

# ophthalmology Team

## 7- Chronic Visual Loss

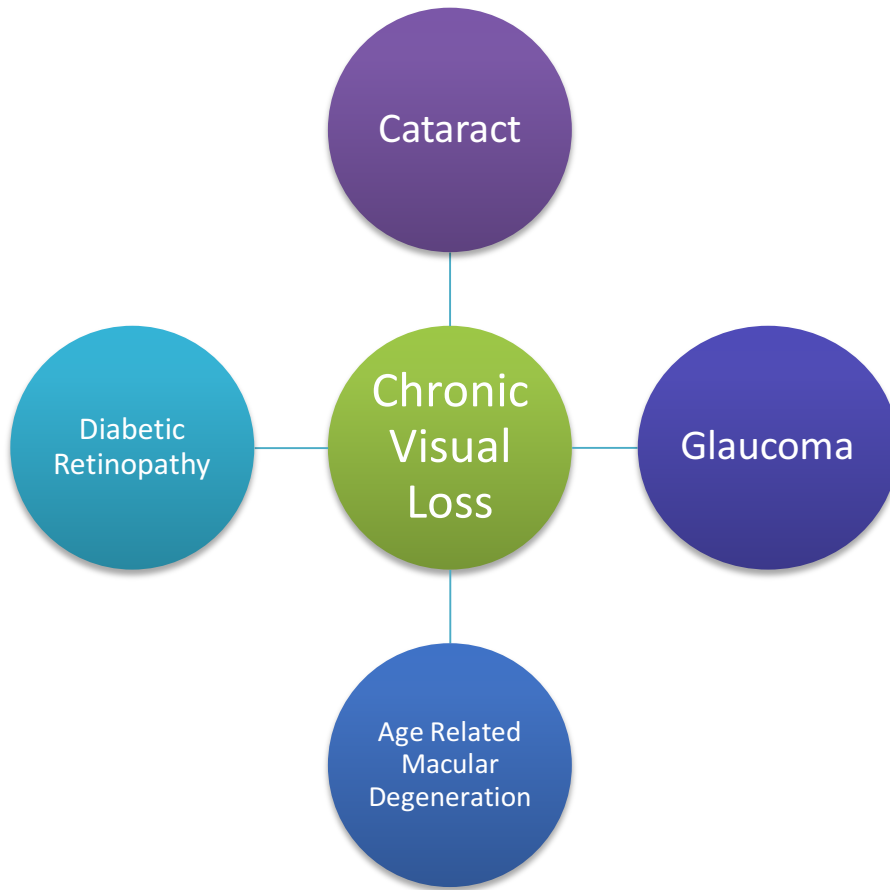
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**Doctor's note**    **Team's note**    Not important

**Important**    **431 teamwork in a yellow box**



## CHRONIC VISUAL LOSS

**Definition:** Slowly progressive visual loss (**chronic means within months to years**).

**Major Causes: (listed starting with the commonest):**

- 1- Cataract
- 2- Diabetic retinopathy (**the prevalence of diabetes in our country is 1 in 4**)
- 3- Glaucoma
- 4- Macular degeneration

**One should recognize the normal first to be able to identify the abnormal e.g.:**

- 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

## 1. Cataract

### Definition:

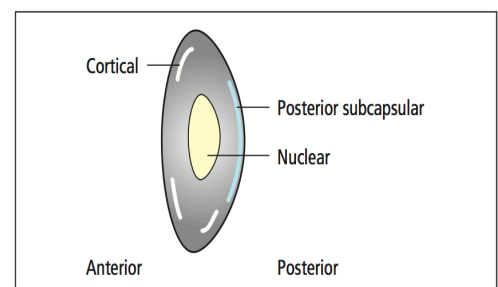
Cataract is the name given to any light-scattering opacity within the lens wherever it is located, when it lies on the visual axis or is extensive; it gives rise to visual loss.

**\*Cataract is the commonest cause of treatable blindness in the world.**

### Classification

**Based on morphology:**

1. Nuclear
2. Subcapsular
3. Cortical.



**Figure 8.1** The location of different types of cataract.

From 11<sup>th</sup> edition of Ophthalmology Lecture Notes Book:

- Age-related cataract is commonly nuclear, cortical or sub- capsular in location
- Steroid-induced cataract is commonly posterior subcapsular.

## Based on maturity:

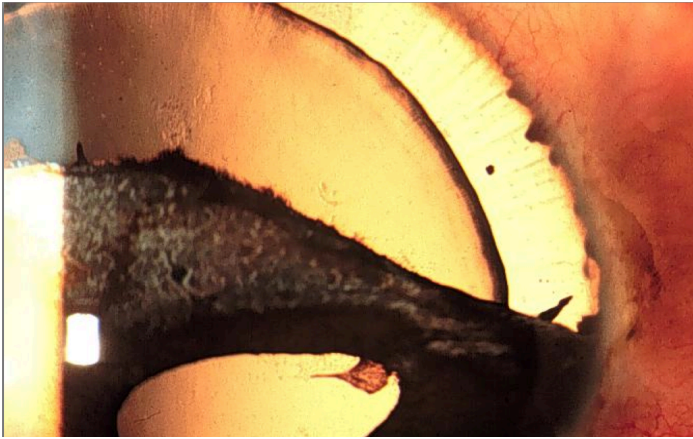
1. Immature (**part of lens involved**)
2. Mature (**entire lens involved**)
3. Tumescient (**congested**)
4. Hyper-mature (**happens when you leave the mature cataract for long time the lens may become dehydrated and the capsule become wrinkled and fibrosed**).

## Based on age of onset:

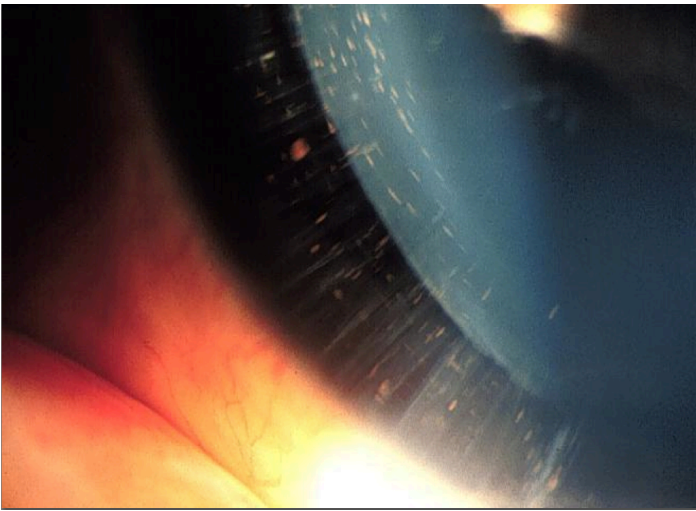
1. Congenital:
  - a. Galactosemia
  - b. Hypoglycemia
  - c. Myotonic Dystrophy
  - d. Congenital ichthyosis
  - e. Rubella Cataract
2. In infants (**cataract causes amblyopia (a failure of visual maturation) by depriving the retina of a formed image at a critical stage of visual development**).
3. Pre-senile
4. Senile

## Secondary cataract:

1. Traumatic
2. Inflammatory
3. Neoplasm
4. Toxic



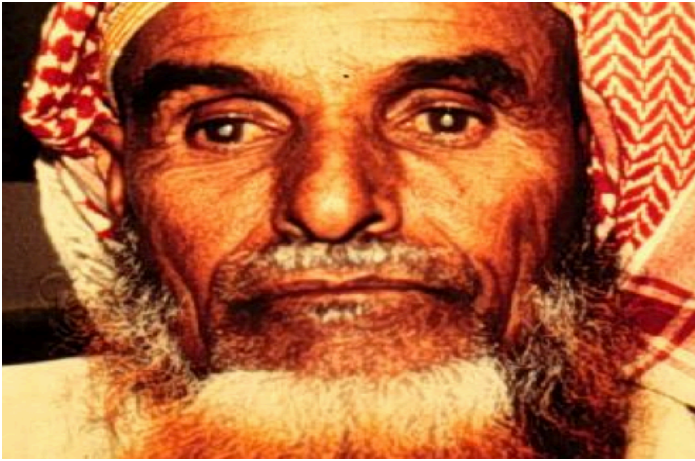
**This is a case of blunt trauma showing dialysis of the iris (the iris detached from it's origin)**



**The same patient in this picture showing the very fine zonules**



**The right eye is abnormal:  
- Congenital cataract (caused by Rubella) and microphthalmia "small eyes".**



**Both eyes are abnormal:**

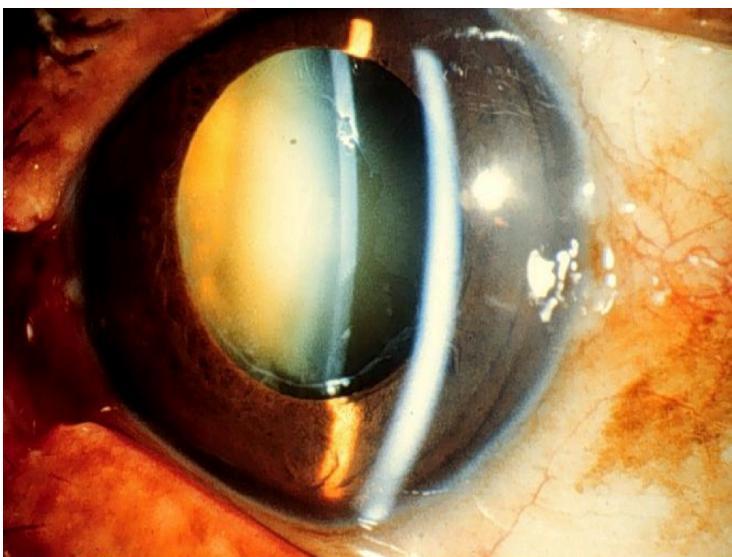
-Showing leukocoria, which is a sign of disease, could be caused by congenital cataract, retinal detachment, or organized vitreous hemorrhage.

- (in this case it is senile cataract)



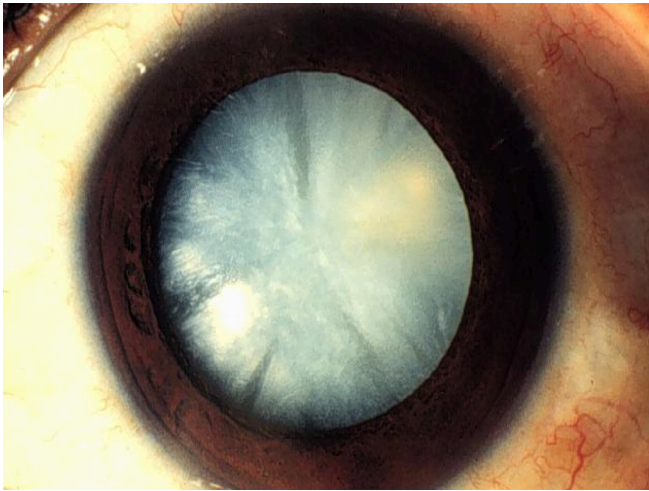
**This is nuclear cataract**

-Usually with aging the nucleus become hard, that's why it is the **most common type of senile cataract.**



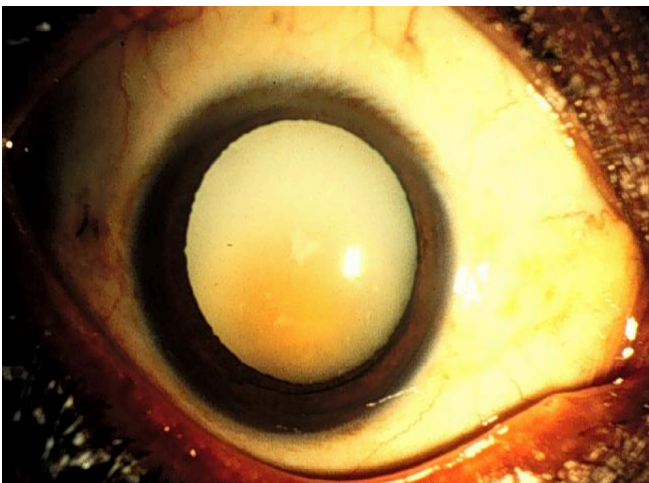
- This is nuclear sclerosis (the nucleus is opaque) and the cortex on top is clear.

-The advantage of using Slit lamp here that it gives section of the tissue so you can identify the level of the pathology.

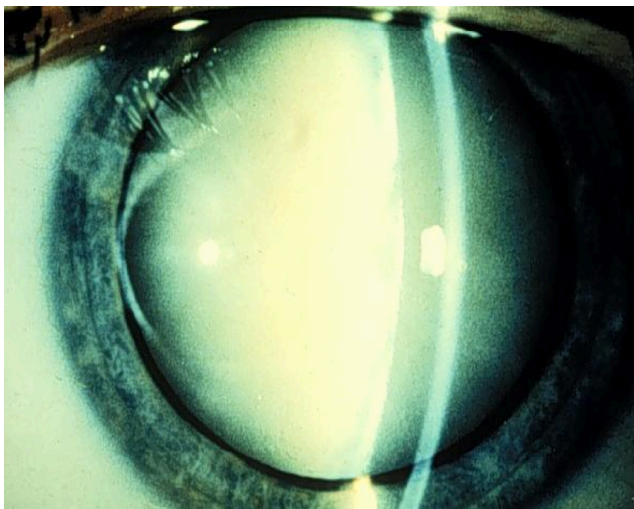


This is cortical cataract:

- Whenever you see glaucoma secondary to cataract **most probably it is cortical cataract** because the lens will be swollen > which increases the pressure > results in glaucoma.
- It is a type of senile cataract.



- Nucleus is displaced down because of liquefied cortex. This is a type of senile cataract (the nucleus moves with the gravity.)
- It is called **Morgagnian cataract**.
- (normally the solid cortex holds the nucleus in position).



-Narrow anterior chamber means swollen lens (happens in glaucoma).

- It is called **intumescent cataract**.

- If we see patient with intumescent cataract and he has normal intraocular pressure he might develop glaucoma.

The management is: iridotomy “create a hole in the iris by laser”.

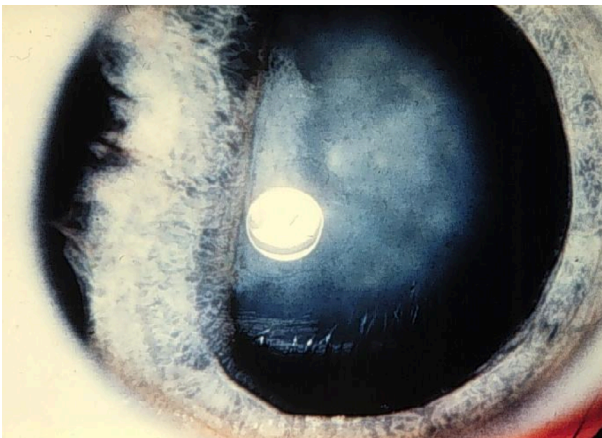
- Note:

**Iridectomy:** is the surgical removal of part of the iris.



**-Hyper mature cataract showing:**

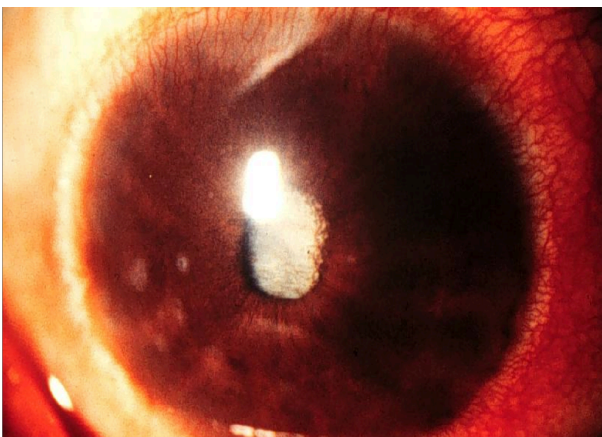
**Wrinkled lens, the anterior capsule is fibrosed, arcus senilis (white-gray ring surrounding the iris.)**



**- Trauma resulted in iridodialysis and traumatic cataract.**

**- Blunt trauma > cause chronic cataract  
cataract takes time to develop.**

**- Penetrating trauma > cause acute  
cataract.**



**-This is cataract secondary to inflammation.**

**- Iridoscleritis: inflammation of the iris and ciliary body of the eye.**

**-the Eye is congested.**

**- If you try to dilate the pupil it will not dilate properly because of posterior synechiae (adhesion between the iris and the lens).**

**Note:**

**Anterior synechiae (adhesion between the iris and cornea).**





- **Angry-looking eyes**

- **caused by allergy burner keratoconjunctivitis.**

- **Note:**

**Some patients with this condition when you give them steroids, the symptoms and itching will be relieved and they will continue buying it from the pharmacy, and this may cause posterior subcapsular cataract which may lead to glaucoma.**

## **Diagnostic tests:**

- Visual acuity
- Flashlight examination (**any patient with very dense cataract can see and react with light, if not then the optic nerve is not functioning**)
- Direct ophthalmoscope
- Slit lamp
- Refraction and retinoscopy
- Red and green light (**for the macula**)
- Ultrasonography

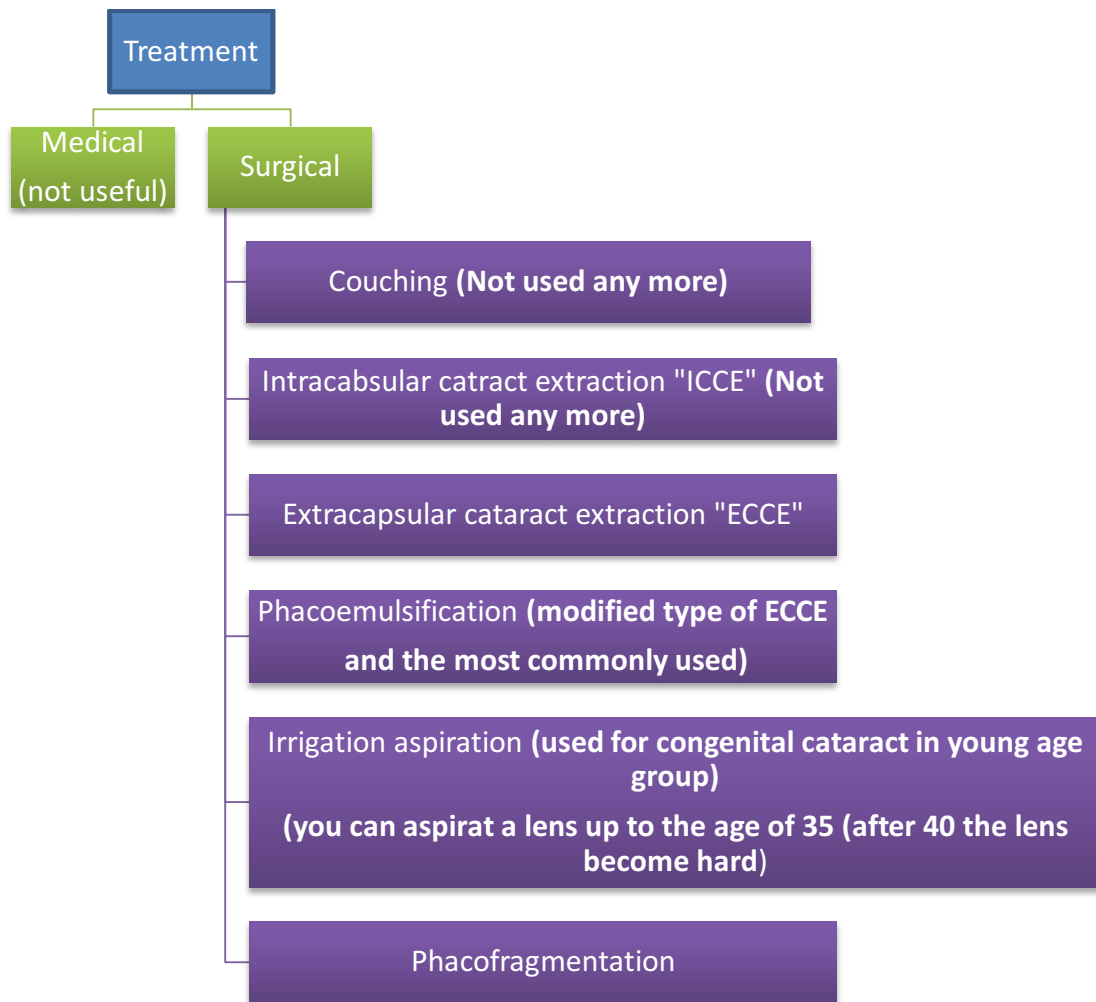


- **Eye ultrasound (B-scan): showing funnel shape detachment of the retina.**

**Note:**

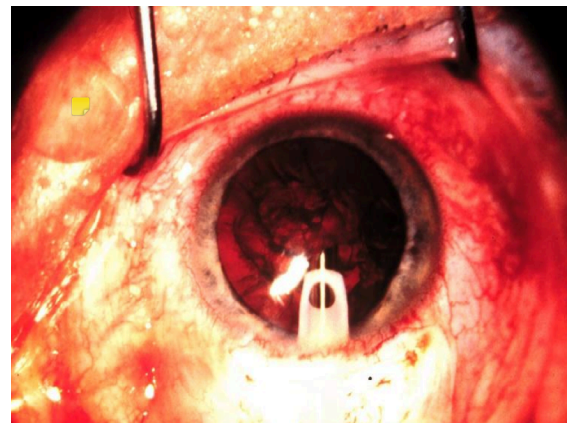
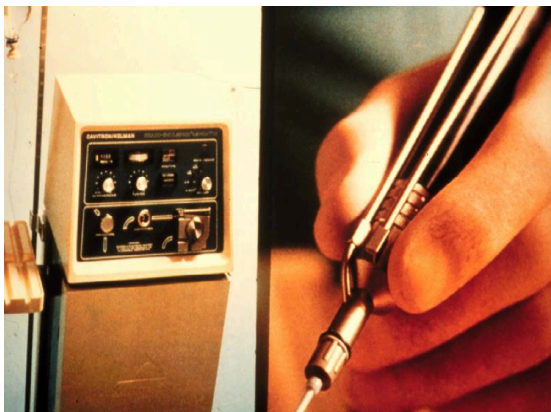
**Always do B-scan before surgery when you can't see the posterior segment to detect any pathology like retinal detachment.**

**-So, B-scan US provides data regarding the ocular structures. A-scan US gives measurements.**



## Phacoemulsification:

Through small opening then you break the nucleus inside and aspirate it with the cortex and the posterior capsule remain intact.



From 431 Team

ICCE: Intracapsular Cataract Extraction

ECCE: Extracapsular Cataract Extraction

ECCE IOL: Extracapsular Cataract Extraction with Implantation of the Intraocular Lens.

PHACO IOL: phacoemulsification with Intraocular Lens Implantation

## 2. Glaucoma

**Narrow angle glaucoma** is an acute condition characterized by severe pain, which makes the patients come to the ER immediately. It might cause total vision loss within hours if left untreated because of optic nerve ischemia. It is considered less serious than **open angle glaucoma**, which is a chronic painless disease may not be even recognized by the patient until it reaches advanced stage.

### Definition:

Glaucoma is a combination of factors that leads to optic nerve damage presented by visual field defect. **The most common risk factor is increased IOP**, which is the most common cause of glaucoma. Less common type of glaucoma is normal or low tension glaucoma, and both have the same optic nerve damage.

**Low or normal tension glaucoma:** optic nerve supping with normal IOP.

- a) If detected early and treated, blindness can be prevented.
- b) Most patients in early glaucoma are asymptomatic.

"High elevations of intraocular pressure (IOP), up to 40 mmHg in patients with open-angle glaucoma, generally cause no pain, redness, or visual symptoms. There is no loss of visual acuity as long as central vision is preserved. **Central visual field loss is a late manifestation of open-angle glaucoma**, usually preceded by ganglion cell loss and optic nerve damage. Some patients are unaware of field loss even when it has progressed to central "tunnel vision" of 10 to 20 degrees. **Visual field loss cannot be recovered once it has occurred.**"<sup>(1)</sup>

## Investigation:

1. IOP using tonometer
2. Visual Field.
3. Exam ONH “optic nerve hypoplasia” to detect abnormal cupping
4. Gonioscopy to visualize the angle

## Basic Information:

- 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."<sup>(6)</sup>

"The major risk factors for developing open-angle glaucoma include age, black race, family history, and elevated intraocular pressure."<sup>(1)</sup>

- Acute angle-closure glaucoma. “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”.<sup>(2)</sup>

Symptoms and signs including loss of visual acuity, pain, conjunctival erythema, and corneal edema.<sup>(1)</sup>

- Optic disc: Physiological cupping

Is a small central cupping with clear margin and the color of the disc is usually orange and the vessels are arising from the center. The cup-disc ratio is usually **0.3 or less**.

Pathological cupping: large and the disc is slightly ischemic and whitish, the vessels are shifted toward the side and the **cup-disc ratio here is 8**.

- So, the mechanism of glaucoma is reduced aqueous drainage, either by narrowing or obstruction of the outflow. Screening is important in the **families with history of glaucoma**.

## What happens when the IOP is raised?

The optic nerve enters the eye from the lamina cribrosa “weak area”. So with reduced aqueous drainage the pressure starts to build up inside the eye and start to compress the optic nerve.

### Examination:

#### When to start screening?

Every 2 to 4 years after the age of 40. Particular attention is needed with +ve family history.

#### How to screen?

By: 1. Tonometry, which measures the IOP.

2. Cup-to-disc ratio, refer the patient to the ophthalmologist if the ratio  $\geq 0.5$  even if the IOP was normal.

#### Referral criteria:

1. IOP over 21 mmHg.
2. C/D ratio 0.5 or greater.
3. One cup significantly larger in one eye.

## 3. Age Related Macular Degeneration “ARMD”

### The macular changes affect:

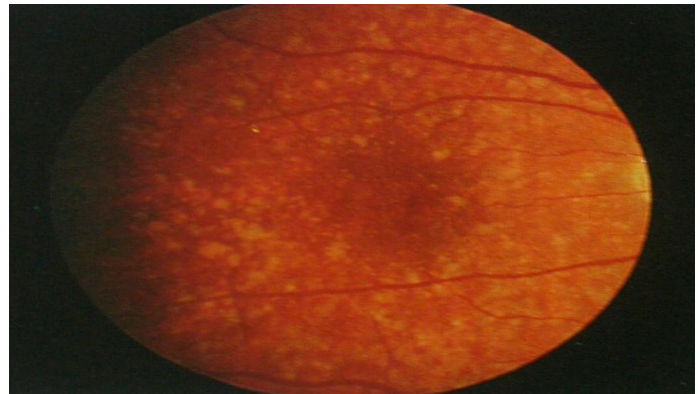
1. Retinal pigment epithelium "RPE", is the outermost layer of retina.
2. Bruch's membrane, is between RPE and choriocapillaris.
3. Choriocapillaris: it is the 3rd layer of choroid.

ARMD is actually the leading cause of blindness in the elderly. These patients develop extracellular breakdown deposits called **“drusen” that form deep in Bruch’s membrane**. This blockage keeps nutrition from percolating up from the choroid to the retina, and conversely blocks photoreceptor waste products from draining down into the choroidal bed. <sup>(2)</sup>

Drusen deposition appears as yellowish small deposits **underneath the retinal pigment epithelium** “fatty depositions”. Can cause **atrophy of the RPE**, and subsequent changes in the choriocapillaris, subretinal neurovascular and burch’s membranes.

### ARMD has two types:

- 1. Dry “90%”:** without bleeding or exudates.
- 2. Wet “10%”:** with bleeding or exudates or both, major cause of blindness.



Dry Macular Degeneration



Wet Macular Degeneration

### **Symptoms:**

- 1. Decreased visual acuity.**
- 2. Metamorphopsia or distortion of vision.**
- 3. Scotoma or blind spot.**

## 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), it is one of the new and very useful techniques providing good information regarding the retinal thickness.

## Treatment:

- Laser treatment of neovascular membrane especially for the wet type.
- Low-vision aid.

## 4. Diabetic Retinopathy

The most accurate predictor of diabetic retinopathy is duration of diabetes. After 10 years, more than half of the patients will show signs of retinopathy, and after 15 years this number increases to nearly 90%. The relative control of glucose during this time is also important, and studies have shown that patients who maintain lower hemoglobin A1C levels have delayed onset and slower progression of eye disease. <sup>(2)</sup>

## Types of diabetic retinopathy:

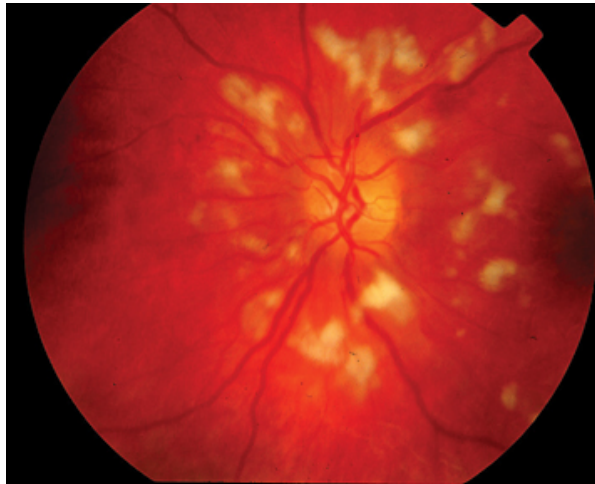
**1. Non-proliferative diabetic retinopathy “NPDR”.** Most patients (95%) have NPDR. This is the earliest stage of retinopathy and it progresses slowly.

Signs: cotton wool spots “soft exudates”, hard exudates, microvascular abnormalities and intraretinal hemorrhage.

The earliest signs of retinal damage are **microaneurysms** and **dot-and-blot hemorrhages** <sup>(2)</sup>.

**1. Proliferative diabetic retinopathy “PDR”.** Characterized by **neovascularization** leading to **preretinal and vitreous hemorrhage, fibrosis** and finally **retinal detachment**.

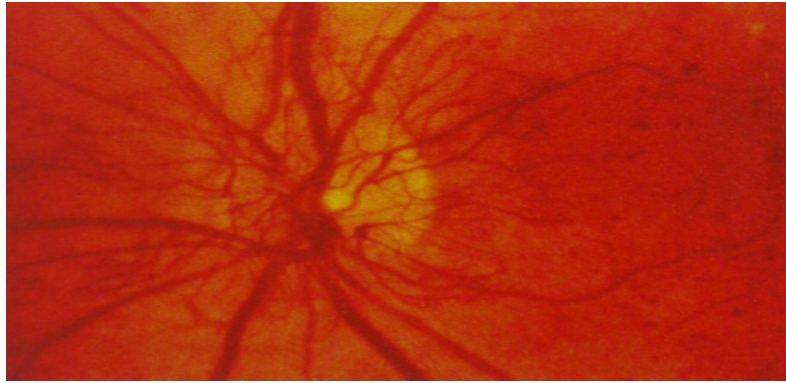
**Soft exudates “Cotton wool spots”:** fuzzy margins and whitish = sign of ischemia “infarction”.



**Hard exudates:** clear margins and yellowish = leaking from the vessels.



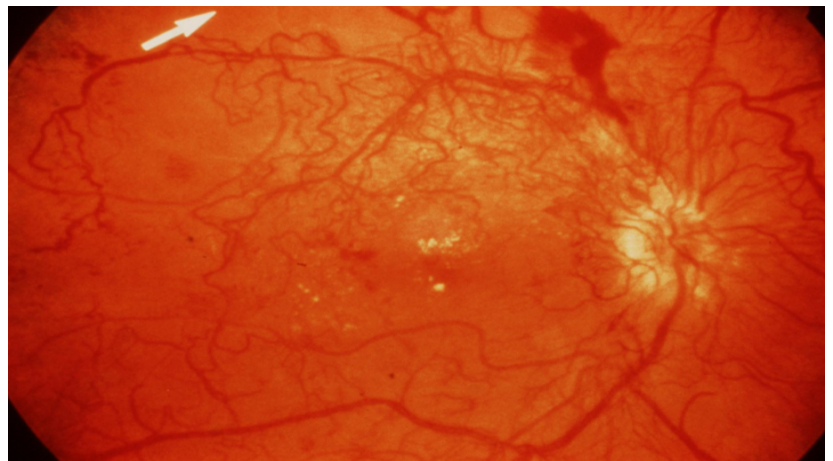




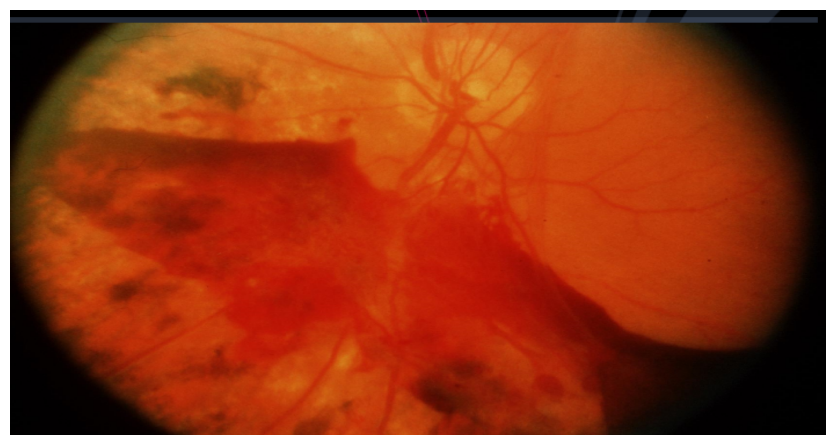
This picture shows a proliferative diabetic retinopathy with neovascularization.

**Neovascularization at the disc (NVD) is more serious and dangerous than neovascularization elsewhere in the periphery (NVE) because neovascular proliferation is associated with fibrovascular proliferation, and the NVD is central and close to the macula and patient could have central tractional detachment which is more serious than the peripheral detachment. “treat with pan-retinal photocoagulation”**

This is advanced PDR with neovascularization everywhere and areas of hemorrhage.



Subhyloid hemorrhage in the top of retina\ preretinal hemorrhage.



## Summary

- Chronic visual loss is a gradual decrease in visual acuity.
- Most common causes are:
  - 1-Cataracts: **most common cause of reversible chronic visual loss**
  - 2-Glaucoma: open angle glaucoma and long term steroids use are associated with chronic visual loss.
  - 3- age-related macular degeneration: is the leading cause of irreversible central visual loss in the United States,
  - 4- Diabetic retinopathy: **is the common cause of irreversible chronic visual loss in our country.**

## References

1. <http://www.uptodate.com/contents/open-angle-glaucoma-epidemiology-clinical-presentation-and-diagnosis>
2. OphthoBook.

## MCQs:

1- Aqueous fluid is produced in which chamber?

- a. anterior chamber
- b. vitreous chamber
- c. posterior chamber
- d. trabecular chamber

2- Which condition would result in an inaccurately high reading with applanation pressure measurement?

- a. thin cornea
- b. thick cornea
- c. edematous cornea
- d. keratoconus

3-What's the difference between dry and wet age-related macular degeneration?

Dry ARMD is when you have drusen and macular RPE atrophy. Wet ARMD implies choroidal neovascularization that has grown up through Bruch's membrane and bleed into the retina. "Wet" essentially means "bloody" in this instance.

4- What are the mechanisms in diabetic retinopathy that might lead to decreased vision? What causes the majority of vision loss in diabetic patients?

There are several mechanisms for potential vision loss in these patients, including:

- Macular edema (probably the leading cause of vision loss)
- Vitreous hemorrhage
- Retinal detachment

5- How do we treat advanced diabetic retinopathy?

Proliferative diabetic retinopathy is treated with PRP (pan retinal photocoagulation)

Answers:

1-C

2-A

If you have any questions/suggestions regarding Ophthalmology teamwork please via:

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