433 Teams

OPHTHALMOLOGY

5

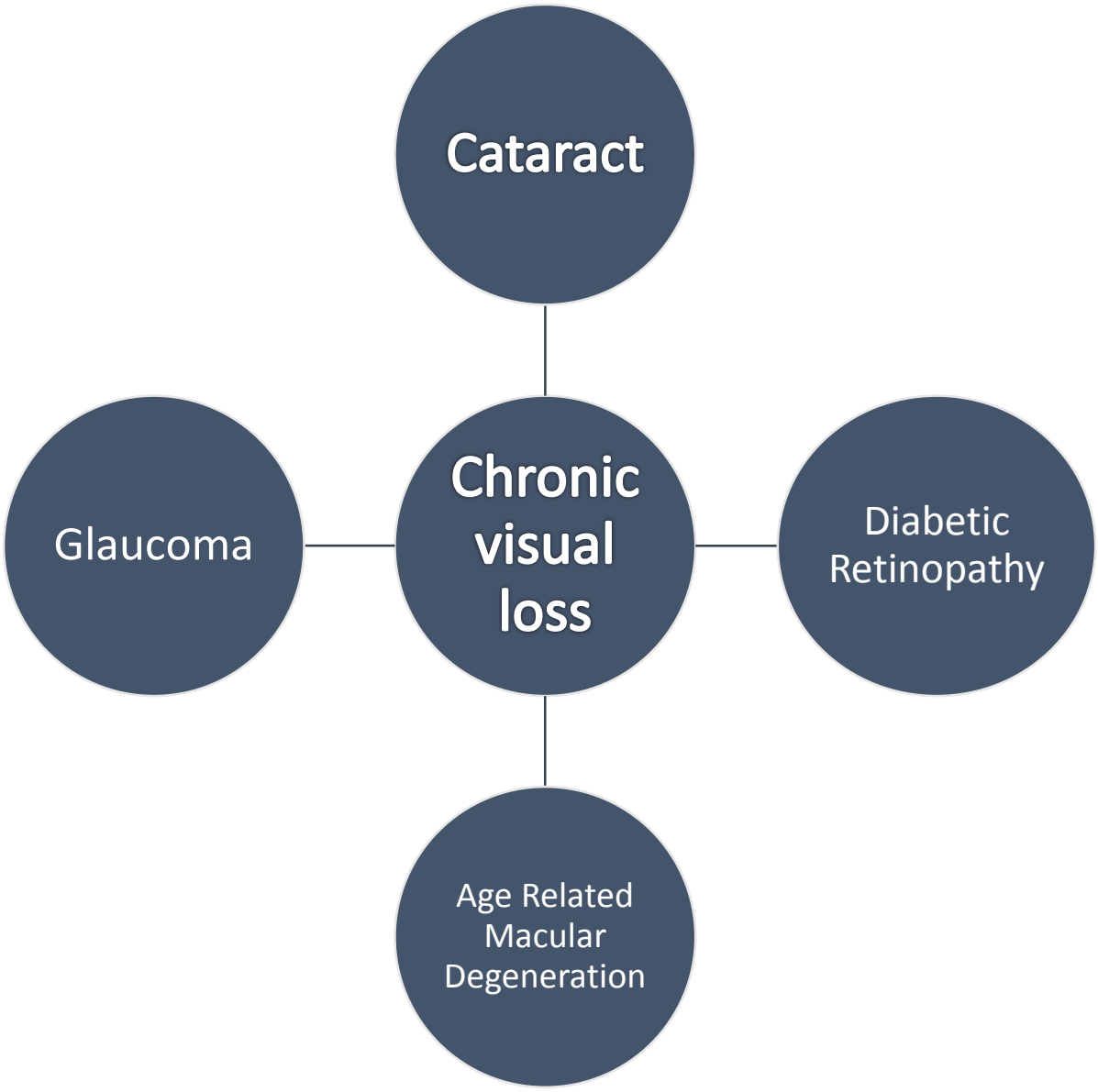
Chronic Visual Loss

Color index:

432 Team – **Important** – **433 Notes** – **Not important**

ophthalmology433team@gmail.com

جامعة الملك سعود
King Saud University


Cataract
Glaucoma
Chronic visual loss
Diabetic Retinopathy
Age Related Macular Degeneration

Chronic visual loss

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

Major causes: (listed 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 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

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

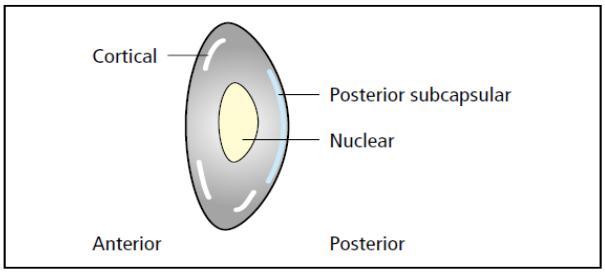


Figure 8.1 The location of different types of cataract.

Based on maturity:

1. Immature **(part of lens involved)**
2. Mature **(entire lens involved)**
3. Tumescent **(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).**

-Age-related cataract is commonly nuclear, cortical or sub-capsular in location

-Steroid-induced cataract is commonly posterior subcapsular.

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

Symptoms

A cataract of sufficient degree causes:

- Painless loss of vision;
- Glare;
- In some instances, a change in refraction.

Signs

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

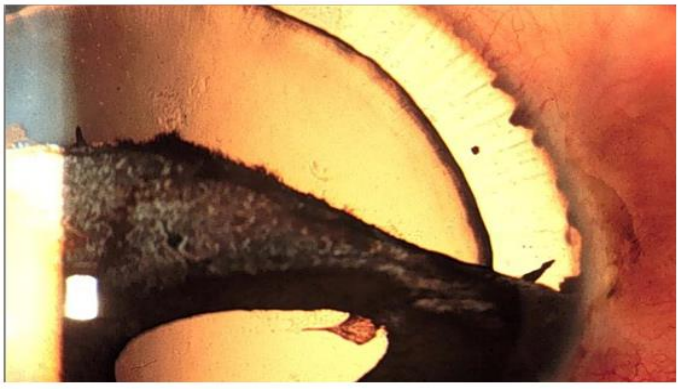
Systemic causes of cataract

- Diabetes.
- Other metabolic disorders (including galactosaemia, hypocalcaemia, Fabry disease).
- Systemic drugs (particularly steroids, chlorpromazine).
- Infection (congenital rubella).
- Myotonic dystrophy.
- Atopic dermatitis.
- Systemic syndromes (Down's, Lowe's).
- Congenital, including inherited, cataract.
- X-radiation.

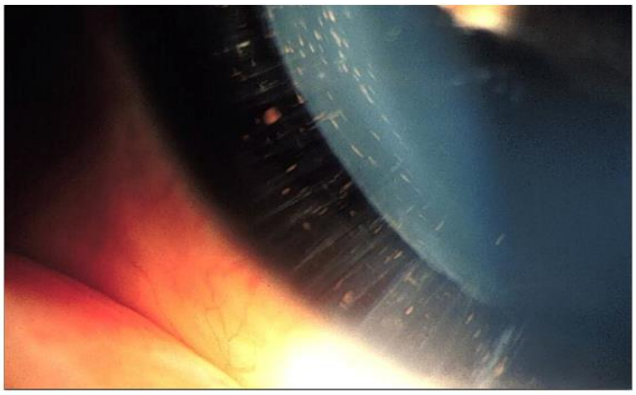
Diagnosis:

- Diagnosis of cataracts is based on a medical history and physical exam. Often tests are used to:
- Confirm the presence of a cataract.
- Rule out other conditions that may be causing vision loss.
  - 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

Cases



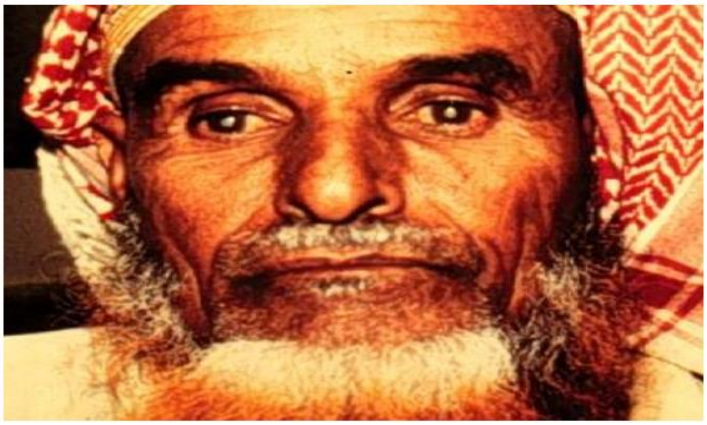
This is a case of blunt trauma showing dialysis of the iris (the iris detached from its origin)



The same patient in this picture showing very fine zonules

The right eye is abnormal:

- Congenital cataract (caused by Rubella) and microphthalmia "small eyes".

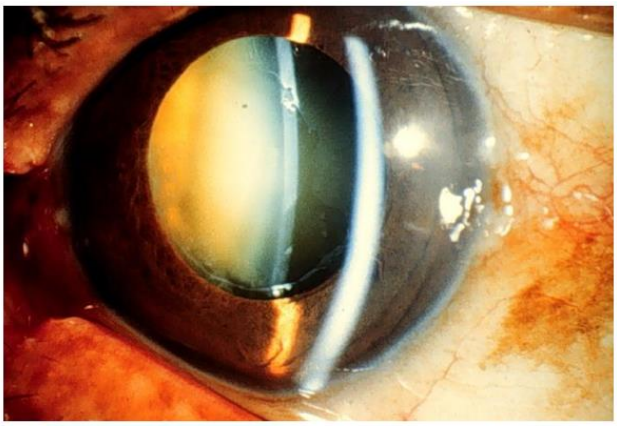


Both eyes are abnormal:

-Showing leukocoria, which is a sign of disease, could be caused by cataract, retinal detachment, or organized vitreous hemorrhage. (Here it is senile cataract)

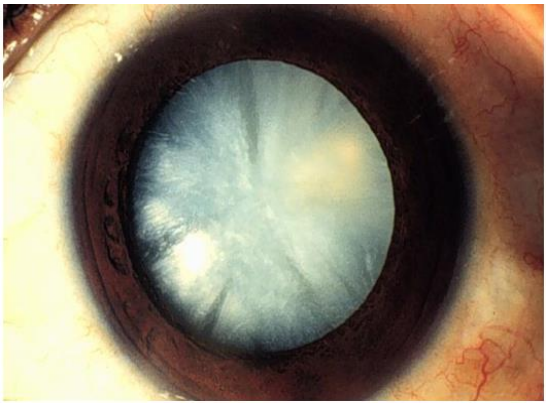
This is nuclear cataract

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



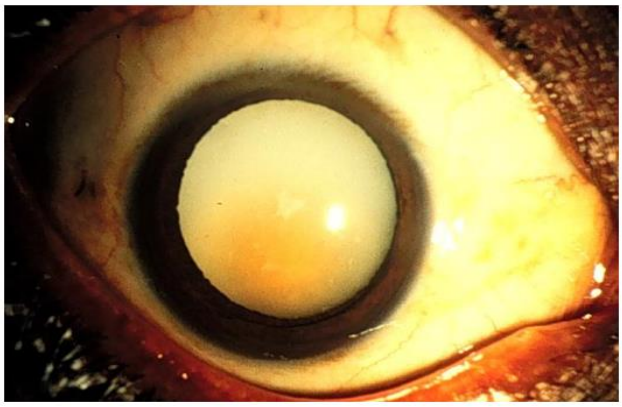
- This is nuclear sclerosis (the nucleus is opaque) and the cortex on top is clear.

-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:

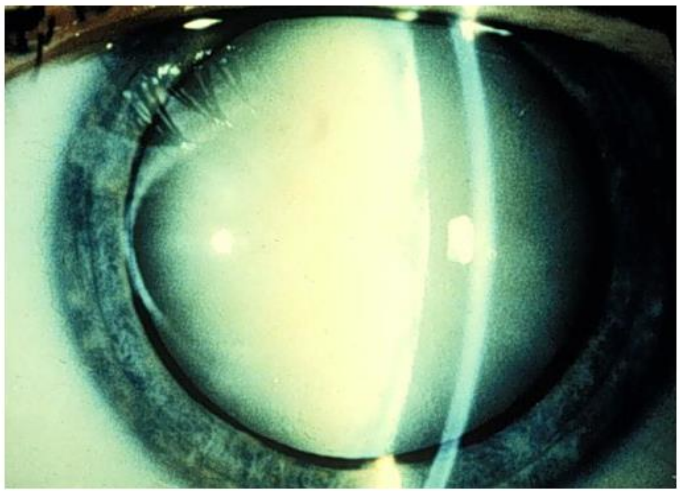
-Whenever you see glaucoma secondary to cataract most probably it is cortical cataract because the lens will be swollen > which increases the pressure > results in glaucoma.



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.

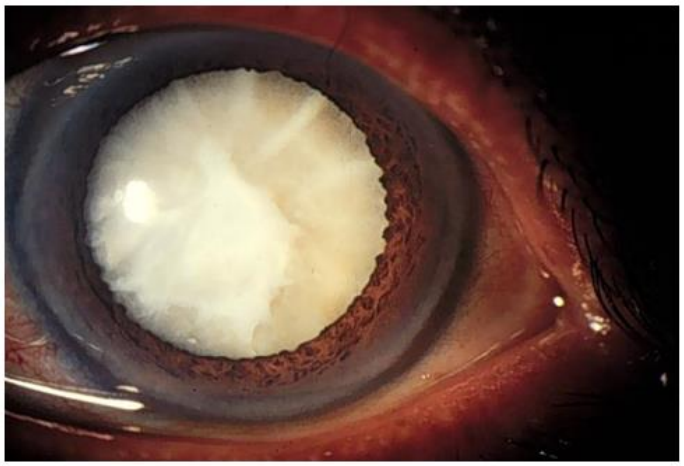
- (normally the solid cortex holds the nucleus in position).



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.

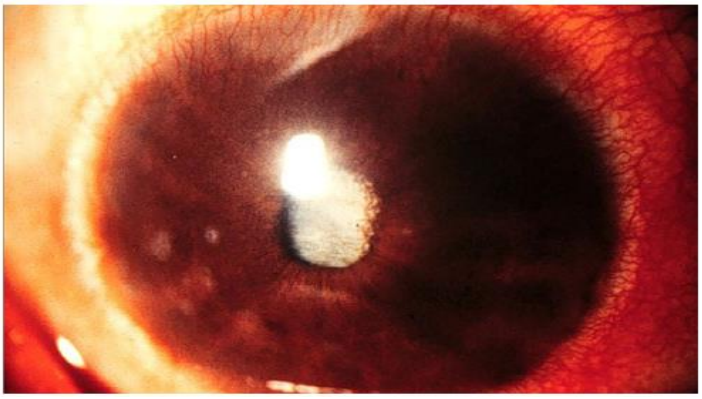
The management is: iridotomy "create a hole in the iris by laser".

Note: Iridectomy: is the surgical removal of part of the iris



-Hypermature cataract showing:

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

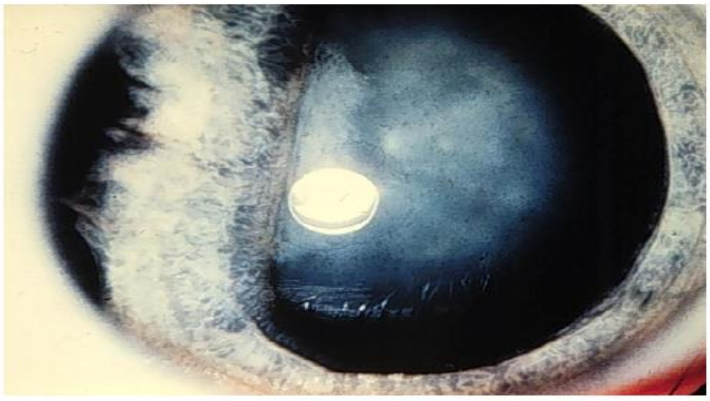


This is cataract secondary to inflammation.

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).

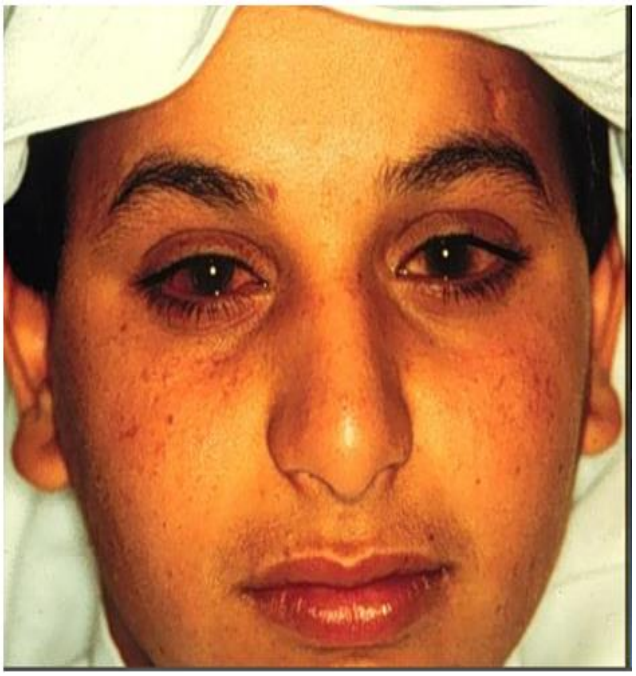
Note: Anterior synechiae (adhesion between the iris and cornea).



- Trauma resulted in iridodialysis and traumatic cataract.

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

- Penetrating trauma > cause acute cataract.



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-Angry-looking eyes

-caused by allergic 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.

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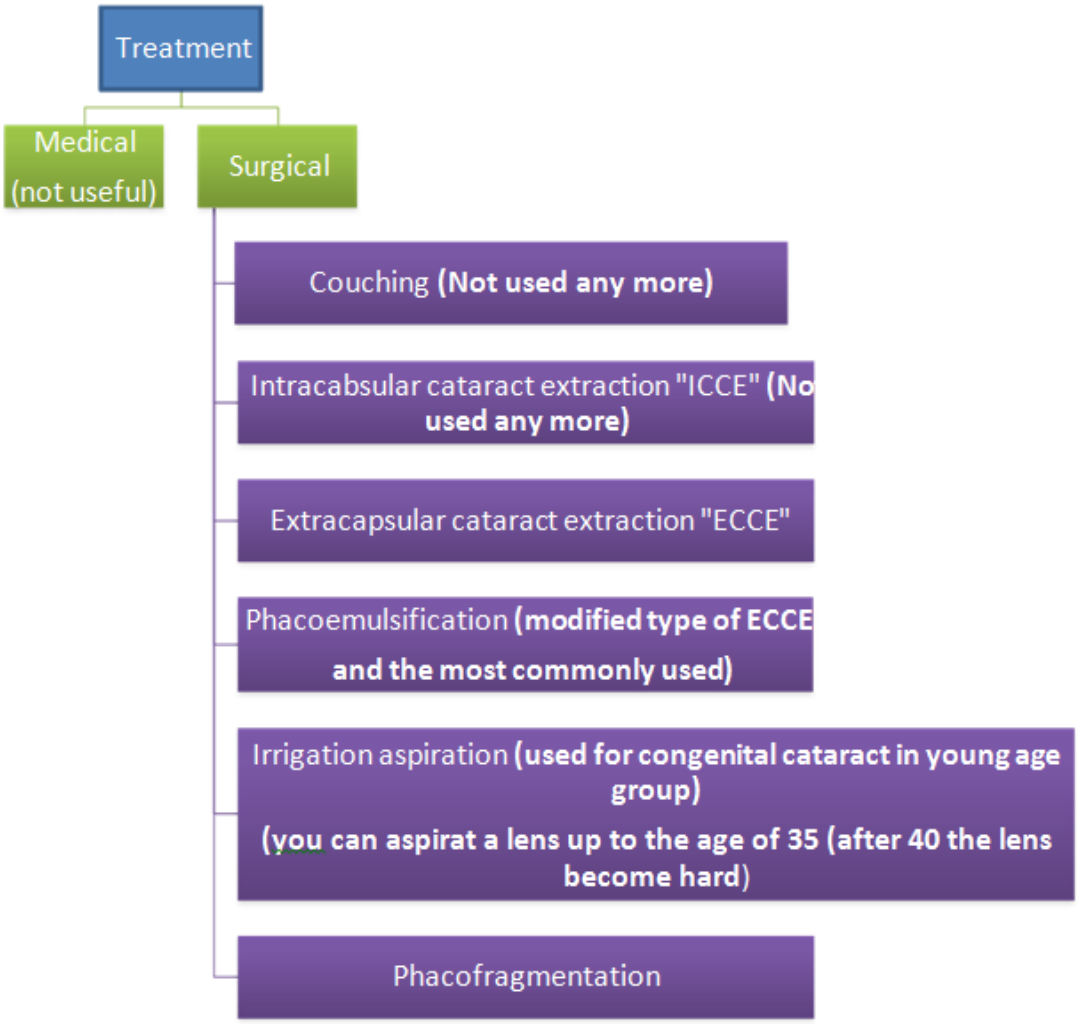
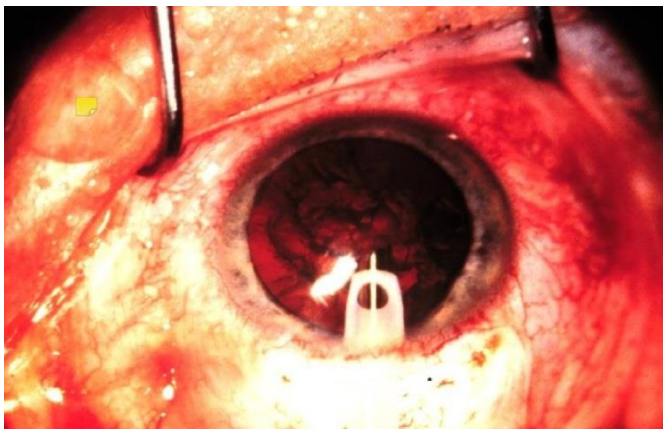


ECCE IOL: Extracapsular Cataract Extraction with Implantation of the Intraocular Lens.
PHACO IOL: phacoemulsification with Intraocular Lens Implantation

Phacoemulsification:

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



Glaucoma

- Definition: Glaucoma is a combination of factors that leads to optic nerve damage presented by visual field defect. **The most important 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.
- If detected early and treated, blindness can be prevented.
- 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**."

- Pathophysiology

Two theories have been advanced to explain how elevated intraocular pressure, acting at the nerve head, damages the optic nerve fibers:

1 Raised intraocular pressure causes mechanical damage to the axons.

2 Raised intraocular pressure causes ischaemia of the nerve axons by reducing blood flow at the nerve head.

The pathophysiology of glaucoma is probably multifactorial and both mechanisms are important.

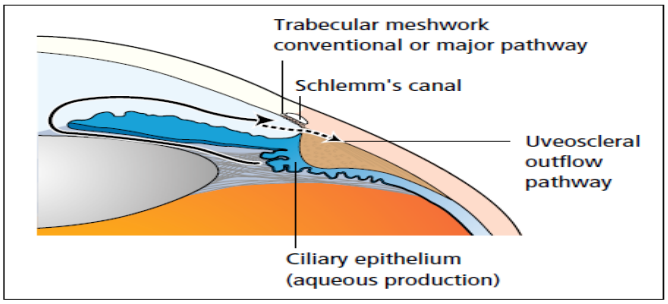


Figure 10.1 Diagram of the drainage angle, showing routes taken by aqueous from production to absorption.

Classification:

Classification of the glaucomas

1 Primary glaucoma:
- Chronic open angle.
- Acute and chronic closed angle.

2 Congenital glaucoma:
- Primary.
- Secondary to maternal rubella infection.
- Secondary to inherited ocular disorders (e.g. aniridia – absence of the iris).

3 Secondary glaucoma (causes):
- Trauma.
- Ocular surgery.
- Associated with other ocular disease (e.g. uveitis).
- Raised episcleral venous pressure.
- Steroid induced.

Primary glaucoma

• 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."

• 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".
Symptoms and signs include loss of visual acuity, pain, conjunctival erythema, and corneal edema.

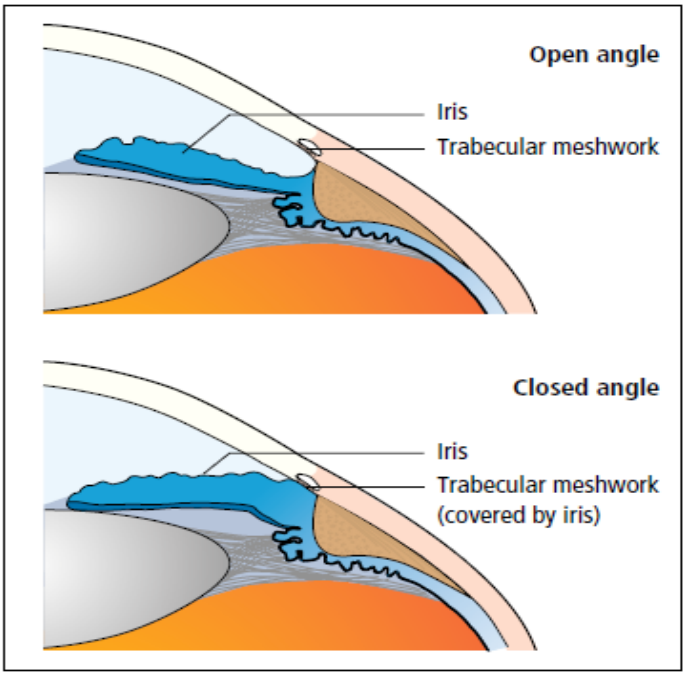


Figure 10.2 Diagram showing the difference between open and closed angle glaucoma. Outflow resistance is increased in each case. In open angle glaucoma the obstruction is due to structural changes in the trabecular meshwork. In closed angle glaucoma the peripheral iris blocks the meshwork.

Symptoms and signs of chronic open angle glaucoma

- Symptomless in its early stages.
- A white eye and clear cornea.
- Raised intraocular pressure.
- Visual field defect.
- Cupped optic disc.

- Investigations:

1. IOP (intraocular pressure) using tonometer

The mean, normal pressure is 15.5 mmHg. The limits are defined as 2 standard deviations above and below the mean (11 – 21 mmHg). In 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.

2. Visual Field exam. Confrontation test, perimetry
3. Exam ONH "optic nerve hypoplasia" to detect abnormal cupping
4. Gonioscopy to visualize the angel

• 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. In the normal eye the cup: disc ratio is usually about 0.3. However, there is some normal variation here. With some people having almost no cup (thus having 1/10 or 0.1), and others having 4/5ths or 0.8 as a cup to disc ratio. If someone has a cup/disc ratio larger than 0.3, then doctors get suspicious that the cup could be getting larger than it used to be. (These numbers according to Glaucoma Research Foundation website)

• 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.**

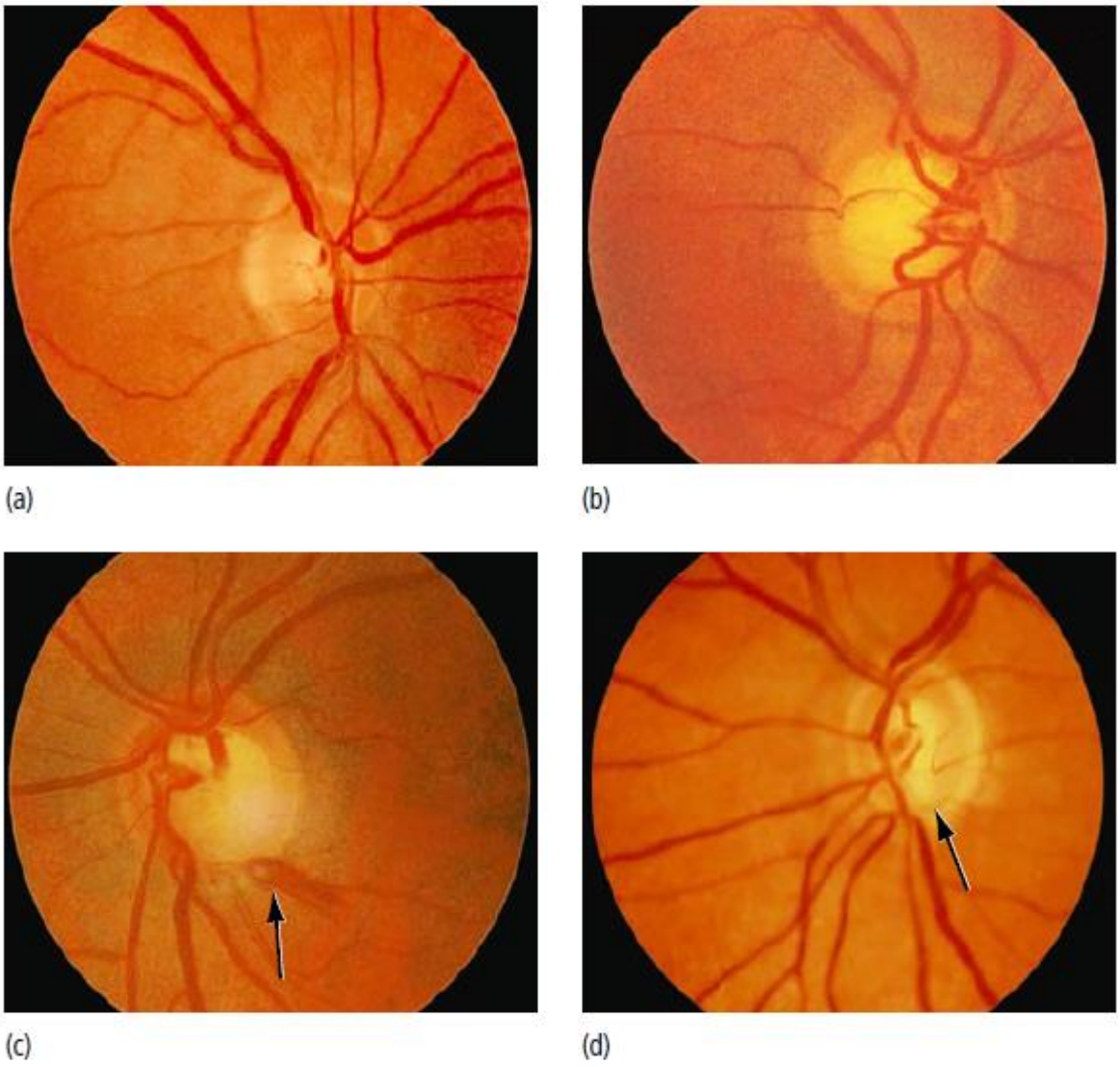


Figure 10.5 Comparison of (a) a normal optic disc, (b) a glaucomatous optic disc. (c) A disc haemorrhage (arrowed) is a feature of patients with normal tension glaucoma. (d) A glaucomatous notch (arrowed) in the disc.

When to start screening for glaucoma

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

How to screen?

1. Tonometry, which measures the IOP.

2. Cup-to-disc ratio, refer the patient to the ophthalmologist if the ratio ≥ 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

Treatment

Treatment is aimed at reducing intraocular pressure. Three modalities of treatment are available

1. Medical treatment;
2. Laser treatment;
3. Surgical treatment. "trabeculectomy"

Notes:

- The thickness of the cornea affects the IOP reading. After Lasik, the cornea becomes weak and a false low reading of IOP could occur, so the diagnosis of glaucoma might be missed.

Table 10.1 Examples and mode of action of drugs used in the treatment of glaucoma. Side effects occur with variable frequency. Systemic effects are due to systemic absorption of the drug.

Drug	Action	Side effects
Topical agents		
Beta-blockers (timolol, carteolol, levobunolol, metipranolol, betaxolol-selective)	Decrease secretion	Exacerbate asthma and chronic airway disease Hypotension, bradycardia, heart block
Parasympathomimetic (pilocarpine)	Increase outflow	Visual blurring in the young Darkening of the visual world due to pupillary constriction Initially, headache due to ciliary spasm
Sympathomimetic (adrenaline, dipivefrine)	Increase outflow Decrease secretion	Redness of the eye Headache palpitations
Alpha-2 agonists (apraclonidine, brimonidine)	Increase outflow through the uveoscleral pathway Decrease secretion	Redness of the eye Fatigue, drowsiness
Carbonic anhydrase inhibitors (dorzolamide, brinzolamide)	Decrease secretion	Stinging Unpleasant taste Headache
Prostaglandin analogues (latanoprost, travaprost, bimatoprost, tafluprost, unoprostone)	Increase outflow through the uveoscleral pathway	Increased pigmentation of the iris and periocular skin Lengthening and darkening of the lashes, conjunctival hyperaemia Rarely, macular oedema, uveitis
Systemic agents		
Carbonic anhydrase inhibitors (acetazolamide)	Decrease secretion	Tingling in limbs Depression, sleepiness Renal stones Stevens–Johnson syndrome

Age Related Macular Degeneration "ARMD"

Age - related macular degeneration (AMD) is the commonest cause of irreversible visual loss in the developed world.

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 neighbouring 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 .

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.

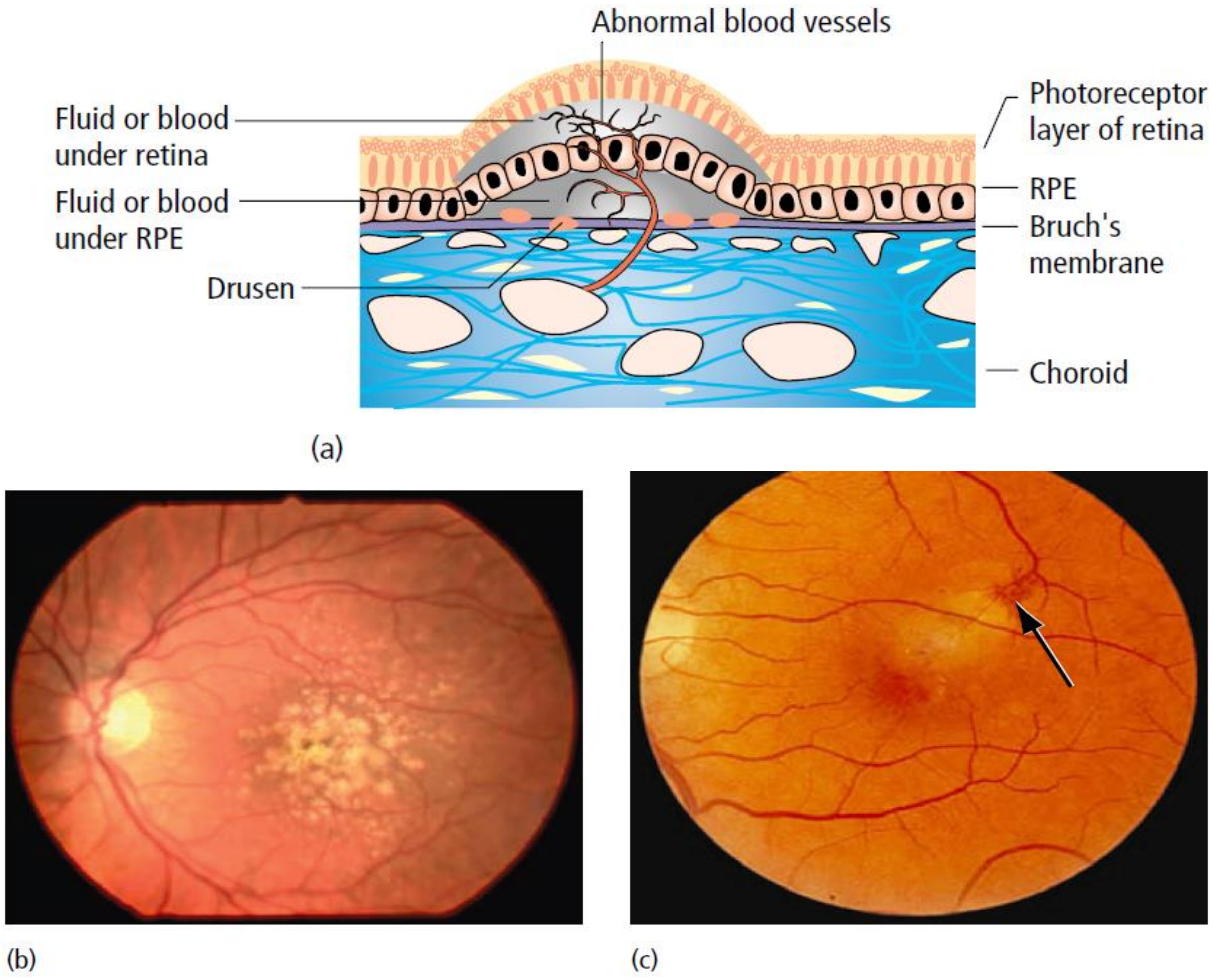
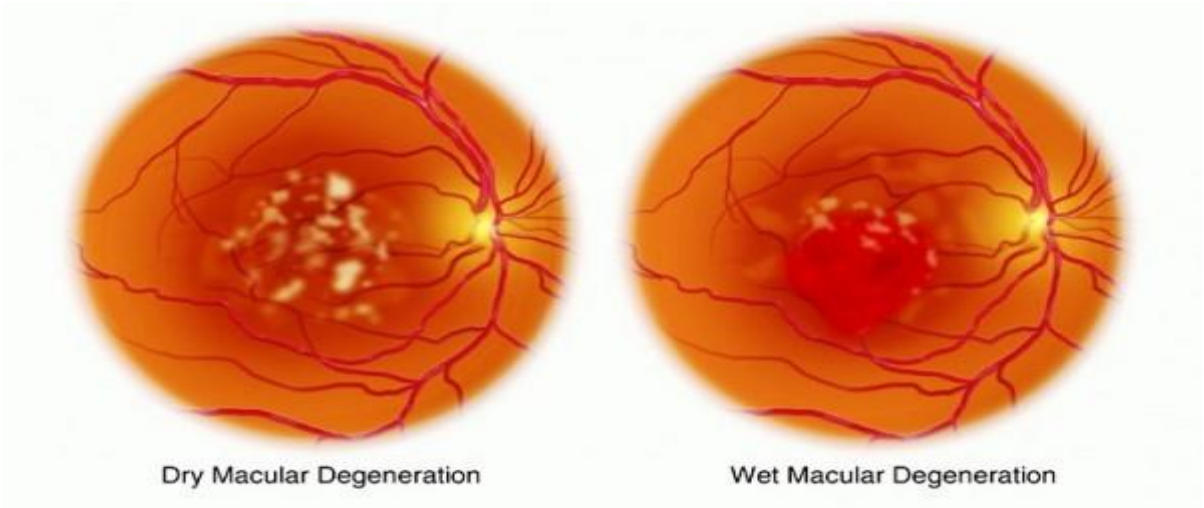


Figure 11.1 (a) The pathogenesis of exudative age-related macular degeneration (RPE, retinal pigment epithelium). Pictures of: (b) dry AMD: note the discrete scattered yellowish sub-retinal drüsen; (c) wet AMD: note the small haemorrhage associated with the sub-retinal membrane.

Two types of ARMD:

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



Symptoms of Macular dysfunction

- 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"

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 new test and very useful technique providing good information regarding the retinal thickness.

Treatment:

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

Diabetic Retinopathy

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.

Factors thought to be important in the development of diabetic retinopathy include:

- Duration of diabetes: 80% have retinopathy after 20 years of disease.
- Poor diabetic control.
- Coexisting diseases, particularly hypertension.
- Smoking.

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. 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.

Screening

Screening should begin by 5 years after diagnosis in patients with type I disease, and may be from the time of presentation in type II disease, since its time of onset is unknown.

Table 12.1 The classification of diabetic retinopathy (note that diabetic maculopathy may coexist with other stages in the classification).

Stage	Description
No retinopathy	There are no abnormal signs present on the retina. *Vision normal*
Background	Signs of microvascular leakage (haemorrhage and exudates) away from the macula. *Vision normal*
Maculopathy	Exudates and haemorrhages within the macula region, and/or evidence of retinal oedema, and/or evidence of retinal ischaemia. *Vision is reduced; sight-threatening*
Preproliferative	Evidence of occlusion (cotton-wool spots). The veins become irregular and may show loops. *Vision normal*
Proliferative	The occlusive changes have led to the release of a vasoproliferative substance from the retina, resulting in the growth of new vessels either on the disc (NVD) or elsewhere on the retina (NVE). *Vision normal; sight-threatening*
Advanced	The proliferative changes may result in bleeding into the vitreous or between the vitreous and the retina. The neuroretina may also be pulled from its overlying pigment epithelium by a fibrous proliferation associated with the growth of the new vessels. *Vision is reduced, often acutely, with vitreous haemorrhage; sight-threatening*

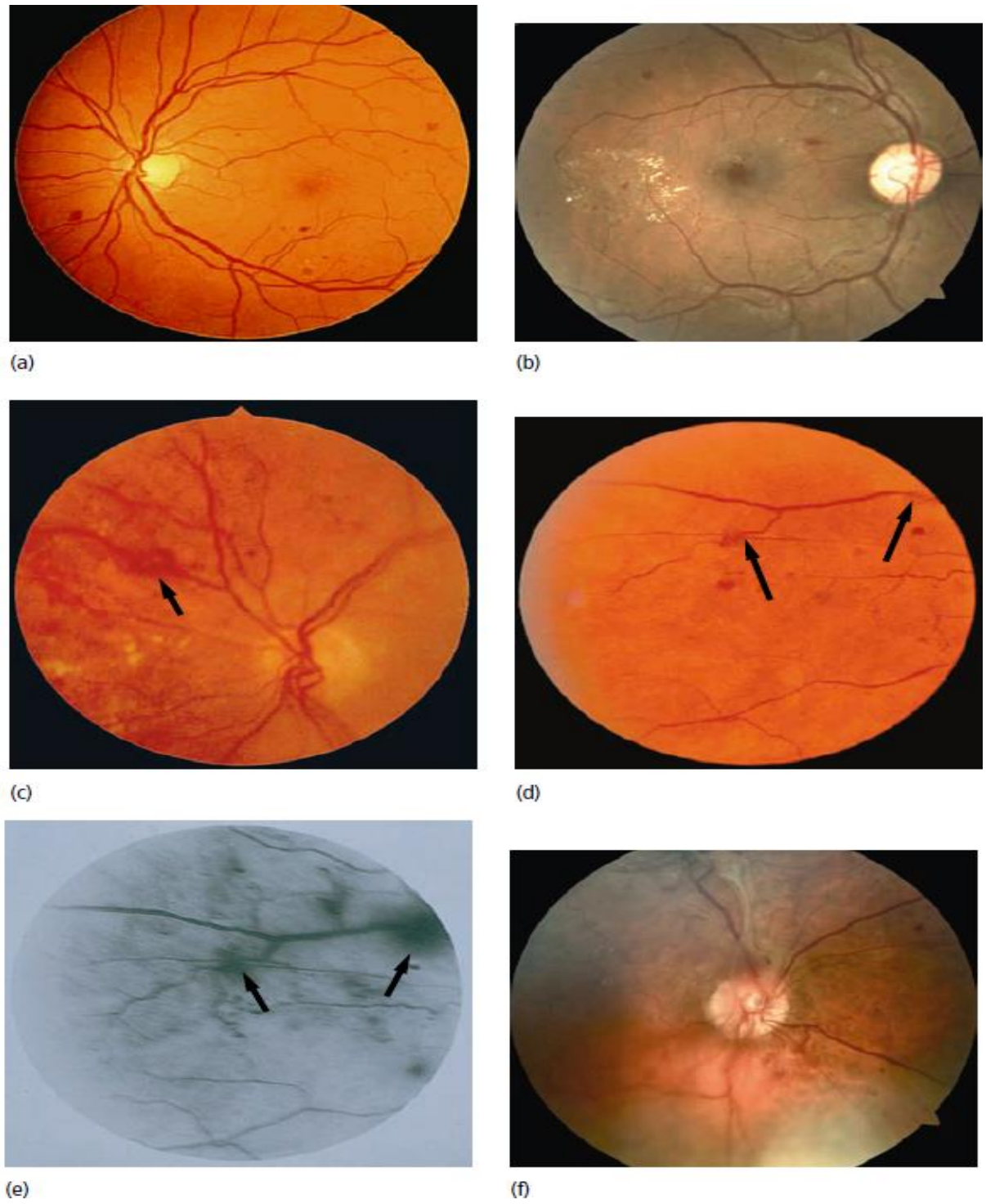


Figure 12.3 The signs of diabetic eye disease. (a) Background diabetic retinopathy. (b) Diabetic maculopathy: note the circinate exudate temporal to the macula. (c) Preproliferative retinopathy with a venous loop. (d, e) Proliferative retinopathy: new vessels have formed on the retina, their presence demonstrated by leakage of fluorescein (hyperfluorescence) on the fluorescein angiogram; closure of some of the retinal capillary network is demonstrated by its failure to fill with fluorescein. (f) Advanced diabetic retinopathy: the neovascularization has caused a traction retinal detachment.

Non-proliferative diabetic retinopathy "NPDR"

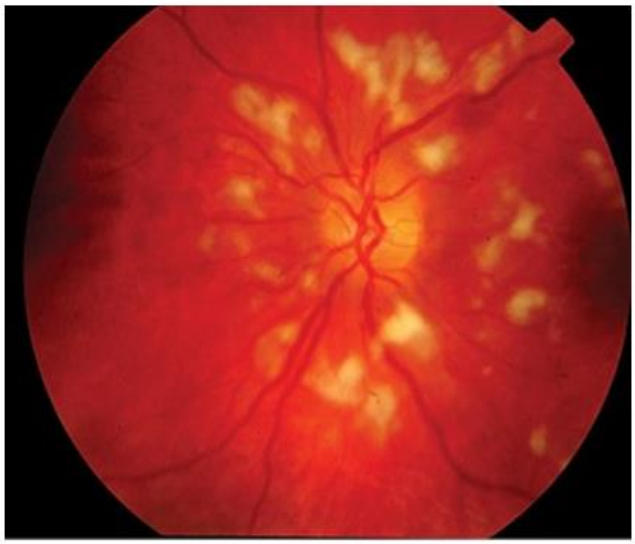
Most patients (95%) have NPDR. This is an early stage of retinopathy and it progresses slowly.

Signs: cotton wool spots "soft exudates", hard exudates, microvascular abnormalities and intra-retinal hemorrhage.

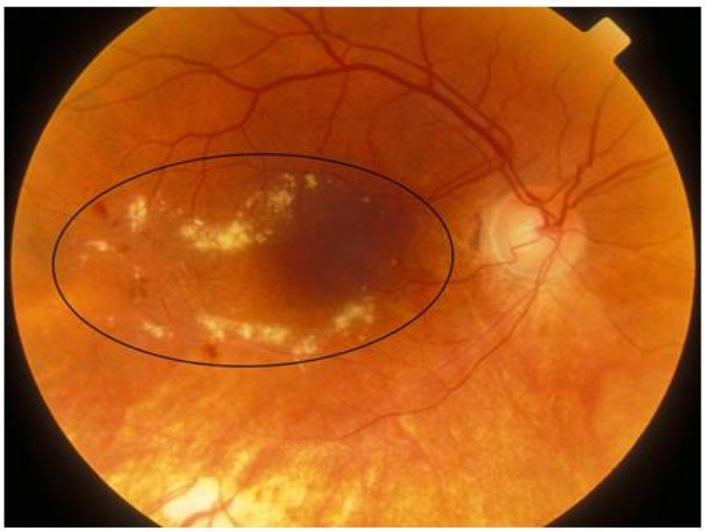
The earliest signs of retinal damage are microaneurysms and dot-and-blot hemorrhages.

Proliferative diabetic retinopathy "PDR"

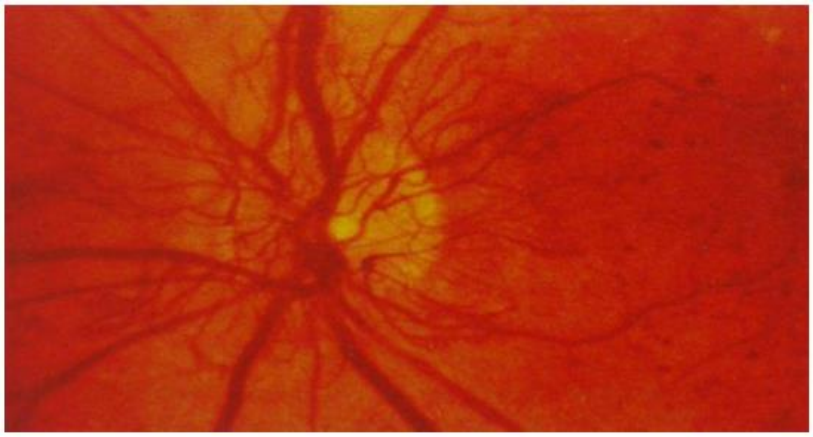
Characterized by neovascularization leading to pre-retinal and vitreous hemorrhage, fibrosis and finally retinal detachment.



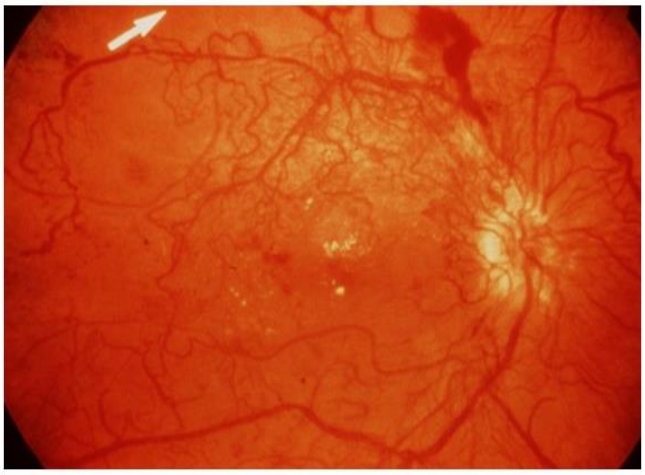
Soft exudates "Cotton wool spots": fuzzy margins and whitish = sign of ischemia "infarction"



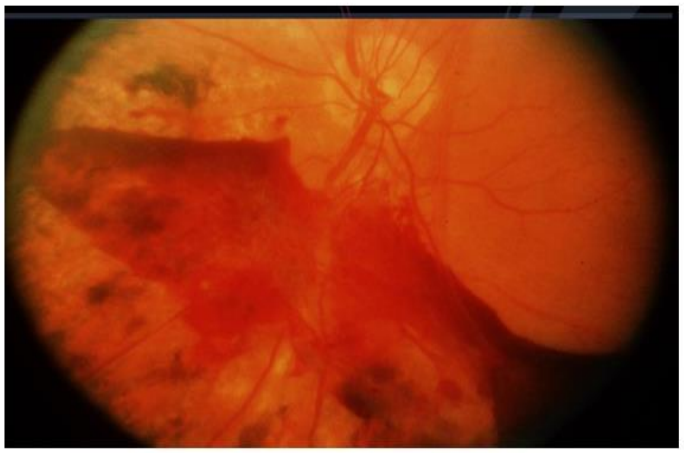
Hard exudates: clear margins and yellowish= leaking from the vessels.



A proliferative diabetic retinopathy with neovascularization



Advanced PDR with neovascularization everywhere and areas of hemorrhage



Subhyloid hemorrhage in the top of retina\preretinal hemorrhage

Note

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. "treated with pan-retinal photocoagulation"

Diabetic retinopathy: clinical observations

- Younger patients are more likely to develop proliferative disease.
- Older patients more commonly develop a maculopathy, but because type II disease is more common, it is also an important cause of proliferative disease.

Treatment

- The mainstay of treatment for sight -threatening diabetic retinopathy is laser therapy (photocoagulation).
- Surgical removal of the vitreous gel (vitrectomy) is done when there is bleeding (vitreous hemorrhage) or retinal detachment.
- Sometimes injections of an anti-VEGF (vascular endothelial growth factor) medicine or an anti-inflammatory medicine may have a role.

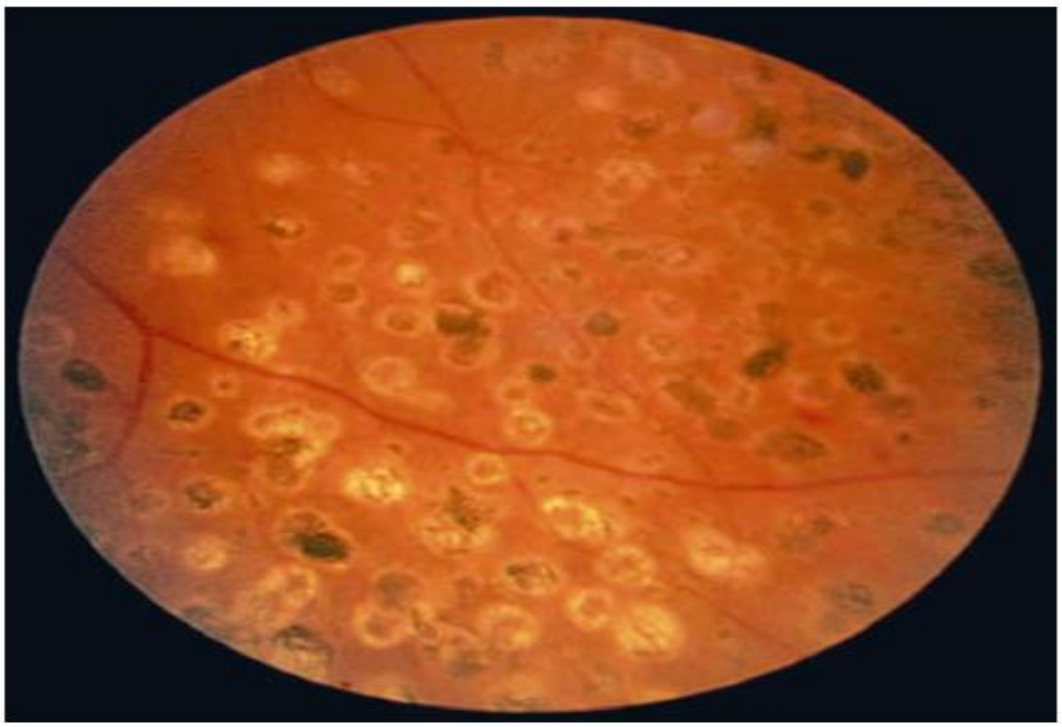


Figure 12.4 Typical appearance of retinal laser burns.

Summary

- Chronic visual loss: **Slowly progressive visual loss (chronic means within months to years).**
- Cataract is the name given to any light-scattering opacity within the lens wherever it is located.
- Cataract is the commonest cause of treatable blindness in the world.
- Glaucoma is a combination of factors that leads to optic nerve damage presented by visual field defect.
  The most important risk factor is increased IOP
- Less common type of glaucoma is normal or low tension glaucoma.
- Glaucoma is treated medically, surgically or by laser.
- Age - related macular degeneration (AMD) is the commonest cause of irreversible visual loss in the developed world.
- Factors thought to be important in the development of diabetic retinopathy include: Duration of diabetes, Poor diabetic control, Coexisting hypertension and Smoking.
- Most patients (95%) with diabetic retinopathy have NPDR. This is an early stage of retinopathy and it progresses slowly.
- PDR is Characterized by neovascularization leading to pre-retinal and vitreous hemorrhage, fibrosis and finally retinal detachment
- The mainstay of treatment for sight -threatening diabetic retinopathy is laser therapy (photocoagulation).

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 low reading with applanation pressure measurement?

a. thin cornea
b. thick cornea
c. edematous cornea
d. Diabetes

Answers: 1-C 2-A

A pachymetry test is a simple, quick, painless test to measure the thickness of the cornea.

Done By:

Othman Abid
Khalid Alsuhaibani