Uveitis Glaucoma

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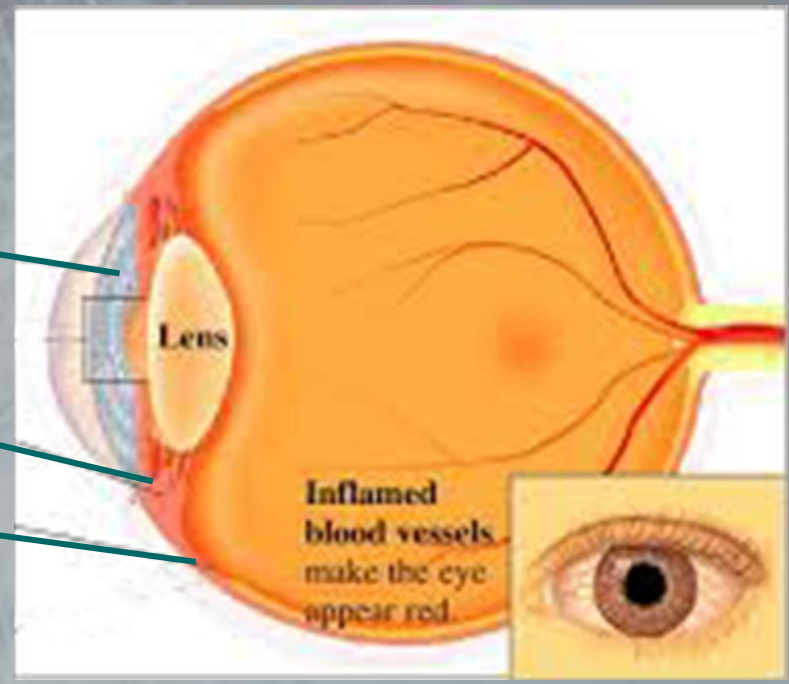
AGENDA

- What is uvea?
- Classification of uveitis
- Evaluation
- Uveitis Glaucoma

  ((Pathology , Diagnosis , Management))
- Uveitis Syndromes
- Key Points

THE UVEA IS THE VASCULAR LAYER OF THE EYE.

- Iris
- Ciliary Body
- Choroide



CLASSIFICATION OF UVEITIS

- Anatomy

  Anterior, Intermediate, Posterior

- Clinical course

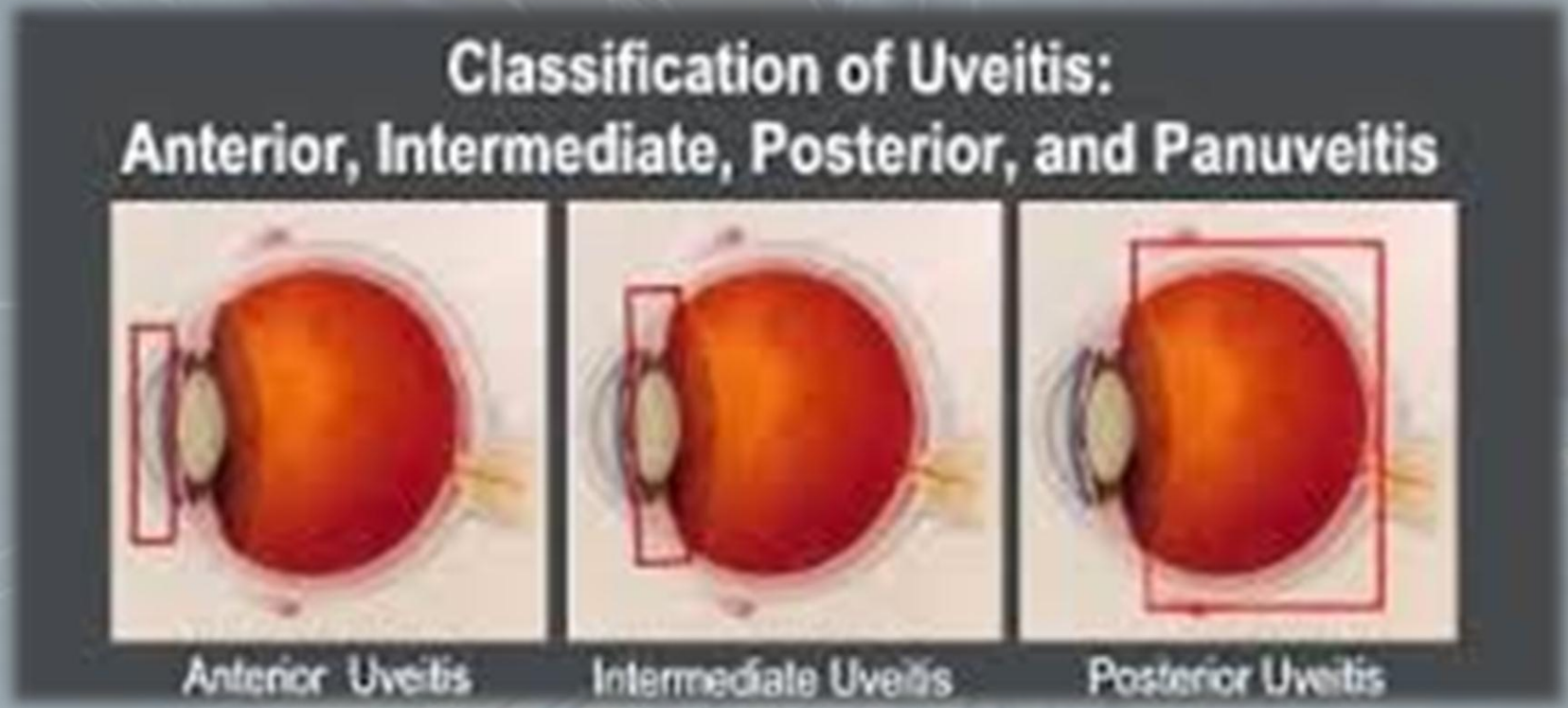
  Acute, Chronic, Recurrent

- Etiology

  Infectious, Non Infectious

- Histology

  Granulomatous , Non Granulomatous


Classification of Uveitis:
Anterior, Intermediate, Posterior, and Panuveitis
Anterior Uveitis
Intermediate Uveitis
Posterior Uveitis

ANTERIOR UVEITIS

- Iritis

Inflammation confined to anterior chamber.

- Iridocyclitis

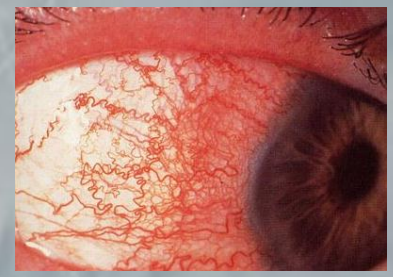
Spills over into the retrolental space

- Keratouveitis

It involved cornea

- Sclerauveitis

It involved sclera and uvula tract



ANTERIOR UVEITIS CONT.

- Acute

  Pain, Photophobia, Redness, blurred vision

- Chronic

  No symptoms, blurred vision may be as a result calcific band keratopathy, cataract , cystoid macular edema CME

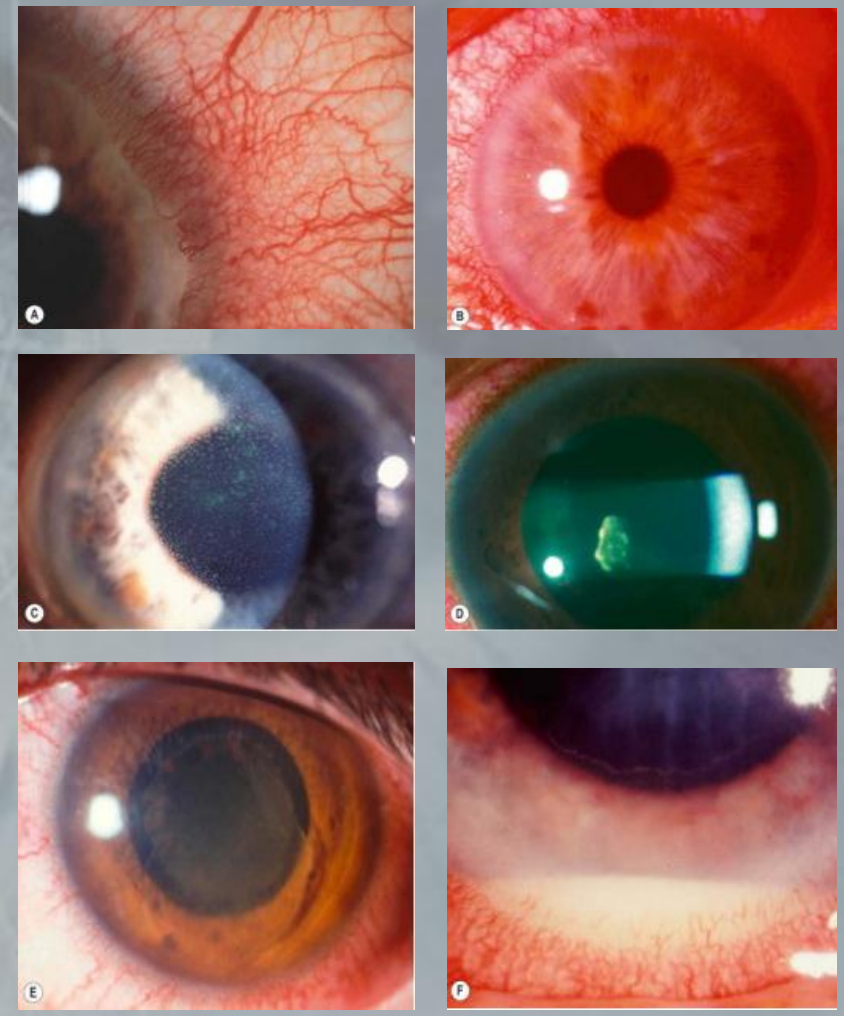
- Recurrent

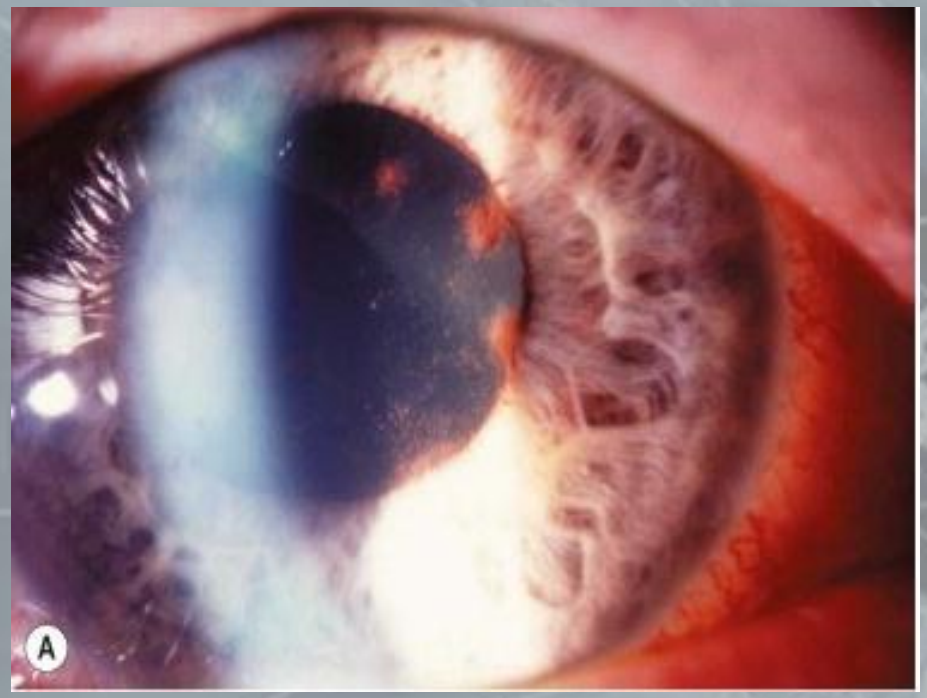
  Repeated episodes separated by periods of inactivity without treatment more than 3 months

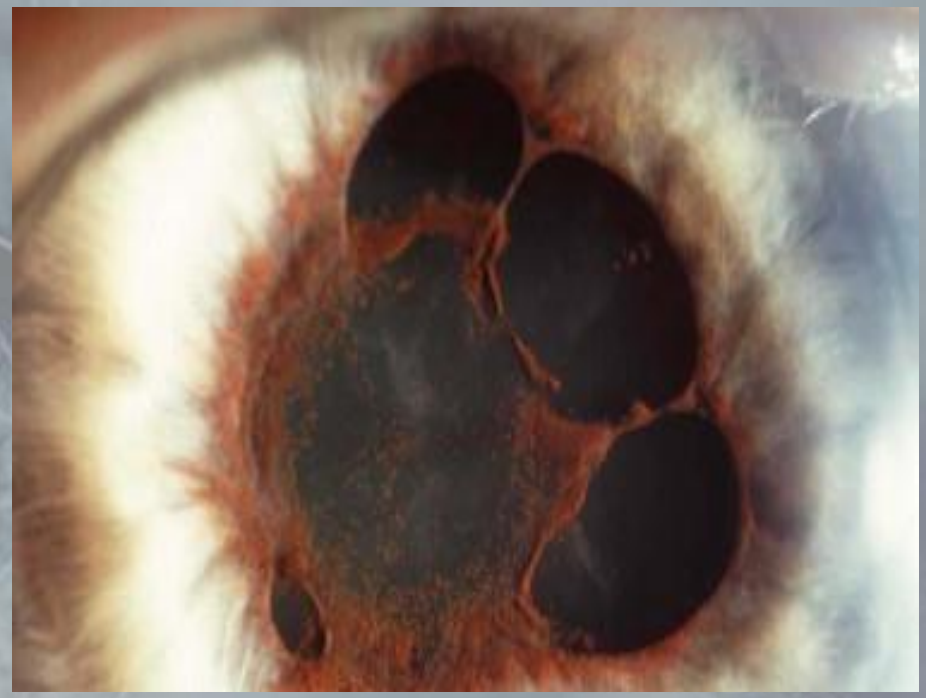
ANTERIOR UVEITIS CONT.

Signs:

- Keratic precipitates.
- Cells.
- Flare
- Hypopyon
- Pupillary Miosis.
- Iris nodules.
- Fibrin.
- Pigment dispersion.







- Posterior synechiae. (A) Early synechiae formation in active acute anterior uveitis; (B) extensive synechiae and pigment on the lens following a severe attack of acute anterior uveitis

INTERMEDIATE UVEITIS

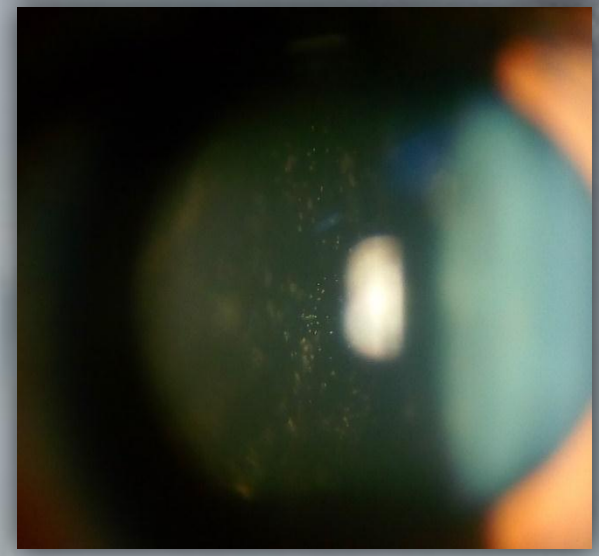
The major site of inflammation is the vitreous.

Symptoms:

Floaters, Blurred Vision

Signs:

- Snowball Opacities.
- Exudates over pars plana.
- Vitreal strands

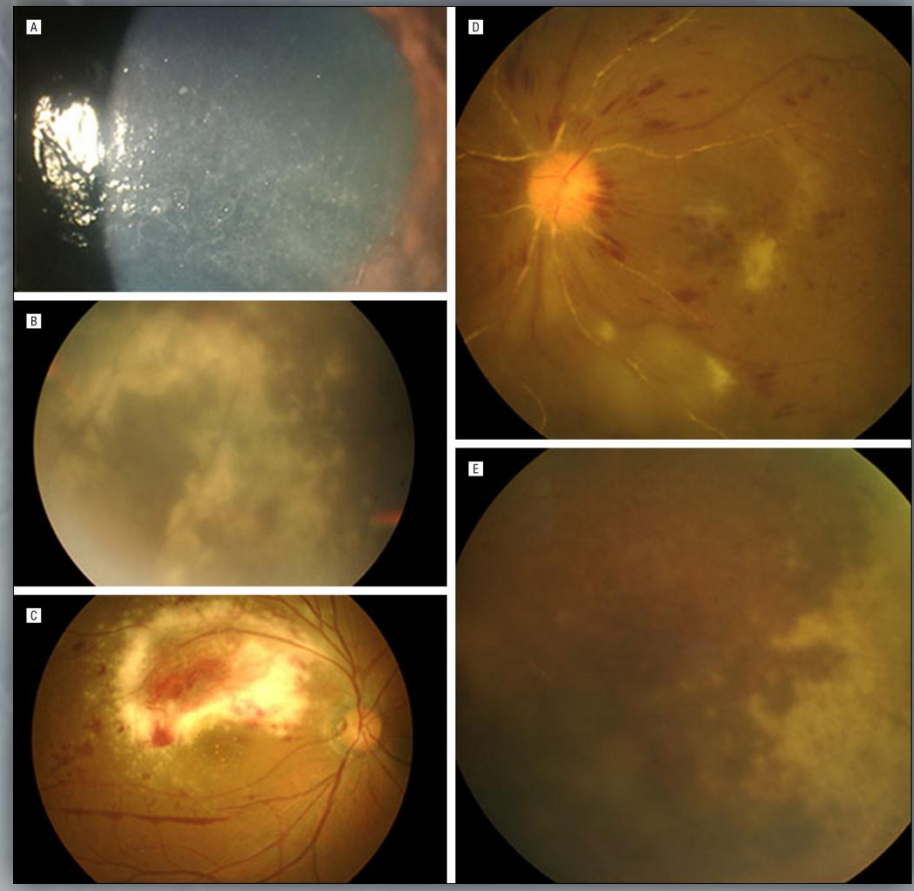


POSTERIOR UVEITIS

The site of inflammation is retina and/ or choroid.

Symptoms:

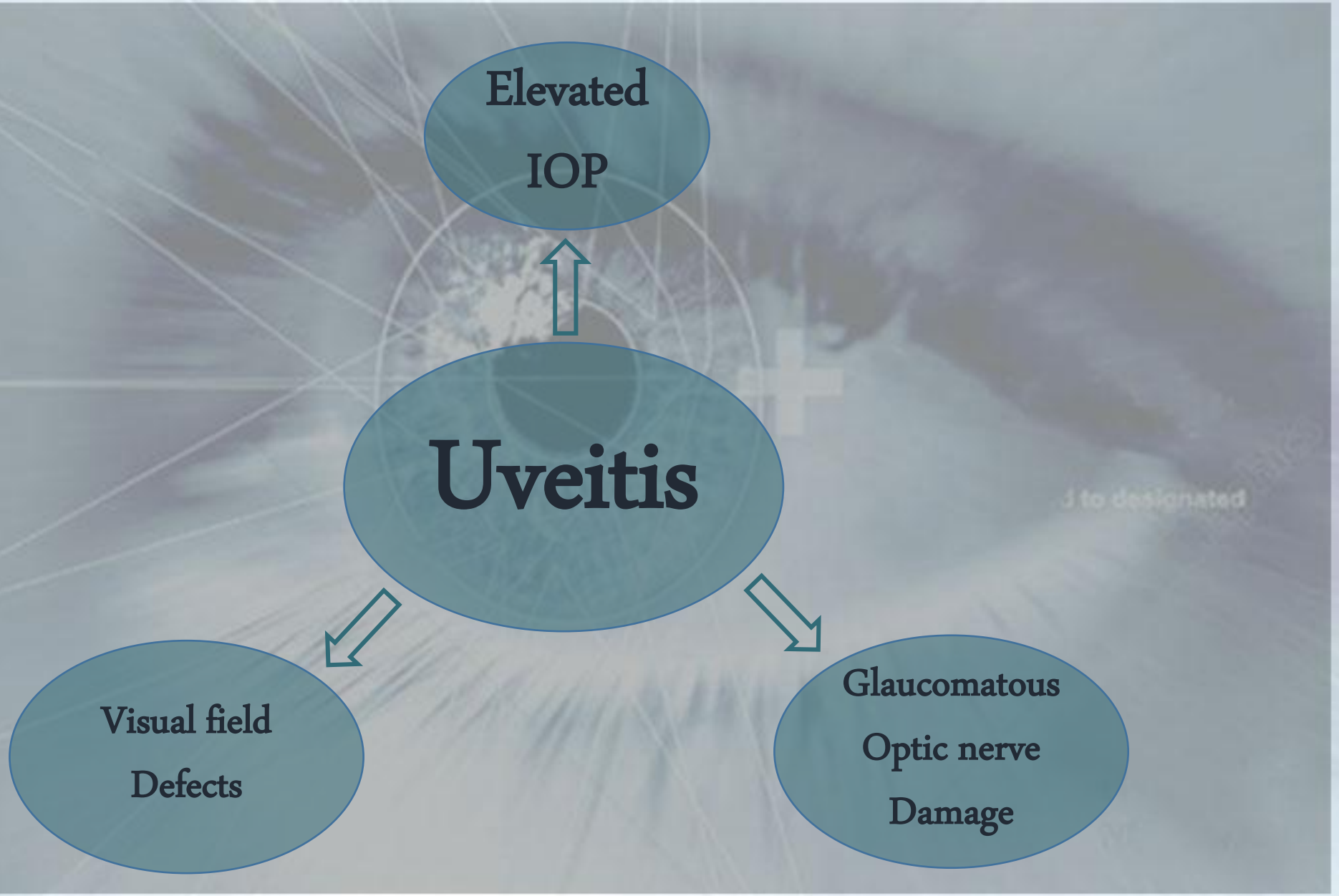
- VA
- Painless
- Floats.
- Photopsiasis.
- Metamorphopsia
- Scotomata
- Blurred Vision



EVALUATION

- Fluorescein Angiography FA
- Ultasonography.
- Optical Coherence Tomography OCT.
- Anterior Chamber Paracentesis.
- Vitreous Biopsy.
- Choroiretinal Biopsy.

Uveitis Glaucoma


Elevated
IOP
Uveitis
Visual field
Defects
Glaucomatous
Optic nerve
Damage

- Uveitis glaucoma is more common in cases of granulomatous the non-granulomatous.
- The prevalence of glaucoma secondary to uveitis

  Adults : 5.2%-19%

  Children : 5%-13%
- Visual prognosis for children with uveitis glaucoma is worse.
- Uveitis is found in patients of all ages esp. 25-44 years.

- In most inflamed eyes the intraocular pressure decreases ,due to a breakdown of the blood-aqueous barrier.
- But in some eyes the outflow is compromised more than production.
- Even if the pressure is normal early on ,it can go up later as aqueous production returns towards normal and the corticosteroids used to treat the inflammatory can also elevated the IOP

Uveitis induced ocular hypertension (ocular hypertension secondary uveitis) refer to:

Uveitis with increased IOP only without visual field defects or optic nerve damage

- posterior Uveitis characterized usually by choroiretinal and optic nerve lesion and can produce visual field defects.

- True Glaucomatous visual field defects are **irreversible** while it may be improved in active inflammatory diseases with appropriate therapy.

Common causes of visual loss in patients with uveitis include:

- Secondary glaucoma
- Cystoid macular edema
- Cataract
- Hypotomy
- Retinal detachment
- Subretinal neovasculazation
- Optic nerve atrophy.

PATHOLOGY OF UVEITIS GLAUCOMA

OPEN ANGLE MECHANISMS:

- Abnormal Aqueous Secretion.
- Aqueous Humor Proteins.
- Inflammatory cell.
- Prostaglandins.
- Trabeculitis.
- Steroids induced ocular hypertension.

ABNORMAL AQUEOUS SECRETION

- Inflammation of ciliary body decrease aqueous production with normal out flow and decrease IOP (like acute Uveitis).

- Decreased aqueous secretion with low outflow

IOP normal or increased.

AQUEOUS HUMOR PROTEINS.

In Uveitis: blood- aqueous barrier is disrupted which lead to increased aqueous proteins in anterior chamber

↓

Obstructing the trabecular mesh work

↓

Increase IOP

+

Posterior or peripheral anterior synechiae

INFLAMMATORY CELLS

- **Inflammatory cells secrete inflammatory mediators (Prostaglandins,Cytokines,..)in AC.**
- **Inflammatory cells can increase IOP by infiltrating the trabecular meshwork and Schlemm's canal → mechanical obstruction to aqueous outflow.**
- **Chronic and sever Uveitis can cause:**
  - ✓ Permanent damage to the trabecular meshwork from injury to trabecular endothelial cell.
  - ✓ Scarring in the trabecular meshwork and Schlemm's canal or from formation of hyaline membrane overlying the trabecular.

TRABECULITIS.

- **Trabeculitis** is diagnosed when the intraocular inflammatory response is localized to the trabecular meshwork with absence of other signs of active intraocular inflammatory.

- Increased IOP with trabeculitis caused by Decrease aqueous outflow due to:
  - ✓ Accumulation of inflammatory cell.
  - ✓ Decrease phagocytosis of the trabecular endothelial cells.

STEROIDS INDUCED OCULAR HYPERTENSION

- Corticosteroids are first line therapy for Uveitis.

- IOP may happen:

✓ Induce physical & mechanical changes in trabecular meshwork microstructure.

✓ Increase the deposition of substances in the trabecular meshwork.

✓ Decrease the breakdown of substances in trabecular meshwork.

✓ Inhibition of prostaglandin synthesis .

STEROIDS INDUCED OCULAR HYPERTENSION

- Steroid responder refer to patients who develop elevated IOP related to corticosteroid therapy.

- 4-6 weeks of topical steroids treatment:

  35% increased IOP at least 5 mmHg

  5 % > 16 mmHg.

RISK OF STEROIDS RESPONSE

- Patients with Glaucoma.
- Glaucoma suspect.
- First degree relatives of Glaucoma patients.
- Elderly.
- Patients with connective tissue disease .
- Diabetes Type I.
- High Myopia.
- Children <10 yr.

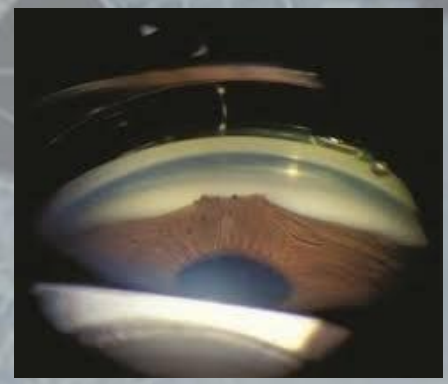
CLOSED ANGLE MECHANISM

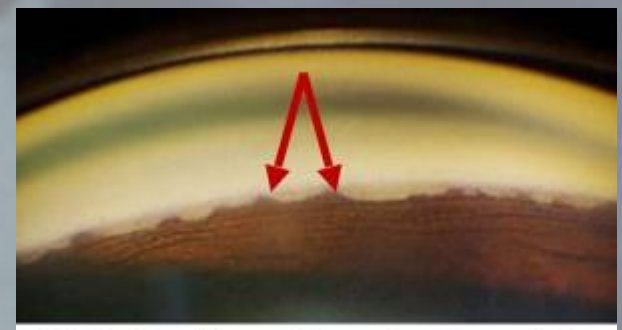
- Peripheral Anterior Synechiae (PAS).
- Posterior Synechiae.
- Forward rotation of ciliary body.

PERIPHERAL ANTERIOR SYNECHIAE

- PAS is adhesions between Iris and the trabecular meshwork or Cornea that can completely block or impair access of the aqueous to the trabecular meshwork.

- Best detection by **Gonioscopy**.





PAS - Peripheral Anterior Synechiae

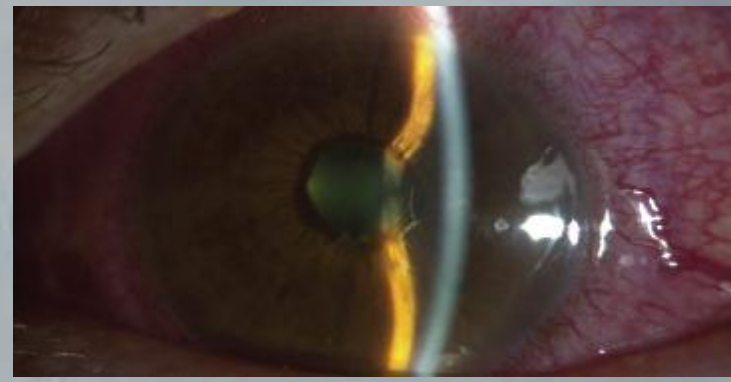
- PAS result from organization of inflammatory material that pulls the Iris surface into the angle affecting only small portions of the trabecular meshwork or cornea.

PERIPHERAL ANTERIOR SYNECHIAE

- Recurrent or chronic Uveitis continued PAS formation can result in complete angle closure.
- Neovascularization of Iris and the angle should be sought in all case of Uveitis presenting with angle closure or extensive PAS.
- Neovascularization Glaucoma secondary to Uveitis is typically resistant to medical and surgical therapy with poor diagnosis.

POSTORIOR SYNECHIAE

- Inflammatory cells , proteins and fibrin in aqueous humor can stimulate posterior synechiae.
- Posterior synechiae are adhesions between posterior iris surface & anterior lens capsule , the vitreous face in aphakic patients or intraocular lens in pseudo phakic individuals.
- Posterior Synechiae that extend for 360 d (pupillary block) cause to Iris bomb and ↑ IOP.



FORWARD ROTATION OF CILIARY BODY

- Acute intraocular inflammation can cause ciliary body swelling and supraciliary or suprachoroidal effusions that may result in the forward rotation of the ciliary body , cause angle closure not associated with pupillary block.

- This type of angle closure occurs on patients with iridocyclitis , annular choroidal detachment and posterior scleritis and can be seen in acute stage of Vogt-Koyanagi-Harada Syndrome

FOR WHICH WOULD ONE DO AN PI?

- Peripheral anterior synechiae (PAS)
- Central posterior synechiae
- Forward rotation of ciliary body
- neovascularization

DIAGNOSIS

The diagnosis of active inflammation is based on:

- Detection of inflammation cells or/ and vitreous
- Flare in AC
- Gonioscopoy

DIFFERENTIAL DIAGNOSIS

I. Adenoma of non pigmented epithelium of ciliary body.

II. Uveitis glaucoma hyphema syndrome.

III. Lens-induced glaucoma.

IV. Non penetrating trauma.

V. Grantos syndrome

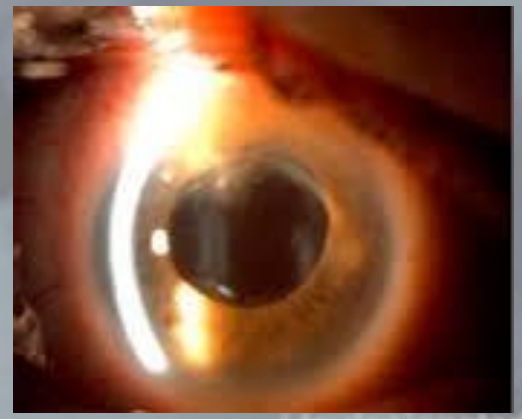
(↑ IOP + inflammatory precipitates on trabecular meshwork + quiet eye)

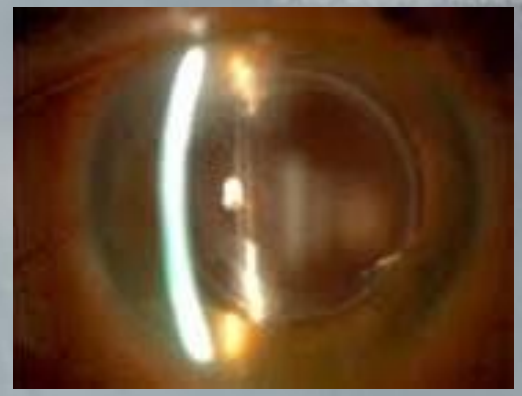
Uveitis-Glaucoma-Hyphema Syndrome

It is almost a complication of intraocular chafing from intraocular lens (IOL) implants leading to:

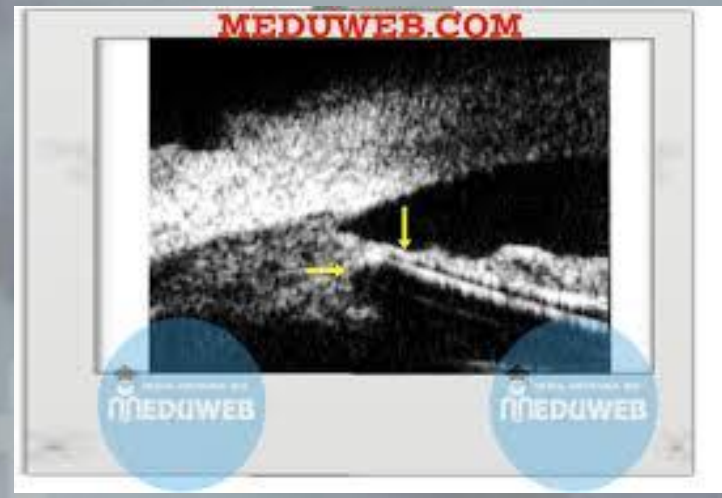
- Iris transillumination defects
- Pigmentary dispersion
- Microhyphemas and hyphema
- Elevated intraocular pressure(IOP)

- **Most commomly caused by chafing from anterior chamber intraocular lenses ,but can occur from any type of pseudophakic lens (even in bag IOL)**
- **usually complicated with:**
  - Chronic inflammation(iridocilitis)
  - Cystoid macularedema
  - Secondary iris neovasculazation
  - Recurrent hyphemas
  - Glaucomatous optic
  - Eventually loss of vision





- This syndroma results of mechanical irritation of anterior segmeny structures from an intraocular lens or even cosmetic iris implants.
- Most commonly in elderly adults ,but reported in the pediatric age group.
- Within 6 months of IOL implant is consistently higher in anterior chamber lenses than in iris plane lenses than in posterior IOL.



MECHANISM OF UGH

Hyphema can be due to:

- Peripupillary contact of iris with lens optic and haptic
- Erosion of uveal structures including the iridocorneal angle,iris,and cillary body.
- Breakdown of the blood-aqueous barrier and subsquent release of pigment,red blood cells,protein,white blood cells into the anterior chamber.

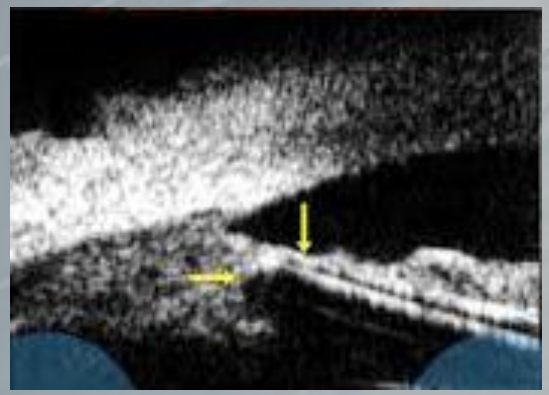
MECHANISM OF UGH

High IOP:

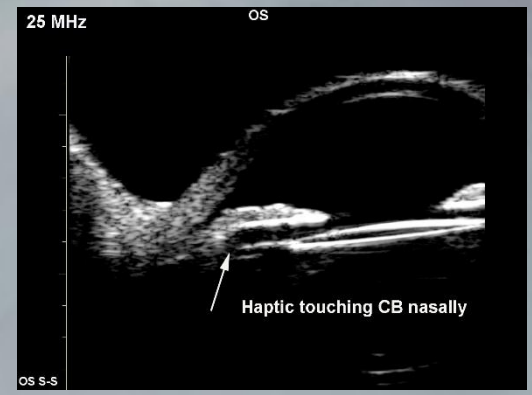
- The release of protein and white blood cells, pigment ,red blood cells in the AC.
- The trabecular meshwork can become blocked ---increase IOP.
- Contact with angle structures by the IOL can cause destruction of outflow structures and increased IOP.

DIAGNOSIS

UBM



OCT

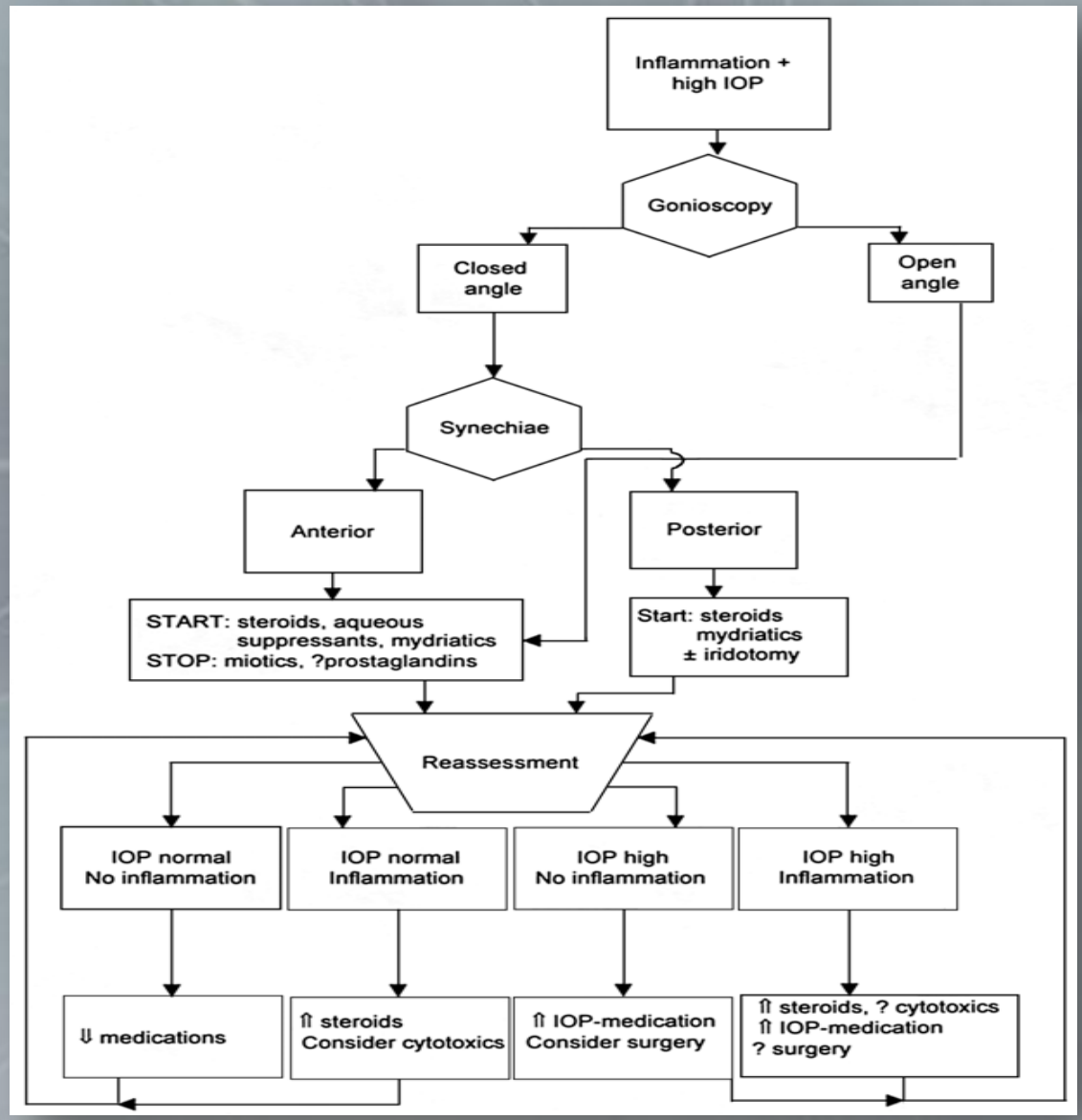


MANAGEMENT

- IOL repositioning
- IOL Removal
- Uveitis ⟷ topical corticosteroids
- Ocular hypertension ⟷ topical and systemic medications
- Hyphema ⟷ head elevation, cycloplegics, topical corticosteroids

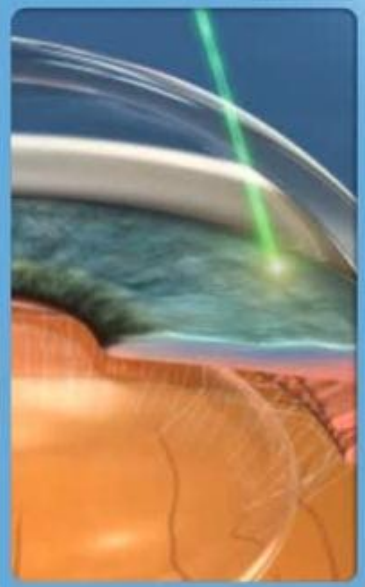
Management of Uveitis Glaucoma

- It is aimed :
- Cotrol intraocular inflammatory
- Contol IOP
- Treat underlying systemic disease


Inflammation + high IOP
Gonioscopy
Closed angle
Open angle
Synechiae
Anterior
Posterior
START: steroids, aqueous suppressants, mydriatics
STOP: miotics, ?prostaglandins
Start: steroids mydriatics ± iridotomy
Reassessment
IOP normal No inflammation
IOP normal Inflammation
IOP high No inflammation
IOP high Inflammation
⇓ medications
⇑ steroids Consider cytotoxics
⇑ IOP-medication Consider surgery
⇑ steroids, ? cytotoxics ⇑ IOP-medication ? surgery

TREATMENT

Laser Surgery



Medications

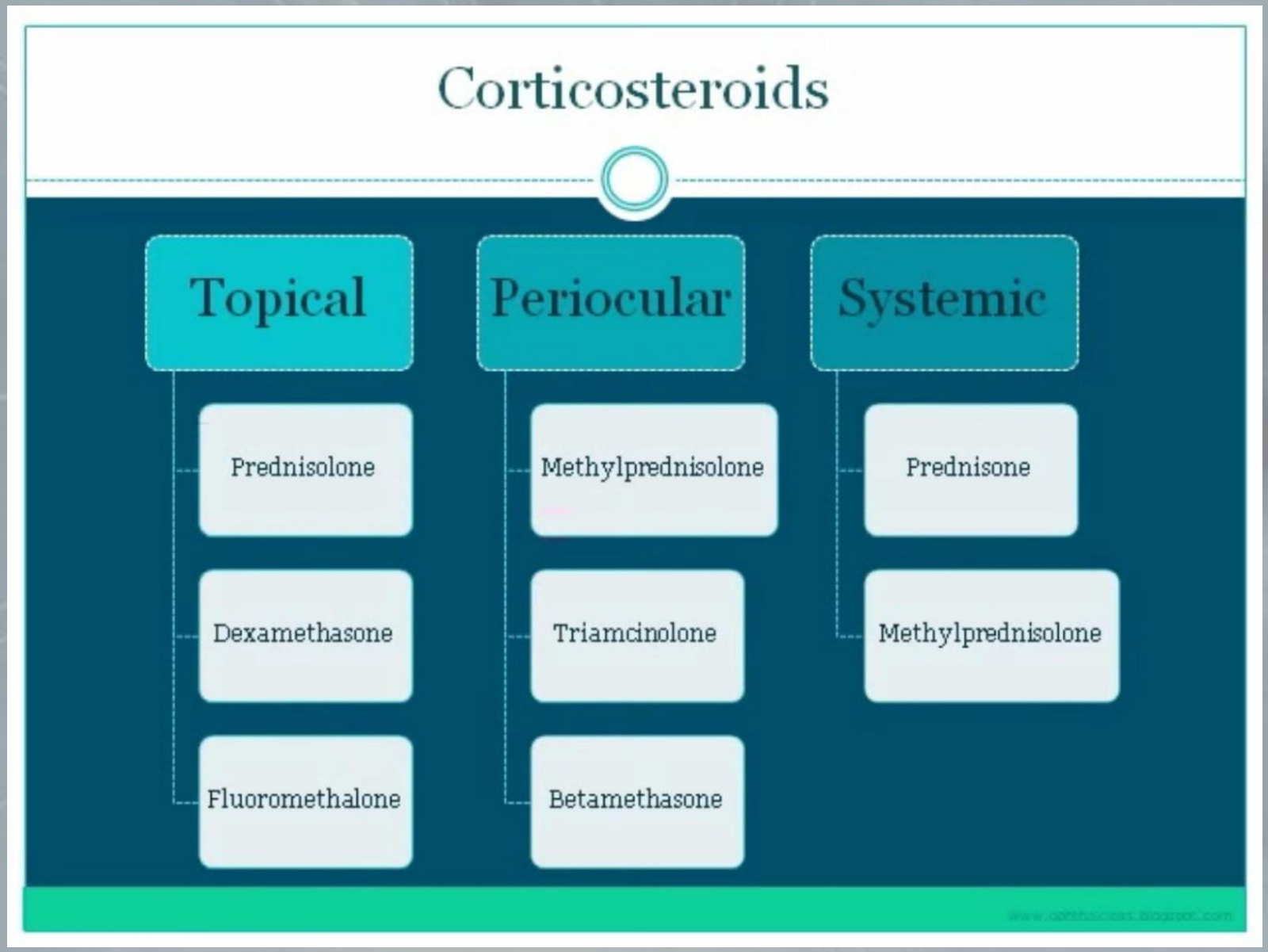
Traditional Surgery

MEDICAL THERAPY

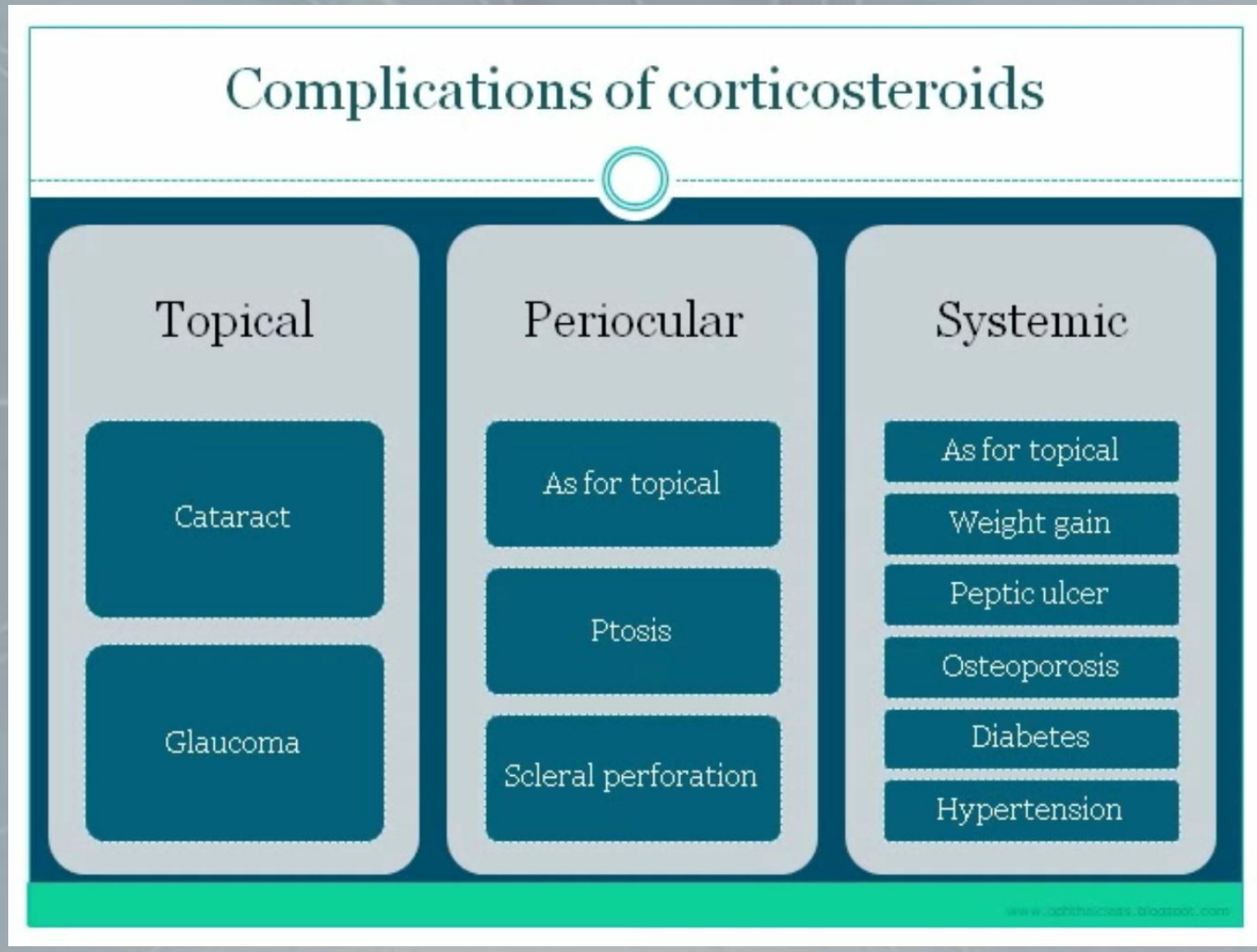
- Corticosteroids.
- NSAID.
- Mydriatic and cycloplegic agents.
- Aqueous suppressants.

  (B blocker, topical carbonic anhydrase inhibitors, alpha2 agonists).

- Hyperosmotic agents (for emergent control of acute pressure elevations)


Corticosteroids
Topical
Periocular
Systemic
Prednisolone
Dexamethasone
Fluoromethalone
Methylprednisolone
Triamcinolone
Betamethasone
Prednisone
Methylprednisolone

Complications of corticosteroids



Corticosteroids – the mainstay of therapy

- Depending on the site of inflammation and severity
  - Topical
  - Periocular
  - Systemic
- Topical drops will not be effective for intermediate, posterior and panuveitis
- 'Use enough soon enough'
- To always start with a higher dose and taper before stopping
- To investigate before starting

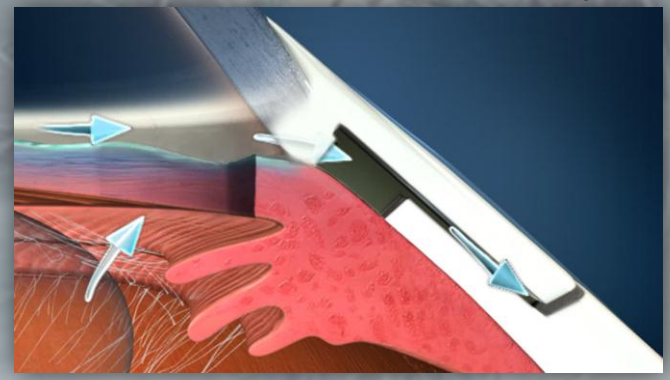
We Should Avoid:

- Miotics.
- Prostaglandins analogs.
- Surgery in eye active inflammation.

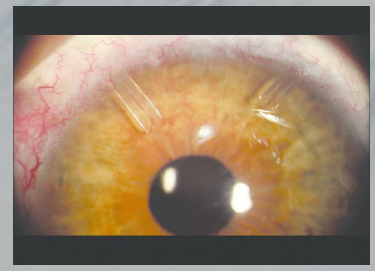
SURGERY

- GLAUCOMA FILTRATION SURGERY

Trabeculectomy with anti-metabolites (mitomycin C ,or 5-fluorouorascil have higher rates).

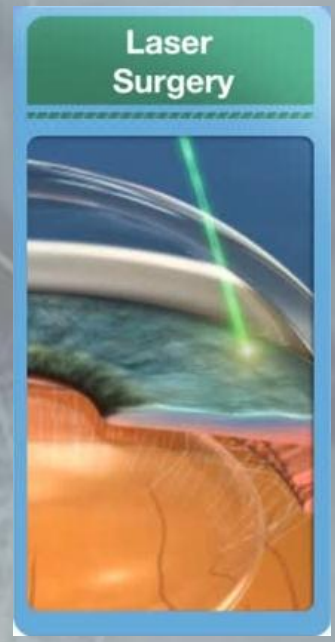


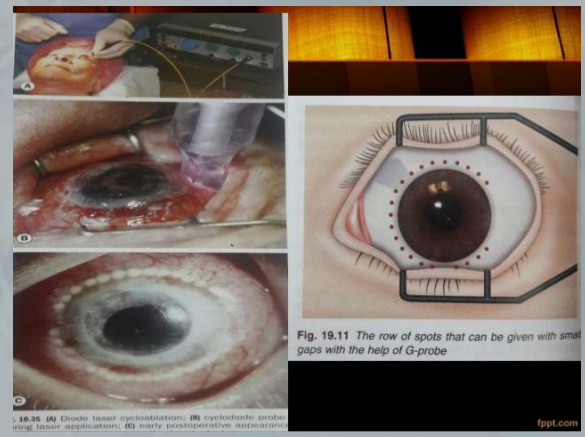
- Aqueous Drainage devices.



LASER THERAPY

- Argon or Nd:YAG laser iridotomy in management of angle closure glaucoma(iris bombe).
- Argon laser trabecularplasty is ineffective
- Cyclodestructive procedure:
  - Cyclocryotherapy.
  - Yag or diode Laser.
  - Transpupillary cyclophotocoagulation

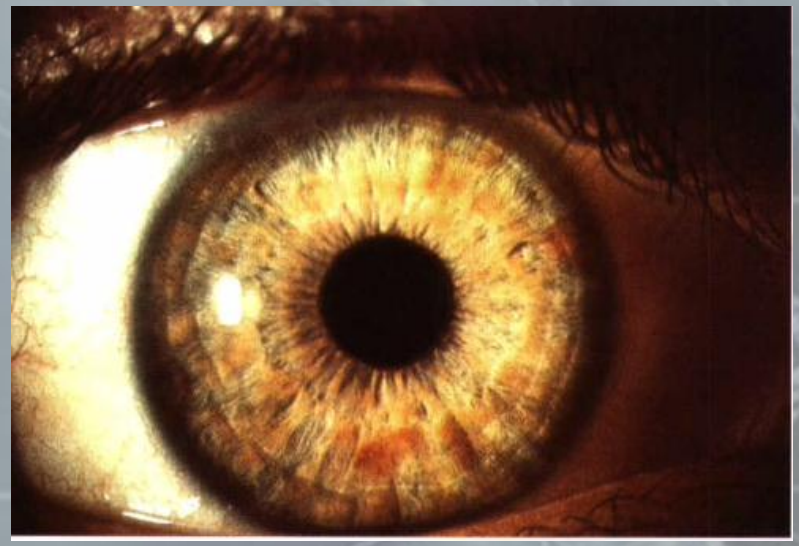


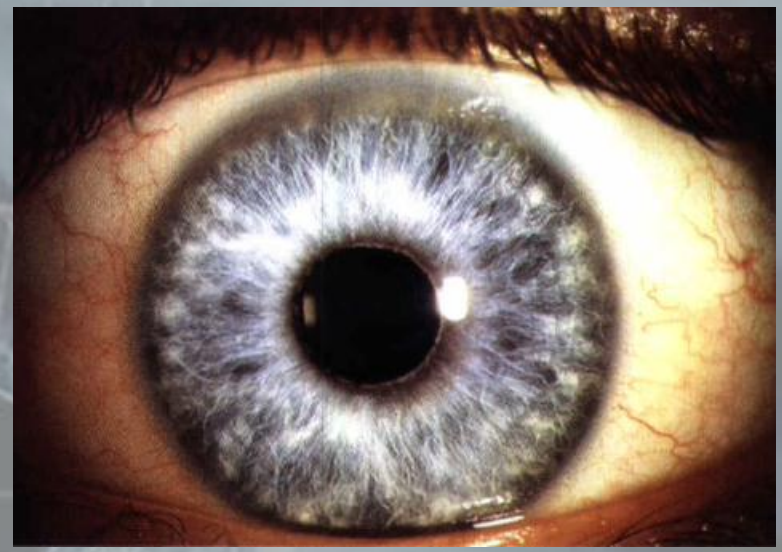


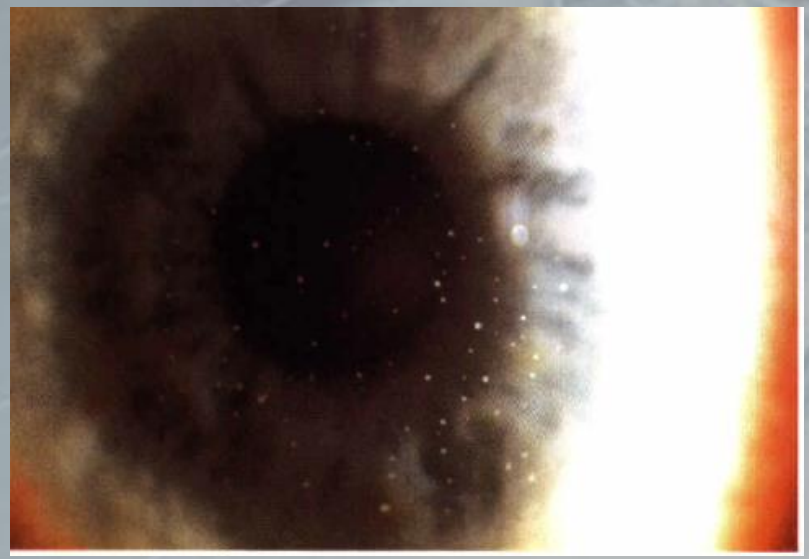
HOW ARE SPECIFIC UVEITIS SYNDROMS HANDLED ?

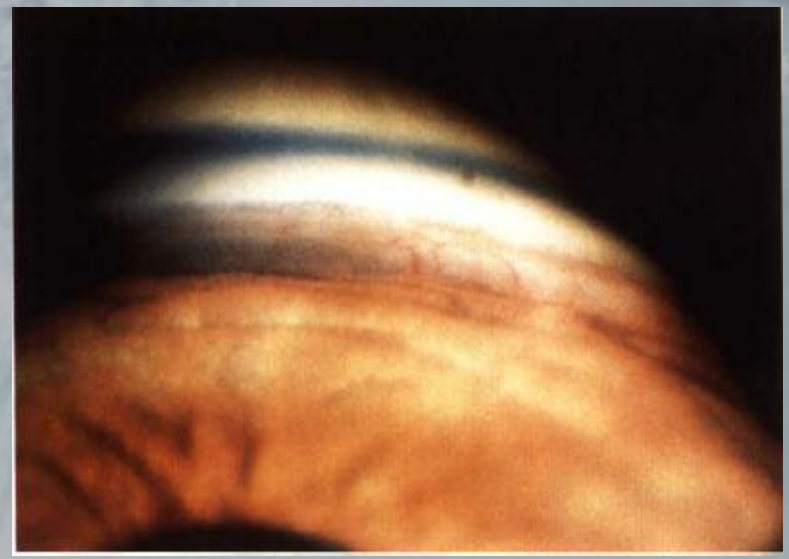
FUCH'S IRIDOCYCLITIS:

- ✓ Stellate ,diffuse keratic precipitates.
- ✓ Heterochromia (in 80%)
- ✓ Cataract.
- ✓ Chronic anterior uveitis without anterior synechiae formation.
- ✓ Low-grade anterior chamber reaction.
- ✓ Typically unilateral – insidious.
- ✓ Presents equally in middle-aged men &women.
- ✓ About 15 % are Secondary open Glaucoma.
- ✓ Gonioscopy: fine blood vessels that cross trabecular meshwork.



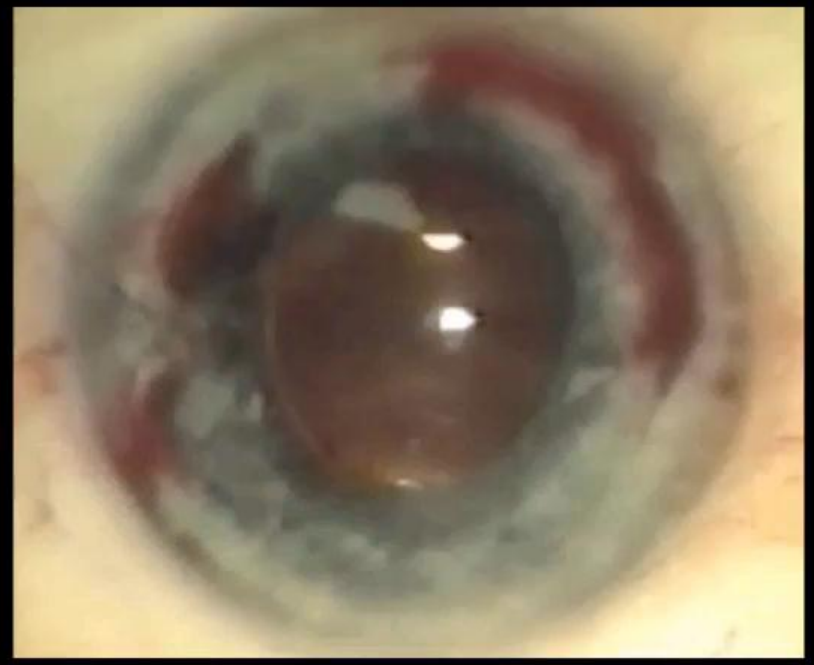






Signs

- Heterochromia
- Keratic precipitates
- Featureless iris
- Iris neovascularization
  - very fragile vessels



MANAGMENT

A. Corticosteroids are not effective

B. Medical therapy starts with aqueous suppressants.

C. Surgical therapy: Filtration surgerey or drainage surgery with anti metabolites.

POSNER- SCHLOSSMAN SYNDROME
(GLAUCOMATOCYCLTIC CRISIS)

- Unilateral- Recurrent episodes of high IOP
- Blurred vision- Mild eye pain.
- Anterior chamber reaction is minimal
- Corneal edema.
- Mild Iritis with few KP(small,discrete, round).
- Usually individuals 20-50 year are affected.
- May have peptic ulcers and gastrointestinal disorders.

POSNER- SCHLOSSMAN SYNDROME-CONT.

- Gonioscopy: open angle.
- Self limited and resolves spontaneously regardless of treatment.
- Aqueous suppressants and topical steroids may be indicated.

KEY POINTS

- Inflamed eyes typically have low pressure
- IOP can occur from both open angle and closed angle mechanisms
- Careful history and follow up can be done
- Closed angle has both block and non pupillary block mechanisms
- Corticosteroid use makes management challenging
- It is important to distinguish between uveitis glaucoma &uveitis –induced ocular hypertension

Take Home **Message**

Thank you.....