

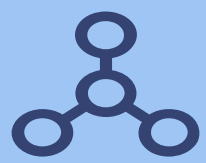
Ophthalmology
Team 437



Acute visual Loss

Objective

- not provided



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Resources :

Dr. Abouammoh slides and notes, 436 teamwork
Book (Lecture notes in Ophthalmology)

- Editing file

Color index

- **Important (F1)**
- Notes
- **Book**
- Extra
- **Golden note**



Acute visual loss

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.

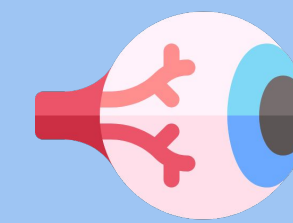
- A disaster for most people and you should be able to evaluate such a patient and be able to recognize situations requiring urgent action.
- It may affect one or both eyes, all or part of visual field, or it may arise from a pathology in any part of the visual pathway.

Etiology:

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

Acute visual loss classified by PAIN 	
Painful	Painless
<ul style="list-style-type: none">❑ Acute Angle Closure Glaucoma: can be acute or chronic (and becomes open angle).<ul style="list-style-type: none">- 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.❑ Uveitis:<ul style="list-style-type: none">- It may be slow or sudden and acute.- Patient is always in pain.❑ Keratitis:<ul style="list-style-type: none">- Infection (microbial keratitis) or inflammation of cornea.- Very severe pain, more than uveitis.❑ Hyphema (Traumatic): can be asymptomatic unless if it's associated with other things.❑ Endophthalmitis: infection of the eye as a whole (infection of the globe).	<ul style="list-style-type: none">❑ Vitreous Hemorrhage:<ul style="list-style-type: none">- It can be painful if it is traumatic.❑ Retinal Detachment:<ul style="list-style-type: none">- It could be caused by trauma or w/o trauma.- The patient may have it and not discover it until covering one eye.❑ Retinal vascular occlusions:<ul style="list-style-type: none">- arteries/veins.❑ Optic neuritis:<ul style="list-style-type: none">- It happens in cases with MS could present with pain or without.- usually not painful but eye movement may be painful.❑ 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.



History (Hx)

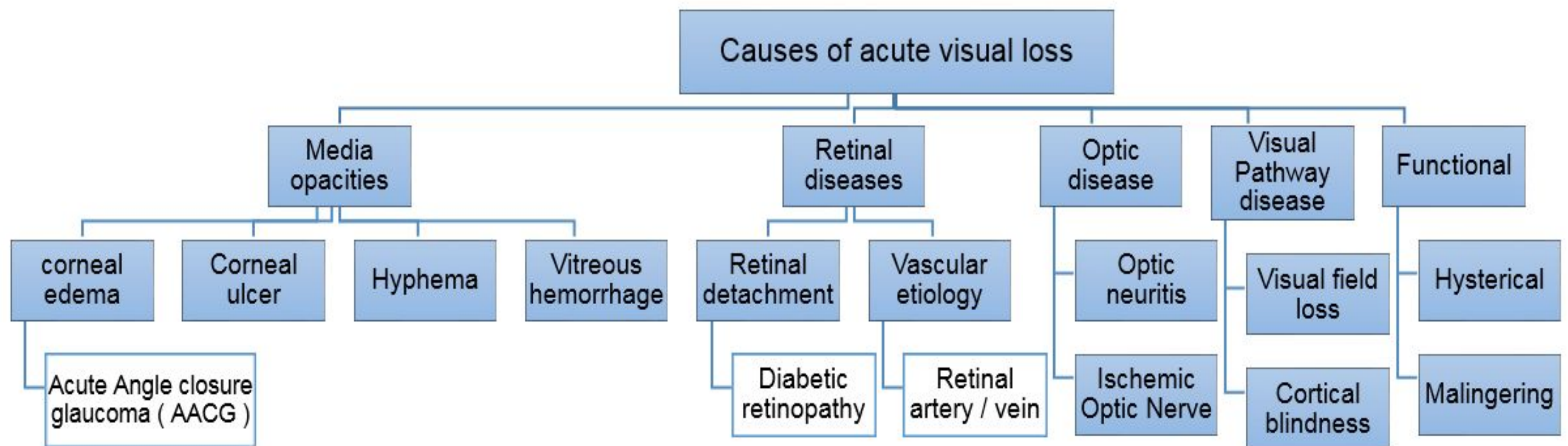


- What is the patient's age and general medical Condition?
 - ❑ For example you don't expect an elderly patient to present with optic neuritis.
 - ❑ If pt is diabetic for example; You would expect certain causes of visual loss
 - ❑ Young with no systemic disease → think about neurological causes: optic 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 (P/E)



- 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).



Media Opacities

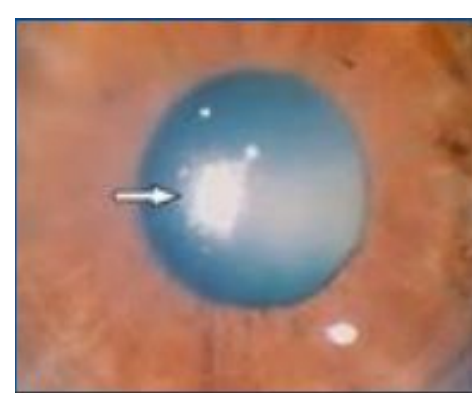
- Corneal opacity is either due to edema or infection (like uveitis) or trauma corneal ulcer

❖ Corneal edema

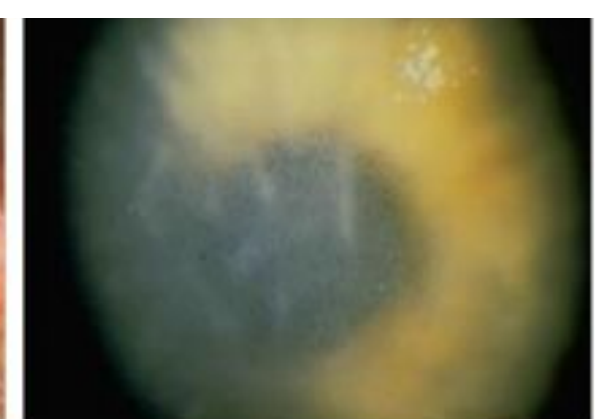
- The cornea usually translucent and made of collagen fibers in a way that makes it clear but here it appears like a ground glass (مائلج أو مطحون) rather than its normal clear transparent appearance (steamy cornea).
- The most common cause of corneal edema is **increased intraocular pressure** & occurs 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 bundling 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 hypotony¹
- 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



Extra

❖ 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.



¹ Hypotony is usually defined as an intraocular pressure (IOP) of 5 mmHg or less.

❖ Acute Angle Closure Glaucoma:

- Signs & symptoms

- Painful acute visual loss

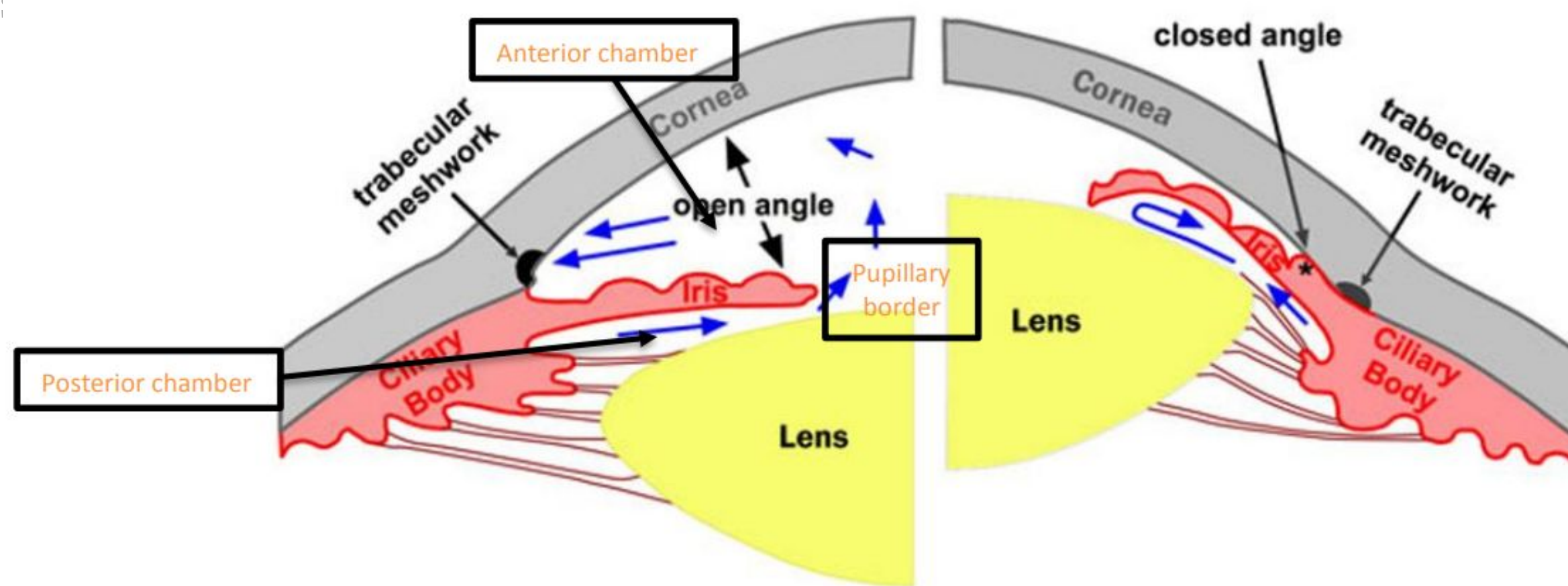
And collection of these:

- 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?

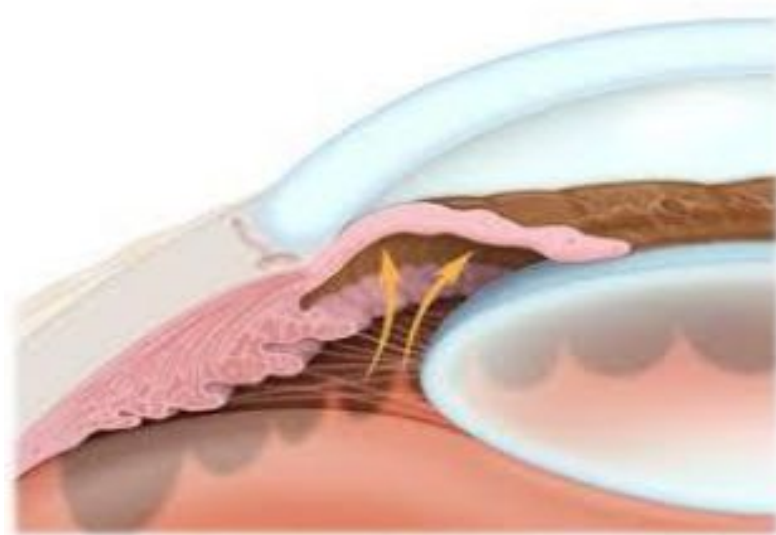
- Normally the pupil either constricts or dilates.
 - In glaucoma, the IOP is so high that the blood supply stops to iris muscles (sphincter pupillae & dilator pupillae) resulting in ischemia.

- 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.
- Redne:



- 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 angle to start with; At a certain point, may be 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; adding over this, the iris may bow anteriorly because of the pressure effect & touch the lens which will make the angle more occluded.

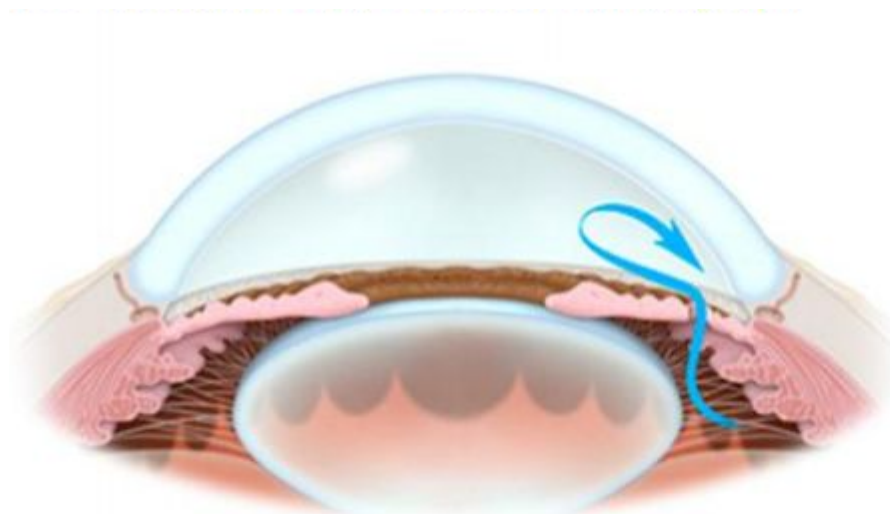


- Peripheral iridotomy

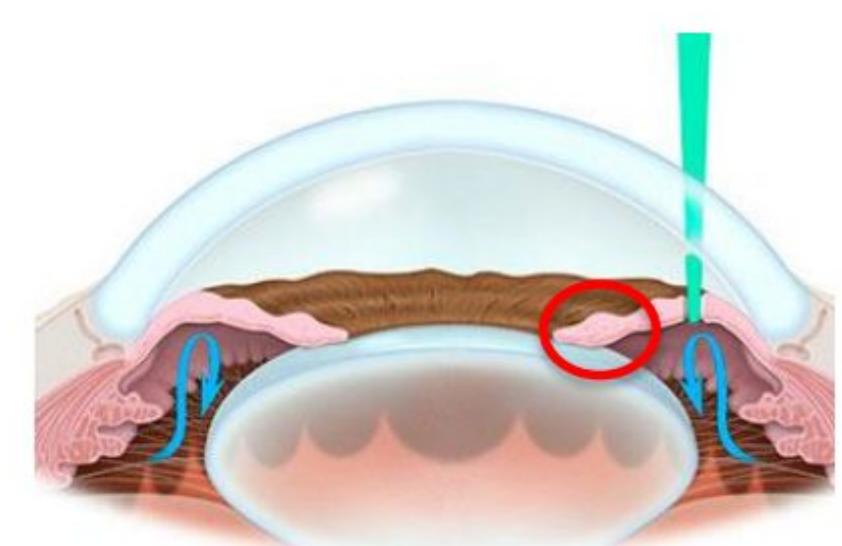
- The angle is narrow & the trabecular meshwork is occluded by the iris touching (if pt presents early) the periphery of the cornea & the pupillary edge of the iris bows against the lens closing the posterior chamber & pressure is building up.

- Aims of acute ACG management:

- Decrease IOP. 1st
- Prevent future attacks in OU (oculus uterque, which means "both eyes") prophylaxis. Even if other eye normal. To prevent attack or glaucoma



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



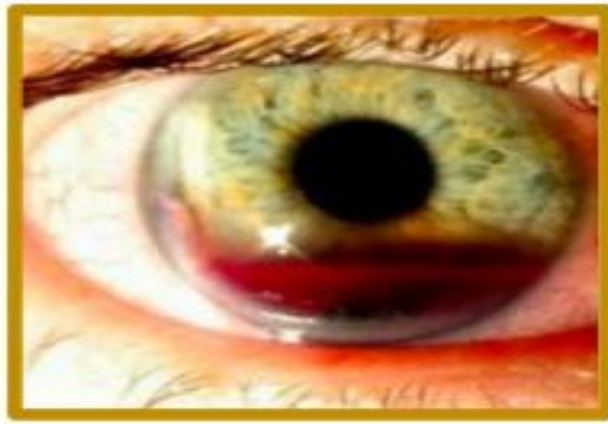
if pt presents late the iris will stick to the lens causing (posterior synechia) *red circle*

❖ 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, intraocular surgery (post-op), chronic inflammation and uveitis which all cause neovascularization. (Tumor and DM)
- Sick cell pts 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”.



We. Measure the height of blood in mm

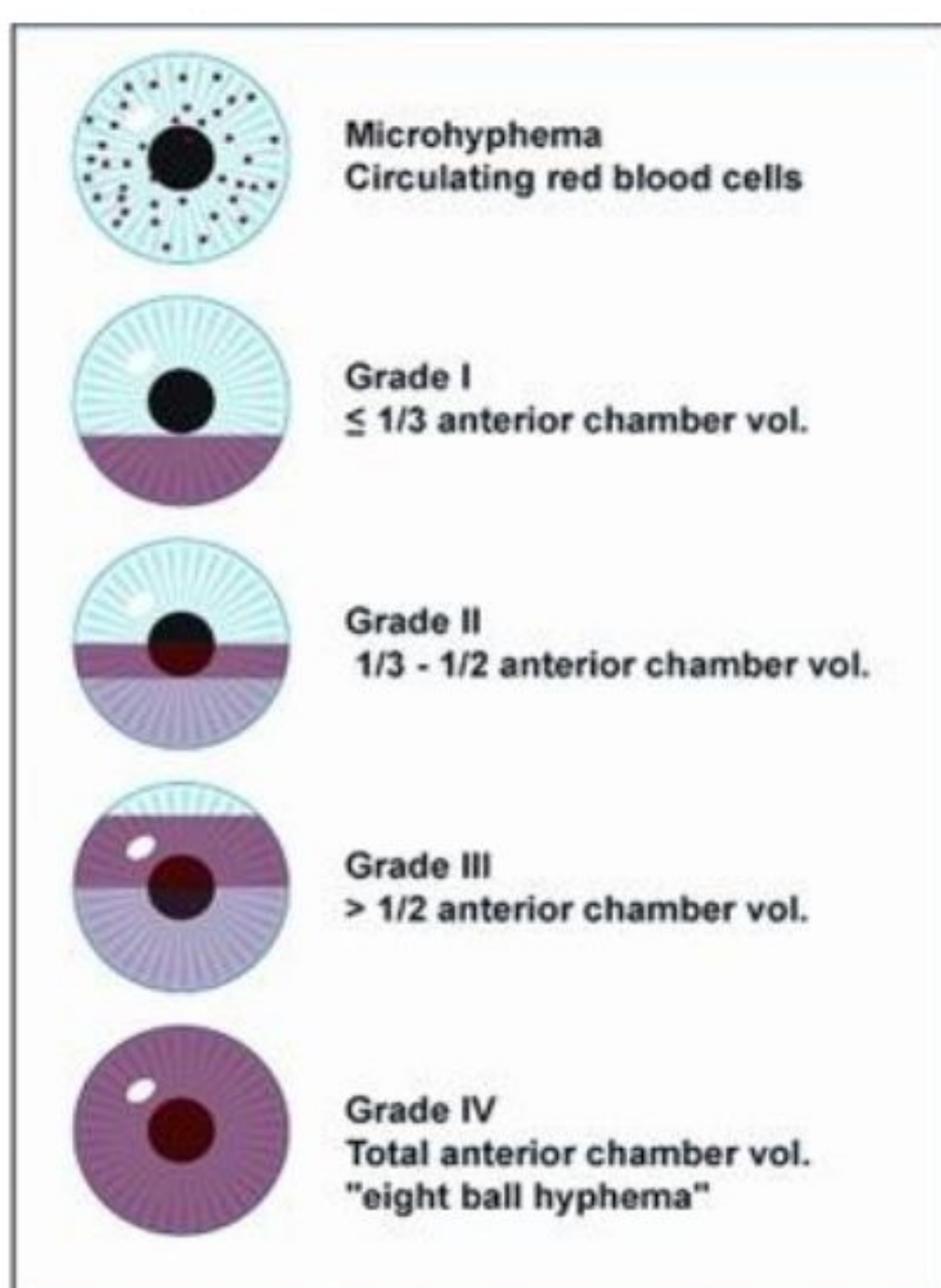


Hyphema
Complications: posterior synechiae & glaucoma.



Eight ball hyphema
Filling of the whole ant. chamber

- 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.

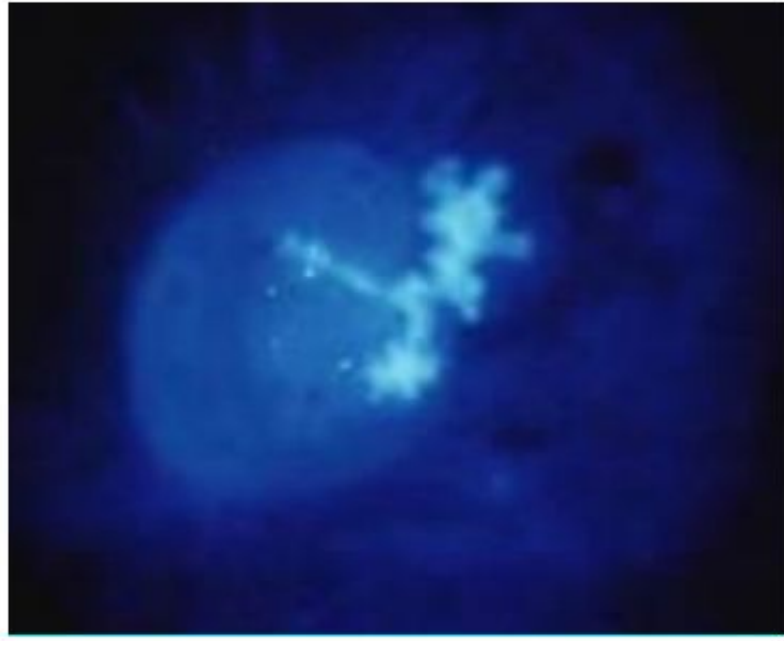


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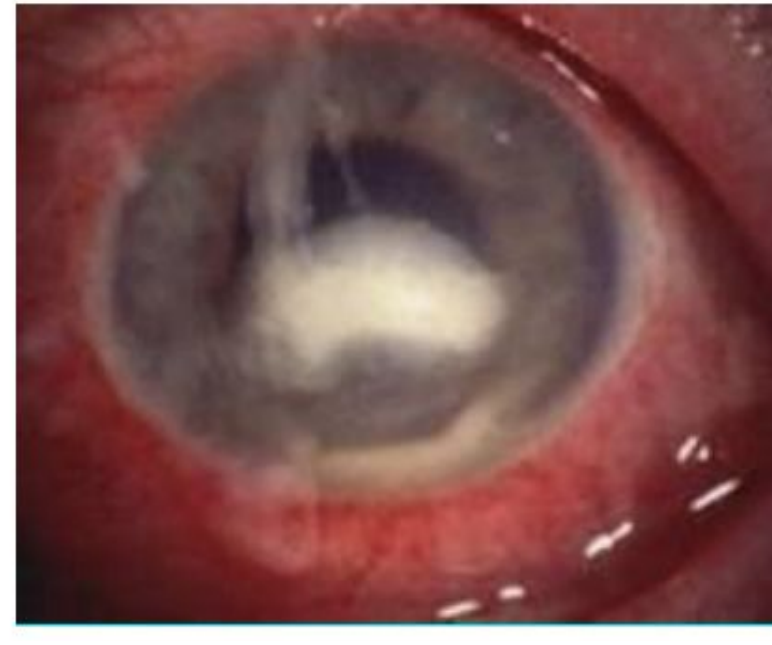
Extra

❖ 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.



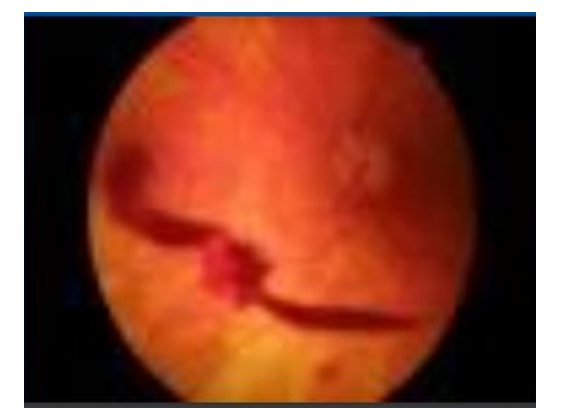
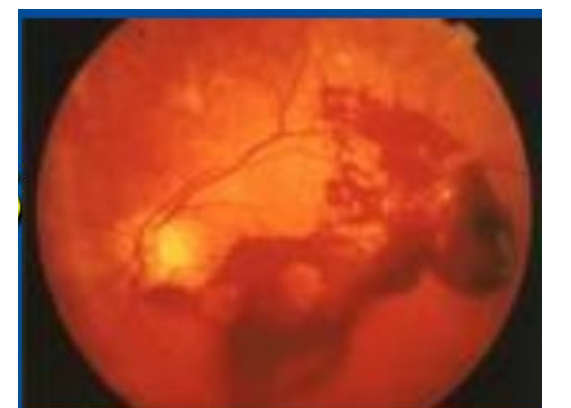
Viral herpetic herpes



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

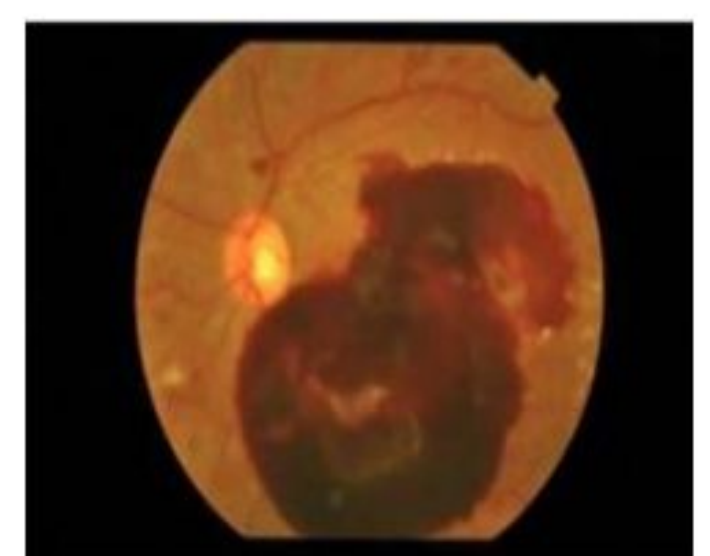
❖ **Vitreous hemorrhage**

- 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.
- It can result from: trauma, diabetes, retinal vein **chronic** occlusion, **acute posterior vitreous detachment and intraocular surgery**; it may also accompany subarachnoid hemorrhage (**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.

- **B scan (bi-model ultrasound) is important** to know the etiology.
 - 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.



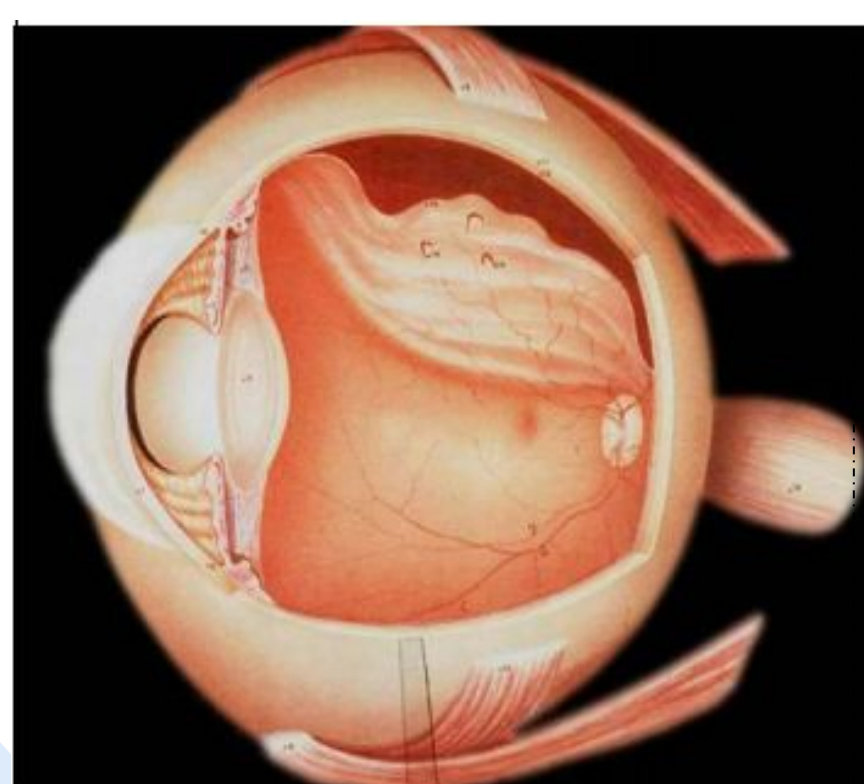
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Retinal diseases

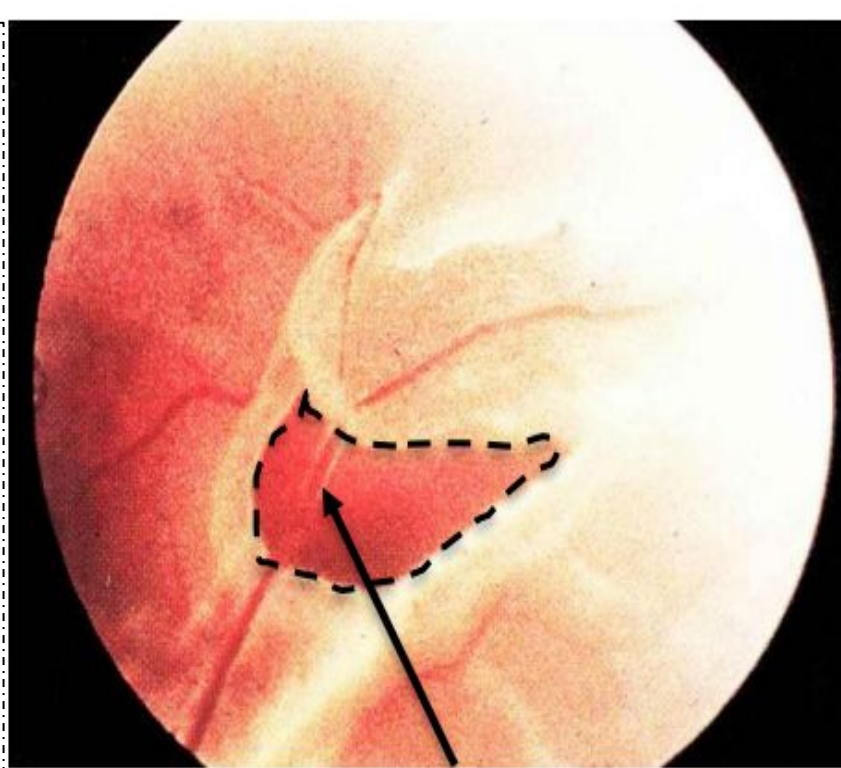
❖ Retinal detachment

- 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 of retinal detachment		
1. Rhegmatogenous RD (rhegma = break) acute	2. Traction RD chronic	3. Exudative RD chronic
<ul style="list-style-type: none"> • 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. 	<ul style="list-style-type: none"> • Slow and gradual loss of vision due to diabetes or TB. • Treatment is surgery (requires intervention). <ul style="list-style-type: none"> ○ 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 	<ul style="list-style-type: none"> • 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. • Pt complains of gradual loss of vision.
<p style="text-align: center;">Extra</p>		<ul style="list-style-type: none"> • Picture: when a break happens, subretinal space allowed vitreous fluid to travel into the break and detach the retina.



Breaks without detachment (If it stretches more > Detachment) Vitreous fluid collects causing detachment



Break (dash line) - Retinal vessel bridging the break

Horseshoe break: if the break gets pulled more (by the vitreous) → can rupture a vessel → RD & hemorrhage.

- 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.

- **Signs & symptoms:** (This is applied in rhegmatogenous RD).

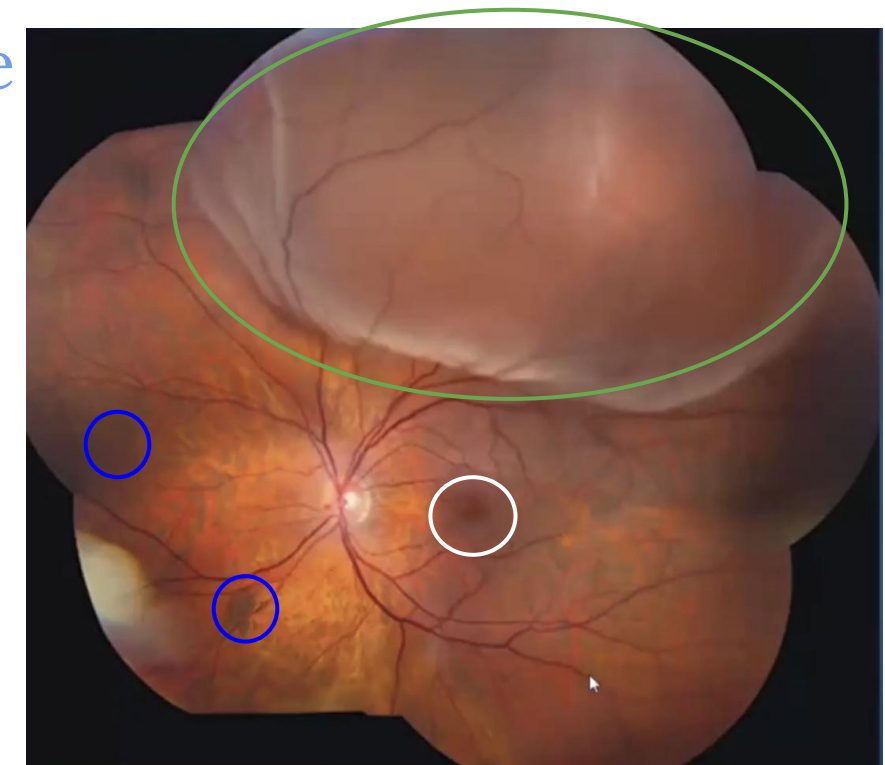
- Prodromal symptoms: **flashes** bolts of thunder + **floaters** like cobwebs, lines or dots.
- If a pt comes early, we may save the eye by surgery which means there is something we can do; But, if the pt is late, pt will present w/ **painless visual field loss-curtain-like** which is the actual retinal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

- **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).
- Hx of surgery because you play w/ vitreous.
- Mechanical
- Trauma.

- blue : myopic fundus
- white : macula
- Green bolus detachment

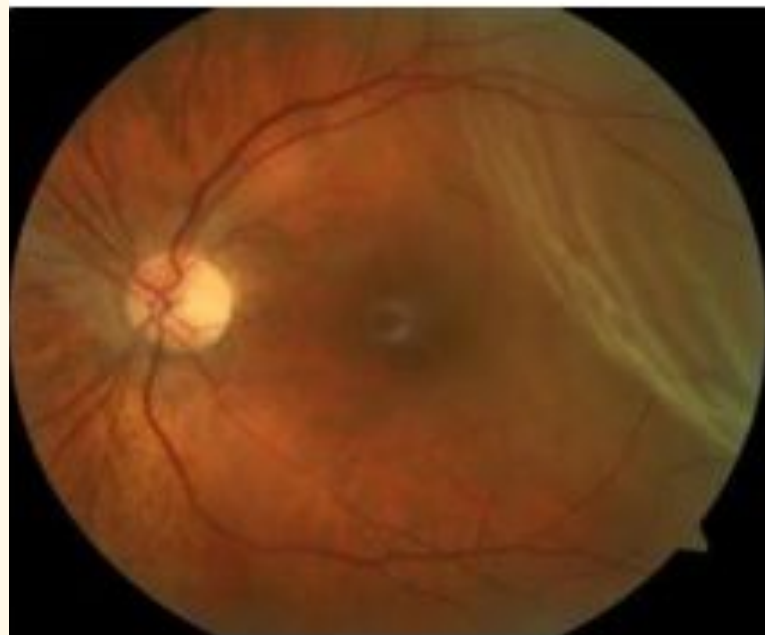


- Aphakia (No lens). Due to Post ocular surgery (any intraocular operation)
 - In the past, they used to deal with cataract aggressively, (traumatic surgeries).
 - because it's a sort of trauma, its abnormal, there should be a lens inside: pseudophakia (artificial lens) → less risk of RD.
- Peripheral retinal degenerations. e.g. lattice degeneration, retinal tufts, etc.
 - Usually peripheral retina is weaker than the central retina. If a pt has weaker retina w/ degenerations, pt will have a higher chance of developing break in the peripheral retina.
- 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.

- **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).
- Macula off: The macula is detached, and intervention is less critical (within 10 days).



Traction

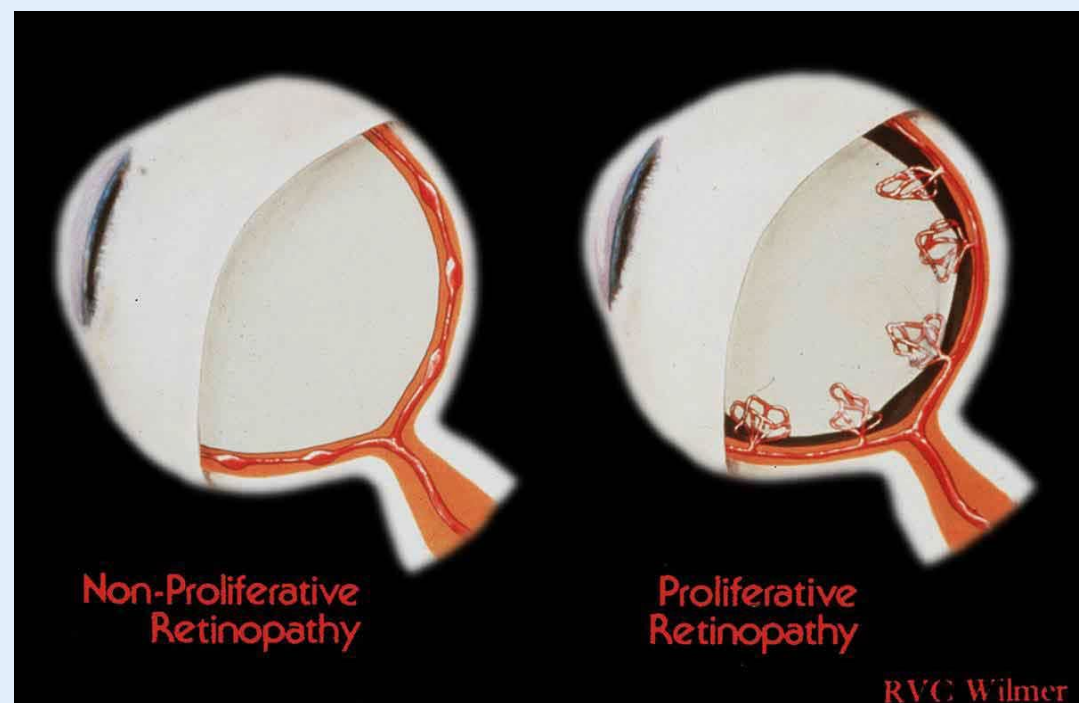
There is a flat retina and the bulging part is detached (if the macula is not involved you can treat this surgically).

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 toxemia 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)

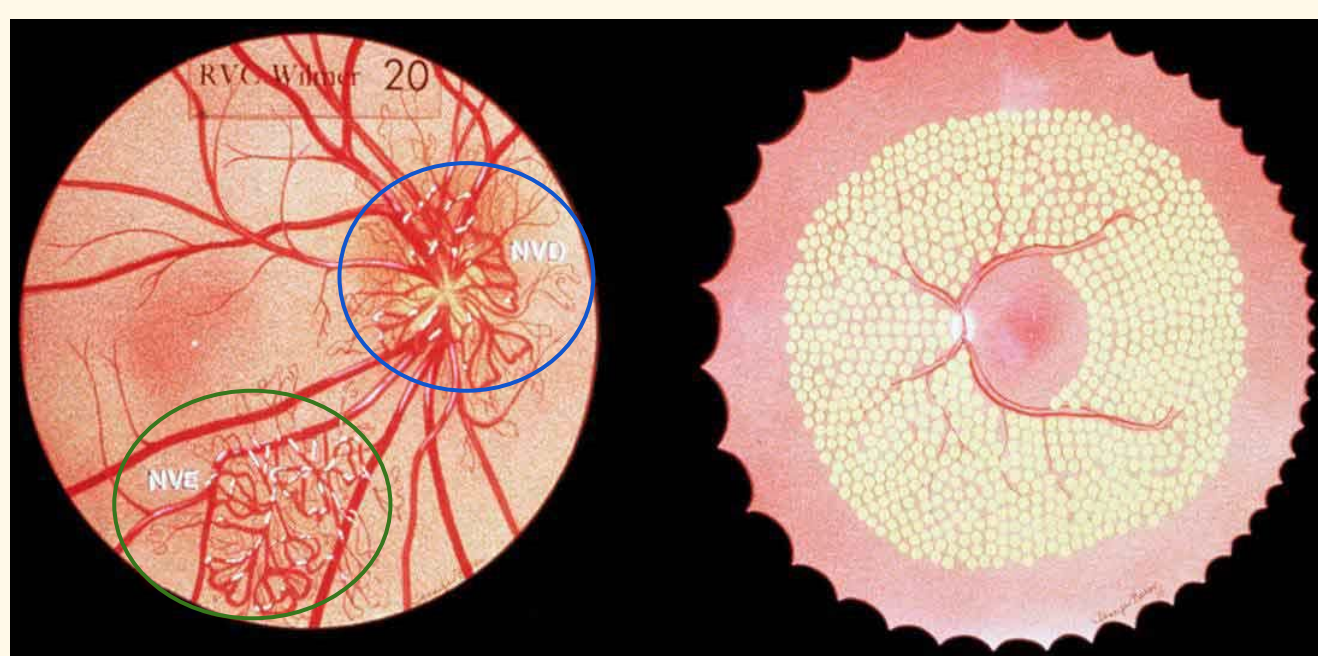


Non-proliferative:

- Vascular changes on the retina.
- Exudates & microaneurysms (dot & blot hemorrhages).

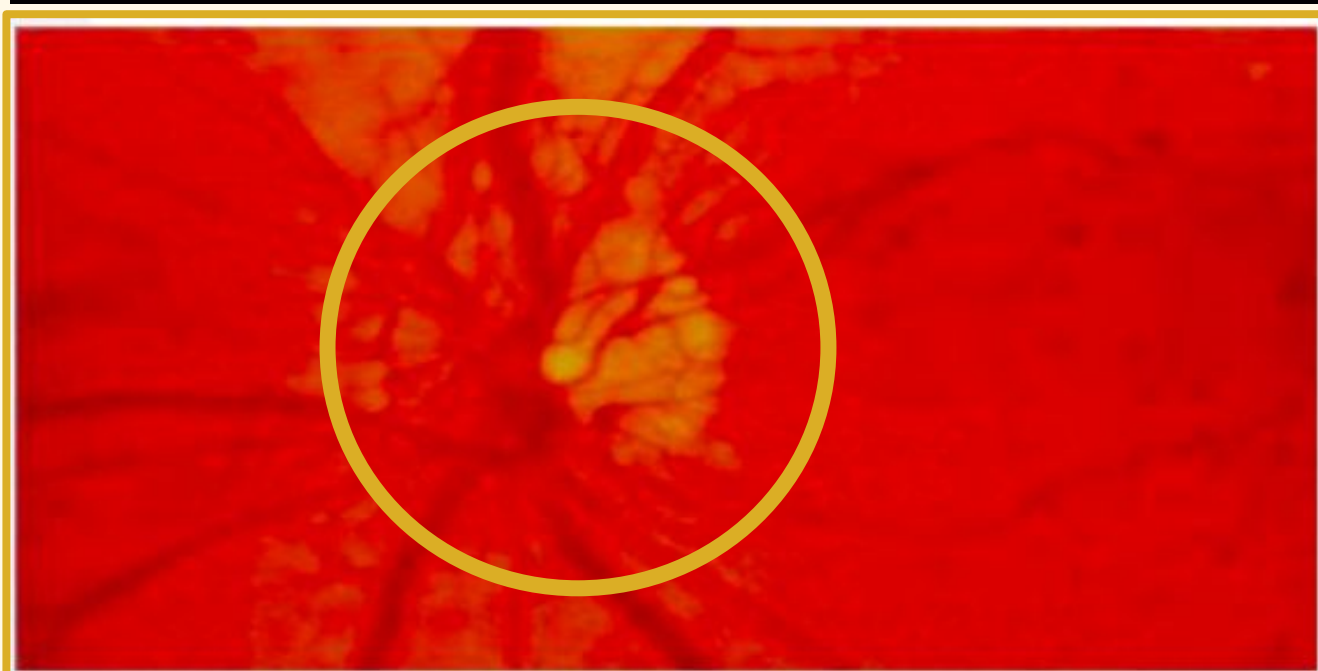
Proliferative:

- 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 + rhegma RD (thus pt may present w/ floaters).

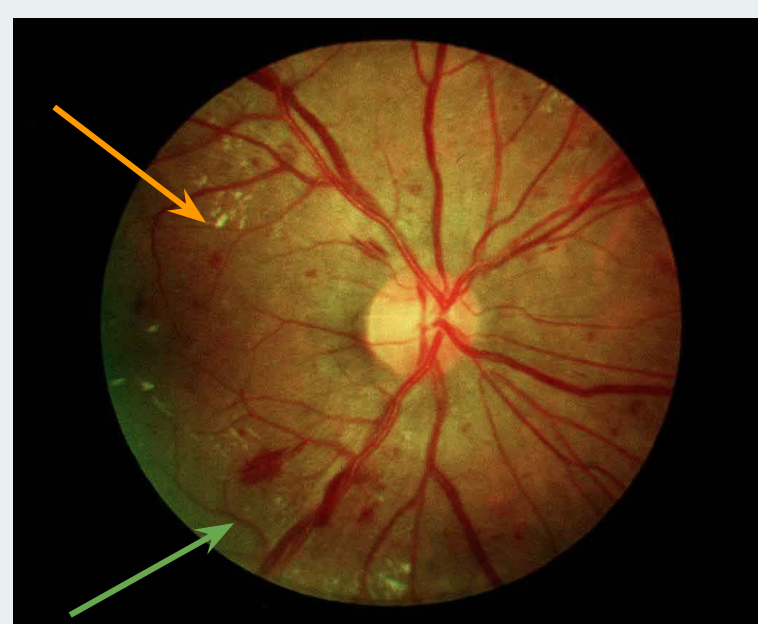


Proliferative retinopathy:

- 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.



Extra pictures



Non-Proliferative retinopathy

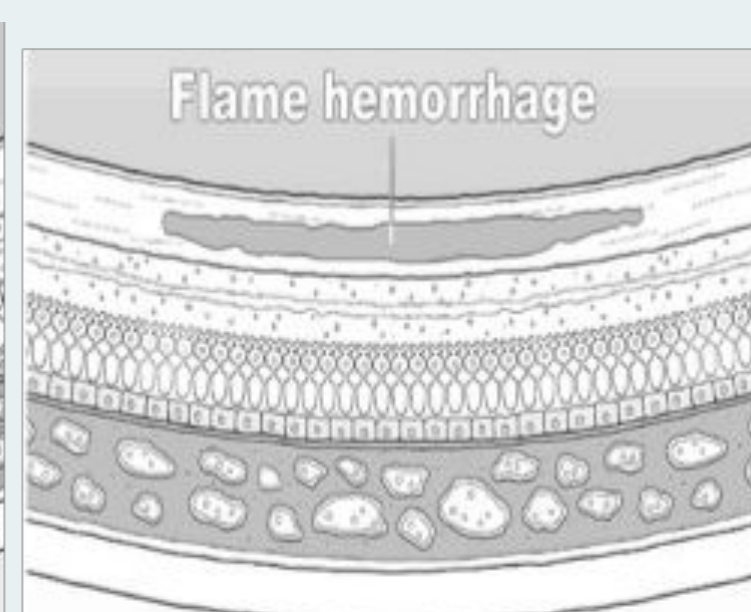
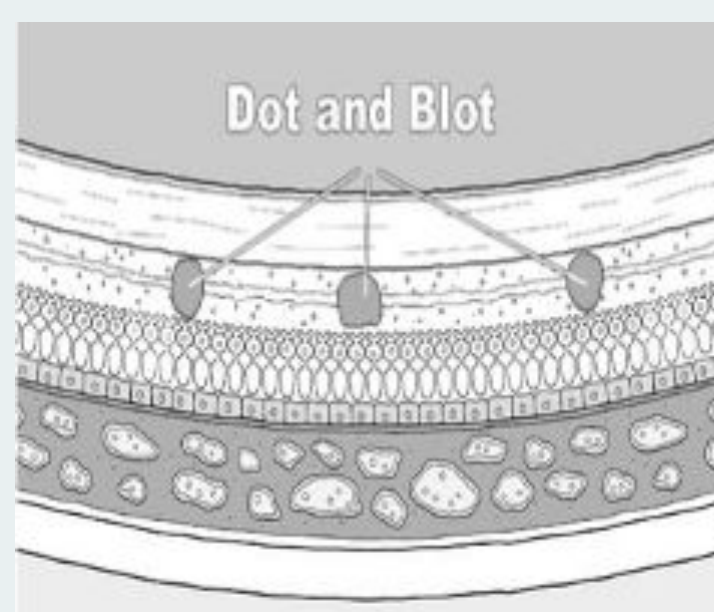
- **Orange** arrow: Exudate
- **Green** Arrow: Microaneurysm

NPDR features:

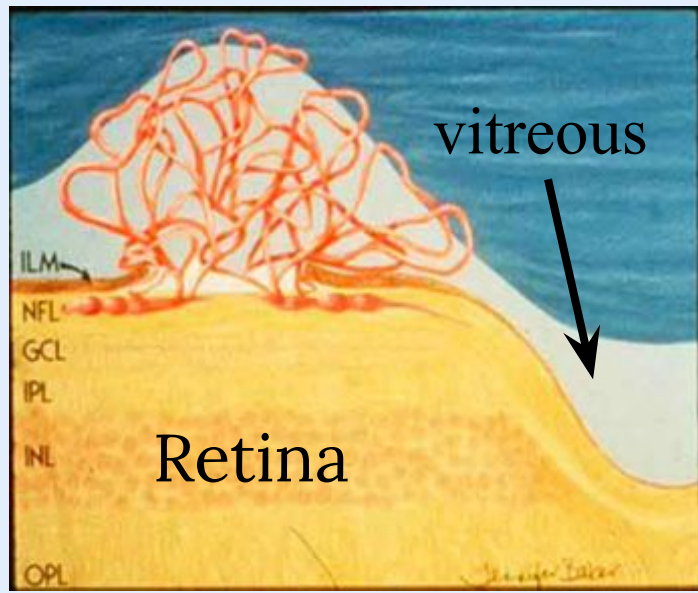
- Injured capillaries can leak fluid into the retina and the aneurysms themselves can burst, forming **"dot-and-blot hemorrhages."**
- Dot-blot hemorrhages look small and round because they occur in the deep, longitudinally-oriented cell layers of the retina. This contrasts with the **"flame hemorrhages"** 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



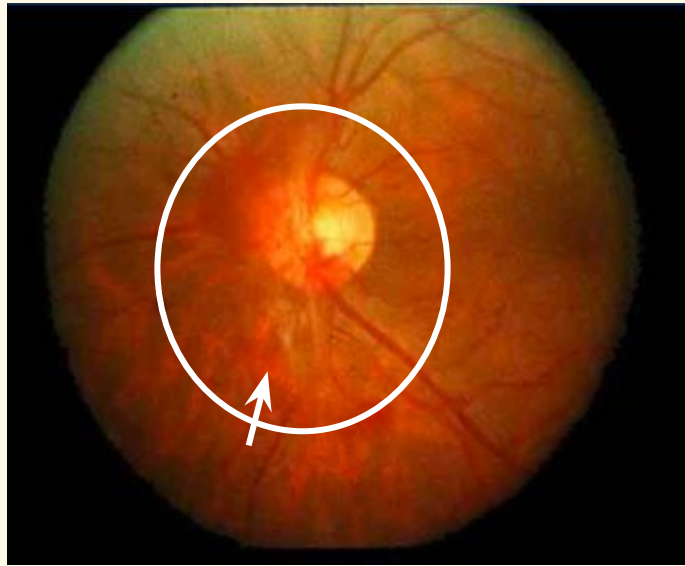
Extra



Diabetic Retinopathy Cont:



- 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.

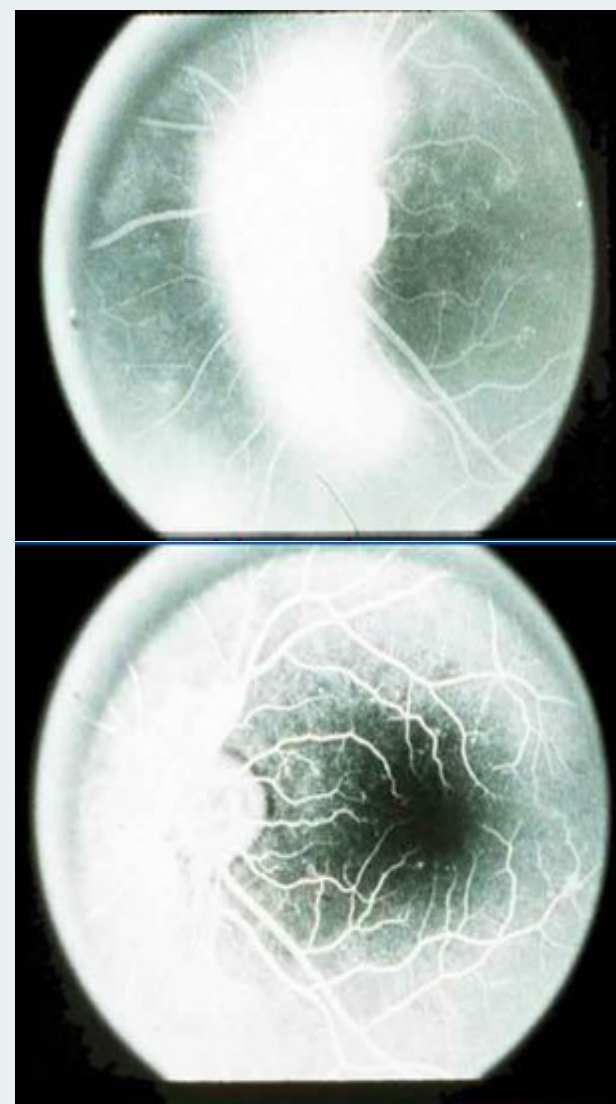


Circle:

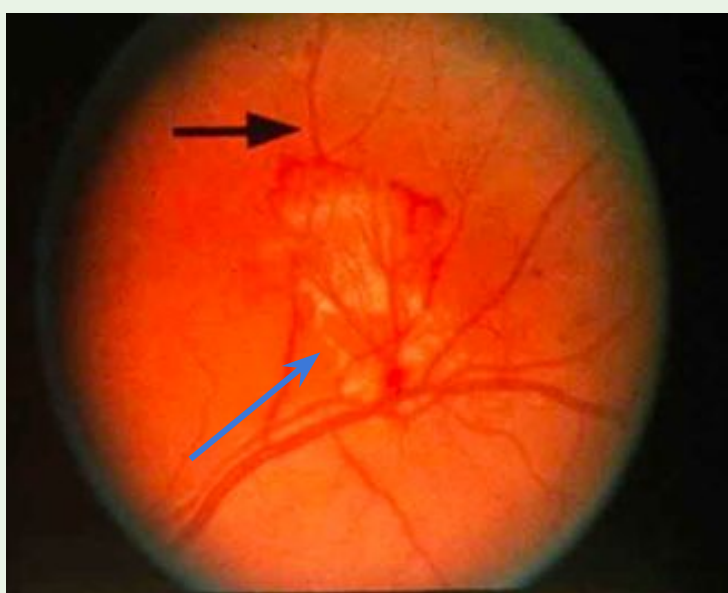
Neovascularizations

White arrows : the white tissue is

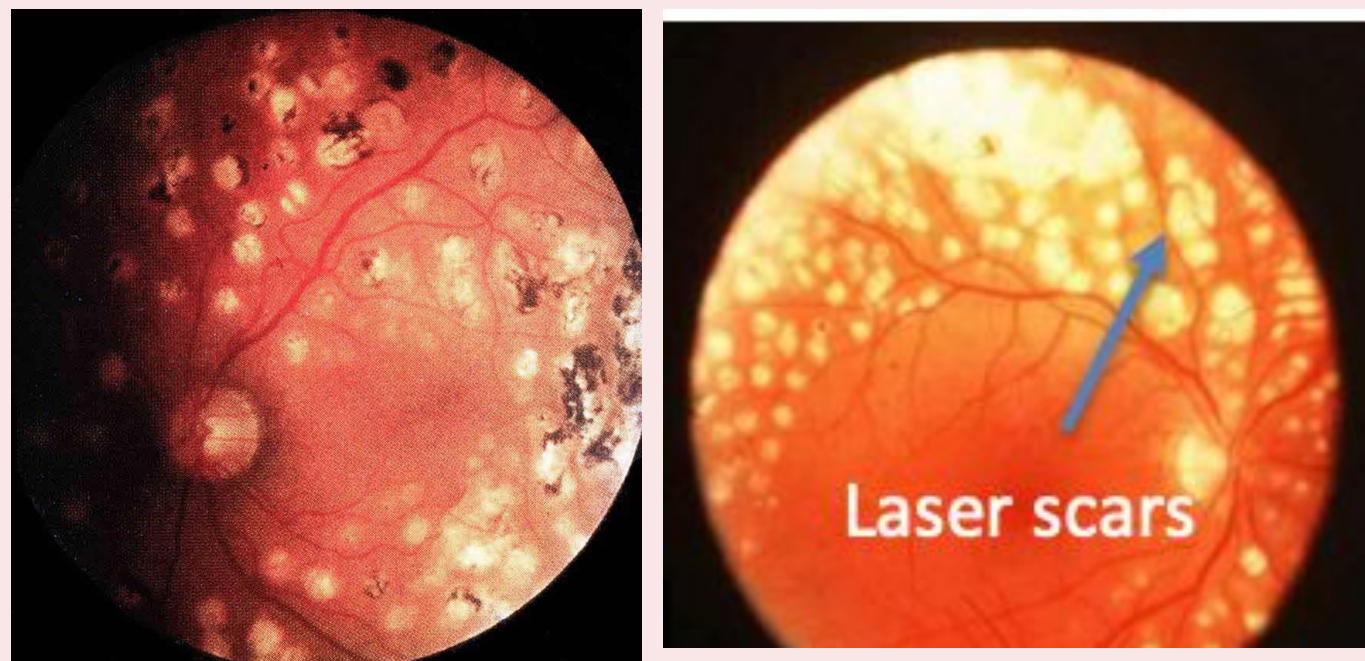
Neovascularizations that is starting to fibrosed



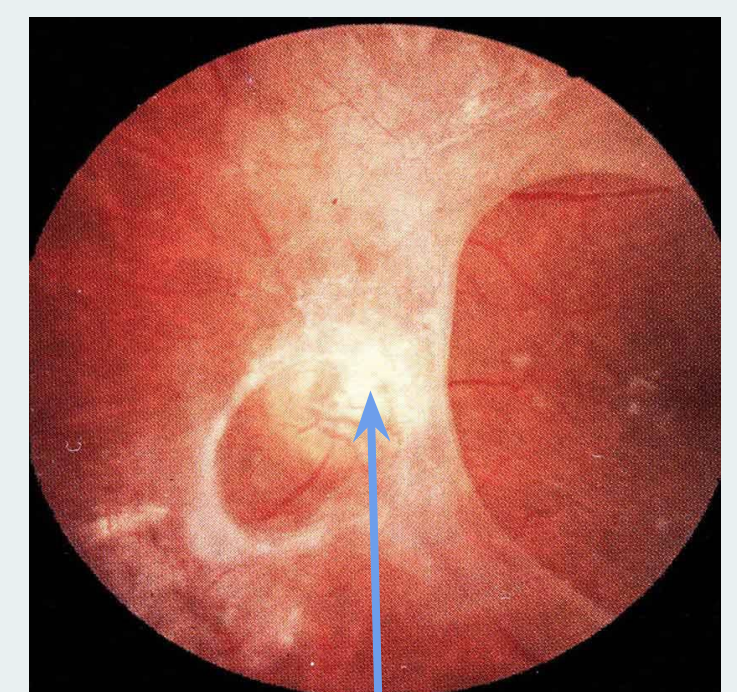
- The neovascularizations always leak, normal vessels don't leak.
- An ischemic retina releases vasogenic factors (e.g. VEGF) which result in the growth of abnormal blood vessels and fibrous tissue onto the retinal surface and forwards into the vitreous. These intravitreal vessels are much more permeable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



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



Laser scars following diabetic retinopathy treatment.



Fibrous tissue



Tractional retinal detachment (complication if pt didn't do laser)



Retinal detachment in histology

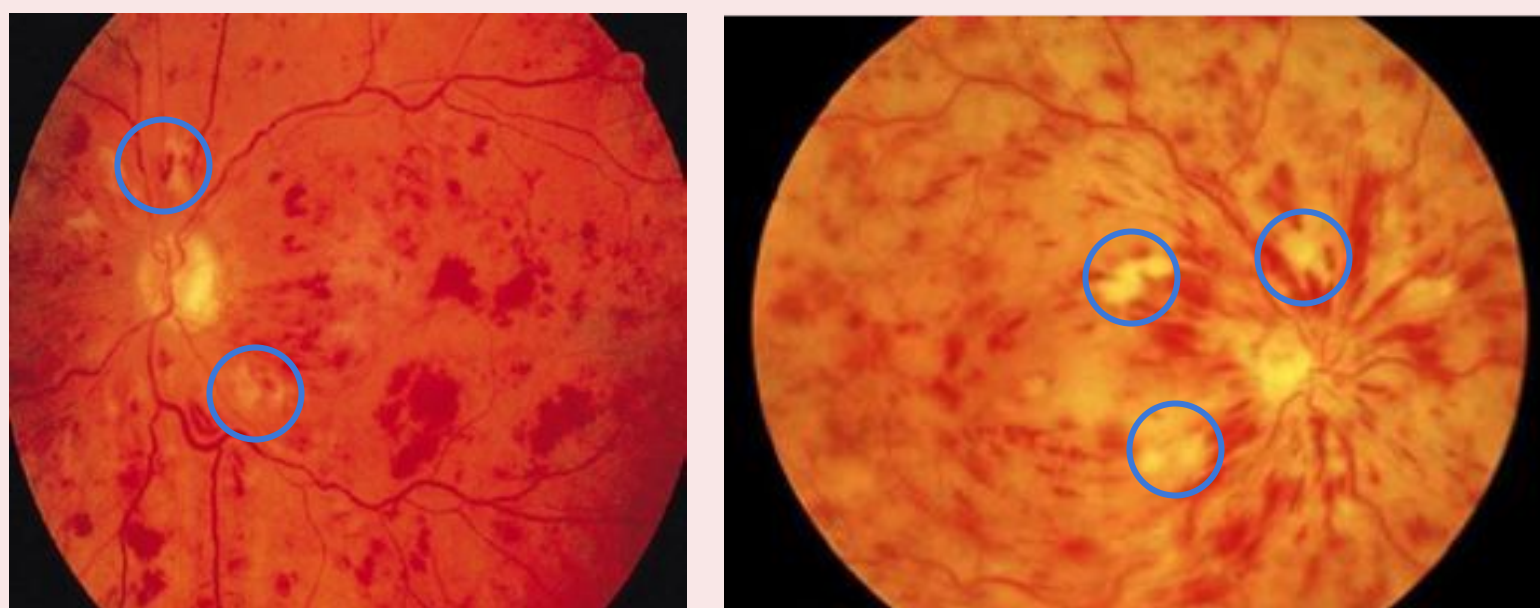
- Fibrosis and contraction of vessels occurs in tractional retinal detachment, the retina is not in its place so it can't function properly.
- **Treatment: laser**
 - We laser everything except the macula
 - The retina goes back in place after laser, but it doesn't mean they'll have good vision. Because 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

Retinal vein occlusion

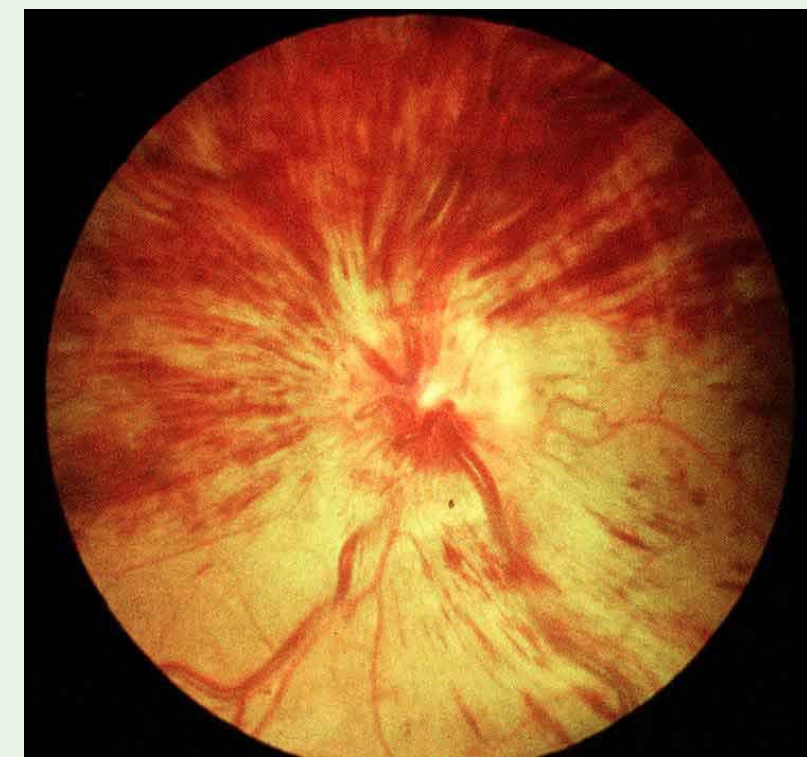
- ophthalmoscopes picture of disc swelling, venous engorgement, cotton wool spots and diffuse retinal hemorrhages like blood and thunder. **Hypertension is a major risk factor.**
- Loss of vision may be severe.
- There is no generally accepted acute management. Central retinal vein occlusion is not a true ophthalmic emergency but it may cause acute visual loss.
 - 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:

- Severe: called ischemic central retinal vein occlusion (associated with **afferent pupillary defect** in addition to poor vision)
- Mild: called nonischemic central retinal vein occlusion.



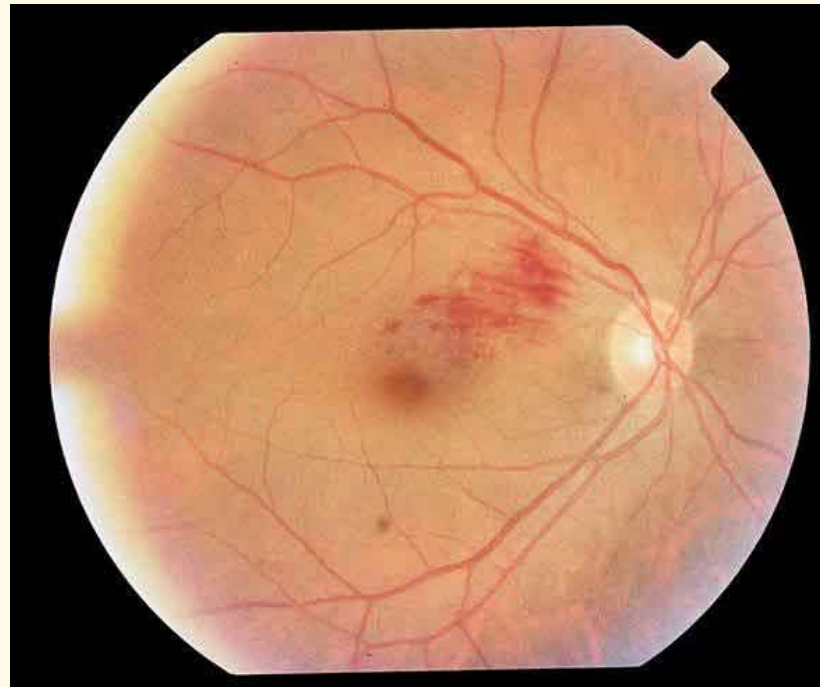
- Flame-shaped hemorrhages all over.
- Cotton wool spots (blue circles).
 - Cotton wool spots are patchy & large unlike exudates which are smaller & well-defined.
 - Cotton wool spots are fluffy white focal lesions with indistinct margins.
 - Cotton wool spots are infarctions in the nerve fiber layer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



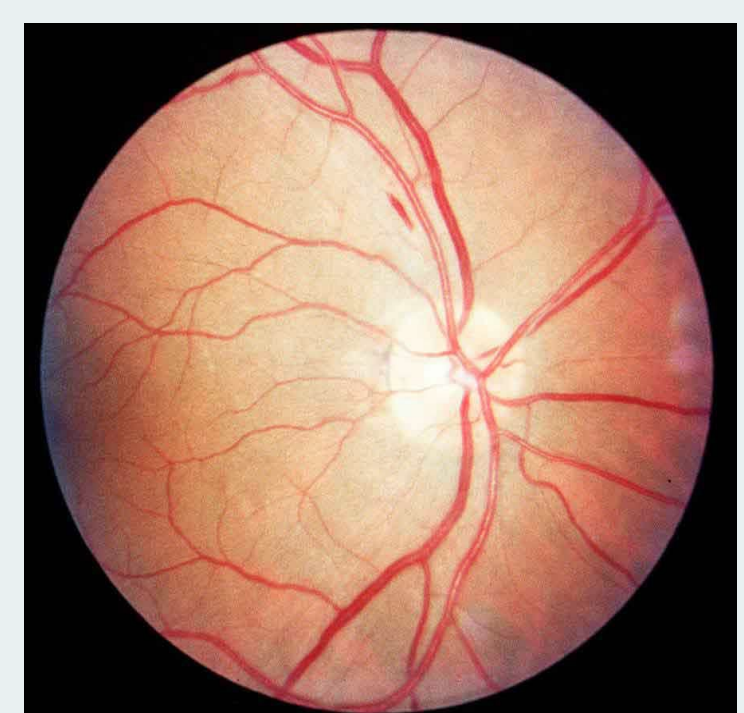
- Hemi-retinal vein occlusion.
- Half of the retina is occluded completely, while the other half is slightly occluded; Still there are tortuous vessels, viscous not well-defined swollen optic disc (non-) flame shaped hemorrhages.



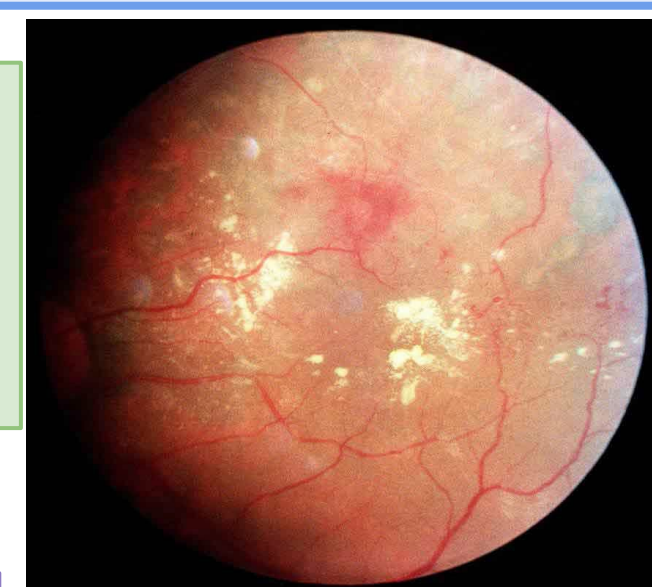
Hemi-retinal (branched) retinal vein occlusion.



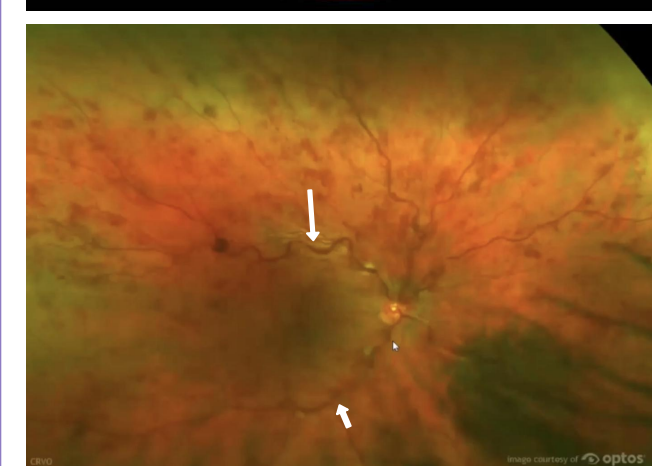
Macular branch retinal vein occlusion causing a small flame shaped hemorrhage.



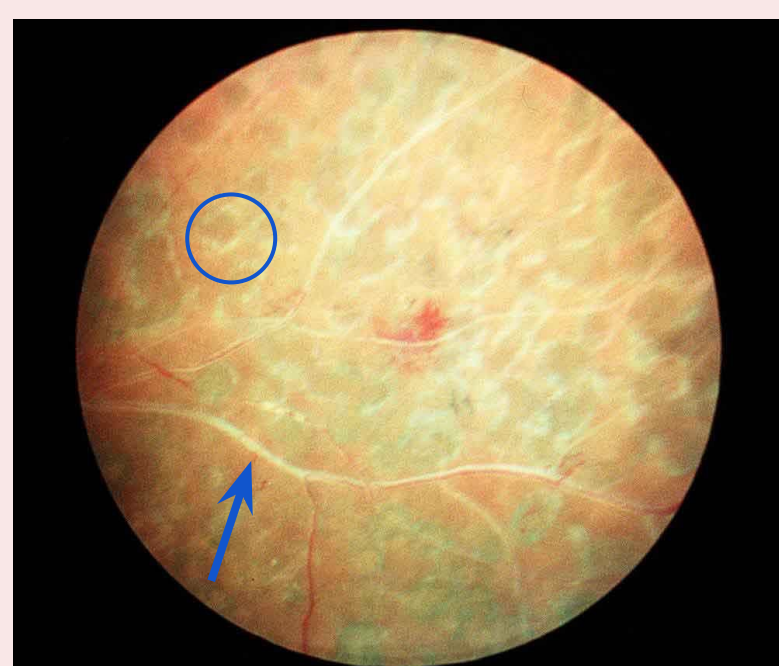
Retinal vein occlusion (with or without central or branched).



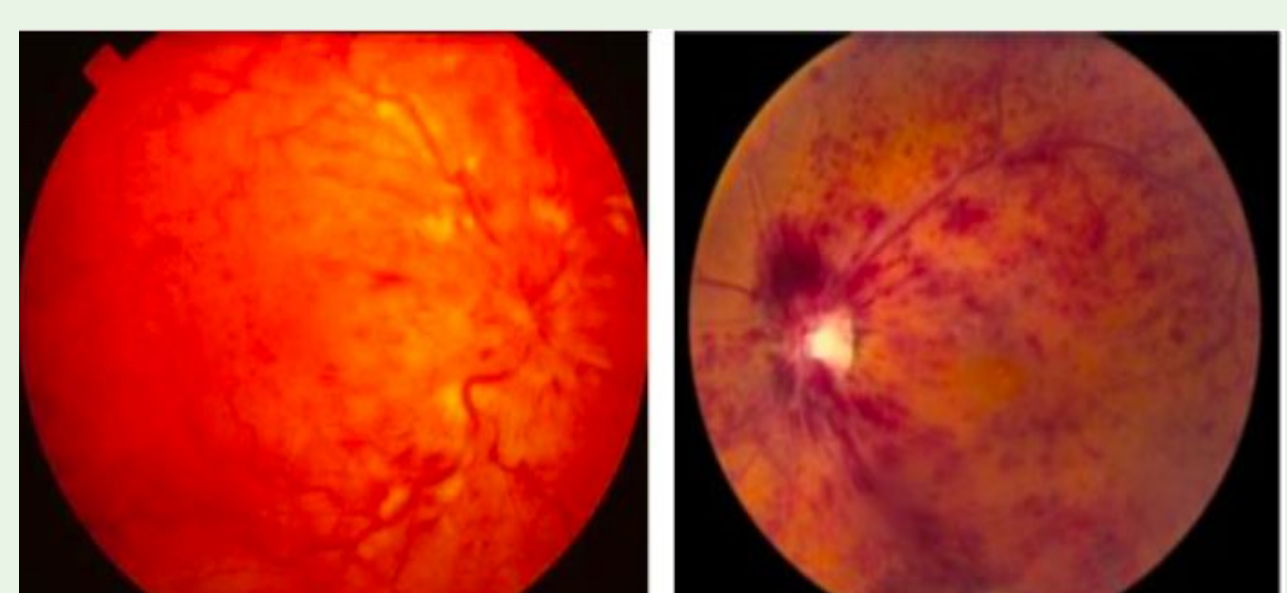
Exudation occurs in chronic edema



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



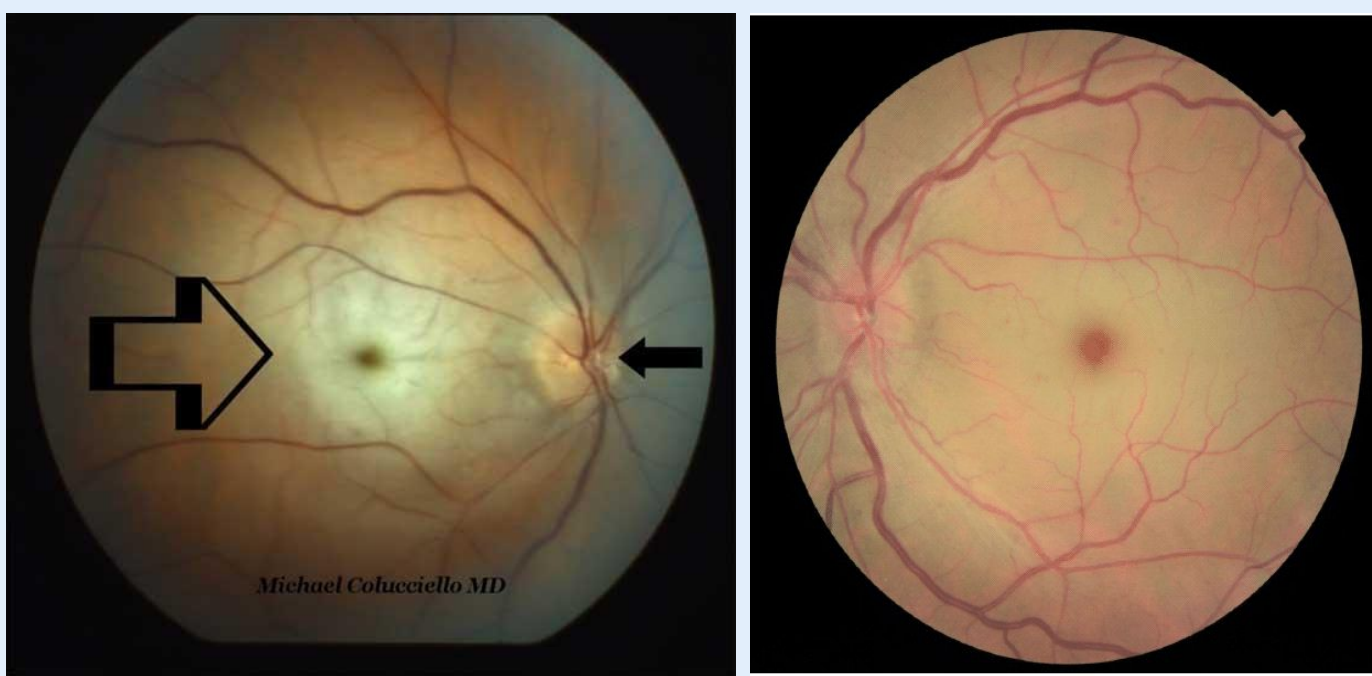
Circle: Laser scar
Arrow: Clear vessel (no blood anymore)



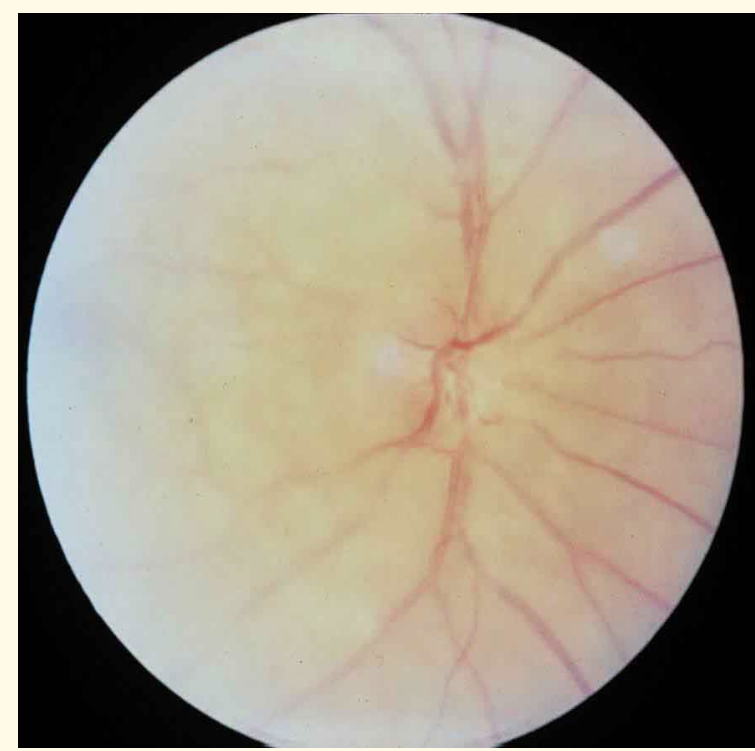
Central vein occlusion characterized by painless visual loss. On fundoscopy: stormy sunset.

Central Retinal artery occlusion

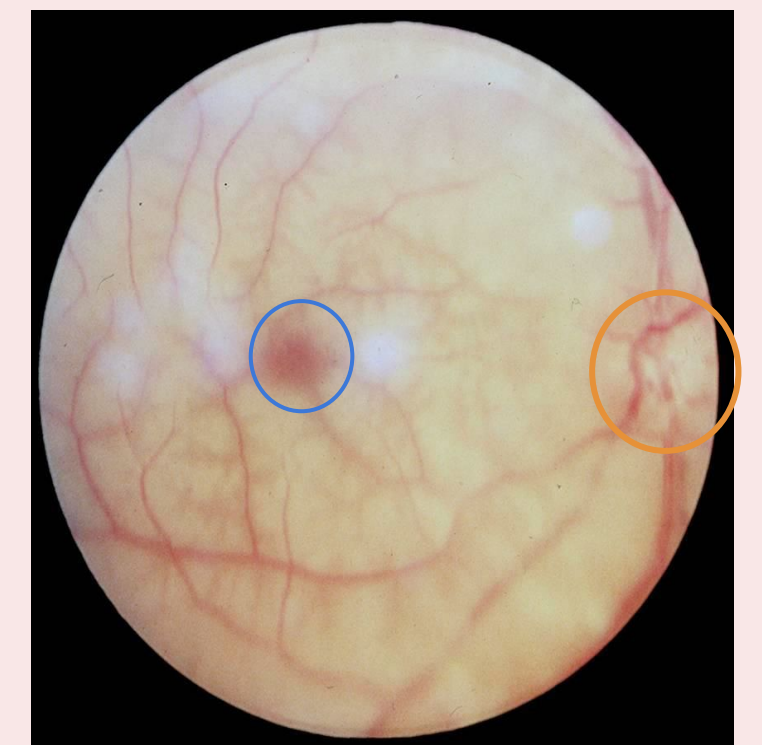
- A sudden, painless and often complete visual loss (no light perception) may indicate central retinal artery occlusion (CARO).
- Several hours after a central retinal artery occlusion, the inner layer of the retina becomes opalescent (opaque, why? because of ischemia).
- A cherry red spot is seen due 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 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.



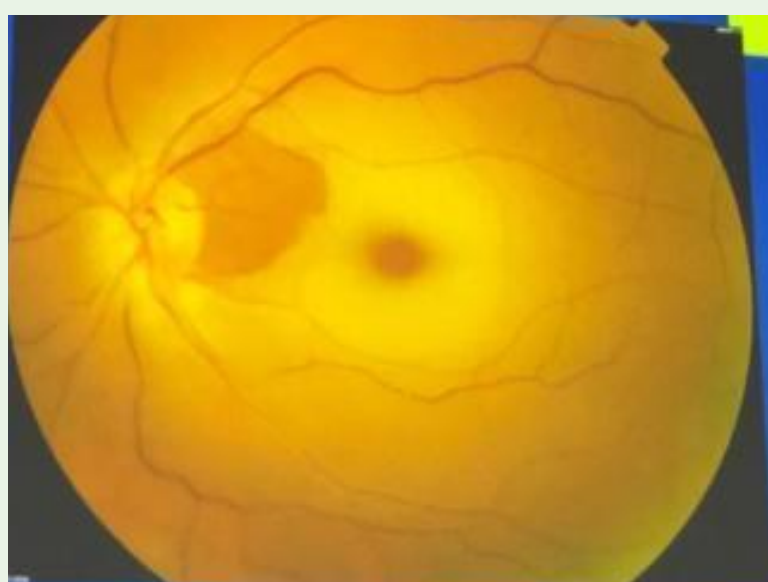
(There is an opaque retina with an edematous disc (small arrow) and the big arrow is pointing at the macula, the dark area is a cherry red spot of the fovea in the center of the macula. Which means: central retinal artery occlusion).



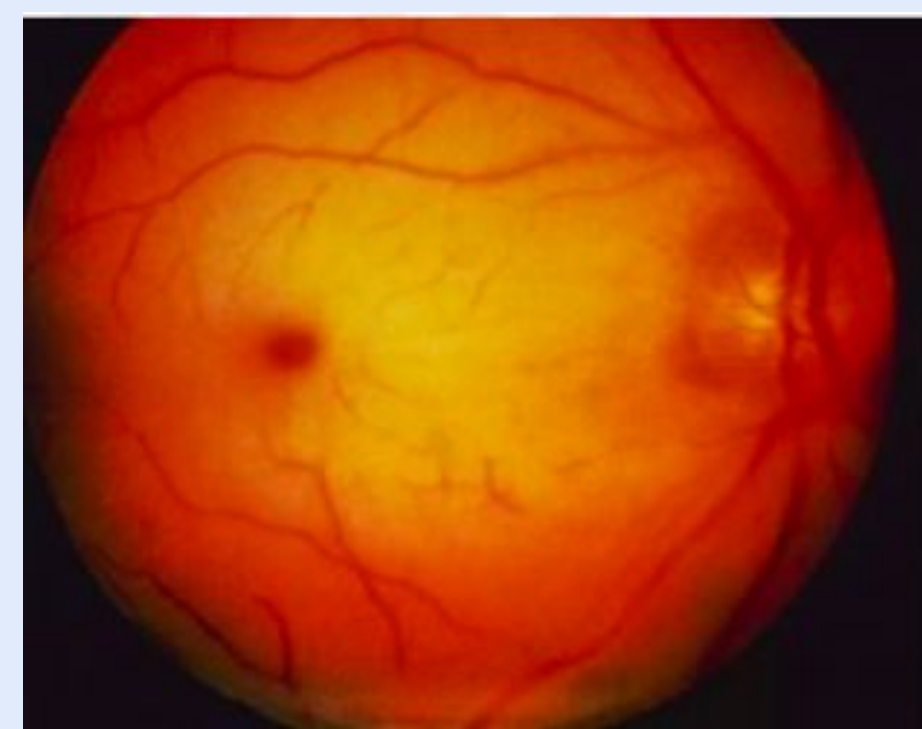
Opaque retina & attenuated artery > central retinal artery occlusion.



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



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.



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 **Check CVD** (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.



Branched retinal artery occlusion.

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.

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.

Temporal: nasal visual field.

Nasal: temporal visual field.

Treatment: should be directed at reducing associated macular edema by injecting anti-vascular endothelial growth factor agents "Anti-VEGF", CRVO is not true ophthalmic emergency.

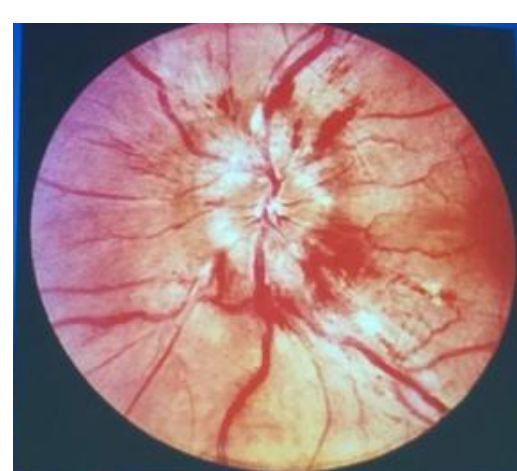
Visual prognosis depends on degree of associated retinal ischemia.

Optic Diseases; Optic neuritis:

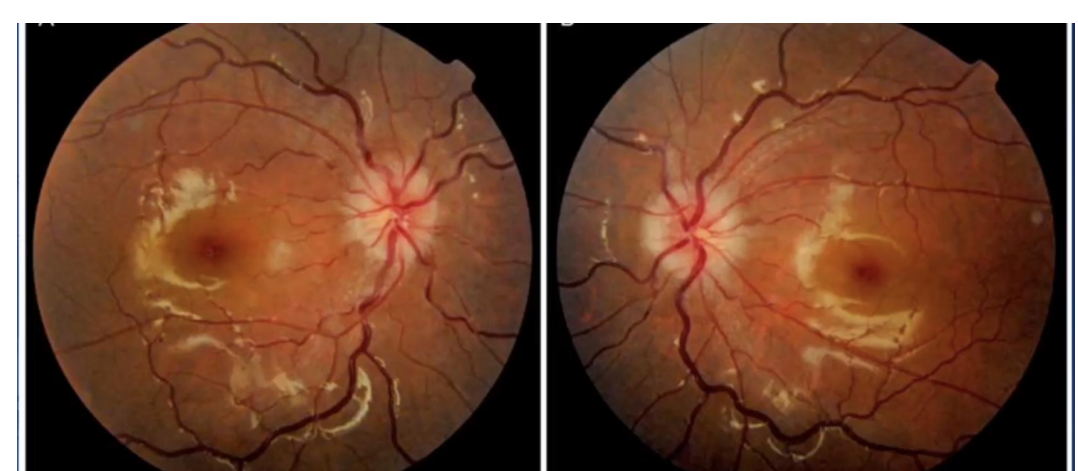
- Optic neuritis is an inflammation of the optic nerve and is usually idiopathic, but it may be associated with **multiple sclerosis** (as first clinical manifestation) in a significant number of cases.
- **The visual acuity is markedly reduced, and an afferent pupillary defect is present (IMP IN OSCE).**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



Swelling of the optic nerve head.



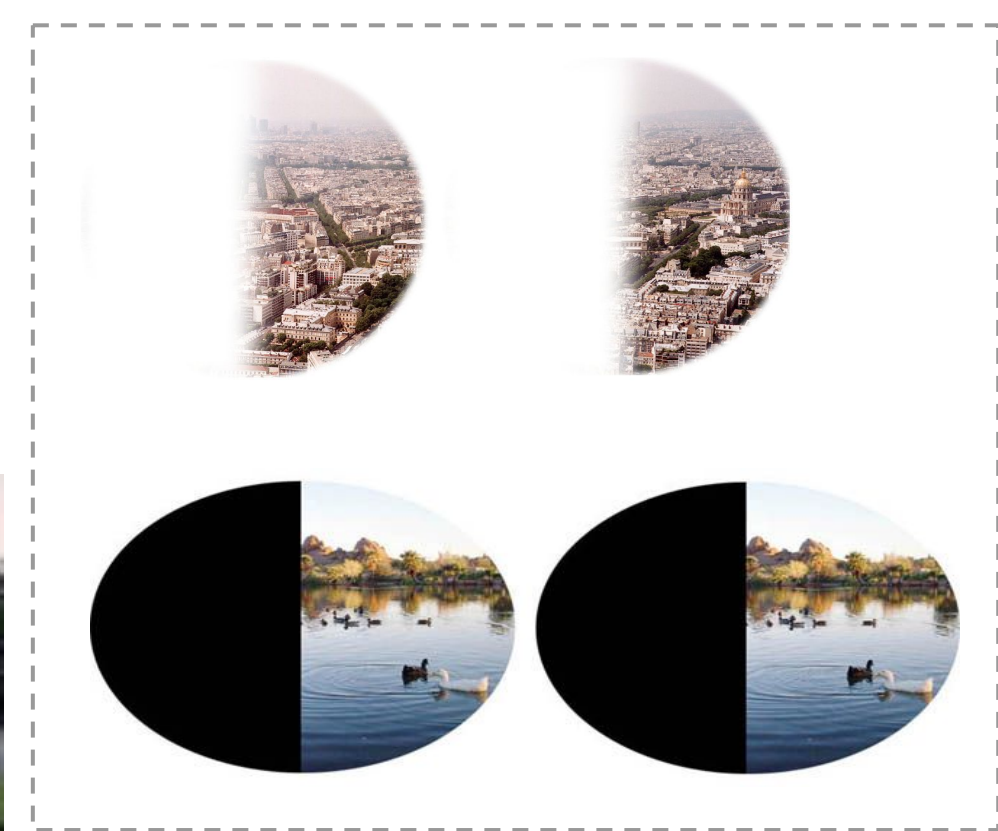
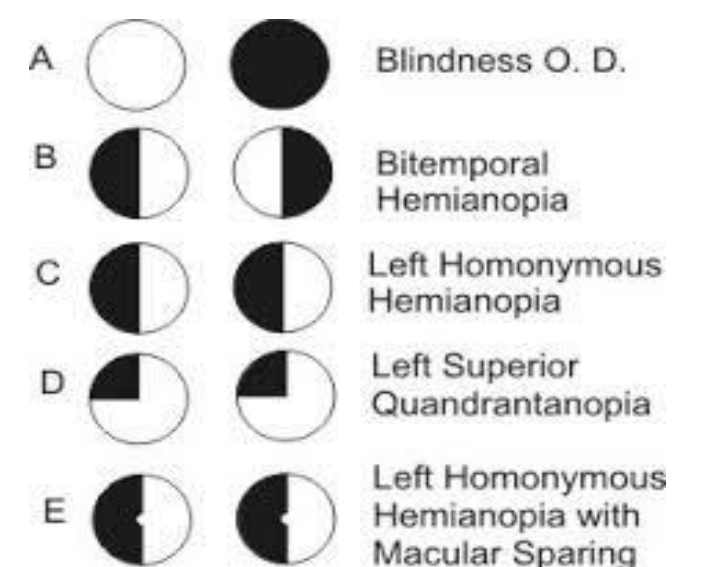
Bilateral Optic nerve swelling. it's **NOT** papilledema; papilledema is Bilateral swelling only due to increased ICP

	Extra information: difference between		
	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**
- Refer to neurology.
- Behind the optic chiasm.



Extra

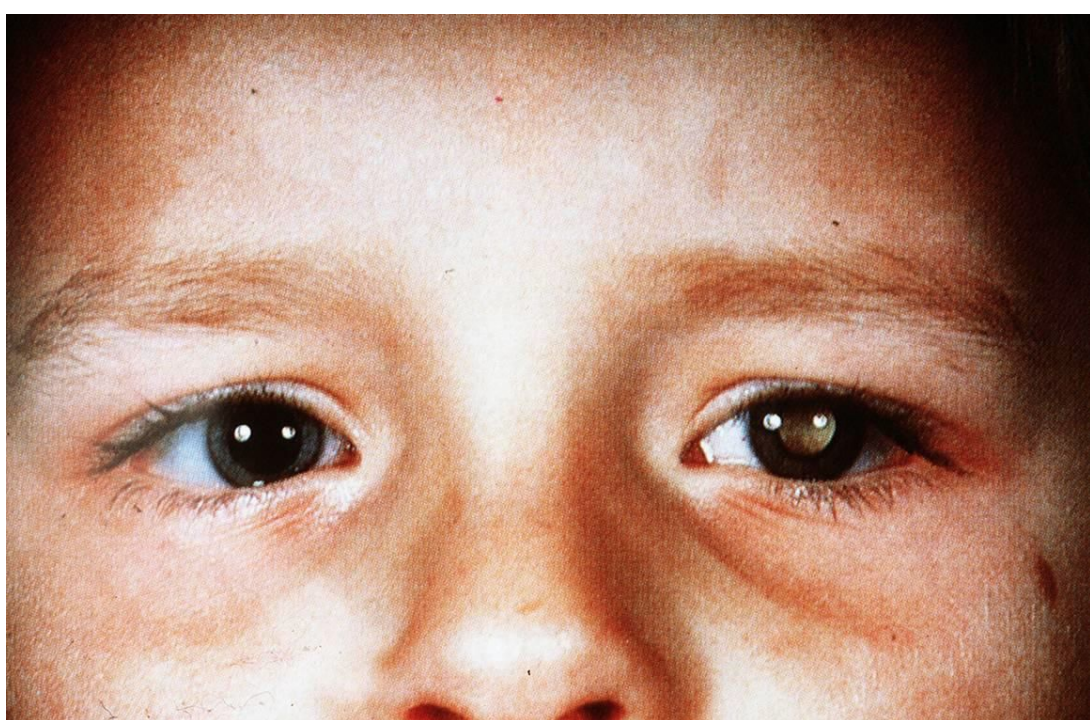
Cortical Blindness

- 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 pt has complete loss of vision).
- Poor vision, loss depends on which part of the cortex was affected.

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.

Pictures at the end of the lecture

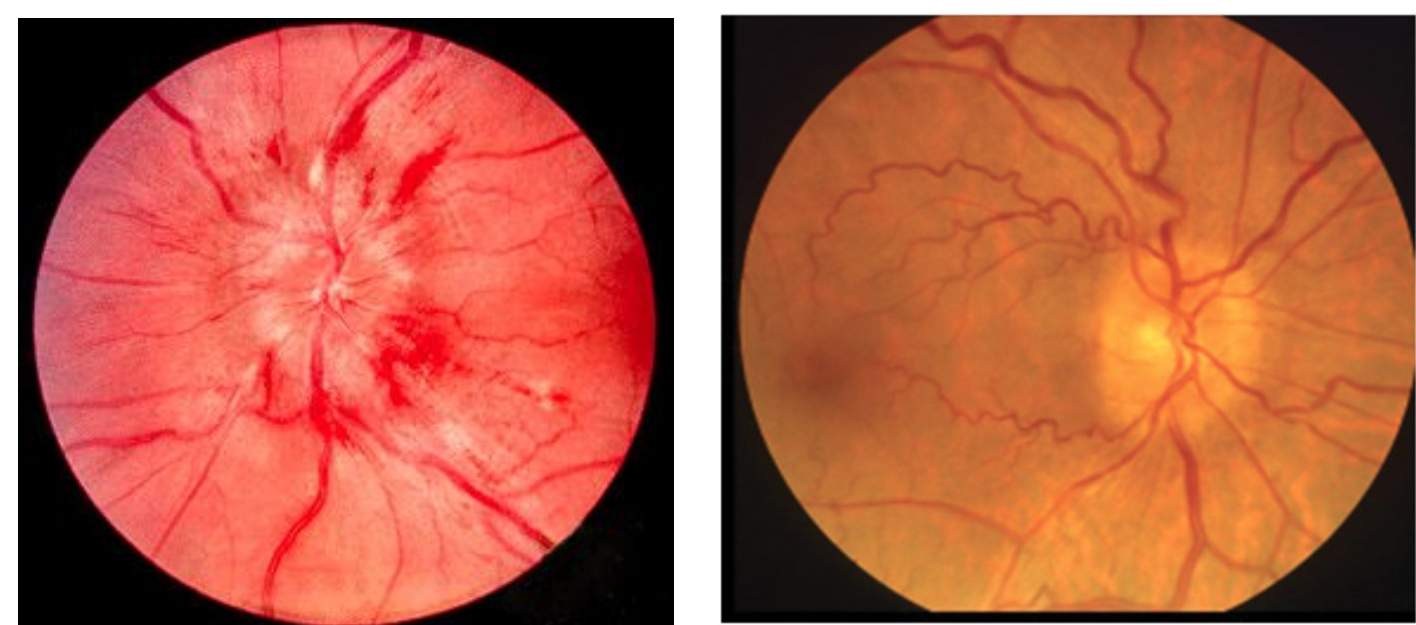


Qs IN EXAM: what is it called?

Leukocoria (in children)

what causes it?

- Retinoblastoma
 - Cataract
- Coat's disease



Optic nerve head swelling



Edema

Practice Questions

Q1. A diabetic patient came complaining of visual loss with ciliary injection and increased IOP what is the most likely diagnose ?

- A. Acute closed glaucoma.
- B. Chronic open glaucoma.
- C. Uveitis glaucoma
- D. Neovascular glaucoma.

Q2. Old man with history of gradual decrease in vision in the last few months, now presented with a cut red and painful eye with increased IOP. What will you see in examination?

- A. Swelling lens.
- B. Deep anterior chamber.
- C. Acute keratitis

Q3. A 30--year--old female presented to eye clinic with acute visual loss and pain on the right eye. Examination reveals a visual acuity of 6/36 in the right eye, 6/6 in the left eye, a central scotoma in the right eye with swollen optic disc and central scotoma on visual field. What is the most likely diagnosis?

- A. Compression of optic nerve
- B. Acute Glaucoma
- C. Optic neuritis
- D. Retinal vein occlusion

Q4. A 65--year--old male known cardiac patient. Presented to ER with acute painless loss of vision. On funduscopic examination, there is pale retina. Which on the following is the most likely diagnosis?

- A. Retinal artery occlusion
- B. Retinal detachment
- C. Optic neuritis
- D. Retinal vein occlusion

Q5. A 75--year--old male with history of jaw claudication, presented to ER with sudden loss of vision in one eye. Erythrocyte sedimentation rate was 100mm/hr. What is the most likely cause of visual loss?

- A. Ischemic optic neuropathy
- B. Central retinal artery occlusion
- C. Rhegmatogenous retinal detachment
- D. Central retinal vein occlusion

Answers

1.D 2.A 3.C 4.A 5.A