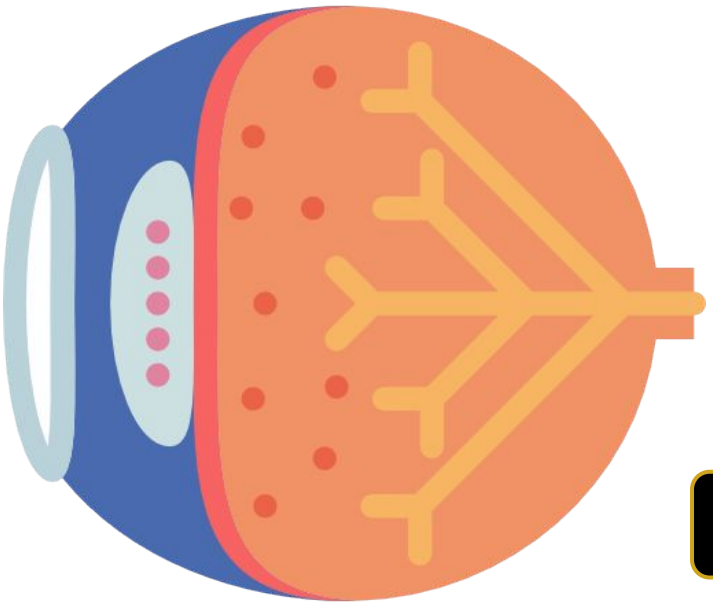
Lecture: 10



Editing file

Acute Visual Loss

- **Presented By: Dr. Marwan Abouammoh/ Dr. Essam Osmn**
- 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.

Important | Doctor's notes | Golden notes | Extra | Book

OPTHALMOLOGY TEAM

Acute Visual Loss

❖ **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.
  - ■ 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

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 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

Clinical Approach

History

- What is the patient's age and general medical Condition?
  - 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: (before optic chiasm decussation) such as optic neuritis.
  - Binocular (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?

Physical Examination

- Visual acuity testing : after vital signs
  - To see if the visual loss is mild, moderate, or severe.
- Confrontation visual fields test:
  - 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)

Acute Visual Loss

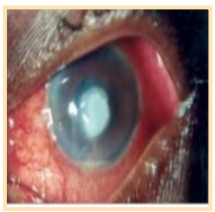
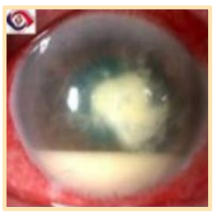
Causes Of Acute Visual Loss

- 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

Media Opacities

Corneal Ulcer

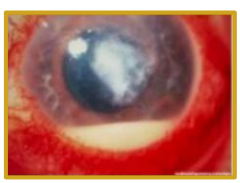
- When there is a corneal opacity due to destruction of tissue by infiltration of microorganisms and WBCs.
- It could be viral, bacterial, fungal, neurotrophic protozoal or in etiology.

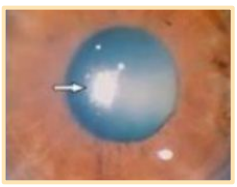
Corneal Edema

- 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 أو مثلج) rather than its normal clear transparent appearance (steamy cornea).
- The most common cause of corneal edema is increased intraocular pressure & occurs typically in acute angle closure glaucoma.
- 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 severe ocular hypotony 1
- The other cause of corneal edema is infection or a cause from edema
  - Any acute infection of the cornea by a corneal ulcer may mimic corneal edema.

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



Abscess



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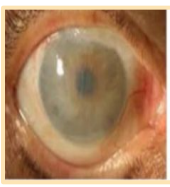
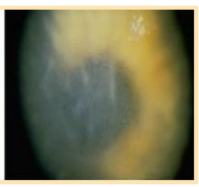



Acute Glaucoma

Classifications of Glaucoma

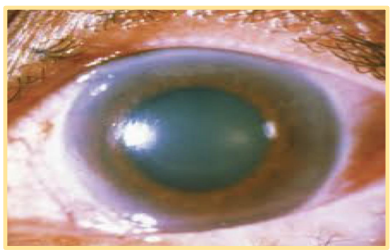
- According to Aetiology
  - Primary
  - Secondary
  - Congenital
- According to Appearance of angle
  - Open angle
  - Closed angle
  - Combined mechanism (mixed)

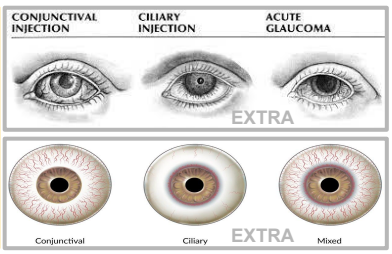
❖ Glaucoma:

- 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. While in secondary it's most likely unilateral.
- To differentiate between open and closed angle glaucoma we use **GONIOSCOPY.**
- **Risk factors:** short AL (axial length) , hyperopia, above 40Y.
- **Signs & symptoms:** Painful acute visual loss And collection of these:
  - Increased intraocular pressure.
  - Media opacity (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 (sphincter papillae & dilator papillae) resulting in ischemia.
  - Acute decreased vision.
  - Severe pain (sometimes people go the ER complaining of headache; they do CT/MRI & they find nothing while pt is screaming) around 27 mins.
  - Redness

Acute Glaucoma Clinical Picture:

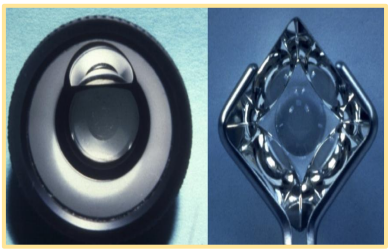
-Painful decrease VA (visual acuity)
-Ciliary injection
-Corneal edema
-Pupil semi dilated
-Disc hyperemic

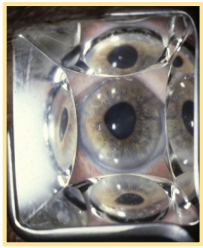


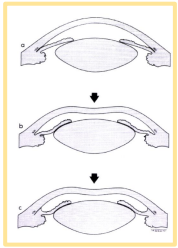


Gonioscopy Lens (SAQ)

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

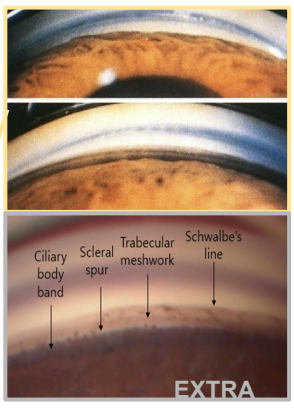


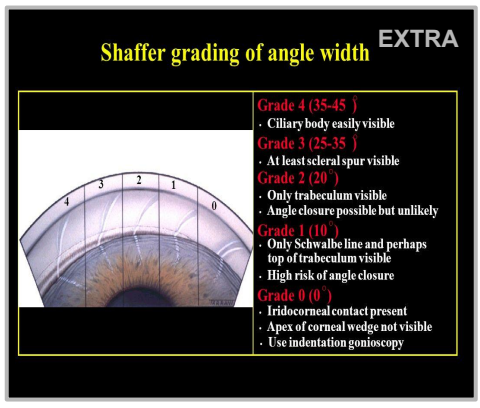




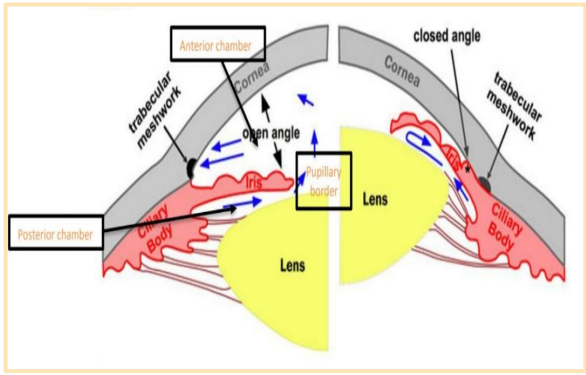
Details about the structures is not required from you. When you can see the structures completely it's called open angle when you can't see it then it's closed angle (iris attached to the lens = closed angle).

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

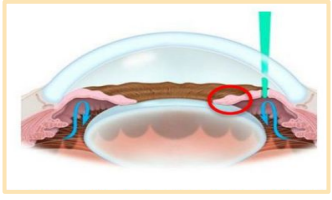


- Normally, the aqueous humor which is produced by the ciliary body travels from the posterior chamber through the pupillary border into the anterior chamber then it gets drained in the trabecular meshwork.
- What happens in acute angle closure glaucoma is that patients have a narrow angel to start with; At a certain point, may be with aging which involves the lens getting bigger the angel will become more narrow until it totally occluded & no fluid can be drained through the trabecular meshwork; adding over this, the iris may bow anteriorly because of the pressure effect & touch the lens which will make the angel more occluded.
- The angel is narrow & the trabecular meshwork is occluded by the iris touching (if pt presents early) the periphery of the cornea & the pupillary edge of the iris bows against the lens closing the posterior chamber & pressure is building up.

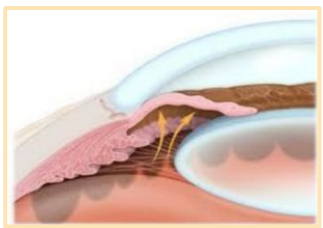
- Closed angle glaucoma is divided into four stages: 1- prodromal attack 2- intermittent attack 3- acute attack 4- chronic closed angle attack (**majority of pts present in this stage**)
- The pathology behind acute glaucoma is **PUPILLARY BLOCK!** The pupil at semi-dilated vision get stuck/blocked with the lens.

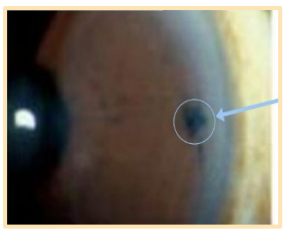
- **Aims of acute ACG management:**
  - Decrease IOP by:
    - systemic **Acetazolamide** (side effects of the drug (**SAQ**): bone marrow depression, renal stones, gastric pain/ulcer, hypokalemia, metabolic acidosis, depression)
    - Topical B Blocker.
    - Pilo 2 (miotics drug)
    - **Peripheral 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.

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*





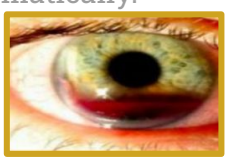
Peripheral laser iridotomy

Media Opacities

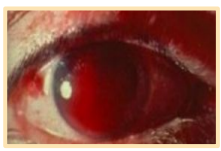
Hyphema

- Hyphema is blood in the anterior chamber.
- The hyphema is a direct consequence of blunt trauma to a normal eye (some people are more prone to bleed).
- If the eye is opened during trauma → emergency the 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. (Tumor and DM)
- Sickle cell patients are more 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".
- 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
- If it's not resolved and the pressure is high it may cause corneal blood staining, which would take years to clear. This will affect the vision dramatically.

We Measure the height of blood in mm

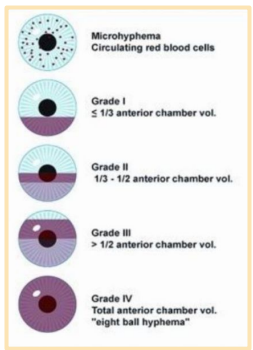


Hyphema Complications: posterior synechiae & glaucoma.



Eight ball hyphema Filling of the whole anterior. chamber

Extra



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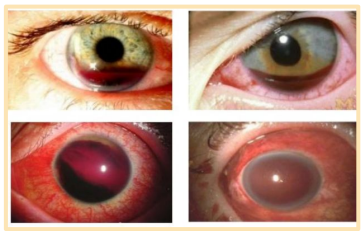
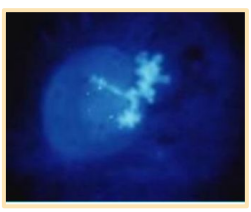


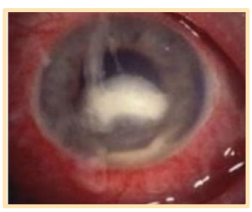
Uveitis

- 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



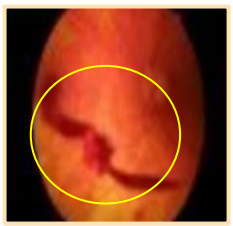
Infection: abscess in the cornea with hypopyon & congested eye.



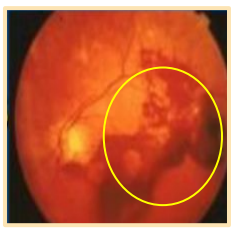
Media Opacities

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 (visual axes) 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.** If slit lamp couldn't confirm it then go for B scan.
  - Suspect ⟶ ophthalmoscope Confirm ⟶ slit lamp
  - **B scan (bi-model ultrasound) is important** to know the etiology. 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



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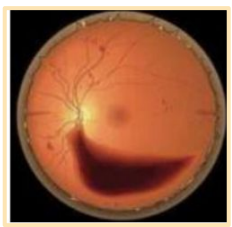

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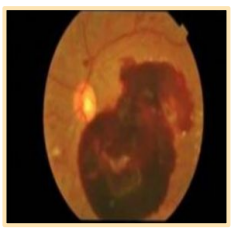
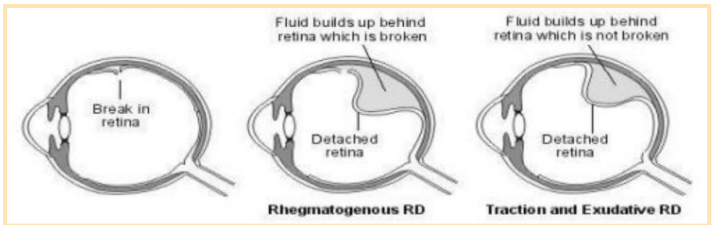


Retinal Detachment

- 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, and without this blood supply becomes ischemic.
- 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.

Types

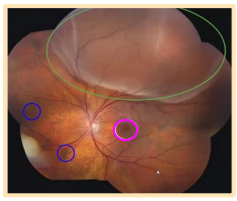
1. Rhegmatogenous RD (rhegma = break) acute	2. Traction RD chronic	3. Exudative RD chronic
• Most common. • An acute cause of visual loss that has prodrome (flashes &/or floaters). • Tear in the retina can allow liquefied vitreous to gain entry to the subretinal space causing a progressive detachment. • Caused by intrinsic cause in the retina. • Most likely need intervention.	• Slow and gradual loss of vision due to diabetes or 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	• 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
		• 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.

Retinal Disease

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 consider 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

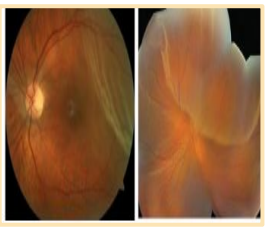
Sign and Symptoms

(This is applied in rhegmatogenous RD).

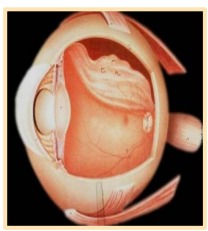
- Prodromal symptoms: flashes bolts of thunder + floaters like cobwebs, lines or dots.
- If the patient came early, we may save the eye by surgery which means there is something we can do; But, if the patient is late, He will present w/ painless visual field loss-curtain-like which is the actual retinal 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.
- 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.

Management

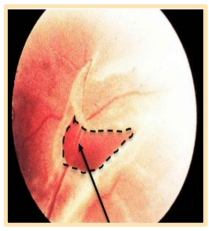
- RD is an urgent condition.
- Needs emergency surgery.
  - If a pt comes early enough w/ only breaks & NO fluid pass by (didn't develop detachment) →you can surround it by laser. - But if w/ detachment → do surgery.
- Scleral buckle, cryotherapy, SRF (subretinal fluid) drainage.
- Vitrectomy most common, 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



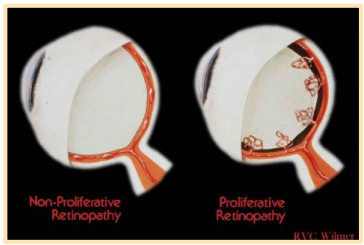
Break (dash line) - Retinal vessel bridging the break
Horseshoe break: if the break gets pulled more (by the vitreous) → can rupture a vessel → RD & vitreous hemorrhage.

Retinal Detachment cont;

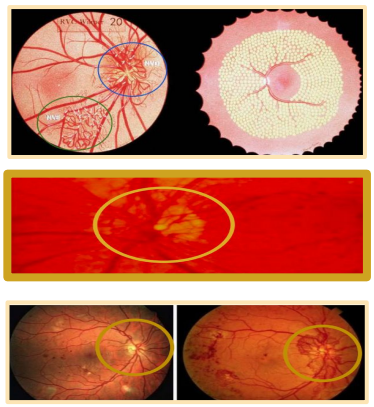
- The potential space between the neuroretina and its pigment epithelium corresponds to the cavity of the embryonic optic vesicle. The two tissues are loosely attached in the mature eye and may become separated:
  - If a tear occurs in the retina, allowing liquefied vitreous to gain entry to the subretinal 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).

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** (Non detachment)

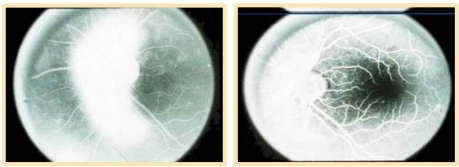


- Non-proliferative:
  - Vascular changes on the retina.
  - Exudates & microaneurysms (dot & plot hemorrhages).
- Proliferative:
  - Neovascularization happens in the choroid but here they pop up from the retina into the vitreous cavity.
  - The vessels undergo fibrosis and contract causing traction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

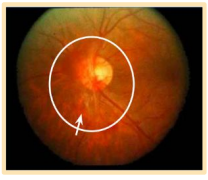


Proliferative retinopathy:

- 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)



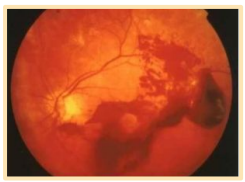
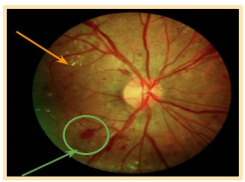
- The neovascularizations always leake, normal vessels don't leake.
- An ischemic retina releases vasogenic factors (e.g. VEGF) which result in the growth of abnormal blood vessels and fibrous tissue onto the retinal surface and forwards into the vitreous. These intravitreal vessels are much more permeable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



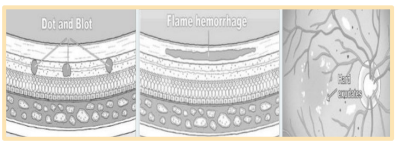
- Circle: Neovascularizations
- White arrows : the white tissue is Neovascularizations that is starting to fibrous

Retinal Disease

❖ Diabetic Retinopathy; cont

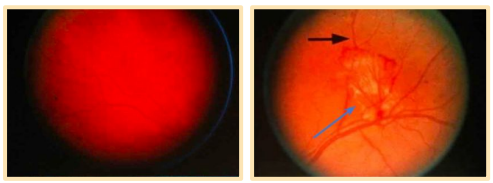



Extra

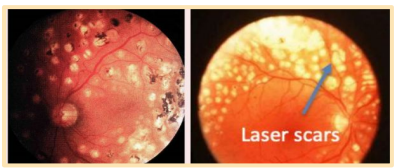


- Non-Proliferative retinopathy
  - Orange Arrow: Exudate
  - Green Arrow: Microaneurysm
- NPDR features:
  - Injured capillaries can leak fluid into the retina and the aneurysms themselves can burst, forming "dot-and-blot hemorrhages."
  - Dot-blot hemorrhages look small and round because they occur 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

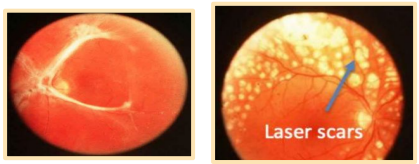
- The neovascularization bulging into the vitreous; they should stay below the ILM. But, in ischemic pts like diabetics the vessels grow into the vitreous cavity > become fibrosed >contract> detach.
- Before undergoing fibrosis, the vessels can bleed causing vitreous hemorrhage which is the cause of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



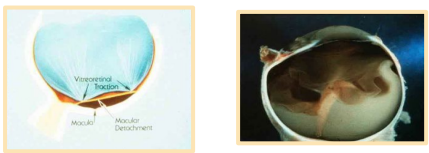
- NVE,
- White (blue arrow) is the fibrous tissue that forms causing the traction (tractional RD).



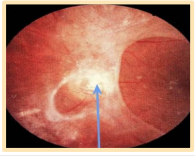
- Laser scars following diabetic retinopathy treatment
- The left represent bad laser while the picture in the right show good procedure (do not do like the one in the left :))



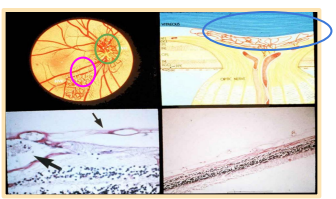
- Tractional retinal detachment on the left (complication if patient didn't do laser)
- The picture on the right represent the retina after treatment with laser



- 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

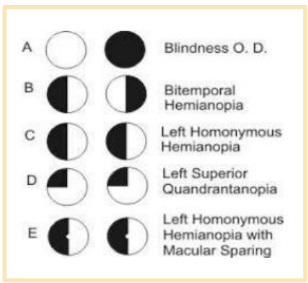
❖ Diabetic Retinopathy; cont

- 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

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
- Refer to neurology.
- Behind the optic chiasm.



Cortical Blindness

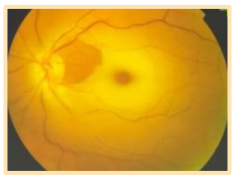
- A rare extensive bilateral damage to the cerebral visual pathways resulting in a complete loss of vision.
- This condition is referred to as cortical, central or cerebral blindness.
- As the pathways serving the pupillary light reflex (midbrain) separate from those carrying visual information at the 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 parinet has complete loss of vision).
- Poor vision, vision loss depends on which part of the cortex was affected.

Retinal Vascular Occlusion

Central Retinal Artery occlusion

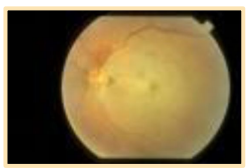
- A sudden, painless and often complete visual loss (no light perception) may indicate central retinal artery occlusion (CARO).
- Several hours after a central retinal artery occlusion, the inner layer of the retina becomes opalescent (opaque, why? because of ischemia).
- A cherry red spot is seen due 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).
- A chronic cherry red spot is also a feature of the storage diseases such as Tay-Sachs pick disease and Niemann-Pick disease.
- 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.

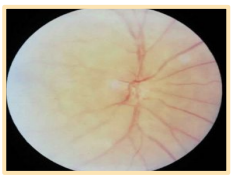
(There is an opaque retina with an edematous disc (small arrow) and the big arrow is pointing at the macula, the dark area is a cherry red spot of the fovea in the center of the macula. Which means: central retinal artery occlusion).



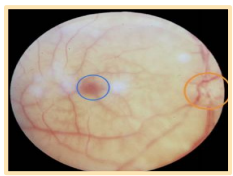
Extra

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.

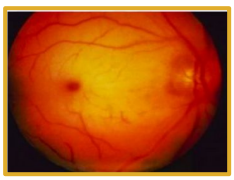




Opaque "pale" retina & attenuated artery > central retinal artery occlusion.



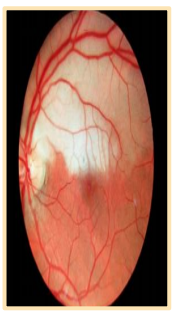
Cherry red spot (blue circle). Disc (orange circle). Opaque retina.



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

Branch Retinal Artery Occlusion

- 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 embolus 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 embolus 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.

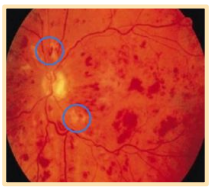


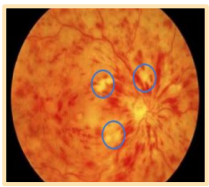
- If it was a central occlusion, you'd see a cherry red spot. The dark fovea is a normal finding due to the retinal pigment epithelium to help with 20/20 vision. Only at the fovea you can see 20/20.
- Notice how white(opaque mostly artery) the affected retina (above) is compared to the normal retina (below).
- Retinal vein occlusion can be hemiretinal or central while in arterial occlusion 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.**
  - Nasal: temporal visual field.
- Lower retina: Upper 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.

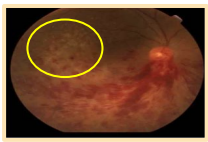
Retinal Vascular Occlusion

- Ophthalmoscopes picture of disc swelling, venous engorgement, cotton wool spots and diffuse retinal hemorrhages like blood and thunder. Hypertension is a major risk factor.
- Loss of vision may be severe.
- 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).
- Treatment? Beyond your level
  - Intravitreal injection of anti-vascular epithelial growth factor (anti-VEGF).
  - Laser is another option.
- Two types:
  1. Severe: called ischemic central retinal vein occlusion (associated with afferent pupillary defect in addition to poor vision)
  2. Mild: called nonischemic central retinal vein occlusion.

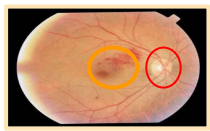
- Flame-shaped hemorrhages all over.
- Visual loss due to macular edema
- Cotton wool spots (blue circles).
  - Cotton wool spots are patchy & large unlike exudates which are smaller & well-defined.
  - Cotton wool spots are fluffy white focal lesions with indistinct margins.
  - Cotton wool spots are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





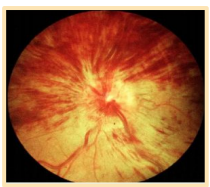


- Retinal vein occlusion.
- Yellow circle: drusen "age related macular degeneration" not bright as much as hard exudate



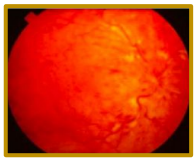
- Macular branch retinal vein occlusion causing a small flame shaped hemorrhage (orange circle).
- Tortuous viens
- No optic nerve swelling cause it affect only branch (red circle)

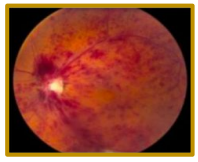
- Hemi-retinal vein occlusion.
- Half of the retina is occluded completely, while the other half is slightly occluded; Still there are tortuous vessels, viscous not well-defined swollen optic disc (non-) flame shaped hemorrhages.

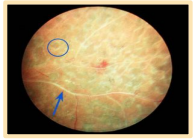


Retinal vein occlusion (wither central or branched).

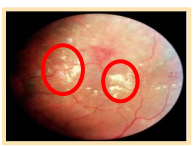
Central vein occlusion characterized by painless visual loss. On fundoscopy: stormy sunset.



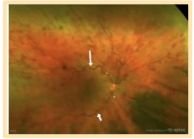




Circle: Laser scar (laser marks)
Arrow: Clear vessel "laser mark" (no blood anymore)



Hard exudate occurs in chronic edema

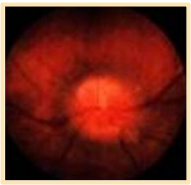


Tortuous vessels (arrows) & flamed shaped hemorrhage = central vein occlusion

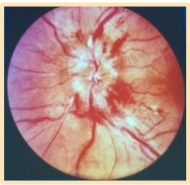
Optic Diseases

Optic neuritis

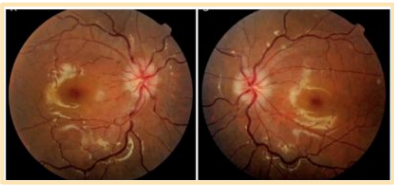
- Optic neuritis is an inflammation of the optic nerve and is usually idiopathic, but it may be associated with multiple sclerosis (as first clinical manifestation) in a significant number of cases.
- The visual acuity is markedly reduced, and an afferent pupillary defect is present (IMP IN OSCE).
- 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 and interferon).
- 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

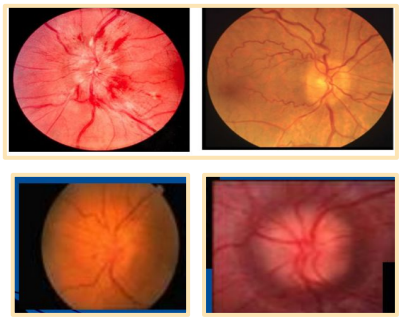
Extra	Papilledema	Papillitis	Retrobulbar neuritis
Definition	Swelling of optic nerve head due to increased ICP	Inflammation or infarction of optic nerve head	Inflammation of orbital portion of optic nerve
Uni/bilateral	Bilateral	Unilateral	Unilateral
Vision impairment	Enlarged blind spot	Central/paracentral 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

Functional visual loss

- A functional disorder is used in preference to hysterical or malingering 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

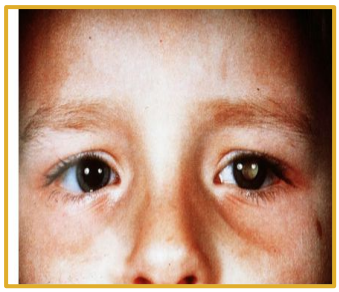
Total hyphema
"8 pool hyphema"

Edema or myelinated nerve fibers

Edema

Qs IN EXAM
What is it called?
Leukocoria (in children)

What causes it?
- Retinoblastoma
- Cataract
- Coat's disease

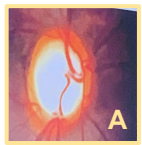


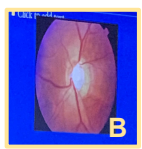
Coats disease: abnormal dilated blood vessel "retinal telangiectasia" leak under the retina and lead to detachment

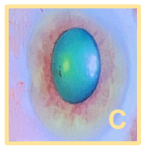
Doctor's Notes (IMP)

- 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.

Neovascular glaucoma: (added by doctor when giving chronic visual loss)

- One of the causes of acute visual loss.
- **Pathophysiology:** neovascularization in the iris (rubeosis iridis) goes and block the TM and it also leaks and make adhesion (synechiae) and causes 2 nd closure angle glaucoma increase the IOP.
- We cannot do laser iridotomy, because the mechanism of closure is different than 1ry closure, which is pushing (pupillary block), But here it is pulling mechanism there is synechiae that is pulling the angle and closes it.
- **Clinical Presentation:**
  1. Decreased VA
  2. Corneal edema
  3. High IOP
  4. Above 40
  5. New vessels in the angle or iris (rubeosis iridis)
  6. Semi dilated pupil
  7. Fundus: Ischemic retina could be caused by diabetic retinopathy, central retinal vein occlusion, or sickle cell anemia.
- **Causes**:
  1. Diabetic retinopathy most common cause in middle east
  2. Central retinal vein occlusion most common cause in Europe
  3. Carotid insufficiency
- **Treatment:**
  1. Initially treat the retinal ischemia
  2. Then Glaucoma surgery to decrease the IOP
- **Scenario:**

65 YO diabetic male, presented with decreased vision and pain in the eye, IOP is 45, semi dilated pupil, fundus show proliferative diabetic retinopathy, what is the Dx?

- Open angle glaucoma
- Closed angle glaucoma
- Secondary glaucoma
- Neovascular glaucoma

- **What is the Initial treatment?**
  - Laser iridotomy
  - Laser trabeculoplasty
  - Laser photocoagulation of the retina or PRP

OPTHALMOLOGY TEAM

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