Ophthalmology 436

Acute Visual Loss

Objectives :

- Recognize situations requiring urgent ophthalmic care to prevent permanent visual loss.
- Properly screen and evaluate patients presenting with acute visual loss.
- Understand the pathophysiology and identify common causes of acute visual loss.

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[Color index: Important | Notes | Extra] Editing File

Introduction to acute visual loss

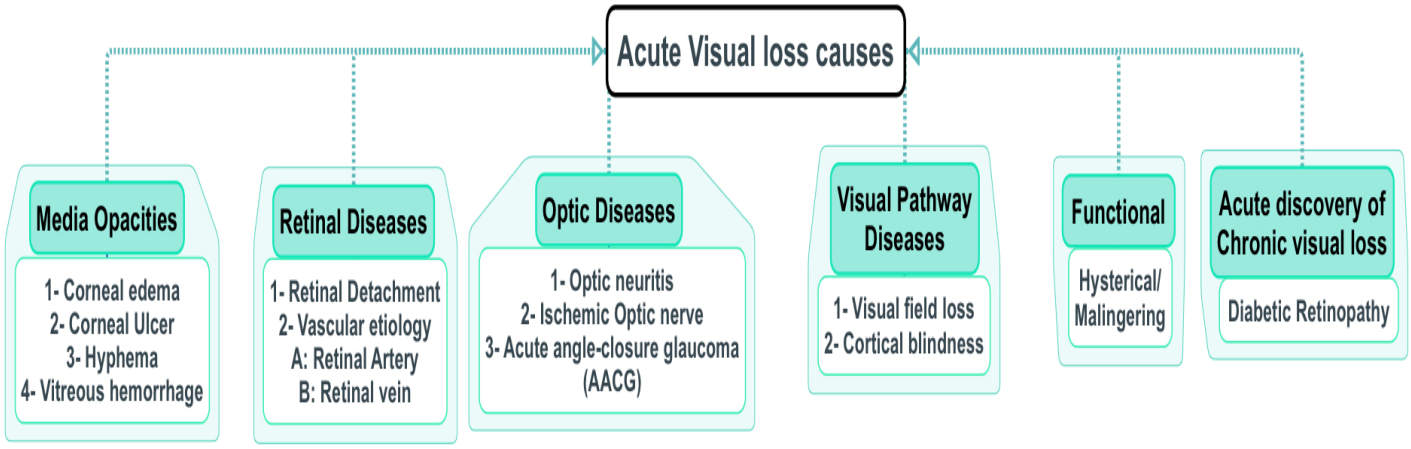
- **What is acute visual loss (AVL)? (disastrous and requires urgent actions)**

– Sudden onset of significant visual impairment or blindness. Loss of vision is usually considered acute if it develops within a few minutes to a couple of days.
– May affect one or both eyes | All OR part of visual field | Arise from pathology of any part of the visual pathway.

Etiology		
AVL classified by PAIN		**AVL classified by STRUCTURE**
Painful	**Painless**	**1. Media opacities:** something interferes with the passing 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. **2. Retinal disease:** improper absorption of light. **3. Optic nerve disease.** **4. Visual pathway or neurological disorders**: Stroke/tumor **5. Functional disorders** **6. Acute discovery of chronic visual loss:** usually **unilateral**
– **Acute (congestive) Glaucoma:** 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:** patient is always in pain. – **Keratitis**: infection or inflammation of cornea "very severe pain, more than uveitis". – **Hyphema (Traumatic):** can be asymptomatic unless if it's associated with other things.	– **Vitreous Hemorrhage:** it can be painful if it is traumatic. – **Retinal Detachment:** the patient may have it and not discover it until covering one eye – **Retinal vascular occlusions**:arteries/veins – **Optic neuritis:** sometimes eye movement may cause mild pain, but usually it is painless. – **Ischemic optic neuropathy** – **Cerebrovascular accident (CVA)** – **Functional**	**All of the above may cause mild, moderate, severe visual loss or total blindness.**

Clinical approach مهم الدكتور عصام قال القسم يفكر يجيب
هستوري في الأوسكي من محاضرة الكرونيك والأكيوت فيجوال لوس

History (Hx)	**Physical examination (P/E)**
– 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: Not 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. **– What is the patient's age and general medical condition?** ○ Young with no systemic disease:think about neurological problems like: Optic neuritis, retinal detachment or trauma. ○ Old with chronic medical condition: Vascular cause. ⋆ Acute glaucoma vs corneal abrasion. **– 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	**1. Visual acuity testing:** to see if the visual loss is mild, moderate, or severe. **2. 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. **3. Pupillary reactions (very important).** **4. External examination of the eye with a pen light:** we look at the eye in general to see if there's any trauma. **5. Biomicroscopic examination (Slit lamp examination)** **6. Ophthalmoscopy exam:** can exclude media opacity, we observe the red reflex, in normal people it is present and equal in both eyes. **7. Tonometry to measure the intraocular pressure**

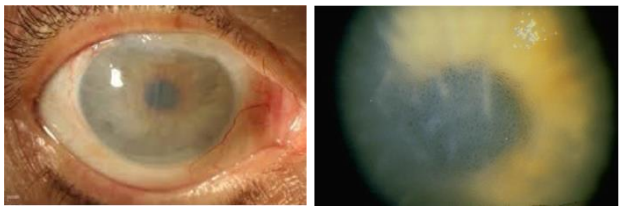


1) Media Opacities

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

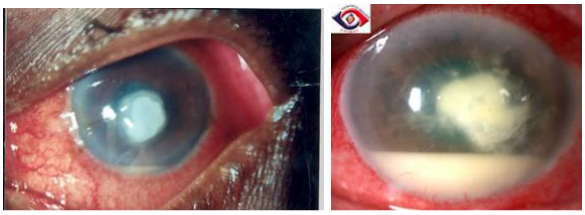
1. Corneal edema

– The cornea appears like **ground glass** rather than its normal clear appearance. (steamy cornea)
– The most common cause of corneal edema is **increased intraocular pressure** typically in **acute angle closure glaucoma** (this is almost always the presentation of corneal edema) so why does it cause edema? that is because 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 hypotony[1]
– Any acute infection of the cornea resulting in a corneal ulcer may **mimic** corneal edema.



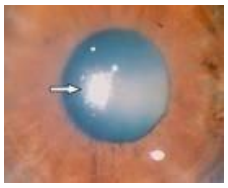
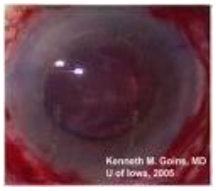
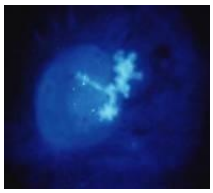
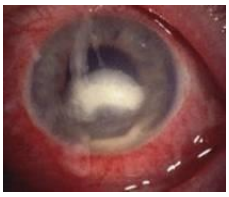
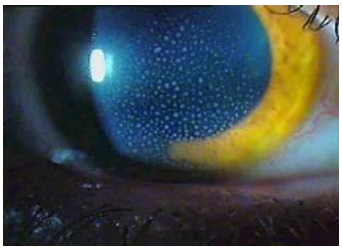
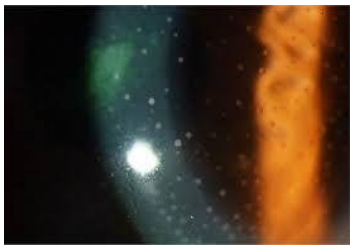
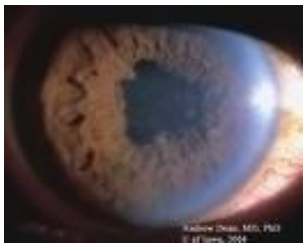
2. Corneal ulcer

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



[1] **Hypotony** is usually defined as an intraocular pressure (IOP) of 5 mm Hg or less.

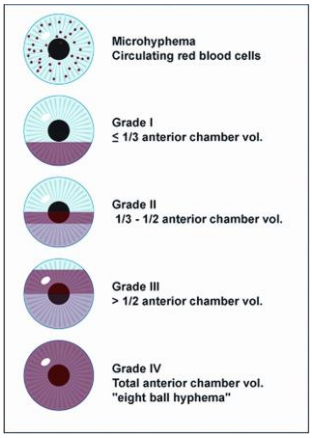
– Uveitis doesn't only cause visual impairment on the corneal side but also on the turbidity of the anterior chamber. In uveitis the inflammation leads to changes in aqueous humor contacts, usually there is a protein present in the anterior chamber and its concentration is 1% of that in the serum. In severe uveitis, the concentration is similar to the serum.
– Posterior synechiae[2] itself doesn't cause visual loss, but the sequences that happen after posterior synechiae.

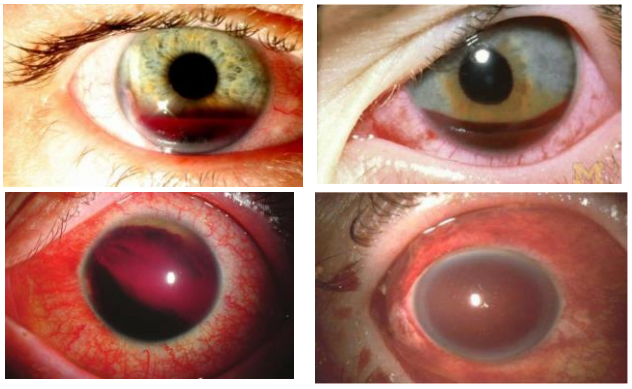
			
Abscess	**Edema**	**Viral herpetic herpes**	**1) Infection: abscess in the cornea with hypopyon[3]. 2) Congested eye.**
			

Uveitis: Mutton fat keratic precipitates appearance due to accumulation of WBC "mainly macrophages" on the corneal endothelium, resembles edema

3. Hyphema

– Hyphema is blood in the anterior chamber.
– The hyphema is a direct consequence of blunt trauma to a normal eye.However, it can occur with tumors, diabetes, intraocular surgery and chronic inflammation which all cause neovascularization.
– The most common cause is trauma. In case of trauma, it usually resolves spontaneously within 3 days (Bed Rest and minimize the activity to avoid repleading).
– 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.
– 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"





[2] **Synechiae** are adhesions that are formed between adjacent structures within the eye usually as a result of inflammation.
[3] A collection of inflammatory cells-puss- in the anterior chamber.

4. Vitreous hemorrhage (VH)

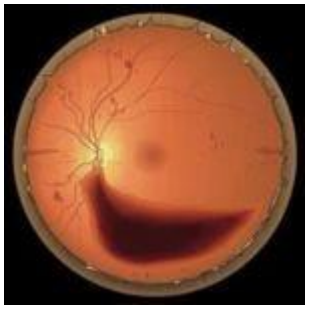
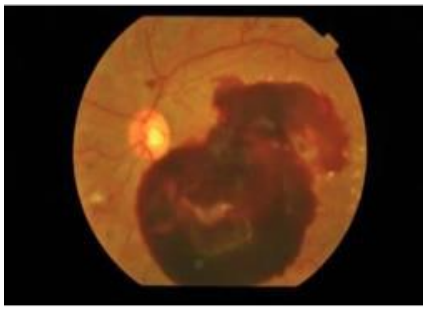
Mechanism

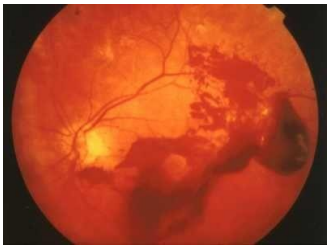
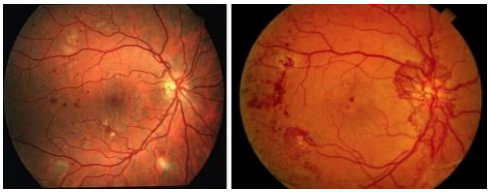
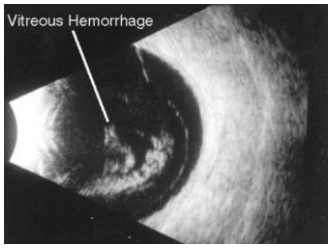
– Extravasation or leakage of blood into the areas in and around the vitreous humor of the eye.
– Any bleeding into vitreous cavity will reduce visual acuity. (because it's a media opacity: opacity in the cornea? Edema. Opacity in the vitreous? Blood. Opacity of lens? Cataract)
– **Can result from:** trauma, diabetic retinopathy (common cause in ksa), retinal vascular occlusion and acute posterior vitreous detachment and intraocular surgery. Many diseases can cause you VH, even TB can cause VH
– Rarely, can accompany subarachnoid hemorrhage. (Terson's syndrome)

In summary, mechanisms of Vitreous Hemorrhage:
A. Abnormal Vessels: DM (accounts for 30-50% of VH), Sickle cell retinopathy, etc
B. Rupture of normal vessels: Retinal tear, trauma, Terson's…
C. Blood from adjacent source: Macroaneurysm (0.6–7%) Age-related macular degeneration (0.6–4%)

Diagnosis

– **Not a diagnosis but rather a sign for many diseases.**
– **First step:** ophthalmoscope. If you cannot appreciate a RED REFLEX → **Second step:** B scan (ultrasound).
– If you cannot appreciate a red reflex with an **ophthalmoscope** and the lens appears clear, you should suspect VH!
– The diagnosis is confirmed with slit lamp examination through a dilated pupil. So you know the lens is definitely clear, further you stop seeing anything inside the vitreous cavity
– **B scan (ultrasound) is important.** (important to know the etiology, it tells you how thick the blood & distribution)

Nonproliferative Diabetic Retinopathy	Proliferative Diabetic	B scan
		

Treatment

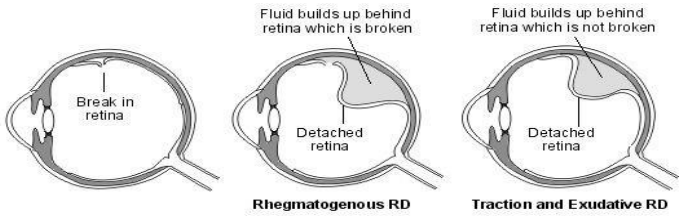
Bed rest and treatment of the underlying cause.

2) Retinal Diseases

1. Retinal detachment (RD)

– 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 normal retina, there is no actual connection or junction between them. It is a potential space, firm, and adherent.
– When the retina breaks, fluid come between the 2 layers and separates them.
– Retinal detachment is one of the painless causes of acute visual loss, and it is not an ocular emergency.
– 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	2. Traction RD	3. Exudative RD
– Most common – Rhegma = break. Tear in the retina allowing liquified vitreous to gain entry to the subretinal space and causing a progressive detachment.	– In Diabetes, SCA. – Here the the retina is detached but continuous with no tear. If it is pulled off by contracting fibrous tissue on the retinal surface.	– Due to inflammation, if we treat the underlying pathology the problem will be solved. (forget it. We treat systematically)
		– Picture: when a break happens, subretinal space allowed vitreous fluid to travel into the break and detach the retina (as a result of an exudative process)

Risk factors

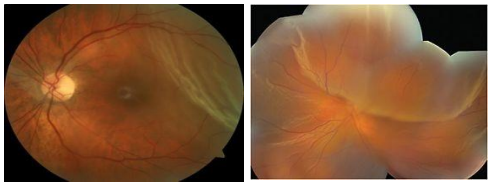
– Posterior Vitreous Detachment (PVD) the vitreous is attached to the eye at the optic head and ora serrata. Due to trauma, surgery, or spontaneous liquefaction "aging", the vitreous detaches and pull the retina and break it.
– Peripheral retinal degenerations. e.g. lattice degeneration, retinal tufts... etc.
– **High myopia.**
– Aphakia: (no lens. In the past they used to deal with cataract aggressively, traumatic surgeries). (bc its a sort of trauma, its abnormal, there should be a lens inside: pseudophakia > less risk of RD)
– History of: trauma, retinal detachment, also previous history of detachment in the other eye, Keratoconus.
– Family history,
– Exudative RD can happen in: renal biliary pts, Vogt–Koyanagi–Harada syndrome, abnormal liver functions, etc
– usually old age.

Signs & Symptoms

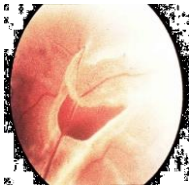
1. Prodromal symptoms: **flashes + floater**s (if you catch the pt here you can prevent RD. but patient almost never come at this point)
2. **Visual field loss- curtain-like** (from below: upper retina, and vice versa)
3. Sudden, painless loss of vision
4. Afferent pupillary defect.
– The diagnosis is confirmed by ophthalmoscopy through a dilated pupil, and retina appears elevated with folds and the choroid background behind the retina is indistinct.

Management

– RD does not need urgent management, unless if its **macula ON.**
– In 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).**
– Scleral buckle, cryotherapy, SRF drainage, Vitrectomy, AFX, endolaser, long acting tamponade (Gas, silicone oil)
(You don't need to know the treatment details)



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

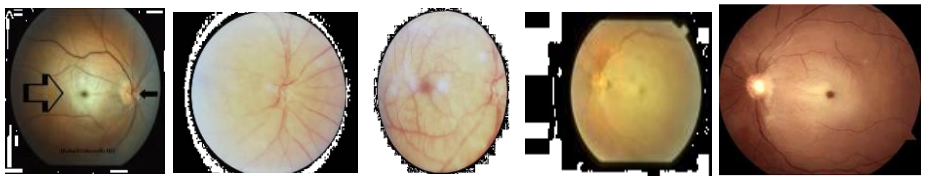


Horseshoe break:
f the break gets pulled more (by the vitreous) → can rupture a vessel → RD & hemorrhage

2. Retinal vascular occlusions

Artery

Central Retinal Artery occlusion



– A sudden, **painless** and often complete visual loss may indicate central retinal artery occlusion.
– Several hours after a central retinal artery occlusion, **the inner layer of the retina becomes opalescent (opaque, why? Bc of edema).**
– A cherry red spot is seen due to the pallor of the perifoveal retina in contrast to the normal color of the fovea. (in picture 1, there is an opaque retina with an edematous disc (small arrow) and then 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)
– A **chronic cherry red spot (big arrow)** is also a feature of the storage diseases such as Tay-Sachs disease and Niemann-Pick disease.
– There is no generally accepted acute management.

Causes of cherry red spot on retina: *mnemonic: **C**herry **T**ree **N**ever **G**row **T**all*
Central retinal artery occlusion | **T**ay-Sachs disease | **N**iemann-Pick disease | **G**aucher's disease | **T**rauma (Berlin edema)

Branch Artery occlusion

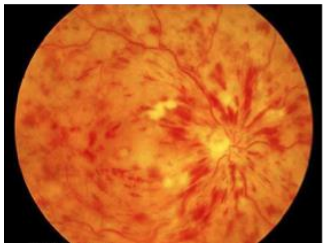
– If only a branch of the central retinal artery is occluded, vision is only partially lost. (refer for VF)
– This is more likely to be the result of an emboli and the source of the emboli should be sought.
– If the visual acuity is affected, attempts should be made to dislodge the emboli by ocular massage

Basically for both branch and central occlusion you try to cause vasodilation, you can try to make the pt rebreathe through a bag so CO2 > vasodilation, but it does not always work > no treatment
***Picture:** notice how white 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)
Upper retina: lower. Lower retina: Upper Temporal: nasal. Nasal: temporal

Vein

Cental retinal vein occlusion

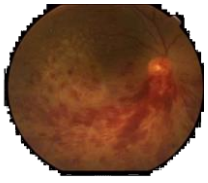
– Ophthalmoscopes picture of disc swelling, venous engorgement and tortuosity, **cotton wool spots (these are more white than exudates, and they are an infarctions of the nerve fiber layer) and diffuse retinal hemorrhages** like blood and thunder (flame shaped).
– Loss of vision may be severe. Bc it causes macular edema.
– 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 depend on degree of associated retinal ischemia.



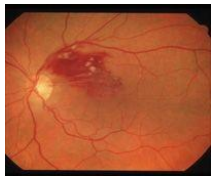
Types

Ischemic	Non ischemic
– It is a disaster that will lead to Permanent visual loss. – It will cause neovascularization which leads to "90-days glaucoma". Explanation: Ischemia causes reduction of oxygen supply => leading to VEGF production " which promotes new blood vessels formation" => Ultimately leads to formation of fibrovascular membranes => The fibrovascular membranes accompany neovascularization and block the trabecular meshwork => Causing glaucoma "Neovascular glaucoma", typically named 90-days glaucoma because it usually takes around 90 days to occur after the onset.	May resolve fully (benign). However, in 50% of the cases it may turn to ischemic. Non-ischemic if there is no hemorrhage the patient will be unaware of it.
Relative afferent pupillary defect **(RAPD +)**	**Intact pupillary reflex**

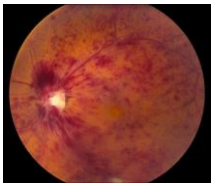
The way to differentiate between the two types is by visual acuity, and pupillary reflex, in ischemic type there must be an afferent defect.



Hemiretinal vein occlusion
/engorged veins/ cotton wool spots/ disc edema



Branch vein occlusion
Veins appear thicker than arteries.



Central vein occlusion
(flame shaped hemorrhage in all quadrants) the disc is not swollen here

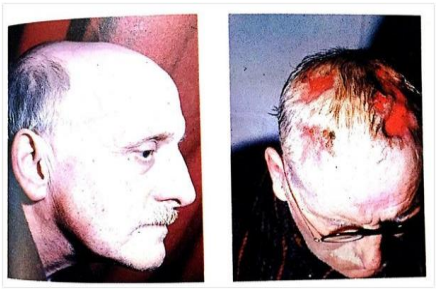
3) Optic Diseases

1. Optic neuritis:

– Optic neuritis is an inflammation of the optic nerve and It is usually idiopathic but may associated with **multiple sclerosis** (as first clinical manifestation.) in a number of cases.
– Visual acuity is markedly reduced **and an afferent pupillary defect is present. (+)**
– 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. (so the goal of management is to prevent recurrence)
– 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)

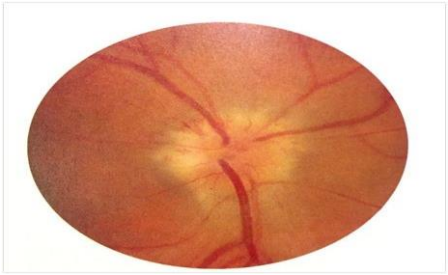
	Extra information: difference between		
	Papilledema	**Papillitis**	**Retrobulbar neuritis**
Definition	Swelling of optic nerve head due to increased ICP	Inflammation or infarction of optic nerve head	Inflammation of orbital portion of optic nerve
Uni/bilateral	Bilateral	Unilateral	Unilateral
Vision 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

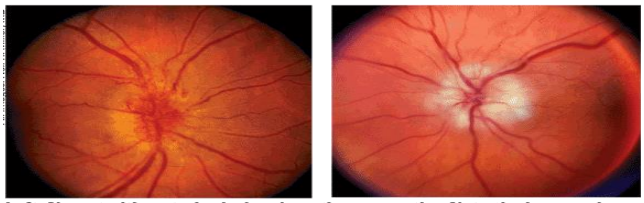
2. Ischemic optic neuropathy



– Anterior ischemic optic neuropathy [AION] is a relatively common cause of severe visual loss.
– The basic lesion is a segmental or generalized infarction of the anterior part of the optic nerve caused by occlusion of the short posterior ciliary arteries.
– Irreversible **painless** visual loss.
– It has two types: Arteritic and non arteritic.

Arteric	Non arteritic
– The loss of vision is due to inflammation of the arteries. – Caused by Giant-cell arteritis "Temporal arteritis". – Causes headache and gangrene of the scalp. On physical examination there is tenderness over the temporal area. – Investigation: ESR and C-reactive protein "if both are elevated => highly suggestive". The gold standard is biopsy. – Treatment is possible if you catch the patient early => Give steroids.	– Due to non-inflammatory disease of the small blood vessels. – Common cause is atherosclerosis. – There is no treatment.



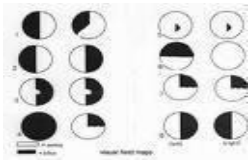


Left: Nonarteritic anterior ischemic optic neuropathy. Note the hyperemic swelling of the optic disc associated with the flame-shaped peripapillary hemorrhage. Right: Arteritic anterior ischemic optic neuropathy. Note the pallid swelling of the optic disc and a peripapillary cotton-wool spot.

3. Acute Angle Closure Glaucoma Discussed in Chronic Visual loss lecture as a part of Glaucoma

4) Visual Pathway loss

1. Homonymous hemianopia



– Loss of vision on one side of both visual fields
– May result from occlusion of one of **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.** Refer to neurology
– Behind the optic chiasm.

2.Cortical Blindness

– A rare bilateral extensive damage to the cortical visual pathways results in **complete loss of Vision**.
– This condition is referred to as cortical, central or cerebral blindness.
– As the pathways serving the pupillary light reflex separate from those carrying visual information at the level of the optic tracts, a patient who is cortically blind has normal pupillary reactions. Thus a patient with normal fundus examination along with normal pupillary reactions, most likely has cortical blindness.
– Poor vision, loss depends on which part of the cortex was affected.

5) Functional visual loss

– Describes vision loss due to hysterical or malingering reasons. ie: not explained by organic basis.
– A patient may report complete blindness in one eye and normal vision in the other eye, and no relative afferent pupillary defect (RAPD)
– Various techniques exist to confirm functional visual loss.

6) Acute discovery of chronic visual loss

Diabetic Retinopathy

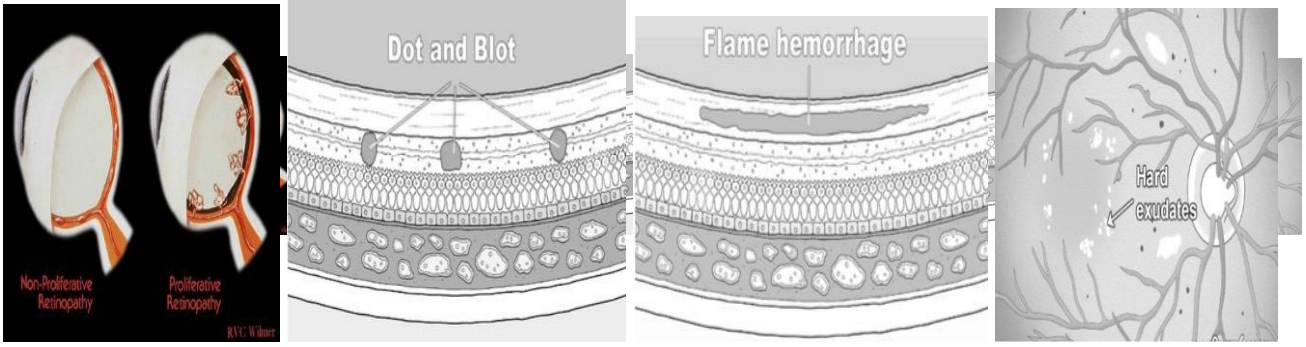
– Diabetic retinopathy is the term used to describe the retinal damage causing this visual loss. Diabetics have a high prevalence of retinopathy, and one out of every five patients with newly diagnosed diabetes will also show signs of retinopathy on exam.

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.

– the retina begins to show early signs of ischemia. Cotton-wool spots, indicate ischemia/infarction of the superficial retinal nerve fibers. As vessel damage progresses, you can also see beading of the larger retinal veins and other vascular anomalies.



– **Retinal detachment in DM: i**n the areas where the neovascularization happens, fibroproliferation ensues and pulls on the retina.

-Complications: visual field loss (tunnel vision)

Questions

What are some common causes of acute visual loss based on patient's age ? (from Dr. Almousa slides)

1. Wet age-related macular degeneration.
2. Commotio retinae[6]
3. Rupture globe.
4. Orbital cellulitis.

MCQ: A 69-year-old woman presents with acute onset of ocular pain, decreased vision, and halos around lights in the right eye associated with nausea and vomiting. The most likely diagnosis is:

a. Primary open-angle glaucoma.
b. Lens induced glaucoma.
c. Pigmentary glaucoma.
d. Acute primary angle-closure glaucoma.

Answer: d

MCQ: A 30 -year-old woman presents with sudden vision loss of the right eye and mild pain on upgaze movement. Examination reveals that vision is 20/50 on the right and 20/20 on the left. There is a +RAPD on the right and a Visual field testing showed an inferior altitudinal defect on the same side. The left side is normal. Optic discs and fundi are normal in both eyes. What is the most likely diagnosis?

a. Branch retinal vein occlusion.
b. Anterior ischemic optic neuropathy.
c. Retrobulbar optic neuritis.
d. Compressive optic neuropathy.

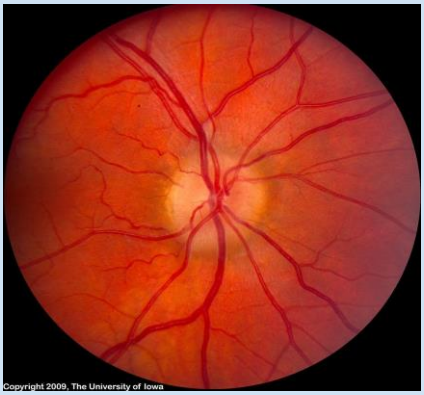
Answer: c

[6]The term describes the damage to the outer retinal layers caused by shock waves that traverse the eye from the site of impact following blunt trauma

Identify Pictures

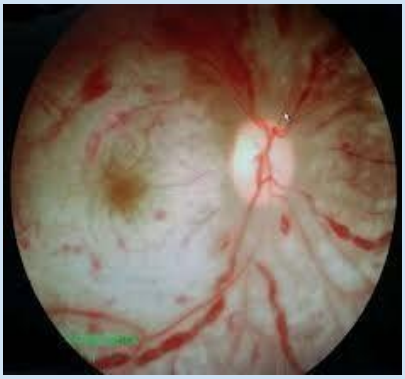
– **What is this?**
Leukocoria in a child

– **What could it be?**
– **DDx for peds leukocoria can be summed up in PREDICT**
Persistent hyperplastic primary vitreous
Retinoblastoma / **R**etinopathy of prematurity
Endophthalmitis
Dysplasia of the retina
Inflammatory cyclitic membrane
Congenital cataract / **C**oat's disease
Toxocariasis

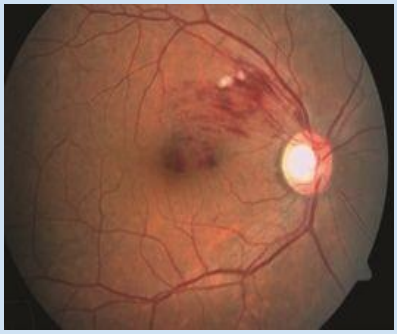


– **What is this?**
Disc edema

– **If it's bilateral it's called?**
Papilledema



– Concurrent Central Retinal Artery and Vein Occlusion
– You can see a pale retina, a cherry red spot (artery)
– Cotton wool spots, flame shaped hemorrhage (vein) and if you follow the artery you can see points of occlusion



– Branch retinal vein occlusion (the whitish spots are cotton wool spots)