



Chronic Visual Loss

2017-2018
Objectives:



Not given

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(special thanks to Lina Aljurf)

Resources: Team 433, Doctors Notes

433 Team

Important

Doctor's Notes

Explanation

Chronic visual loss


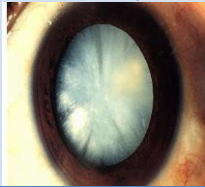
Definition: **Slowly progressive** visual loss (chronic means within months to years).

Major causes: **(starting from the most common)**

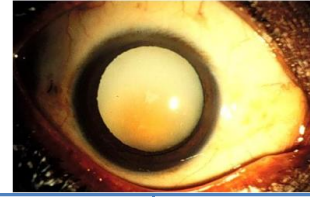
- 1- Cataract
- 2- Diabetic retinopathy
- 3- Glaucoma
- 4- Macular degeneration One should recognize the normal first to be able to identify the abnormal e.g.:
 - Normal macula
 - Lens clarity **(normally it has a shade if you don't know this normal appearance you may think it is cataract)**
 - Optic nerve head
 - Normal retina

Cataract

- 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 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	<p style="text-align: center;">Nuclear</p> <p>Early stages of cataract: new fibers compress old fibers causing sclerosis. Associated with Myopia</p> 	<p style="text-align: center;">Sub-capsular</p> <p>Anterior: Fibrous metaplasia in the central zone, Posterior: granular or plaque like; migration of epithelial cells (DM, steroid, ocular inflammation)</p>	<p style="text-align: center;">Cortical</p> <p>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.</p> 	
	<p style="text-align: center;">Zonular or lamellar</p> <p>Usually Bilateral opacities of specific zones; spokes like or wheel appearance. congenital</p>	<p style="text-align: center;">Sutural</p> <p>Opacity only in the embryonic nucleus Y shaped : congenital</p>	<p style="text-align: center;">Morgagnian</p> <p>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</p>	

osmosis) it loses its support of the nucleus and allow free movement of it within the capsule bag.

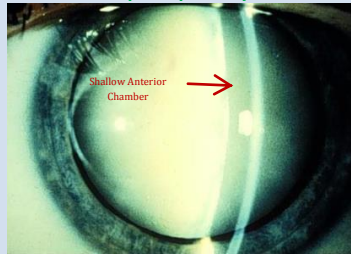


Types Based on Maturity

Immature
(part of lens involved)

Intumescent
(congested)

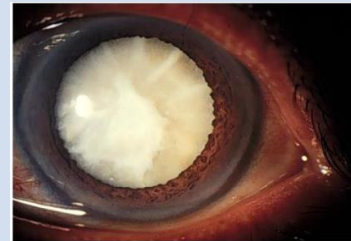
In a stage between mature and immature.
Rapid swelling of the lens causes shallow anterior chamber and block the aqueous from circulating causing (phacomorphic glaucoma) which is treated by Peripheral Iridotomy (hole or canal in the periphery of the iris).



Mature
(entire lens involved)

Hyper-Mature

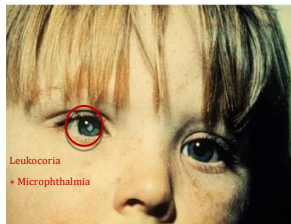
happens when you leave the mature cataract for long time, the lens may become dehydrated and the capsule become wrinkled and fibrosed, calcification might be associated.
Can lead to phacolytic glaucoma due to the leakage of proteins which block the mesh network causing open angle glaucoma



Based on onset:

Congenital
(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



Infantile

(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)

Pre-senile




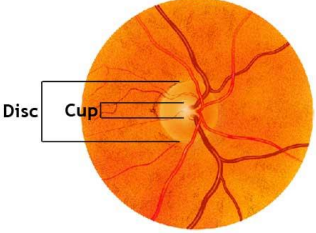

Early onset cataract

Senile

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.

<p>Based on cause (secondary cataract):</p>	<p>Traumatic Blunt or penetrative and takes time to appear!</p> 	<p>Neoplasm Melanoma or Retinoblastoma</p>	<p>Inflammatory Anterior Uveitis: Iritis or Iridoscleritis</p> 	<p>Toxic Foreign Body</p> <p>Steroid induced cataract:</p> <p>This pt is case of vernal keratoconjunctivitis severe allergic inflammatory disease common in the southern areas, treated by steroids, but when over dosed cataract (post. Subcapsular) or glaucoma can result.</p> 
<p>Signs and symptoms</p>	<p>Symptoms : Painless loss of vision; Glare; difficulty seeing in the presence of bright light In some instances, a change in refraction (Myopia).</p>		<p>Signs :</p> <ul style="list-style-type: none"> - Visual acuity is reduced. - Cataract appears black against the red reflex when - The eye is examined with a direct ophthalmoscope 	
<p>Diagnosis and Treatment :</p>	<p>Diagnosis:</p> <ol style="list-style-type: none"> 1- History: gradual visual loss and cloudy vision 2- P/E : <ul style="list-style-type: none"> • Visual Acuity • Flash light examination • Slit lamp examination • Direct ophthalmoscope • Refraction and Retinoscopy • Red and Green Light • Ultrasound (B scan for posterior examination) 		<p>Treatment:</p> <p>Medical Treatment isn't effective !</p> <p>Surgical :</p> <p>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)</p> <ul style="list-style-type: none"> • Couching : old risky and require wearing glasses • ICCE not used anymore. • ECCE (endoscopic capsular cataract extraction) :  (4min with steps written) • Phacoemulsification :  (Animated 2min) • Modified ECCE; most commonly used. • Aspiration and Irrigation : young lenses because they are soft , after 35 it hardens and difficult to aspirate; used for congenital cataract. • Phacofragmentation. 	

Glaucoma

<p>Definition</p>	<p>Optic nerve damage presented by visual field defect. Commonly caused by increased intra ocular 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).</p>			
<p>Types</p>	<p>Open Angle Glaucoma</p> <p>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, <u>gradual vision loss.</u>"</p> <p>"The major risk factors for developing open-angle glaucoma include age, black race, family history, and elevated intraocular pressure , Myopia, DM, OCP" <i>More serious because it's asymptomatic</i></p>	<p>Closed Angle Glaucoma</p> <p>1- Pupillary Block : After the age of 40 ; prolonged 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!</p> <p>2- Non-pupillary block: Younger age ,Far eastern ethnicity, plateau iris with thick peripheral iris roll, so not fully relieved by iridotomy.</p> <p>Occurs when the angle between the cornea and iris closes abruptly.</p> <p>With this closure, aqueous fluid can't access the drainage pathway entirely, causing ocular pressure to increase rapidly.</p> <p>This is an ophthalmological emergency and patients can <u>lose all vision in their eye within hours</u>". Symptoms and signs include loss of visual acuity, pain, conjunctival erythema, and corneal edema.</p>		
<p>Investigations</p>	<p>IOP (intraocular pressure) Using Tonometer Normal Is 11 – 21 mmHg</p> 	<p>Visual Field exam</p> <ul style="list-style-type: none"> - Confrontation test. - perimetry <div style="display: flex; justify-content: space-around;">   </div> <p><small>Confrontation visual field exam Humphrey automated perimetry</small></p>	<p>Exam ONH (optic nerve head) Fundoscopy</p>  <p><i>Comment on: optic nerve color, margins, vessels, the presence of cupping!</i></p>	<p>Gonioscopy To measure the angle</p> 

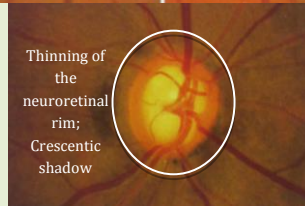
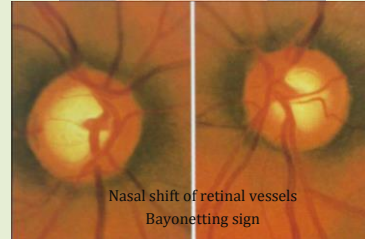
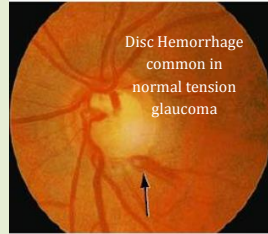
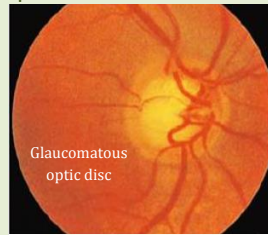
Changes in Glaucoma

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
 Pathological cupping: Cup:disc ratio more than 0.5



Treatment & Prevention

Start screening after the age of 40, every 2 to 4 years by Tonometry and cup to disc ratio.



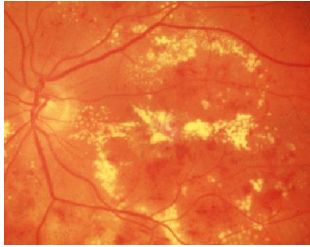
Patient is referred for treatment when:

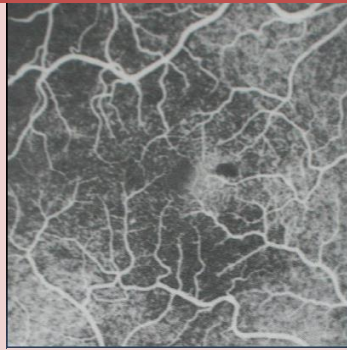
- 1- IOP more than 21mmgh.
- 2- C:D ratio more than 0.5
- 3- One cup significantly larger than the other one.

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

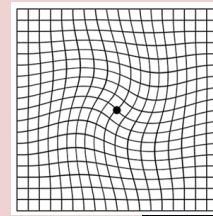
<p>Definition</p>	<p>Changes in the macula affects outer retinal layer, retinal pigment epithelium, Bruch's membrane and choriocapillaris.</p>	
<p>pathogenesis</p>	<p>Over time, undigested lipid products, such as the age pigment lipofuscin, accumulate in the RPE (Retinal pigment epithelium) and the excess 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 <u>dry or non – exudative form</u> of AMD.</p> <p>In the less common, <u>exudative or ' wet ' form</u>, 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 <u>sub-retinal neovascular membrane</u>.</p> <p>Types of ARM:</p> <ol style="list-style-type: none"> 1. Dry "90%": without bleeding or exudates. 2. Wet "10%": with bleeding or exudates or both, major cause of blindness. 	
<p>Signs and Symptoms</p>	<p>Symptoms:</p> <ul style="list-style-type: none"> • Blurred central vision. • Distorted vision (metamorphopsia) • Reduction (micropsia) or enlargement (macropsia) of object size • Loss of the central visual field (scotomata) <p>Signs:</p> <ul style="list-style-type: none"> • foveal reflex is absent • Yellow, well circumscribed drüsen may be seen • Sub-retinal, pre-retinal, haemorrhages may be seen. "wet type" 	 <p>Normal Macula : Darker than surroundings. Avascular Contains only cones</p>  <p>Drusen deposits : yellow spots</p>  <p>Macular Exudate</p>
<p>Examination</p>	<ol style="list-style-type: none"> 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 <ul style="list-style-type: none"> • Fluorescein angiography, inject IV fluorescein to visualize the retinal vessels. • Indocyanine green dye • OCT (Optical Coherence Tomography) 	



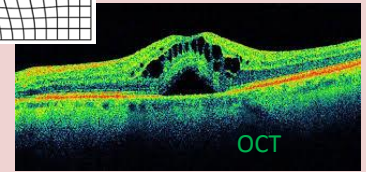
Normal Fluorescein Angiography



Blood entrapped in the membrane
Fluorescein Angiography



Abnormal
Amsler grid Test



OCT

Treatment

- 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 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

<p>Background Diabetic Retinopathy</p> <p>Micro aneurysm Dot and blot hemorrhage Exudate</p> <p>-Earliest signs but persist-</p>	<p>Diabetic Maculopathy</p> <p>Macular Edema Ischemia</p>	<p>Proliferative Diabetic Retinopathy</p> <ul style="list-style-type: none"> • Cotton wool spot :(accumulation of debris within the nerve fiber layers) • Venous changes :(increased tortuosity, looping, beading, sausage like segmentation) • Arterial changes:(narrowing or silver wiring, Obliteration) • Intraretinal microvascular Anomalies) <ul style="list-style-type: none"> • Deep Retinal Hemorrhage. 	<p>Advanced diabetic disease</p> <ul style="list-style-type: none"> • Traction Retinal Detachment (there are three types of retinal detachment : Rhegmatogenous retinal detachment + Exudative (serous) retinal detachment +Tractional retinal detachment) • Viterous Hemorrhage • Neovascular Glaucoma
<p>Treatment</p>	<p>Medical : FenoFibrate (antilipid) Anti VEGF (intraretinal) Surgical : Focal Laser Photocoagulation in case of macular edema. In case of neovascularization pan retinal photocoagulation is the treatment of choice!</p>		