🔥 UVEITIS

Definition:

Inflammation of the uveal tract (iris, ciliary body, choroid)

1️⃣ Classification of Uveitis

A. Anatomical Classification 🏷️

Depends on part of uvea involved:

Flowchart:

Anterior Uveitis → Iritis / Iridocyclitis / Anterior Cyclitis

Intermediate Uveitis → Pars Planitis / Posterior Cyclitis / Hyalitis

Posterior Uveitis → Choroiditis / Chorioretinitis / Retinochoroiditis

Pan Uveitis → Involvement of all uveal parts

B. Clinical Classification 🩺

Depends on onset, duration, and symptoms:

Type	Onset	Duration	Features
Acute uveitis	Sudden	<6 weeks	Pain, redness, photophobia, lacrimation
Subacute	Insidious	6 weeks–3 months	Mild symptoms
Chronic	Insidious	>3 months	Repeated episodes; less acute symptoms

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C. Histological Classification 🔬

- Non-granulomatous → lymphocytes, polymorphs, plasma cells

- Granulomatous → epithelioid cells, giant cells, mutton-fat KPs

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D. Etiology 🧬

1. Infective Uveitis

Source	Examples
Exogenous (entry via eye perforation)	Perforating injuries, corneal ulcers, post-surgery (cataract/glaucoma)
Endogenous (via bloodstream)	Bacteria: TB, Syphilis Virus: Herpes zoster, HIV Fungi: Candida, Histoplasma Protozoa: Toxoplasma Parasites: Toxocariasis

2. Non-Infective Uveitis

- Secondary: toxins from nearby ocular structures (e.g., corneal ulcer, scleritis, retinitis)

- Autoimmune:
  - Ocular: Phacoantigenic uveitis, Sympathetic ophthalmia
  - Systemic: Ankylosing spondylitis, Juvenile idiopathic arthritis, Behcet's disease, Sarcoidosis, IBD
- Idiopathic: ~50% of cases unknown

🌟 ACUTE IRIDOCYCLITIS (ACUTE ANTERIOR UVEITIS)

Definition:

Acute inflammation of the iris + ciliary body

Most common form of uveitis (>90% cases)

❑ Etiology

- Idiopathic → most common

- Other causes: HLA-B27 associated systemic diseases

2️⃣ Pathogenesis 🧩

- Acute inflammation → iris & ciliary body
- Spasm of ciliary muscle
- Vascular dilation + ↑ capillary permeability
- Chemotaxis → inflammatory cells into eye
- Can lead to secondary glaucoma

3️⃣ Clinical Features

Symptoms

- Sudden ocular pain (deep, worse at night) 😣
- Decreased vision 👁️
- Photophobia 🌞
- Lacrimation
- Redness of the eye 🔴

Signs

Structure	Findings
Visual acuity	Reduced
Limbus	Circumcorneal congestion
Cornea	Keratic precipitates (KPs) → pathognomonic
Anterior chamber	• Aqueous flare (protein leak) • Aqueous cells (inflammation) • Fibrinous exudates (HLAB27 cases) • Hypopyon (severe)
Iris	Muddy appearance (exudate)
Pupil	Miosis, sluggish reaction
Posterior synechiae	Adhesion pupil ↔ anterior lens capsule
Anterior vitreous	Inflammatory cells may be present

Media	Red reflex poor; fundus usually normal
Tenderness	Present on palpation
Intraocular pressure	May be low, normal, or high

Differential Diagnosis:

- Acute painful red eye → differentiate from acute glaucoma

4️⃣ Investigations 🧪

Investigation	Purpose
CBC, ESR, CRP	Rule out systemic inflammation
Blood sugar	Rule out diabetes-related uveitis
Serology	Syphilis, toxoplasmosis, histoplasmosis

ANA, RF, LE cells	Autoimmune screening
ANCA	Systemic vasculitis
Serum ACE	Granulomatous diseases (sarcoidosis, TB)
Skin tests	Tuberculin, Kveim, Histoplasmin
Radiology	X-ray sacroiliac joints, lumbar spine
HLA typing	HLA-B27 in acute anterior uveitis (~50%)
OCT	Macular edema
Vitreous biopsy	Refractory uveitis

S Treatment 💊

Goals:

1. Relieve symptoms 😌
2. Treat underlying cause
3. Prevent complications

A. Cycloplegics (relieve pain + prevent synechiae formation)

- Atropine 1% drops/ointment → 2–3×/day
- Homatropine 2% drops → 2–3×/day
- Cyclopentolate 1% drops → 3–4×/day

B. Corticosteroids (reduce inflammation)

- Topical drops: Prednisolone / Dexamethasone 4–6×/day
- Topical ointment: At bedtime
- Periocular injections: Severe cases
- Systemic steroids: Prednisolone 1–1.5 mg/kg/day

C. Immunosuppressives (for steroid-resistant cases)

- Azathioprine: 2 mg/kg/day
- Methotrexate: 10–15 mg/week

- Cyclosporin: 2 mg/kg/day → drug of choice in Behcet's disease

D. Antibiotics

- Used in infective uveitis → topical, periocular, systemic

E. Intravitreal steroids

- Triamcinolone acetonide 4 mg/0.1 ml → for cystoid macular edema unresponsive to other therapy

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📊 Flowchart – Acute Anterior Uveitis (Iridocyclitis)

Idiopathic / Infective / Autoimmune → Iris & ciliary body inflammation → Pain, photophobia, ↓ vision → KPs, miosis, aqueous flare → Posterior synechiae, secondary glaucoma → Cycloplegics + Steroids ± Immunosuppressives

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💡 Exam Tips:

- Keratic precipitates (KPs) → pathognomonic for anterior uveitis
- Posterior synechiae → check pupil shape
- HLA-B27 association → ankylosing spondylitis, reactive arthritis, psoriasis

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🌟 CHRONIC IRIDOCYCLITIS (CHRONIC ANTERIOR UVEITIS)

Definition:
Chronic inflammation of the iris and ciliary body with persistent inflammation and relapses <3 months

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❑ Clinical Features

- Less common than acute type
- Often asymptomatic unless complications develop

Symptoms

- Usually none until complications arise

Signs

Feature	Description
Eye appearance	White, quiet eye
Anterior chamber	Flare + cells present
Keratic precipitates (KPs)	Greasy, mutton-fat → granulomatous uveitis
Posterior synechiae	Occlusion pupillae or ring synechiae
Iris nodules	• Koeppe nodules: pupillary margin • Busacca nodules: iris surface

Exam Tip: Koeppe & Busacca nodules are hallmarks of chronic granulomatous iridocyclitis 🔬

2️⃣ Investigations

- Similar to acute iridocyclitis
- Targeted based on suspected underlying cause

3️⃣ Treatment 💊

Goals:

- Control inflammation
- Prevent complications
- Treat underlying cause

Management:

- Non-specific therapy: Symptom control (cycloplegics, topical steroids)
- Specific therapy: Treat underlying systemic or ocular disease

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4️⃣ Complications ⚠️

Complication	Mechanism/Notes
Complicated cataract	Most common; due to chronic inflammation
Secondary glaucoma	Trabecular meshwork obstruction, acute trabeculitis, posterior synechiae → iris bombe
Cyclitic membrane	Organization of exudate behind lens
Choroiditis	May develop due to continuity with posterior segment
Retinal complications	• Cystoid macular edema (CME) → main cause of visual loss • Exudative retinal detachment
Optic disc edema	Severe chronic inflammation
Band keratopathy	Long-standing uveitis

Vitreous opacities / vitritis	Chronic inflammation of vitreous
Hypotony	Acute → reversible low IOP; chronic → ciliary atrophy, permanent hypotony
Phthisis bulbi	End-stage eye due to prolonged hypotony

🌈 INTERMEDIATE UVEITIS

Definition:

Inflammation of the pars plana, peripheral retina, ciliary body, and vitreous base

- Accounts for 10% of all uveitis cases, 20% pediatric
- Often bilateral (80%) but asymmetrical

1️⃣ Pars Planitis (Most common intermediate uveitis)

Etiology

- Idiopathic: 50%
- Associated systemic diseases: Sarcoidosis, Syphilis, TB

Clinical Features

Symptoms:

- Insidious onset
- Floaters: main complaint
- Blurred vision occurs late
- No pain, photophobia, or redness

Signs:

Feature	Description
Anterior chamber	Few cells, scattered KPs

Vitreous	Vitritis; inflammatory cells in anterior vitreous
Snowballs	Aggregates of inflammatory cells in peripheral vitreous
Snowbanking	Gray-white exudates over inferior pars plana; correlates with severity
Retina	Peripheral periphlebitis, venous sheathing, optic disc swelling in young patients

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2 Complications

Complication	Notes
Cystoid macular edema (CME)	Main cause of visual loss
Retinal neovascularization	May lead to vitreous hemorrhage
Tractional retinal detachment	Due to vitreoretinal fibrosis

Complicated cataract	From chronic inflammation
Secondary glaucoma	From chronic inflammation & synechiae
Band keratopathy	Chronic uveitis
Vitreous opacities	May persist for years

3️⃣ Diagnosis 🧪

- Primarily clinical
- Laboratory tests to exclude systemic causes (sarcoidosis, syphilis, TB)

4️⃣ Prognosis

- ~10% → self-limiting, benign course
- ~30% → smoldering course, remission & exacerbation

- ~60% → prolonged course with flares
- Pars planitis may remain active for >30 years

🇸 Treatment 💊

A. Medical Therapy

Therapy	Notes
Topical steroids	Limited role (poor posterior segment penetration)
Periocular steroid injection	Posterior sub-tenon → effective 2–4 weeks
Intravitreal triamcinolone	Severe, refractory cases
Systemic steroids	When local therapy ineffective
Immunosuppressives	Azathioprine → bilateral cases

Anti-VEGF	Intravitreal → retinal neovascularization

B. Surgical Therapy

Procedure	Indication
Laser photocoagulation	Peripheral retinal neovascularization → prevent vitreous hemorrhage
Pars plana vitrectomy	Severe visual loss due to vitreous opacities, hemorrhage, or CME
Cryotherapy	Rarely used; peripheral retina ablation in refractory cases

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📊 Flowchart – Intermediate Uveitis (Pars Planitis)

Idiopathic / Systemic Disease → Inflammation (pars plana, vitreous) → Floaters, blurred vision → Vitritis, snowballs, snowbanking, periphlebitis → CME, retinal neovascularization, cataract, glaucoma → Steroids

(local/systemic), Immunosuppressives, Anti-VEGF, Laser / Vitrectomy

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💡 Exam Tips:

- Floaters → hallmark symptom
- Snowballs & snowbanking → hallmark signs
- CME → main cause of vision loss
- Pars planitis can persist decades → chronic management important

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🌟 POSTERIOR UVEITIS (CHOROIDITIS)

Definition

Posterior uveitis refers to inflammation of the choroid.

Because the retina lies directly adjacent to the choroid, the inflammatory process almost always spreads to the retina.

➡ Therefore the lesion is usually called chorioretinitis.

Key Concept for Exams 🧠

Choroiditis rarely occurs alone.

Posterior uveitis → Choroid + Retina involvement = Chorioretinitis

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Aetiology

1️⃣ Infective Causes

- Bacterial infections
- Viral infections
- Fungal infections
- Parasitic infections (most important: toxoplasmosis)

2️⃣ Non-Infective Causes

- Sarcoidosis
- Collagen vascular diseases
- Idiopathic causes (unknown)

Clinical Features

Symptoms

Symptom	Explanation
Gradual painless visual loss	Due to retinal involvement and vitreous haze
Floaters	Dark spots moving in front of eye caused by inflammatory debris in vitreous
Metamorphopsia	Distortion of objects due to irregular retinal surface
Micropsia	Objects appear smaller due to separation of photoreceptors
Macropsia	Objects appear larger due to crowding of rods and cones
Photopsia	Flashes of light due to irritation of photoreceptors

Positive Scotoma	Fixed dark spot in visual field corresponding to lesion

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Signs

Clinical signs vary depending on the causative organism.

Typical findings include:

- Vitreous inflammation (vitritis)
- Chorioretinal inflammatory lesions
- Retinal edema
- Retinal scarring in healed lesions

Treatment

Non-Specific Treatment

- Corticosteroids
- Immunosuppressive drugs

These help control inflammation.

Specific Treatment

Depends on underlying cause, for example:

- Antimicrobials for infections
- Immunotherapy for autoimmune disease

🦠 TOXOPLASMOSIS

Definition

Toxoplasmosis is an infectious disease caused by Toxoplasma gondii, a single-celled obligate intracellular protozoan parasite.

📌 Most common cause of infectious retinochoroiditis worldwide.

Forms of the Parasite

Form	Description
Oocyst (sporocyst)	Contains sporozoites; present in cat intestine
Tissue cyst	Contains bradyzoites; found in brain, eye, heart, skeletal muscle, lymph nodes
Tachyzoites (trophozoites)	Active proliferating form causing tissue destruction

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Hosts of the Parasite

Host Type	Role
Definitive host	Cat (sexual reproduction occurs)
Intermediate host	Humans and livestock

Cats shed oocysts in feces, contaminating food, soil, and water.

Mode of Transmission

Infection occurs through:

- Eating contaminated fruits or vegetables
- Drinking contaminated water
- Eating undercooked infected meat containing cysts
- Pica in children (eating contaminated soil)
- Transplacental transmission from infected mother to fetus

Organs Commonly Affected

Toxoplasmosis mainly affects:

- Brain
- Retina
- Heart muscle
- Lymph nodes

🌟 Clinical Forms of Ocular Toxoplasmosis

Three clinical forms exist:

1️⃣ Congenital toxoplasmosis
2️⃣ Acquired toxoplasmosis
3️⃣ Recurrent toxoplasma retinochoroiditis

👶 Congenital Toxoplasmosis

Definition

Occurs when tachyzoites cross the placenta and infect the fetus during pregnancy.

Classic Systemic Features

The classical triad includes:

Feature	Explanation
Hydrocephalus	Enlargement of ventricles
Intracranial calcification	Brain calcifications seen on imaging
Convulsions	Due to CNS involvement

Ocular Features

Main manifestation: Retinochoroiditis

This occurs in two stages.

1 Active Stage

Characteristics:

- Necrotizing granulomatous retinochoroiditis
- Thick cream-colored retinal lesion
- Marked vitritis

- Often occurs near old scar

➡ This adjacent lesion is called a satellite lesion

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2 Inactive Stage

Many infants are born with healed lesions.

Typical appearance:

- Bilateral chorioretinal scars
- Punched-out lesions
- Heavy pigmentation
- Often located in the macular region

Flowchart – Congenital Toxoplasmosis

Maternal infection during pregnancy → Parasite crosses placenta → Fetal infection → CNS involvement + retinal infection → Retinochoroiditis → Healed pigmented chorioretinal scars

🧑 Acquired Toxoplasmosis

Previously it was thought that all ocular toxoplasmosis was due to reactivation of congenital infection.

However, it is now known that primary acquired infection can also cause ocular disease in children and adults.

Symptoms

Depend on location of retinal lesion.

Common symptoms include:

- Floaters
- Blurred vision
- Unilateral hazy vision

Ocular Examination Findings

Structure	Findings
Conjunctiva	Eye usually white
Anterior chamber	Mild–moderate granulomatous anterior uveitis
Keratic precipitates	May be present
Posterior synechiae	May occur
Vitreous	Vitritis
Retina	Focal white retinitis lesion

Flowchart – Acquired Ocular Toxoplasmosis

Ingestion of parasite cysts → Systemic infection → Parasite reaches retina → Retinal necrosis + inflammation → Vitritis + focal retinitis → Chorioretinal scar after healing

🧠 Exam Pearls

⭐ Most common cause of infectious posterior uveitis:
→ Toxoplasmosis

⭐ Classic lesion:
White retinal lesion + overlying vitritis ("headlight in fog" appearance)

⭐ Congenital triad:
Hydrocephalus
Intracranial calcification
Retinochoroiditis

⭐ Satellite lesion:
New active lesion adjacent to old scar

🔁 Recurrent Toxoplasmosis (Recurrent Toxoplasmic Retinochoroiditis)

Definition

Recurrent toxoplasmosis is reactivation of latent Toxoplasma gondii infection in the retina, leading to recurrent episodes of necrotizing retinochoroiditis.

📌 It is one of the most common causes of posterior uveitis worldwide.

🧬 Pathogenesis

After the initial infection (often congenital), the parasites become encysted in retinal tissue in an inactive form.

These cysts may remain dormant for many years.

When cysts rupture, parasites are released and cause local retinal infection and inflammation, leading to recurrent lesions near old scars.

Flowchart - Pathogenesis

Congenital or acquired infection → Parasite reaches retina → Formation of retinal cysts (latent stage) → Parasites remain dormant for years → Cyst rupture → Release of tachyzoites → Local retinal invasion → Necrotizing retinochoroiditis → New lesion forms adjacent to old pigmented scar

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Mechanism of Tissue Damage

Two main mechanisms contribute:

1. Direct retinal invasion by parasites
2. Immune reaction (antigen–antibody response) in retina, iris, and choroid

This leads to:

- Inflammation
- Retinal necrosis
- Vitritis

👁️ Clinical Features

Recurrent toxoplasmic retinochoroiditis is very common clinically.

Symptoms

Symptom	Explanation
Floaters	Due to inflammatory cells in vitreous
Unilateral blurred vision	Caused by retinal inflammation
Hazy vision	Due to vitritis and vitreous haze

Signs

General Appearance

- Eye usually white (no severe redness)

Retinal Lesion

- Whitish-yellow raised retinal lesion
- Located adjacent to old pigmented chorioretinal scar
- Represents active retinitis

Vitreous Findings

Severe vitritis may obscure the retina.

📌 Classic description:

"Headlight in the fog appearance"

Meaning:

White retinal lesion → Seen through dense vitreous inflammation

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Other Fundus Findings

Finding	Explanation

Posterior pole lesions	Most commonly affected
Retinal perivasculitis	Inflammation around retinal vessels
Vitritis	Universal finding
Macular involvement	Causes severe visual loss

Anterior Segment Findings

Mild nongranulomatous anterior uveitis may occur.

Findings include:

- Keratic precipitates (KPs)
- Cells in anterior chamber
- Flare

Causes of Visual Loss

Visual loss occurs due to:

- Macular involvement
- Optic nerve head involvement
- Severe vitritis

🔬 Diagnosis

Diagnosis is primarily clinical, supported by laboratory tests.

🧫 Serological Tests

Used to detect anti-toxoplasma antibodies.

Antibody	Significance
IgG	Appears 1-2 weeks after infection, peaks in 1-2 months, persists for life

IgM	Appears earlier but declines faster; indicates recent infection

📌 IgM positivity → recent infection

2️⃣ PCR (Polymerase Chain Reaction)

Highly sensitive test.

Detects Toxoplasma DNA in:

- Aqueous humour
- Vitreous fluid

💊 Treatment

Although the disease can be self-limiting, treatment aims to:

- Reduce severity of inflammation
- Reduce risk of permanent visual loss
- Reduce risk of recurrence

Medical Treatment

1️⃣ Corticosteroids

Used to control inflammation.

- Topical steroids
- Systemic steroids (Prednisolone)

Typical dosage:
Prednisolone 1 mg/kg, then gradually tapered.

⚠️ Always given with anti-toxoplasma drugs.

2️⃣ Anti-Toxoplasma Drugs

Drug	Dose
Pyrimethamine	Loading dose 75–100 mg for 1–2 days, then 25–50 mg daily
Sulfadiazine	1 g four times daily for 3–4 weeks

Folinic acid	Prevents bone marrow toxicity of pyrimethamine

Classic Therapy (Triple Therapy)

- Drug Combination -
Prednisolone
Pyrimethamine
Sulfadiazine

📌 Standard treatment with good clinical response.

Alternative Treatments

Drug	Use

Co-trimoxazole	Better tolerated but slightly less effective
Clindamycin	Can replace pyrimethamine or be added to therapy

Flowchart – Treatment Strategy

Active toxoplasmic lesion → Control inflammation → Steroids + Anti-toxoplasma drugs → Reduce retinal necrosis → Prevent macular damage → Reduce recurrence

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🦠 Tuberculous Uveitis

Definition

Tuberculosis is a chronic granulomatous infection caused by Mycobacterium tuberculosis.

Characteristics:

- Acid-fast bacillus
- Obligate aerobe
- Primarily affects lungs

However, it may spread hematogenously to other organs, including the eye.

Ocular Tuberculosis

Ocular manifestations occur due to:

1. Direct infection by Mycobacterium tuberculosis
2. Hypersensitivity immune reaction to the organism

Nature of Disease

Tuberculous uveitis is typically:

- Chronic
- Granulomatous
- May affect anterior or posterior segments

👁️ Intraocular Manifestations

1️⃣ Anterior Uveitis

Common findings include:

- Granulomatous uveitis
- Mutton-fat keratic precipitates
- Fibrinous exudate
- Iris nodules

2️⃣ Intermediate Uveitis

Inflammation involves:

- Pars plana
- Vitreous

3️⃣ Vitritis

Vitreous inflammation is a very common feature.

4️⃣ Disseminated Choroiditis

Most common ocular manifestation of TB.

Characteristics

- Multiple yellowish deep choroidal lesions
- Size: 0.5 – 3 mm
- Number: 5 to several hundred

These lesions are called:

⭐ Choroidal tubercles

Associated Findings

- Disc edema
- Retinal hemorrhages
- Vitritis

Most commonly located in the posterior pole.

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5 Choroidal Tuberculoma

Second most common manifestation.

Features

- Single large lesion
- Dome-shaped
- Whitish elevation

Size:

4 – 14 mm

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6 Secondary Retinal Changes

May cause:

- Retinal detachment
- Macular star

Due to spread of choroidal inflammation.

7️⃣ Other Ocular Manifestations

Tuberculosis may also cause:

- Eyelid nodules
- Conjunctivitis
- Phlyctenulosis
- Scleritis
- Interstitial keratitis

🔬 Diagnosis of Tuberculous Uveitis

1️⃣ Tuberculin Skin Test (Mantoux Test)

Indicates exposure to tuberculosis.

2️⃣ Interferon-Gamma Release Assay (IGRA)

Blood test detecting immune response to TB antigens.

Sensitivity ≈ 80%

3️⃣ Isolation of Mycobacterium Tuberculosis

From ocular fluids.

4️⃣ PCR Testing

Detection of mycobacterial DNA.

5️⃣ Fundus Fluorescein Angiography (FFA)

Findings:

Early → Hyperfluorescence

Late → Leakage

Indicates active choroidal inflammation.

6️⃣ Indocyanine Green Angiography (ICG)

Shows:

- Early hyperfluorescence
- Late staining of choroidal lesions

💊 Treatment of Tuberculous Uveitis

1️⃣ Anti-Tubercular Therapy (ATT)

Long-term multi-drug therapy is required.

Typical drugs include:

- Isoniazid
- Rifampicin
- Pyrazinamide
- Ethambutol

2️⃣ Corticosteroids

Used to reduce:

- Tissue damage
- Inflammatory response

Can be given:

- Topically
- Systemically

Flowchart – Pathogenesis of Ocular TB

Pulmonary tuberculosis → Hematogenous spread → Mycobacterium reaches choroid → Granulomatous inflammation → Choroidal tubercles / tuberculoma → Vitritis + retinal complications

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-> The End <-