Chronic Visual Loss

Objectives:

- Not given.

[Color index : Important | Notes | Extra]

Resources: Slides+434team+Notes+Lecture notes of ophthalmology.

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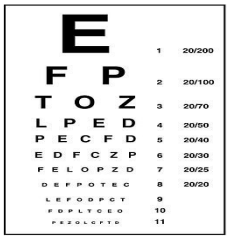
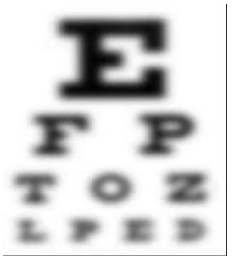
Chronic visual loss:



- **Definition**: Slowly progressive painless visual loss. (chronic means within months to years).
- Vision: So how can we assess the vision?
  1. Quantity: VA (Visual acuity)
  2. Quality (like if someone has a 20/20 vision but he can't see sharp details of objects): VF, clarity of vision, color vision



- Causes: (Always be systematic- امشوا بترتيب الأناتومي- cornea, iris, lens etc)
  - Refractive
  - Cornea
  - Lens
  - Vitreous
  - Retina
  - Optic Nerve
  - Neurologic

- One should recognize the normal first to be able to identify the abnormal :
  - Normal macula.
  - Lens clarity (normally it has a shade if you don't know this normal appearance you may think it is cataract).
  - Optic nerve head.
  - Normal retina.

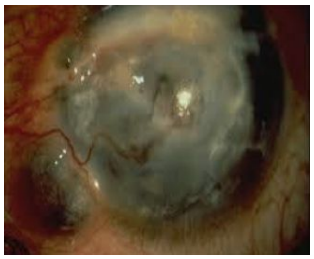
Refractive:

- Mostly in young patients
- Myopia, hyperopia or astigmatism
- Amblyopia!! (كسل العين: brain tends to ignore the weak eye, brain will be confused, thus can't fuse images)
- Signs: Normal exam. Refraction needed to show errors (everything will be normal except for visual acuity)
- Rx: Glasses, CL, Refractive surgery
- NB: lenticular causes needs cataract surgery

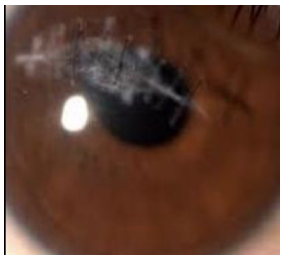
Cornea:

- Scar: trauma, infection (contact lenses might scratch your cornea and cause infection so be aware!)
- Hereditary: corneal dystrophies, keratoconus (munson sign: V-shaped indentation observed in the lower eyelid when the patient's gaze is directed downwards)
- Signs: corneal scar, bulging corneal, stromal opacities. Might have some conjunctival injection with chronicity
- Rx: Refraction, CL (soft or hard), corneal cross linking, keratoplasty (زراعة قرنية)

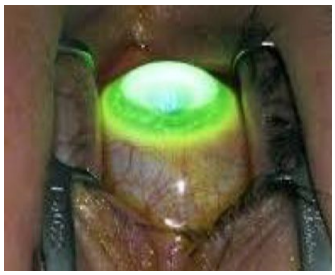
Keratoconus

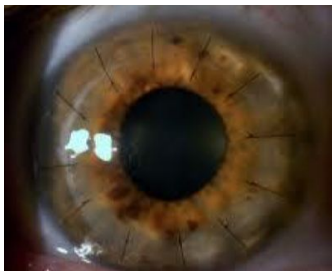


Stromal opacities

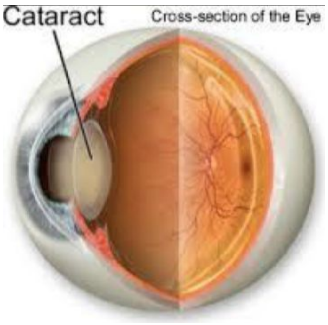


Corneal scar



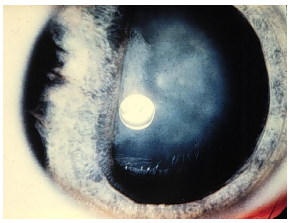


Lens:

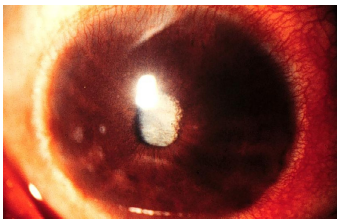


- Lens contains proteins and when they become disorganized, it'll lead to opacification!
- Cataract is the commonest cause of treatable blindness in the world. (posterior cataracts cause more visual complaints than anterior)
- Definition: Cataract is the name given to any light scattering opacity (vision opacification) within the lens wherever it is located, when it lies on the visual axis or is extensive; it gives rise to visual loss.

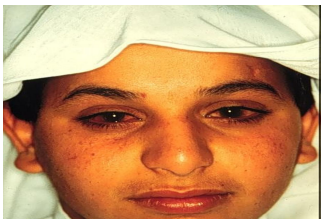
- Causes:
  - Age related (when we age, hair will turn grey, skin will wrinkle, and lens will cataract! Elasticity is lost because of protein disorganization -> loss of accommodation -> presbyopia and this is normal after the age of 40)
  - Metabolic (like diabetes, very imp!)
  - Traumatic (penetrating -> will breach the capsule, and non-penetrating -> cause disorganization of proteins)
  - Congenital
  - Drugs (like steroids)
  - Inflammation (like uveitis)
  - Ocular (like retinitis pigmentosa العشا الليلي)
  - Neoplasms: Melanoma or Retinoblastoma



Iris dilation "It is detached from its common position"



The pupil has not dilated => synechia formation "most common cause of it is inflammation". => caused by uveitis.



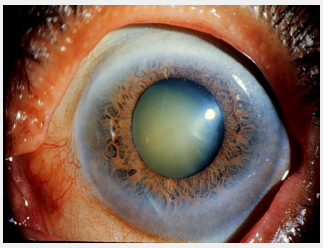
Steroid induced cataract:
This pt is case of vernal(allergy) keratoconjunctivitis severe allergic inflammatory disease common in the southern areas, treated by steroids, but when overdosed cataract (post. Subcapsular) or glaucoma can result.

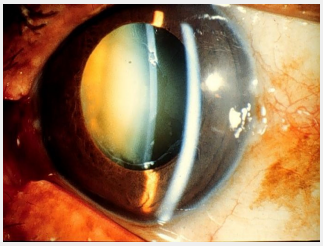
The treatment is disodium cromoglycate and antihistamine(systematic & local).

Clinical classification

Types Based on Morphology (anatomy):

Nuclear



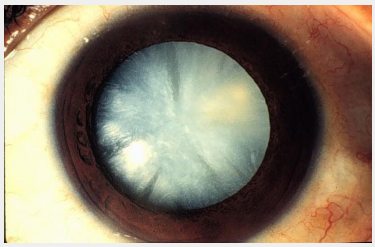


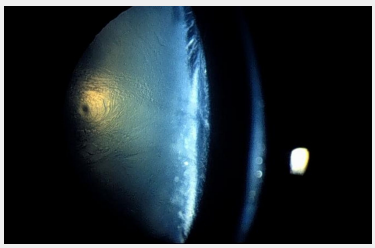
Nuclear Sclerosis.

Early stages of cataract: new fibers compress old fibers causing sclerosis. Associated with Myopia.

★ 2nd pic, the left side kind of yellow (opaque) is the nucleus of the lense (sclerosis happens to this nucleus, hence the name), u can see 2 vertical lines (left one is the ant. Capsule, right one is the cornea), black area B/W 2 lines is Ant. chamber.

Cortical



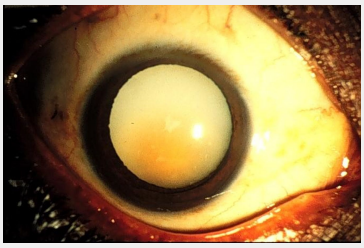


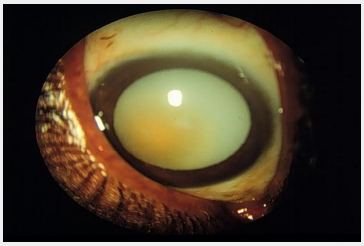
Cortical Opacity.

Slit lamp

If the opacity is located in the cortex, it is called a cortical cataract; Peripheral wedge like opacities or radial spoke like. Glare is commonly associated.

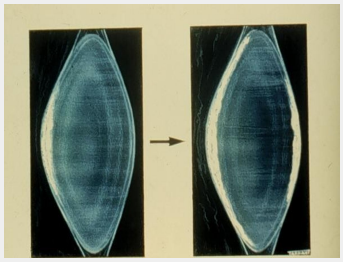
Morgagnian





Normally cortex is solid and hold the nucleus in place but when it liquefies (increased concentration of protein molecules under the lens capsule, water is drawn from the aqueous into the lens capsule via osmosis) it loses its support of the nucleus and allow free movement of it within the capsule bag.

Subcapsular



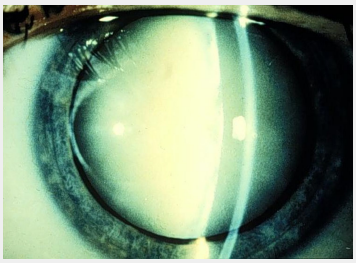
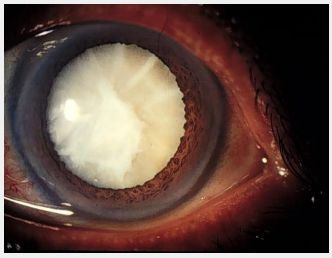
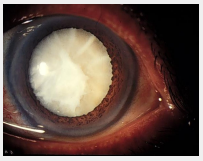
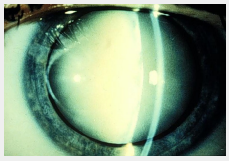
Anterior side is The less convex side.

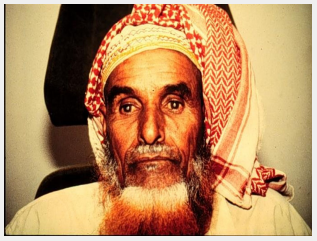
Opacity in the posterior side > posterior subcapsular cataract, it even extends anteriorly if left as seen in the right picture.

Anterior: Fibrous metaplasia in the central zone.

Posterior: granular or plaque like; migration of epithelial cells (DM, steroid, ocular inflammation).

★ Left pic: right side is Anterior and left side is Posterior. Anterior is always flatter than posterior which is more convex.

Types Based on Maturity:			
Immature	**Mature**	**Hyper-Mature**	**Intumescent**
(part of lens involved) (you still see the fundus)	(entire lens involved) (you can't see the fundus)  The whole lens is opaque > mature cataract. The anterior chamber is shallow(narrow) > Risk of cataract, so do a prophylaxis which is iridotomy. In this type the lens is swollen and thus closing the pupil, called Intumescent cataract.	(pushes the iris against the cornea -> closed angle glaucoma)  Happens when you leave the mature cataract for long time, the lens may become dehydrated and the capsule become wrinkled and fibrosed, calcification might be associated. Can lead to phacolytic glaucoma due to the leakage of proteins which block the mesh network causing open angle glaucoma	(congested)   In a stage between mature and immature. Rapid swelling of the lens causes shallow anterior chamber and block the aqueous from circulating causing (phacomorphic glaucoma) which is treated by Peripheral Iridotomy (hole or canal in the periphery of the iris).

Based on onset (age):			
Congenital	**Infantile**	**Pre-senile**	**Senile**
(present at birth) Causes: a. Galactosemia (metabolic disorder affect galactose metabolism) b. Hypoglycemia c. Myotonic Dystrophy d. Congenital ichthyosis (genetic skin disorder) e. Rubella Cataract  Leukocoria(white eye) “right eye” => it is a sign of diseases => the cause here is congenital cataract. With microphthalmia. This case is due to rubella.	(develop during 1st year of life) We are concerned about it because if left untreated causes deprivation Amblyopia (blocked light from reaching retina so no image formed leading to defect visual cortex maturation)	Early onset cataract	Nuclear sclerosis: greenish yellow central opacity Associated with anterior chamber flare (high proteins in the aqueous causes scattering the light from slit lamp). Associated with myopia and poor night vision.  Bilateral cataract “Senile cataract”, due to age only “Leukochoria”.

Signs and symptoms:

Symptoms:

- Painless loss of vision (gradual onset)
- VA: worsening of existing myopia, correction of hyperopia
- Loss of contrast sensitivity in low light (common in females, they can’t differentiate colors)
- Glare in bright light (scatter of light); difficulty seeing in the presence of bright light In some instances, a change in refraction (Myopia).

Signs:

- Visual acuity is reduced.
- Cataract appears black against the red reflex when
- The eye is examined with a direct ophthalmoscope

Diagnosis:

1. History: gradual visual loss and cloudy vision
2. P/E :
   - Visual Acuity
   - Flash light examination
   - Slit lamp examination (tells u type of cataract).
   - Direct ophthalmoscope
   - Refraction and Retinoscopy
   - Red and Green Light (macular function).
   - Ultrasound (B scan for posterior examination)(check the structure & shape from inside)

Funnel shaped total Retinal Detachment.

Treatment

Medical Treatment isn't effective!

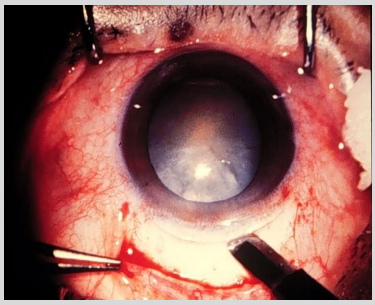
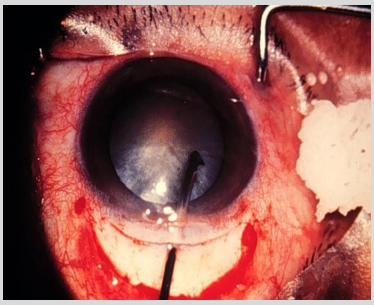
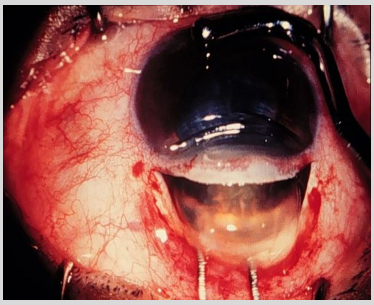
Surgical :

If the patient has No light perception don't do the surgery because there's another pathology beside the cataract(even complete thick cataract won't block light)

- Congenital: lens aspiration ± IOL (remove the optical lens -> replace it with new one and keep the capsule)
- Acquired: ECCE(very severe cataract) + PCIOL / Phaco + PCIOL
- Couching : old risky and require wearing glasses
- -ICCE (intracapsular cataract extraction) not used anymore.
- ECCE (Extracapsular cataract extraction):
- Phacoemulsification : Modified ECCE; most commonly used. Nowadays, small opening and putting a foldable lens.
- Phacofragmentation. same as above but from posterior segment.

This procedure isn’ extra “it was mentioned in the lecture”:

1 2 3

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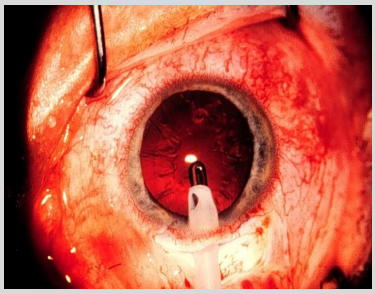
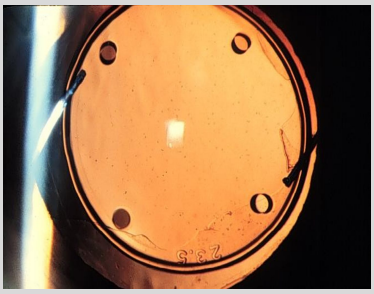
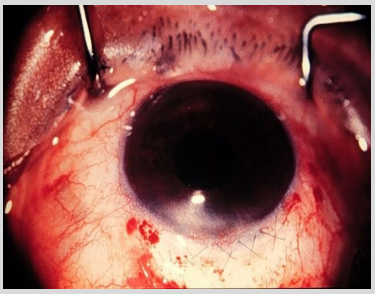
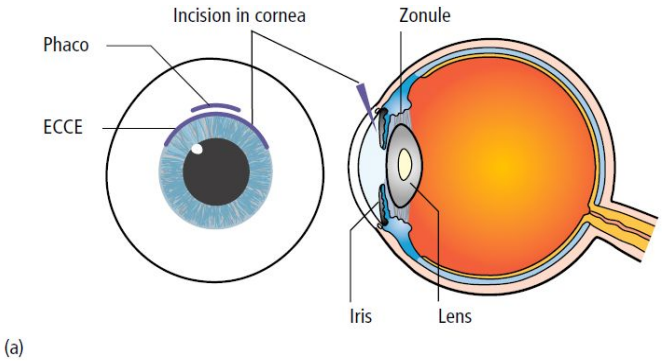
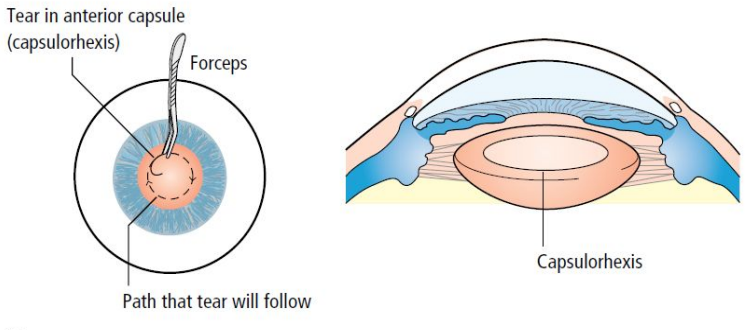




1) ECCE.
2) Opening the anterior capsule.
3) Taking out the lens. We keep the capsule to support the new lense.
4) Cortex aspiration.
5) Putting the artificial lens.
6) Closing the eye.

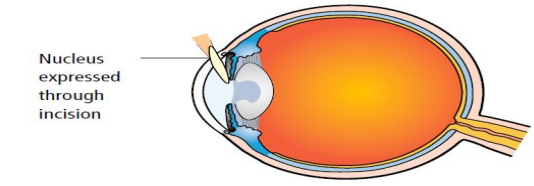
Extra for those who are interested :



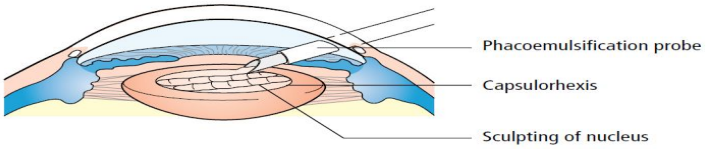
(a)



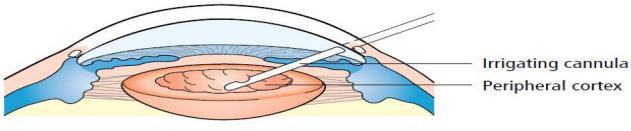
(b)



(c) **Extracapsular extraction**



(d) **Phacoemulsification**



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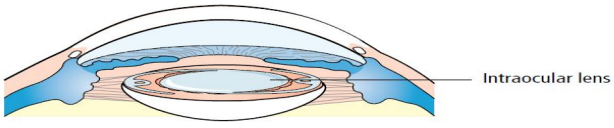


(f)

★ Stages in the removal of a cataract and the placement of an intraocular lens.

(a) An incision is made in the cornea or anterior sclera. A small, stepped self - sealing incision is made for phacoemulsification and a wider, limbal incision, for extracapsular surgery (ECCE).

(b) A circular disc of the anterior capsule is removed. In ECCE a ring of small incisions is made with a needle to perforate the capsule, allowing the central portion to be removed. In phacoemulsification the capsule is torn in a circle leaving a strong smooth edge to the remaining anterior capsule. A cannula is then placed under the anterior capsule and fluid injected to separate the lens nucleus from the cortex, allowing the nucleus to be rotated within the capsular bag.

(c) In ECCE the hard nucleus of the lens is removed through the incision, by expression . Pressure on the eye causes the nucleus to pass out through the incision.

(d) Alternatively the nucleus can be emulsified in situ . The phacoemulsification probe, introduced through the small corneal or scleral incision, shaves away the nucleus.

(e) The remaining soft lens matter is aspirated, leaving only the posterior capsule and the peripheral part of the anterior capsule.

(f) An intraocular lens is implanted into the remains of the capsule. To allow implantation through the small phacoemulsification wound, the lens must be folded in half or injected through a special introducer into the eye. The incision is repaired with fine nylon sutures. If phacoemulsification has been used the incision in the eye is smaller and a suture is usually not required.

Vitreous:

- Vitreous Hge: trauma, PDR, uveitis, PR
- Vitreous condensation, opacification
- Vitritis: uveitis
- Rx: underlying cause

Glaucoma: مية زرقاء, سويرق.

Definition:
Optic nerve damage presented by visual field defect.
Commonly caused by increased intraocular pressure, less common type is normal tension Glaucoma (a variant of open angle glaucoma, normal IOP, optic nerve damage with NO features of secondary glaucoma or other causes).

- ❖ Second leading cause of blindness!
- ❖ Early diagnosis is crucial to prevent loss of vision
- ❖ High IOP + Characteristic optic nerve head changes + visual field loss secondary to nerve fiber layer loss
- ❖ IOP is the single factor to be controlled

❖ **Aqueous Humor:**

- Active secretion:
1- Na/K ATPase. 2- Cl secretion. 3-Carbonic anhydrase.

- Passive secretion:

1- Ultrafiltration. 2- Diffusion.

❖ **Aetiology:**

- Primary: No detectable reason and often bilateral

- Secondary: Predisposing factor and often unilateral

❖ **Angle:** 1- Closed. 2- Open. 3- Combined mechanism.

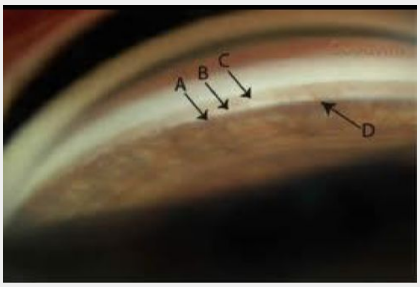
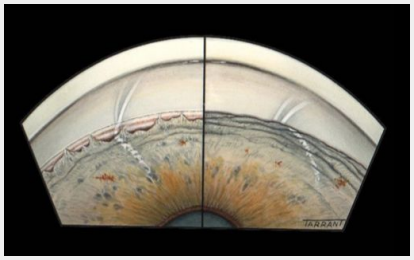
Glaucoma starts with peripheral (navigational) vision involvement.
Initially asymptomatic(usually pts will come in later stages when they lose their sight or incidentally by following up)
Usually detected on routine examination.

Risk factors:

- IOP
- Age
- Family history
- DM
- Myopia

Signs:

- High IOP (but it's not always the case, there is a normal tension glaucoma!)
- Gonioscopy: open or closed
- Optic nerve head damage
- Visual field loss

Types:	
Open Angle Glaucoma:	**Closed Angle Glaucoma:**
It occurs from blocked aqueous drainage caused by an unidentified dysfunction or microscopic clogging of the trabecular meshwork. This leads to chronically elevated eye pressure, and over many years, gradual vision loss." "The major risk factors for developing open-angle glaucoma include age, black race, family history, and elevated intraocular pressure , Myopia, DM, OCP" More serious because it's asymptomatic Iris not covering TM? Open angle  	1. Pupillary Block : After the age of 40 ;longed pupil dilatation like (watching TV in the darkness) Iris and lens get adherent and with dilatation the iris pushed against the meshwork and causes blockage! 2. Non-pupillary block: Younger age ,Far eastern ethnicity, plateau iris with thick peripheral iris roll, so not fully relieved by iridotomy. Occurs when the angle between the cornea and iris closes abruptly. With this closure, aqueous fluid can't access the drainage pathway entirely, causing ocular pressure to increase rapidly. This is an ophthalmological emergency and patients can lose all vision in their eye within hours". Symptoms and signs include loss of visual acuity, pain, conjunctival erythema, and corneal edema. Iris covering TM? Closed angle 

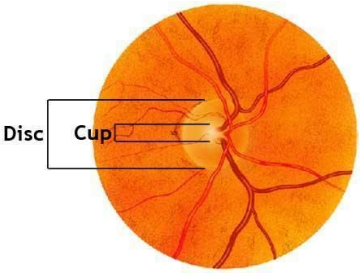
Investigations:

IOP
(intraocular pressure)
Using Tonometer
Normal Is 11 –21 mmHg

Visual Field exam
(assesses the pt's condition, if severe نسحب منه الرخصة)

- Confrontation test.
- perimetry

Exam **ONH** (optic nerve head) Fundoscopy: you'll

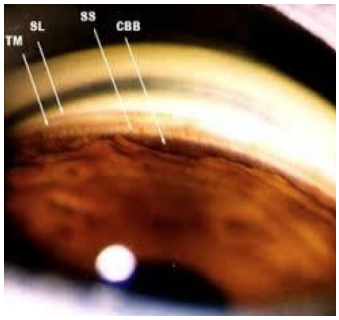


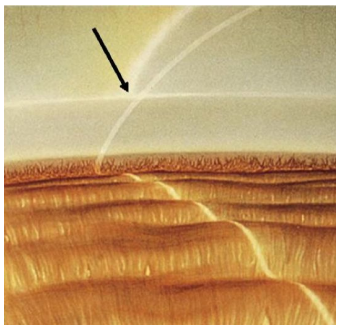
examine the optic nerve+blood vessels+surrounding structures

- Disc margin and disc diameter
- Neuroretinal rim
- Cup/disc ratio(normal value is 0.3, bigger cup=more nerve tissue loss! Causes for large cups: steroids and DM)
- Disc size
- PPA
- NFL defect
- Optic disc haemorrhage

Comment on: optic nerve color, margins(clear), vessels, the presence of cupping!

Gonioscopy
To measure the angle (focusing the light on the lens-> reflects on the iris, so we can see between the iris and cornea)

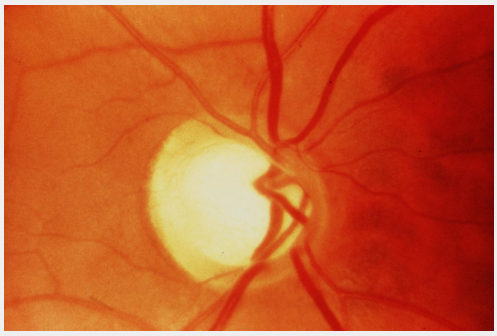


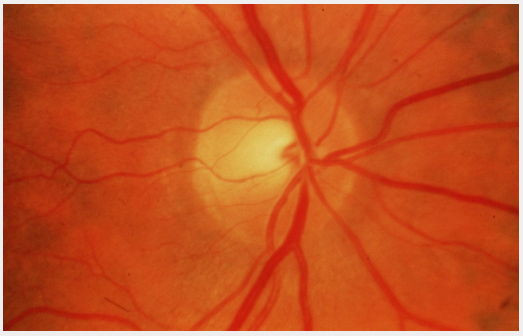


Changes in Glaucoma:

- Chronic open angle glaucoma on presentation, the pressure is typically in the 22–40 mmHg range. In angle closure glaucoma it rises above 60 mmHg.

- To confirm the diagnosis of glaucoma
  - Scotoma (blind spot)
  - Restriction of visual field .
- Edema: cupping
  - Physiological cupping: cup:disc ratio is less than 0.5, Central, healthy rim outside
  - Pathological cupping: Cup:disc ratio more than 0.5





Normal

RIGHT EYE
PALE
VESSELS shifted TO NASAL SIDE
CUPPING "The optic nerve fibers passes through the lamina cribrosa".
Engorgement of vessels.

Treatment & Prevention:

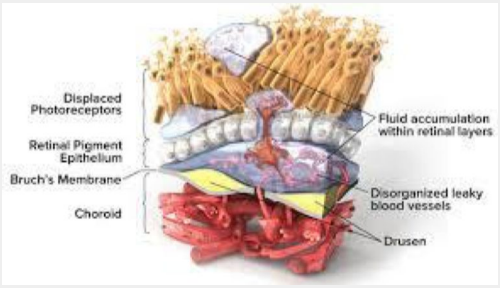
Start screening after the age of 40, every 2 to 4 years by Tonometry and cup to disc ratio.
Patient is referred for treatment when:

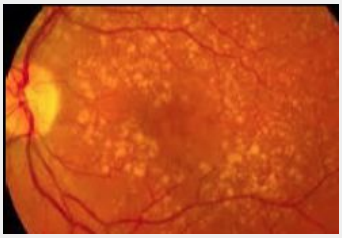
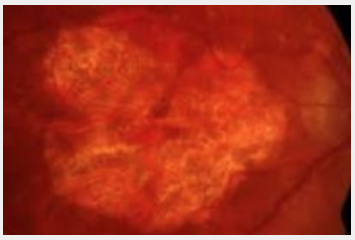
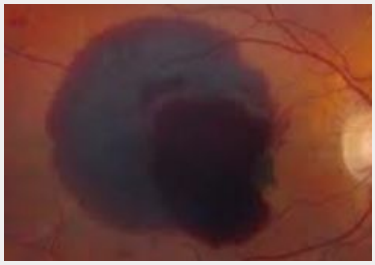
1. IOP more than 21mmgh.
2. C:D ratio more than 0.5
3. One cup significantly larger than the other one.

Treatment is aimed at reducing intraocular pressure by 3 modalities available (some pts don't have any symptoms and they're living their life happily, so don't interfere)

1. Antiglaucoma medications
2. Laser treatment; SLT(open angle -> laser burns trabecular meshwork -> induce changes), PI(laser opening behind the iris -> aqueous goes behind the iris -> pressure will decrease)
3. Surgical treatment "trabeculectomy".

Macular degeneration (age related macular changes):

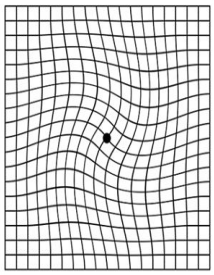
Definition	**Impaired central vision.** **Peripheral vision preserved.** **Leading cause of legal blindness in developed world.** **Changes in the macula affects outer retinal layer, retinal pigment epithelium, Bruch's membrane (b/w retina and choroid) and choriocapillaris (the innermost layer of choroid).**
pathogenesis	**Multifactorial: age, smoking, vascular disease, UV light, diet, and FHx.** Over time, undigested lipid products, such as the age pigment lipofuscin, accumulate in the RPE (Retinal pigment epithelium) and the excess material is transferred to Bruch's membrane, impairing its diffusional properties. Extracellular deposits form between the RPE and Bruch's membrane called Drüsen. Collections of these Drüsen in the macula give rise to the condition termed **Age - Related Maculopathy or ARM** where vision is normal. The neighboring RPE and photoreceptors may also show degenerative changes, producing the dry or non – exudative form of AMD. In the less common, exudative or ' wet ' form, new vessels from the choroid, stimulated by angiogenic factors such as vascular endothelial growth factor (VEGF), grow through Bruch's membrane and the RPE into the sub-retinal space, where they form a sub-retinal neovascular membrane . Types of ARM: 1. Dry "90%": without bleeding or exudates. 2. Wet "10%": with bleeding or exudates or both, major cause of blindness.
Signs and Symptoms	**Symptoms:** • Metamorphopsia: distorted vision • Micropsia: reduction of size of objects • Macropsia: enlargement of size of objects • Scotoma: VF loss • Blurred central vision. **Signs:** • foveal reflex is absent • Yellow, well circumscribed drüsen may be seen • Subretinal, preretinal, haemorrhages may be seen. "wet type"
	Macular involvement: •Outer retinal layer •Retinal pigment epithelium •Bruch's membrane (photoreceptors will secret lipids -> accumulates in bruch's membrane) •choriocapillaris  • Drusens(yellowish discoloration): lipid products from photoreceptor outer segments, found under retina • New vessels from choroid grow into the subretinal space forming subretinal neovascular membrane • Hemorrhage into subretinal space or even through the retina into the vitreous (significant loss of vision)

Types	Atrophic Exudative:	Exudative:
	•Often asymptomatic •Gradual over years Signs: •Drusen  •Geographic atrophy  •Photoreceptor degeneration •scotoma when light adapting	•Rapidly progressive (weeks) Signs: •Choroidal (subretinal) neovascularization •Preretinal hemorrhage  •Elevation of retina •Subretinal fibrosis  •Metamorphopsia • Central scotoma 

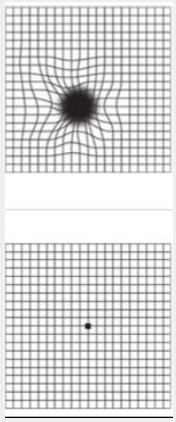
Examination

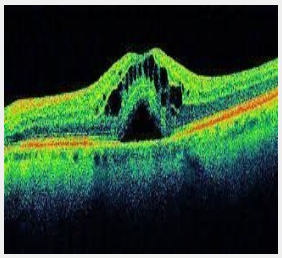
1. Visual acuity
2. Amsler grid testing for the macula. If the patient saw wavy lines, then the macula is abnormal.
3. Ophthalmoscopy
4. Others

• Fluorescein angiography, inject IV fluorescein to visualize the retinal vessels.
•ICG
•Indocyanine green dye
• OCT (Optical Coherence Tomography)

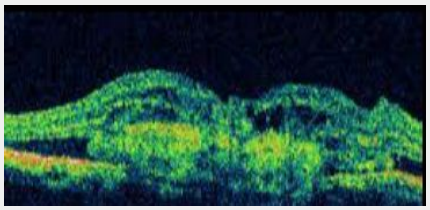


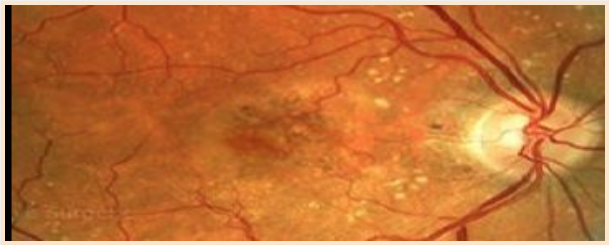
Amsler grid testing





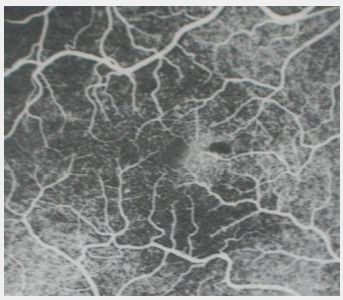
OCT > show layers, sign of drusen

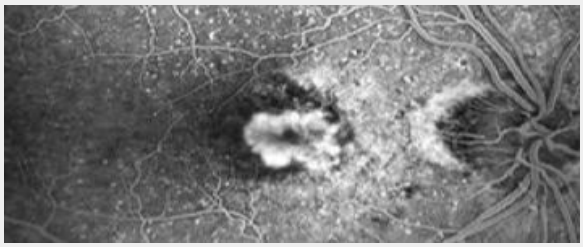




Ophthalmoscopy

Normal(left pic) and abnormal (right pic) Fluorescein Angiography



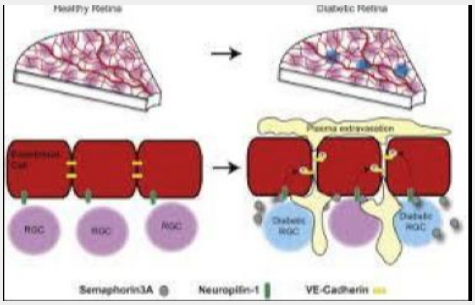
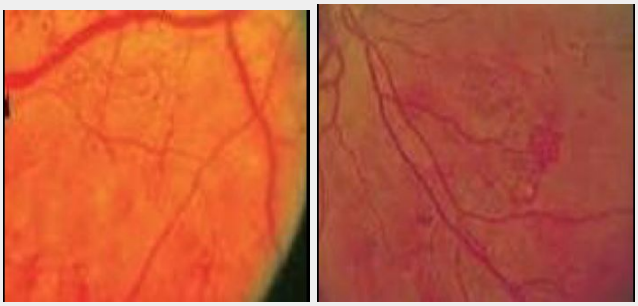
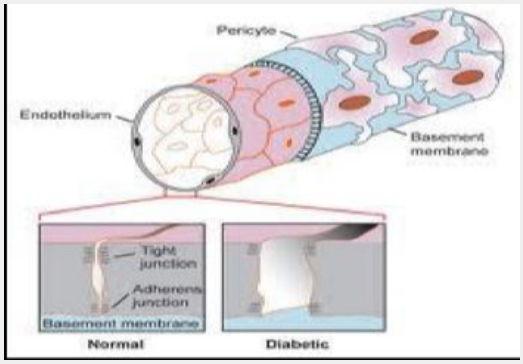
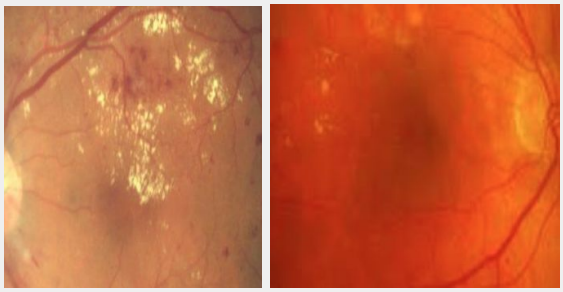


Treatment (Dry macular degeneration) irreversible condition

•Lifestyle
•Stop smoking, reduce UV exposure, Zinc & antioxidants
• Low-vision aid
• Monitoring with Amsler chart
•Observation
• Laser treatment of neovascular membrane especially for the wet type.
• Anti - VEGF agents. Wet type
•Verteporfin photodynamic therapy (PDT): injection of photosensitizer into systemic circulation followed immediately by laser targeting new vessels in macular area

Diabetic Retinopathy: Will be discussed in details in systemic diseases lecture

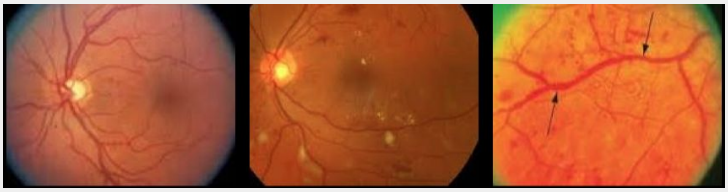
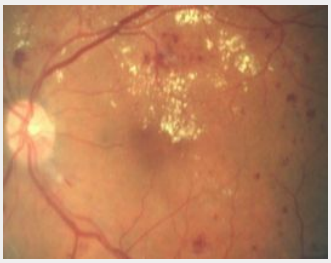
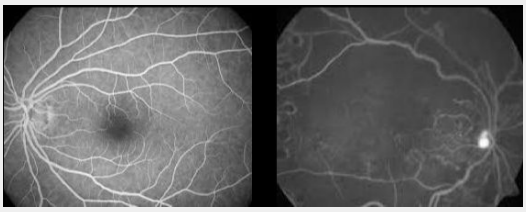
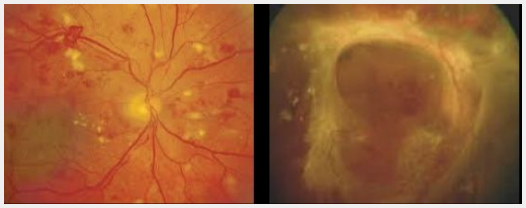
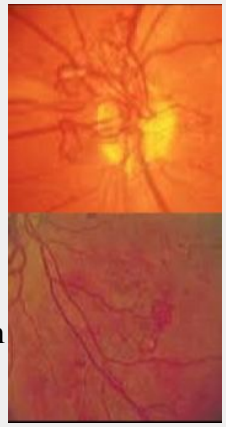
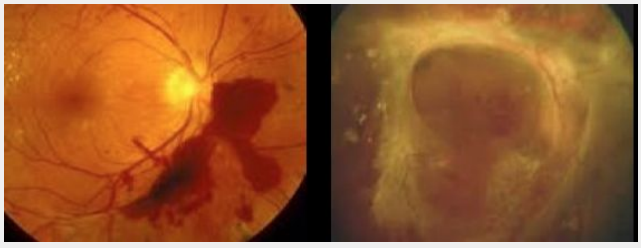
- ❖ Diabetes is the leading cause of blindness in KSA
- ❖ Microangiopathy which involves precapillary arterioles, capillaries and postcapillary venule
- ❖ Diabetes is associated with the following ocular events:
- ❖ Microvascular occlusion
- ❖ Microvascular leakage
- ❖ Retinopathy
- ❖ Cataract
- ❖ Glaucoma (e.g. rubeotic glaucoma, but an association with chronic open
- ❖ angle glaucoma is disputed).
- ❖ Extraocular muscle palsy due to microvascular disease of the third, fourth or sixth cranial nerves.

Microvascular Occlusion:	Microvascular Leakage:
-Thick capillary basement membrane -Capillary endothelial cell damage -Changes in red blood cells  -All this will lead to retinal ischemia -> AV shunts and NVs (this is bad! New fragile blood vessels that are not useful) 	Loss of pericytes between endothelial cells-> leakage into retina -> exudates and edema  

Risk Factors:

- ❖ Duration
- ❖ Poor metabolic control
- ❖ Pregnancy
- ❖ HTN
- ❖ Nephropathy
- ❖ Smoking
- ❖ Obesity and Hyperlipidemia

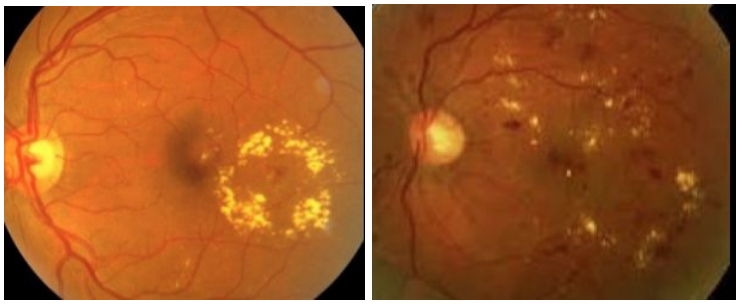
Clinically classified into two type:

Non-proliferative diabetic retinopathy (NPDR): (بيجي بالاختبار ومشنقة لو ماحليتوه!)	Proliferative diabetic retinopathy (PDR):
A.Mild B. Moderate C. Sever  •Asymptomatic •Decreased visual acuity:A. CSME B. macular ischemia  	A.Early B. Advance  •Symptomatic •Can also cause macular ischemia and/or edema •Neovascularization •NVD: neovascularization of the disc •NVE: neovascularization elsewhere  • Fragile (intra-retinal or vitreous hemorrhage) • Associated with fibrous proliferation TRD 

❖ 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



❖ Rx:

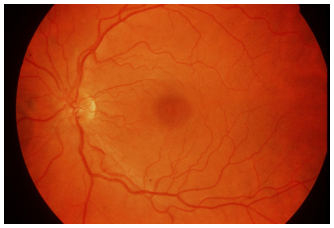
• Laser

• intravitreal steroid injection

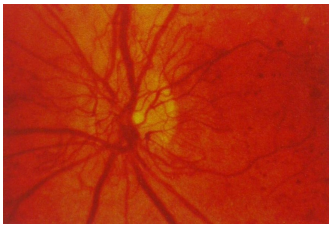
• intravitreal anti-VEGF injection

• Pars plana vitrectomy

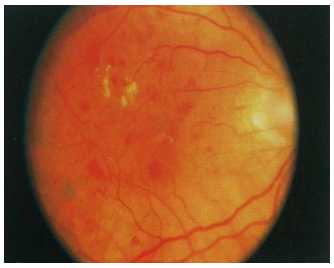
Categories of Diabetic retinopathy			
Background Diabetic Retinopathy Micro aneurysm Dot and blot hemorrhage Exudate -Earliest signs but persist-	**Diabetic Maculopathy** Macular Edema Ischemia	**Proliferative Diabetic Retinopathy** ● Cotton wool spot :(accumulation of debri within the nerve fiber layers) ● Venous changes :(increased tortuosity, looping, beading, sausage like segmentation) ● Arterial changes:(narrowing or silver wiring, Obliteration) ● Intraretinal microvascular Anomalies) ● Deep Retinal Hemorrhage.	Advanced diabetic disease ● Traction Retinal Detachment (there are three types of retinal detachment : Rhegmatogenous retinal detachment + Exudative (serous) retinal detachment +Tractional retinal detachment) ● Vitreous Hemorrhage ● Neovascular Glaucoma



Normal



Extensive neovascularization



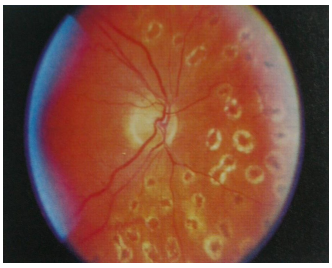
RIGHT

Early stage

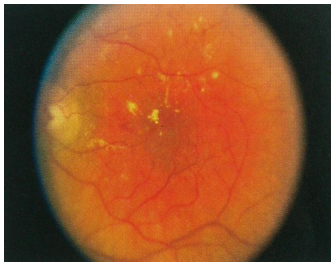
Hemorrhagic spots

hard exudates yellowish

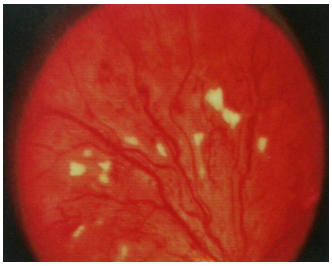
microaneurysms



Laser Treatment

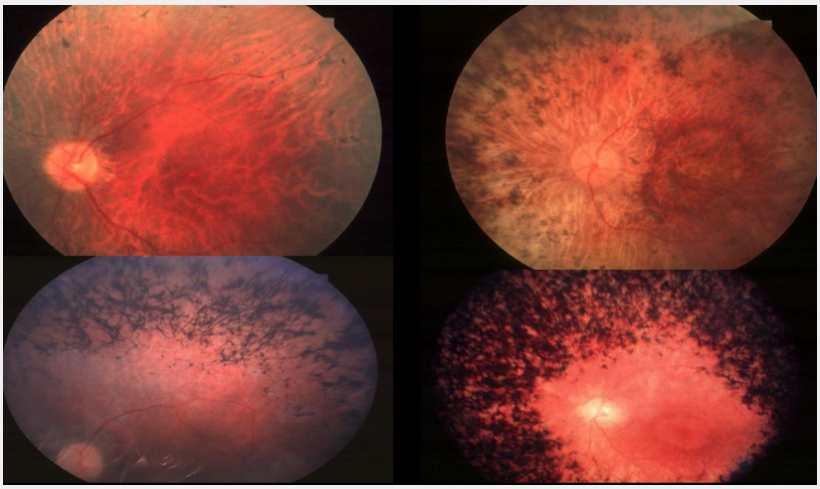


Exudates



COTTON WOOL SPOTS

Retinitis Pigmentosa:

	•Group of genetic disorders affect the retina ability to respond to light •Slow loss of vision: nyctalopia, loss of peripheral vision, blindness •Most are legally blind by 40s •Central visual field of less than 20 degrees •XR: males: more often and more severe •females: carry the genes and experience vision loss less frequently •Target photoreceptors • Associated with pigmentary changes in the RPE, which may be primary or secondary to the photoreceptor loss
Signs & symptoms	**Symptoms:** • Nyctalopia (loss of night vision) • Tunnel vision (loss of peripheral vision) **Signs:** •VA:20/20-NLP •+-APD •PSCC •RPE hyperpigmentation (bone spicules) alternate with atrophic regions •Attenuation of the arterioles •Waxy pallor of the optic nerve head •CME (severe cases of RP) 
Investigations	•VF test • Color testing (mild blue-yellow axis color defects) • Dark adaptation study (reduced contrast sensitivity relative to VA) • Genetic subtyping •OCT (CME)• FFA • ERG • EOG

Systemic Associations	**•Hearing loss and RP:** Usher syndrome Alport syndrome Refsum disease **• Kearns-Sayre syndrome:** External ophthalmoplegia Lid ptosis Heart block Pigmentary retinopathy **• Abetalipoproteinemia** **• Mucopolysaccharidoses** **• Bardet-Biedl syndrome** **• Neuronal ceroid lipofuscinosis**
Treatment	• CAI: CME • Vitamins?? • Cataract: surgery• Low vision aids • Gene therapy!!