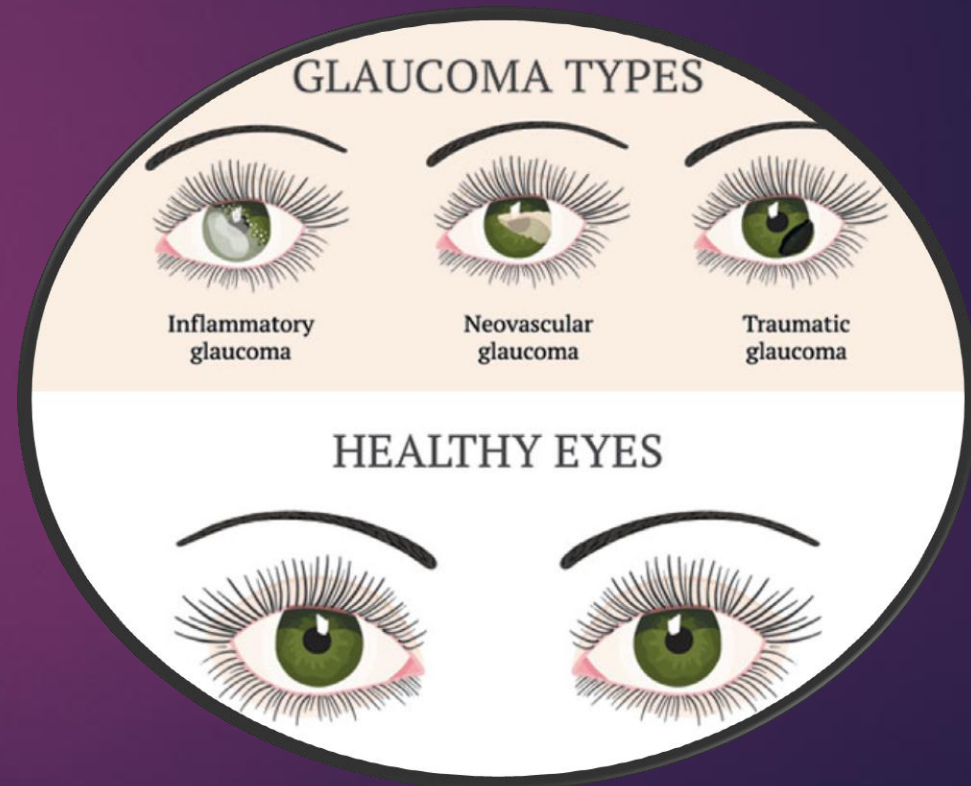


# Secondary Glaucoma

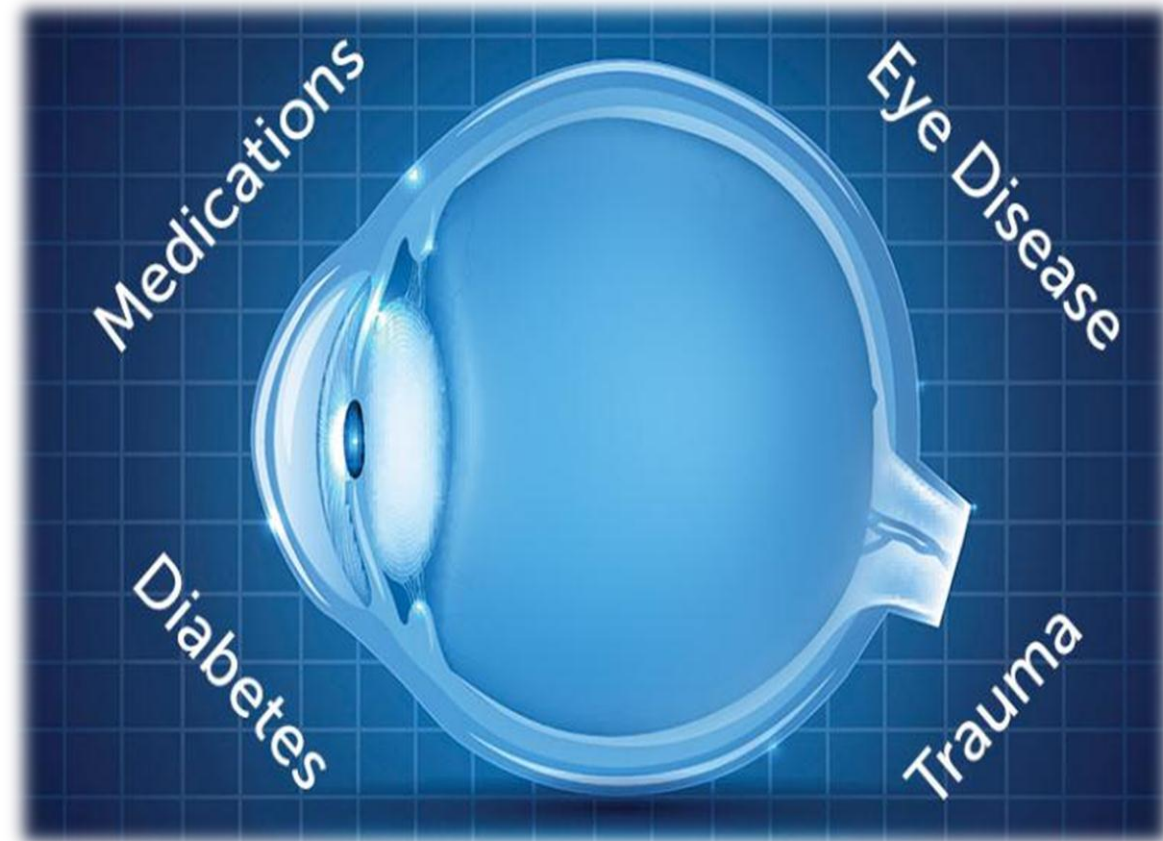
PRESENTED BY: DR. MOHAMAD ALKOUKOU

SUPERVISED BY: DR SHEREN KHODER



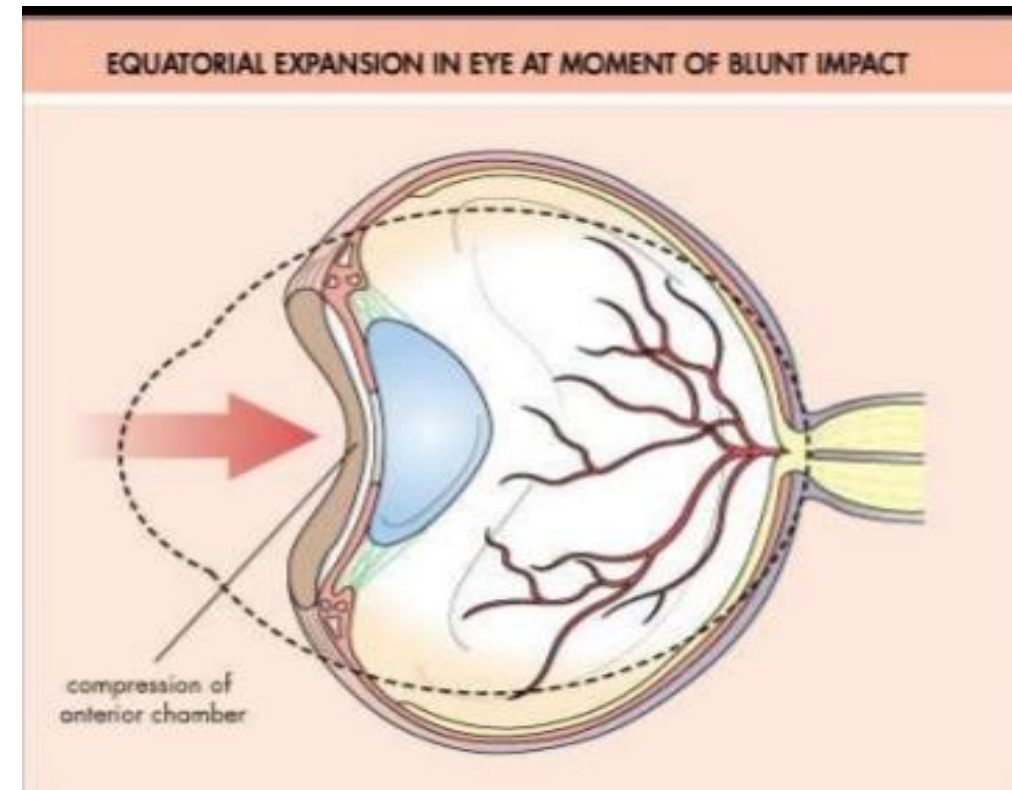
# Secondary Glaucoma

- ▶ **Glaucoma associated with trauma.**
  - ❖ Angle recession glaucoma
  - ❖ Hyphema
- ▶ **Steroid induced glaucoma.**
- ▶ **Malignant glaucoma.**



# Angle Recession Glaucoma ARG

- ▶ Secondary open glaucoma that is associated with ocular trauma.
- ▶ Recession of anterior chamber angle is common after ocular trauma **BUT** small percentage go on to develop glaucomatous optic neuropathy.
- ▶ ARG can occur after days, months or even years later after trauma.



# Angle Recession Etiology

- ▶ Tear in ciliary body between longitudinal and circular muscle layers.
- ▶ Clinically , abnormal widening of ciliary body on gonioscopy.
- ▶ **60%** of eyes with non-penetrating or concussive trauma will develop some degree of angle recession
- ▶ **60% to 100%** of eyes with traumatic hyphema will develop angle recession.



# Risk factors

- ▶ 1) Numbers of clock hours of AR: **180 to 240** ((higher risk))
- ▶ 2) Increased pigmentation at angle.
- ▶ 3) Elevated baseline IOP.
- ▶ 4) Hyphema.
- ▶ 5) lens displacement.

Note: 4% to 6% of people with angle recession will go on to develop ARG at 10 years.

# Pathophysiology

▶ Blunt trauma



▶ Tear between circular & longitudinal muscle



▶ Decrease outflow facility from



▶ Direct damage to TM  
(Scarring & sclerosis)



endothelial proliferation over TM

▶ POAG is more common in fellow uninjured eye

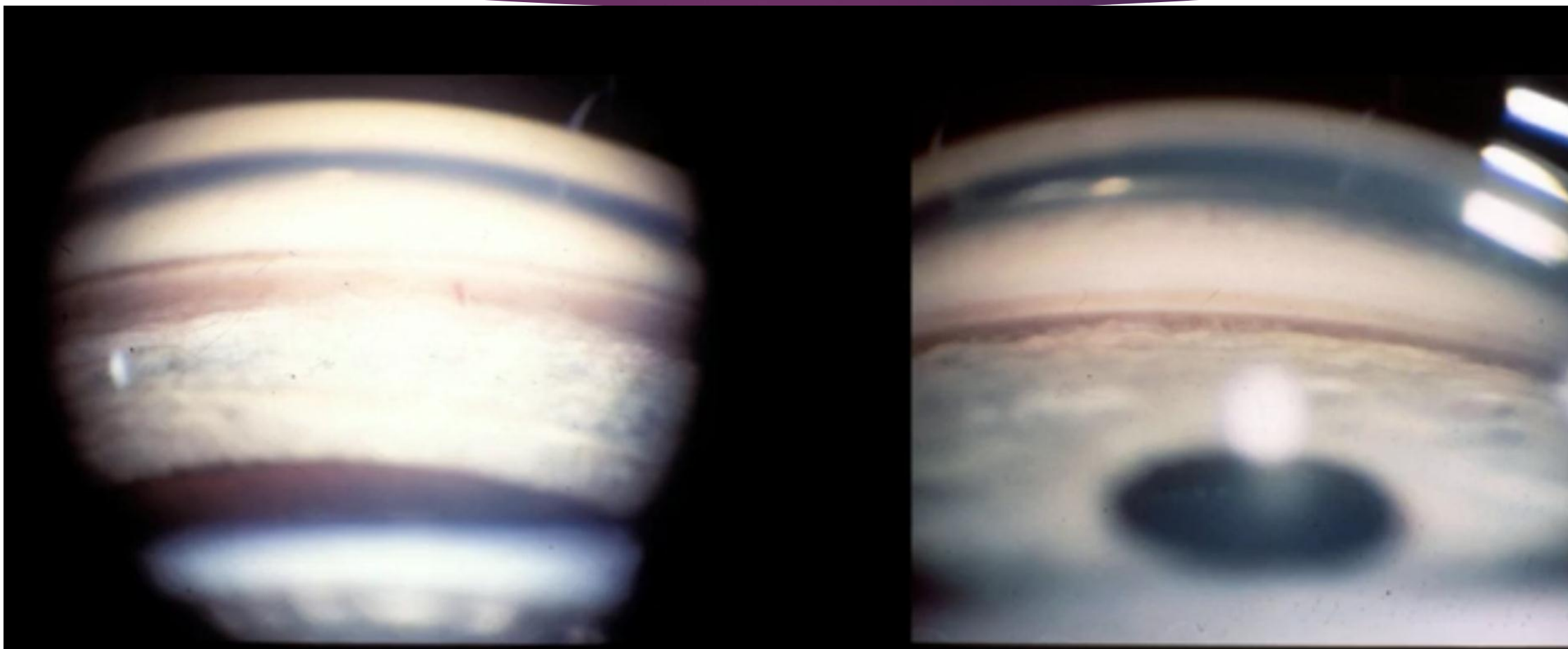
▶ 50% of these patients will suffer increase IOP in the contralateral eye.

# Diagnosis

- ▶ Physical examination:
- ▶ Gonioscopy: (( key exam finding ))
- ▶ Irregular widening of ciliary body
- ▶ Torn iris process
- ▶ Increased prominence of scleral spur
- ▶ Best done with **direct gonoscope**



*We should compare*





# ARG Diagnosis

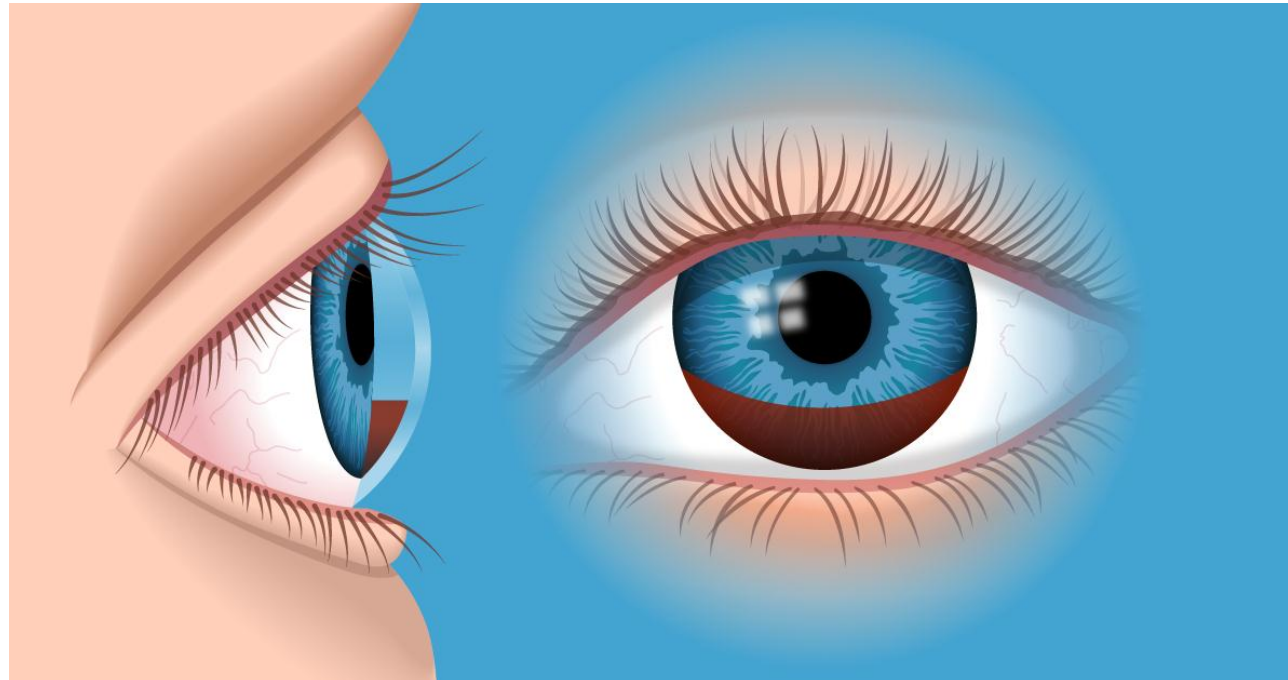
- ▶ *High IOP + nerve damage+ angle recession on gonioscopy.*
- ▶ Should be your **first** thought when encountering **unilateral glaucoma**.

# Management

- ▶ Observation up to 10 years or more if IOP ,discs are normal.
- ▶ Medically:
  - Same as POAG except:
    - miotics
    - Fair to poor response to medication
- ▶ Laser: SLT?!
- ▶ Surgery: trabectomy with MMC.

# Hyphema

- ▶ Blood in anterior chamber range from microscopic bleeding to total hyphema.
- ▶ Trauma or Recent ocular surgery are the most common risk factors
- ▶ Hyphema can cause **open** or **closed** angle glaucoma



# Etiology

## ▶ 1) Traumatic:

Most common blunt trauma maybe penetrating

## ▶ 2) Intraocular surgery or laser:

Higher in patients with past medical history that predispose them to irregular vasculature within AC

Ocular ischemia neovascularization

## ▶ 3) Neovascularization:

Abnormal blood vessels on IRIS.CILIARY BODY or within AC ANGLE

Result of posterior segment ischemia; DM.CRVO.....

## ▶ 4) Neoplastic:

Melanoma of iris .ciliary body

Retinoblastoma

## ▶ 5) Inflammatory/infectious:

HSV/HZV uveitis.

Fuchs heterochromia iridocyclitis.

## ▶ 6) Vascular anomaly:

Juvenile Xanthogranuloma JXG

## ▶ 7) Others:

Leukemia/Aneimia/hemophilic disorders and sickle cell anemia.

# Pathophysiology

## ▶ Blunt Trauma



Compressive forces .



Injury to iris, trabecular and ciliary body vessels.



Damage of arterial circle of iris.

## ▶ Penetrating Trauma

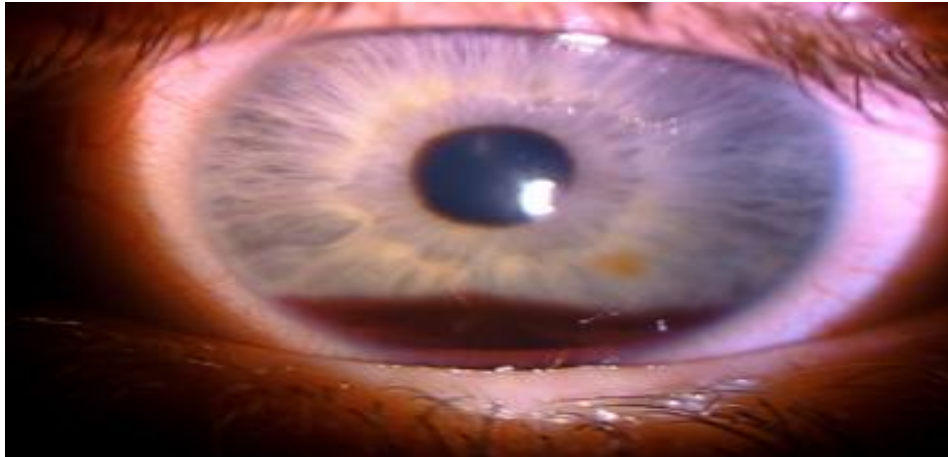


Direct damage to blood vessels

# ↑ IOP

## ▶ Open angle

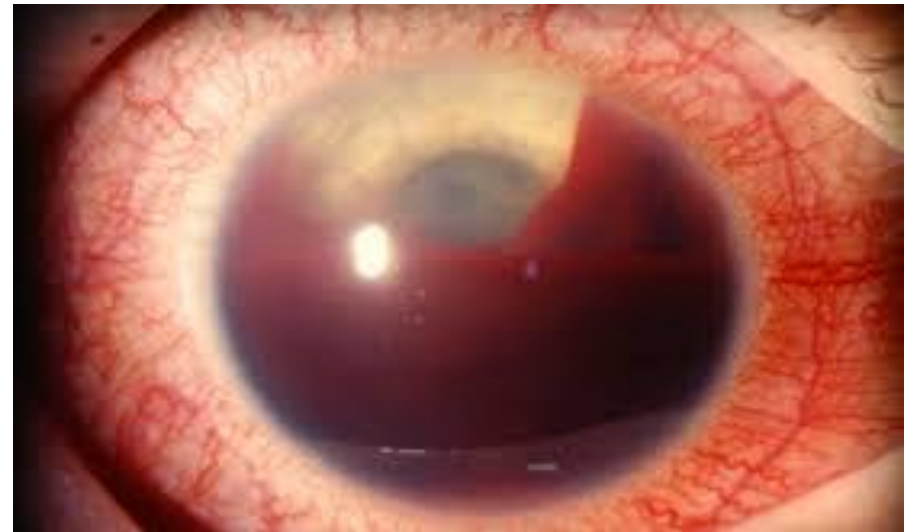
- ❖ Red blood cells, inflammatory cells.
- ❖ Obstruct trabecular meshwork.



Note: 50-90% of these patients develop angle recession or red cell glaucoma.

## ▶ Closed angle

- ❖ pupillary block.
- ❖ clots in anterior chamber.



# Diagnosis

- ▶ *History*
- ▶ *Trauma .....*
- ▶ *Ocular surgery or laser....*
- ▶ *Systemic disease*
- ▶ *Drugs*

# Physical Examination

## ► Routine ophthalmic workup:

1: VA

2: Pupil examination






3: Anterior segment.....

4: IOP

5: Gonoiscopy

6: posterior segment



| Grade        | Anterior chamber filling                           | Diagram   | Best prognosis for 20/50 vision or better |
|--------------|--|---|---|
| Microhyphema | Circulating red blood cells by slit lamp exam only |    | 90 percent                                |
| I            | <33 percent  |    | 90 percent                                |
| II           | 33-50 percent                                      |   | 70 percent                                |
| III          | >50 percent  |  | 50 percent                                |
| IV           | 100 percent  |  | 50 percent                                |



# HYPHEMA



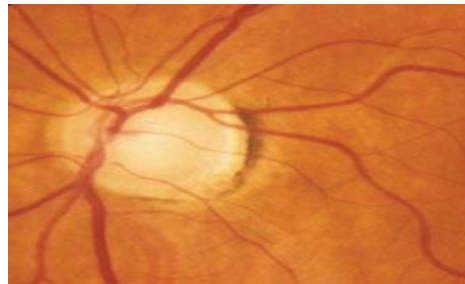
SANTANINA MUSAHARI, R.N.



| <b>Size of Hyphema</b> | <b>% with IOP Increase</b> |
|------------------------|----------------------------|
| <50%                   | 14%                        |
| >50%                   | 27%                        |
| 100% (not 8-ball)      | 52%                        |
| 100% (8-ball)          | 100%                       |

# Complications

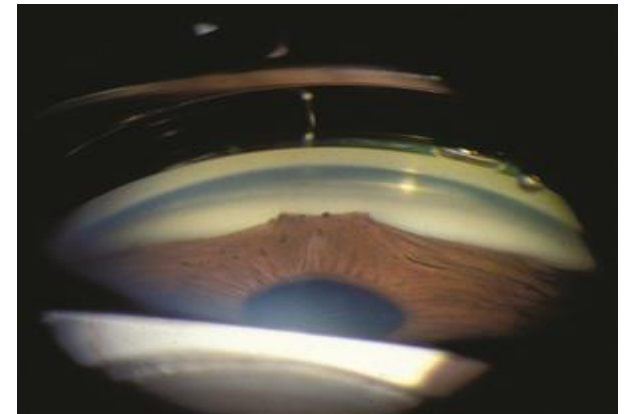
▶ **↑IOP & optic atrophy**



▶ **posterior synechiae**



**peripheral anterior synechiae**





▶ **Rebleeding : 3.5% to 38%**

- ▶ Occurs when the initial clot retracts and lyses usually within the first 5 days after injury
- ▶ More sever & more likely to cause complication

▶ **Risk factors:**

Hypotony or elevated IOP

Grade 3 hyphema and above

Black patients

systemic hypertension

use of aspirin

▶ **Corneal blood staining:**

▶ occurs as result from impregnation of the corneal stroma with hemoglobin&hemoseiderin

▶ **Predisposing factors:**

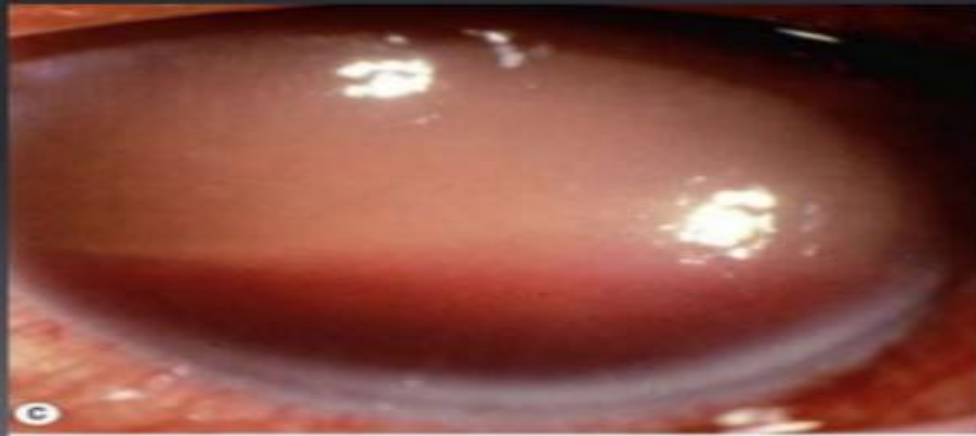
- 1) prolonged & large hyphema
- 2) dysfunction of corneal endothelium
- 3) ↑ IOP

It takes months if not years for cornea to clear gradually, maybe PKP

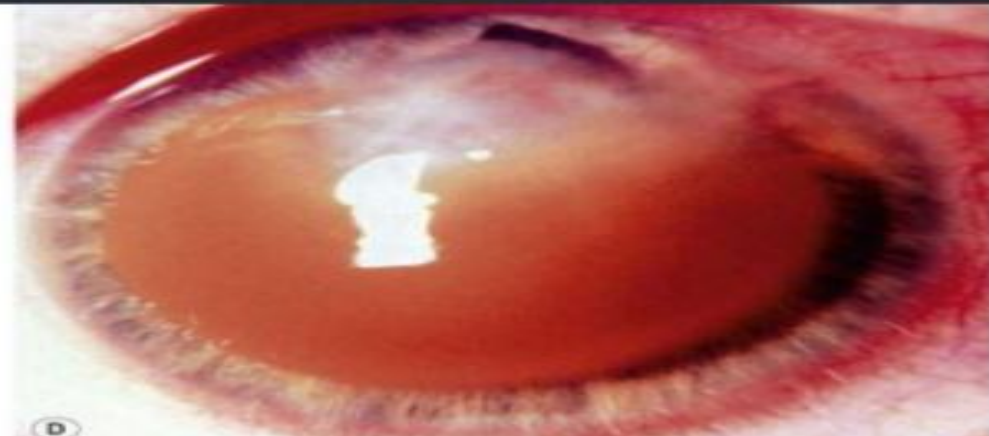


# Hyphaema

- Total hyphaema



Corneal Blood staining



# *Two main groups of patients require special attention from us*

- ▶ **1) children:**
- ▶ Risk of amblyopia.





## ► 2) sickle cell disease:

Patients have a much harder time clearing blood from AC & greater risk of pressure rise

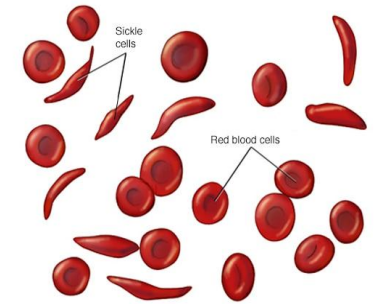
They are more sensitive to high pressures

Difficult to manage with drugs :

CAI: avoided may increase sickling tendency in AC

Adrenergic agonists may promote intravascular sickling by their vasoconstrictive

Systemic hyperosmotic agents may induce a sickle crisis in dehydrated patients



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# *Prognosis*

Based upon size of initial hemorrhage

- ▶ **Good** 1/3<sup>rd</sup>
- ▶ **Fair** from 1/3<sup>rd</sup> to 2/3<sup>rd</sup>
- ▶ **Poor** 2/3<sup>rd</sup>

# Management

## ▶ *General instruction:*

keep head of bed elevated

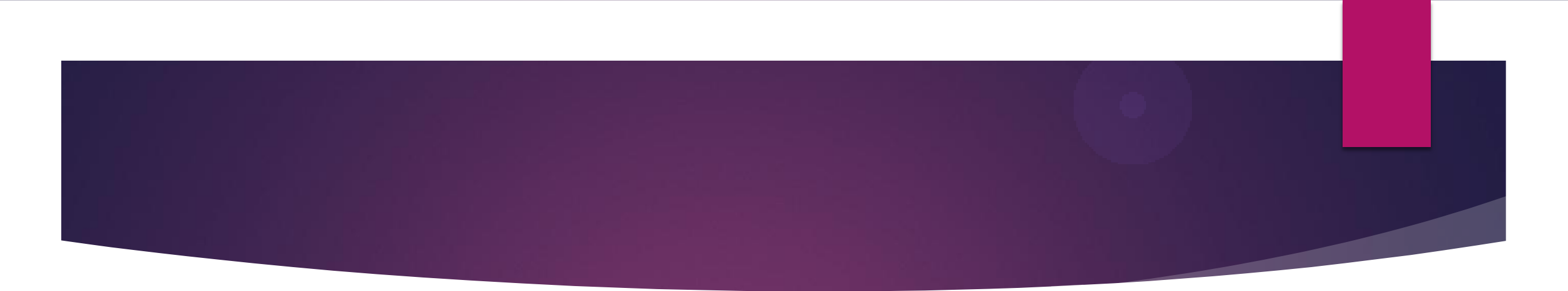
- ▶ Activity restriction
- ▶ Avoid Aspirine&NSAIDS

## ▶ *Medication:*

- ▶ Topical steroid
- ▶ Topical cyclopegics
- ▶ ↑IOP:
  - ▶ B-blocker
  - ▶ A-agoinsts
  - ▶ Avoid Miotics&prostaglaindine

CAIS

hyperosmotic agents

- 
- ▶ ant- fibrinolysis :Aminocaproic acid ACA &traexamis acid TA
  - ▶ Has been shown to be benefit to prevent re-bleeding
    - ▶ but has a host of side-effects
  - ▶ NOT used routinely
  - ▶ Beneficial in patients at higher risk for rebleeding or other hyphema associated complications

# Indication For Hospital Admission

- ▶ 1) non-compliant patient.
- ▶ 2) patients with bleeding diathesis or bleeding dyspraxia.
- ▶ 3) severe ocular or orbital injuries.
- ▶ 4) ↑ IOP with sickle cell anemia.



# *Indication For Surgical interventions (paracentesis and washout AC)*

- ▶ 1) large Hyphema > 10 days
- ▶ 2) total Hyphema > 5 days
- ▶ 3) IOP > 50 mmhg for 2 days (with maximum therapy)
- ▶ 4) IOP > 35 mmhg for 5 days (with maximum therapy)
- ▶ 5) IOP > 24 mmhg for 24 hours in sickle cell anemia
- ▶ 6) corneal blood staining

# Steroid induced Glaucoma

- ▶ Secondary open angle glaucoma that results from use of steroids.
- ▶ incidence of steroid induced glaucoma in general population is unknown.
- ▶ The incidence is related to type,dose,route of steroid administration and presence of risk factors.
- ▶ Significant elevation in IOP in response to topical steroids have been reported in
- ▶ 50% to 90% in glaucoma patients
- ▶ 5% to 10% in normal people.

# *Risk factors*

- ▶ 1) primary open angle glaucoma POAG.
- ▶ 2) first degree relative with POAG.
- ▶ 3) history of previous induced IOP elevation.
- ▶ 4) type 1 DM.
- ▶ 5) very young age ((less than 6 years)) or older age.
- ▶ 6) high myopia.
- ▶ 7) penetrating keratoplasty especially in eyes with fuchs endothelial dystrophy or keratoconus.
- ▶ 8) connective tissue disease.



# *Etiology*

- ▶ Increased glycosaminoglycan's in trabecular meshwork impede aqueous outflow
- ▶ Reduce membrane permeability of the trabecular meshwork
- ▶ Reduce local phagocytic activity by cells and break down of extracellular and intracellular structural proteins.



▶ **Topical ocular preparations:**

Risk of IOP-rise increase with duration of use & it's anti-inflammatory effect.

Dexamethasone & prednisolone increase IOP more than flurometholone ,hydrocortisone .rimexolone

▶ **Periocular:**

Subconjunctival,subtenon,retrobulbar

▶ **Intravitreal:**

50% of patients that receive intravitreal triamcinolone develops IOP elevation

Between 2 to 4 weeks after injection

▶ **Dermatologic**

▶ **Systemic**

# Management

## ▶ 1)discontinue steroid:

Acute form of IOP elevation normalize in days

Chronic form of IOP elevation normalize in 1 to 4 weeks

### **Note:**

If topical steroid should be used → weaker or less-pressure inducing steroid may help

In small subset of patients IOP remain chronically elevated despite discontinuation of steroid

## ▶ 2)Glaucoma treatment as usual

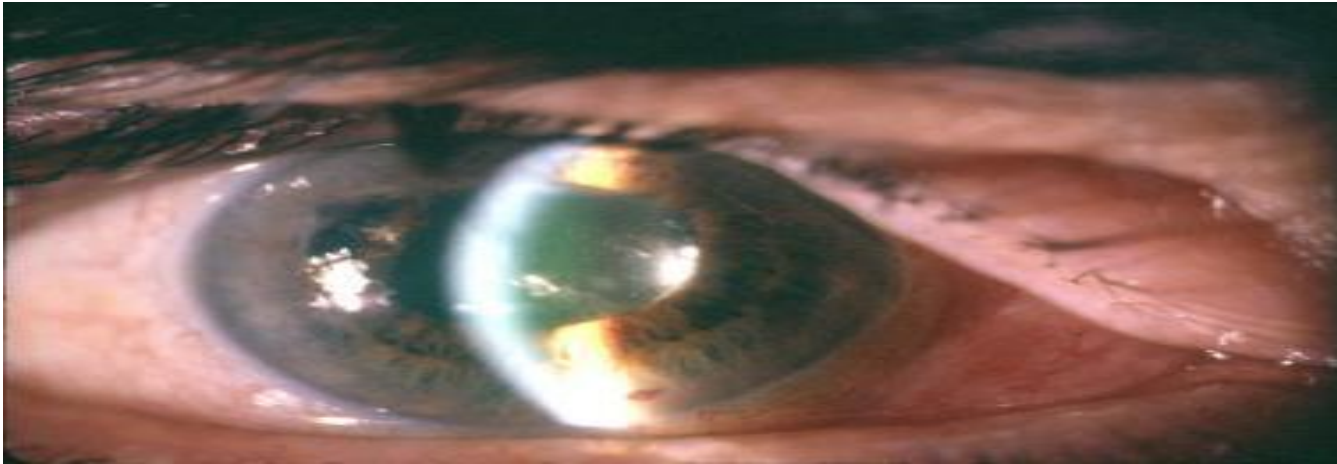
▶ First medically with anti-glaucoma drugs

▶ Laser : less effective

▶ Filtration surgery: similar result to POAG

# *Malignant Glaucoma Aqueous Misdirection*

- ▶ Secondary angle closure that presents with:
- ▶ **↑ IOP+ Shallowing of central peripheral AC**
- ▶ It is diagnosis of exclusion, requires exclusion of other clinical entities such as
- ▶ Choroidal hemorrhage, choroidal effusion and pupillary block
- ▶ This syndrome usually occurs following penetrating surgery of the eye, although it has certainly been reported following laser procedures



# *Pathophysiology*

- ▶ Eye changes the direction of aqueous humor flow, instead of moving forward the pupil, the aqueous goes into the vitreous
- ▶ Flattening of AC

# Management

- ▶ Typically there is recent history of eye surgery .
- ▶ ↑IOP + central & peripheral flat AC (not iris bombe) + normal b-scan.
- ▶ Often the episode can be treated with cycloplegics & aqueous suppressants
- ▶ If fails ....
- ▶ The key component to resolving the attack is disruption of anterior hyaloid face via
- ▶ Laser-or ppv



***THANK YOU.....***