Chronic Visual Loss

Objectives:

- Not given.

[Color index : Important | Notes | Extra]

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

Done by : Abdulrahman Al-Shammari.

Revised by : Adel Al Shihri.

Chronic visual loss:

- **Definition**: Slowly progressive painless visual loss. (chronic means within months to years).
- Major causes: (starting from the most common)
  1. Cataract.
  2. Diabetic retinopathy.
  3. Glaucoma.
  4. Macular degeneration.

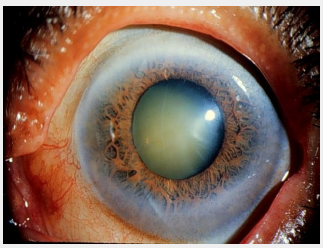
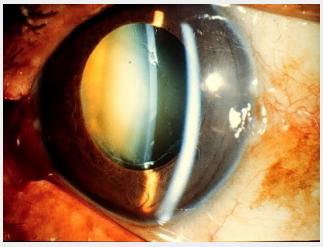
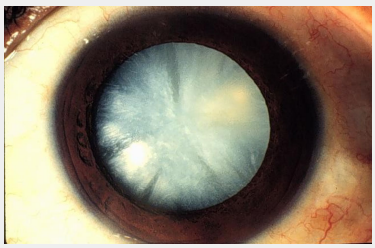
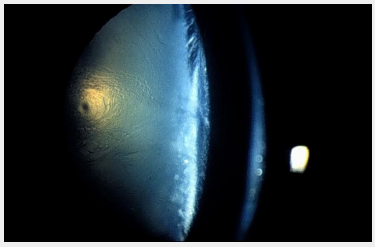
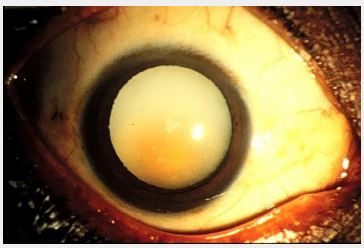
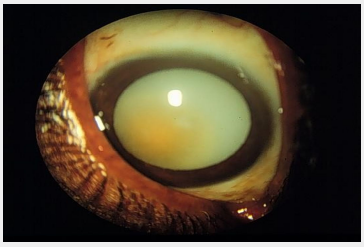
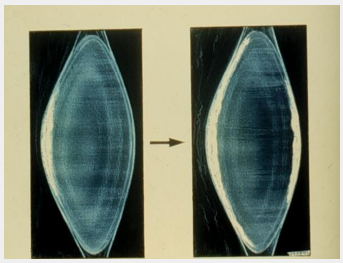
- One should recognize the normal first to be able to identify the abnormal :
  - Normal macula.
  - Lens clarity (normally it has a shade if you don't know this normal appearance you may think it is cataract).
  - Optic nerve head.
  - Normal retina.

Cataract:

- 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.

Types Based on Morphology

Nuclear	Cortical	Morgagnian	Subcapsular
 	 	 	
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 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.	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.	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.

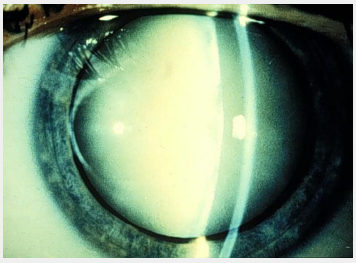
Types Based on Maturity:

Immature

(part of lens involved)

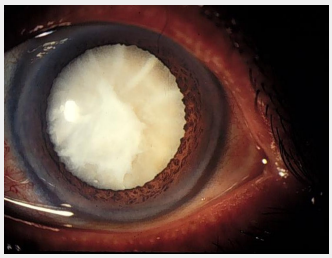
Mature

(entire lens involved)



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.

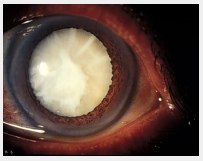
Hyper-Mature

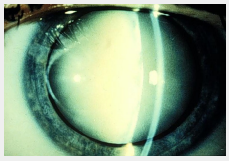


Happens when you leave the mature cataract for long time, the lens may become dehydrated and the capsule become wrinkled and fibrosed, calcification might be associated.
Can lead to phacolytic glaucoma due to the leakage of proteins which block the mesh network causing open angle glaucoma

Intumescent

(congested)



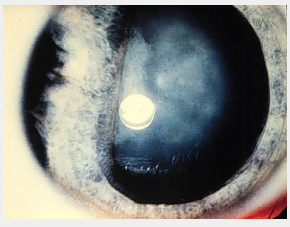


In a stage between mature and immature.

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 cause (secondary cataract):

Traumatic



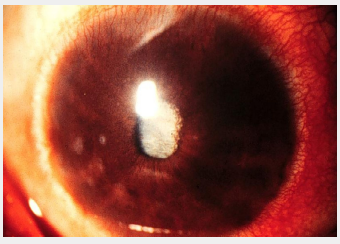
Blunt or penetrative and takes time to appear!

Iris dilation “It is detached from its position”

Neoplasm

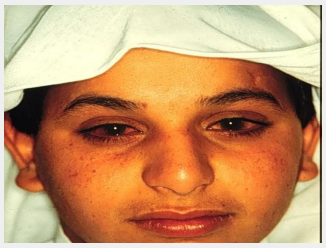
Melanoma or Retinoblastoma

Inflammatory



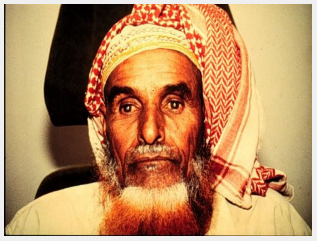
The pupil has not dilated => synechia formation “most common cause of it is inflammation”. => caused by uveitis.

Toxic



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).

Based on onset:			
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:	**Signs:**
Painless loss of vision; Glare; difficulty seeing in the presence of bright light In some instances, a change in refraction (Myopia).	• 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 !

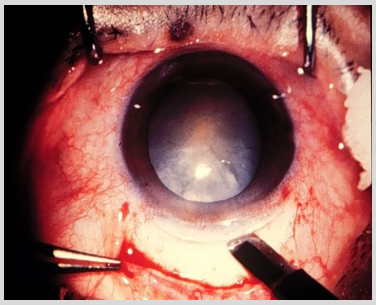
Surgical :

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)

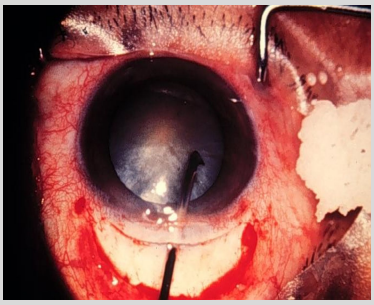
- Couching : old risky and require wearing glasses
- -ICCE (intracapsular cataract extraction) not used anymore.
- ECCE (Extracapsular cataract extraction):
- Phacoemulsification : Modified ECCE; most commonly used. Nowadays, small opening and putting a foldable lens.
- Phacofragmentation. same as above but from posterior segment.

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

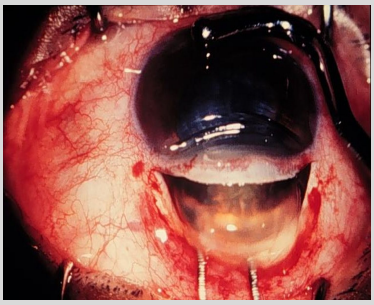
1



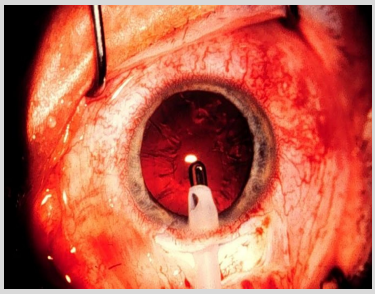
2



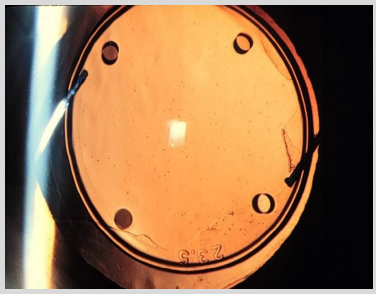
3



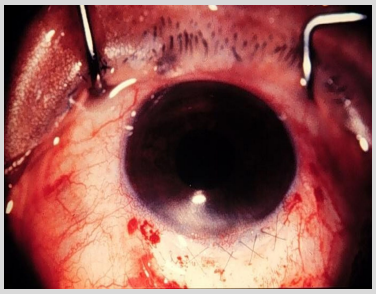
4



5

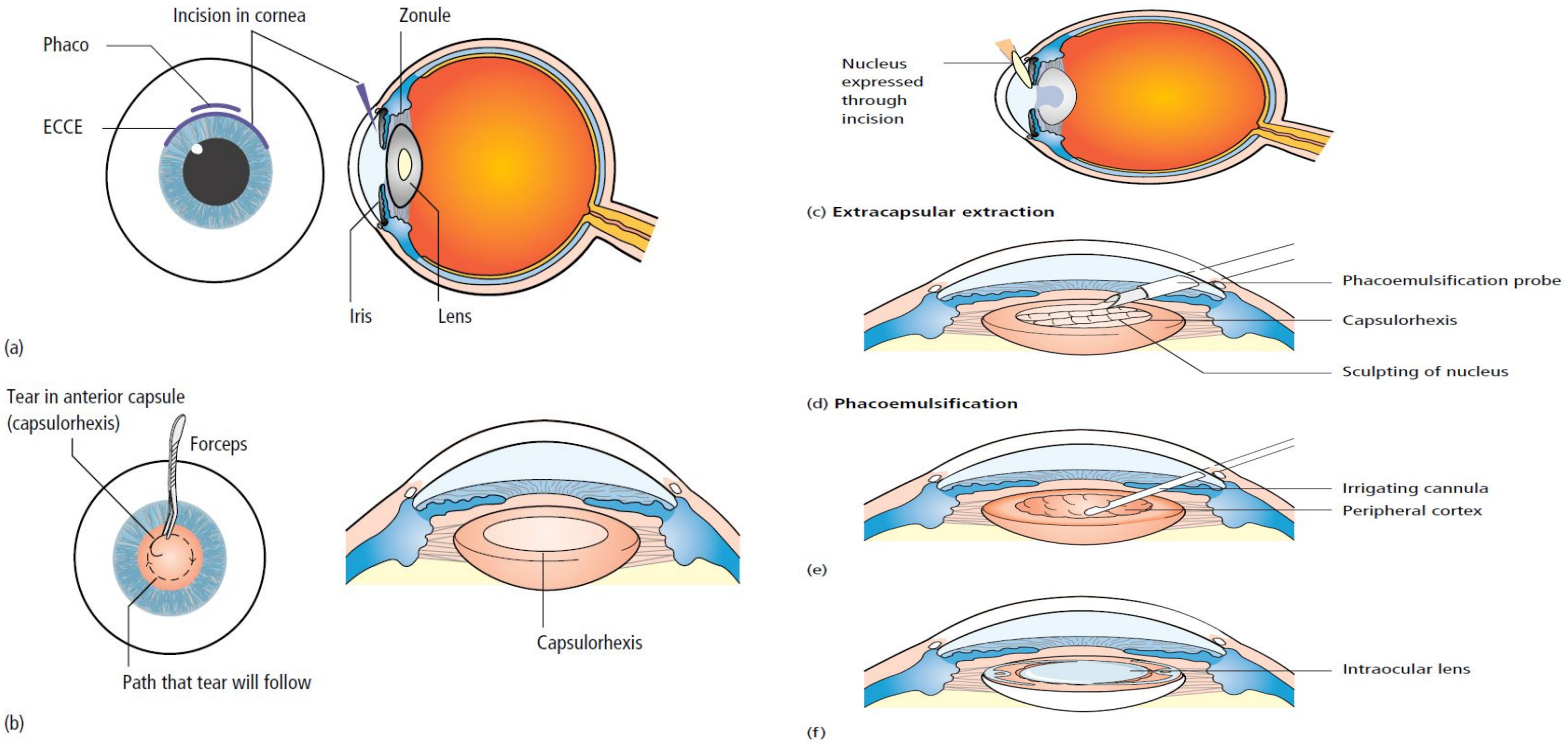


6



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 :



★ 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.

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).

Types:

Open 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

Closed Angle Glaucoma:

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.

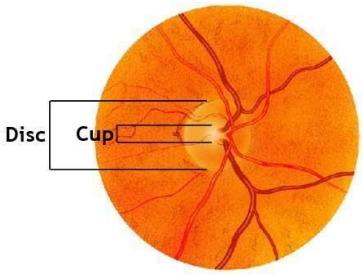
Investigations:

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

Visual Field exam

- Confrontation test.
- perimetry

Exam **ONH** (optic nerve head) Fundoscopy:



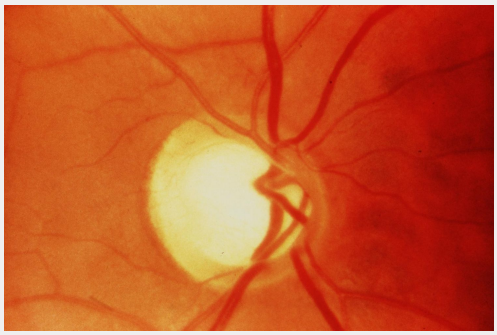
Comment on: optic nerve color, margins(clear), vessels, the presence of cupping!

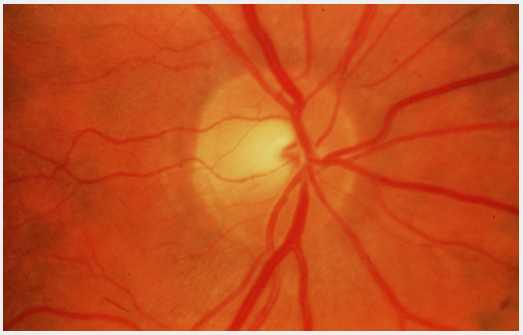
Gonioscopy
To measure the angle

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.

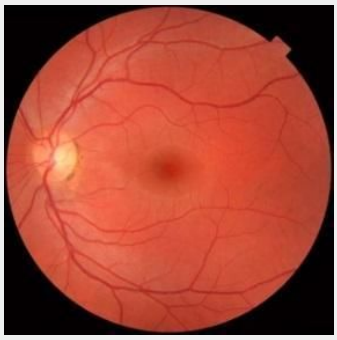
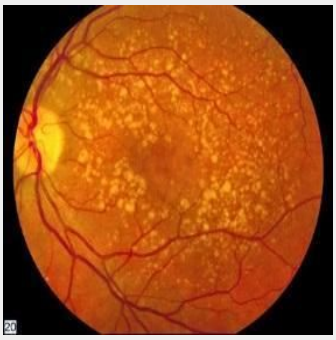
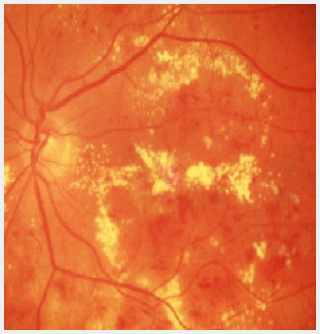
Patient is referred for treatment when:

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.

1. Medical treatment; carbonic anhydrase inhibitors
2. Laser treatment;
3. Surgical treatment “trabeculectomy".

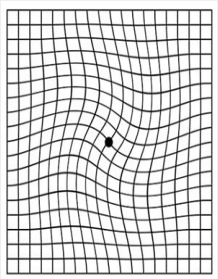
Age Related Macular Changes:

Definition	**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	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:** • Blurred central vision. • Distorted vision (metamorphopsia) • Reduction (micropsia) or enlargement (macropsia) of object size • Loss of the central visual field (scotomata) **Signs:** • foveal reflex is absent • Yellow, well circumscribed drüsen may be seen • Sub-retinal, pre-retinal, haemorrhages may be seen. "wet type"
	Normal Macula : Darker than surroundings. Avascular Contains only cones Drusen deposits (depositions of yellow spots "lipids like material" underneath the retina) : yellow spots    Macular Exudate

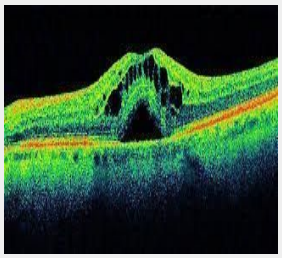
Examination

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

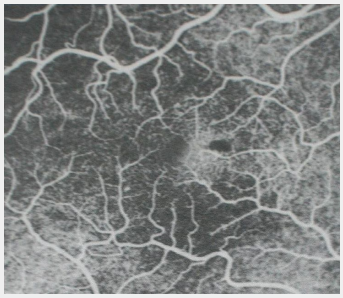
- Fluorescein angiography, inject IV fluorescein to visualize the retinal vessels.
- Indocyanine green dye
- OCT (Optical Coherence Tomography)



Amsler grid testing



OCT > show layers, sign of drusen



Normal Fluorescein Angiography

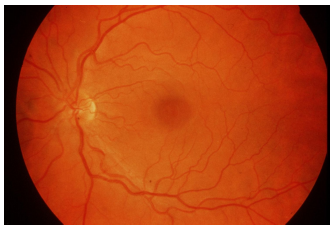
Treatment
irreversible condition

- Low-vision aid
- Laser treatment of neovascular membrane especially for the wet type.
- Anti - VEGF agents. Wet type

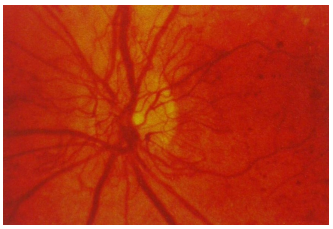
Diabetic Retinopathy: Will be discussed in details in systemic diseases lecture

- Diabetes is the leading cause of blindness in KSA
- Diabetes is associated with the following ocular events:
- 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.

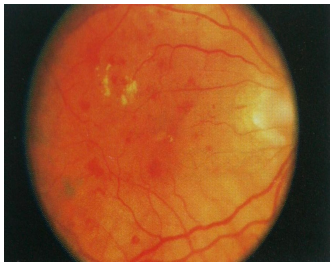
Categories of Diabetic retinopathy			
Background Diabetic Retinopathy Micro aneurysm Dot and blot hemorrhage Exudate -Earliest signs but persist-	**Diabetic Maculopathy** Macular Edema Ischemia	**Proliferative Diabetic Retinopathy** • Cotton wool spot :(accumulation of debri within the nerve fiber layers)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



Normal



Extensive neovascularization



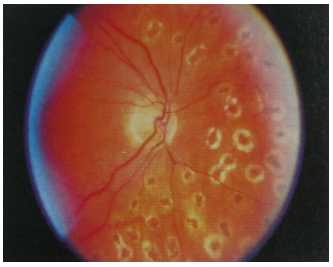
RIGHT

Early stage

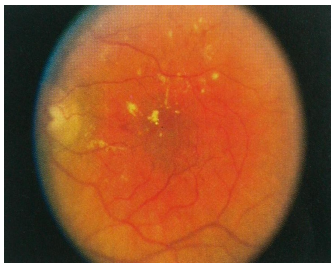
Hemorrhagic spots

hard exudates yellowish

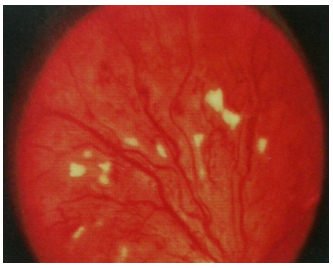
microaneurysms



Laser Treatment



Exudates



COTTON WOOL SPOTS