUMOUR IN CHILDREN

1. DEFINITION

Retinoblastoma is a malignant tumour arising from the immature neuroepithelial cells (retinoblasts) of the developing neurosensory retina in one or both eyes.

It is the **most common intraocular tumour of childhood** and the **second most common malignant intraocular tumour after uveal melanoma.**

Key Point
Retinoblastoma is a potentially curable tumour when detected early.

2. EPIDEMIOLOGY

- **Prevalence:** 1 in 15,000–20,000 live births
- Accounts for about 3% of all childhood cancers
- **Age:** Usually occurs between 1–2 years
- **Sex:** No sex predisposition
- **Laterality:**
  - Unilateral in 70–75%
  - Bilateral in 25–30%
  - One eye is usually affected earlier and more extensively.

3. GENETICS & HEREDITY

Retinoblastoma gene (RB) located on **chromosome 13q14 is a tumour suppressor** (anti-oncogenic) gene.

Knudson two-hit hypothesis:
Deletion/inactivation of both alleles of RB gene leads to tumour.

1. Heritable (Germline) Retinoblastoma
- 40% of cases
- Autosomal dominant inheritance
- Usually bilateral, multifocal
- Presents earlier

13q14

2. Non-heritable (Somatic) Retinoblastoma
- 60% of cases
- Usually unilateral, unifocal
- Sporadic, presents later

4. PATHOLOGY

Origin
Arises as malignant proliferation of immature retinal neural cells.

Histopathology
Tumour composed of small round blue cells with scant cytoplasm and large nuclei.

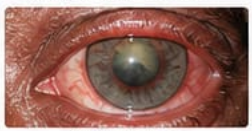
Rosettes (Characteristic)
- **Flexner-Wintersteiner rosettes:** Highly specific. Central lumen surrounded by tumour cells.
- **Homer-Wright rosettes:** No lumen, tumour cells arranged around neurofibrils.
- **Fleurettes:** Photoreceptor differentiation.
- **Pseudorosettes:** Tumour cells arranged around blood vessels; indicate poor differentiation.

5. CLINICAL FEATURES

I. QUIESCENT STAGE
- Leukocoria (60%) (white pupillary reflex/ "cat's eye reflex")
- Strabismus
- Defective vision
- Nystagmus (usually in bilateral cases)


Leukocoria

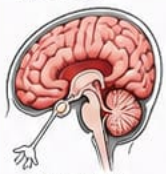
II. GLAUCOMATOUS STAGE (PAINFUL RED EYE)
- Secondary / neovascular glaucoma
- Redness, watering
- Corneal haze, raised IOP
- Enlarged eyeball (buphthalmos)
- Orbital cellulitis-like picture


Glaucomatous eye

III. STAGE OF EXTRAOCULAR EXTENSION
- Extension through sclera
- Fungating orbital mass
- Proptosis
- Orbital cellulitis-like features


Orbital extension

IV. STAGE OF DISTANT METASTASIS
- Lymphatic spread
- Direct extension to optic nerve and brain


Brain metastasis

6. TYPES OF RETINOBLASTOMA

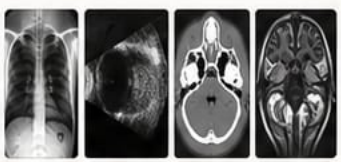
1. **Endophytic:** Grows inward into vitreous cavity. White, creamy mass; vitreous seeding common; "cottage cheese" appearance.
2. **Exophytic:** Grows outward; causes exudative retinal detachment.
3. **Mixed:** Features of both endophytic and exophytic growth.
4. **Diffuse infiltrating:** Placoid retinal thickening; diagnosed late.

7. DIFFERENTIAL DIAGNOSIS OF LEUKOCORIA (PSEUDOGLIOMA)

- Congenital cataract
- Persistent hyperplastic primary vitreous (PHPV)
- Retinopathy of prematurity
- Toxocariasis
- Coats disease
- Endophthalmitis

8. DIAGNOSIS

- **Examination under anaesthesia (EUA):**
  - Fundus examination
  - Indirect ophthalmoscopy
  - IOP measurement
- **Plain X-ray orbit:** Calcification in ~75% cases.
- **B-scan ultrasonography:** Detects tumour mass and calcification.
- **CT scan:** Detects calcification, shows extent of intraocular and extraocular spread.
- **MRI:** Best for optic nerve involvement, intracranial extension and pinealoblastoma.


X-ray (Calcification) | B-scan USG | CT scan | MRI (Optic nerve involvement)

9. INTERNATIONAL CLASSIFICATION OF RETINOBLASTOMA (ICRB)

Group	Description
A	Small tumour confined to retina
B	Larger tumour, may be near fovea or disc
C	Local subretinal / vitreous seeds
D	Diffuse subretinal / vitreous seeds
E	Extensive tumour (≥50% retina) or any with high-risk features (poor prognosis)

10. TREATMENT

A. CONSERVATIVE (GLOBE SALVAGE)
Used in Groups A–D

1. Chemoreduction (CVE regimen)
- Carboplatin
- Vincristine
- Etoposide

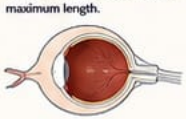
2. Focal Therapies
- Cryotherapy – small anterior tumours
- Laser photocoagulation – posterior tumours
- Thermotherapy – small posterior tumours
- Plaque radiotherapy – localized vitreous disease

3. External Beam Radiotherapy (EBRT)
Now used less due to risk of secondary malignancy.

B. ENUCLEATION

Indications
- Group E tumours
- Optic nerve involvement
- Neovascular glaucoma
- Extensive retinal involvement

Optic nerve should be cut with maximum length.



C. PALLIATIVE THERAPY

Indications
- Orbital extension
- Intracranial spread
- Distant metastasis

Includes
- Chemotherapy
- Radiotherapy
- Orbital exenteration

11. METASTASIS

Spread occurs by:
- Lymphatic spread (preauricular & neighbouring nodes)
- Direct extension via optic nerve to brain

12. PROGNOSIS

Good prognosis:
- When detected early and treated before extraocular extension.

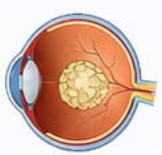
Poor prognostic factors:
- Optic nerve invasion
- Massive choroidal invasion
- Undifferentiated tumour cells
- Extraocular extension
- Late stage at presentation

13. IMPORTANT PEARLS

- ★ Leukocoria is the most common presenting sign.
- ★ Bilateral disease is usually heritable and earlier onset.
- ★ Flexner-Wintersteiner rosettes are pathognomonic.
- ★ Calcification on imaging is highly suggestive.
- ★ ICRB guides treatment and prognosis.
- ★ Early diagnosis improves globe salvage and survival.

14. SUMMARY

Retinoblastoma is a highly curable intraocular cancer of childhood when detected early and managed appropriately. A multidisciplinary approach with accurate staging, appropriate therapy and regular follow-up is essential for preserving life, vision and the eye.



Abbreviations: RB – Retinoblastoma gene; IOP – Intraocular pressure; CT – Computed Tomography; MRI – Magnetic Resonance Imaging; USG – Ultrasonography; CVE – Carboplatin, Vincristine, Etoposide; EBRT – External Beam Radiotherapy; ICRB – International Classification of Retinoblastoma

★ Dr Ashik Sureshkumar ★

OPTIC NEURITIS

INFLAMMATION AND DEMYELINATION OF THE OPTIC NERVE

DEFINITION

Optic neuritis is an inflammatory and demyelinating disorder of the optic nerve that causes acute or subacute visual loss, pain and colour vision defects.

ANATOMY (Optic Nerve)

- Anterior (intraocular) part – up to lamina cribrosa
- Posterior (intraorbital) part – up to optic canal
- Intracanalicular part – within optic canal
- Intracranial part – from optic canal to optic chiasm

ETIOLOGY

1. Idiopathic (most common)
2. Demyelinating diseases – Multiple sclerosis, Neuromyelitis optica (Devic's disease), ADEM
3. Infections – Viral (measles, mumps, chickenpox, EBV), Bacterial (TB, syphilis, Lyme), Others
4. Autoimmune – Sarcoidosis, SLE, Polyarteritis nodosa, Granulomatosis with polyangiitis, Sjögren syndrome
5. Toxic – Alcohol, tobacco, methanol, ethambutol, isoniazid, etc.
6. Traumatic – Optic nerve contusion or compression
7. Paraneoplastic, Radiation, Others (rare)

TYPES (ANATOMICAL / CLINICAL)

1. Typical (Retrobular) Optic Neuritis – Most common; demyelinating cause, unilateral, painful eye movements.
2. Neuroretinitis – Optic disc + surrounding retina (macular star may be present).
3. Papillitis – Optic disc only (less common).
4. Retrobulbar Neuritis – Posterior to globe; most common type.
5. Para-infectious Optic Neuritis – Following infections.
6. Toxic / Nutritional Optic Neuritis – Due to toxins or nutritional deficiencies.

EVALUATION OF ANISOCORIA

1. Pupil size in dim and bright illumination should be noted first.
   - *Anisocoria same in dark and bright* illumination → usually physiological.
   - *Anisocoria greater in dim illumination* → suggests Horner's syndrome (sympathetic palsy).
2. Pupillary light reflex should be tested after noting the pupil size.
   - *Poor light reaction* → defect in parasympathetic system (III nerve palsy, tonic pupil, anticholinergic drugs).

Papillitis (swollen disc)

CLINICAL FEATURES

A. Symptoms

- Subacute visual loss – unilateral, develops over hours to days; reaches nadir in 1–2 weeks.
- Pain on eye movements (retrobulbar pain) – common, aggravated by movement.
- Visual loss – monocular, central/ceccentral scotoma common.
- Decreased colour vision (red desaturation).
- Photopsia (visual obscurations in bright light).
- Impaired contrast sensitivity.
- May be associated with headache.

B. Signs

1. Visual acuity – reduced (variable).
2. Colour vision – severely impaired (red desaturation).
3. Relative Afferent Pupillary Defect (RAPD) – present in most cases; detected by swinging flashlight test.
4. Fundus – may be normal (retrobulbar ON) or show disc swelling (papillitis) / macular star (neuroretinitis).
5. Visual field defect – central scotoma most common; altitudinal defects may occur.
6. Contrast sensitivity – reduced.
7. Visual evoked response (VEP) – reduced amplitude, delayed P100 latency.

RAPD – SWINGING FLASHLIGHT TEST

- Shine light in both eyes – direct and consensual reflexes are normal.
- Swing light to the affected eye → pupils dilate.
- Swing light to the normal eye → pupils constrict. (Indicates afferent defect in the affected eye.)

UHTHOFF PHENOMENON

Transient worsening of visual symptoms on exertion or exposure to heat; improves on rest or cooling. Seen in demyelinating optic neuritis (multiple sclerosis).

PULFRICH PHENOMENON

Patients complain of mild, dull eyeache (retrobulbar pain). Pain increases with eye movements, especially upward and downward gaze.

INVESTIGATIONS

A. Visual Function Tests

- Visual acuity – Reduced
- Colour vision – Red desaturation
- Visual field – Central scotoma common
- Contrast sensitivity – Reduced
- VEP – Reduced amplitude, delayed P100 latency.

B. Imaging

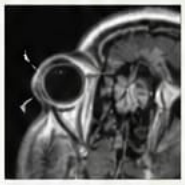
- MRI brain and orbits with contrast – Investigation of choice.
  - Optic nerve enhancement (T2 hyperintensity with gadolinium enhancement).
  - Brain lesions suggestive of demyelination (MS plaques).
- CT scan – Rarely used.

C. Other Tests

- Optical Coherence Tomography (OCT) – RNFL thinning in subacute/chronic cases.
- Serological tests – As indicated (VDRL, ANA, ACE, NMO-IgG, anti-MOG, etc.)
- Lumbar puncture – If demyelinating disease or infection suspected.

MRI FINDINGS IN OPTIC NEURITIS

- T2 hyperintensity of optic nerve
- Gadolinium enhancement (within 10 days of onset)
- May show brain lesions (periventricular, juxtacortical, infratentorial) in MS



DIFFERENTIAL DIAGNOSIS

- Papillitis – Disc swelling without star formation
- Neuroretinitis – Disc edema with macular star
- Papilloedema – Bilateral, without pain, due to raised ICP
- Ischaemic optic neuropathy – Sudden painless loss in older patients, altitudinal defect
- Anterior ischaemic optic neuropathy – Pale swollen disc
- Compressive optic neuropathy – Slow progressive, painless
- Leber hereditary optic neuropathy – Young males
- Toxic / Nutritional optic neuropathy – Bilateral, symmetrical

OPTIC NEURITIS TREATMENT TRIAL (ONTT)

Large randomized trial which showed:

- Oral prednisolone hastens visual recovery but does not change final outcome.
- IV methylprednisolone reduces risk of recurrence of ON.

Recommendations (for typical demyelinating ON):

1. IV methylprednisolone 1 g daily for 3 days → followed by oral prednisolone 1 mg/kg/day for 11 days → then taper over 4 days.
2. Oral prednisolone alone is not recommended.
3. No treatment / placebo – similar final visual outcome but slower recovery and higher recurrence rate.

ASSOCIATION WITH MULTIPLE SCLEROSIS (MS)

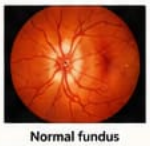
- Optic neuritis may be the first manifestation of MS in 15–20% of patients.
- Risk of developing MS after a single episode of ON is:
  - 30–50% if MRI shows demyelinating lesions.
  - 12–25% if MRI is normal.
- Risk is higbids alth:
  - Younger age (< 40 years)
  - Abnormal brain MRI
  - Bilateral ON
- ONTT showed IV methylprednisolone delays conversion to clinically definite MS by about 2 years.

EVOLUTION, PROGNOSIS & COMPLICATIONS

- Evolution – Visual loss develops over hours to days, reaches nadir in 1–2 weeks, then recovers gradually over 4–6 weeks.
- Prognosis –
  - About 75–90% recover good visual acuity (6/9 or better).
  - Poor prognosis with severe initial loss, bilateral ON, associated myelitis, or atypical features.
- Complications –
  - Recurrent optic neuritis
  - Optic atrophy (with severe/Repeated attacks)
  - Association with MS or NMO
  - Chronic visual field defects, colour vision deficit

FUNDUS FINDINGS

Typical (Retrobular) ON	Papillitis
• Fundus appears normal. • In some cases – mild blurring of disc margins.	• Disc edema • Hyperaemia • Blurring of margins



Normal fundus

Swollen disc

TREATMENT – SUMMARY

1. Treat the underlying cause if identified (infection, toxin, autoimmune disease, etc.).
2. For typical demyelinating ON – IV methylprednisolone 1 g/day for 3 days → followed by oral prednisolone 1 mg/kg/day for 11 days → taper over 4 days.
3. Oral steroids alone – not recommended.
4. Recurrent or steroid-dependent cases – disease modifying therapy (if MS/NMOSD confirmed).
5. Symptomatic treatment – analgesics for pain.

KEY POINTS

- Most common cause of acute unilateral visual loss in young adults.
- Usually retrobulbar, painful eye movements, central scotoma, RAPD present.
- Uhthoff phenomenon – worsening on heat or exertion; improves on rest.
- MRI brain and orbits with contrast is investigation of choice.
- IV methylprednisolone speeds recovery and reduces recurrence risk.
- Important association with multiple sclerosis – needs follow-up and counselling.

★ Dr Ashik Sureshkumar ★

BACTERIAL CORNEAL ULCER
(BACTERIAL KERATITIS)

A suppurative inflammatory condition of the cornea caused by pyogenic bacteria to epithelial epithelial defect, stromal infiltration, ulceration and potential visual loss. It is an ophthalmic emergency because rapid progression may lead to perforation and blindness.

ETIOLOGY

Prerequisites for infective keratitis
1. Break in corneal epithelium allowing pathogen entry into stroma
2. Pathogenic organisms reaching the damaged cornea

Predisposing factors

A. Factors causing epithelial breach

- Trauma
- Corneal abrasions
- Foreign body
- Contact lens wear
- Exposure keratitis
- Xerosis
- Neuroparalytic keratitis
- Bullous keratopathy

B. Factors lowering host resistance

- Topical steroids
- Diabetes mellitus
- AIDS
- Immunosuppressive therapy

CAUSATIVE ORGANISMS

GRAM-POSITIVE COCCI

- Staphylococcus aureus
- Staphylococcus epidermidis
- Streptococcus pneumoniae

GRAM-NEGATIVE COCCI

- Neisseria gonorrhoeae
- Neisseria meningitidis

GRAM-NEGATIVE BACILLI

- Pseudomonas aeruginosa
- Klebisella
- Proteus
- E. coli
- Moraxella

★ Most common overall organism: Staphylococcus
★ Most common in contact lens wearers: Pseudomonas

PATHOGENESIS

1 Progressive infiltration → 2 Active ulceration → 3 Regression → 4 Cicatrization

The ulcer may:
- Remain localized
- Perforate deeply
- Spread rapidly causing sloughing ulcer

CLINICAL FEATURES

Symptoms
- Pain
- Foreign body sensation
- Watering
- Photophobia
- Blurring of vision
- Redness

Signs
- Lid edema
- Blepharospasm
- Circumcorneal congestion
- Corneal epithelial defect
- Greyish-white stromal infiltrate
- Ulcer with necrotic floor
- Overhanging margins
- Stromal edema
- Hypopyon may be present
- Muddy iris
- Small pupil (iridocyclitis)

CHARACTERISTIC FEATURES

PNEUMOCOCCAL ULCER
- Oval, yellowish-white ulcer
- Dense infiltrate
- Usually associated with hypopyon
- Called ulcus serpens

PSEUDOMONAS ULCER
- Rapidly progressive
- Ring abscess
- Greenish mucopurulent exudate
- Ground-glass surrounding cornea
- May perforate within 48–72 hours

COMPLICATIONS

1. Toxic iridocyclitis
2. Secondary glaucoma
3. Descemetocele
4. Corneal perforation
5. Prolapse of iris
6. Anterior staphyloma
7. Corneal fistula
8. Endophthalmitis / Panophthalmitis
9. Corneal scarring causing permanent visual impairment

INVESTIGATIONS

A. Clinical examination
- Diffuse illumination
- Fluorescein staining
- Slit lamp biomicroscopy
- Assessment of ulcer size, depth, margins, vascularization and anterior chamber reaction

B. Microbiological investigations

Corneal scraping for:
- Gram stain
- Giemsa stain
- KOH mount
- Culture on blood agar
- Sabouraud's agar

TREATMENT – GENERAL MEASURES

- Hospitalization
- Eye rest
- Dark goggles
- Analgesics
- Vitamins

TREATMENT – MEDICAL

A. Topical fortified antibiotics (Mainstay)

Common combinations
- Fortified Cefazolin 5%
- Fortified Tobramycin 1.3%
- Fortified Vancomycin 5%

Fluoroquinolones
- Ciprofloxacin 0.3%
- Ofloxacin 0.3%
- Gatifloxacin 0.3 – 0.5%
- Moxifloxacin 0.5%

Frequency
- Every 5 minutes initially
- Then every 15 minutes
- Hourly for first 48 hours
- Gradual tapering after healing begins

B. Adjunctive therapy

1. Cycloplegics
- Atropine 1%
- Homatropine 2%

Benefits:
- Relieve pain
- Prevent posterior synechiae
- Reduce congestion

2. Systemic antibiotics

Indicated in:
- Perforation
- Scleral involvement
- Gonococcal infection

TREATMENT OF IMPENDING PERFORATION

- Pressure bandage
- Acetazolamide
- Tissue adhesive (cyanoacrylate glue)
- Bandage contact lens
- Conjunctival flap
- Amniotic membrane grafting

SURGICAL TREATMENT

- Therapeutic keratoplasty for perforated or non-healing ulcer

CONCLUSION

Bacterial corneal ulcer is a vision-threatening emergency requiring early diagnosis, microbiological confirmation, and intensive topical antibiotic therapy. Prompt treatment prevents complications such as corneal perforation, endophthalmitis, and permanent blindness.

Dr Ashik Sureshkumar

VERNAL KERATOCONJUNCTIVITIS (VKC)

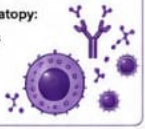
A CHRONIC, RECURRENT, BILATERAL ALLERGIC INFLAMMATION OF CONJUNCTIVA AND CORNEA COMMONLY AFFECTING BOYS DURING SUMMER

DEFINITION

Vernal keratoconjunctivitis is a chronic, recurrent, bilateral allergic inflammation of the conjunctiva and cornea occurring commonly in children and young boys, especially during summer months.

ETIOPATHOGENESIS

- IgE–mediated hypersensitivity reaction
- Increased mast cells and eosinophils
- Associated with atopy:
  - Allergic rhinitis
  - Asthma
  - Eczema



TYPICAL PATIENT PROFILE

- Boys commonly affected
- Age group: 5–15 years
- More common in hot, dry climates
- Seasonal exacerbation during summer / spring

TYPES OF VKC

1. **Palpebral type** – giant cobblestone papillae on upper tarsal conjunctiva
2. **Limbal type** – limbal hypertrophy with Horner–Trantas dots
3. **Mixed type** – features of both palpebral and limbal type

CLINICAL FEATURES

SYMPTOMS

- Severe itching (hallmark symptom)
- Redness
- White ropy / stringy discharge
- Watering
- Photophobia
- Foreign body sensation
- Burning sensation

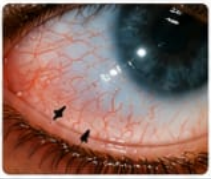
CLINICAL SIGNS

1. PALPEBRAL TYPE

- Giant cobblestone papillae on upper tarsal conjunctiva
- Conjunctival hyperemia
- Thick mucus strands

2. LIMBAL TYPE

- Gelatinous limbal hypertrophy
- Horner–Trantas dots – collections of eosinophils at limbus



3. CORNEAL SIGNS

- Superficial punctate keratitis
- Shield ulcer
- Pannus formation
- Corneal scarring in severe disease

INVESTIGATIONS

Usually clinical diagnosis.

Supportive findings:

- Conjunctival scraping – eosinophils
- Raised serum IgE

DIFFERENTIAL DIAGNOSIS

- Seasonal allergic conjunctivitis
- Atopic keratoconjunctivitis
- Trachoma
- Giant papillary conjunctivitis
- Phlyctenular conjunctivitis

COMPLICATIONS

- Shield ulcer
- Keratoconus due to repeated eye rubbing
- Secondary bacterial infection
- Corneal scarring and visual impairment
- Steroid-induced glaucoma
- Steroid-induced cataract

GENERAL MEASURES

- Avoid allergens and dust exposure
- Cold compresses
- Dark glasses
- Eye hygiene
- Avoid eye rubbing

TREATMENT

1. MILD DISEASE

- Lubricating eye drops
- Topical antihistamines (Olopatadine, Azelastine)

2. MODERATE DISEASE

- Mast cell stabilizers:
  - (Sodium cromoglycate, Lodoxamide)
- Dual action agents (Olopatadine, Ketotifen)

3. SEVERE DISEASE

- Short course topical steroids (Loteprednol, Fluorometholone)

* Monitor IOP during prolonged steroid therapy

4. STEROID-SPARING AGENTS

- Topical cyclosporine
- Tacrolimus ointment

TREATMENT OF CORNEAL COMPLICATIONS (SHIELD ULCER)

May require:

- Debridement
- Antibiotic drops
- Bandage contact lens

KEY EXAM POINTS

- Seasonal recurrent allergic conjunctivitis in boys
- Severe itching is the hallmark symptom
- White ropy discharge is characteristic
- Cobblestone papillae on upper tarsal conjunctiva
- Horner–Trantas dots at limbus are classical sign
- Corneal complications can lead to visual loss
- Steroids should be used cautiously due to risk of glaucoma and cataract

FACTORS AFFECTING DISEASE SEVERITY

- Atopy (allergic rhinitis, asthma, eczema)
- Climate (hot, dry, dusty environment)
- Allergen exposure (pollen, dust, etc.)
- Eye rubbing
- Delayed treatment

IMPORTANT PEARLS

- Usagnosis is mainly clinical.
- Conjunctival scraping may show eosinophils.
- Recurs every year, worse in summer.
- Early treatment prevents corneal complications.
- Compliance with therapy is important.

VIVA QUESTIONS (HIGH YIELD)

Q.	QUESTION	ANSWER
1.	What is VKC?	A chronic, recurrent, bilateral allergic inflammation of conjunctiva and cornea.
2.	Age and sex predilection?	Boys, 5–15 years.
3.	Seasonal variation?	Worse in summer and spring.
4.	Cardinal symptom?	Severe itching.
5.	Characteristic discharge?	White ropy / stringy mucus.
6.	Classical palpebral sign?	Giant cobblestone papillae.
7.	Classical limbal sign?	Horner–Trantas dots.
8.	Corneal complications?	Shield ulcer, pannus, scarring, keratoconus.
9.	Investigations?	Usually not needed. Conjunctival scraping – eosinophils, raised serum IgE.
10.	First line treatment in mild disease?	Lubricants + topical antihistamines / dual action agents.
11.	Drug of choice in moderate disease?	Mast cell stabilizers – sodium cromoglycate / lodoxamide.
12.	Treatment in severe disease?	Short course topical steroids.
13.	Steroid sparing agents?	Cyclosporine eye drops, Tacrolimus ointment.
14.	Important complication?	Shield ulcer and steroid-induced glaucoma.
15.	General advice to patient?	Avoid allergens, cold compresses, avoid eye rubbing.

ONE-LINE CONCLUSION

Vernal keratoconjunctivitis is a chronic, recurrent allergic ocular disorder of childhood characterized by severe itching, ropy discharge, giant papillae and limbal inflammation. Early diagnosis and proper anti-allergic therapy help prevent corneal complications and visual morbidity.

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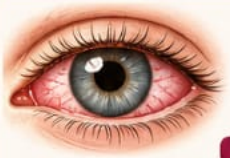
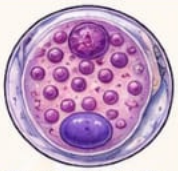


TRACHOMA

(Chronic Keratoconjunctivitis due to Chlamydia trachomatis)



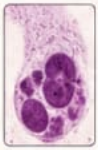
Chlamydia trachomatis

DEFINITION

Trachoma (previously Egyptian ophthalmia) is a chronic keratoconjunctivitis caused by *Chlamydia trachomatis* (biovar TRIC), primarily affecting the superficial epithelium of conjunctiva and cornea. It is characterized by mixed follicular and papillary response, pannus formation and late cicatrization leading to blindness.

1. ETIOLOGY

- Causative organism: *Chlamydia trachomatis*, biovar TRIC (TRIC agent)
- Obligate intracellular, gram-negative bacterium.
- Produces intracytoplasmic inclusion bodies: **Halberstaedter–Prowazek bodies (HP bodies).**



SEROVARS

Associated with blinding trachoma	Associated with inclusion conjunctivitis
A, B, Ba, C	D to K

2. TRANSMISSION

1. Direct spread
- Eye to eye spread
- Contact transmission
- Airborne or waterborne

2. Vector transmission
- Through flies

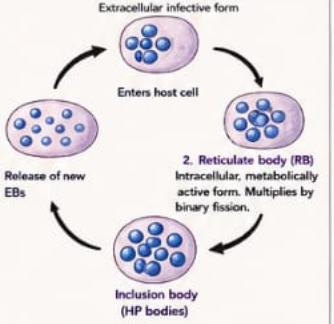
3. Material transfer
- Contaminated fingers, towels, handkerchiefs, bedding, tonometers, surma rods, etc.

Source of infection: Conjunctival discharge of cases. Superadded bacterial infection increases secretions and transmission.

3. PREDISPOSING FACTORS

- Early childhood infection
- Female predominance
- Dry and dusty climate
- Poor socioeconomic conditions
- Overcrowding
- Poor hygiene
- Lack of water supply
- Abundant fly population
- Shared towels and handkerchiefs
- Poor health education

4. LIFE CYCLE OF CHLAMYDIA



5. CLINICAL FEATURES

I. ACTIVE INFLAMMATORY TRACHOMA

(Usually in childhood)

- **Incubation period:** 7–14 days

Symptoms (without secondary infection)
- Mild foreign body sensation
- Occasional lacrimation
- Slight stickiness of lids
- Scanty mucoid discharge

Symptoms (with secondary infection)
- Acute mucopurulent conjunctivitis

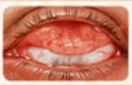
A. Conjunctival Signs
- Congestion of upper tarsal conjunctiva
- Follicles on upper tarsal conjunctiva (sagolike)
- Papillary hypertrophy – flat topped, velvety appearance

B. Corneal Signs
- Superficial keratitis
- Herbert follicles (limbal area)
- Progressive pannus (vascularization of superior cornea)
- Corneal ulcer at advancing edge of pannus

II. CHRONIC CICATRICIAL TRACHOMA

(Due to recurrent infection and chronic inflammation)

Conjunctival Sequelae
- Conjunctival scarring (Arlt's line)
- Concretions
- Pseudocysts, Xerosis, Symblepharon



Corneal Sequelae
- Regressive pannus, Herbert pits
- Corneal opacity, Corneal xerosis
- Corneal ectasia, Corneal blindness

Lid Sequelae
- Trichiasis, Entropion, Tylosis, Ptosis
- Madarosis, Ankyloblepharon

Lacrimal Sequelae
- Chronic dacryocystitis
- Chronic dacryoadenitis

6. WHO CLASSIFICATION (FISTO)

TF Trachomatous inflammation, follicular	≥ 5 follicles on upper tarsal conjunctiva	
TI Trachomatous inflammation, intense	Inflammatory thickening obscuring >50% of deep tarsal vessels	
TS Trachomatous scarring	Scarring on tarsal conjunctiva	
TT Trachomatous trichiasis	At least one eyelash rubbing the eyeball	
CO Corneal opacity	Corneal opacity obscuring pupillary margin	

7. DIAGNOSIS

A. Clinical Diagnosis
- Typical clinical signs
- WHO grading (FISTO)

B. Laboratory Diagnosis
1. Conjunctival cytology – Giemsa stain
2. Detection of inclusion bodies – Iodine stain, Immunofluorescence
3. ELISA
4. PCR (Polymerase chain reaction)
5. Isolation in tissue culture
6. Serotyping by micro immunofluorescence

Differential Diagnosis
- Adenoviral follicular conjunctivitis
- Spring catarrh
- Other follicular conjunctivitides

Complications
- Corneal ulcer
- Corneal opacity
- Trichiasis
- Entropion
- Blindness

8. MANAGEMENT

A. Treatment of Active Inflammatory Trachoma

Antibiotics (systemic)
- Azithromycin 20 mg/kg single dose (max 1 g)
- Erythromycin 250 mg QID or 500 mg BD for 14 days
- Doxycycline 100 mg BD for 10 days (≥ 8 yrs)

Topical therapy
- Tetracycline 1% eye ointment or
- Erythromycin 1% eye ointment

2 times daily for 6 weeks

B. Treatment of Cicatricial Trachoma

- Artificial tears for xerosis
- Trichiasis – Electrolysis / Cryolysis / Bilamellar tarsal rotation surgery
- Entropion – Surgical correction
- Corneal opacity – Penetrating keratoplasty

9. PREVENTION – SAFE STRATEGY (WHO)

S SURGERY — For trichiasis and entropion

A ANTIBIOTICS — Community-wide antibiotic therapy (Azithromycin)

F FACIAL HYGIENE — Face washing, clean face, avoid sharing towels

E ENVIRONMENTAL IMPROVEMENT — Better sanitation, fly control, improved water supply

10. ADULT INCLUSION CONJUNCTIVITIS

- **Etiology:** Serotypes D–K of *Chlamydia trachomatis*
- **Transmission:** Genital-hand-eye spread, swimming pools
- **Clinical features:**
  - Acute follicular conjunctivitis
  - Hyperemia, mucopurulent discharge
  - Preauricular lymphadenopathy
  - Superior keratitis

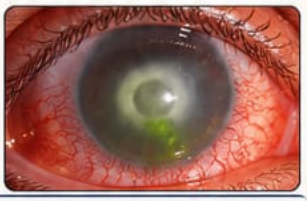
11. KEY POINTS

- Trachoma is caused by *Chlamydia trachomatis* (biovar TRIC). Mainly affects children in poor socioeconomic conditions.
- Spread is by direct contact, flies and fomites.
- Active disease shows follicles, papillae and pannus.
- Chronic disease leads to scarring, trichiasis, entropion and corneal opacity.
- WHO grading (FISTO) is used for diagnosis and surveillance.
- Treatment is with antibiotics and surgery for complications.
- SAFE strategy is key to global elimination of trachoma.

Dr Ashik Sureshkumar

MICROBIAL KERATITIS
(CONTACT LENS–ASSOCIATED CORNEAL ULCER)

A 25-year-old male complains of acute onset redness, pain, and photophobia in his right eye for the last two days. He has a history of contact lens use and admits to occasional non-compliance with lens hygiene. On examination, there is circumcorneal congestion, and fluorescein staining shows a central corneal ulcer with surrounding edema.



a) MOST LIKELY DIAGNOSIS

Microbial keratitis (bacterial corneal ulcer)

Most likely organism:
Pseudomonas aeruginosa

Points supporting diagnosis

- Acute onset pain, redness, photophobia
- Contact lens user with poor hygiene
- Circumcorneal congestion
- Central corneal ulcer with fluorescein staining
- Corneal edema

Fluorescein staining showing central ulcer

b) DIFFERENTIAL DIAGNOSES

1. Herpes simplex keratitis
2. Acanthamoeba keratitis

Other possible differentials:

- Fungal keratitis
- Sterile contact lens infiltrate
- Marginal keratitis

ETIOLOGICAL ORGANISMS IN CONTACT LENS USERS

Most common:

- *Pseudomonas aeruginosa*

Others:

- Staphylococcus aureus
- Serratia
- Acanthamoeba
- Fungal pathogens

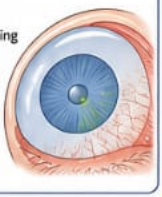
CLINICAL FEATURES OF BACTERIAL CORNEAL ULCER

Symptoms

- Severe pain
- Redness
- Watering
- Photophobia
- Blurred vision
- Foreign body sensation

Signs

- Circumcorneal congestion
- Corneal epithelial defect staining with fluorescein
- Stromal infiltrate
- Corneal edema
- Mucopurulent discharge
- Hypopyon (in severe cases)
- Reduced vision



c) MANAGEMENT

1. IMMEDIATE MEASURES

- Stop contact lens use immediately
- Remove and preserve lenses/lens case for culture
- Urgent ophthalmology referral

2. EVALUATION

- Visual acuity assessment
- Slit lamp examination
- Corneal scraping for:
  - Gram stain
  - KOH mount
  - Culture and sensitivity

3. MEDICAL TREATMENT

A. Intensive topical antibiotics
(For severe central ulcer)

- Fortified cefazolin 5% + fortified tobramycin/gentamicin 1.3% hourly

OR

- Monotherapy with topical fluoroquinolone:
  - Moxifloxacin 0.5%
  - Gatifloxacin 0.3%

B. Cycloplegics

- Atropine 1% or homatropine
- Relieves ciliary spasm and pain

C. Lubricants

- Preservative-free artificial tears
- Frequent lubrication

4. SUPPORTIVE MEASURES

- Oral analgesics for pain
- Avoid eye patching
- Close daily follow-up

5. AVOID

- Topical steroids initially
- Continued contact lens wear

6. SURGICAL MANAGEMENT
(if severe / non-responsive)

- Tissue adhesive
- Therapeutic keratoplasty

COMPLICATIONS

- Corneal perforation
- Descemetocele
- Endophthalmitis
- Corneal scarring
- Secondary glaucoma
- Permanent visual loss

FEATURES SUGGESTIVE OF PSEUDOMONAS ULCER

- Rapid progression
- Dense suppuration
- Greenish discharge
- Ring abscess
- Corneal melting / perforation

FEATURES SUGGESTIVE OF ACANTHAMOEBA KERATITIS

- Pain disproportionately severe compared to clinical findings
- Ring infiltrate
- History of swimming / tap water exposure with lenses

INDICATIONS FOR HOSPITAL ADMISSION

- Large central ulcer
- Threatened perforation
- Poor compliance
- Monocular patient
- Severe pain or vision loss

d) PREVENTIVE MEASURES FOR CONTACT LENS USERS

Proper hand hygiene before handling lenses

Regular cleaning and disinfection of lenses

Avoid overnight wear unless prescribed

Avoid using tap water on lenses

Replace lens cases regularly

Avoid swimming / showering with lenses

Follow replacement schedule strictly

Regular ophthalmic follow-up

Discontinue lens use during eye irritation / infection

KEY POINT

Contact lens–associated **microbial keratitis** is an **ophthalmic emergency** requiring prompt microbiological evaluation and intensive topical antimicrobial therapy to prevent corneal scarring and blindness.

Dr Ashik Sureshkumar

ACANTHAMOEBA KERATITIS

(Amoebic Keratitis)

A sight-threatening protozoal infection of the cornea, commonly associated with contact lens use and water exposure.

ETIOLOGY

- Caused by free-living protozoan amoeba *Acanthamoeba* (most commonly *A. castellanii, A. polyphaga, A. hatchetti*)
- Not a bacterium
- Ubiquitous in nature – soil, dust, freshwater, tap water, swimming pools, hot tubs, contact lens solutions (contaminated)
- Forms cysts (resistant) and trophozoites (active form)

Acanthamoeba trophozoite

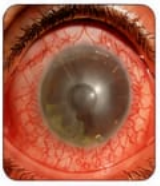
RISK FACTORS

- Contact lens wear, especially poor hygiene
- Exposure to tap water – swimming, showering, washing face
- Use of tap water or non-sterile solutions for cleaning lenses
- Hot tubs, swimming pools, rivers, ponds

CLINICAL PEARL

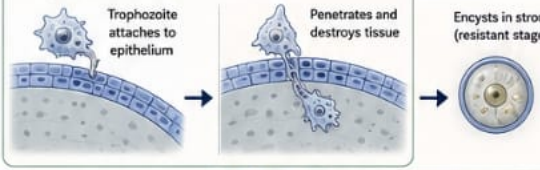
"Pain out of proportion to clinical findings"

Severe pain, often disproportionately severe compared to the signs.



PATHOGENESIS

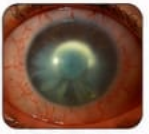
- Trophozoites attach to corneal epithelium → penetrate via epithelium or through micro-erosions
- Cause cytolysis, stromal necrosis and inflammation
- Spread along corneal nerves → severe pain
- Encyst in stroma → resistant to treatment



CLINICAL FEATURES

Symptoms

- Severe ocular pain (disproportionate)
- Redness
- Photophobia
- Foreign body sensation
- Tearing, blurred vision
- Decreased vision



Ring infiltrate

Signs

- Circumcorneal congestion
- Radial keratoneuritis (pathognomonic)
- Ring-shaped stromal infiltrate
- Epithelial defect
- Corneal edema
- Perineural infiltrates
- Punctate epithelial erosions
- Hypopyon (in advanced cases)

Radial keratoneuritis

DIAGNOSIS

- Clinical suspicion in at-risk patient with characteristic features (especially pain out of proportion)
- Slit lamp examination
- Corneal scraping for:
  - Wet mount: look for trophozoites (motile)
  - Calcofluor white stain: cysts appear bright refractile (double-walled)
  - Culture on non-nutrient agar with *E. coli* overlay
  - PCR (if available)
- In vivo confocal microscopy (highly sensitive)
- Histopathology (rarely required)

Cyst on Calcofluor white stain

Double-walled cyst (light microscopy)

DIFFERENTIAL DIAGNOSIS

- Bacterial keratitis (e.g., *Pseudomonas*)
- Fungal keratitis
- Herpes simplex keratitis
- Fungal marginal keratitis
- Sterile contact lens infiltrate
- Mooren's ulcer

TREATMENT (Prolonged and aggressive therapy is essential)

1. Anti-amoebic therapy (Topical)

- Polyhexamethylene biguanide (PHMB) 0.02% – 0.06%
- Chlorhexidine 0.02%
- Propamidine isethionate 0.1%
- Neomycin 3%

Typically, dual therapy (PHMB + Chlorhexidine) is used

2. Supportive therapy

- Cycloplegics (e.g., Atropine 1%)
- Lubricants
- Oral analgesics
- Avoid topical steroids initially (may worsen infection)

3. Adjuncts (in severe / recalcitrant cases)

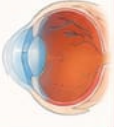
- Oral Itraconazole/Fluconazole (some benefit)
- Corneal debridement
- Therapeutic penetrating keratoplasty (in non-responsive or advanced cases)

Treatment is prolonged (months) – continue till lesions are inactive and cultures negative

Regular follow-up is crucial

COMPLICATIONS

- Corneal scarring
- Persistent epithelial defect
- Corneal perforation
- Descemetocele
- Secondary glaucoma
- Endophthalmitis (rare)
- Permanent visual loss



KEY DIFFERENCE FROM BACTERIAL KERATITIS

Feature	Acanthamoeba keratitis	Bacterial keratitis
Organism	Protozoan amoeba	Bacteria
Pain	Severe, out of proportion	Severe, proportionate
Infiltrate	Ring infiltrate	Suppurative ulcer
Risk factors	Water exposure + contact lens	Contact lens misuse
Response to antibiotics	Poor	Good (if sensitive)

PREVENTION

- Proper hand hygiene before handling lenses
- Use only sterile contact lens solutions
- Avoid tap water for cleaning/rinsing lenses
- Avoid swimming/showering with lenses
- Regular cleaning and timely replacement of lenses and cases
- Avoid overnight wear (unless prescribed)
- Discontinue lens use if eye irritation occurs
- Regular eye check-ups

ONE-LINE TAKE HOME MESSAGE

Think of Acanthamoeba keratitis in any contact lens wearer with severe pain disproportionate to signs, especially with water exposure – early diagnosis and prolonged anti-amoebic therapy can save vision.

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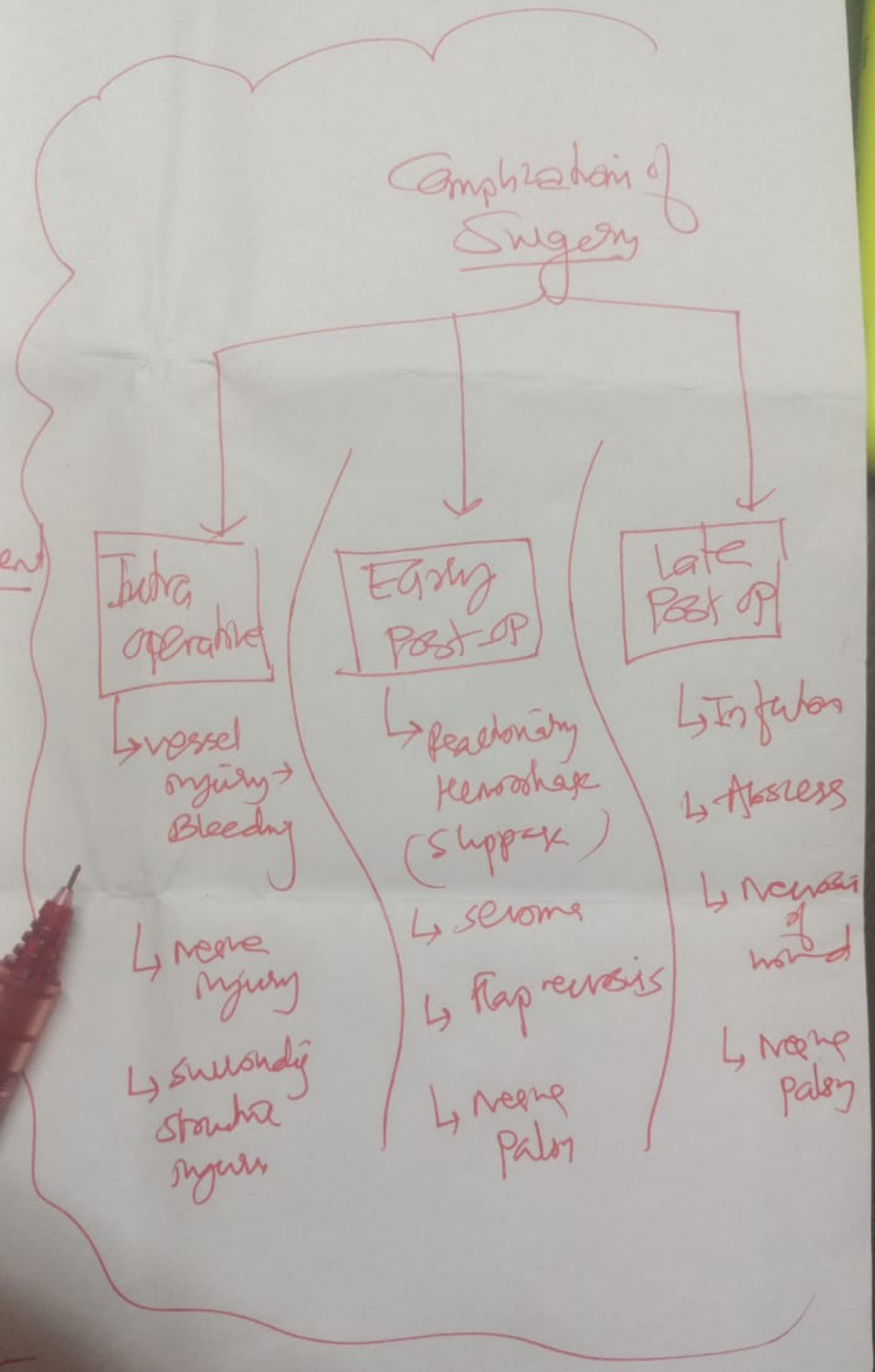
Disease

- Defn
- Etiology
- Pathology
- Clinical feature
  - Symptoms:
  - Signs
- Investigation
  - ↳ Routine
  - ↳ Imaging
  - ↳ Invasive
  - ↳ Definite
- Treatment
  - ↳ Lifestyle modification
  - ↳ Medical
  - ↳ Surgical
  - ↳ Advanced

Management


Complication of Surgery
Intra operative
Early Post-op
Late Post op
vessel injury → Bleeding
nerve injury
surrounding structure injury
Reactionary Hemorrhage (Slipped)
seroma
flap necrosis
wound dehiscence
Infection
Abscess
Necrosis of wound
Nerve palsy
Nerve palsy

Surgery

1. Defn →
2. Indication →
3. Contraindication →
4. Anaesthesia → GA / LA
5. Position
6. Technique / steps → 1. 2. 3. 4.
7. Post op care
   - ↳ NPO
   - ↳ IV fluid
   - ↳ Pain medicine
8. Complications
   - ↳ Intraop — Bleeding, nerve injury
   - ↳ Early post op — Bleeding, seroma
   - ↳ Late - Infection, Failure

STEPS IN CONSENT WRITING FOR SURGERY

DEFINITION: Informed surgical consent is a voluntary authorization given by a competent patient after understanding the nature, benefits, risks, alternatives, and consequences of a surgical procedure.

ETHICAL OBLIGATION | LEGAL REQUIREMENT | ESSENTIAL COMPONENT OF PATIENT AUTONOMY

COMPONENTS OF INFORMED CONSENT

1. Disclosure
2. Understanding
3. Capacity/Competence
4. Voluntariness
5. Authorization

CHARACTERISTICS OF VALID CONSENT

- ✔ Informed
- ✔ Voluntary
- ✔ Specific
- ✔ Written
- ✔ Competent

TYPES OF CONSENT

Type	Example
Implied consent	Physical examination
Express verbal consent	Minor procedures
Written consent	Surgery / anesthesia
Informed consent	Major surgery
Proxy consent	Guardian consent

STEPS IN SURGICAL CONSENT WRITING

1 IDENTIFICATIONS OF PATIENT

- Name
- Age / Sex
- Hospital number
- Diagnosis

2 NAME OF PROCEDURE

Procedure should be written clearly and specifically.

Examples:
- Open appendicectomy
- Tracheostomy
- Modified radical mastectomy

Avoid abbreviations.

3 EXPLANATION OF DIAGNOSIS

- Nature of disease
- Indication for surgery
- Consequences if untreated

4 EXPLANATION OF PROCEDURE

- Nature of surgery
- Type of anesthesia
- Major operative steps
- Expected duration of surgery

Use simple and understandable language.

5 BENEFITS OF SURGERY

- Curative
- Diagnostic
- Palliative
- Functional improvement
- Symptom relief

6 RISKS AND COMPLICATIONS

COMMON COMPLICATIONS
- Pain
- Bleeding
- Infection
- Scar formation

PROCEDURE-SPECIFIC COMPLICATIONS

Examples:
- Facial nerve injury
- Hearing loss
- Anastomotic leak
- Voice change

SERIOUS COMPLICATIONS
- ICU admission
- Reoperation
- Organ failure
- Death

High-risk complications must be specifically mentioned.

7 ALTERNATIVES TO SURGERY

- Medical treatment
- Observation
- Other procedures

8 CONSEQUENCES OF REFUSAL

Patient should understand possible consequences if surgery is refused.

Examples:
- Disease progression
- Organ dysfunction
- Mortality risk

9 BLOOD TRANSFUSION CONSENT

Mention possibility of:
- Blood transfusion
- Blood products
- Associated risks

Separate consent may be required.

10 ANESTHESIA CONSENT

Separate anesthesia consent required.

Explain:
- Type of anesthesia
- Risks
- Possible complications

11 POSSIBILITY OF ADDITIONAL PROCEDURES

Mention possibility of:
- Conversion from laparoscopic to open surgery
- Stoma creation
- Organ removal
- Extension of procedure if needed intraoperatively

12 POSTOPERATIVE COURSE

- ICU stay
- Catheters / drains
- Ventilator support
- Recovery period
- Rehabilitation

13 OPPORTUNITY FOR QUESTIONS

Patient / guardian should be allowed to:
- Ask doubts
- Clarify concerns

14 VOLUNTARY AGREEMENT

Consent should be:
- Free from coercion
- Free from pressure
- Voluntary

Patient can withdraw consent anytime before surgery.

15 DOCUMENTATION

Consent form should contain:
- Patient signature / thumb impression
- Doctor signature
- Witness signature
- Date and time

CAPACITY TO CONSENT

Patient must be able to:
- Understand information
- Retain information
- Weigh risks and benefits
- Communicate decision

WHO CAN GIVE CONSENT?

Adult competent patient
Age > 18 years

Proxy / Surrogate Consent

Needed in:
- Minors
- Unconscious patients
- Mentally incapacitated patients

Can be given by:
- Parent
- Spouse
- Legal guardian

HIGH-RISK CONSENT

Should specifically mention:
- ICU admission
- Ventilator support
- Major disability
- Organ failure
- Mortality risk

SPECIAL CONSENTS

Separate consent may be required for:
- Blood transfusion
- Organ donation/removal
- Sterilization
- Photography / video recording
- Research participation

WITHDRAWAL OF CONSENT

Patient can withdraw consent at any time before surgery, even after signing, provided patient is mentally competent.

REFUSAL OF TREATMENT

If patient refuses:
- Explain consequences clearly
- Document refusal
- Obtain patient / witness signature if possible

LANGUAGE OF CONSENT

Consent should:
- Be in language understood by patient
- Avoid medical jargon
- Use interpreter if necessary

WITNESS IN CONSENT

Especially important in:
- Illiterate patients
- High-risk procedures
- Thumb impression may be used
- Independent witness preferred

AUDIO-VISUAL CONSENT

May be required in:
- Clinical trials
- Certain high-risk procedures

IMPORTIONS WHEPLES SURGENT IS NOT REQUIRED

EMERGENCY DOCTRINE

Treatment may proceed without consent when:
- Life-threatening emergency exists
- Patient unconscious
- Delay may endanger life

THERAPEUTIC PRIVILEGE

Rare situation where disclosure may severely harm patient psychologically. Used very cautiously.

COMMON MEDICO-LEGAL ERRORS

- Incomplete consent
- Blanket / general consent
- Wrong-site consent
- Illegible documentation
- Failure to mention complications
- Consent after sedation
- Undated consent

WHO SURGICAL SAFETY CHECKLIST

- **SIGN IN** — Before induction of anesthesia
- **TIME OUT** — Before incision
- **SIGN OUT** — Before patient leaves operating room

Helps improve:
- Patient safety
- Team communication
- Reduction in surgical errors

DOCUMENTATION PEARLS

- Use blue / black ink
- Avoid overwriting
- Corrections should be countersigned
- Mention date and time
- Mention surgeon and procedure clearly

IMPORTANT PRINCIPLES OF SURGICAL CONSENT

- Consent is procedure-specific.
- Blanket consent is invalid.
- Consent for anesthesia is separate.
- Consent should be taken before premedication / sedation.
- Patient autonomy must be respected.

CONCLUSION

Proper surgical consent is an ethical, legal, and professional obligation. A valid informed consent establishes patient autonomy, improves doctor–patient communication, and protects both patient and surgeon medico-legally. Accurate documentation, proper disclosure of risks, and voluntary participation are essential for safe surgical practice.

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atrophy).

III. Primary Angle-Closure Glaucoma

Pathogenesis

Primary angle-closure glaucoma (PACG) results from gradual synechial closure of the angle of anterior chamber. Untreated patients with PAC may over the period covert to PACG with or without history of subacute or acute attack of PAC.

Clinical features

Clinical features of PACG given below, are similar to that of POAG except that angle closure is present:

- *Intraocular pressure (IOP)* remains constantly raised.
- *Eyeball* remains white (no congestion) and painless.
- *Optic disc* shows glaucomatous cupping.
- *Visual field defects* similar to POAG occur (*See* page 218).
- *Gonioscopy* reveals more than 270° of angle closure along with peripheral anterior synechiae (PAS).

Clinical diagnostic impression: Angle is abnormal in function (elevated IOP) and structure (PAS + ve) with optic neuropathy.

Treatment

POAG vs PACG

HOW TO DIFFERENTIATE AND SOLVE QUESTIONS

Based on a Clinical Question

CLINICAL QUESTION

A 57 years female presented to OPD with history of diminution of vision since 2 years. On examination patient was found to have **IOP of 52 mmHg, biarcuate scotoma** on Humphrey visual field analysis.

KEY CLUE FROM THE CASE

- Gradual diminution of vision over 2 years (chronic)
- No pain, no halos, no redness, no vomiting
- Biarcuate scotoma on visual field
- Raised IOP (52 mmHg)

MOST PROBABLE DIAGNOSIS

PRIMARY OPEN ANGLE GLAUCOMA (POAG)

Because chronic course + typical glaucomatous field defect (biarcuate scotoma) + absence of acute symptoms of angle closure strongly suggest POAG.

POAG vs PACG – KEY COMPARISON

FEATURE	POAG (Primary Open Angle Glaucoma)	PACG (Primary Angle Closure Glaucoma)
Onset & Course	Insidious, chronic, slowly progressive (Years)	Acute (sudden attack) or intermittent (Subacute/Chronic)
Symptoms	Usually asymptomatic early Gradual diminution of vision	Severe eye pain, redness, headache, halos around lights, nausea, vomiting, sudden diminution of vision
Age	> 40 years	Usually > 40 years (acute more in hyperopes and elderly females)
IOP	Raised (can be very high in advanced cases)	Very high during acute attack
Cornea	Clear	Early: Clear Acute attack: Corneal edema
Anterior Chamber	Normal depth	Shallow peripherally, may be shallow centrally in acute attack
Pupil	Normal, reactive	Mid-dilated, sluggish in acute attack
Gonioscopy	Open angle (SS grade III–IV)	Narrow/closed angle (SS grade 0–I)
Optic Disc	Cupping, increased C:D ratio, notching, rim thinning	Similar (cupping in chronic/advanced cases)
Visual Field Defects	Paracentral scotoma, nasal step, arcuate/Bjerrum scotoma, biarcuate scotoma (classic)	Early: May be normal or non-specific Later: Similar glaucomatous defects
Systemic Association/Risk Factors	Family history, myopia, older age	Hypermetropia, shallow AC, female, older age, crowded anterior segment
Typical Presentation in Exam	Chronic visual loss + raised IOP + arcuate/biarcuate scotoma	Painful red eye + halos + vomiting + raised IOP + shallow AC
Management (Initial)	IOP lowering (medications), Laser/filters as needed	Acute attack: IOP reduction first + definitive treatment (PI/laser iridotomy) Chronic: medical/surgical

HIGH-YIELD POINTS TO SOLVE QUESTIONS

- If the case mentions chronic, gradual, painless diminution of vision with arcuate scotoma → Think POAG.
- If the case mentions acute pain, redness, halos, vomiting, shallow AC → Think PACG.
- Biarcuate scotoma on visual field is typical for chronic open angle glaucoma.
- Gonioscopy is the gold standard to differentiate – Open angle (POAG) vs Closed/Narrow angle (PACG).
- High IOP can be present in both, but symptoms and angle findings are the key.

GONIOSCOPY – WHAT IT SHOWS

OPEN ANGLE (POAG)
SS Grade III–IV

CLOSED / NARROW ANGLE (PACG)
SS Grade 0–I

REMEMBER THIS DIFFERENCE IN ONE LINE

POAG
"Silent thief of sight" – Chronic, painless, open angle, typical field defects (arcuate, biarcuate).

VS

PACG
"Emergency in eye" – Acute, painful, closed angle, halos, vomiting.

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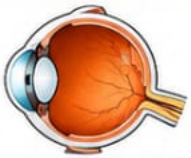
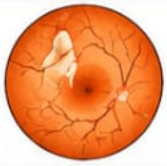


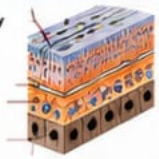
RETINAL DETACHMENT

SEPARATION OF NEUROSENSORY RETINA FROM THE PIGMENT EPITHELIUM



ANATOMY (NORMAL)

- Two layers are loosely attached:
  1. Neurosensory retina
  2. Retinal pigment epithelium (RPE)
- Potential space in between



CLASSIFICATION (CLINICO-ETIOLOGICAL)

1. Rhegmatogenous (Primary) RD
2. Tractional RD
3. Exudative RD

CLINICAL FEATURES – GENERAL

- Photopsia (flashes of light)
- Floaters (dark spots)
- Field defect (curtain / veil / shadow)
- Progressive loss of vision

① RHEGMATOGENOUS (PRIMARY) RETINAL DETACHMENT

Most common type

ETIOLOGY / PREDISPOSING FACTORS

- Age: Most common 40–60 yrs
- Male > Female (M:F = 3:2)
- Myopia (≈ 40% cases)
- Aphakia / Pseudophakia
- Lattice degeneration
- Trauma
- Senile posterior vitreous detachment (PVD)
- Family history

PATHOGENESIS

PVD / Trauma / Lattice degeneration (holes / tears in retina)

↓

Subretinal fluid (SRF) passes through the retinal break (hole or tear)

↓

Separates the neurosensory retina from RPE

TYPES OF RETINAL BREAKS

SIGNS

1. External exam – usually normal
2. IOP – normal or slightly low
3. Marcus Gunn pupil – relative afferent pupillary defect (extensive RD)
4. Plane mirror exam / Distant ophthalmoscopy – reflex altered in detached area (greyish reflex)
5. Indirect ophthalmoscopy is the best method
6. Grey reflex of detached retina (convex configuration)
7. Folds / Corrugations / Ballooned retina in large RD
8. Retinal vessels – dark, tortuous, oscillating
9. Retinal breaks – often in upper temporal quadrant
10. Shaffer sign – pigment in anterior vitreous
11. Scleral indentation – to localize breaks

SYMPTOMS – LOCALISED RELATIVE LOSS IN FIELD OF VISION (EARLY)

- Noted in early stages
- Progresses to total loss when macula involved

INVESTIGATIONS

1. Detailed ocular & systemic evaluation
2. B-scan ultrasonography – useful when media hazy
3. Fundus fluorescein angiography – for break localization
4. OCT – for macula status
5. CT / MRI – in intraocular tumours

MANAGEMENT (RHEGMATOGENOUS RD)

STAGE 1 (Asymptomatic Holes / Tears)	STAGE 2 (Recent onset RD < 1 year, Macula-off)	STAGE 3 (Chronic RD > 1 year) Or PVR / Complicated RD
No treatment (observe) Most close spontaneously Follow up in 50% cases	• Surgery recommended • Options: – Scleral buckling – Pars plana vitrectomy (PPV) – Pneumatic retinopexy (selected cases) • Face down position 7–14 days	Surgery mandatory PPV + Membrane peeling + Endolaser + Tamponade (Silicone oil / Gas)

COMPLICATIONS

- Cataract
- PVR (proliferative vitreoretinopathy)
- Uveitis
- Phthisis bulbi
- Re-detachment
- Raised IOP / Glaucoma
- Endophthalmitis

PROGNOSIS

- Better if macula on at presentation
- Poorer in chronic RD / PVR / Large breaks / Multiple operations

② TRACTIONAL RETINAL DETACHMENT

CAUSES

- Proliferative diabetic retinopathy (most common)
- Post-traumatic scarring
- Post-haemorrhagic retinitis proliferans
- Retinopathy of prematurity
- Sickle cell retinopathy
- Eales' disease
- Vitreomacular traction syndrome

PATHOGENESIS

Fibrovascular membrane on retina contracts → exerts traction → retinal detachment

(Usually does not cross ora serrata)

CLINICAL FEATURES

- Gradual, painless loss of vision
- Floaters, flashes may be absent
- Severe reduction in vision
- Fundus: Elevated retina with folds, fibrovascular proliferation

TREATMENT

- Pars plana vitrectomy
- Membrane peeling
- Endolaser photocoagulation
- Tamponade (Silicone oil / Gas)

③ EXUDATIVE (SEROUS) RETINAL DETACHMENT

DEFINITION

Accumulation of subretinal fluid without any retinal break or traction.

CLINICAL FEATURES

- Painless, gradual diminution of vision
- No photopsia, no floaters
- Mild hypermetropization
- Fundus: Smooth, dome-shaped elevation of retina

MANAGEMENT

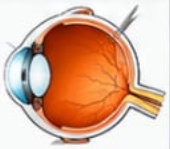
Treat the underlying cause (Usually resolves when cause is treated)

CAUSES

1. Systemic diseases
   - Toxaemia of pregnancy
   - Hypertension, Blood dyscrasias
   - Polyarteritis nodosa
2. Ocular diseases
   - Nanophthalmos, Optic pit, Coloboma
   - Familial exudative vitreoretinopathy (FEVR)
   - Harada's disease, Sympathetic ophthalmia
   - Orbital cellulitis
3. Vascular causes
   - CSR (Central serous retinopathy)
   - Coats' disease
   - Malignant melanoma of choroid
   - Haemangioma, Metastatic tumours
4. Others
   - Sudden hypotony (perforation, surgery)
   - Uveal effusion syndrome
   - Choroidal neovascularization

SURGICAL PRINCIPLES (RHEGMATOGENOUS RD)

1. Seal the retinal breaks
2. Reduce vitreous traction
3. Drain / remove SRF
4. Reattach the retina
5. Provide internal / external tamponade
6. Prevent re-detachment



POSTOPERATIVE POSITIONING

- Face down position in macula-off RD
- Variable duration: 3–14 days
- Depends on tamponade used (Gas bubble / Silicone oil)

IMPORTANT POINTS

- Early diagnosis improves prognosis
- Indirect ophthalmoscopy + scleral indentation is gold standard
- Upper temporal quadrant is the commonest site of breaks
- Macula status at presentation determines visual outcome

RED FLAG SYMPTOMS

- Sudden floaters
- Flashes of light
- Curtain / veil in vision

→ Urgent ophthalmic referral

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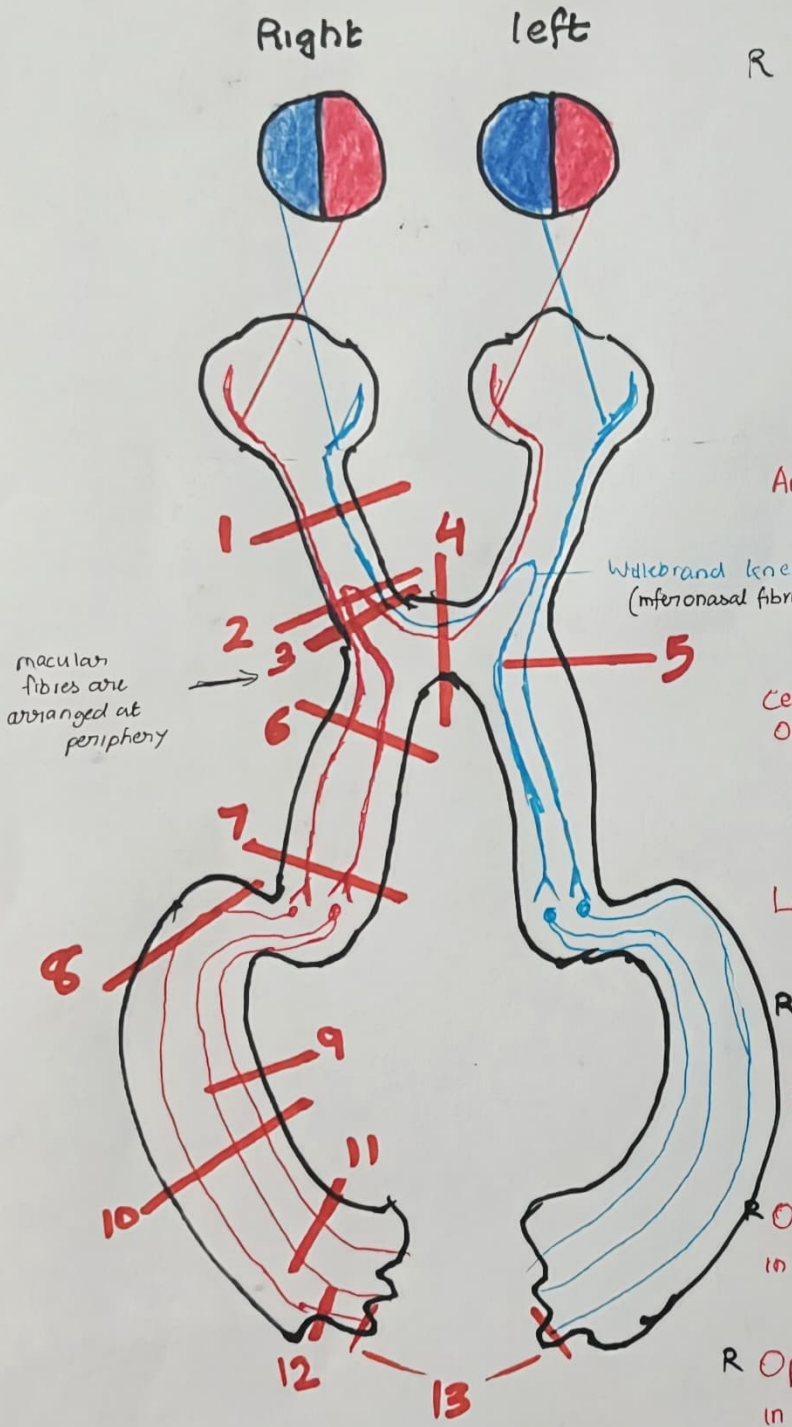
VISUAL PATHWAY



R Optic nerve 1

Ⓡ Ipsilateral blindness

anterior chiasma 2

blindness

~~Junctional scotoma~~

Anterior chiasma 3

Central Scotoma hemianopia

Junctional scotoma

Central Optic chiasma 4

Bitemporal hemianopia

Lateral chiasma 5

Binasal hemianopia

R Optic tract geniculate body 6,7

Ⓛ Homonymous hemianopia

R Optic radiations in temporal lobe 8

Superior quadrantopia

R Optic radiations in parietal lobe 9

Ⓛ inferior quadrantopia

R Entire optic radiations 10

Homonymous hemianopia

R Visual cortex sparing macula 11

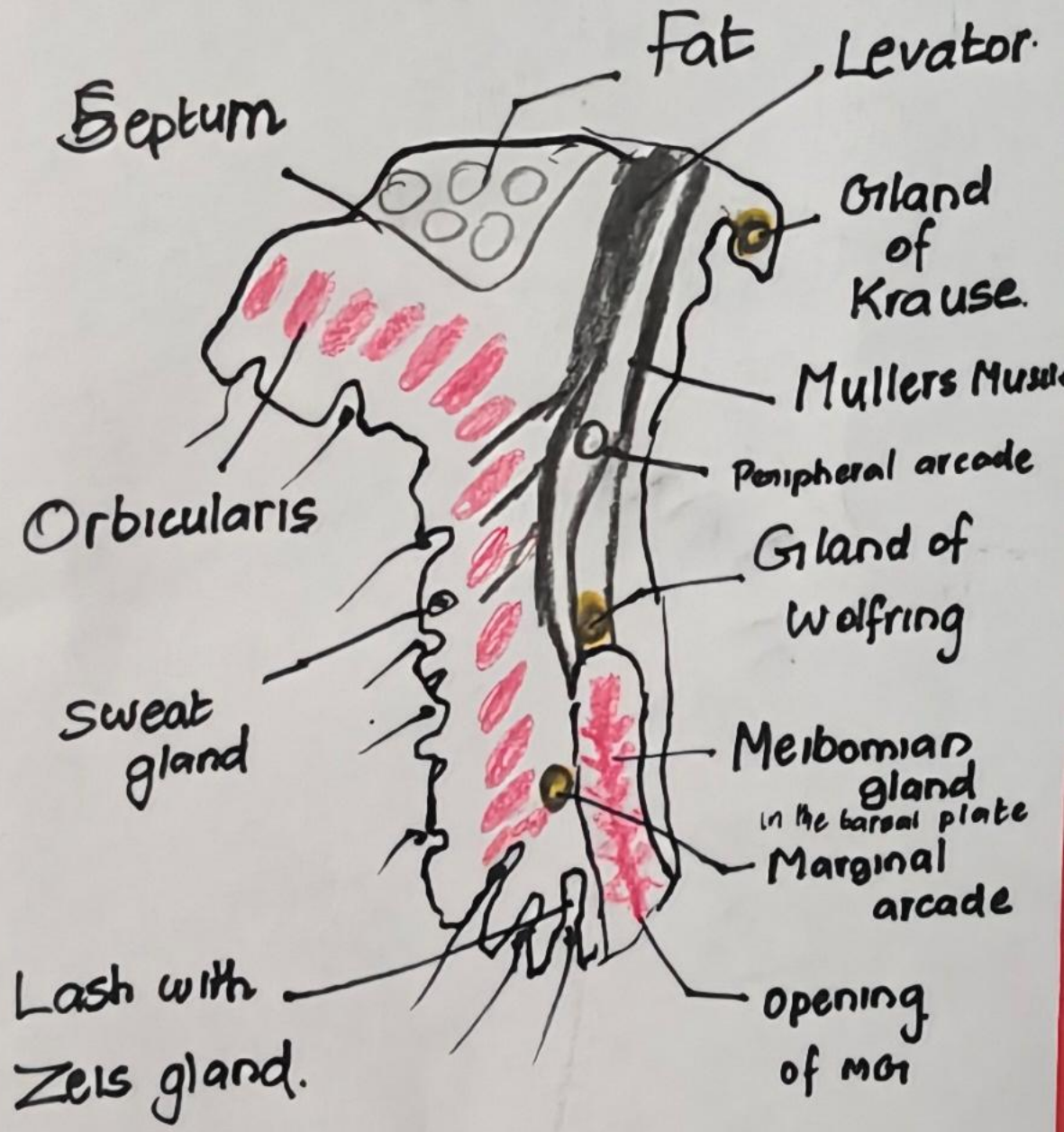
Contralateral H Heminopia with macular sparing

R Visual cortex only macula 12

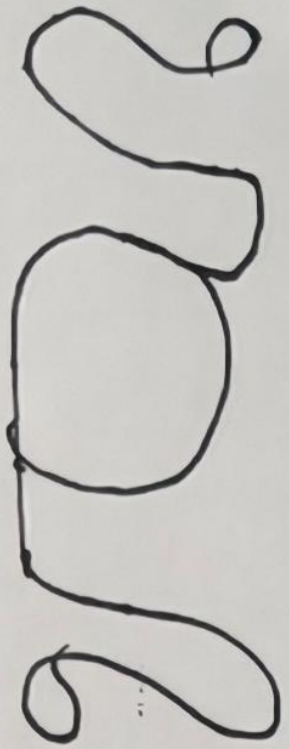
Bilateral visual cortex only macula 13

maculas defects

EYELID


Fat
Levator
Septum
Gland of Krause.
Mullers Muscle
Peripheral arcade
Orbicularis
Gland of Wolfring
Sweat gland
Meibomian gland in the tarsal plate
Marginal arcade
Lash with Zeis gland.
opening of MG

TYPES OF IOL



ANTERIOR CHAMBER IOL

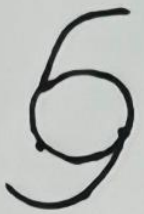
Singh & Worst Iris claw lens

PC IOL Modified C loop

PC IOL Quadriloop

Aniridia IOL

Toric

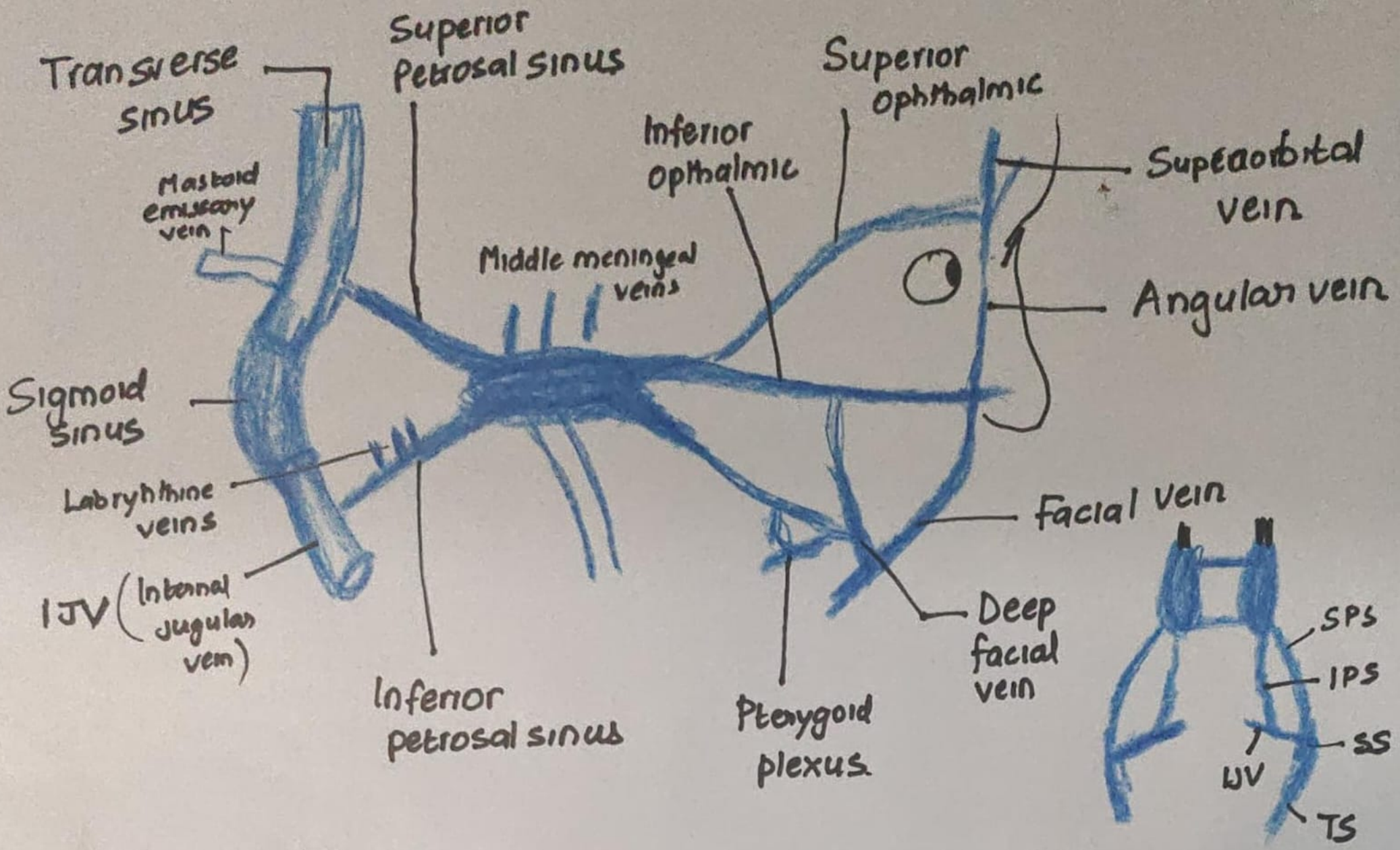


Unifocal

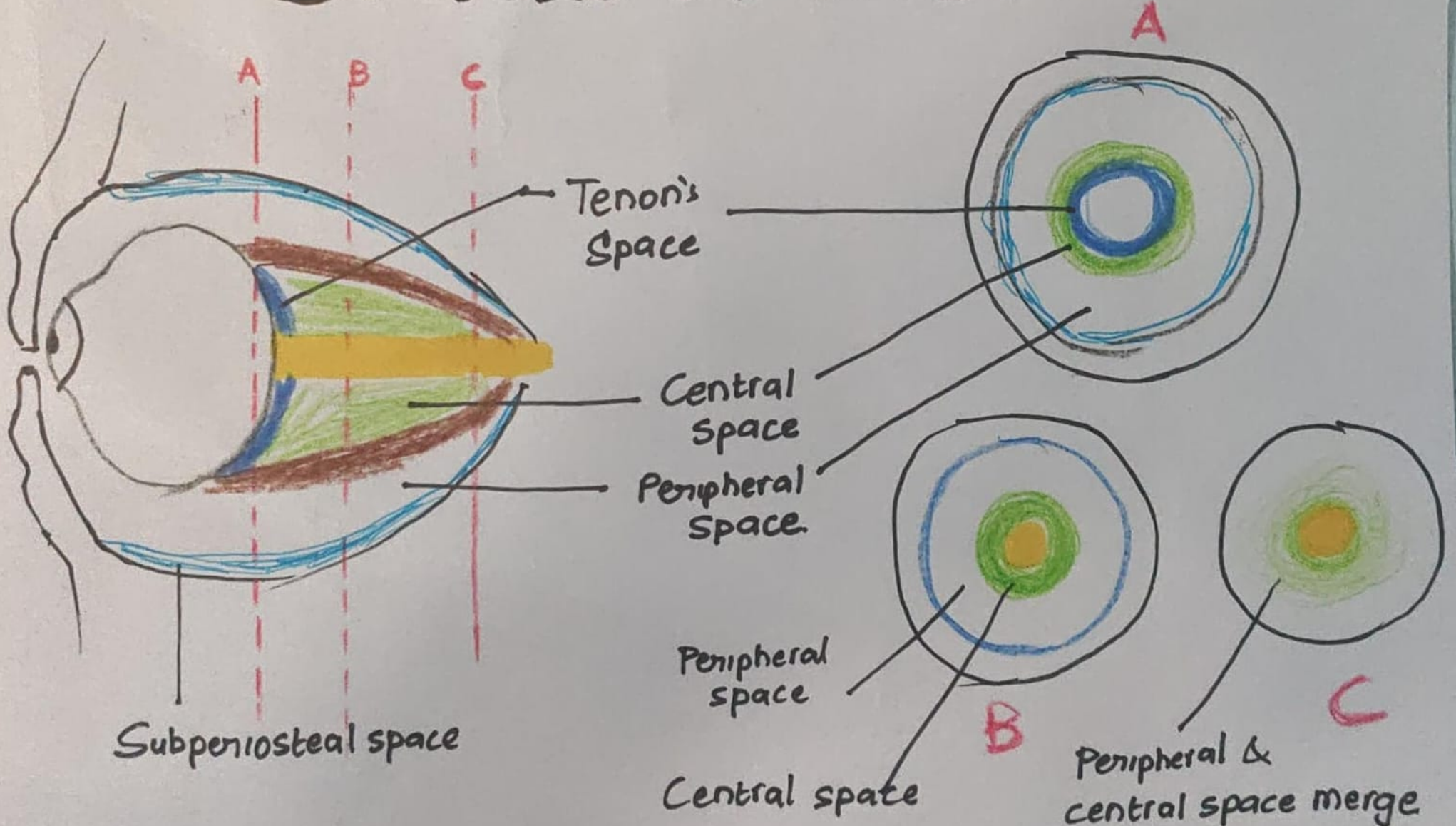
Bifocal

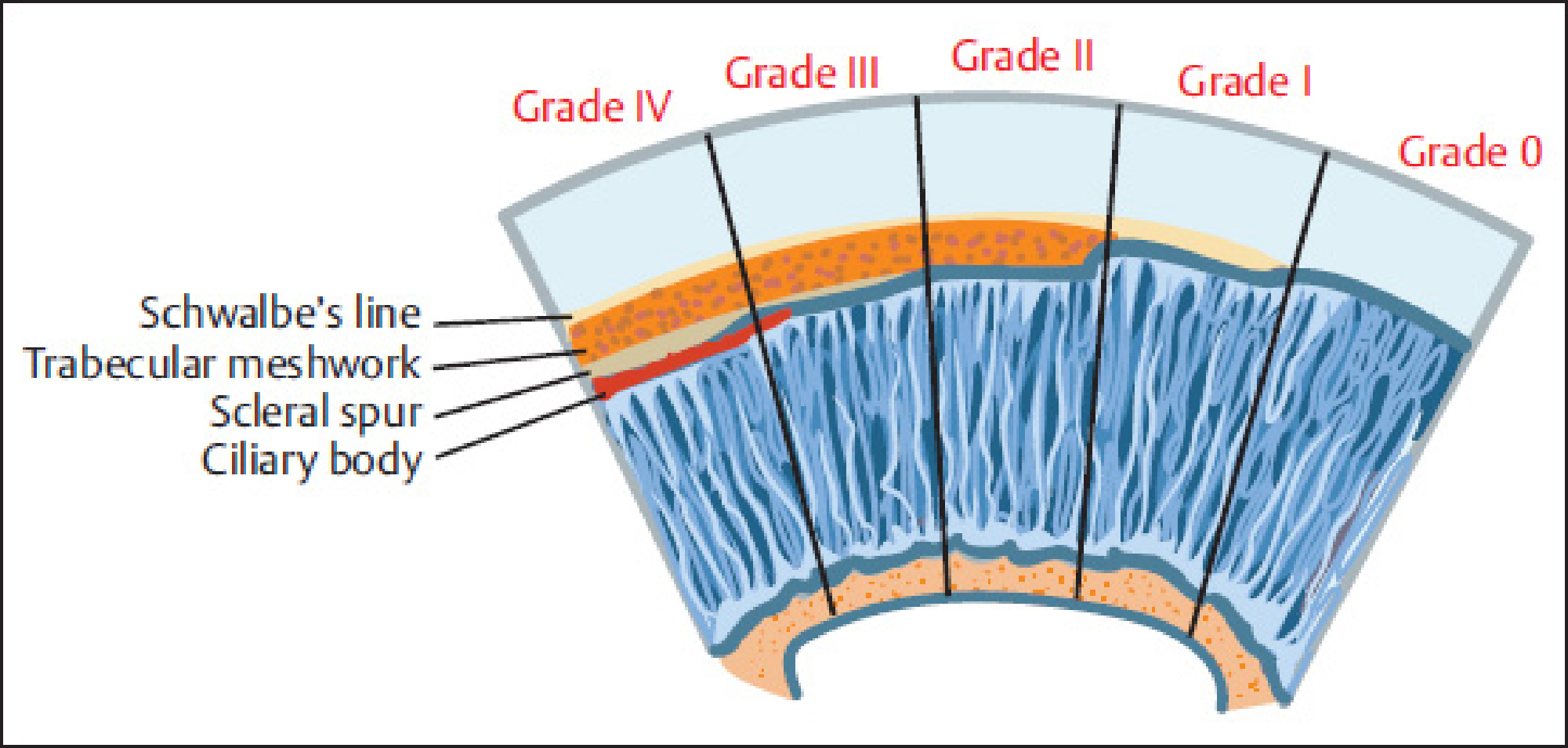
Multifocal

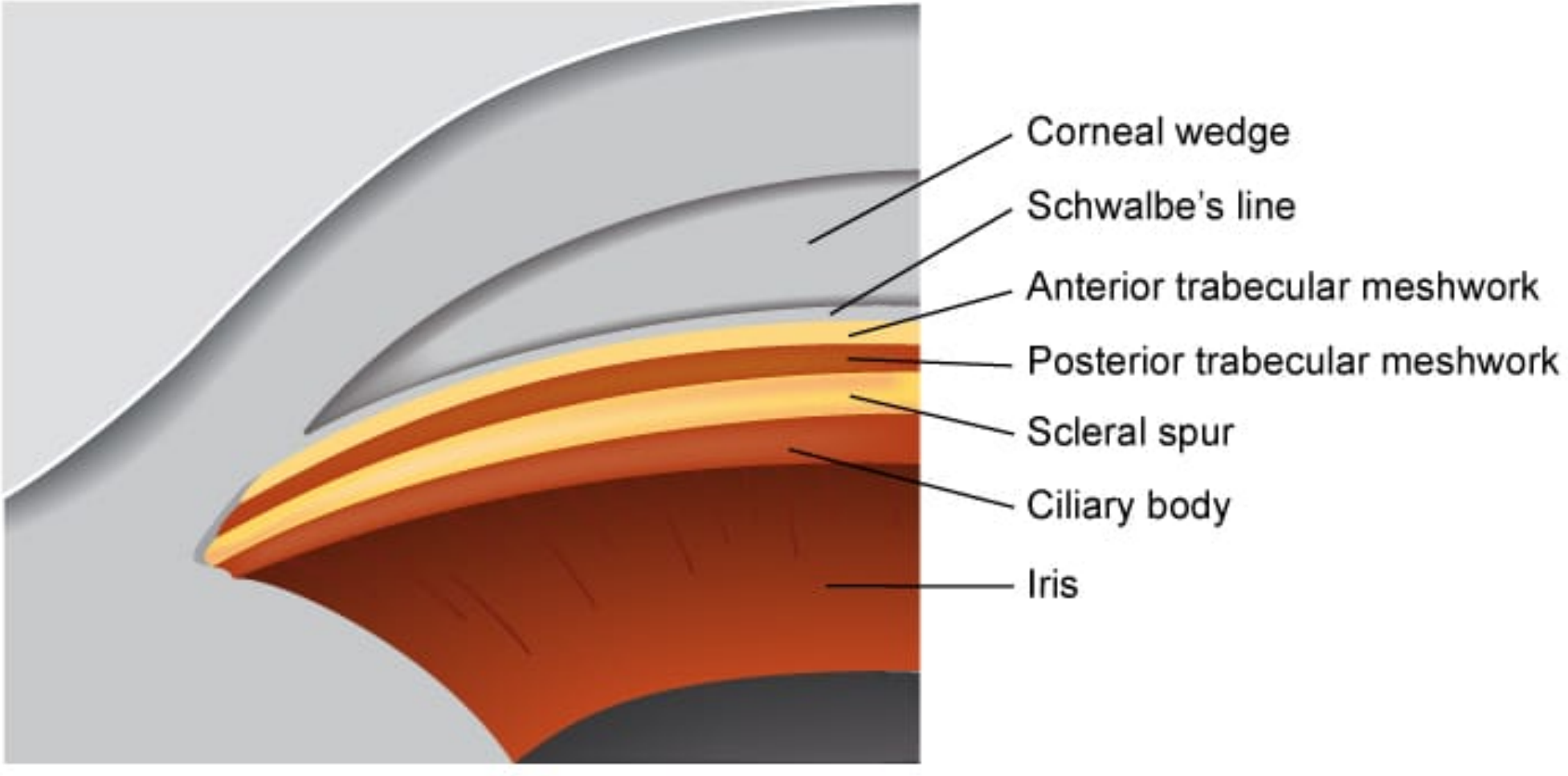
CAVERNOUS SINUS CONNECTIONS



SPACES OF ORBIT




Grade IV
Grade III
Grade II
Grade I
Grade 0
Schwalbe's line
Trabecular meshwork
Scleral spur
Ciliary body


Corneal wedge
Schwalbe's line
Anterior trabecular meshwork
Posterior trabecular meshwork
Scleral spur
Ciliary body
Iris

OPHTHALMOLOGY CASE ESSAY

Q. 60 years old female patient with type II diabetic mellitus since 12 years, presents to OPD with complaining of sudden painless loss of vision in one eye. Answer the following:

- What is your more probable diagnosis
- What are the differential diagnosis
- What are the clinical features of the condition
- How will you confirm the diagnosis
- How will you manage the case

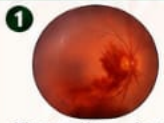
MOST PROBABLE DIAGNOSIS

VITREOUS HEMORRHAGE SECONDARY TO PROLIFERATIVE DIABETIC RETINOPATHY (PDR)

Why?

- ☑ Long-standing type 2 DM (12 years)
- ☑ Sudden painless unilateral loss of vision
- ☑ Diabetic retinopathy → Neovascularization
- ☑ Fragile vessels → Vitreous bleed

DIFFERENTIAL DIAGNOSIS

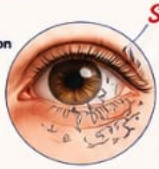


1. Vitreous Hemorrhage (PDR)
2. Central Retinal Artery Occlusion (CRAO)
3. Central Retinal Vein Occlusion (CRVO)
4. Retinal Detachment
5. Macular Hemorrhage / Diabetic Macular Edema
6. Anterior Ischemic Optic Neuropathy (AION)
7. Posterior Vitreous Detachment
8. Dense Cataract
9. Occipital Stroke (Rare)

CLINICAL FEATURES OF VITREOUS HEMORRHAGE

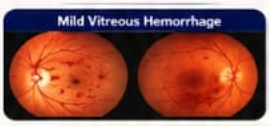
Symptoms

- Sudden painless loss of vision
- Floaters, cobwebs
- Black spots, red haze
- Decreased vision
- Flashes (may be present)
- Visual field defects



Signs (Examination)

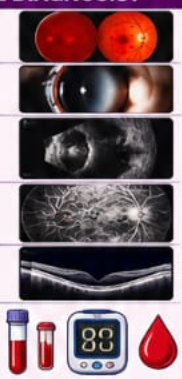
- Visual acuity ↓
- Fundus hazy view (mild VH)
- Dense VH → No fundus view
- Red reflex reduced or absent



Associated with Proliferative Diabetic Retinopathy

HOW WILL YOU CONFIRM THE DIAGNOSIS?

1. **Ophthalmoscopy / Fundus Examination**
   Primary diagnostic step
2. **Slit Lamp Biomicroscopy**
   Shows blood cells in vitreous
3. **B-Scan Ultrasonography**
   (Very important if fundus not visible)
   Shows echogenic particles in vitreous
4. **Fundus Fluorescein Angiography (FFA)**
   (After clearing of hemorrhage)
   to assess ischemia & neovascularization
5. **Optical Coherence Tomography (OCT)**
   To assess macular edema
6. **Blood Investigations**
   - Blood sugar (FBS, PPBS, HbA1c)
   - BP, Renal function tests, Lipid profile



HOW WILL YOU MANAGE THE CASE?

A. INITIAL MANAGEMENT

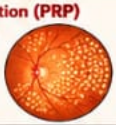
- ✓ Bed rest with head elevation
- ✓ Avoid strenuous activity & heavy lifting
- ✓ Control blood sugar (HbA1c < 7%)
- ✓ Control hypertension & lipids
- ✓ Stop anticoagulants if medically permissible

B. TREAT UNDERLYING CAUSE (Proliferative Diabetic Retinopathy)

1. Pan-Retinal Photocoagulation (PRP)

Treatment of choice once retina visible

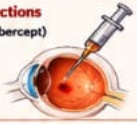
- Regress neovascularization
- Prevent recurrent bleed



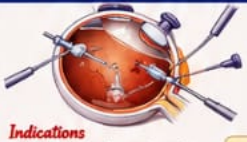
2. Intravitreal Anti-VEGF Injections

(Bevacizumab / Ranibizumab / Aflibercept)

- For active neovascularization
- For diabetic macular edema



C. PARS PLANA VITRECTOMY (PPV)



Indications

- Non-resolving vitreous hemorrhage
- Bilateral VH
- Tractional retinal detachment
- Recurrent VH
- Dense VH with severe vision loss

Advantages

- ✓ Clears blood
- ✓ Removes fibrovascular tissue
- ✓ Allows endolaser treatment
- ✓ Improves vision

COMPLICATIONS IF UNTREATED

Tractional Retinal Detachment

Neovascular Glaucoma

Recurrent Hemorrhage

Permanent Vision Loss

Dr Ashik Sureshkumar

VH

Q. 60 years old female patient with type II diabetic mellitus since 12 years, presents to OPD with complaining of sudden painless loss of vision in one eye.

Answer the following:

- What is your more probable diagnosis
- What are the differential diagnosis
- What are the clinical features of the condition
- How will you confirm the diagnosis
- How will you manage the case

1. MOST PROBABLE DIAGNOSIS

VITREOUS HEMORRHAGE
secondary to
PROLIFERATIVE DIABETIC RETINOPATHY (PDR)

WHY?

- Long-standing type 2 DM (12 years)
- Elderly patient (60 years)
- Sudden painless unilateral loss of vision
- Diabetic retinopathy → neovascularization → fragile vessels → vitreous bleed

2. DIFFERENTIAL DIAGNOSIS

A. RETINAL CAUSES

1. Central Retinal Artery Occlusion (CRAO)
2. Central Retinal Vein Occlusion (CRVO)
3. Retinal Detachment
4. Vitreous Hemorrhage (Most likely)
5. Macular Hemorrhage / Diabetic Macular Edema

B. OPTIC NERVE CAUSES

6. Anterior Ischemic Optic Neuropathy (AION) (Arteritic / Non-arteritic)

C. LENS / VITREOUS CAUSES

7. Posterior Vitreous Detachment (PVD)
8. Dense Cataract (usually gradual)

D. NEUROLOGICAL CAUSES

9. Occipital Stroke (rarely true monocular loss)

4. HOW WILL YOU CONFIRM THE DIAGNOSIS?

1. **Ophthalmoscopy / Fundus Examination**
   Primary diagnostic step.

2. **Slit Lamp Biomicroscopy**
   Shows blood cells in vitreous.

3. **B-Scan Ultrasonography**
   Very important if fundus not visible.
   Detects: Vitreous hemorrhage, retinal detachment, PVD, intraocular foreign body.

4. **Fundus Fluorescein Angiography (FFA)**
   Done after hemorrhage clears.
   Assesses: retinal ischemia, neovascularization.

5. **OCT (Optical Coherence Tomography)**
   To assess macular edema.

6. **Blood Investigations**
   Blood sugar, HbA1c, BP evaluation, renal function.

3. CLINICAL FEATURES OF VITREOUS HEMORRHAGE

A. SYMPTOMS

- Sudden painless diminution / loss of vision
- Floaters, cobwebs
- Black spots
- Red haze
- Visual field defects
- Flashes (if associated with retinal tear)

DEPENDING ON SEVERITY

- Mild VH
  - – Blurring of vision
  - – Floaters
- Severe VH
  - – Marked visual loss
  - – Only perception of light

B. SIGNS / EXAMINATION

- Visual acuity – reduced variably
- Pupils – usually normal; RAPD may occur
- Slit lamp – RBCs in vitreous
- Fundus examination –
  - – Mild VH: hazy view, scattered blood in vitreous
  - – Dense VH: fundus not visible, red reflex absent/reduced
- Associated diabetic retinopathy signs: NVE/NVD, microaneurysms, dot-blot hemorrhages, hard exudates, cotton wool spots

VITREOUS HEMORRHAGE

5. HOW WILL YOU MANAGE THE CASE?

A. INITIAL MANAGEMENT

- Bed rest with head elevation

- Avoid strenuous activity

- Control diabetes
- Control hypertension

- Stop anticoagulants if medically permissible

B. TREAT UNDERLYING CAUSE (PROLIFERATIVE DIABETIC RETINOPATHY)

1. PAN-RETINAL PHOTOCOAGULATION (PRP)

- Treatment of choice once retina visible.
- Purpose: regress neovascularization, prevent recurrent bleed.

2. INTRAVITREAL ANTI-VEGF INJECTIONS

- Drugs: Bevacizumab, Ranibizumab, Aflibercept
- Used for active neovascularization, diabetic macular edema.

C. PARS PLANA VITRECTOMY (PPV)

INDICATIONS

- Non-resolving vitreous hemorrhage
- Bilateral VH
- Tractional retinal detachment
- Recurrent VH
- Dense VH with vision loss

ADVANTAGES

- Clears blood
- Removes fibrovascular tissue
- Allows endolaser treatment

COMPLICATIONS IF UNTREATED

Tractional Retinal Detachment

Neovascular Glaucoma

Permanent Vision Loss

Recurrent Hemorrhage

6. ONE-LINE CLINICAL DIFFERENTIATION TABLE

CONDITION	KEY FEATURES	FUNDUS FINDINGS	RISK FACTORS	VISUAL ACUITY
Vitreous Hemorrhage (PDR)	Sudden painless loss, floaters, haze	Hazy view, blood in vitreous	Long-standing DM	Variable – from mild ↓ to PL
Central Retinal Artery Occlusion	Sudden profound painless loss	Pale retina with cherry-red spot	DM, HTN, Atherosclerosis	Counting fingers or worse
Central Retinal Vein Occlusion	Sudden painless blurring	"Blood and thunder" retina	DM, HTN, Glaucoma	Variable
Retinal Detachment	Flashes, floaters, curtain-like defect	Detached retina	Myopia, PVD, Trauma, PDR	Variable
Macular Edema / Hemorrhage	Central blurring, distortion	Macular edema / hemorrhage	DM	Mild to moderate ↓
Anterior Ischemic Optic Neuropathy	Sudden painless loss, altitudinal defect	Optic disc edema	DM, HTN, Age > 50	Variable
Posterior Vitreous Detachment	Floaters, flashes	Weiss ring may be seen	Age > 50, Myopia	Mild ↓
Dense Cataract	Gradual painless loss	Opaque lens	Age, DM	Gradual ↓
Occipital Stroke	Homonymous field defect	Normal eye exam	HTN, DM, Stroke risk factors	Variable

SHORT EXAM-ORIENTED CONCLUSION

A 60-year-old diabetic patient with sudden painless monocular vision loss most likely has **vitreous hemorrhage due to proliferative diabetic retinopathy.**
Diagnosis is confirmed by fundus examination and B-scan ultrasonography.
Management includes strict glycemic control, PRP laser photocoagulation, anti-VEGF therapy, and vitrectomy when indicated.

Dr Ashik Sureshkumar

Opthalmology one liners

Outer eye

Causative organism in angular conjunctivitis - Moraxella Axenfield

Reverse hypopyon seen in candida endophthalmitis and dt silicon oil following vitreoretinal surgery

Lens

Iris shadow present in which type of cataract

-Immature senile cataract

Glaucoma

Investigation to measure/visualise

a) angle of anterior chamber

-Gonioscope

b)intraocular pressure

-tonometer

Field defects like Bjerrum scotoma,seidel scotoma are seen in which type of glaucoma

-Primary open angle glaucoma

Mechanism of action of antiglaucoma drugs

Prostaglandin analogue- increase uveoscleral outflow

Beta blocler- reduce secretion

Adrenergic drug -increase outflow

(Brimonidine - increase uveoscleral outflow and decrease production)

CA i- decrease production

Rho kinase inhibitors -increase conventional outflow

Triad of congenital glaucoma

-lacrimation,photophobia,blepharospasm

Eclipse sign is seen in

-Shallow anterior chamber

Laser used in peripheral iridotomy

-Nd YAG,Argon

Inner eye

Uthoff symptoms seen in -

Optic neuritis

Tomato splash appearance - CRVO

Orbit and eye lid

Paralytic ectropion is due to paralysis of which nerve -7th CN

Most common cause of unilateral proptosis in children - orbital cellulitis

Bilateral proptosis in children- neuroblastoma, leukemia/chloroma

Most common cause of unilateral and bilateral proptosis in adults- Thyroid ophthalmopathy

Miscellaneous

Sympathetic ophthalmia is seen in which condition

-penetrating eye injury

Most common site of obstruction in congenital dacryocystitis

-NLD (Valve of Hasner)

Layers of tear film

(Outer to inner)- lipid ,aqueous,mucin layers

Congenital dacryocystitis primary treatment

- Crigler massage