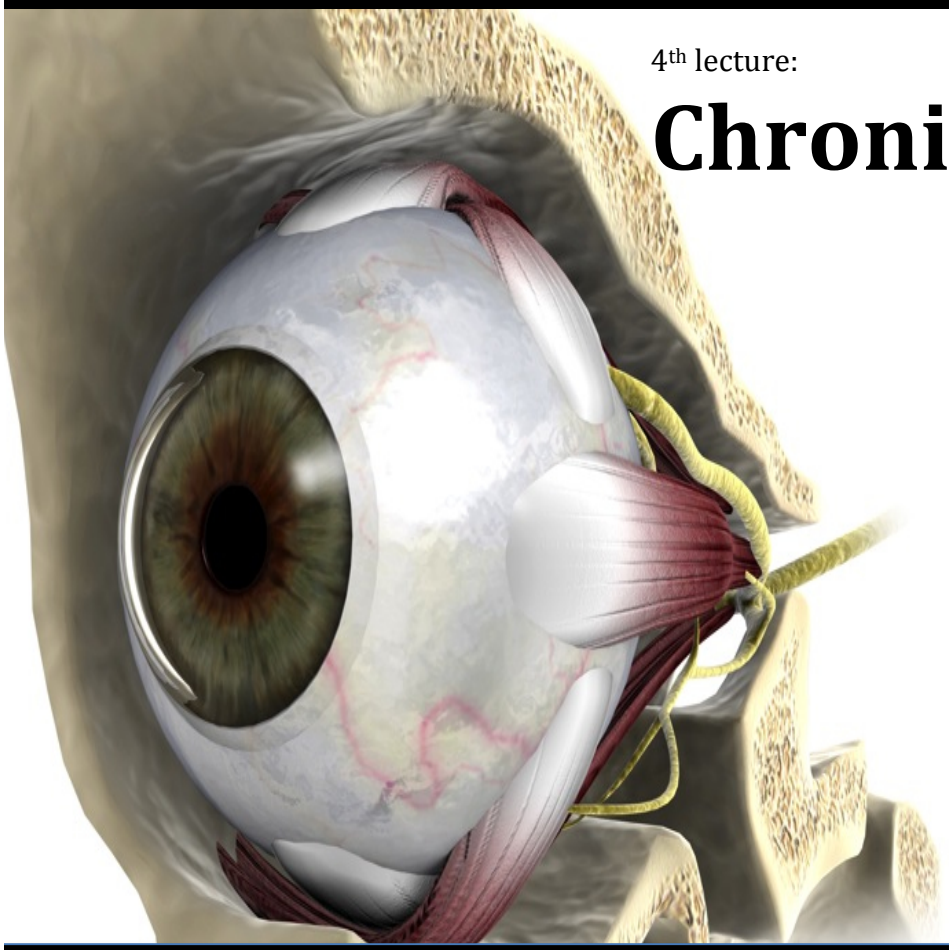
430 Ophthalmology Team



4th lecture:

Chronic visual loss

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The slides were provided by the doctor (Dr. Essam Osman)
Sources: Slides + notes taken during lecture + 429 teamwork
Important Notes in red
Things doctor said blue
Notes in green
Possible MCQs mentioned or pointed by the doctor

❖ ***Definition:*** Gradual decrease of visual acuity.

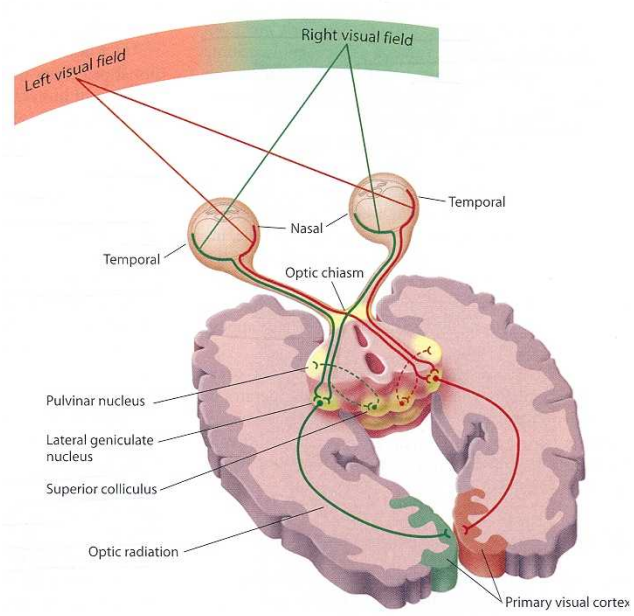
Causes of slowly progressive visual loss in an adult patient: (MCQ)

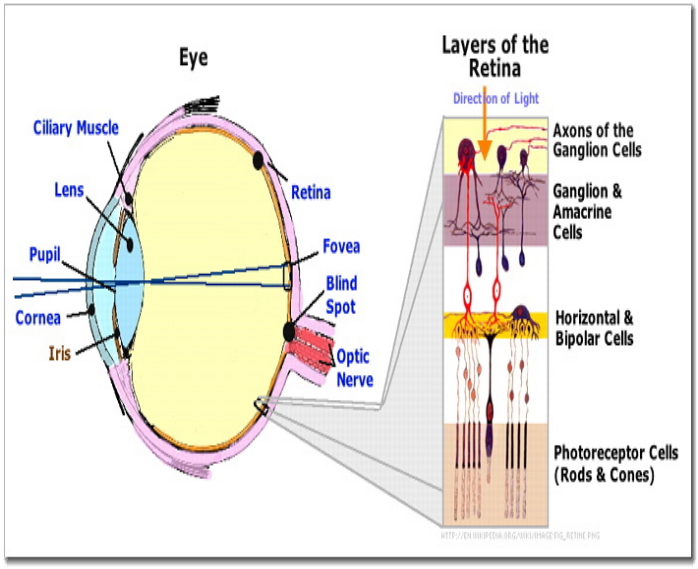
1. Glaucoma (open angle)
2. Cataract.
3. Macular degeneration
4. Diabetic retinopathy.

How to approach a patient with chronic visual loss:

1. Measure intraocular pressure with a Tonometer. (To rule in/out Glaucoma)
2. Evaluate the nerve head, classifying it as normal, or abnormal.
3. Evaluate the clarity of the lens. (Cataract opacity?)
4. Evaluate the function and appearance of the macula. (Macular degeneration)
5. Obtain full history. (Diabetes, HTN?)

The visual pathway:





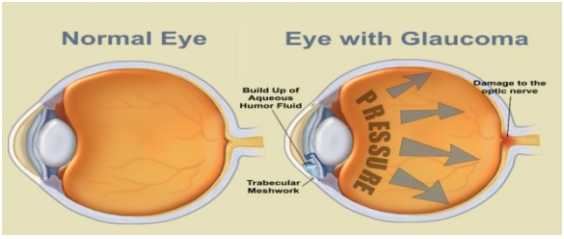
- Light enters the eye via the refractive media, namely the cornea, anterior chamber, lens, and vitreous, and stimulates the retina posteriorly. Through a series of other retinal nerve cells: light stimulates the photoreceptors, i.e. the rods and cones, the end result is that the RGC is stimulated. The RGC sends its axon, or fiber, in the nerve fiber layer to the optic disc and then down the optic nerve.

From the optic nerve, about half of the fibers cross over at the chiasm to the opposite optic tract, and the other half remain on the same side. The fibers in the optic tract synapse in the lateral geniculate nucleus of the thalamus. Neurons in the lateral geniculate nucleus then project to the occipital lobe, to the primary visual cortex. From there, there is further processing with projections to other cells in the visual cortex and elsewhere, resulting in conscious visual perception.

Now that we know how visual information is normally transmitted to the brain, what happens with a disease like glaucoma?

CHRONIC GLAUCOMA: مرض السوي رق

Glaucoma: Optic neuropathy manifested by visual field changes with or without increased IOP
(It can present with normal or low intraocular pressure)
Examine pressure, optic disk and lens.



- Major cause of blindness.
- Often asymptomatic in early stages (because only peripheral vision is affected).
- Damage is irreversible.
- Effective treatment is available.

Peripheral vision is always affected first, then the central vision is affected late; this is why they present late and this is why it is irreversible.)

Why Does Pressure Rise in the Eye Cause Glaucoma?
Glaucoma usually occurs when pressure in your eye increases. This can happen when aqueous humour isn't circulating normally in the anterior compartment. Normally, it, flows out of the eye through a mesh-like channel. If this channel becomes blocked, fluid builds up, causing angle closure glaucoma. The direct cause of this blockage is unknown. It can be inherited.
Less common causes of glaucoma includes blunt or chemical injuries to the eye, severe eye infections, blockage of blood vessels in the eye, inflammatory conditions of the eye, and occasionally eye surgery to correct another condition. Glaucoma usually occurs in both eyes, but it may involve each eye to a different extent.

Angle structures:

1. Schlemm's canal.
2. Trabecular meshwork.
3. Sclera spure.
4. Iris root.
5. Ciliary body.

Normal IOP = 10 – 21 mmHg.
Ocular hypertension = 22 – 30 mmHg.
Definite glaucoma >= 30 mmHg

Drainage of aqueous humour:
Aqueous humor is secreted into the posterior chamber by the ciliary body. It flows through the narrow cleft between the front of the lens and the back of the iris, to escape through the pupil into the anterior chamber, and then to drain out of the eye via the trabecular meshwork. From here, it drains into Schlemm's canal and eventually into the veins of the orbit.

❖ *Classification of Glaucoma:*

1) Acute (Angle closure) Glaucoma.
2) **Chronic (Open angle) Glaucoma.**
3) Congenital Glaucoma.

CHRONIC GLAUCOMA:

Primary(idiopathic): **(diagnosed by exclusion)**

Elevated Intra Ocular Pressure which results from mechanical resistance to aqueous outflow that may be caused by an abnormality in the trabecular meshwork or collapse of Schlemm's canal.

❖ *Types:*

- Juvenile onset
- Late onset
- Normal tension glaucoma

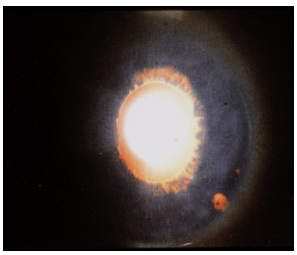
❖ *Risk factors* of primary open angle Glaucoma: (MCQ)

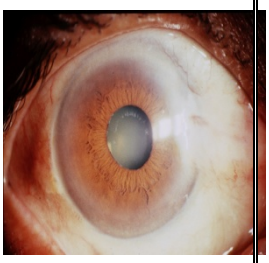
1. Family history.
2. Age.
3. Black.
4. Myopia. (will allow early diagnosis of glaucoma because they seek consultation for their myopia therefore the Glaucoma can be diagnosed early)
5. DM.
6. HTN.

Secondary:

❖ *Causes:*

- Pseudoexfoliation. (Epithelial tissue deposit > close the angle)
- Pigmentary. (Increased pigmentation of Iris leads to Glaucoma)
- Steriod-induced. (Most common cause of secondary Glaucoma) that's why never prescribe steroids for more than 4 – 6 weeks to a patient. Ex. Patients with **Vernal keratoconjunctivitis**, they treat themselves by OTC steroids to relieve itching, they present later with Glaucoma.
- Uveitic.
- Trauma.
- Lens induced





❖ *Symptoms:*

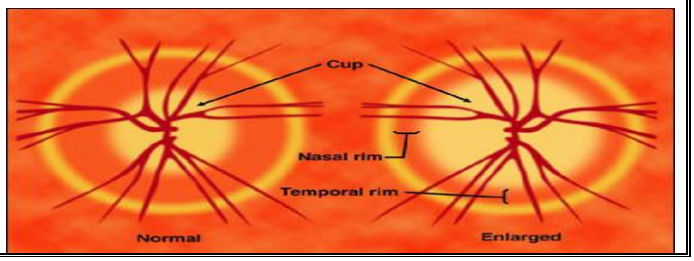
- Asymptomatic.
- Peripheral vision is affected first.
- Central vision is affected in late stage of disease.
- Tunnel vision.
- Gradual increase in IOP.

❖ *Diagnosis?* (2 of 3 must be present)

1) Raised IOP. More than 22 mmHg

IOP measurement:

- Schiotz.
- Applanation.
- Tonopen.
- Pulsair.
- Air puff.
- Paskal.



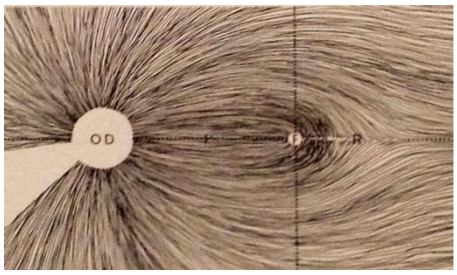
2) Optic disk abnormality. (With Ophthalmoscope)
- —Normally cupping of the disc is 0.3 to 0.4
- —More than 2% of population C:D ratio "cup:disc ratio" more than 0.7

Ex. Patient presented with C/D ratio more than 0.3, what's the next step?
Examine visual field and measure the IOP.

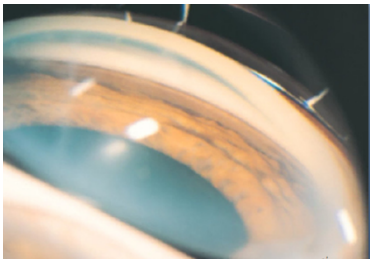
3) Visual field defect. (In respect to horizontal Medline) (MCQ)
- Peripheral vision is lost first.
- Horizontal raphe are affected.

- There are 2 directions of fibers of retina: horizontal and vertical line. In glaucoma the horizontal raphe (MCQ) are damaged, particularly the superior and inferior temporal fiber.
- The vertical midline is involved in neurological diseases.
- Horizontal lines are weaker than vertical ones; this is why they get affected first by pressure.

Papillomacular bundle: is the strongest fiber bundle in the retina and it gives the best vision; it is between the fovea and the optic nerve.



4) Decreased visual acuity in late stages.
5) Gonioscopy: Open anterior chamber angle
- Schwalb's line
- Trabecular meshwork
- Sclera spure
- Iris root
- Ciliary body



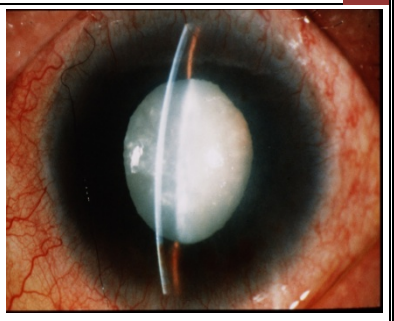
❖ ***Management:***

- The primary goal of POAG therapy is to halt or delay progression of the disease in order to prevent the patient from developing blindness.
- The effect of treatment is temporary.
- Therapy is administered in a stepwise fashion beginning with medicine, laser and finally surgery.
- The choice and timing f treatment depend on the degree of optic nerve damage, level of IOP control, and progression of disease, patient age, and compliance.

Options are as follow

1. Observation
2. Medical: ocular hypotensive agents: beta-blocker, prostaglandin analogues, alpha-adrenergic agonist, and carbonic anhydrase inhibitors.
3. Laser: trabeculoplasty to increase trabecular meshwork outflow, or to apply laser at the limbus to destroy ciliary processes and reduce aqueous production (cyclophotocoagulation).

4. Surgical: The aim of the surgery is to lower the IOP by creating an alternate pathway for aqueous to escape from the eye. The most common procedure is trabeclectomy. Another option is glaucoma drainage implant.



CATARACT: 2nd cause of chronic visual loss

- Opacification of the lens.

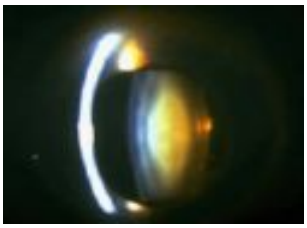
❖ *CAUSES:*

- **Age-Related:** divided based on morphology into:
  - **Sub-capsular cataract:** occurs just beneath the lens capsule
  - **Nuclear cataract:** due to diffuse lens hardening and discoloration from deterioration of older central (nuclear) fibers.
  - **Cortical cataract:** caused by swelling, degeneration and liquefaction of the younger outer (cortical) fibers.
- **Traumatic:** unilateral.

Cataract is the most common cause of chronic visual loss.

- **Metabolic**
  - Diabetes is the most common cause of metabolic cataract
  - Galactosemia.
  - Glacokinase deficiency.
  - Mannosidosis.
  - Fabrys disease.
  - Lowes syndrome.
  - Hypocalcemic syndrome. (MCQ)
- **Cataratogenic Drugs**
  - Chlorpromazine: antipsychotic
  - Miotics: **miotic eyedrop**: increase the outflow of aqueous humor by constricting the pupil
  - Myleran: decreases bone marrow function used in leukemia treatment
  - Amiodarone: antiarrhythmic
  - Gold
- **Complicated Cataract** *(cataract being a complication of the following)*
  - Uveitis
  - Retinal dystrophy, retinitis pigmentosa.
  - High myopia
  - Acute glaucoma attacks can cause anterior subcapsular cataract
- **Intrauterine Cataract**
  - Rubella, toxoplasmosis and CMV.
- **Syndromes**
  - Down syndrome,
  - Werner and
  - Rothman syndromes.
- **Hereditary**
  - 1/3 of cases

❖ *Classification:*



- **Based on morphology:**
  - Nuclear
  - Subcapsular
  - Cortical.
- **Based on maturity:**
  - Immature (part of lens involved)
  - Mature (entire lens involved)
  - Tumescent (congested)
  - Hyper-mature
- **Based on age of onset:**
  - Congenital
  - Infantile
  - Pre-senile
  - Senile

❖ *Signs:*

- Decreased visual acuity.
- Color vision defect. (Especially blue color discrimination)
- Lens opacification

❖ *Management:*

Indications of surgery: numbered based on priority. (MCQ)

1. Patient's needs
2. Decreased visual acuity
3. Therapeutic
4. Complicated

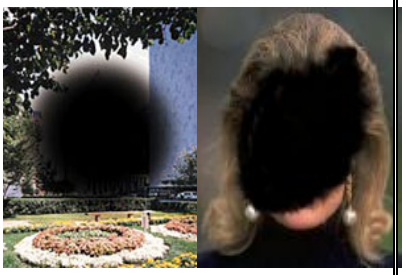
Types of management:

- **Congenital**: lens aspiration ± IOL (Intraocular lens)

- **Surgeries for acquired:**

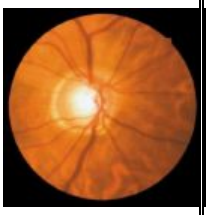
1. ICCE (intra capsular cataract extraction): removal of the whole lens, not done any more.
2. ECCE (extra capsular cataract extraction): a hole is made into the capsule allowing the removal of the nucleus and insertion of a prosthetic lens into the remaining capsular bag.
3. ECCE + IOL: as above with intra ocular lens insertion.
4. PHACO IOL: micro incision cataract surgery. (Most common surgery done now)

MACULAR DEGENERATION:



❖ Macular Anatomy: *Irreversible central visual loss due to loss of corocapillaris and photoreceptors above 65 or 70 years.*

- The macula is an oval area situated about 2 disc diameters temporal to the optic disc. The macula is composed of both rods and cones and is the area responsible for detailed, fine central vision.
- The central macula is avascular and appears darker than the surrounding retina. The fovea is an oval depression in the center of the macula, it has a high density of cones but has no rods.
- The central depression of the fovea may act like a concave mirror during an ophthalmoscope exam, producing a light reflection (i.e. foveal reflex).



❖ Definition of Macular degeneration:

- Some degree of visual loss associated with drusen(hyaline nodules), atrophy of Retinal pigmented epithelium (RPE) and subretinal neovascularization (CNV).

❖ Types:

- **Non-exudative (dry)** 90%: slow progressive atrophy of Retinal pigmented epithelium (RPE) and photoreceptors. Most patients are asymptomatic, however those with atrophy may notice metamorphopsia (visual distortion). (progressive atrophy and loss of photoreceptors thus decreased vision)
- **Exudative (wet)** 10%: characterized by choroidal neovascularization and Retinal pigmented epithelium (RPE) detachment. (Can be treated effectively with laser) more treatable. (RPE detachment, atrophy of corocapillaris in retina and neovascularization which causes leakage causing fibrosis in macular area causing decreased vision)

Fortunately there is peripheral vision therefore those with age-related macular degeneration do not completely loss their vision they decreases gradually from 6/6 to 6/60 (20/20 to 20/200).

❖ Relevance:

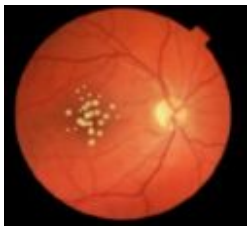
- In the United States, age-related macular degeneration is the leading cause of ***irreversible central visual loss*** (20/200 or worse).
- Because certain types of macular degeneration are treated effectively with laser, it is important to recognize this entity and to refer for appropriate care.
- It is important to distinguish between the possible causes of visual loss, whether cataract (surgically correctable), glaucoma (medically or surgically treatable), or macular degeneration (potentially laser treatable).

❖ Tests for macular function:

- **Visual Acuity**
- **Pupillary light reaction.**
- **Color vision.**
- **Ophthalmoscopy.**
- **Amsler grid** (important test: a 10 by 10 square grid held at normal reading distance which you give to the patient; as long as the patient sees it as squares he is normal but once he starts

seeing it distorted this is a sign of early macular degeneration. Now we need to do Flourescine angiography..)

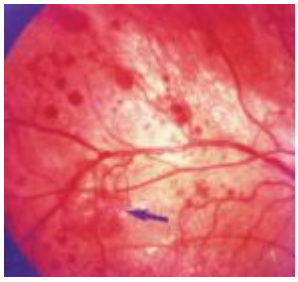
- **Phtosters test.**
- **Laser inferometry.**
- **Flourescine angiography.** to assess degree of neovascularization and differentiate between the two types: early staining wet type late staining in the dry type.



- Drusen are hyaline nodules (or colloid bodies) deposited in Bruch's membrane, which separates the inner choroidal vessels from the retinal pigment epithelium. Drusen maybe small and discrete or larger, with irregular shapes and indistinct edges. Patients with drusen alone tend to have normal or near normal visual acuity, with minimal metamorphopsia (change in the size of objects)
- As the most common cause of vision loss among people over the age of 60, macular degeneration impacts millions of older adults every year. The disease affects central vision and can sometimes make it difficult to read, drive or perform other activities requiring fine, detailed vision.

Drusens can be present in normal people; however, their presence is a risk factor for the development of the disease.

❖ ***Risk factors:*** (MCQ)



- **Non-modifiable:**
  - Age
  - Race
  - Gender
  - Genetics
- **Modifiable:**
  - Smoking
  - High Blood Pressure
  - High Cholesterol
  - Poor Nutrition
  - Unprotected Exposure to Sunlight
  - Ultraviolet (UV) light has been
  - Excessive Sugar Intake
  - Obesity
  - Sedentary Lifestyle

❖ ***Management:***

- Dry type:
  - Monitor, Amsler grid allows patients to check for metamorphopsia.
  - Low vision aids (magnifier, closed-circuit television).
  - Anti-oxidants, green leafy vegetables.
  - Sunglasses, visors.

- Wet type:
  - Laser photocoagulation for neovascularization.
  - 50% of choroidal neovascularization cannot be treated initially.
  - No definitive treatment for disciform scarring.
  - Photodynamic therapy with verteporfin

- intravitreal injection of anti-angiogenesis growth factor.

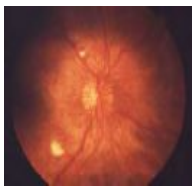
DIABETIC RETINOPATHY: most common in Saudi Arabia

❖ Definition:

Progressive dysfunction of the retinal blood vessels caused by chronic hyperglycemia.

❖ Risk factors:

- Duration of the disease. (Most important risk factor)
- Type of diabetes mellitus.
- Metabolic control: chronic hyperglycemia increases the risk of developing diabetic retinopathy.
- Hypertension
- Renal diseases.
- Hyperlipidemia
- Pregnancy
- Anemia



❖ Pathogenesis:

- **Microvascular Occlusion**

Thickened capillary basement membrane > Capillary endothelial cell damage > Changes in RBC > Retinal ischemia > AV shunt and growth factors release > Neovascularization > New weak vessels > Hemorrhage > present with vitreous hemorrhage. "It is treated with laser".

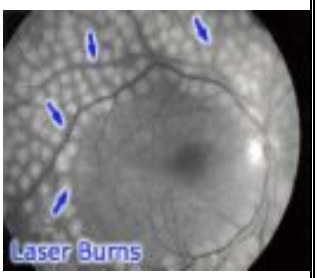
- **Microvascular Leakage**

Loss of pericyte cells between endothelial cells > Leakage of plasma constitute (proteins and lipids) in the retina (exudate).

❖ Types: imp

- Non-proliferative. (Early stages)
- Proliferative. (Presence of neovascularization and traction retinal detachment)
- Macular edema. (Presence of exudate)

❖ Management: MCQ



- **Non-Proliferative diabetic retinopathy:** observation.
- **Proliferative diabetic retinopathy:** Pan-retinal photocoagulation. (PRP)
- With or without **Macular edema:** Focal and GRID laser.

MCQ:

Neovascularization > diabetic patient refuses to do laser > Secondary open angle Glaucoma develops > Leakage of the vessels > Fibrosis > Closed angle Glaucoma > Sudden increase in the IOP > Neovascular Glaucoma.

- If the angle is closed > can't treat.
- If the angle is still open > we can treat with steroids (decreased edema) and Atropine (decreases ciliary body spasms and dilates the pupils).

Causes of neovascular Glaucoma:

- Diabetic retinopathy. (Chronic visual loss)
- Central vein occlusion. (Acute visual loss).

Doctor talked about congenital glaucoma and said it is not part of our course but this is what he said:
The child has a large cornea and high pressure. (high pressure is a must).