Ophthalmology
Team 437

MED437
KING SAUD UNIVERSITY

Chronic Visual Loss

Objective

Not provided

- This lecture is very important for the exam.
- It was previously given by Dr AlMobarak to both group A and 436, thus we added 436 teamwork to ours to include all topics in this lecture.
- Dr AlKharashi mentioned “My slides include everything that is needed for the exam and my notes are for your understanding”.
- 436 teamwork will be highlighted as: blue = mentioned by Dr alkharashi grey: extra

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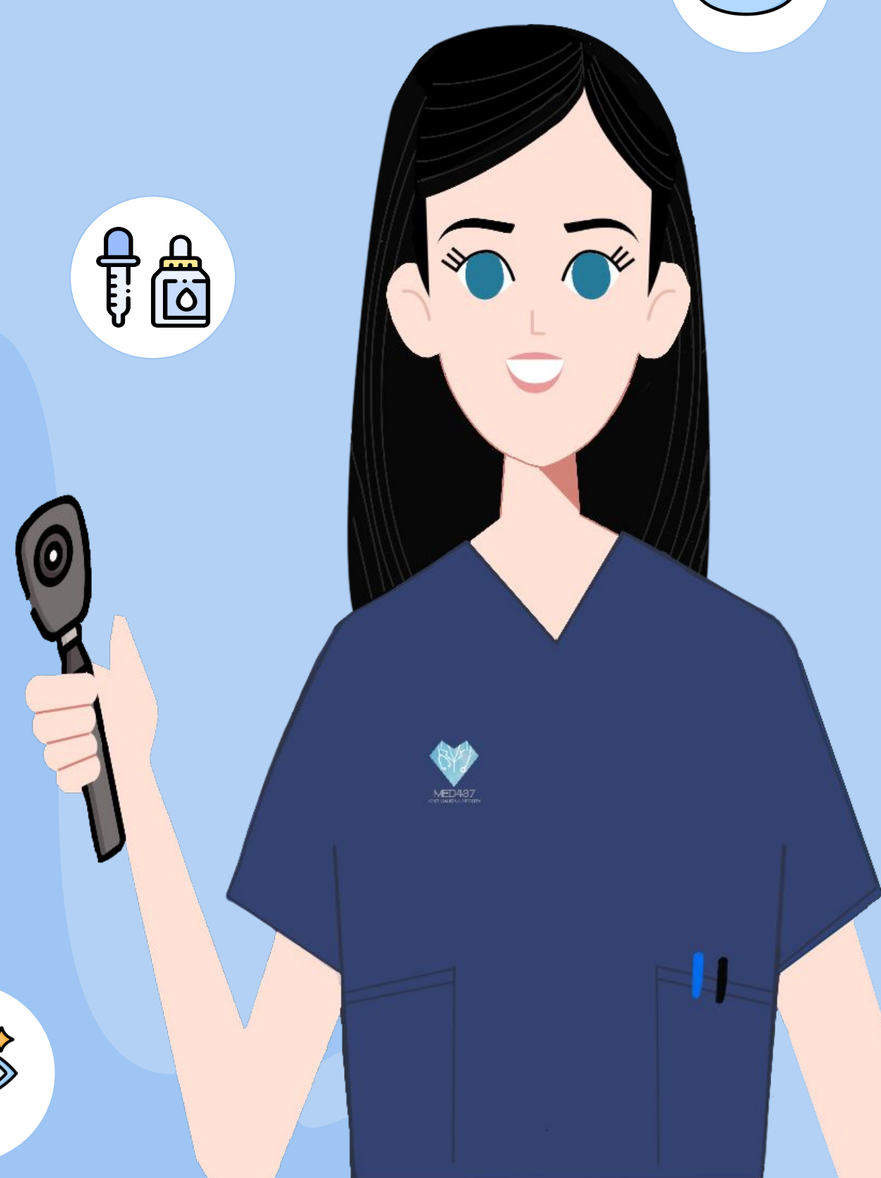
Resources :

Dr. Alkharashi & Prof. Essam Osman slides and notes, 436 teamwork

Book (Lecture notes in Ophthalmology)

- Editing file

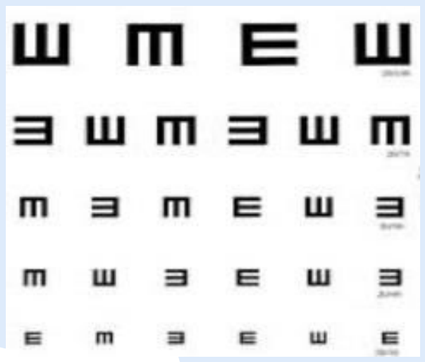


Color index

- Important
- Notes
- Book
- Extra
- Golden note
- 436 Teamwork

Introduction to chronic visual loss

- Dr discussed the visual pathway. You can return to the anatomy lecture to understand it.



- **Definition:** a gradual progressive event (painless visual loss).
- There is no pain no redness (quite eye)
- **Duration:** > 2 weeks.
- **Vision:** (how can we assess vision?)
  - Quantity: VA (visual acuity).
    - The fovea and macula contain cons photoreceptors(central)
  - Quality: VF (visual Field) peripheral retina , clarity of vision, color vision.
    - Peripheral vision detects the surroundings, ex. Movement of nearby object.
    - Rods receptive field is huge , كانه عندك عامل في المزرعة يعلمك اذا احد دخل مزرعتك
    - Cons narrow receptive field aid in accuracy, (الحارس يعلمك تفاصيل الدخيل (اي بوابة، نوع السيارة، عدد الاشخاص،شكلهم)
- **Causes:**
  - Refractive (the most common).
  - Cornea.
  - Lens.
  - Vitreous.
  - Retina.
  - Optic nerve. Ex. benign tumor-> start with visual field loss
  - Neurologic.Ex. benign tumor, pituitary adenoma -> start with visual field loss

Tunnel vision in advanced glaucoma

- **Causes of slowly progressive visual loss in an adult patient:**
  1. **Glaucoma**
  2. **Cataract**
  3. **Macular degeneration**
  4. **Diabetic retinopathy**
  5. **Retinitis pigmentosa.**

Good quantity but bad quality.

- **Diagnosis of chronic visual loss** (by exclusion)**:**

  1. Measure intraocular pressure with a tonometer, if normal then most likely it is not glaucoma**.**

  2. Evaluate the nerve head, classifying it as normal or abnormal, normal optic disc=most likely not glaucoma.

  3. Evaluate the clarity of the lens, clear lens= most likely not cataract.

  4. Evaluate the function and appearance of the macula, if affected= most likely retinitis pigmentosa.

Refractive:

Mostly in young adults.

Causes:

- Myopia, hyperopia or astigmatism.
- Amblyopia (كسل العين).

Signs:

- Normal exam.
  - You will have completely normal examination apart from affected visual acuity with refraction error.
  - However, sometimes patients with astigmatism have corneal scar on examination.
- Refraction needed to show errors.

Treatment:

- Glasses, contact Lenses, refractive surgery.
  - There are different types of refractive surgery:
    - **Lasik and epilasik:** we are maintaining the epithelium &r applying it again. Faster visual recovery.
    - **PRK:** there is scraping of the epithelium by focusing the laser emission on the corneal stroma. Higher safety margin in patients who have borderline corneal thickness.
- NB: lenticular causes need cataract surgery.
  - Lenticular causes of refractive error (not important to know, but good for your knowledge).
  - If you have:
    - A bulky lens, it will focus the light rays in front of the retina, and it will induce more myopia.
    - Or if you have weak zonules, the lens will either move forward or backward.
      - If it moves forward, the light rays will be more focused in front of the retina, so it will induce more myopia.
      - If it moves backward, light rays will be focused behind the retina, so it will induce hyperopia.

Cornea:

- ❖ living tissue that has the ability to regenerate very quickly
- ❖ Why is it transparent ?

1. because it is avascular
2. the corneal epithelium is tightly packed together to avoid scattering of light rays as it passes it
3. The corneal lamellae within the stroma is arranged in a unique pattern - the distance from one collagen lamellae to the other is shorter than half of the light's wavelength.
4. Corneal hydration is fixed around 78% ; corneal edema reduces the transparency
   - the anterior chamber provides corneal nutrition by passing the fluid through the corneal endothelium junction, the corneal endothelium pumps out the extra fluid from the cornea to maintain hydration at 78%) <- will be mentioned in glaucoma topic

- ❖ Corneal endothelium pathology: if a patient has corneal abrasion, post surgery endothelial detachment or dysfunction to the endothelial pump -> corneal edema (white and opaque)
- ❖ corneal ulcer will heal with a scar (white spot in the cornea)
- ❖ patient with corneal abrasion left untreated -> corneal ulcer (invaded the stroma)-> infection = body considers it as an emergency and irregular collagen will close the opening in an irregular way (opaque scar)

Causes:

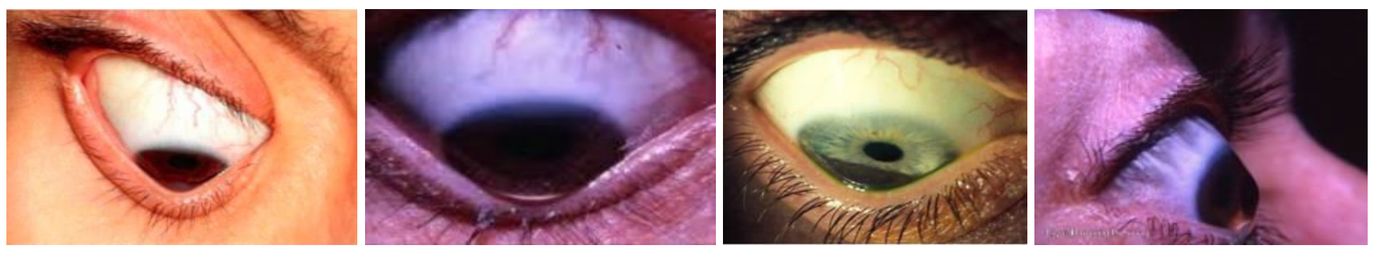
- ❖ Scar from previous trauma or infection.
- ❖ Hereditary: corneal dystrophies, keratoconus.
  - ➢ Why there is a scar whenever you have a trauma or an infection? because the collagen fibers intercalated with each other instead of being parallel causing scaring.
  - ➢ We suppress the scarring by steroids (we give steroids after eye surgeries).

Signs:

- ❖ Corneal scar.
- ❖ Bulging cornea.
- ❖ Stromal opacities.
- ❖ Patients might have some conjunctival injection with chronicity.

Keratoconus قرنية مخروطية (very important for exam):

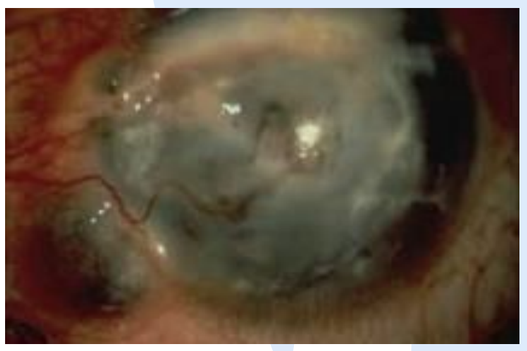
- ➢ In keratoconus, instead of having a smooth regular corneal surface, it is bulging anteriorly; It will induce more myopia and more astigmatism because the regularity of the cornea will be lost.
- ➢ It is impossible for someone with keratoconus to have hyperopia.
- ➢ In early phases, astigmatism is regular; Then with advanced keratoconus it becomes irregular astigmatism.
- ➢ When the lid margin bulges down when someone looks down, this is a sign called Munson's sign that occurs with advanced Keratoconus.
- ➢ Descemet's membrane will rupture, then aqueous will enter to the corneal stroma. With chronicity, it will induce corneal haze, corneal edema. It also causes apical scarring.
- ➢ Advanced keratoconus:
  - Irregular myopic astigmatism.
  - Munson's sign.
  - Apical scarring.



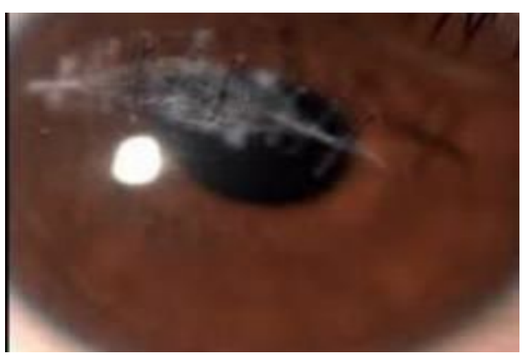
Keratoconus
Sign: Munson's sign.

Cornea cont:

- **Stromal opacities**
  - Conjunctival injection with chronicity. Conjunctival injection is almost always seen in acute event, but sometimes you might see it as chronic event because of chronic infection like fungal keratitis or herpetic keratitis.

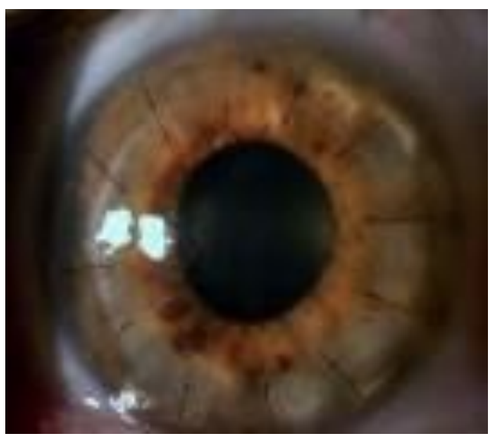


- **Corneal scar**
  - This pt. had a penetrating globe injury by a metallic object (full thickness corneal laceration). It needed suturing, we sutured it by (10 nylon) which is non absorbable, we need to remove it after a while.
  - The cornea will heal by scarring which will induce chronic visual loss. Also, corneal scarring induces astigmatism that is a cause of chronic visual loss (this is the main reason).
  - Sometimes the scar is central, within the pupillary area, so corneal haze will obscure the entry of light rays within the pupil, resulting in astigmatism and a barrier toward full light entry through the pupil.
  - Also, infection. If you are a contact lens wearer, you might have a corneal ulcer (microbial keratitis). The most common organism in contact lens wearer is pseudomonas. It causes a very rapid corneal perforation.



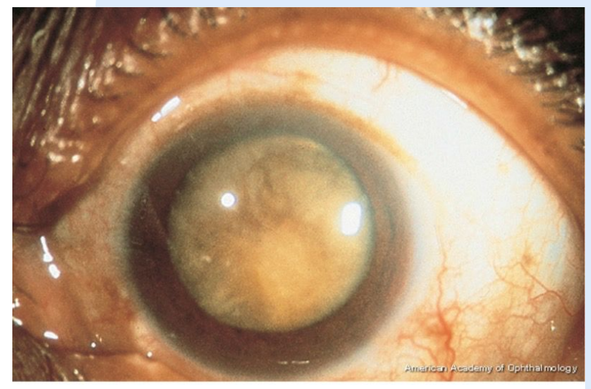
Important picture

- **Treatment:**
  - Refraction to correct astigmatism or myopia.
  - Contact lens (soft or hard) for keratoconus.
    - It is used to depress the conning cornea back.
  - Corneal cross linking.
    - UV light is applied to strengthen the corneal stroma and as a result the bulging will stop. but we should protect the retina (by vitamin B drops).



  - Keratoplasty زراعة قرنية:
    - If the conservative measures fail.
    - A cornea is taken from a donor & is implanted in a recipient eye, there are 3 types:
      - Penetrating keratoplasty: implanting all layers of cornea.
      - Lamellar Keratoplasty: transplanting only the stromal and bowman layers.
    - Indications:
    - Keratoconus without scaring or with anterior stromal scarring
    - **Not** for deep posterior stromal scarring.

Lens “cataract”: (إعتام عدسة العين)



The most common cause of chronic visual loss.
Opacity of the lens. Lens is transparent, and it contains proteins which are well-organized. When there is disorganization of lens protein = from soluble to insoluble, there will be clouding of the lens (cataract).

Pathophysiology: Video

- Disorganization of lens proteins → opacification.

Causes:

- Age related (the most common cause).

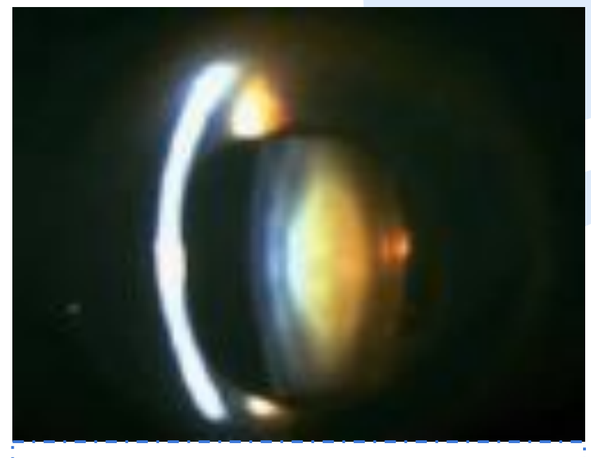
Subtypes: subcapsular (anterior or posterior), Nuclear and cortical.

- The origin of the lenses is the surface ectoderm, same as hair and skin. After age 40, changes happen to hair, skin and vision. People develop (presbyopia) because accommodation is slightly affected (the lens loses its elasticity) secondary to disorganization of lens proteins.

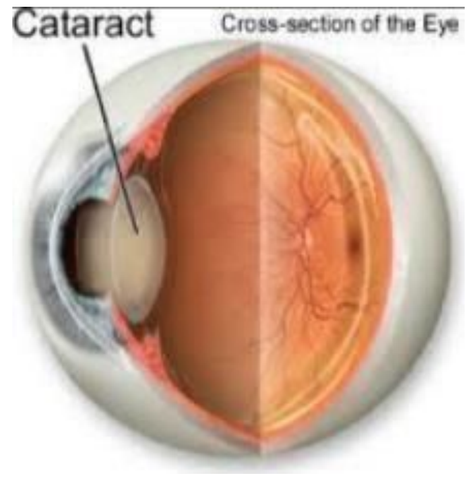
- Metabolic:
  - Diabetic
  - Galactosemia
  - Galactokinase deficiency
  - Mannosidosis
  - Fabry's disease
  - Lowes syndrome
  - Hypocalcemic syndrome.

DM is the most common cause of cataract development & progression

- Systemic diseases
  - Myotonic dystrophy
- Traumatic.
- **Medications: Cataractogenic drugs**
  - **Steroid. it will cause posterior sub-capsular cataract (it will cause glaucoma as well).**
  - Chlorpromazine
  - Miotics
  - Myleran
  - Amiodarone
  - gold
- Congenital.
- Inflammation: Uveitis
- Complicated cataract: Uveitis, Retinal dystrophy, retinitis pigmentosa, High myopia and Acute glaucoma.
- Intrauterine causes: Rubella, toxo and CMV.
- Syndromes: down syndrome, werner syndrome, rothman syndrome.
- Hereditary: 1/3
- Ocular: Patient with retinitis pigmentosa.



-Nuclear cataract
-Can be treated 1-3 months after diagnosis



Cataract Clinical Classification (important):

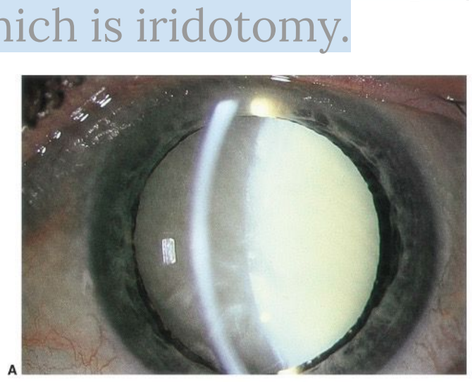
1. According to The Maturity
(progression from immature, mature, intumescent, hyper-mature).

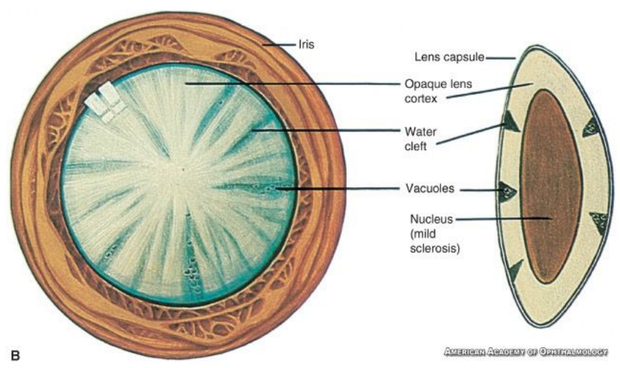
Immature

You can see the posterior pole (retina).

Mature

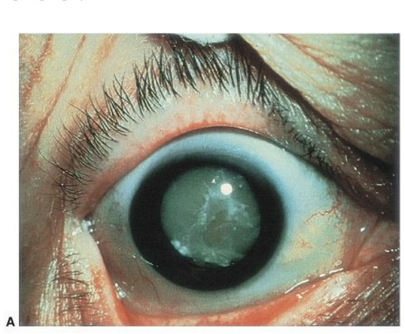
- The whole lens is completely white.
- You cannot see the posterior pole "retina".
- The anterior chamber is shallow (narrow) → risk of glaucoma, so do a prophylaxis which is iridotomy.

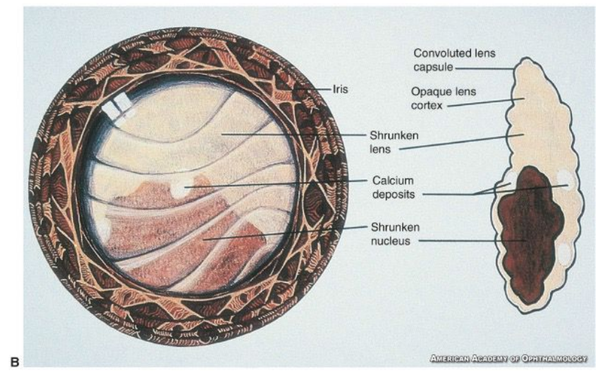




Hyper-Mature

The lens proteins leak through the intact capsule, Lens is composed of nucleus, cortex & capsule. When the cortical proteins start to leak through an intact capsule, the nucleus will sink down within the capsule & the capsule will wrinkle.
Urgent case.

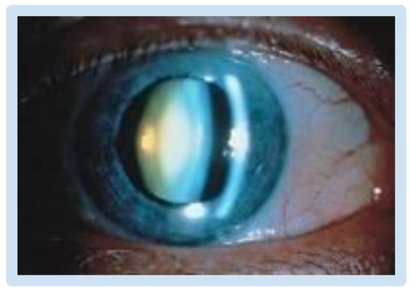




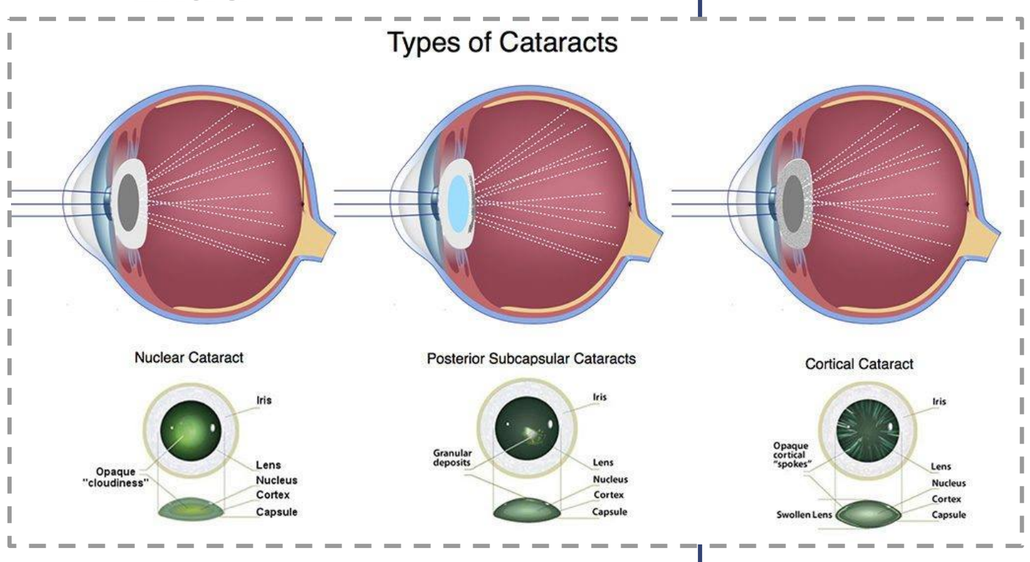
2. Morphologic (According to the Anatomic location)
important for surgical intervention, it can be a mixture of all types.

Nuclear

The lens nucleus itself becomes opacified.



Extra

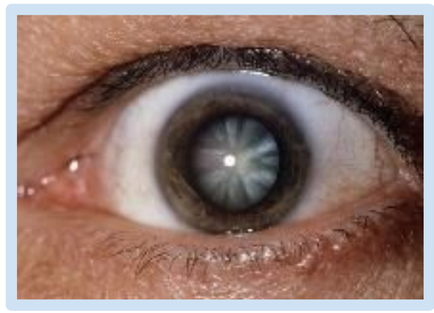


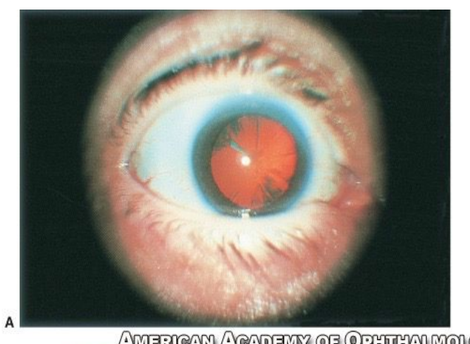
Subcapsular

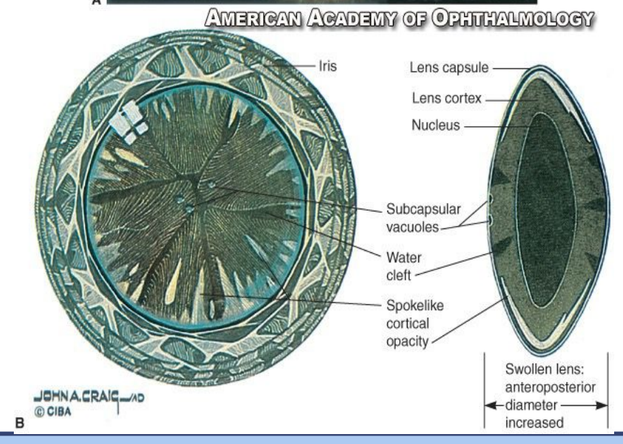
- Nucleus is not significantly affected.
- It precipitates in the posterior sub-capsular area.
- Bothersome why? 1- cataract at visual axes-> prevents light rays to pass through 2-posterior capsule opacity is close to the nodal point(area of refraction) = hard to treat.

Cortical

Cortex is opacified.







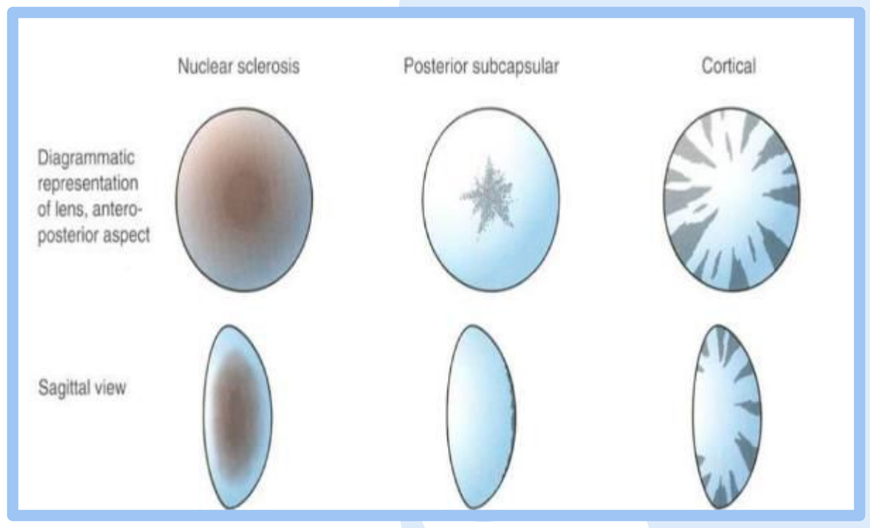
3. According to the Age of onset

Congenital
treat early or they will have amblyopia

- Hereditary ⅓
- Metabolic
- Syndromes:
  - Down syndrome, trisomy 13 & 18
- Intrauterine infection:
  - Rubella, Toxoplasmosis, CMV

Infantile

Pre senile & Senile

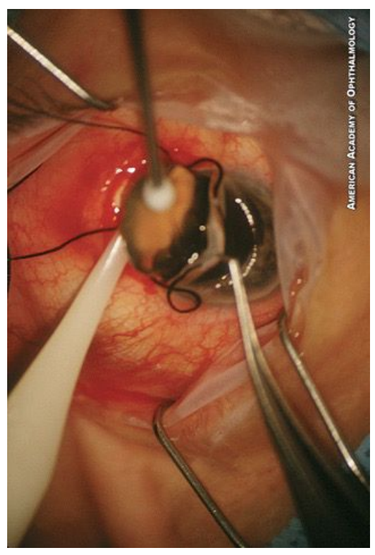


Clinically:

- Gradual onset of painless visual loss.
- **Visual acuity:**
  - Worsening of existing myopia
    - In cataract the lens will enlarge & the anterior-posterior diameter increases, so light rays will be focused more anterior to retina).
  - Correction of hyperopia
    - In hyperopia, the light rays fall behind the retina; thus, some cataract patients, especially old ones who need near vision, will be happier with nuclear sclerosis, WHY? The diameter increases, light rays fall more anterior into the retina. So old patients who do not drive at night, will be happier & should be informed that they will need glasses for near vision after surgery.
- Loss of contrast sensitivity in low light المريض ما يقدر يفرق بين درجات الألوان, فيحتاج ضوء اقوى عشان يفرق
- Glare in bright light (scatter of light):
  - Classically happens with posterior subcapsular cataract during night drive. Even if the patient has a visual acuity of 20\20; this affects visual quality.
  - The posterior sub-capsular cataract will not allow equal penetration of light through the pupil. The light that passes through areas of condensation of posterior subcapsular cataract will have different intensity than the light that passes freely.

Management:

- Only surgical (you have to know it) Medical Treatment isn't effective! The more opaque the more you need to treat
  - ICCE (Intracapsular cataract extraction) removed lens and cataract and capsule, rare now.
  - ECCE (Extracapsular cataract extraction) removed center of lens that contains cataract, leave capsule. Bigger incision , more suture , more corneal damage, more infection risk
  - ECCE IOL (Extracapsular cataract extraction) + (intraocular lens) keeping the capsule in the eye acts as a bag for the artificial lens
  - **PHACO IOL (Phacoemulsification) + (intraocular lens)** small incision (2-3 mm), break cataract through ultrasound and place a foldable artificial lens (best surgical method) **BEST**
  - Congenital: lens aspiration ± IOL
  - Acquired:
    - ECCE + PCIOL (posterior chamber intraocular lens) “very severe cataract”.
    - Open anterior capsule & empty the lens from the opacified proteins; After that, you do ECCE or phacoemulsification then you put an artificial lens.
    - The problem with artificial lens: it does not allow accommodation. But new lenses allow patients to look far, intermediate and near without correction, it is called multi- or tri-focal IOL.
    - Phaco (Phacoemulsification) + PCIOL (Phaco is modified ECCE: small opening and putting a foldable lens).
- Simply you have an opacified lens proteins you need to exchange the lens with an artificial clear lens).



Vitreous:

Vitreous hemorrhages:

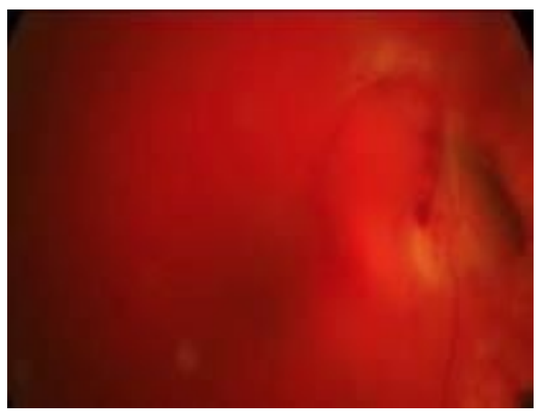
- ➢ Causes:
  - Trauma (bleeding blood vessels will cause gradual loss of vision).
  - PDR (Proliferative diabetic Retinopathy).
  - Uveitis.
  - PR (proliferative retinopathies in general).

Vitreous condensation, opacification:

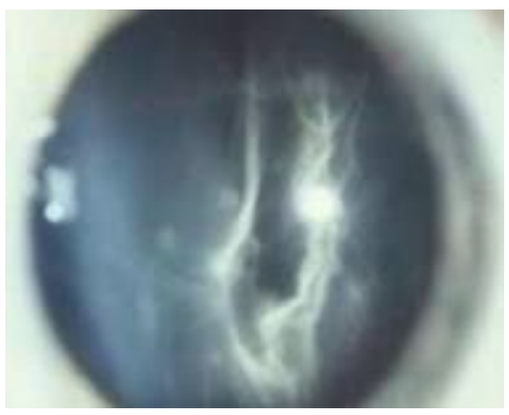
- ❖ Causes:
  - ❖ In chronic inflammation or posterior vitreous detachment (PVD), there will be accumulation of inflammatory cells within the vitreous.
  - ❖ With inflammation, there will be fibrosis, scaring and haziness.
  - ❖ Patient will complain of flying fly, especially with old patient.
  - ❖ The vitreous is a jelly like transparent structure once it detached
  - ❖ from its insertion it will shrink and cause condensation. sometimes it will affect the vision.

Vitritis:

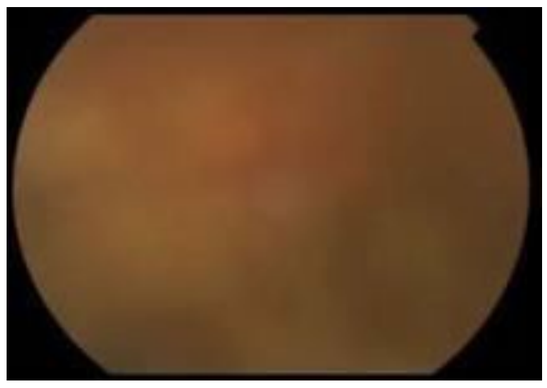
- ➢ Causes:
  - Uveitis. with chronic uveitis, the vitreous will be full of inflammatory cells.
  -
  - **Treatment:**
- ❖ Treat the underlying causes
  - ➢ Uveitis: steroids and immunosuppressive medication.
  - ➢ Vitreous hemorrhage: observation, if it not resolved by itself you need to do vitrectomy.



Vitreous hemorrhage, you cannot see the posterior pole



Vitreous condensation (flying fly)



Glaucoma الماء الأزرق Video

Increase in intraocular pressure

EGS definition: "progressive optic neuropathies, that have in common characteristic morphological changes at the optic nerve head and retinal fiber layer in the absence of other ocular disease or congenital anomalies.
Progressive retinal ganglion cell death and visual field loss are associated with these changes"
Prof. Essam: " it's important to know that in the past glaucoma was thought to be an increased intraocular pressure, but actually glaucoma is optic neuropathy, so the first thing you should see is optic nerve damage, manifested by visual field change, and not necessarily associated with increased intraocular pressure, we call it "Normal Tension/Pressure Glaucoma".
Extra: Definition of glaucoma: clinical and experimental concepts

The significance:

- Second leading cause of blindness in the United States.by damaging the optic nerve single most important cause of blindness in African Americans.
- Often Asymptomatic; in early stage (peripheral vision) a silent disease.
  - Early diagnosis is crucial to prevent loss of vision. If glaucoma is detected early and treated medically or surgically, blindness can be prevented. Most patients with early glaucoma are asymptomatic.
- Damage is irreversible irreversible blindness
- Effective treatment is available

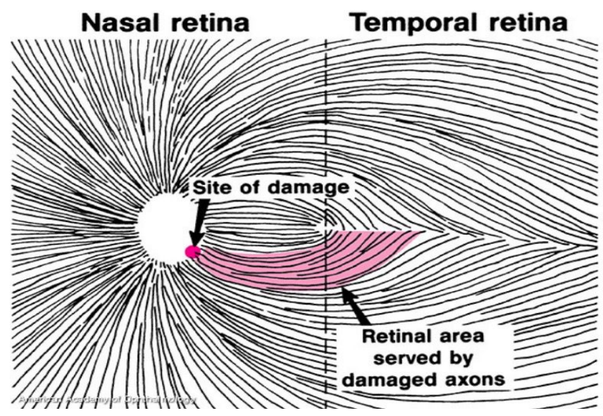
Types: (Acute-chronic-congenital)

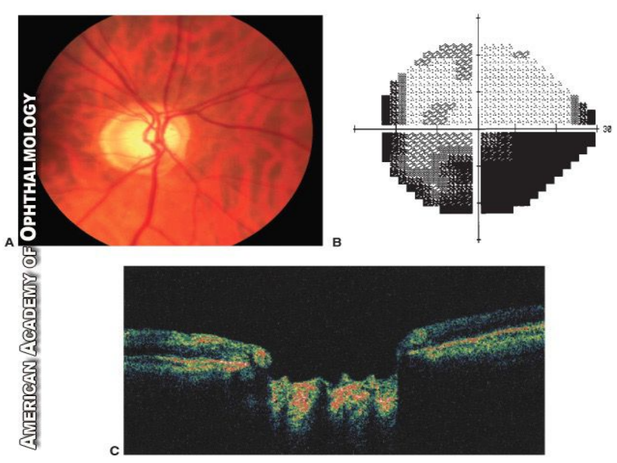
- Open Angle
- Acute Closed angle

Risk factors:

- IOP (most imp).
- Age.
- Family history.
- DM/HTN.
- Medication "Steroids".
- Myopia.

- Horizontal rafi separates the retina and optic nerve into superior and inferior retina
- Superior retinal fibers (responsible for the inferior part of the visual field) accumulates at the superior pool of the optic nerve
- Inferior retina fibers (responsible for the superior part of the visual field) accumulates at the inferior pool of the optic nerve
- Images are inverted by the retina; therefore each side of the retina is responsible for the opposite visual field
- Pink damage seen on image results in part of the superior visual field loss

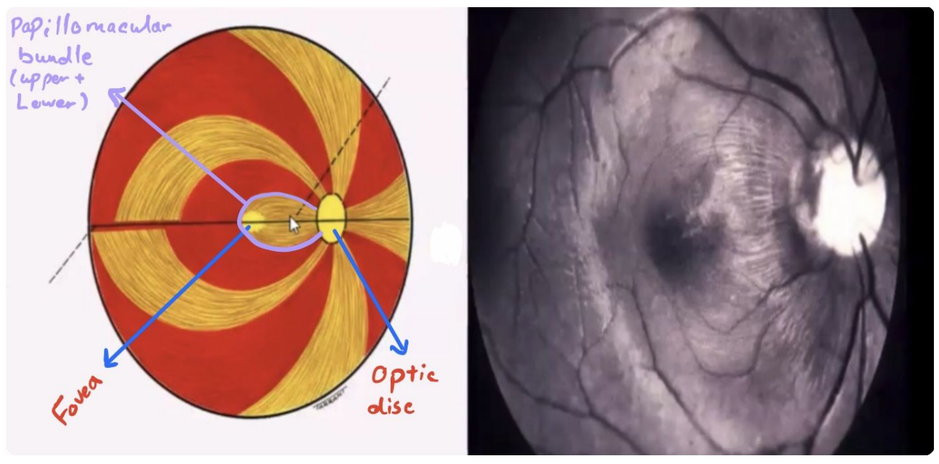




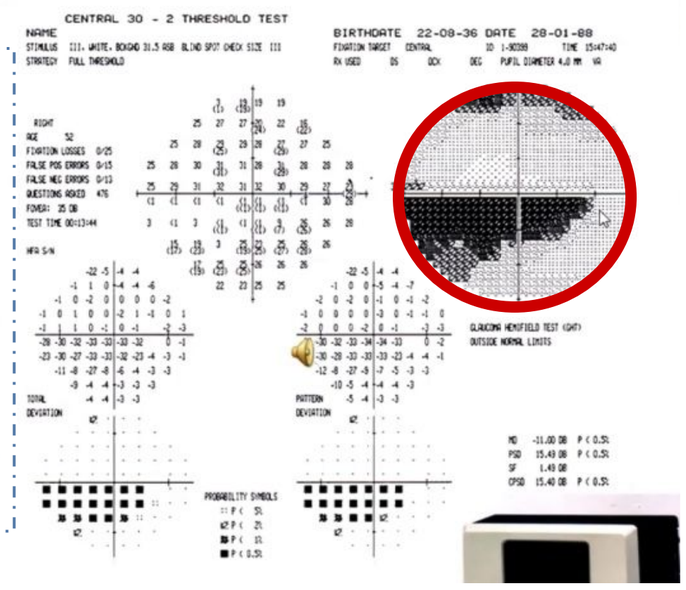
Symptoms:

- Initially asymptomatic. Patients won't seek medical advice early and present late because: The great majority of patients lack pain, ocular inflammation.
- Glaucoma starts with peripheral(navigational) vision involvement (NO central vision involvement), usually the center is preserved till the end of the disease. Much peripheral vision can be lost before the patient notices visual impairment.
- Usually detected on routine examination.
- Patients present late to the clinic when they have progressive loss of vision & their visual field is markedly affected.

Papillomacular bundle is responsible for central vision. In glaucoma it'll be affected late hence the late presentation.



Here, the visual field defect respecting the horizontal midline. But in neurological diseases it'll be respecting the vertical midline.



Glaucoma cont:

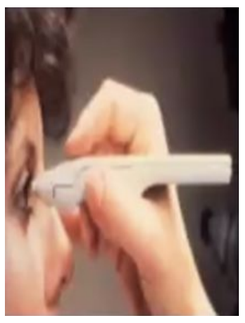
Signs:

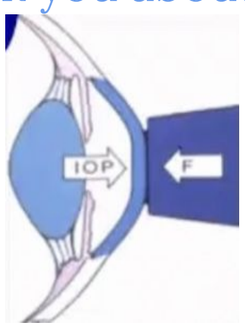
- Because glaucoma involves elevated pressure in the eye, routine measurement of Intraocular pressure is a valuable means of screening for glaucoma. Elevation of intraocular pressure can lead to optic nerve damage; therefore, examination of the optic disc is another way to detect glaucoma.
- Most of the world calls Glaucoma "blue water", however in some areas it can be called "black water" مثل السودان or even Swerek Disease "مرض سويرق لأنه يسرق النظر بهدوء ومن دون أعراض".
- **Glaucoma triad:**
  - **High IOP (most commonly, but not always MCQ),**
  - **Characteristic optic nerve head damage**
  - **And visual field loss.** secondary to nerve fiber layer loss.
    - In end stage glaucoma, patients lose their peripheral visual field resulting in what's called tunnel vision. Patients will tilt their heads toward you when you speak to them.

Neovascular glaucoma, with fibrous tissue

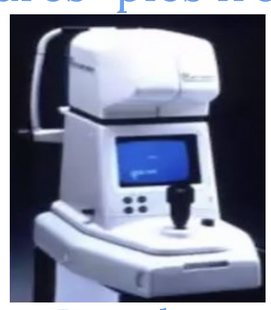
- IOP is the single factor to be controlled **(normal is 10-21 mmHg)**.
- Mean= 15.9mmHg ± 2 SD, IOP > 21.7 is abnormal.
- Factors affecting IOP:
  Age, sex, race, hereditry, diurnal and seasonal variation, blood pressure, obesity, drugs, posture, exercise, neural, hormonal, refractive error, eye movement, eyelid closure, inflammation and surgery.
- How to measure IOP "in the exam they may ask you about the pictures" pics from Prof.Essam slides:

Tonopen





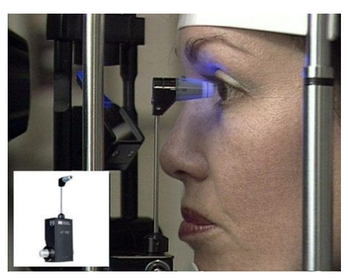
Applanation tonometry



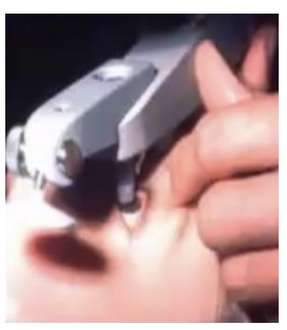
Pascal

I care

Pascal differs from Goldmann in which the corneal thickness is included in the result of IOP measurement.



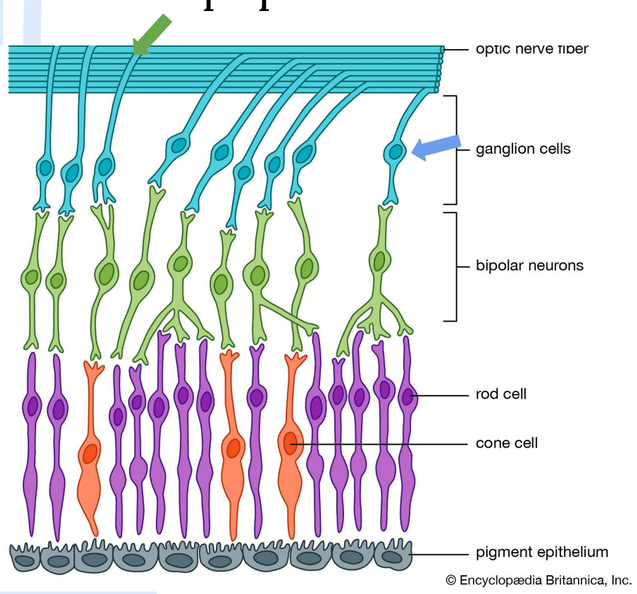
Goldmann



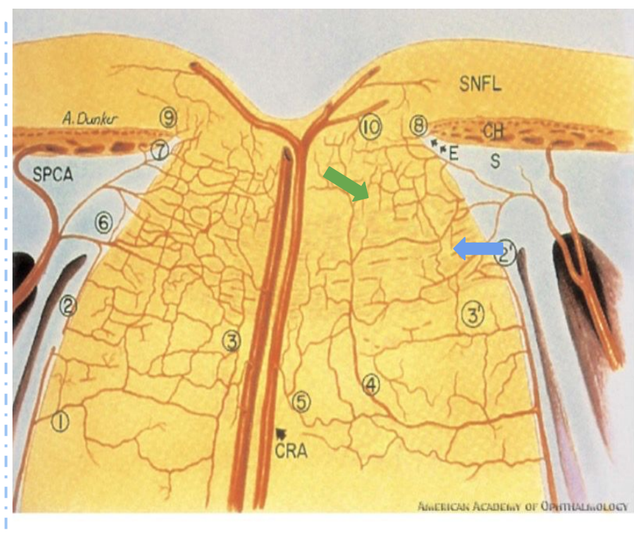
- In clinic, you need to
  - Examine the vision to assess the patient stage.
  - Measure IOP:
    - There are several methods to check IOP, most important is Goldmann Applanation (the gold standard method).
  - Use gonioscopy lens, to check the angle of the eye, where iris meet the cornea.

- **Factors that influence the retinal ganglion cell health:**

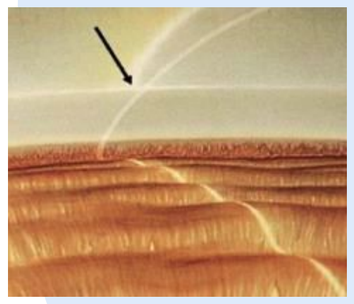
1. Increased intraocular pressure.
2. Excessive glutamate stimulation in the retina.
3. Increase in the inflammatory cytokines secreted by glial and microglial cells.
4. Aberrant immunity.
5. Retinal ischemia.
6. Apoptosis in the trabecular meshwork cells.



- The anterior retinal ganglion cells will start to move their axons in the outermost part of the retina (blue arrow) towards the optic nerve. As they move, the adjacent axons will appear and will move above (superficial) to the previous axons. (green arrow)
- Increase in IOP leads due compression of these axons, pressing the deeper peripheral axons towards the connective tissue = loss of peripheral vision as the first symptom.



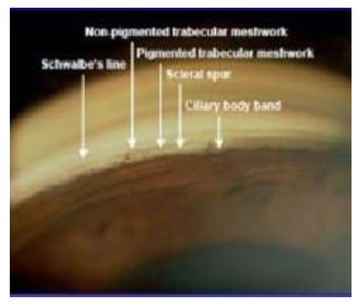
Glaucoma cont:

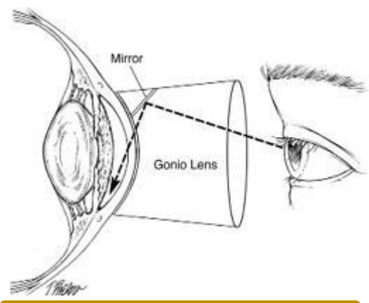


- Apoptosis in the trabecular meshwork of glaucomatous patients

Aqueous humor:

- **Active secretion:**
  - Na/K ATPase.
  - Cl secretion.
  - Carbonic anhydrase (available within the ciliary body and corneal endothelium).
  - The endothelium pumps fluid away from the cornea to keep the stroma dry.
  - The principal physiological function of the corneal endothelium is to allow leakage of solutes and nutrients from the aqueous humor to the more superficial layers of the cornea while at the same time pumping water in the opposite direction, from the stroma to the aqueous.
  - Accumulation of fluid within the stroma will result in a cloudy cornea instead of the normally transparent cornea.
  - For example, a patient underwent a penetrating keratoplasty and developed glaucoma. I want to give the patient a medication to stop the action of carbonic anhydrase pump in order to reduce the aqueous production. At the same time, the medication will stop the function of the carbonic anhydrase pump within the, and fluid will accumulate in the stroma (resulting in a cloudy cornea). In such a case, carbonic anhydrase pump inhibitors are not my 1st option.
- **Passive secretion:**
  - Ultrafiltration.
  - Diffusion.





Gonioscopy or zeiss gonioscopy lens:

Are we dealing with open or closed, acute or chronic?

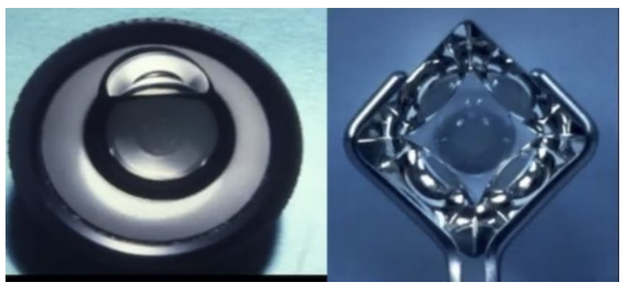
U should do gonioscopy and check the angle.

- Normal angle structures (important):
  - Schwalbe's line (SL).
  - Trabecular meshwork (TM).
    - Non pigmented.
    - Pigmented.
  - Sclera spur (SS).
  - Ciliary body.
- It is important to know these structures because if we do not see them, it means the angle is closed.
- In the aqueous humor pathway, the area of maximum resistance is between the lens and iris.
- **Open angle Vs closed angle**

Open angle: trabecular meshwork not occluded by peripheral iris.

Closed angle: trabecular meshwork occluded by peripheral iris.

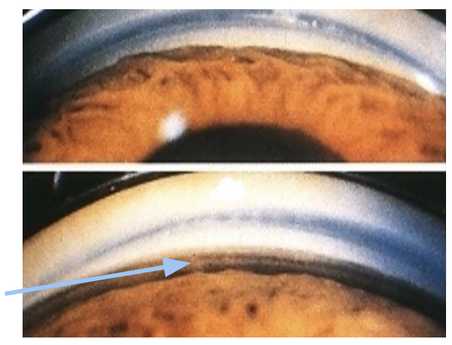
Instrument: gonioscope/goniolens. It is used to view the iridocorneal angle in case of glaucoma.



Gonioscopy lens (IMP)

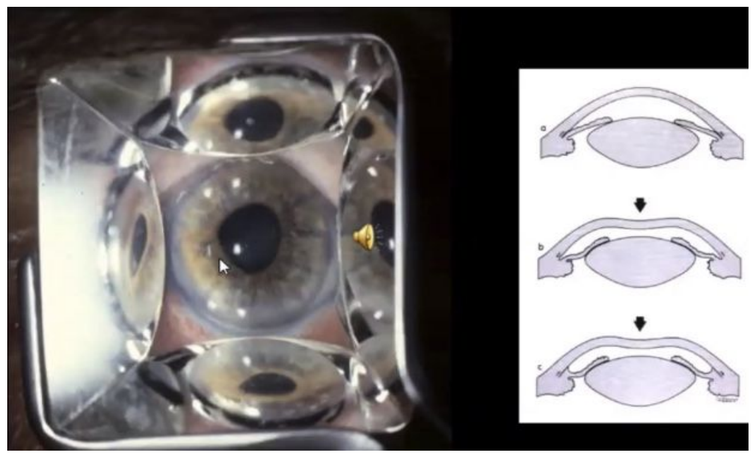
Above: no angle structure seen.

Below: when u press by the lens, it shows u that the angle is open.



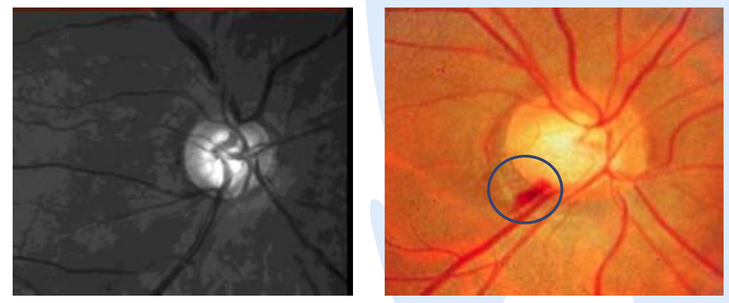
Left: When u put the gonioscopy lens in center, u can see the angle in all quadrants at the same time.

Right: "Indentation gonioscopy", when u indent by these lens u may open a narrow angle. So it can differentiate between open angle, closed angle and narrow or positional angle.



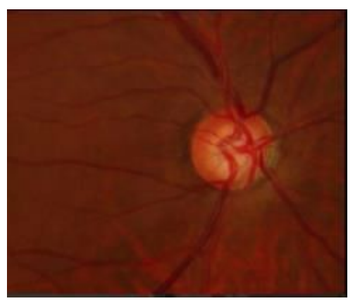
Glaucoma cont:

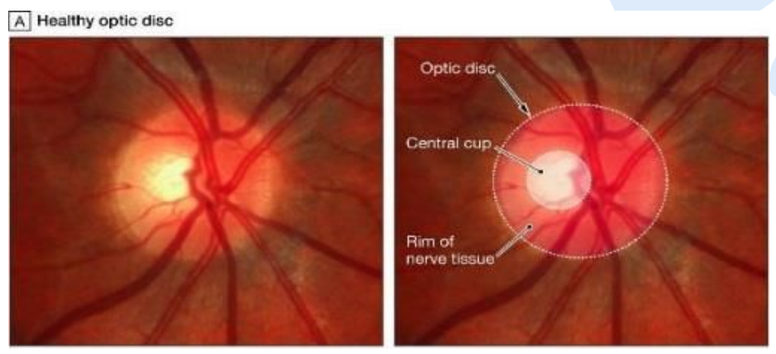
Optic Nerve Head (ONH) complex evaluation:



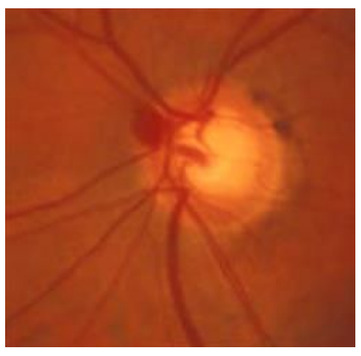
Disc hemorrhage in normal tension glaucoma

- You will definitely have cupping in your exam.
- In clinic do detailed optic nerve exam because you might have:
  - glaucomatous optic nerve head damage.
  - Anomalous disc.
  - Disc pallor because of CNS or DM.
- **What to evaluate:**
  - Disc margin and disc diameter.
  - Cup/disc ratio: (important)
    - How to estimate it? Take the vertical ratio; if more than 0.3 we should worry.
    - Normal value is 0.3, bigger cup = more nerve tissue loss!
    - Cause for large cups:
      - Glaucoma.

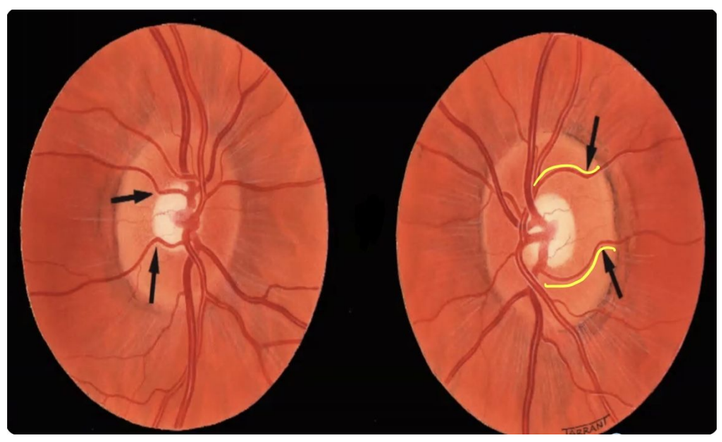




  - Neuro-retinal rim:
    - Neuro-retinal rim is the neural tissue in the disc.
    - The cup is almost circular. There are almost 1 million axons passing within the optic canal to go to the brain. These axons will form the optic disc. There is an empty space in the middle because 1 million axons cannot completely fill the canal. The space is called cup. When there is an enlargement of the cup, it means you are losing some axons because of glaucoma.
    - How to assess the cup?
      - 1- disc margin
      - 2- neuro-retinal rim. نسبة وتناسب
      - How much is the ratio of the cup to the disc margin vertically.
    - Disc size.
    - PPA (peripapillary atrophy).
    - NFL defect (nerve fiber layer).
    - Optic disc hemorrhage (important).
      - It happens commonly with normal tension glaucoma. you will see spot of disc hemorrhage at the disc margin. Most of the times, you need to pay attention to the cupping itself.

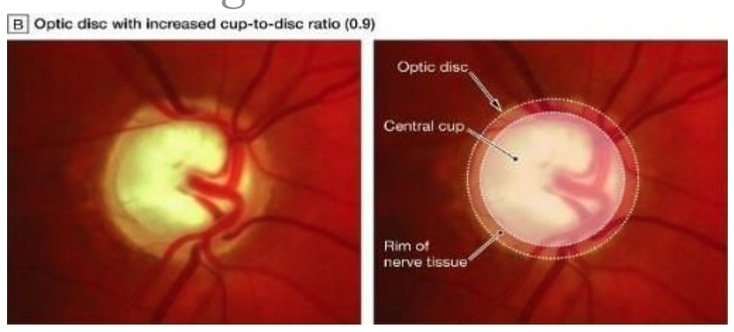


Optic disc hemorrhage



Left: normal physiological cupping (round pale area in the center of the optic disc).
Right: In glaucoma cupping we are looking for the kinking of the vessels (in the pic it's more than the pale area), and not just the pale area.

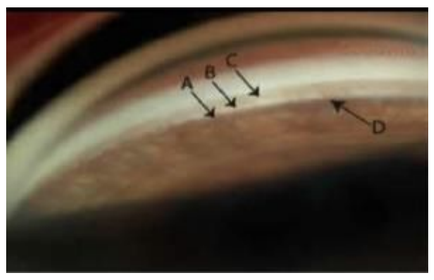
Notice: increase cupping, thinning of neuroretinal rim

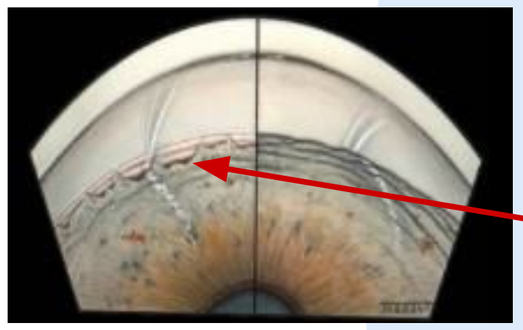


Glaucoma cont:

Classification of glaucoma:

Open





Closed

The open shouldn't have the tents like synechia

- **According to the etiology:**
  Primary, secondary and congenital.
  - Most common causes of secondary glaucoma: DM (neovascular glaucoma), uveitis (unilateral or bilateral).

Primary Vs Secondary:

Primary No detectable ocular or systemic abnormality, often bilateral and often familial.
Secondary predisposing ocular or systemic abnormality, often unilateral and often sporadic.

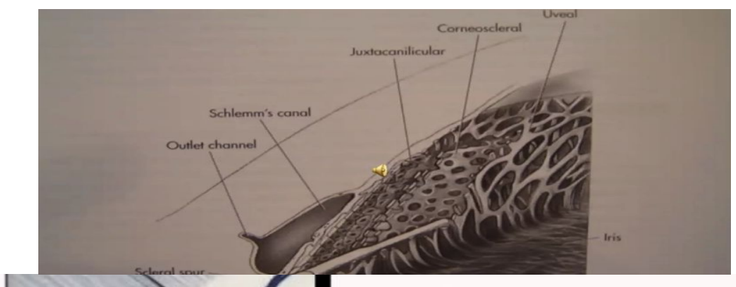
- **According to the appearance of the angle:**
  - ➢ It can be open, closed or combined mechanism.
  - ➢ Open → iris not covering TM.
    - In open angel you can see the angel structures (without pressing on the cornea by gonioscopy lens).
    - The problem with the pores in the TM.
  - ➢ Closed → iris covering TM
  - ➢ In peripheral anterior synechiae there will be adhesion between iris and angle (MCQs or SAQ).

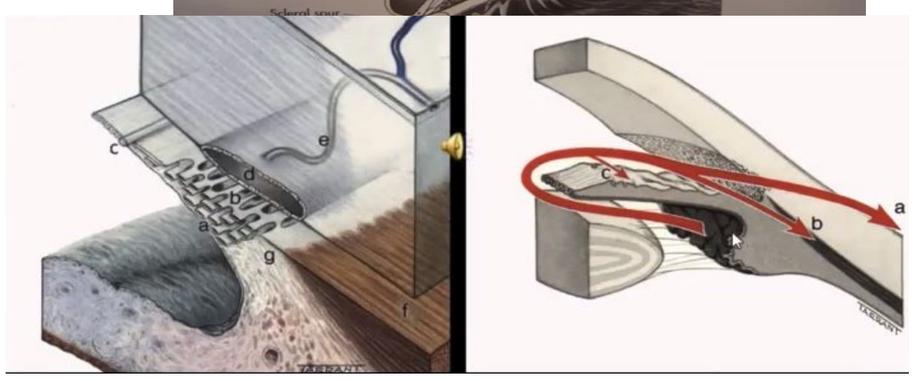
- **Open angle glaucoma:** Intrinsic defect in outflow tract.

"In case of open angle glaucoma the angle is anatomically open, but physiologically impaired, why? Because the layer of trabecular meshwork isn't functioning as required, so when the aqueous humor goes from the anterior chamber and circulate between the iris and the lens, it won't go through the trabecular meshwork"

Extra: "Aqueous humor exits the eye through a well-structured tissue called the trabecular meshwork (TM). after crossing the TM, aqueous humor reaches the Schlemm's canal, which drains directly to the aqueous veins (Aqueous humor outflow). Open angle glaucomas are thought to be caused by an increase in the resistance to aqueous humor drainage through the TM and/or Schlemm's canal. As a consequence, aqueous humor volume and IOP increase."

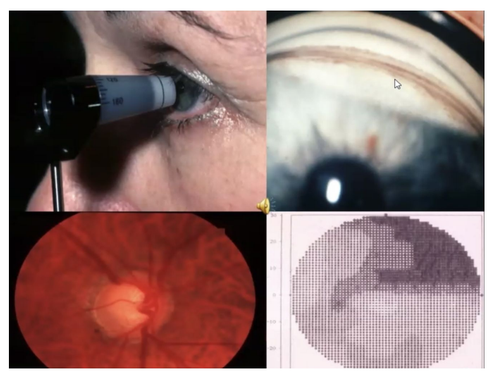
Source: https://journals.physiology.org/doi/full/10.1152/nips.01443.2003



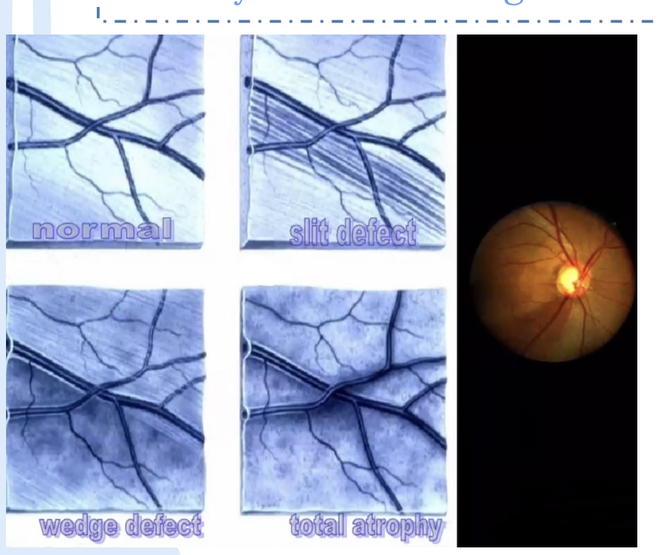


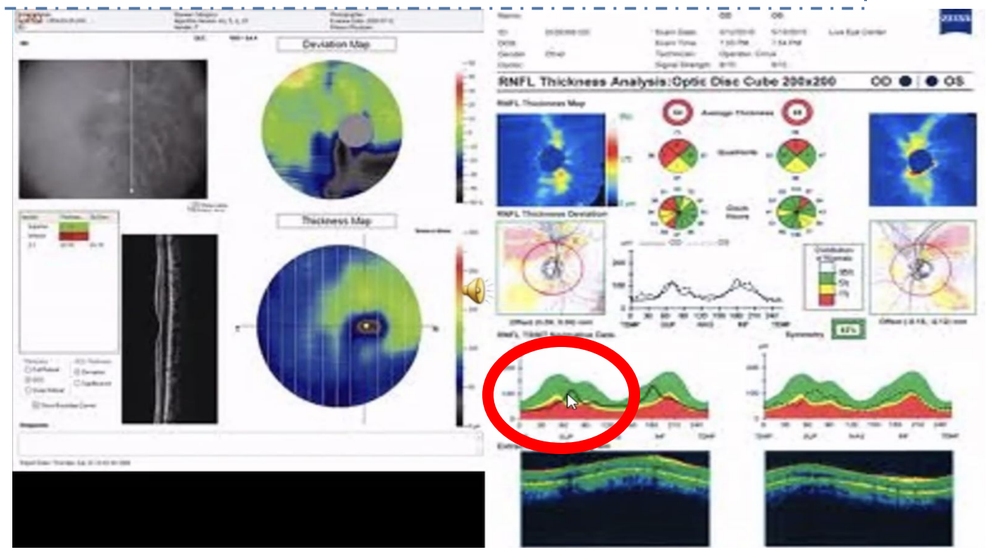
- **Risk factors of open angle glaucoma:** IMP
  - Family history
  - Black
  - Old age
  - Myopia
  - Diabetes
  - Hypertension

- **To diagnose an open angle glaucoma: (Diagnosis by exclusion)**
  1- Measure IOP (you should have high pressure by Goldmann applanation Tonometry)
  2- Look for optic disc (optic disc cupping)
  3- Visual field (defect respecting the horizontal midline)
  4- Gonioscopy (the angle is widely open)

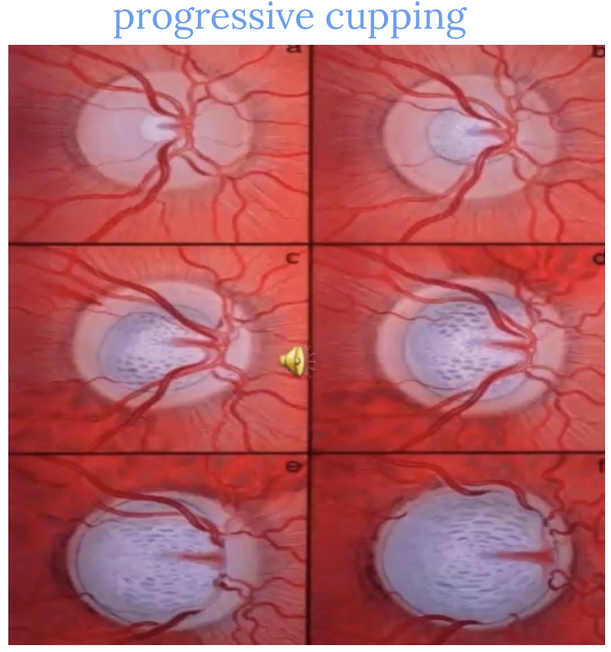


When you do Ocular Coherence Tomography (OCT), you can see the nerve fiber changes (see Pics below). Pic (2) the line below the green area = thinning of nerve fiber layer = advanced glaucoma.

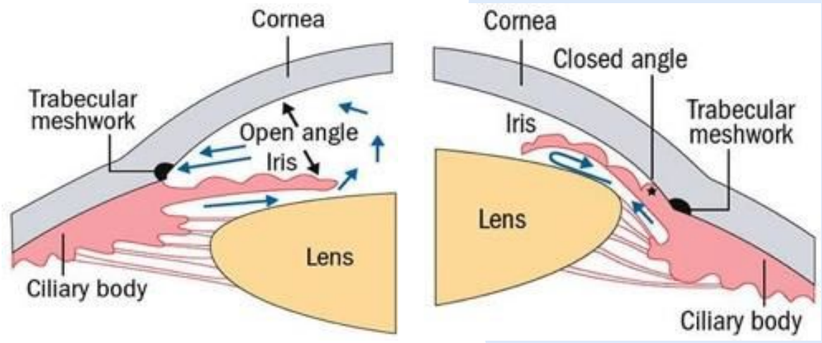




Here, you can see progressive cupping



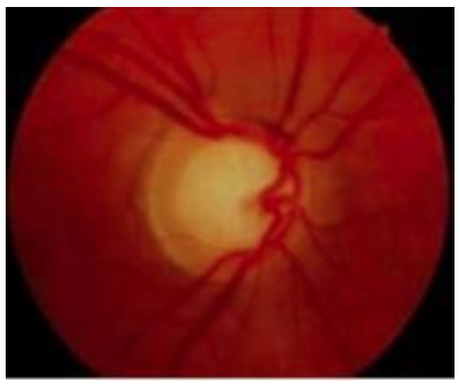
Glaucoma cont:



- Generally, if you have a glaucoma patient in your clinic, you need to decide the type of glaucoma, **open** or **closed** angle. Why? For management.
- Patient in which the structures are visible with gonioscopy, but is having high IOP, optic nerve head & visual field changes → **open angle glaucoma.**
- Patient in which the iris is rolling on the angle structures with gonioscopy lens → **angle closure glaucoma.**
- Sometimes, you will have the iris covering the angle structures. In this case, press the gonioscopy lens gently on the cornea. Why? We want to force the aqueous in the anterior chamber to go to the angle and push the iris posteriorly. If the angle will open with pressure, and you can see the structures, then this is appositional angle closure. If it opened and you see adhesions, then this is a chronic process and the patient is having a primary angle closure glaucoma.
- Why does the angel close? remember we said the maximum area of resistance in between the iris & lens. Sometimes, patients who have increased anterior posterior diameter of the lens (as in cataract for example), resistance will be higher. So aqueous is accumulating behind the iris. Then it will push the iris anteriorly. Iris will roll against the angle. So, instead of 2 microliters\min coming out, 1 microliter\min is coming out which means the is an accumulation of 1 microliter\min. This will push the iris & close the angel. With chronicity, adhesions will be developed.

Treatment:

- Treatment is aimed at reducing intraocular pressure by 3 modalities available:
  - Antiglaucoma medications.
  - Laser treatment: SLT (selective laser trabeculoplasty for open angle), PI (peripheral iridotomy for closed angle).
    - SLT is applied on the trabecular meshwork to stretch the pores of the sclerosed meshwork.
    - 2 different laser entities for 2 different glaucoma entities (be careful).
  - Glaucoma surgery
    - Surgically, we do peripheral iridectomy.

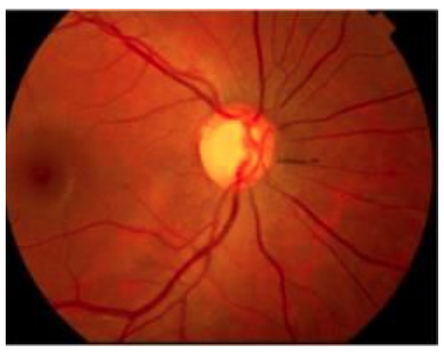


Diagnosis:
- Disc cupping

Investigation to confirm the diagnosis:
- Visual field examination.

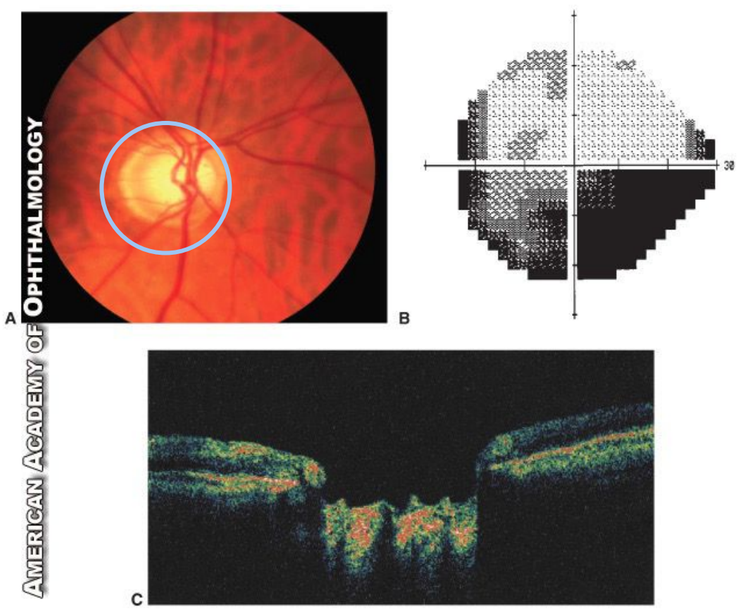
2 types of visual field defect:
- Nasal step.
- Arcuate scotoma.
- Peripheral visual field defect.

- Large cupping of the optic disc
- Superior retinal damage-> inferior visual field loss



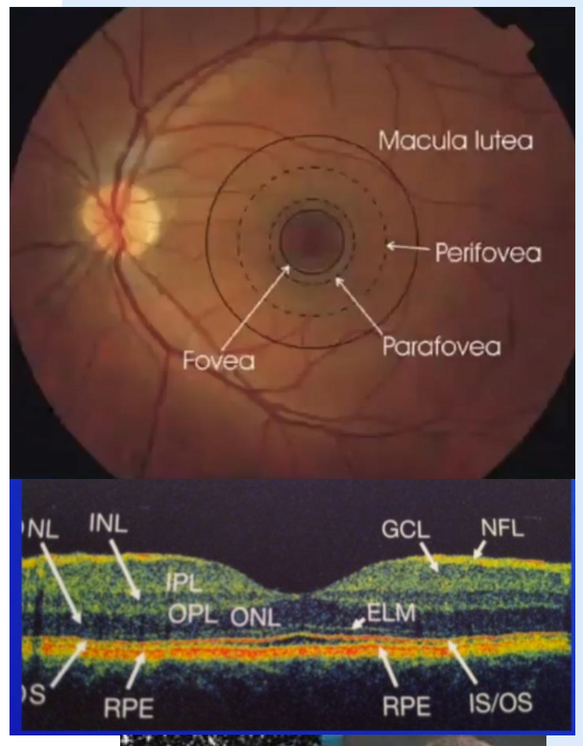
Diagnosis:
- Disc cupping

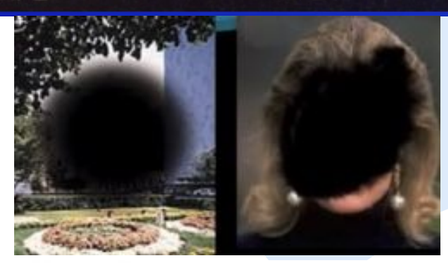
Next step:
- Goldmann applanation tonometry.



Macular degeneration (AMD):

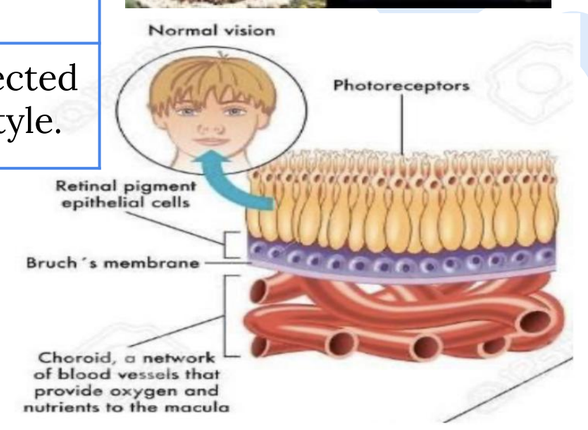
❖ **Prof. Essam revised the macular anatomy and said it's IMP to understand it.**





- ❖ Impaired central vision, while peripheral vision preserved (opposite to glaucoma) IMPORTANT, that is why patients with macular degeneration will complain faster than patients with glaucoma
- ❖ leading cause of irreversible central visual loss (20/200 or worse) among people with old age, Leading cause of legal blindness in developed world. "احفظوها زين"
- ❖ It Is known as age related macular degeneration because it comes with age. It affects the macula because it is highly used.
- ❖ Lipid deposition under the photoreceptors -> toxic reactions. Inflammatory element
- ❖ Symptoms:
  - ➢ Metamorphopsia distorted vision "الرؤية مزعوجة كأني أشوف اللي قدامي مكسر"
  - ➢ Micropsia: reduction of size of objects.
  - ➢ Macropsia: enlargement of size of objects.
  - ➢ Scotoma: visual field loss.
- ❖ **Risk factors:** important

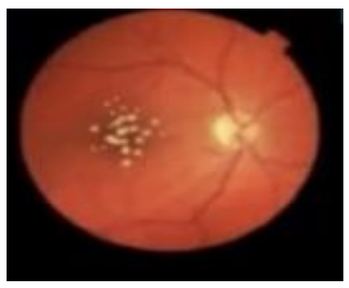
Uncontrollable risk factors	Risk factors you can control
Age (above 60), Race (Caucasian), gender (females), genetics (family history)	Smoking, high BP, high cholesterol, poor nutrition, UV light, Unprotected exposure to sunlight, excessive sugar intake, obesity, sedentary lifestyle.



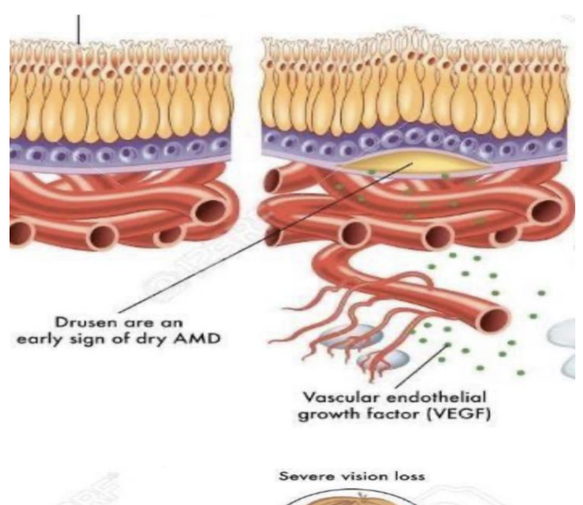
- ➢ Pathogenesis:

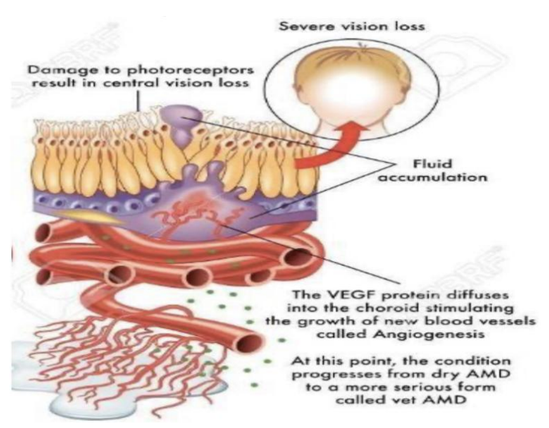
Macular involvement:

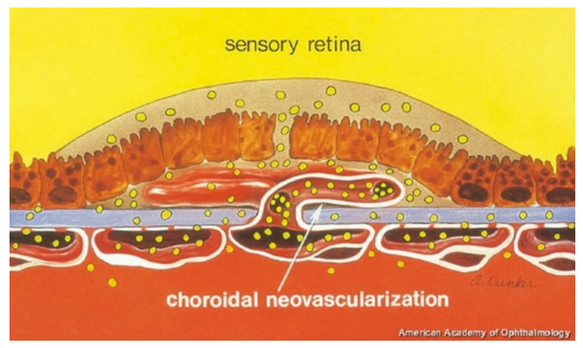
- ➢ Outer retinal layer "photoreceptors".
- ➢ Retinal pigment epithelium (RPE).
- ➢ Bruch's membrane.
- ➢ Choriocapillaris.



- ❖ Drusens (yellowish discoloration):
  - ➢ Drusens are lipid products from photoreceptor outer segment, found under retina.
  - ➢ Drusens are hyaline nodules (or colloid bodies) deposited in Bruch's membrane, which separates the inner choroidal vessels from the RBE.
  - ➢ Drusens may be small and discrete or larger, with irregular shapes and indistinct edges.
  - ➢ Patients with drusens alone tend to have normal or near normal visual acuity, with minimal metamorphopsia.
  - ➢ With age certain people will develop lipid material deposition under the photoreceptors this will cause some sort of toxic reaction to the photoreceptors.
  - ➢ Normally, the lipid deposits will be drained and absorbed by the choroid. If there is accumulation of lipid deposits, what will happen to the oxygenation from the choroid reaching the retina? decreased> ischemia> atrophy. So the patient won't be able to see.
  - ➢ Bruch's membrane will be stretched because of drusen accumulation. This is precipitated by hypoxia resulting in break of bruch's membrane.
  - ➢ In response to retinal hypoxia, vascular endothelial growth factor (VEGF) will be elaborated resulting in neovascularization, which is a bad thing in the retina, because blood vessels are abnormal and fragile, and almost always come with fibrosis and scarring.

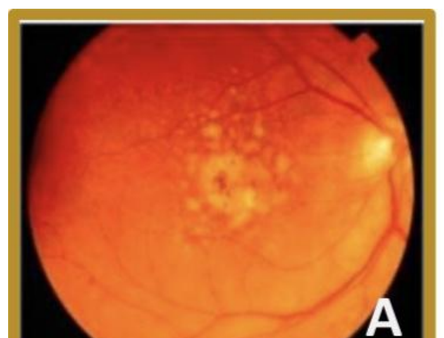


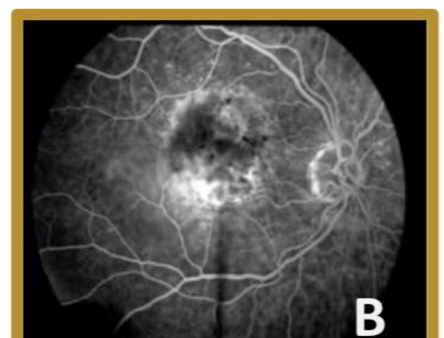




- ❖ Neovascularization:
  - ➢ With ischemia new vessels from choroid grow into the subretinal space forming subretinal neovascular membrane.
- ❖ Hemorrhage:
  - ➢ It can be multilayer: sub-retinal, sub-choroidal and sub/intra-vitreal depending on the extent that neovascularization reaches.
  - ➢ New vessels are bad either it continues to vitreous causing vitreous hemorrhages or it will bleed under the RPE causing sub-RPE hemorrhages or it will bleed under the internal limiting membrane and causes sub-hyaloid hemorrhage.
  - ➢ Hemorrhage into subretinal space or even through the retina into the vitreous results in significant loss of vision.
  - ➢ Diagnosis: age-related macular degeneration.
    2 risk factors: age & smoking.

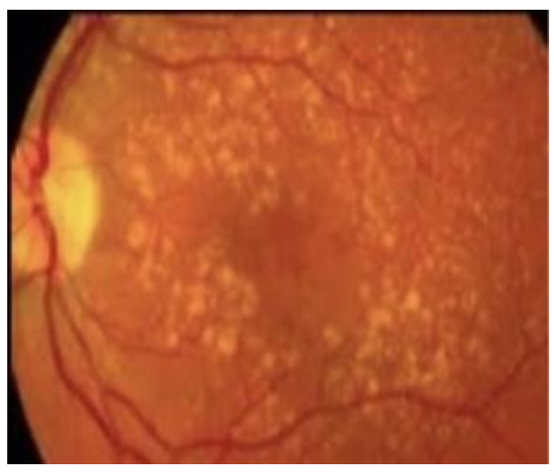
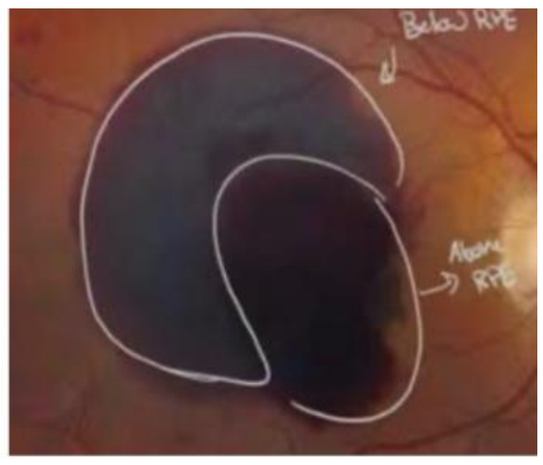
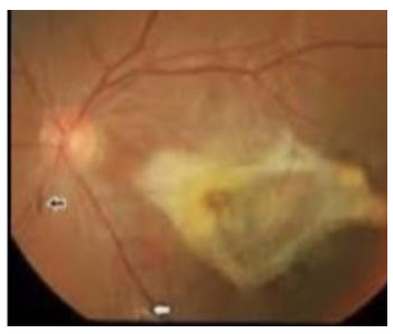
-Name of the investigation in pic. B is fundus fluorescein angiography.



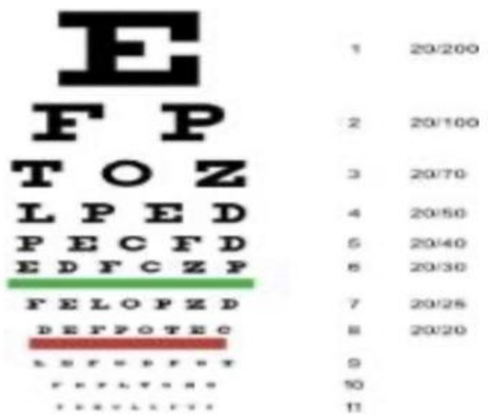
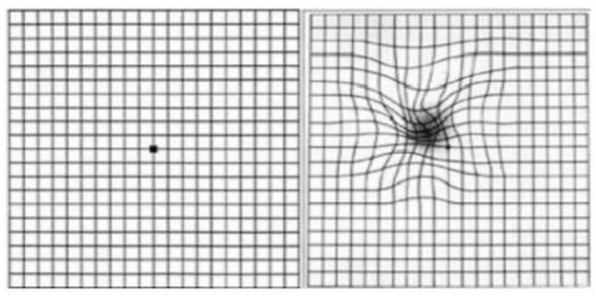
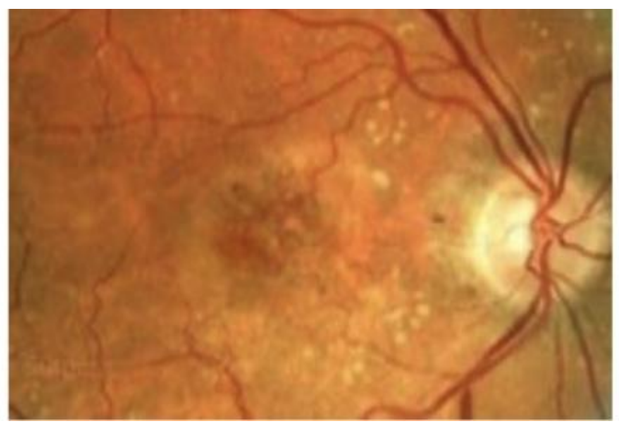
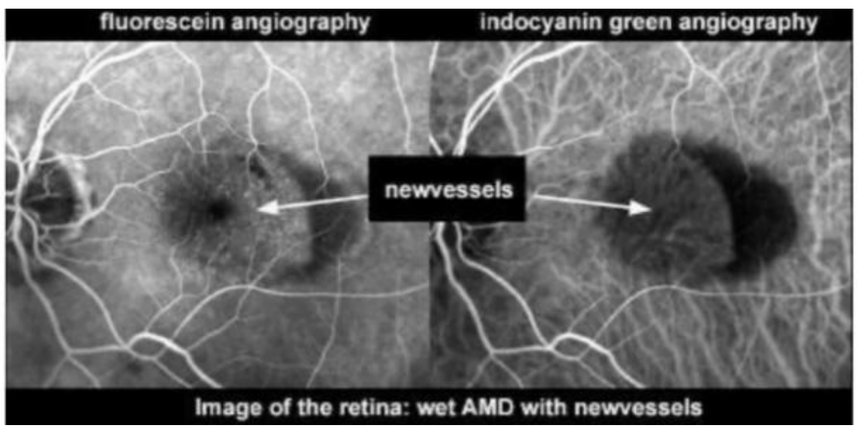
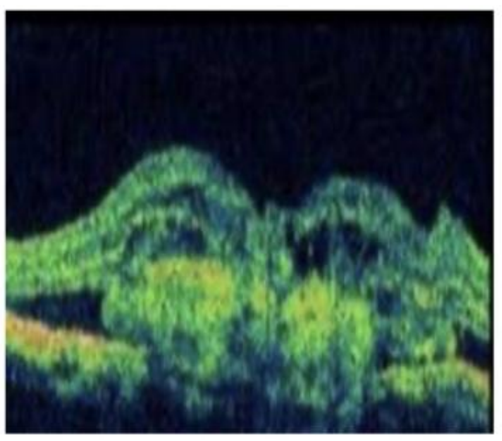


- Types:

<table>
<tr><th>Atrophic = dry</th><th>Exudative = wet</th></tr>
<tr><td>• 90% common, Slow progressive atrophy of RPE and photoreceptors.</td><td>• 10%, RPE detachment and choroidal neova.Blood vessels leak fluid.</td></tr>
<tr><td>• Often asymptomatic.
• Gradual over years.</td><td>• Rapidly progressive (weeks). It can be an acute or chronic cause of visual loss.</td></tr>
<tr><td>• Signs:
○ Drusen.
○ Geographic atrophy.
○ Photoreceptor degeneration.
○ Scotoma when light adapting</td><td>• Signs:
○ Choroidal (subretinal) neovascularization.
○ Preretinal hemorrhage (shiny blood).
○ Elevation of retina because of accumulation of blood.
○ Subretinal fibrosis.
○ Metamorphopsia (a classical complain of patients with macular degeneration). المريض يشوف صورة متعرجة
○ Central scotoma (because of blood and scarring within the macular area).
  </td></tr>
<tr><td>• Drusens are soft, mostly circular & deep.
• Its color is dark similar to that of the normal retina.

Dry type ; atrophic and lipofuscin accumulation
  </td><td>• Subretinal hemorrhage
○ If choroidal (subretinal) neovascularization bleeds, it will appear dark grey hemorrhage. That means it is under RPE (RPE is always dark in color).
○ If it is bight, that means it is above RPE.
  </td></tr>
<tr><td>• The accumulation of drusens (lipids) for so many years will create a barrier that prevents oxygenation to reach the retina (photoreceptors) causing atrophy of the photoreceptors & RPE resulting in geographic atrophy. Patient then can’t see.
</td><td>• Subretinal fibrosis
○ Remember we said the neovascularization almost always comes with fibrosis.
○ Once the patient has sub-retinal fibrosis, nothing can be done; thus, it needs urgent management.
  </td></tr>
<tr><td>WE can’t treat the patient because photoreceptors already are destroyed.</td><td>We can give the patient some medications to stop these blood vessels to invade the retina like Anti-VEGF agents or lasers.</td></tr>
<tr><td colspan="2">What’s the difference between dry and wet age-related macular degeneration? (extra)
• Dry ARMD is when you have drusen and macular RPE atrophy.
• Wet ARMD implies to when there is a break in Bruch’s membrane and choroidal neovascularization has grown up through it and bleed into the retina. BM not intact</td></tr>
</table>

- Diagnostic Tests;

Visual acuity	Amsler grid	Ophthalmoscope
• Because central vision will be affected first. 	• If the patient sees wavy lines, it means there is disorganization of photoreceptors secondary to accumulation of drusens. The black spot (blind spot) is caused by accumulated drusens, localized ischemia, localized loss of photoreceptors, hemorrhage & fibrosis. 	• Here you can see drusens, geographic atrophy, hemorrhage or fibrosis. Depends on the pt's stage. 
Fluorescein angiography	**ICG (Indocyanine Green)**	**OCT (Optical Coher Tomography)**
• It will show you the choroidal neovascular membrane.  		• To show subretinal hemorrhages and fibrosis, which is dense homogenous membrane under the retina. 

- Take proper history (age above 60-65).
- Exclude other causes of chronic visual loss (Cataract, glaucoma, diabetic retinopathy).

❖ Other diagnostic tests (mentioned by Prof. Essam):

Pupillary light reaction
Color vision
Photostress test
Laser interferometry

❖ Treatment:

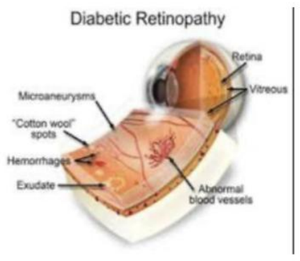
If dry:

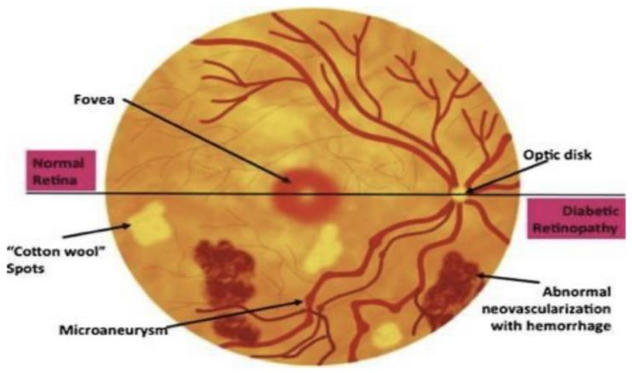
- ❖ Lifestyle: stop smoking, reduce UV exposure, Zinc & antioxidants.
- ❖ Low-vision aid.
- ❖ Monitoring with Amsler chart to follow up the progression (IMPORTANT).
- ❖ Observation.
- ❖ Laser photocoagulation, for neovascular membrane especially for the wet type.
- ❖ Anti - VEGF agents e.g.: avastin, Lucentis & eylea Wet type (stop the retina from signaling VEGF & as a result will stop formation of abnormal blood vessels).

- You don't need to know this: Verteporfin photodynamic therapy (PDT): injection of photosensitizer into systemic circulation followed immediately by laser targeting new vessels in macular area.

Diabetic retinopathy (IMPORTANT):

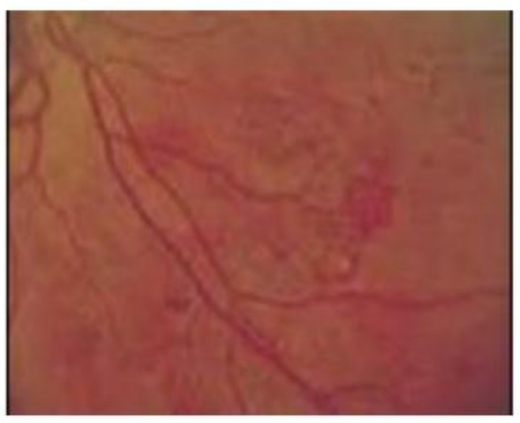
- Pathophysiology:
  - Microangiopathy which involves precapillary arterioles, capillaries and postcapillary venules.
  - 2 mechanisms: IMP
1. Microvascular occlusion.
2. Microvascular leakage.





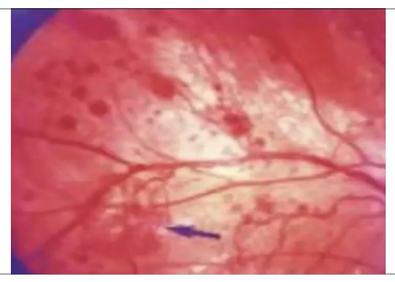
Microvascular occlusion

Thick capillary basement membrane; it means lumen is smaller.

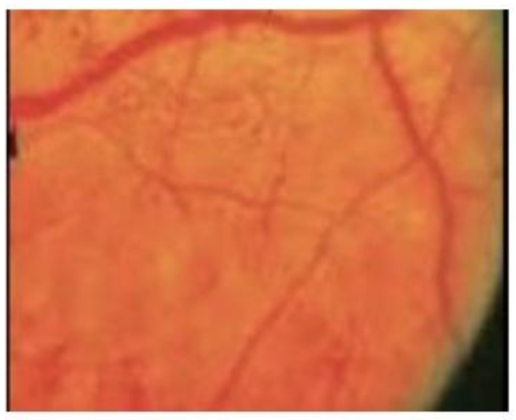


Abnormal blood vessels (fan-shape)

Capillary endothelial cell damage with changes in RBCs→ Retinal ischemia
→ AV shunt and neovascularization of the retina and around the optic disc.
So the cause of neovascularization of the retina which is seen in proliferative type is related to microvascular occlusion.



Change in red blood cell and increased viscosity in diabetic patients.



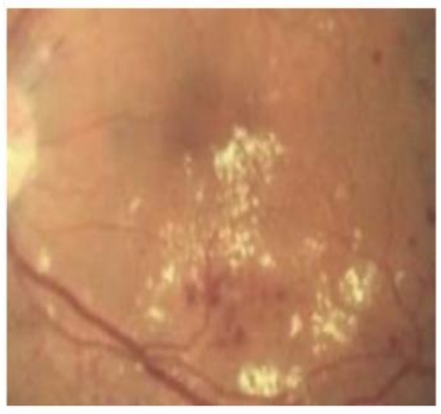
Abnormal blood vessels

- All of these will lead to decreased blood supply > retinal ischemia > neovascularization and AV shunt formation.
- The retinal surface is dry (there should be no fluid within the retina).

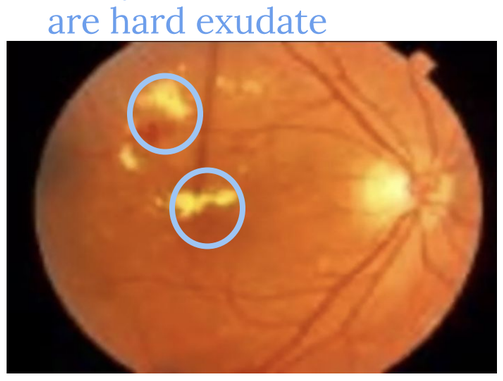
Microvascular leakage

Loss of pericytes between endothelial cells (then there will be gap) → plasma leakage into retina (retina should be dry) (with plasma there will be lipid leakage) → exudates and edema.

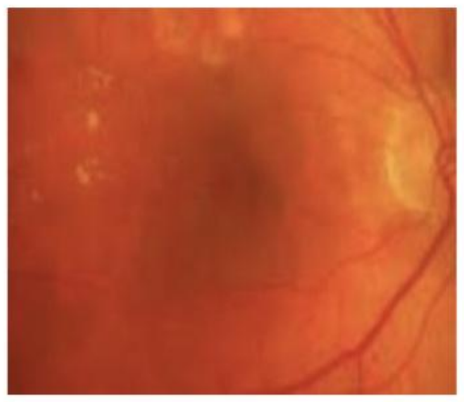
- Exudate of lipid in Retina.
- ill-defined exudate,no pattern.
- More superficial.



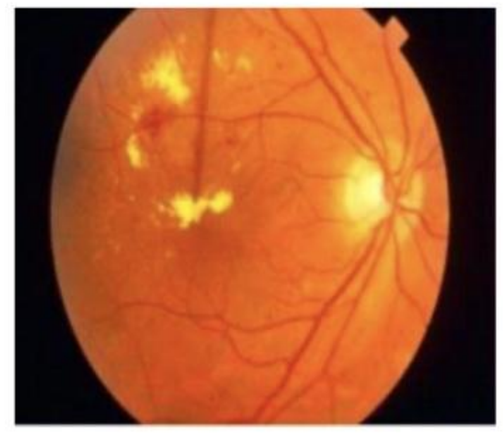
The yellowish deposits are hard exudate



- Retinal Edema.

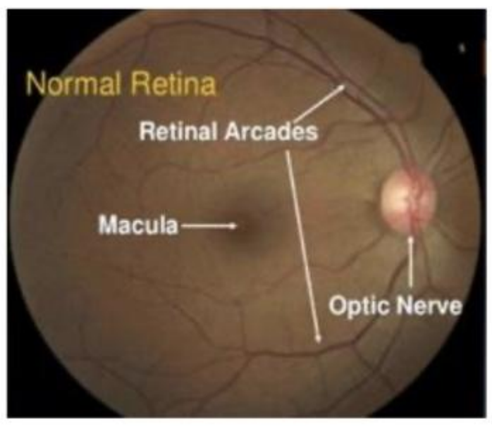


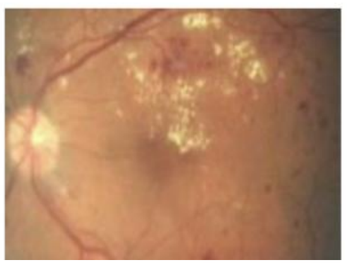
- Risk factors:
  - Duration of the disease is the most important factor and not the high blood sugar, pregnancy, nephropathy, poor metabolic control, smoking, HTN, obesity and hyperlipidemia, anemia.
- 2 Types of diabetic retinopathy: Proliferative with or without macular edema, non-proliferative with or without macular edema.
- Clinical classifications (IMPORTANT): you will have a slide in the exam.
  - Remember! The hallmark feature of non-proliferative diabetic retinopathy is micro aneurysms; while in proliferative diabetic retinopathy, it is neovascularization.
- Non proliferative diabetic retinopathy (NPDR):
  - Mild (micro-aneurisms, the only patients developed).
  - Moderate (exudates,hemorrhages as well as micro-aneurisms).
    - Hemorrhage in 1-3 quadrants is considered moderate.
    - Venous bleeding in 1 quadrant is considered moderate.
    - Hemorrhage in 3 quadrants along with venous bleeding in 1 quadrant is still considered moderate.



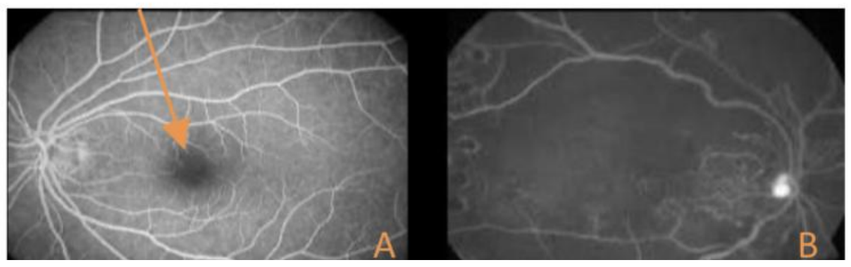
Diagnosis: non-proliferative diabetic retinopathy. Treatment: Laser photocoagulation.

- Severe (hemorrhages in 4 quadrantes, venous bleeding or slugging (means diameter of the vein is increasing) in 2 quadrates, intra-retinal malformation of blood vessels in 1 quadrate).
  - Memorize it like this: “HIV” H: hemorrhages, I: intra-retinal vascular malformation, V: venous bleeding.
  - The intra-retinal vascular malformation is deep within the retina (deep to the arcade).
  - Intra-retinal vascular malformation is also known as intra-vascular micro-vascular abnormalities (IRMA).
- Symptoms:
  - Asymptomatic
  - Decreased visual acuity: clinically significant macular edema & macular ischemia.
  - Causes of visual loss of pts w/ non-proliferative is macular edema or macular ischemia.



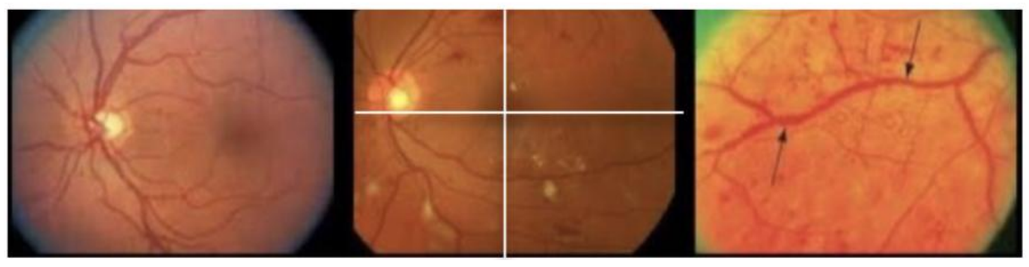


Clinically significant macular edema (CSME)



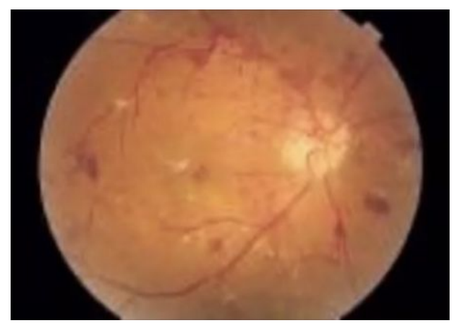
- Fovea or FAZ (foveal avascular zone).
- Fundus fluorescein angiography: Delineation of the vascularity of retina.

A. Normal.
B. Ischemic retina (dye will not pass through the ischemic area).

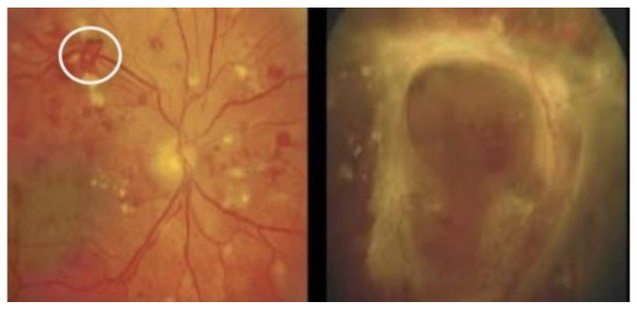


Mild Moderate

Hemorrhage and exudate, but no proliferation of vessels

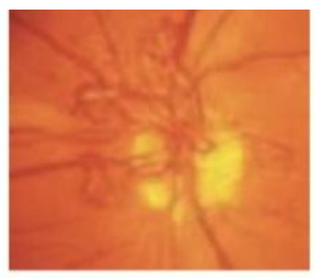


- Proliferative diabetic retinopathy (PDR):
  - Early (neovascularization, fan-like and disorganized).
  - Advanced (when it progresses & bleeds, it will form fibrosis which is attached to the retina. When it contracts it will pull on the retina causing tractional retinal detachment).
- Advanced proliferative diabetic retinopathy = there should be tractional retinal detachment.
- The neovascularization forms on the retinal surface (superficial).



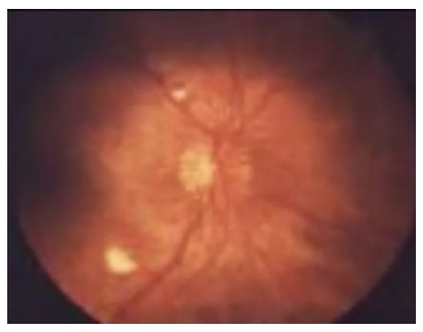
Early Advanced

  - Symptoms:
    - Symptomatic.
    - It can also cause macular edema & macular ischemia.



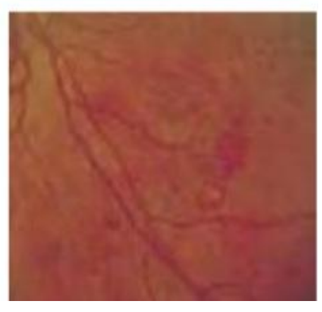
NVD

New vessels at the level of optic disc



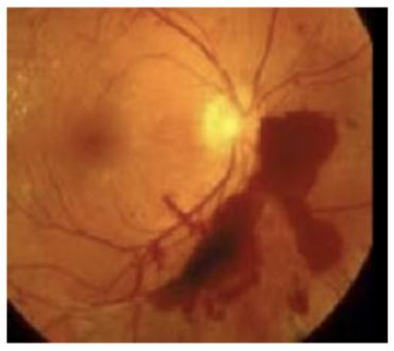
  - Neovascularization:

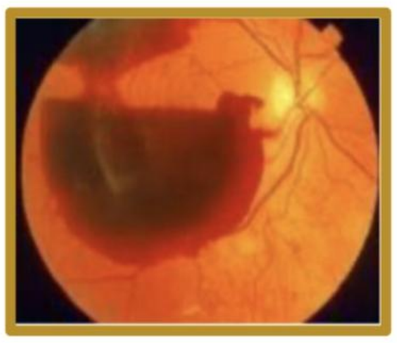
A. NVD: neovascularization of the disc.
B. NVE: neovascularization elsewhere.



NVE

- Fragile (intra-retinal or vitreous hemorrhage).
- Associated with fibrous proliferation which can cause at a later stage tractional retinal detachment (TRD).

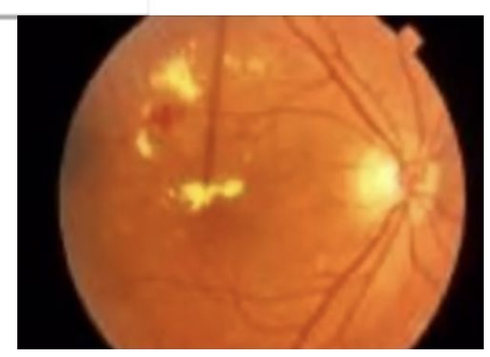




Vitreous hemorrhage.
Cause: Diabetic retinopathy.

Types	
Nonproliferative diabetic retinopathy (NPDR)	**Proliferative diabetic retinopathy (PDR)**
– Most patients (95%) have NPDR. This is the earliest stage of retinopathy and it progresses slowly. Because so many diabetic patients have NPDR, this stage is commonly described as "background retinopathy." The earliest signs of retinal damage arise from capillary wall breakdown, seen on the fundus exam as vessel microaneurysms. 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. – Recall the vascular section of this lecture: With worsening retinopathy and vessel damage, 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.	– With ongoing injury to the retinal vasculature, eventually the vessels occlude entirely, shutting down all blood supply to areas of the retina. In response, the ischemic retina sends out chemicals that stimulate growth of new vessels (fragile and easy to bleed). This new vessel growth is called neovascularization, and is the defining characteristic of proliferative retinopathy. Far fewer patients have proliferative retinopathy, which is fortunate as this stage can advance rapidly with half of these patients going blind within five years if left untreated. – The most common cause of blindness in diabetic patients is from macular edema. Macular edema occurs in about 10% of patients with diabetic retinopathy and is more common with severe retinopathy. On exam the macula looks mildly elevated, and you can see past evidence of edema in the form of yellow- colored "hard exudates." These exudates are fatty lipids that are left behind after past macular swelling subsides.

❖ Diabetic Macular Edema:
  ◇ Retinal edema threatening or involving the macula.
  ◇ Evaluate: location of retinal thickening relative to the fovea and the presence and location of exudates.

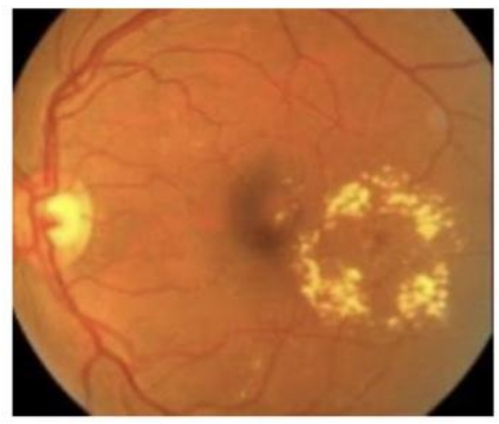


  ◇ In clinically significant macular edema:

- We have 2 classes because the management is different.

A. Focal macular edema:
   - Circinate ring, exudate in a circular fashion.
   - Treated with focal laser.

B. Diffused macular edema:
   1. Haphazardly diffused exudates and thickening → diffuse macular edema.
   2. Treated with grid laser or injection.



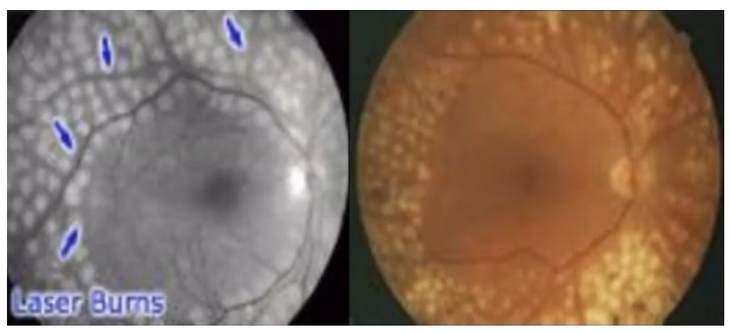
Circinate ring in focal macular edema

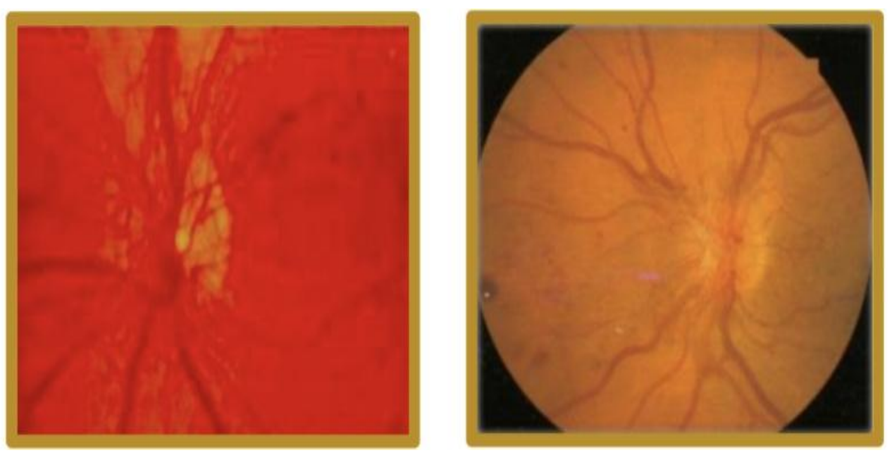
  ◇ Treatment:
   - laser > For PDR → pan retinal photocoagulation (treat the hole retina except the macula).
   - Intravitreal steroid injection.
   - Intravitreal anti-VEGF injection. (Avastin, Lucentis, Eylea).
  ◇ Pars plana vitrectomy.
- For patients having vitreous hemorrhage or tractional retinal detachment.

Management of diabetic retinopathy:

❖ NPDR > Observation (no treatment expect control the blood sugar).
❖ PDR > Pan retinal photocoagulation (PRP). The laser changes the ischemic retina to anoxic retina and this will decrease the vascular endothelial growth factor responsible for the formation of new vessels > stops the neovascularization.
❖ Macular edema > Focal or Grid laser, injections of anti-VEGF can decrease the edema.

Laser marks from the PRP

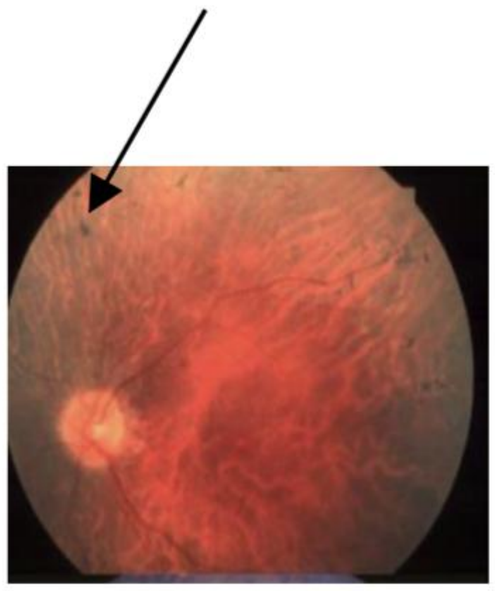
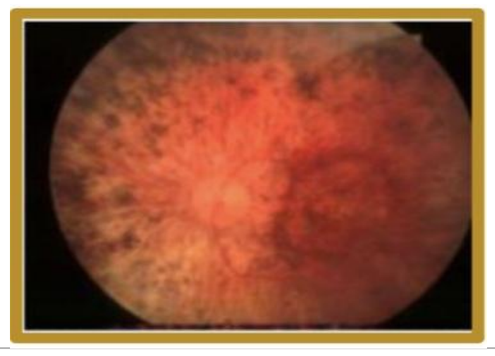
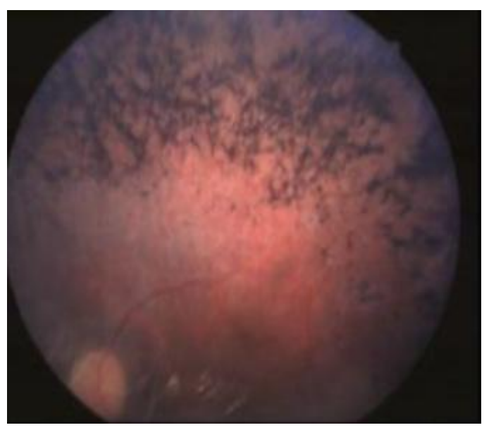




Diagnosis: proliferative diabetic retinopathy (neovascularization at the optic disc).
Management: Pan-retinal photocoagulation.

Retinitis pigmentosa "العشى الليلي":

- General characteristic:
  - A group of genetic disorders affecting the retinal ability to respond to light.
  - Most are legally blind (central visual field of less than 20 degrees) by 40s.
    - Legal blindness is 20/200 vision & worse.
  - X-linked recessive: Males: more often and more severe.
    - Females: carry the genes and experience vision loss less frequently.
  - Target photoreceptors and associated with pigmentary changes in the RPE, which may be primary or secondary to the photoreceptor loss.
  - The retina has 2 types of cells that gather light: rods and cones. The rods are around the outer ring of the retina and are active in dim light. Most forms of RP affect the rods first. So night and peripheral vision will be affected. Cones are mostly in the center of the retina, they help in seeing color and fine details. When RP affects them, there'll be slow central vision loss and inability to see colors.
- Symptoms: Slow loss of vision:
  - Nyctalopia: loss of night vision, sometimes with progression > tunnel vision.
  - Tunnel vision "loss of peripheral vision".
  - Blindness.
  - Photosensitivity.
  - Low or blurry vision at night especially while driving.
  - Very slow dark-light adaptation.
- Signs:
  - Visual acuity varies from: 20/20 to no light perception (NLP) at all.
  - +-APD (afferent pupillary defect).
  - PSCC (posterior sub-capsular cataract).
  - RPE hyperpigmentation (bone spicules) it is the dark part of the eye, alternate with atrophic regions.
  - Attenuation of the arterioles "thinning".
  - Waxy pallor of the optic nerve head.
  - Cystoid macular edema (CME) in severe cases of RP.

RP stages and signs			
Early stage	2nd stage	3th stage	4th stage "severe"
Black spots in periphery "bone spicules, why? RPE clumping" (black arrow) 	• bone spicules Increased more and more. • Start to loss photoreceptors. • Waxy disc appearance. • Attenuation of arteriole. • Diagnosis: RPE hyperpigmentation (bone spicules) & waxy disc appearance. • Test: VF, color testing. • Treatment: carbonic anhydrase inhibitors. • Retinal changes secondary to photoreceptors loss. 	• More loss of photoreceptors. • More clumping of RPE. 	• Severe photoreceptors loss. • Tunnel vision. • Severe RPE loss and clumping. • Bone spicules. • Waxy pallor. • Circumferential involvement of retina. It will spare the macula. • Cystoid macular edema.

- **Investigation:**
  - **Visual field test** (it shows constricted visual field & in advanced stage tunnel vision).
  - Color testing (mild blue-yellow axis color defects).
  - Dark adaptation study (reduced contrast sensitivity relative to VA).
  - **Genetic subtyping** to confirm the presence of the particular gene-defects.
  - **Optical Coherence Tomography (OCT)**, to look for (CME), to confirm damage of photoreceptors and to show the extent of their loss. .
  - FFA florican angiography.
  - **ERG electroretinography** IMP is an eye test that detects function of the retina including photoreceptors, to confirm the dysfunction or loss of functionality of photoreceptors.
  - EOG electrooculography to record eye movement.

- **Systemic associations:** rule out systemic association (IMPORTANT)

  - Hearing loss & RP:
    - Usher syndrome (IMPORTANT)a condition characterized by partial or total hearing loss and RPE (common).
    - Alport syndrome is a genetic condition characterized by RP, kidney disease, and hearing loss.
    - Refsum disease is an autosomal recessive neurological disease that results in the over- accumulation of phytanic acid in cells and tissues.

  - Kearns-Sayre Syndrome (IMPORTANT)
    - External ophthalmoplegia (thus restricted ocular motility).
    - Lid ptosis (unilateral or bilateral).
    - Heart block.
    - Pigmentary retinopathy.

  - Abetalipoproteinemia is a disorder that interferes with the normal absorption of fat and fat- soluble vitamins)↓vitamin A →RP

  - Mucopolysaccharidoses.

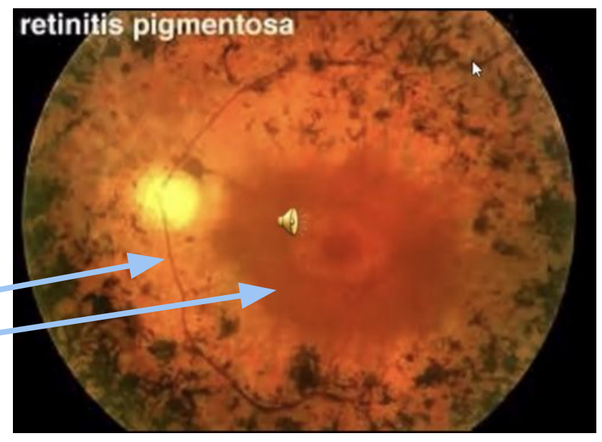
  - Bardet-Biedl syndrome genetic disorder characterized principally by obesity, RP, pigmentosa, hypogonadism, and kidney failure in some cases.

  - Neuronal ceroid lipofuscinosis lysosomal storage disorders characterized by dementia, RP, and epilepsy.

- **Treatment:**
  - Unfortunately, nothing can be done to prevent the progression of the disease, but associated ocular problems can be treated:
    - CAI: CME (carbonic anhydrase inhibitor for CME).
    - Vitamins.
    - Cataract: surgery.
    - Low vision aids.
    - Gene therapy.

Prof. Essam said it's very important pic for the SAQs Exam:

You can see:
- Bone spicules appearance of the pigmentation in the periphery.
- Very pale optic disc.
- Attenuated vessels.
- Macular star.



Dr notes

- What other symptoms will appear to a patient with chronic visual loss?
  - increase in severity
  - loss of visual acuity or field
  - involves both eyes -> loss of daily function
  - HE WILL NOT HAVE :
    - NO PAIN (ACUTE)
    - NO RED EYE (ACUTE)
- How do we see a candle or a car?
  - light rays reflected off of the candle in divergence fashion and it will pass freely the cornea -> lens-> focused in the macula.
    - Divergence: light will fan out or spread (in nature, light is always divergence)
    - Convergence : is the physical joining together of light rays. Light rays tend to come together at a point (called point of convergence) from different directions. [ex. magnifier]
- what provides the eye with refraction?
  - cornea 40 diopters
  - lens 20 diopters (More important than cornea. Due to accommodation, it can provide up to 40 diopters)
- what is the average dioptric power of the eye?
  - 60 diopters
- name conditions that can disturb the ocular medium (increases opacity)?
  - cataract

Dr notes

Very important questions for the exam, that were asked at the end of the lecture by Prof. Essam.

Q1: What are the indications of cataract surgery?

1- The patient needs. For example, an engineer or an IT whose work got affected by his condition.
2- Visual loss.
3- Therapeutic indications, which means we remove the lens to treat other diseases. Like age-related macular degeneration or diabetic retinopathy. For example, in diabetic retinopathy we might not be able to see the retina bc of the lens so we remove them to treat the retina.
4- When the lens complicate (Cataract causing Phacomorphic or Phacolytic Glaucoma) .

Q2: How can the cataract cause glaucoma?

- When the lens increase in size, it pushes the iris and make pupillary block and angle closure, it's called **(Phacomorphic Glaucoma).**
- In leaking mature or hypermature cataract, the protein leaks from the lens into the anterior chamber, it causes anaphylactic reaction, so the macrophages will block the trabecular meshwork >reduce the drainage > leading to secondary open angle glaucoma, **(Phacolytic Glaucoma)**.

Q3: What is the most important risk factor for Diabetic retinopathy?

Duration of diabetes, (Controlling the diabetes has its effect on the development but not as the duration).

Q4: What are the types of diabetic retinopathy? And what is the treatment?

- Proliferative with or without macular edema.
  Treatment: Pan-retinal Photocoagulation (PRP).
- Non-proliferative with or without macular edema.
  Treatment: observation/no treatment.
  Treatment of macular edema: Focal & Grid laser, anti-VEGF injections (Avastin, Lucentis, Eylea).

Q5: How does the PRP work?

- The laser changes the ischemic retina to anoxic retina and this will decrease the vascular endothelial growth factor responsible for the formation of new vessels > stops the neovascularization.

Practice Questions

Q1. Which one of these in NOT an indication of photocoagulation surgery?

A. Micro aneurysm.
B. Neovascularization elsewhere.
C. Neovascularization on optic nerve.
D. Significant macular edema.

Q2. A 45-year-old male with a history of renal transplant on steroid therapy complaining of gradual painless diminution of vision both eyes, on examination there is lens opacity. What is the most likely diagnosis?

A. Nuclear cataract.
B. Dot cataract.
C. Posterior subcapsular cataract.
D. Cortical cataract.

Q3. 75 years old man is complaining of decrease in visual acuity for the last 5 years, on examination red reflex was present, there is retinal pigment changes and retinal atrophy, what is the diagnosis?

A. Retinitis pigmentosa.
B. Retinal detachment.
C. Macular degeneration.
D. Proliferative retinopathy.

Q4. A 30-year-old male presented with night blindness, tunnel visual field, cystoid macular edema and cataract. What is the diagnosis?

A. Vitamin A deficiency.
B. Retinitis pigmentosa.
C. Retinal detachment.
D. Diabetic Retinopathy.

Q5.A 65 years old male presented with gradual reduction in his central vision, which one of the following is the most likely diagnosis?

A. Central retinal artery occlusion.
B. Central retinal vein occlusion.
C. Age related macular degeneration.
D. Retinal detachment.

Answers:

1:A 2:C 3:C 4:B 5:C