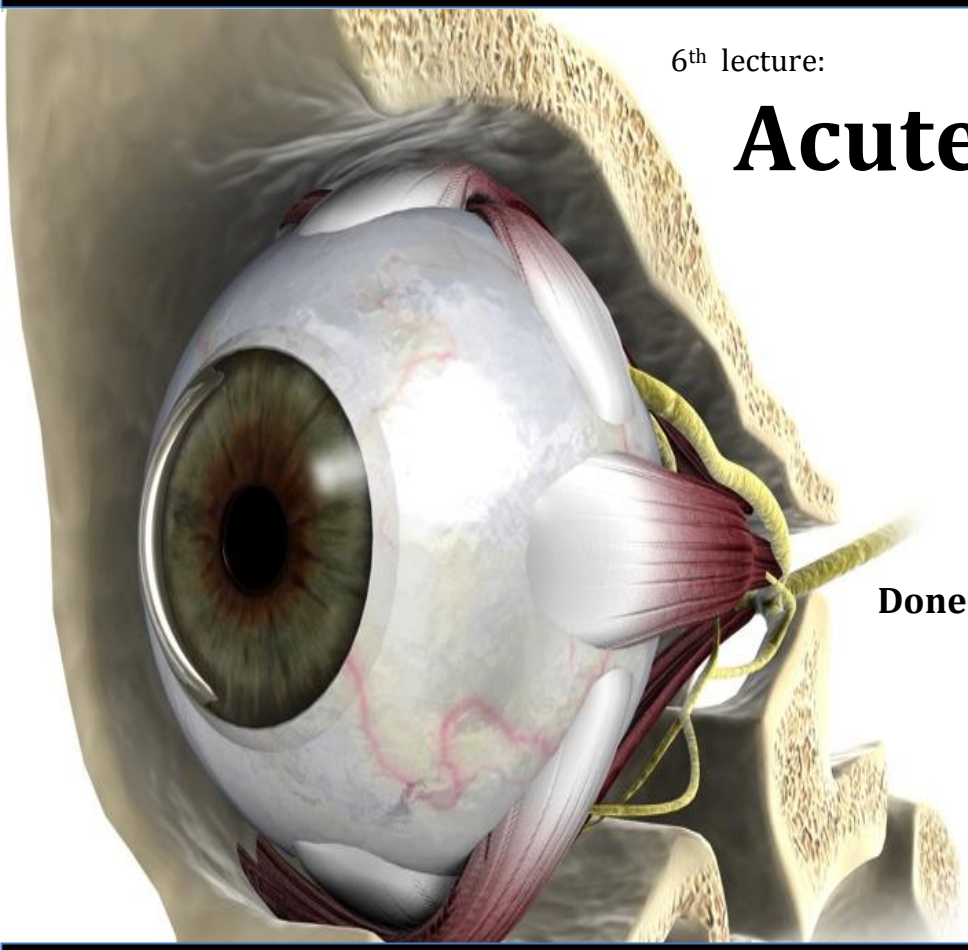
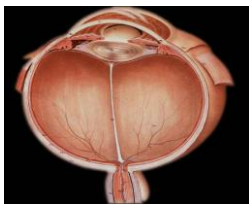
430 Ophthalmology Team



6th lecture:

Acute Visual Loss



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The slides were provided by doctor (Samir AlMansouri, MD)
Other resources: (team 429 and ophthalmology lecture notes 11th edition)
Important Notes in red
Copied slides in blacK.
my notes (my understanding or other resource) in blue
doctor explanation notes in green
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

- **Definition of Acute Visual Loss:**
  - Sudden onset of blindness.
  - It is a disaster for most people and you should be able to evaluate such a patient and be able to recognize situations requiring urgent action.
  - Chronic visual loss ➔slowly progress
  - Acute visual loss➔sudden (acute means seconds or minutes to few days)
    Acute visual loss (grades) not necessary mean complete loss.

- **Causes of Acute Visual Loss:**
  - Can be classified as painful and painless causes :

Painless	**Painful**
Vitreous hem	Keratitis – inflmation of cornea
RD seconds	Acute Angle Closure glaucoma
Retinal vascular Occlusions seconds	Closure angle glaucoma➔acute Open angle glaucoma➔chronic
Optic neuritis +	Uveitis
Ischemic optic neuropathy	
CVA	
Functional	

Or can be classified as : (doctor recommend revising the eye anatomy)

1. **Media opacities** . (cornea – equas champer –lens – vetrous)
2. **Retinal disease.**
3. **Optic nerve disease**. central causes
4. Visual pathway disorders.
5. Functional disorders.
6. Acute discovery of chronic visual loss.

- **History :**

The history questions to be asked of a patient of sudden visual loss include:

1. Is the visual loss transient, persistent, or progressive?
2. Is the visual loss monocular or binocular?
3. Did the visual loss occur suddenly or it developed over hours, days or weeks?
4. What is the patient's age and general medical condition?
5. Did the patient have normal vision in the past and when was vision last tested
6. Was pain associated with the visual loss?

- **Examination:**
  - Visual acuity testing
  - Confrontation visual fields. Compare patient with yourself , occlude the opposite eyes, move something in between. It is helpful even if it is not accurate.
  - Pupillary reactions. Afferent➔ optic nerve. Efferent➔ occulomotor
  - Ophthalmoscopy. visualize the fundus , direct and indirect ophthalmoscope, indirect is with more light can see more.
  - External examination of the eye with a pen light
  - Tonometry to measure the intraocular pressure

- **Media opacities** :

Easy fast way to examine medial opacity is RED REFLEX .

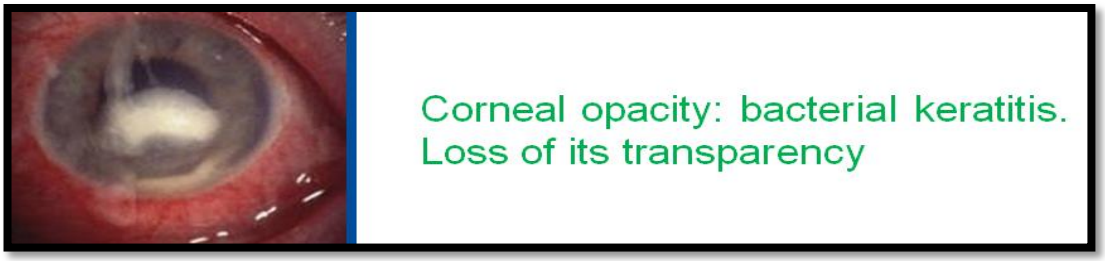
1- CORNEAL Oedema:

The cornea appears like a ground glass rather than its normal clear appearance.
The most common cause of corneal edema is **increased intraocular pressure** and occurs typically in **angle closure glaucoma**.

2- Corneal Infection {Keratitis=(inflammation of the Cornea)**}**

Any acute infection of the cornea by a cornealulcer may mimic corneal edema.
Most common cause of corneal infection is bacterial and viral . Fungal slowly in progress.

In acute inflammation there are :
1-infiltrate:(white exudates on cornea)
2-edema:(water, the cornea become thicker)
We look to those When we want to see the prognosis after treatment ,
usually infiltration is cleared and the cornea thickness become normal.



Herpes simplex keratitis Treated by acyclovir and(26:45 min sorry!)

3- CORNEAL Hyphema:

Hemorrhage lead to acute visual loss, it can be from posterior segment (vitreous hemorrhage) or anterior segment (anterior chamber: 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.
Also , in proliferative diabetic retinopathy they may have vessels on the iris (throbiosis iridis) abnormal fragile vessels can break and cause hemorrhage ➔acute visual loss.

4- Vitreous Hemorrhage:

It is Not a diagnosis rather a sign of many diseases.

Any bleeding into vitreous will also reduce the visual acuity. after trauma, seen in Diabetics or after a retinal vein occlusion and it may also accompany subarachnoid hemorrhage. Also can be due to retinal detachment with tear (rhegmatogenous type) but it is rare.

If you cannot appreciate a red reflex with an ophthalmoscope and the lens appears clear, you should suspect a vitreous hemorrhage. The diagnosis is confirmed with slit lamp examination through a dilated pupil. B scan is important.

5- Uveitis :

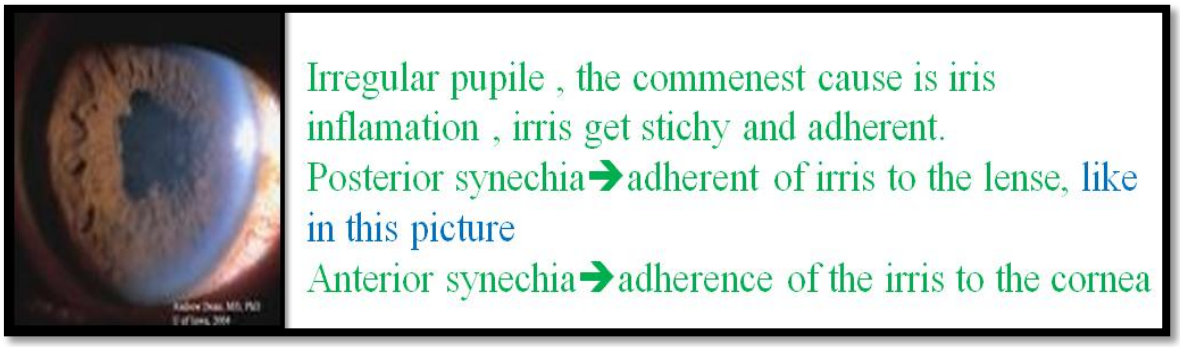
It is inflammation of uveal tissues (the iris, cilliary body and choroid) . it is painful.

Can be classified anatomically :

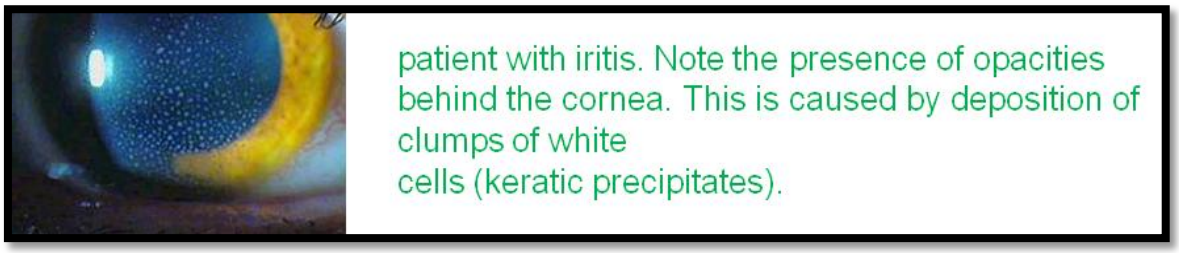
1-inflamation of the iris called (iritis) or **(anterior uveitis).**

2-inflamation of cilliary body called cyclitis , can associated with veterous inflammation (virtitis) and pars planitis as a group termed ➔**(intermediate uveitis).** (iridocyclitis)➔inflammation of iris+ ciliary body.

3-inflamation of posterior uvea is termed **(posterior uvitis)** and may involve the coroid (coroiditis) the retina (retinitis) or both (chorioretenitis).



Irregular pupile , the commenest cause is iris inflamation , irris get stichy and adherent.
Posterior synechia➔adherent of irris to the lense, like in this picture
Anterior synechia➔adherence of the irris to the cornea



patient with iritis. Note the presence of opacities behind the cornea. This is caused by deposition of clumps of white
cells (keratic precipitates).

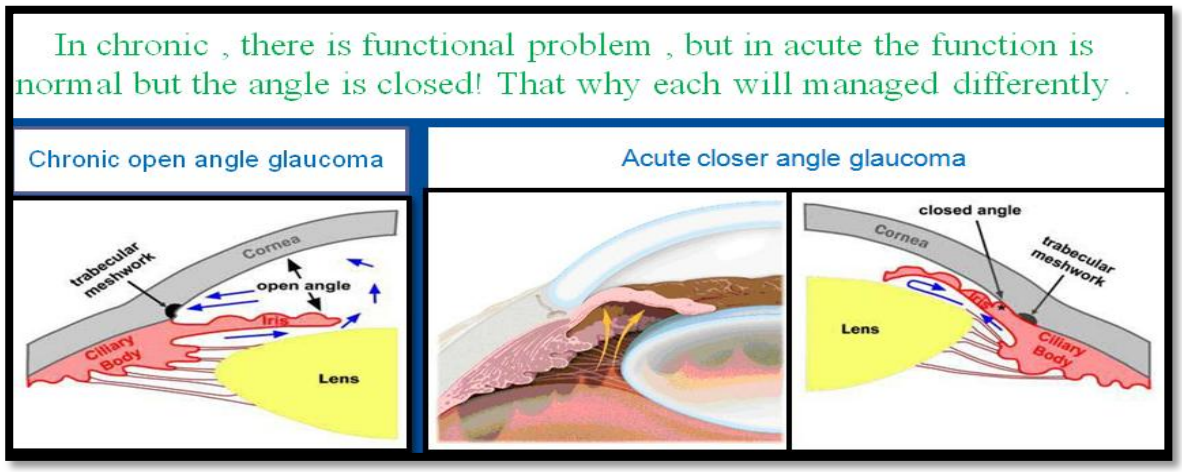
6- Acute Angle Closure Glaucoma:

Close glaucoma present with pain , on examination :

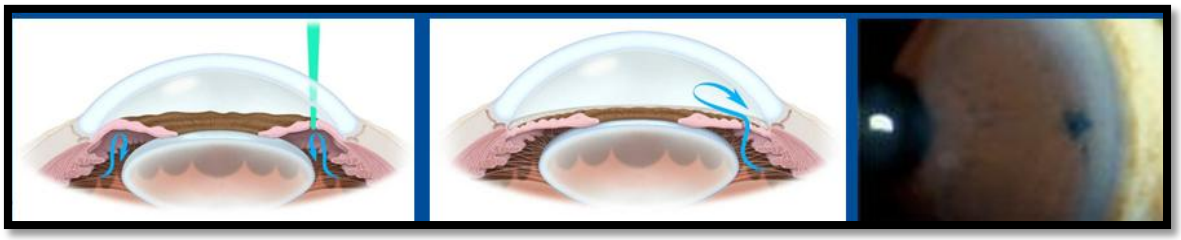
Corneal edema , the pupil is dilated and fixed , ciliary injection (congestion , redness of the eye but it is just around the limbs .)

In angle closer glaucoma the presser may reach over 40 , it is painful ,
it may reach up to 60-70 , very high presser.

Which is more serious : acute or chronic glaucoma??
The chronic is more serious , because patient does not aware of it ,
no symptoms no pain so ➔ patient does not present with it early , he\she will remain with it till the vision turbid or loss. But in acute (close angle glaucoma)
it is very painful , patient will jump to hospital seeking for management .



Management of acute closure angle glaucoma:
Laser YAG PI (peripheral iridectomy)
Make an Opening at the peripheral of the iris , will allow the aqueous that accumulated behind the iris to come immediately to the anterior champer then drained.



**before we have laser(before IP), we used to do surgical iridectomy (cutting part of the tissue to make a hole)

management should be done early, don't leave it as days. Otherwise there will be :

1- **Adhesions (synechia):** that **synechia** will occlude the angle permanently , then even if you do PI , it will not help.
2- **Nerve fibers affected**. Increase IOP➔pushing all the tissue and the optic nerve head with the weakest area on it (lamina criprosa: which is like a mish work of collagenuos fibers through which the nerve fibers are passing) that high presser will push all that out➔optic nerve will look cupping and fibers will affected➔patient can loss vision within few days.
(more general information about laser at 47:19)

- Aims of Acute ACG management :
Decrease IOP and Prevent future attacks in OU

(OU=Both eyes, OS=left eye, OD=right eye). Usually PI done in the other eye as a protection.

Retinal Detachment (RD):

It is retinal splitting. And it happens between 2 layers, the neurosensory retina and retinal pigmented epithelium. In normal retina there is no actually connection or junction between them. It is potential space. it is firm and adherent. When retina gets break, fluid come between the 2 layers and separate them.

- **Symptoms:**
  - Prodromal
    a. Flashes : because of retinal stimulation.
    b. Floaters: patient has break in retina. So, retinal particles will float in vitreous in front of retina and the patient will see them as floaters.
    ** flashes and floaters are worning sign or early sign of RD.
    after them there will be :
  - **VF** (visual failed) **loss** – curtain-like.
  - Sudden, painless **loss of vision**. (means RD involving the macula)

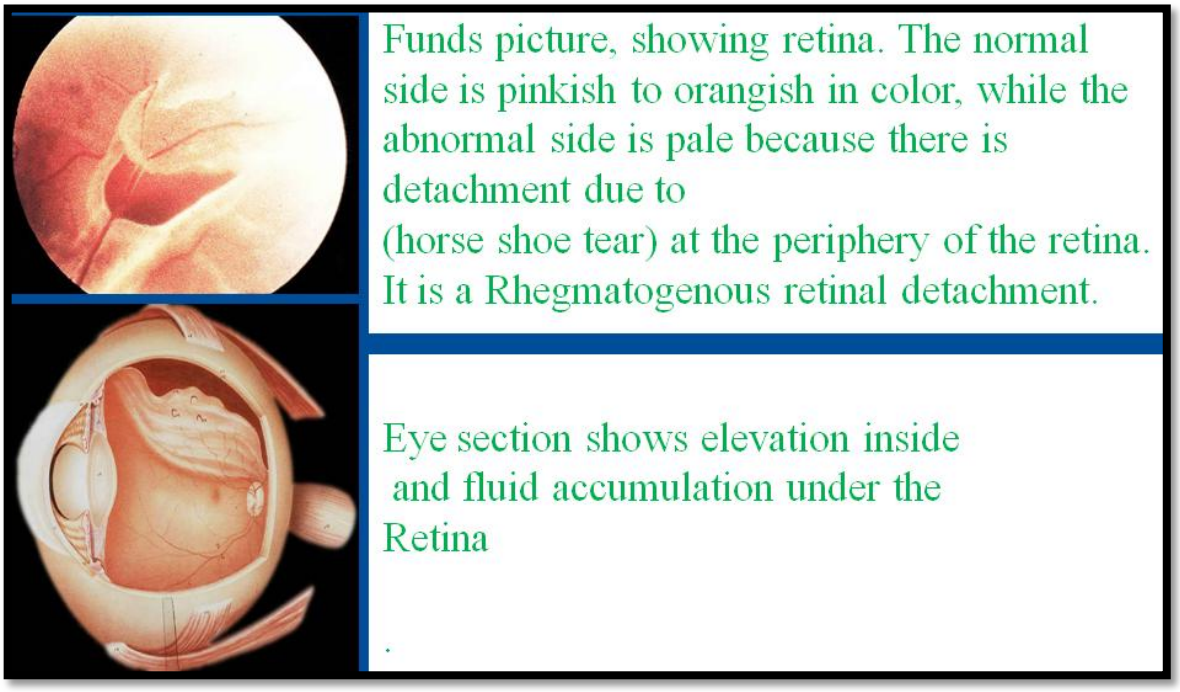
- **Types of RD :**
  1. Rhegmatogenous RD : related to break in retina either due to degeneration or trauma-more common- , the fluid comes under the retina causing detachment.
  2. Traction RD: like in diabetic retnopathy , the fibrovascular membrane contacts and pulls the retina. (will explain it again in diabetic retinopathy later)
  3. Exudative RD: accumulation of fluid under the retina such in choroid diseases.

- **RD Risk Factors:**
  - **Peripheral retinal degenerations, e.g. lattice degeneration**, retinal tufts…etc.
  - High myopia: the retina is stretched, high risk of RD, especially in trauma.
  - Aphakia: no lense, more space for vitreous, it will move more.
  - Trauma.
  - H/O RD: or even family history, also some syndromes such: marfan syndrome.

- **Management OF RD :**
  The aims of management are: 1-drain the fluids under the retina. 2-close the hole.

  - RD is an urgent condition. Especially if the macula is not detached.
  - Needs emergency surgery. Within 48hr not more.
    Early management➔good prognosis.
    Late management, even if retina back➔ retinal function less
  - Scleral buckle, cryotherapy=cold, cause inflammation➔induce adhesion and close the hole. , SRF(sub-retinal fluid) drainage.
  - Vitrectomy, AFX(air fluid exchange), endolaser, long-acting tamponade=to allow adhesion (Gas, Silicone oil) then remove it later.

    Example of RD and its management:



Mangment : the aim of sugury is to drain the fluid . to allow the retina to go back . then to close the hole (the horse shoe tear)to close the detachment and that done by 2 ways: Either by internal fixation (gas or fluid exchange) to push the retina then do laser or cryotherapy .
or by external fixation by doing buckle from outside to push the sclera opposite the retina then make laser at the margins of the break.

The laser will cause adhesion between neurosessory retina and retinal pigmented epithelium.

❖ Diabetic Retinopathy :

Diabetes is a common disease , the statistic studies shows that its percentage in KSA increasing , (you can find the percentages at 0:35s) . diabetic retinopathy most common finding in diabetic patients. Diabetic retinopathy become one of major causes of visual loss, diabetic retinopathy per say is a chronic cause of visual loss , but also can cause acute visual loss either due to Bleeding or Traction retinal detachment .

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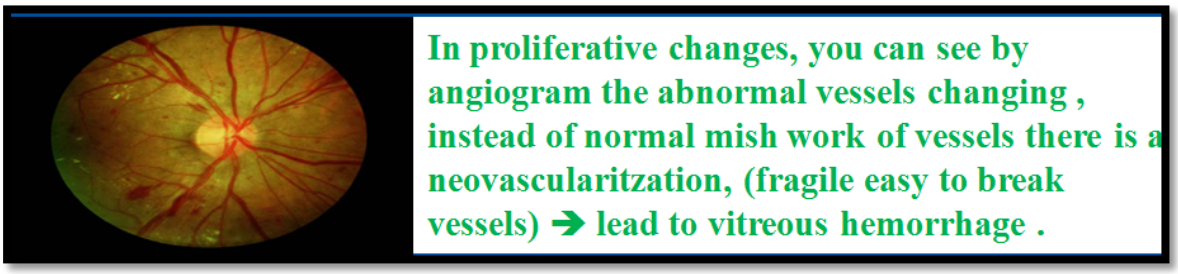
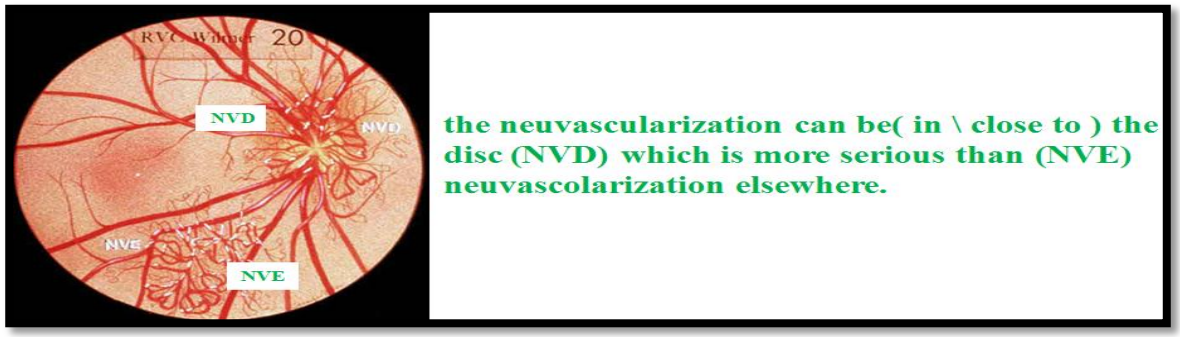
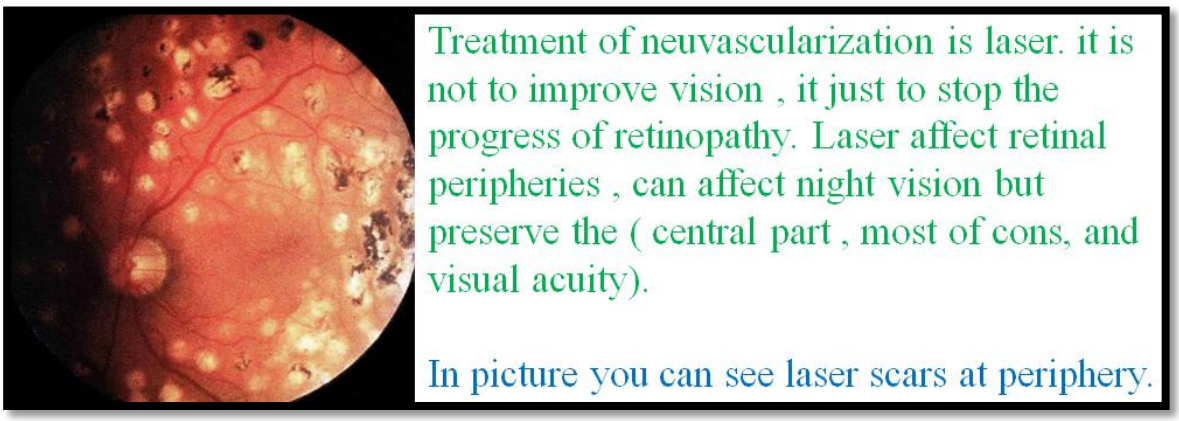


In proliferative changes, you can see by angiogram the abnormal vessels changing , instead of normal mish work of vessels there is a neovascularitzation, (fragile easy to break vessels) ➔ lead to vitreous hemorrhage .

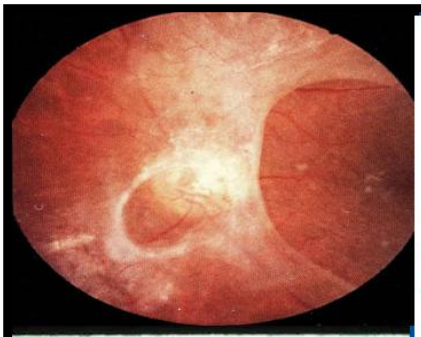


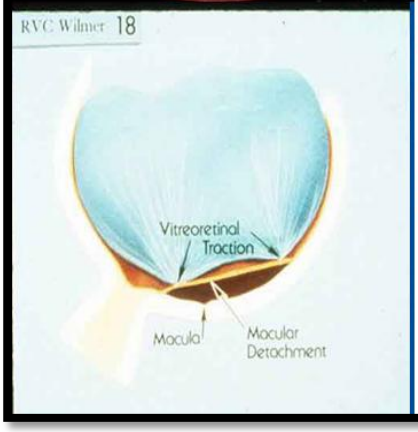
the neuvascularization can be(in \ close to) the disc (NVD) which is more serious than (NVE) neuvascolarization elsewhere.



Treatment of neuvascularization is laser. it is not to improve vision , it just to stop the progress of retinopathy. Laser affect retinal peripheries , can affect night vision but preserve the (central part , most of cons, and visual acuity).

In picture you can see laser scars at periphery.





Neuvascularization can be associated with fibrosis . (fibro-vascular membrane) on the retina when the membrane contract it will pull the retina and detach it ➔ (tractional detachment). to manage that we need surgical intervention , we have to go in and separate the membrane to release the traction (sometimes we can separate it but not always)

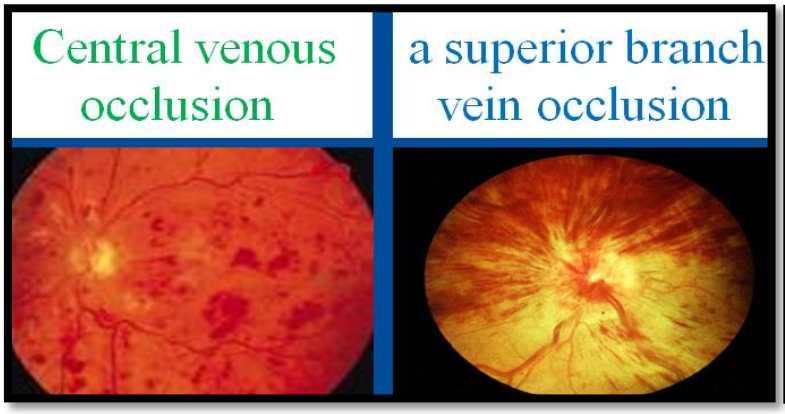
At the picture below you can see Detachment of the macula , that's why (NVD) more serious than (NVE).

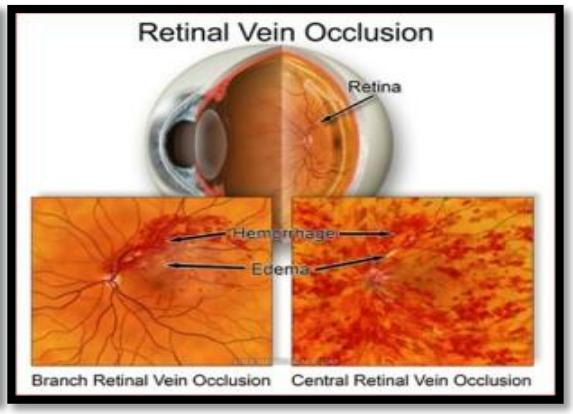
❖ Retinal vascular occlusion:

1. Retinal vein occlusion :

ophthalmoscopes picture of disc swelling, venous engorgement, cotton wool spots and diffuse retinal hemorrhages like blood and thunder. Loss of vision may be severe. There is no generally accepted acute management. Central retinal vein occlusion is not a true ophthalmic emergency. Venous occlusion could lead to ischemia and atrophy of the retina .Venous occlusion is(not acute and need urgent management) such as arterial occlusion .

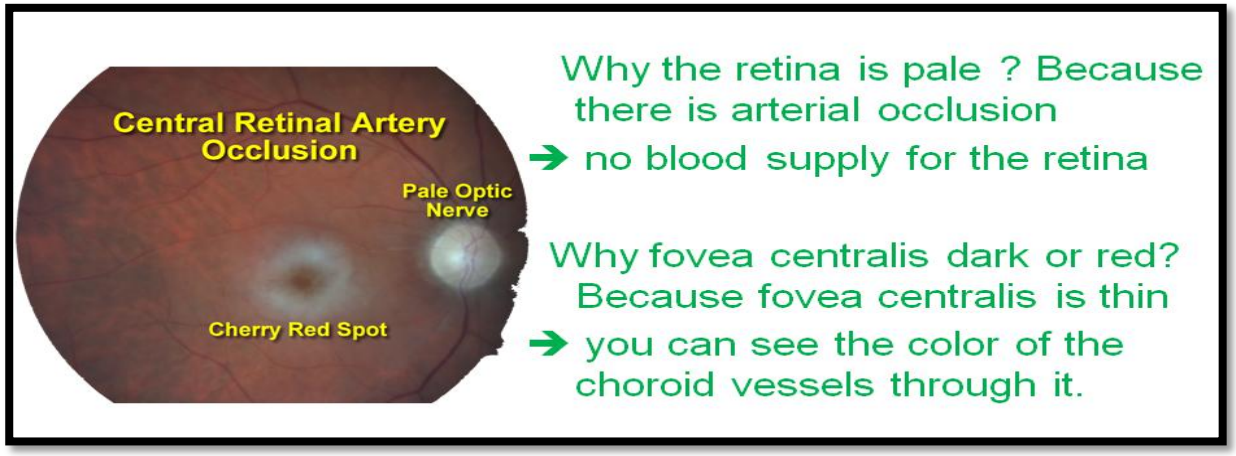
Venous occlusion can be **central** (all retina is involved , scattered hemmarhage, tursious engorged vessels all around) or **peripheral** (hemorrhage at the occluded branch site)



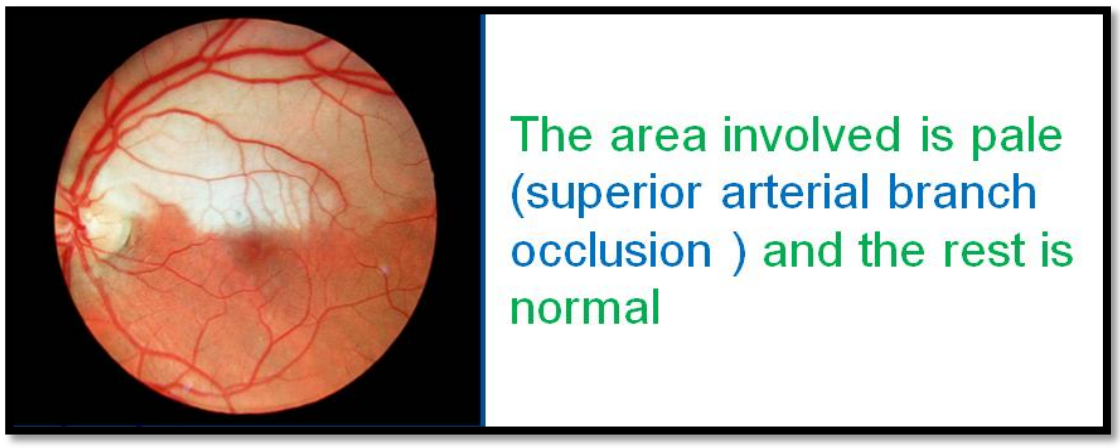


2. Central Retinal artery occlusion:

Arterial occlusion should be treated as soon as possible . it is 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. **A cherry red spot** is seen due to the pallor of the perifoveal retina in contrast to the normal color of the fovea. A chronic cherry red spot is also a feature of some metabolic storage diseases such as Tay-Sachs Pick diseas disease and Niemann-Pick disease.



Branch Retinal Artery Occlusion :
when only a branch of the central retinal artery is occluded, vision is only partially lost. 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.



❖ Optic Nerve Disease:

▪ Optic Neuritis:

Optic Neuritis is inflammation of the optic nerve and is usually idiopathic but maybe Associated with multiple sclerosis in a significant number of cases. The visual acuity is markedly reduced and an **afferent pupillary defect is present**. 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.

Inflation of the optic nerve called➔optic neuritis
One of optic neuritis Symptoms that it is painless but retrobulbar neuritis could be painful with ocular motility .

Inflammation of the optic nerve head called➔papillitis

How to differentiate between papilitis and papillary edema? Both have fuzzy margins and engorgements, but in papilitis thier will be a decrease of vision while the vision in papillary edema is not affected.

❖ Visual Pathway Disorders :

Homonymous hemianopia - is loss of vision on one side of both visual fields and 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. Any patient with a hemianopia needs at CT or MRI to localize and identify the cause.

Visual pathway : optic nerve➔optic chiasm➔tract➔radiasion➔then cortex
Studying the visual failed will help to localize the site of lesion.
For example: in pituitary adenoma there will be copresion on the chiasm ➔ bilateral temporal hemianopia.
Affected optic nerve➔ visual failed affected in one eye
Affected optic tract ➔visual failed affected in both eyes
And so on, each lesion has its visual failed impact.

❖ Cortical Blindness :

Cortical Blindness: A rare extensive bilateral damage to the cerebral 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 lights reflex separate from those carrying visual information at the level of the optic tracts, a patient who is cortically blind has normal papillary reactions. Thus a patient with normal fundus examination along with **normal papillary reactions,** most likely has cortical blindness..

❖ Functional Disorders :

A functional disorder is used in preference to hysterical or malingering to describe visual loss without organic basis. A patient may report complete blindness in one eye and normal vision in the other eye, and no relative afferent pupillary defect.

(I understood that patient psychologically denying his\her ability to see)

Note that , in functional disorders and cortical blindness the papillary reactions are normal

Done ☺ بالتوفيق