Lecture 4

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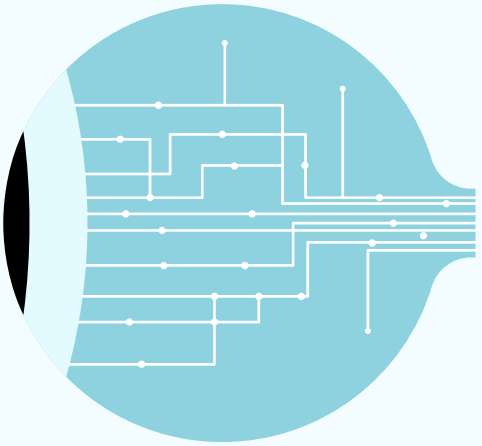


Acute Visual Loss

Presented by: Prof.Essam Osman

Objectives of the course:

- To define acute visual loss.
- To know common conditions that present with acute visual loss (causes, clinical manifestation and management):
  - Acute glaucoma, retinal artery and vein occlusions, retinal detachments, uveitis and keratitis.
- How to use ophthalmic instruments and results of investigations to differentiate between different causes.
- To know what are treatment options for different conditions.
- To know when to refer a case to a specialist.
- We might ask you about dx of a case, features/dx of a microscope, or to list the symptoms of a specific disease.

Color index:

Original content | Important | Golden Notes | Doctor's notes[439] | Doctor's notes[441] | Extra | Textbook

Acute Visual Loss

Acute= sudden
Chronic = gradually
*not related to time.

❖ Importance of eyes: "The eye is the window to the body"

- ◇ What is acute visual loss (AVL)?
  - ■ **Sudden** onset of blindness or significant visual impairment. Loss of vision is usually considered acute if it develops within a few minutes to a couple of days.
  - ■ It is a disaster for most people and you should be able to evaluate such a patient and be able to recognize situations requiring urgent action.
  - ■ It may affect one or both eyes, all or part of visual field, or it may arise from a pathology in any part of the visual pathway

❖ Etiology:

- ◇ Acute visual loss (AVL) can be classified by:
  1. Presence of pain.
  2. Structure affected

439 Slides

Acute Visual loss classified by STRUCTURE

- Media opacities:
  - Something interferes with the passage of light from cornea to vitreous; Usually the pathology is not within the lens as it only causes visual loss in cases of very severe trauma.
- Retinal disease:
  - Improper absorption of light.
- Optic nerve disease.
- Visual pathway or neurological disorders:
  - Stroke/neuritis.
- Functional disorders.
- Acute discovery of chronic visual loss:
  - Having visual loss for long time and they just noticed it and came to ER
  - Usually unilateral, the patient will close one eye (the good eye) and notice he can't see from the other eye and rushes to the emergency.
- All of the above may cause mild, moderate, severe visual loss or total blindness.

❖ Trauma causing :

1. Rupture of globe
2. Retinal detachment
3. Traumatic cataract
4. The lens falls backward into the vitreous if the zonules are ruptured (traumatic lens subluxation
5. Bleeding

Acute Visual Loss

439 Slides

Acute Visual loss classified by PAIN

PAINFULL	PAINLESS

• Acute Angle Closure Glaucoma:
  ○ Can be acute or chronic (and becomes open angle).
  ○ In the past, they misdiagnosed it with MI due to pain severity, they presented with severe headache, drop of vision, severe eye pain, nausea and vomiting
	• Vitreous Hemorrhage:
	  ○ It can be painful if it is traumatic.

• Uveitis:
  ○ It may be slow or sudden and acute.
  ○ Patient is always in pain.
	• Retinal Detachment:
	  ○ It could be caused by trauma or w/o trauma.
	  ○ The patient may have it and not discover it until covering one eye.

• Keratitis:
  ○ Infection (microbial keratitis) or inflammation of cornea.
  ○ Very severe pain, more than uveitis
	• Retinal vascular occlusions:
	  ○ Arteries/veins.

• Hyphema (Traumatic):
  ○ Can be asymptomatic unless if it's associated with other things.
	• Optic neuritis:
	  ○ It happens in cases with MS could present with pain or without.
	  ○ usually not painful but eye movement may be painful.

• Endophthalmitis: infection of the eye as a whole (infection of the globe).	• Ischemic optic neuropathy.
	Cerebrovascular accident (CVA) (or stroke).
	Functional "no underlying cause"

Acute Visual Loss

Causes Of Acute Visual Loss

- Media Opacities
  - Vitreous Hemorrhage
  - Corneal Ulcer
  - Hyphema
  - Corneal Edema
    - Acute Angle Closure Glaucoma
- Retinal Disease
  - Retinal detachment
    - Diabetic Retinopathy
  - vascular Etiology
    - Retinal Artery/ vein
- Optic Disease
  - Optic neuritis
  - Ischemic optic nerve
- Visual Pathway Disease
  - Visual Field loss
  - Cortical Blindness
- Functioning
  - Hysterical
  - Malingering

👨‍⚕️ **dr note ; Order is based on commonest causes, in the exam I will put a case about either of these**

Chronic visual loss

- Cataract 80-90%
- Chronic glaucoma
- Diabetic retinopathy
- Age related macular degeneration
- Retinitis pigmentosa

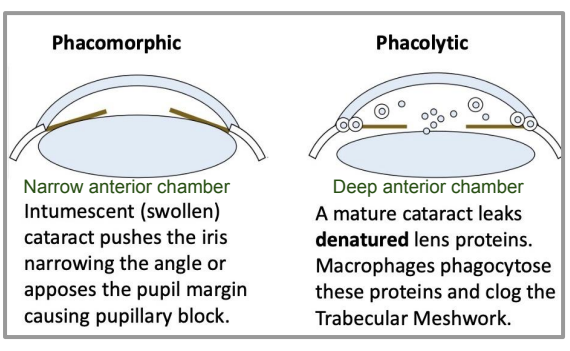
Acute Visual Loss

Important to differentiate between them!!!!!

- Acute glaucoma
- Vascular occlusion
- Retinal detachment
- Optic neuritis
- Neovascular glaucoma

SAQ :Acute presentation on top of chronic etiology e.g. cataract induce acute angle glaucoma
How chronic cataract ct change to acute vision loss(acute glaucoma)?
By one of these mechanism :
1-Phacomorphic : The lens gets enlarge and close pupile (angle closure)
2-Phacolytic : the lens become degeneration,get out of capsule and make some source uveitis

Possible SAQ
Pt with cataract suddenly present to the hospital with increase IOP



History of sudden visual loss

- What is the patient's **age** and general medical Condition? Optic neuritis <40 , acute glaucoma >40
  - For example you don't expect an elderly patient to present with optic neuritis.
  - If pt is diabetic for example; You would expect certain causes of visual loss
  - Young with no systemic disease ⟶ think about neurological causes: optic neuritis, retinal detachment or trauma.
  - Old with chronic medical condition ⟶ vascular cause.
- Is the visual loss transient, persistent, or progressive?
  - Transient: Vascular, migraine (ex: Amaurosis fugax).
  - Persistent (continuous) such as retinal detachment, hemorrhage, or optic neuritis.
  - Progressive: non-vascular, could be the progression of optic neuritis.
- Is the visual loss monocular or binocular?
  - Mononuclear: e.g.retinal detachment,optic neuritis (before optic chiasm decussation) such as optic neuritis.
  - Binocular e.g. migraine, DM retinopathy (after optic chiasm decussation) such as cortical blindness.
    - Think about central causes and confirm it by pupillary reflex ⟶ it is 100% normal.
- Did the visual loss occur suddenly, or it developed over hours, days or weeks?
  - Sudden ⟶ vascular (ischemic, central retinal artery occlusion).
  - Hours ⟶ acute angle closure glaucoma.
  - Days-Weeks ⟶ optic neuritis and retinal detachment.
- Did the patient have normal vision in the past and when was vision last tested?
  - Some people will only realize loss of vision from one eye; when they cover the good Eye.
- Was pain associated with visual loss?
- Contact lens use? corneal ulcer.
- History of trauma?
- quite a number of people will realize loss of vision from one eye when they cover the good eye.

Physical Examination

- Visual acuity testing : after vital signs
  - To see if the visual loss is mild, moderate, or severe.
- Confrontation visual fields test:
  Is it complete or partial vision loss?
  - It is useful if there is a pathology in the distal part of visual pathway.
  - If it is suspected in the history, so it is useful in neurological deficit.
- Pupillary reactions (very important). For the pathway
- External examination of the eye with a pen light:
  - We look at the eye in general to see if there's any trauma.
- Ophthalmoscopy exam: (Direct)
  - It can exclude media opacity; we observe the red reflex: In normal people it is present and equal in both eyes.
- Tonometry to measure the intraocular pressure. Acute Angle Glaucoma
- Biomicroscopic examination (Slit lamp examination)

Sudden painless VL and absence of red reflex by direct ophthalmoscope -> B scan is the best

MCQ: \
1-65 year old patient complaining of chronic visual loss? Age related macular degeneration.

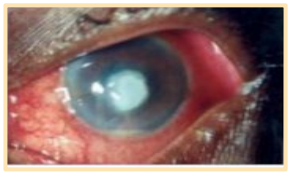
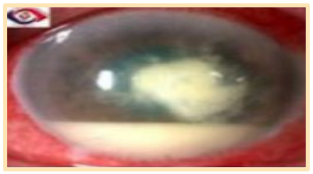
2-35 year old female presented with acute visual loss, On exam she had APD, diagnoses? Optic Neuritis.

Media Opacities

439 Slides

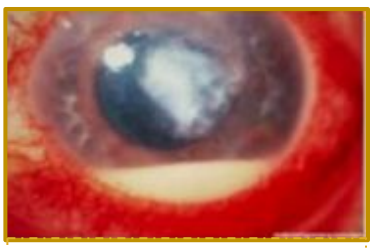
Corneal Ulcer

- When there is a corneal opacity due to destruction of tissue by infiltration of microorganisms and WBCs.
- It could be viral, bacterial, fungal, neurotrophic protozoal or in etiology.

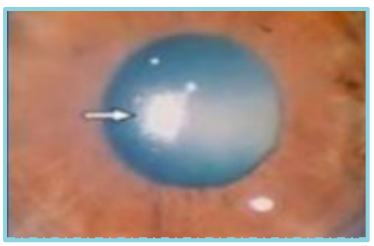



Corneal Edema

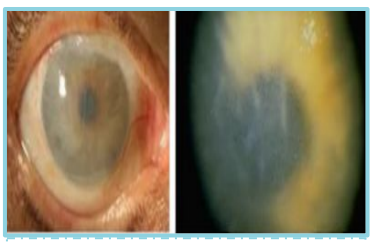
- Definition: Increased corneal thickness due to the abnormal accumulation of fluid in the corneal epithelium and stroma
- The cornea usually translucent and made of collagen fibers in a way that makes it clear but here it appears like a ground glass (مطحون او مسلح) rather than its normal clear transparent appearance (steamy cornea).
- The most common cause of corneal edema is increased intraocular pressure & occurs typically in **angle closure glaucoma.** Why in acute only not in chronic? Due to sudden increase in pressure
- Any acute infection of the cornea by a corneal ulcer may mimic corneal edema.
- This is almost always the presentation of corneal edema. Why does it cause edema? because the high intraocular pressure interferes with the function of the endothelium which is pumping the aqueous humor from the stromal cells to detergent the cornea. This is true for abnormal ocular pressure of any cause!
- Other causes of corneal edema include:
  - Contact lens
  - Corneal ulcer
- Dx + Tx: OCT is best for Dx. Tx is by hypertonic saline, managing glaucoma +/- steroids.



Corneal opacity & hypopyon
Complication: corneal ulcer (scarring) & glaucoma.



Abscess

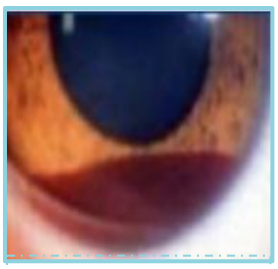


Extra

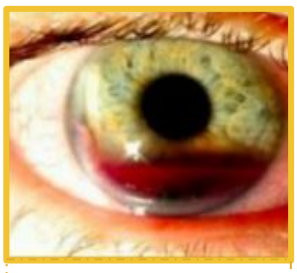
Media Opacities

Hyphema

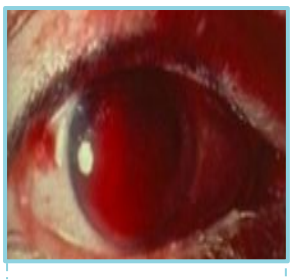
- Hyphema is blood in the anterior chamber.
- The hyphema is a direct consequence of blunt trauma to a normal eye. High IOP contribute in corneal blood staining
- If the eye is opened during trauma → emergency that requires surgery
- If closed → requires treatment to avoid rupturing vessels.
- However, it can occur with tumors in front of the eyes, advanced stages of diabetes (diabetic retinopathy: new vessels on the surface of the eyes which can bleed= neovascular glaucoma), intraocular surgery (post-op), chronic inflammation and uveitis which all cause neovascularization.
- **You have to rule out Sickle cell patients,** because prone to develop the bleed especially after trauma It may need evacuation in sickle cell patient, to avoid vascular accidents "there is high IOP and the deformed RBCs can't pass through the trabecular meshwork".
- Blood in the AC makes levels as it fills up and those levels are described by percentages, when it's filling the whole anterior chamber it's called "Eight ball Hyphema"(IMP).
- The most common cause of hyphema is trauma. In case of trauma, it usually resolves spontaneously within 3 days (bed rest and minimize the activity to avoid re-bleeding).
- This usually settles with rest, but a rebleed may occur in the first 5–6 days after injury.
- Steroid eye drops are given for a short time, together with dilation of the pupil. Steroids reduce the risk of rebleeds.
- The commonest complication is a raised ocular pressure, particularly if there is a secondary bleed



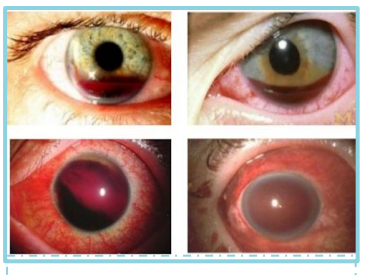
We Measure the height of blood in mm



Hyphema
Complications: posterior synechiae & glaucoma.



Eight ball hyphema
Filling of the whole anterior chamber



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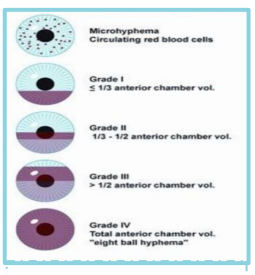
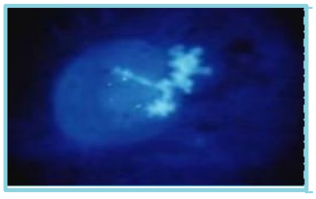


Extra

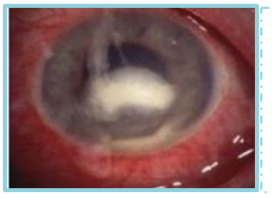
439 Slides

Uveitis

- Uveitis doesn't only cause visual impairment on the corneal side but also on the turbidity of the anterior chamber. In uveitis, the inflammation leads to changes in aqueous humor contacts, usually there is a protein present in the anterior chamber and its concentration is 1% of that in the serum. In severe uveitis, the concentration is similar to the serum



Viral herpetic herpes

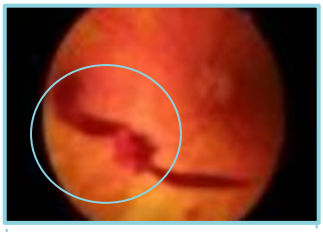


Infection: abscess in the cornea with hypopyon & congested eye.

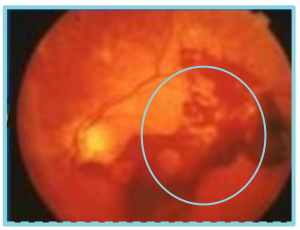
Media Opacities

Vitreous hemorrhage

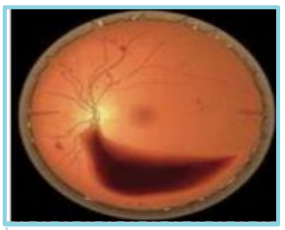
- Not a diagnosis rather is a sign of many diseases.
- Extravasation or leakage of blood into the areas in & around the vitreous humor of the eye.
- Any bleeding into the vitreous cavity will also reduce the visual acuity.
- Because it's a media opacity:
  - Opacity in the cornea? Edema.
  - Opacity in the vitreous? Blood.
  - Opacity of lens? cataract.
- After **trauma**, seen in **diabetics** or **after retinal vein occlusion**, acute posterior vitreous detachment and intraocular surgery; and it may accompanied subarachnoid hemorrhage because optic nerve is covered in the meninges (Terson's syndrome).
- Many diseases can cause VH, even TB can cause VH.
- **The most common cause of vitreous hemorrhage is diabetes** (MCQ).
- 2nd most common cause of vitreous hemorrhage is branched retinal vein occlusion.
- On examination:
  - If you cannot appreciate a red reflex with a direct **ophthalmoscope** and the lens appears clear, **you should suspect a vitreous hemorrhage.**
  - The diagnosis is **confirmed with a slit lamp examination through a dilated pupil.**
  - Suspect By ophthalmoscope Confirm By slit lamp
  - **B scan (bi-model ultrasound) is important** . also if there is dense blood obscuring the view
    - US tells you how dense the vitreous hemorrhage & if the retina is in place or detached.
  - Absence of red reflex means **MEDIA OPACITY** (where? It can be anywhere, in the cornea, AC, lens, vitreous, retina not in place) and then you assess it with a direct ophthalmoscope.
  - Leukocoria (leuko = white, coria= pupil), white pupils, is used to describe a reflex of pupils in pediatric patients.
- Management:
  - Bed rest & treat underlying cause.



Hemorrhage



Flame shaped hemorrhage is indicative that it happened due to malignant HTN (very important)



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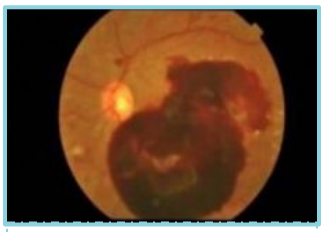


Extra

Acute Glaucoma

Classifications of Glaucoma

- According to Aetiology
  - Primary
  - Secondary
  - Congenital
- According to Appearance of angle
  - Open angle
  - Closed angle
  - Combined mechanism (mixed)

Dr. :
-don't forget about congenital glaucoma
-Imp to know the subtype:
Open or closed angle? Then is it Primary or sec?
-Primary : the lens clear but enlarge in size + already have shallow anterior chamber
-Sec: cataracts + + increase in size and obstructive (not necessary shallow anterior chamber to be)

Example sec - closed angle **Phacomorphic -Phacolytic**

❖ Glaucoma:

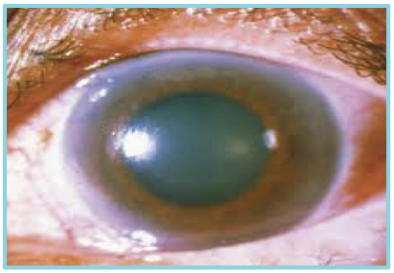
Damage to Optic Nerve! Not increase I.O.P
So optic nerve disease

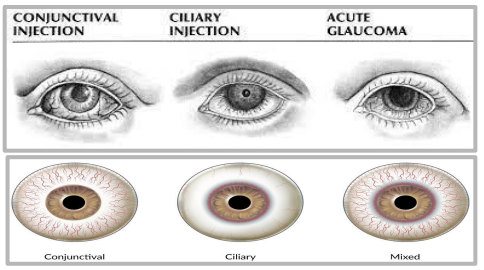
- A group of diseases with: characteristic **optic nerve damage** + **visual field loss** + **elevated IOP (variable)**
- In primary glaucoma there's no detectable ocular or systemic abnormality, often bilateral and often familial. It is the most common cause of optic neuropathy.
- To differentiate between open and closed angle glaucoma we use **GONIOSCOPY,** OCT too.
- **Risk factors:** short axial length (so female>male) , hyperopia in close angle(myopia in Open-Angle), shallow anterior chamber (as I said hyperopia is a RISK FACTOR!) ,above 40Y in close angle (open angle happen in any age).

- **Signs & symptoms(IMP):** Painful acute visual loss And collection of these:
  - Increased intraocular pressure. Absent in chronic cases due to adaptation.
  - Corneal edema.
  - Congested injected eye (angry looking eye) w/ mid-dilated non-reactive pupil.
    - Why mid-dilated non-reactive pupil? Normally the pupil either constricts or dilates. In glaucoma, the IOP is so high that the blood supply stops to iris muscles resulting in ischemia.
  - Redness

Acute Glaucoma Clinical Picture:

-Painful decrease VA (visual acuity)
-Ciliary injection
-Corneal edema
-Pupil semi dilated =mid-dilated non-reactive pupil
-Disc hyperemic (swollen)
-Congested injected eye (angry looking eye)

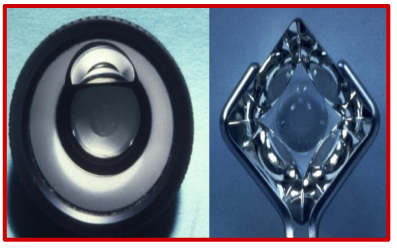


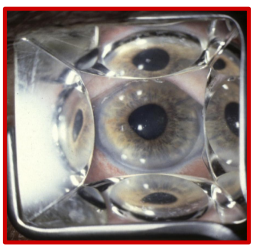


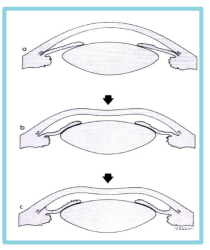
Imp to know the item!
COME IN EXAM!

Gonioscopy Lens (SAQ)

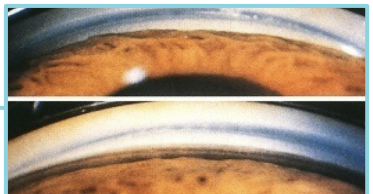
- Open Angle: trabecular meshwork will not be occluded by peripheral iris.
- Closed Angle: trabecular meshwork will be occluded by peripheral iris.

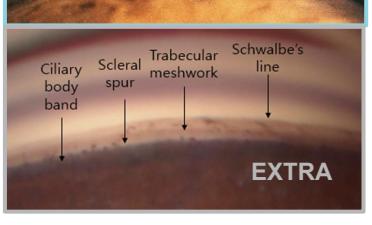


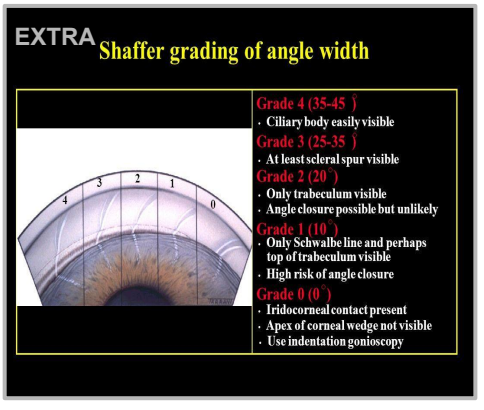




Dr : When you can see the structures completely it's called **open angle** when you can't see it then it's **closed angle** (iris attached to the lens = closed angle).
NO NEED FOR MORE DETAIL.
You can see in the pic that the angle is not completely seen so the pt might have tendency to get acute closed angle glaucoma we need to try and keep it open by laser.

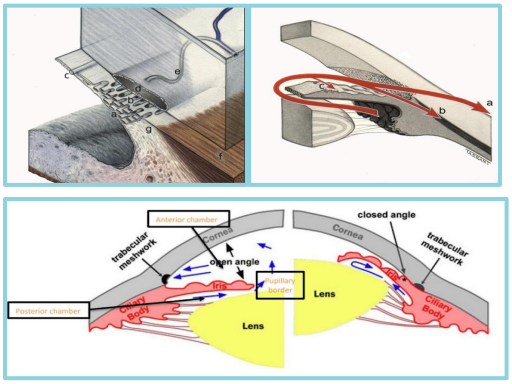






Acute Angle Closure Glaucoma

- Normally, the aqueous humor which is produced by the ciliary body non pigmented cells travels from the posterior chamber through the pupillary border into the anterior chamber then it gets drained in the angle 85% through the direct pathway trabecular meshwork, 15% through uveoscleral indir ect route.
- In Primary acute angle closure glaucoma the patients have a narrow angle to start with, and with aging which involves the lens getting bigger the angle will become more narrow until it totally occluded & no fluid can be drained through the trabecular meshwork.
- This causes the periphery of the cornea & the pupillary edge of the iris bow against the lens closing the posterior chamber & pressure is building up.



- The pathology behind acute glaucoma is **PUPILLARY BLOCK!** The pupil at semi-dilated vision get stuck/blocked with the lens at the center and it happens usually at night.

-The only 2 organs that increase in size with age (physiological) : pregnancy and **lens** (for enlarge lens not all old people will have glaucoma -need predisposing patient- so already angle is narrow and the lens when enlarge will precipitate acute attack.

-glaucoma will come because decrease drainage . But **nothing can increase secretion**
But can decrease secretion like in ciliary body atrophy
-

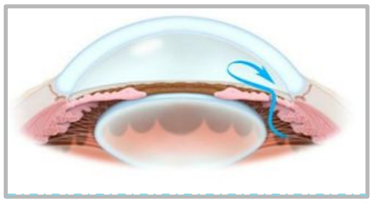
- **Aims of acute ACG management:**
  - Decrease IOP by:
    - Systemic + topical **Acetazolamide** (side effects of the drug (**SAQ**): bone marrow depression, renal stones, gastric pain/ulcer, hypokalemia, metabolic acidosis, depression)

Mnemonic : **Bone**. St**one**. Abdominal gr**oan**. Psychic m**oan**.

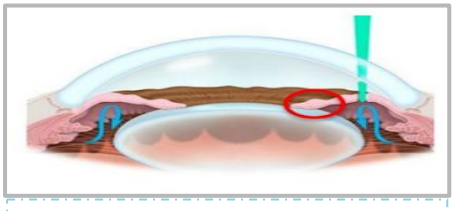
For hypokalemia the patient will present with tremor so we advise him to eat banana and orange (high potassium)

    - Topical B Blocker.
    - Pilo 2 (**pilocarpine) pupil constricted** (patient come with pupil dilated so we need to stretch pupil to make the laser iridotomy easier) (miotics drug)
      -decrease the pressure first by Acetazolamide then give pilocarpine.
    - **Peripheral laser iridotomy** (create an opening to allow the aqueous humor to drain).
  - Prevent future attacks in OU (oculus uterque, which means "both eyes") by **prophylactic peripheral laser iridotomy to the other eye.** Even if other eye normal. To prevent attack or glaucoma. **IMP**

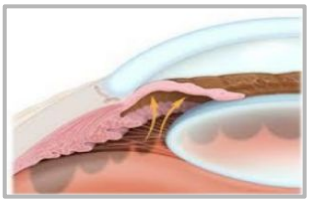
Production and absorption of the aqueous humor at the same level to prevent eye collapse (hypotony) low IOP

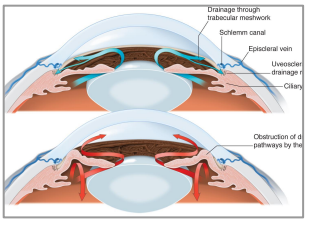


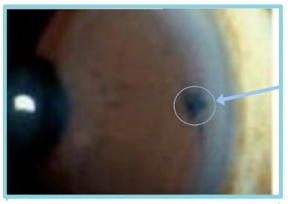
Management: Making a gap by laser (Yag peripheral iridotomy) You only need one opening



- If pt presents late the iris will stick to the lens causing (posterior synechia) *red circle*
-If the iris sticked to the trabecular meshwork or the cornea it is called peripheral anterior synechia (PAS)

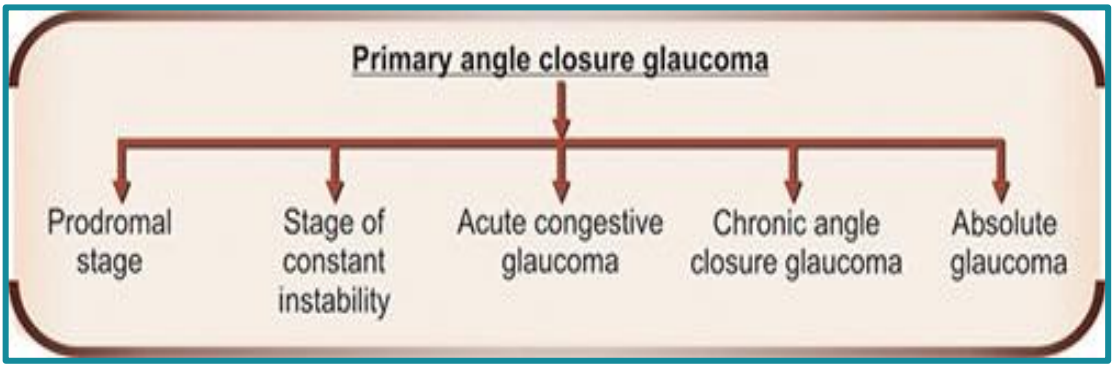






Peripheral laser iridotomy

Glaucoma



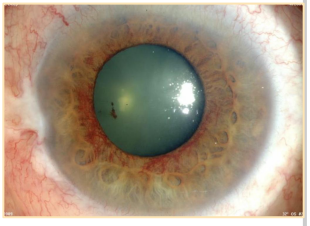
Scenario: about old female watching a movie in a dim light..
-Prodromal stage: starts when the patient turns off the light and **resolve when the light turned on** with other risk factors "MCQ 40 YO female driving at night,she develops sudden eye pain>> attack of acute glaucoma"
-intermittent attack: the patient stays in dim light despite the pain for longer time, the angle will not open completely and some **adhesions** will take place>> normal pressure but **Gonioscopy will show the adhesions.**
- Acute angle glaucoma **increase IOP**, severe pain and vomiting.
-if the patient go through recurrent intermittent attacks >>> chronic angle closure glaucoma **majority of pts present in this stage**
- absolute glaucoma

Neovascular glaucoma (NVG)

- **Common Causes:** **Diabetic retinopathy (in middle east)** , **CRVO(in europe)** , Carotid insufficiency
- **Pathophysiology: Retinal ischemia** (any disease cause retinal ischemia can cause NVG)cause neovessels in iris and angle release of proteins leading to inflammation in the angle and closure of the anterior chamber results in synechial angle closure glaucoma With sudden rise of IOP
- DM > Ischemia > retinal cells secreting VEGF > Neovascularization and vitreous hemorrhage
- Clinical features :acute Decrease VA , Corneal edema , Iris NVI , semi dilated Pupil , FUNDUS RETINAL ISCHEMIA

Treatment

- Acute management: Atropine, Diamox, Anti-VEGF
- **Main treatment** Of retinal ischemia is **pan retinal photocoagulation (PRP)**
- **+/- ANTI VEGF** (Avastin) to decrease new new vessel in iris and angle.
- +/- Glaucoma surgery
- The most severe type of glaucoma, very painful because inflamed eye (leakage protein..)
- IMP to understand the scenario if it is in acute situation or main treatment.



Rubeosis iridis
Diagnose ? NVG
What is in the picture?
Rubeosis iridis

Retinal vessel occlusion

- Retinal vein occlusion is much more common than retinal artery occlusion. Retinal vein occlusion is the second most common vascular disease of the retina (after diabetic retinopathy)

Central Retinal Artery occlusion CRAO

Retinal Vascular Occlusion

Clinical feature :

- A sudden, painless and often complete visual loss (no light perception) **in one eye** may indicate central retinal artery occlusion (CRAO).

Etiology:

- **1) Embolism as result of carotid artery atherosclerosis (most common) , Emboli as result of AF or endocarditis**
- **2) thrombosis**
- **3)rare causes** (vasculitis , arterial vast spasm)

Ophthalmoscopic findings :

- **A cherry red spot (HALLMARK DIAGNOSIS OF CRAO)** is seen at the fovea centralis due to the pallor of the perifoveal retina in contrast to the normal color of the fovea & underlying choroid (fovea is the thinnest part of the retina; thus, the opacity in the fovea is less compared with its surroundings).
- Grayish-white (cloudy) discoloration of the entire retina
- Several hours after a central retinal artery occlusion, the inner layer of the retina becomes opalescent (opaque, why? because of ischemia).
- A chronic cherry red spot is also a feature of the storage diseases such as Tay-Sachs pick disease and Niemann-Pick disease. محدش حيسألك في الكلام دا

Causes of cherry red spot on retina:

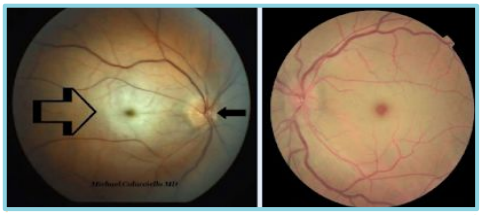
- Mnemonic: Cherry Tree Never Grow Tall : Central retinal artery occlusion | Tay-Sachs disease | Niemann-Pick disease | Gaucher's disease | Trauma (Berlin edema).
- There is no generally accepted acute management.

- Clinical scenario : Pt underwent cardiac valve transplant came with sudden painless loss of vision? CRAO

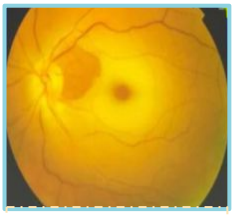
Case will become (valve replacement + acute unilateral vision loss (embolism) car accident and multiple bone fracture + acute unilateral vision loss (fat embolism)

- Treatment should be initiated as soon as possible, as permanent retinal damage occurs within 1.5 hours of central retinal artery occlusion.

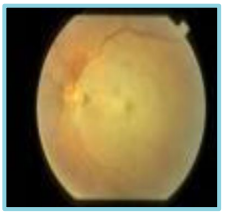
Normal perfused retina; why? 20% of people have a branched artery (cilioretinal artery) that pierces through the retina before the central retinal artery is formed & supplies the macula causing good vision even in the case of CRAO.

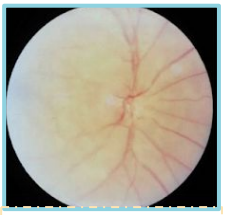


(There is an opaque retina with an edematous disc (small arrow) and the big arrow is pointing at the macula, the dark area is a cherry red spot of the fovea in the center of the macula. Which means: central retinal artery occlusion).

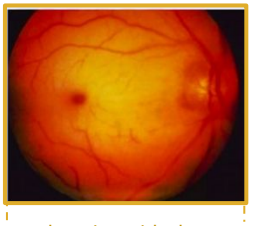


Extra





Opaque "pale" retina & attenuated artery > central retinal artery occlusion.



Pale retina with cherry red spot. Indicating central retinal artery occlusion.

Management

Managed within one hour or patient will loss the vision

- Decrease IOP. Diamox (Acetazolamide) to lower the IOP
- Asking the pt to rebreathe into a bag so CO2 causes vasodilation. Basically, for both branch and central occlusion you need to cause vasodilation.

Retinal vessel occlusion

This whole page wasn't mentioned by the Dr.

Branch Retinal Artery Occlusion

Retinal Vascular Occlusion Not interested for you

- When only a branch of the central retinal artery is occluded, vision is only partially lost upper nasal part of visual field (refer for VF).
- This is more likely to be the result of an embolus and the source of the embolus should be sought you should investigate more with doppler ultrasound or TEE looking for vegetation on the valves etc. (if the embolus is a cholesterol embolism it's called hollenhorst plaque).
- Loss of vision may be severe because it causes macular edema.
- If the visual acuity is affected, attempts should be made to dislodge the emboli by ocular massage and decreasing IOP.
    - Another way to dislodge the embolus is by asking the pt to rebreathe into a bag so CO2 causes vasodilation.
    - Basically, for both branch and central occlusion you try to cause vasodilation.
    - Diamox (Acetazolamide) to lower the IOP

- If it was a central occlusion, you'd see a cherry red spot. The dark fovea is a normal finding due to the retinal pigment epithelium to help with 20/20 vision. Only at the fovea you can see 20/20.
- Notice how white (opaque mostly artery) the affected retina (above) is compared to the normal retina (below).
- Retinal vein occlusion can be hemiretinal or central while in arterial it can not be hemiretinal (very very rare)
- The ischemia stopped just short of the macula, so this patient most likely didn't have his vision completely affected **(but his lower visual field is gone)**. **'Cant see footsteps, fall down the stairs'**
- **Upper retina: lower visual field.**
- Lower retina: Upper visual field.
- Nasal: temporal visual field.
- Temporal: nasal visual field.

- Treatment: both **central retinal artery and branch retinal artery occlusion** has **no treatment**
- Visual prognosis depends on degree of associated retinal ischemia.

Extra from AMBOSS

Clinical features of central vs. branch retinal artery occlusion

	CRAO	BRAO
Clinical features	• Sudden, painless loss of vision in **one eye** (often described as a "descending curtain") • A past history of amaurosis fugax may be present.	• Sudden onset of visual field defects (**scotomas**) in the affected eye • A past history of amaurosis fugax may be present.
Relative afferent pupillary defect	• **Present**	• Absent

Ophthalmoscopic findings	• Grayish-white (cloudy) discoloration of the entire retina
	• **Cherry-red spot** at the fovea centralis
	• Retinal plaques/emboli
	  ◦ **Hollenhorst plaque**: cholesterol embolus that presents as a refractive, iridescent lesion (~ 20% of cases)
	  ◦ Whitish-gray platelet thrombi
	  ◦ White calcific plaques
	• Box-carring of all retinal vessels in the acute phase
	• Narrowing of all retinal vessels
		• Grayish-white discoloration of the retinal quadrant supplied by the affected vessel
		• Box-carring of retinal vessels during the acute phase in the affected retinal quadrant
		• Narrow retinal vessels in the affected retinal quadrant
		• Retinal emboli/plaques (~ 60–70% of cases)

General physical examination	• A bruit over the carotid artery is a sign of carotid atherosclerosis. • An irregular pulse may indicate atrial fibrillation. • Scalp tenderness and/or jaw claudication is a sign of temporal arteritis.	

Retinal vessel occlusion

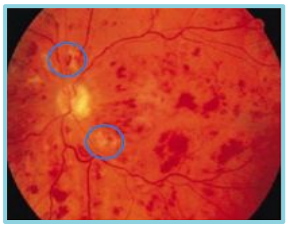
More common than artery occlusion, Hypertensive pt with acute visual loss

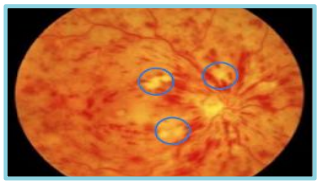
Retinal Vascular Occlusion

Retinal Vein Occlusion CRVO

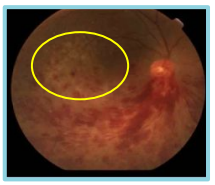
- Ophthalmoscopes picture of disc swelling, venous engorgement, cotton wool spots and diffuse retinal hemorrhages like blood and thunder. **Hypertension is a major risk factor** + can occur in young people by **Antiphospholipid +ve (مش عليك الكلام دا).**
- Loss of vision may be severe in. case of ischemic CRVO .
- **Case will become : Hypertensive patient or use anti-hypertsenive medication.**
  Hallmark of examination (retinal hemorrhage) and in history (hypertensive).
- There is no generally accepted acute management. Central retinal vein occlusion is not a true ophthalmic emergency but it may cause acute visual loss.
- What is the cause of acute visual loss in retinal vein occlusion? **MACULAR EDEMA**
- How can vein occlusion cause acute visual loss?
  - Blood covering the fovea.
  - Macular edema (acute) & exudation (chronic).

  ما يهمني الأنواع والعلاج. يهمني تعرف كيف تفرّق بين الخمس أمراض اللي ذكرناها
- Treatment? Beyond your level control of HTN
  - Intravitreal injection of anti-vascular epithelial growth factor (anti-VEGF).
  - Laser is another option.
- Two types of Central retinal vein occlusion:
  1. Mild: called **non-ischemic** central retinal vein occlusion.
  2. Severe: called **ischemic** central retinal vein occlusion (associated with afferent pupillary defect in addition to poor vision)

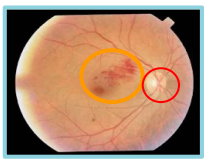




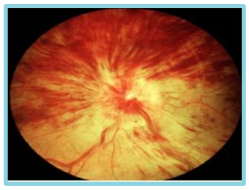
- Flame-shaped hemorrhages all over.
- Visual loss due to macular edema
- Cotton wool spots (blue circles).
- Cotton wool spots are patchy & large unlike exudates which are smaller & well-defined.
- Cotton wool spots are fluffy white focal lesions with indistinct margins.
- **Cotton wool spots are infarctions** in the nerve fiber layer (2nd layer of retina thus the cotton wool spots are on the surface



- Retinal vein occlusion.
- Yellow circle: drusen "age related macular degeneration" not bright as much as hard exudate

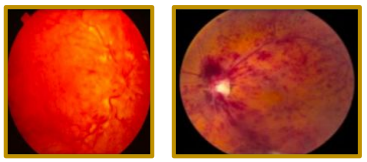


- Macular branch retinal vein occlusion causing a small flame shaped hemorrhage (orange circle).
- Tortuous viens
- No optic nerve swelling cause it affect only branch (red circle)



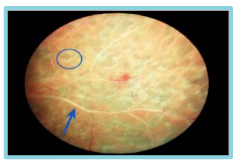
- Hemi-retinal vein occlusion.
- Half of the retina is occluded completely, while the other half is slightly occluded; Still there are tortuous vessels, viscous not well-defined swollen optic disc (non-) flame shaped hemorrhages.

- Retinal vein occlusion (wither central or branched)



- **Central vein occlusion characterized by painless visual loss. On fundoscopy: stormy sunset.**

- Tortuous vessels (arrows) & flamed shaped hemorrhage = central vein occlusion



- Circle: Laser scar (laser marks)
- Arrow: Clear vessel "laser mark" (no blood anymore)

- Hard exudate occurs in chronic edema

Retinal Disease

Young, myopic, male — Retinal Detachment

- An abnormal separation between the sensory retina and the underlying retinal pigmented epithelium (RPE) and choroid plexus. the outer third (the part furthest from the inner vitreous) of the retina gets its nourishment primarily from the underlying choroidal vascular bed. With a detachment, **the photoreceptor layer separates from the choroid, leads to retinal ischemia and retinal degeneration.**
- In a normal retina, there is no actual connection or junction between them. It is a potential space, firm, and adherent.
- When the retina breaks, fluid leaks between the 2 layers and separates them.
- Retinal detachment is one of the painless causes of acute visual loss.
- It will cause sudden or acute visual loss if it was in the macula, but macular involvement takes time, so the pathophysiology is chronic, but the visual loss will be acute.

Case : 13Y.O wearing myopic glass suddenly he got flashes of light + floater then he devople sudden decrease in visual acuity
Physical examination : reff reflux is red
Diagnose ? Retinal detachment

Types — Dr : عبر مطالب فيه الاهم السيناريو اللي فيه

1. Rhegmatogenous RD (rhegma = break) acute, common

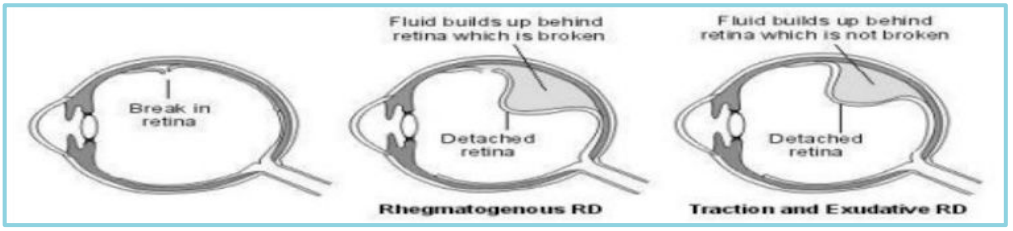
- Most common.
- An acute cause of visual loss that has prodrome (flashes &/or floaters).
- **Tear** in the retina can allow liquefied vitreous to gain entry to the subretinal space causing a progressive detachment.
- Caused by intrinsic cause in the retina.
- Most likely need intervention.
- High **myopia** is risk factor due to axial elongation
- Treated surgically

2. Traction RD chronic

- Slow and gradual loss of vision due to diabetes (Diabetic retinopathy) TB.
- Treatment is surgery (requires intervention).
  - Here the retina is detached but continuous with no tear. If it is pulled off by contracting fibrous tissue on the retinal surface.
- Retinal changes sec. to another cause.
- Blind and painful eye due to blocked trabecular meshwork

3. Exudative RD chronic

- Uveitis
- If we treat the underlying pathology, the problem will be solved. Here it's usually a systematic disease. Eg: renal failure, uncontrolled HTN.
- DON'T touch the eye.
- Chronic & slow.
- Patient complains of gradual loss of vision
- The only one is treated Medically

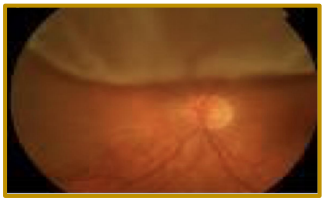


- Picture: when a break happens, subretinal space allowed vitreous fluid to travel into the break and detach the retina.

- If this break was big enough, this retinal vessel will break & cause vitreous hemorrhage; thus, we will not see what is behind the vitreous hemorrhage, so we do B-scan to find if there is a break & retinal detachment.
- What causes this? vitreous changes. In newborns, the vitreous is a gel-like structure & with time the vitreous gel liquefies to water & solid. The solid component is the floaters that pt sees.
- Diabetics & myopic pts are more prone to develop liquefaction of the vitreous.
- When the gel liquifies it contracts & pulls on the retina causing this retinal detachment.
- If the vitreous is still gel-like, pt can have breaks without retinal detachment.

★★★

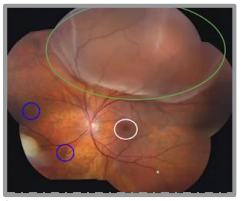
SAQ EXAM:
What is the pic shown?
Retinal detachment



Retinal Disease

439 Slides — Risk Factors

- **High myopia: Retina is thin and stretched at the periphery. More prone to break. The Higher the eye the more the eye stretches and detach.**
- H/O retinal detachment (in the other eye or family hx).
- Trauma. intraocular surgeries are considered risk factor because trauma can occur in surgery
- Aphakia (No lens). Due to Post ocular surgery (any intraocular operation)
  - In the past, they used to deal with cataract aggressively, traumatic surgeries).
  - Because it's a sort of trauma, its abnormal, there should be a lens inside: pseudophakia (artificial lens) → less risk of RD.
- Peripheral retinal degenerations. e.g. lattice degeneration, retinal tufts, etc.
  - **Usually peripheral retina is weaker than the central retina. If a patient has weaker retina w/ degenerations, patient will have a higher chance of developing break in the peripheral retina.**
- **Hx of surgery because you play w/ vitreous.**
- Mechanical
- Keratoconus.
- Posterior Vitreous Detachment (PVD)
  - The vitreous is attached to the eye at the optic head and ora serrata. Due to trauma, surgery, or spontaneous liquefaction secondary to aging, the vitreous detaches and pulls the retina and break it



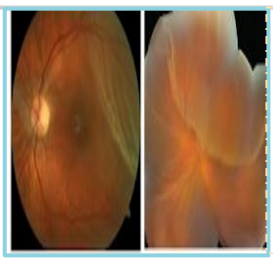
Myopic fundus
Macula
Bolus detachment

Sign and Symptoms (This is applied in rhegmatogenous RD)

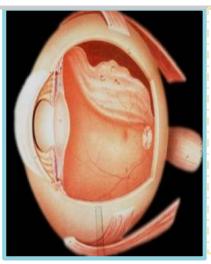
- An extensive retinal detachment involving the macular area would produce acute visual loss and this patient will complain of flashing lights followed by a large number of floaters and then a shade or blind covering the visual field
- Prodromal symptoms: **flashes** bolts of thunder + **floaters** like cobwebs, lines or dots. (Photopsia)
- If the patient came early, we may save the eye by surgery which means there is something we can do; But, if the patient is late, He will present w/ painless visual field loss-curtain-like which is the actual retinal detachment happening.
- Sudden, painless loss of vision; The course may be so short that no matter what you do you're late.
- Afferent pupillary defect is usually present
- The diagnosis is confirmed by ophthalmoscopy through a dilated pupil, and retina appears elevated with folds and the choroid background behind the retina is indistinct.

439 Slides — Management

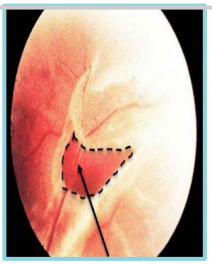
- **RD is an urgent condition.**
- **Needs emergency surgery.**
  - If a pt comes early enough w/ only breaks & NO fluid pass by (didn't develop detachment) →you can surround it by laser. - But if w/ detachment → do surgery.
- Scleral buckle, cryotherapy, SRF (subretinal fluid) drainage.
- Vitrectomy most common, AFX (air fluid exchange), endolaser, long acting tamponade (gas, silicone oil).
- In the management, there are two types based on the status of the macula, macula on and off.
- Macula on: the macula is still attached, and the intervention is required within 24 hours (Because central visual acuity is still preserved). "better prognosis"
- Macula off: The macula is detached, and intervention is less critical (within 10 days) "worse prognosis"



Traction
There is a flat retina and the bulging part is detached (if the macula is not involved you can treat this surgically)



Breaks without detachment (If it stretches more > Detachment) Vitreous fluid collects causing detachment. the break separate retina from retinal pigment epithelium



Break (dash line) - Retinal vessel bridging the break
Horseshoe break: if the break gets pulled more (by the vitreous) → can rupture a vessel → RD & vitreous hemorrhage.

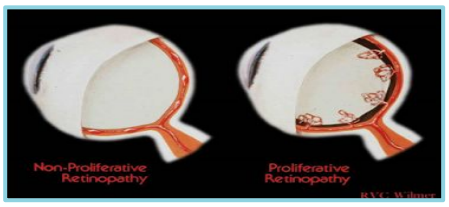
Retinal Disease

439 Slides — Retinal Detachment cont;

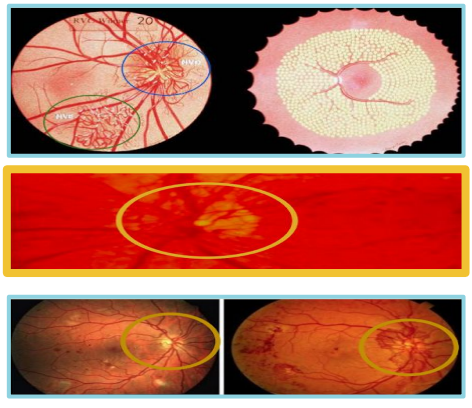
- The potential space between the neuroretina and its pigment epithelium corresponds to the cavity of the embryonic optic vesicle. The two tissues are loosely attached in the mature eye and may become separated:
  - If a tear occurs in the retina, allowing liquefied vitreous to gain entry to the subretinal space. This causes a progressive, rhegmatogenous, retinal detachment which may be partial or total.
  - If it is pulled off by contracting fibrous tissue on the retinal surface, e.g. in the proliferative retinopathy of diabetes mellitus (traction retinal detachment).
  - When, rarely, fluid accumulates in the subretinal space as a result of an exudative process, which may occur with retinal tumours or during toxaemia of pregnancy (exudative retinal detachment).

439 Slides — Diabetic retinopathy

- Diabetic retinopathy is the term used to describe the retinal damage causing visual loss. Diabetics have a high prevalence of retinopathy.
- Diabetic retinopathy causes neovascularizations when ruptured causing vitreous hemorrhages resulting in acute visual loss.
- They start off as **non-proliferative** and if the DM isn't taken care of then It progresses to **proliferative**.
- The cause of Acute visual loss in diabetic retinopathy is **vitreous hemorrhage** (Not detachment)

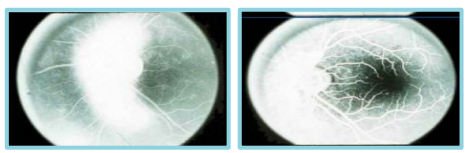


- Non-proliferative:
  - Vascular changes on the retina.
  - Exudates & microaneurysms (dot & plot hemorrhages).
- Proliferative:
  - Neovascularization happens in the choroid but here they pop up from the retina into the vitreous cavity.
  - The vessels undergo fibrosis and contract causing traction or tractional + rheugma RD (thus patient may present w/ floaters)

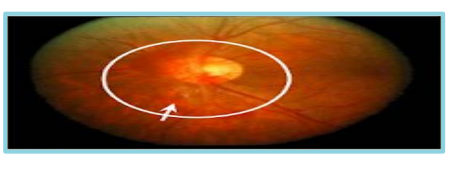


Proliferative retinopathy:

- If it's on the disc > NVD "NeoVascularization on Disc"
- If it's outside the disc > NVE "NeoVascularization Elsewhere"
- Gold circle: Neovascularization at the disc & is treated by photocoagulation.
- We treat it with laser (photocoagulation) the periphery to reduce the oxygen demand and the oxygen will be enough for the rest (macula)



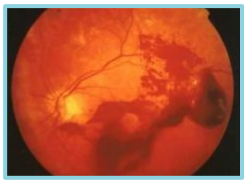
- The neovascularizations always leake, normal vessels don't leake.
- An ischemic retina releases vasogenic factors (e.g. VEGF) which result in the growth of abnormal blood vessels and fibrous tissue onto the retinal surface and forwards into the vitreous. These intravitreal vessels are much more permeable than normal retinal vessels, so that they leak dye during retinal fluorescein



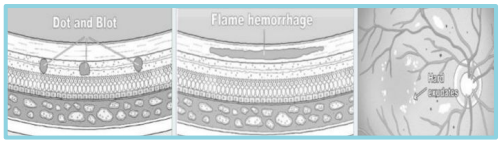
- Circle: Neovascularizations
- White arrows : the white tissue is Neovascularizations that is starting to fibrous

Retinal Disease

❖ Diabetic Retinopathy; cont

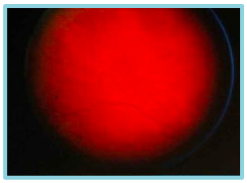
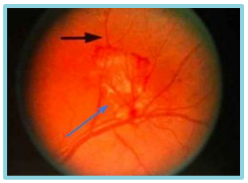



Extra

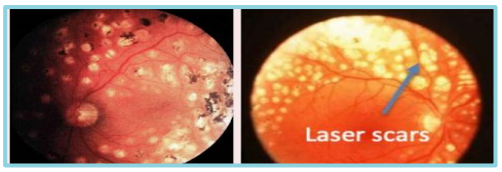


- Non-Proliferative retinopathy
  - Orange Arrow: Exudate
  - Green Arrow: Microaneurysm
- NPDR features:
  - Injured capillaries can leak fluid into the retina and the aneurysms themselves can burst, forming "dot-and-blot hemorrhages."
  - Dot-blot hemorrhages look small and round because they occur in the deep, longitudinally-oriented cell layers of the retina. This contrasts with the "flame hemorrhages" of hypertension that occur within the superficial ganglion nerve layer, and thus spread horizontally.
  - As vessel damage progresses, you can also see beading of the larger retinal veins and other vascular anomaly
  - If the hard exudate accumulate and reach the macula it will lead to visual loss due to macular edema

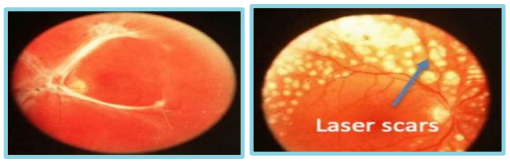
- The neovascularization bulging into the vitreous; they should stay below the ILM. But, in ischemic pts like diabetics the vessels grow into the vitreous cavity > become fibrosed >contract> detach.
- Before undergoing fibrosis, the vessels can bleed causing vitreous hemorrhage which is the cause of acute visual loss

- NVE,
- White (blue arrow) is the fibrous tissue that forms causing the traction (tractional RD).

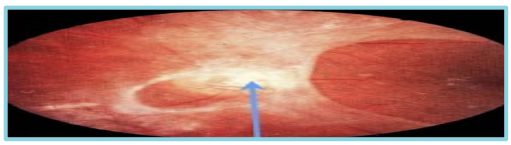


- Laser scars following diabetic retinopathy treatment
- The left represent bad laser while the picture in the right show good procedure (do not do like the one in the left :))

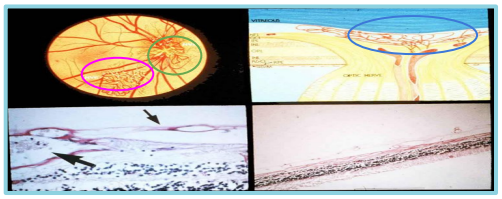


- Tractional retinal detachment on the left (complication if patient didn't do laser)
- The picture on the right represent the retina after treatment with laser

- Right picture: Retinal detachment in histology



- Blue Arrow: Fibrous tissue
- Treatment: panretinal photocoagulation



- Green circle: newvascularizaion of the disc (NVD)
- Pink scircle: newvascularizaion elsewhere (NVE)
- Blue circle: neovascularization in the posterior surface of the vitreous and if the vitreous contract it will lead to hemorrhage.

Retinal Disease

❖ Diabetic Retinopathy; cont

- Fibrosis and contraction of vessels occurs in tractional retinal detachment, the retina is not in its place so it can't function properly.
- Treatment: laser
  - We laser everything except the macula
  - The retina goes back in place after laser, but it doesn't mean they'll have good vision. Because the traction is chronic & pts already have photoreceptor loss & other retinal structural abnormality.
  - The goal of the surgery is that patients don't worsen NOT to regain 20/20. It's a measure we take along with tight control of blood sugar so patients don't go blind.
- The retina is a part of the brain with neurons, so it is not just an ischemic problem.
- Neovascularization + vitreous hemorrhage > go away immediately with laser

❖ Very helpful video !

???

Optic Nerve Disease

Optic neuritis Pain with eye movement

- Optic neuritis is an inflammation of the optic nerve and is usually idiopathic, but it may be associated with **multiple sclerosis** (as first clinical manifestation) in a significant number of cases.
- The visual acuity is markedly reduced, and an **afferent pupillary defect is present** (IMP IN OSCE).
- Associated with **pain** on extraocular muscle movement in 90% of patients.
- The optic disc initially appears hyperemic and swollen.
- The visual acuity usually recovers. However, repeated episodes of optic neuritis may lead to permanent loss of vision > That's why we treat. (so, the goal of management is to prevent recurrence, you have to treat with steroids IV followed by oral and interferon).

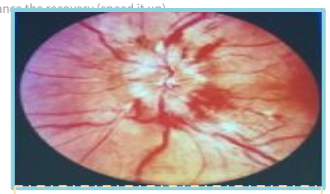
- **Case will talk about multiple sclerosis then developed acute vision loss**
- **Example : 35 Y.O she has hyperthermia when she take shower she has tingling, etc…**

HALLMARK : afferent pupillary defect - Not complete vision loss (from 6/6 to 6/60)
SAQ: what important examination in optic neuritis? afferent pupillary defect (INDIRECT QUESTION)
Disc : hyperemic or normal
Investigation : MRI for brain
Tx: IV steroid 5 days followed by oral steroid
9:12 A.M doctor cough and use microphone (may had URTI)

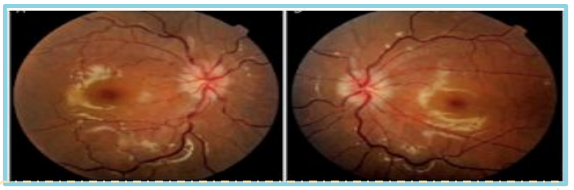
It has three types:
- Optic papillitis (Optic nerve head is involved)
- Retrobulbar neuritis (the posterior part of the nerve is involved)
- Neuroretinitis (Optic nerve head with contagious retinal inflammation).

- Most common type is retrobulbar neuritis. Here, the fundus looks normal, but the vision is severely affected with central visual fields defect (most common presentation).
- Most of the time It is reversible with return of normal vision within 4-6 weeks (self-limiting).
- But if one eye only is affected you may use steroids to enhance the recovery (speed it up).

"swollen optic nerve"
Swollen hyperemic disk



Swelling of the optic nerve head



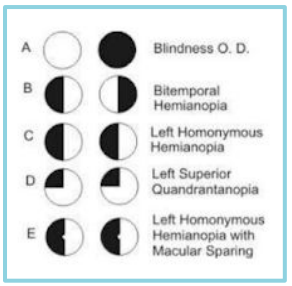
Bilateral Optic nerve swelling. it's NOT papilledema; papilledema is Bilateral swelling only due to increased ICP

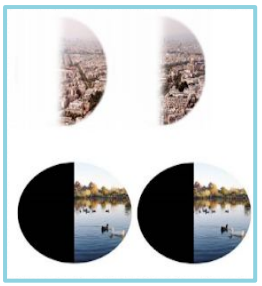
Extra	Papilledema	Papillitis	Retrobulbar neuritis
Definition	Swelling of optic nerve head due to increased ICP	Inflammation or infarction of optic nerve head	Inflammation of orbital portion of optic nerve
Uni/bilateral	Bilateral	Unilateral	Unilateral
Vision impairment	Enlarged blind spot	Central/paracentral scotoma to complete blindness	Central/paracentral scotoma to complete blindness
Fundus appearance	Hyperemic disk	Hyperemic disk	Normal
Vessel appearance	Engorged, tortuous veins	Engorged vessels	Normal
Hemorrhages?	Around disk, not periphery	Hemorrhages near or on optic head	Normal
Pupillary light reflex	Not affected	Depressed	Depressed
Treatment	Normalize ICP	Corticosteroids if cause known	Corticosteroids with caution

Visual Pathway Disorders

Homonymous Hemianopia

- Loss of vision on one side of both visual fields.
- May result from occlusion of one of the posterior cerebral arteries with infarction of the occipital lobe.
- Other vascular abnormalities occurring in the middle cerebral artery distribution may produce a hemianopia, but usually other neurological signs are prominent (like in stroke).
- Any patient with hemianopia needs a CT or MRI to localize & identify the cause. +Treat if treatable
- **Vascular stroke can cause acute vision loss but the problem here the visual acuity 6/6 so you will not detected!**
  Even he complain from vision loss!
  What should you do? Visual Field
  Will take it in neuro-ophthalmology lec.
- Refer to neurology.
- Behind the optic chiasm.





- If one have afferent pupil defect **the lesion become between optic nerve and LGB**
  If it is behind LGB ? he will never have APD!
  So if patient have sudden loss of vision and pupil reactive nicely? The lesion behind LGB

Cortical Blindness

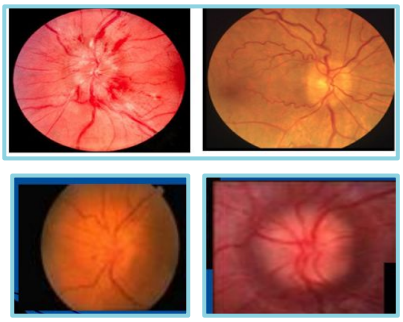
- A rare extensive bilateral damage to the cerebral visual pathways resulting in a complete loss of vision.
- This condition is referred to as cortical, central or cerebral blindness.
- As the pathways serving the pupillary light reflex (midbrain) separate from those carrying visual information at the level of the optic tracts (cortex), a patient who is cortically blind has normal pupillary reactions. Remember that the Lateral Geniculate Body is in the brainstem at the midbrain and the optic nerve won't be atrophied because the problem is BEHIND the optic chiasm.
- Thus, a patient with a normal fundus examination along with normal pupillary reactions, most likely has cortical blindness (if the patient has complete loss of vision).
- Poor vision, vision loss depends on which part of the cortex was affected.
- Role out bY neuroimaging and neuro ophthalmology

Functional visual loss

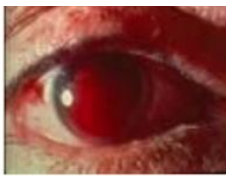
- A functional disorder is used in preference to hysterical no clinical issue but the patient believes that he/she cannot see or malingering seeking attention or subvention يمارض to describes visual loss without organic basis (there is no actual visual loss).
- A patient may report complete blindness in one eye and normal vision in the other eye, and no relative afferent pupillary defect (RAPD).
- The patient has no real visual loss but psychological/hysterical problem (eg; munchausen syndrome by proxy), simple way to check: cover good eye and hide mirror behind your back then quickly show it. Patient will usually look

❖ Picture at the end of the lecture

Optic nerve head swelling



Hyphema

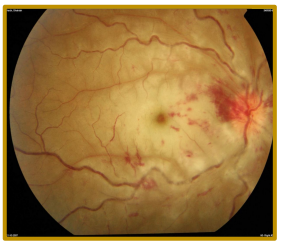


Total hyphema
"8 pool hyphema"
IMP

Edema or myelinated nerve fibers

Edema

combined artery
vein occlusion

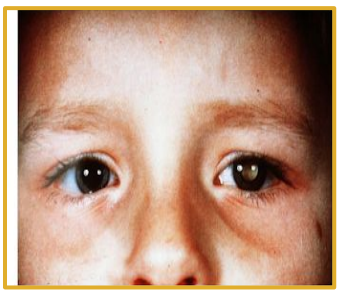


Qs IN EXAM

What is it called?
Leukocoria (in children)

What causes it?

- Retinoblastoma
- Cataract
- Coat's disease



Coats disease: abnormal dilated blood vessel "retinal telangiectasia" leak under the retina and lead to detachment

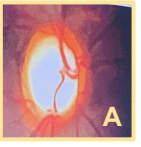
- How can we differentiate between acute and chronic visual loss? By the timing (sudden vs gradual).
- **What is the most common cause for CHRONIC VISUAL LOSS**: 1) cataract 2) chronic open angle glaucoma 3) diabetes retinopathy 4) age-related macular degeneration "AMD" 5) Retinitis pigmentosa.
- **What is the most common cause for ACUTE VISUAL LOSS**: 1) acute closed angle glaucoma 2) central retinal artery occlusion 3) central retinal vein occlusion 4) retinal detachment 5) optic neuritis.
- In Visual pathway disorders the visual acuity might be normal, so you should consider visual field evaluation for all patients.
- **A 35 yrs old pt came with acute visual loss could it be caused by acute glaucoma?** NO, because acute glaucoma usually occurs after 40s. That's why the age is important. If elderly with 75 yrs old think about age related macular degeneration (chronic) or vascular cause (acute). If a cardiac pt with vegetations think about vascular occlusion, if hypertensive pt think about central retinal vein occlusion. If young female pt presented with tingling, numbness and headache and while showering she had loss of vision think about optic neuritis caused by multiple sclerosis. (**MCQ**)
- **Why pupillary reaction is important?** Normal pupil reaction should be round, regular and constricted but if you do the examination and it was dilated instead of constricted then it indicates a pathology such as acute glaucoma (mild fixed dilated/non reactive pupil), also it help figure other abnormalities such as afferent pupillary defect in optic neuritis.
- **What is the definition of glaucoma?** Optic nerve is the target organ here so we can define it as 'Damage of the optic nerve (optic neuropathy) manifested by visual field changes', IOP is a risk factor.
- **Scenario of Closed Glaucoma (SAQ):** A pt above 40s who has pain at night while watching TV and hazy vision. **What is the pathology?** Pupillary block. **How would you manage?** Acetazolamide + peripheral iridotomy. **What are the side effects of acetazolamide?** Bone marrow depression/gastric ulcer/hypokalemia/renal stones..etc. **what would you do to the other eye?** Prophylactic peripheral laser iridotomy because he can get in the other eye.
- **Scenario of Retinal Detachment (SAQ)**: young myopic pt with history of flashing of lights/curtains light or visual loss. Treatment is surgery depending on the type macula off or on (just write surgery)
- **Scenario of Retinal Artery Occlusion (SAQ)**: 65 yrs old with history of endocarditis/thrombosis? Or heart disease presented with sudden loss of vision. Or RTA pt with multiple bone fractures presented with sudden loss of vision (fat embolism). (Cherry red spot)
- **Scenario of Retinal Vein Occlusion (SAQ)**: pt with history of hypertension. It's very important to know the pictures because it'll come in the exam (flame-shaped diffuse retinal hemorrhage, disc swelling, venous engorgement, cotton wool spots). you should control hypertension, details about the actual treatment (laser/anti-VEGF) not required from you.
- **Scenario of Optic Neuritis (SAQ)**: middle aged female (35 yrs old) with multiple sclerosis, or systemic lines, she came for mild pain loss of vision, **what is the hallmark on examination?** Afferent pupillary defect is present. **What is the treatment?** IV steroids.
- **Cortical blindness vs optic neuritis:** Pupillary reaction is negative/no RAPD/ in cortical blindness (the lesion is behind LGN).
- **Scenario of Diabetic retinopathy (SAQ):** diabetic pt with diabetic retinopathy presented suddenly to ER with acute visual loss, IOP is 45, with examination there's new vessels in the iris. **What's the diagnosis?** Neovascular glaucoma. **What's the treatment?** PRP

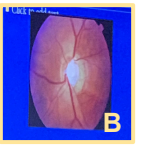
Doctor showed pics at the end of the lectures and made some points:

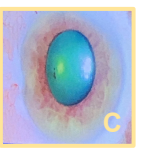
(A): optic disc cupping, normally it's 3% only but here it's around 8% which might be physiological or pathological glaucoma. **we should do visual field to differentiate between normal and abnormal (SAQ)**

(B): typical glaucoma optic disc: cupping, thinning, atrophy.

(C): neovascular glaucoma: when do you have new vessels in the retina? When there's retinal ischemia caused by diabetic retinopathy. This new vessels can be formed in the angle which cause leaking and fibrous tissue that close the angle and cause secondary closed angle glaucoma but here iridotomy is **CONTRAINDICATED** due to Bleeding, you need to treat the cause: control retinal ischemia by PRP (pan-retinal-photocoagulation) to stop new vessels from forming > then treat by trabeculectomy not iridotomy.







*Doctor showed some gonioscopy examples pics about open/closed angle.

CASES FROM DR 441

Case 1:
45 Y.O female who present with acute vision loss on examination the eye is very quiet (no redness no edema) and optic nerve show **hyperemic disc**
What possible diagnose ?
Answer: Typical Optic neuritis.

Case 2:
Diabetic patient present to you in clinic with sudden loss of vision
Examination: IOP = normal and eye quiet and no abnormality and NO APD
What possible diagnose ?
Answer: vitreous hemorrhage
EXPLANATION:
IOP normal = not glaucoma, no APD = not Optic neuritis. No flash and no glass= not retinal detachment

Case 3:
Diabetic patient (take insulin for 20 years) present to emergency with sudden severe pain in the eye
Examination: diabetic retinopathy and IOP =35
What possible diagnose ?
Answer:neovascular glaucoma

Case 4:
35 Y.O cardiac patient known case of cardiac stent and past history of infective endocarditis. He develop sudden visual loss
Examaninatio : retinal pale
What is the possible finding i retuna ?
cherry red spot

Case 4:
Hypertiensive patient known case of hyppertion who develop sudden vision loss
Examination : quiet eyes - IOP normal - on AOD
What is the possible finding i retuna ?
Retinal hemorrhage (CRVO)

Lecture Quiz

Q1- A 35 year old female presented with sudden decrease in visual acuity, eye pain on movement and the symptoms worsened with hot shower, examination revealed afferent pupillary defect, what is the cause of sudden decrease in vision?

A. Acute glaucoma
B. Vitreous hemorrhage
C. Vascular occlusion
D. Optic neuritis

Q2- Acetazolamide is used to lower IOP in acute closed angle glaucoma, which of the following is true regarding the side effects of the drug?

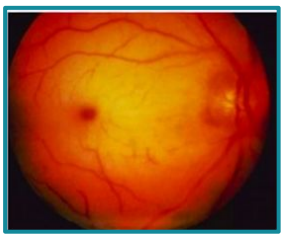
A. Hyperkalemia
B. Metabolic alkalosis
C. Hypokalemia
D. Hypocalcemia

Q3- Risk factor for acute Closed angle glaucoma?

A. Myopia
B. Hyperopia
C. Presbyopia
D. Astigmatism

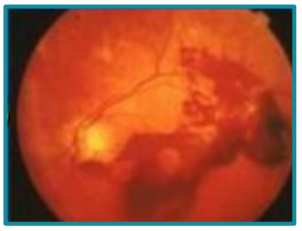
Q4- 65 year old male underwent cardiac valve transplant came with sudden painless loss of vision, what is the hallmark in the examination?

A. Cherry red spots
B. Retinal hemorrhage
C. Disc swelling
D. Cotton Wool spots



Q5- 55 year old diabetic patient presented with sudden visual loss, eye is quite IOP is normal with no afferent pupillary defect what is the finding in picture below?

A. Neovascular glaucoma
B. Acute glaucoma
C. Vitreous hemorrhage
D. Optic neuritis



Answers: Q1: D | Q2: C | Q3: B | Q4: A | Q5: C

Short Answer Questions

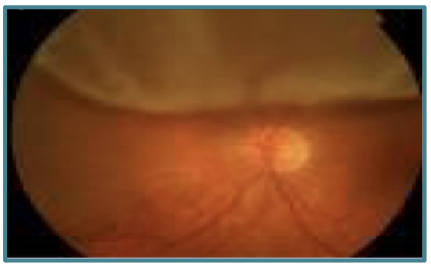
Case 1

A: Mention two changes that will cause acute glaucoma secondary to chronic cataract?

B: What is the pathology behind acute closed angle glaucoma?

Case 2

30 year old male wearing myopic glasses suddenly experienced flashes and floaters examination of the retina shown:

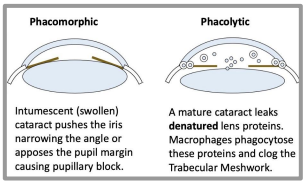


A: What is the diagnosis?

B: Mention two risk factor that can lead to this condition

Answers:

Case 1
A: Phacomorphic, phacolytic
B: Pupillary block

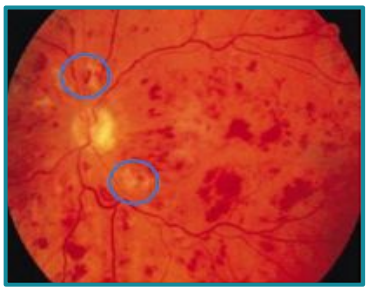


Case 2
A: Retinal detachment
B: Myobia, trauma

Short Answer Questions

Case 3

65 year old male diagnosed with hypertension 30 years ago, retinal examination shown:

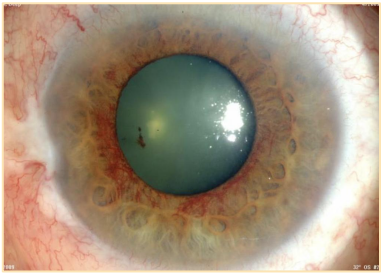


A: What is the diagnosis?

B: Mention 2 abnormal findings in the retina?

Case 4

55 year old with long standing DM complicated by diabetic retinopathy, presented with sudden decrease in visual acuity and corneal edema, IOP is 50 mmhg, examination of the eye shown:



A: What is the diagnosis?

B: What is the acute management for this patient ?

Answers:

Case 1
A: Central retinal vein occlusion
B: intraretinal hemorrhage, swollen disc, cotton wool spots

Case 2
A: Neovascular glaucoma (if the question asked about finding write Rubeosis iridis)
B: **Atropine, Diamox(acetozolamide), Anti-VEGF**

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