Chronic Visual Loss

Objectives:

- Chronic Glaucoma:
    A. Causes.
    B. Types.
    C. Management.
- Senile Cataract.
- Diabetic macular edema.
- Age Related Macular Degeneration (ARMD).

Sources:

- Lecture slides.
- Essentials of Ophthalmology.
- 427 Team.

Chronic visual loss

Definition: Gradual decrease of visual acuity.

Causes of slowly progressive **visual loss** in an adult patient (MCQ):

1. Glaucoma.

2. Cataract.

3. Macular degeneration.

4. Diabetic retinopathy .

- Age by itself is not a cause of decrease of visual acuity.
- **Central vein occlusion** causes **acute visual loss**.

First evaluation:

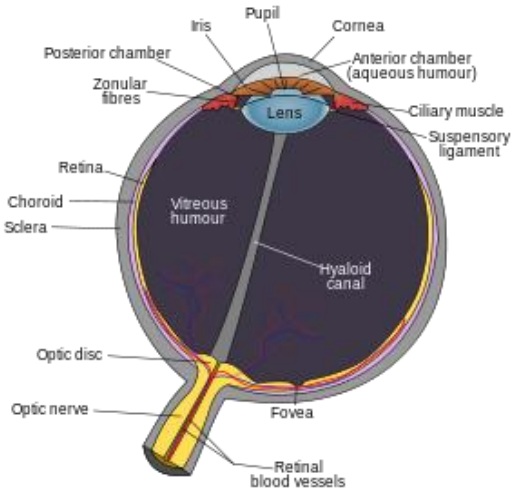
Measure intraocular pressure with a tonometer.

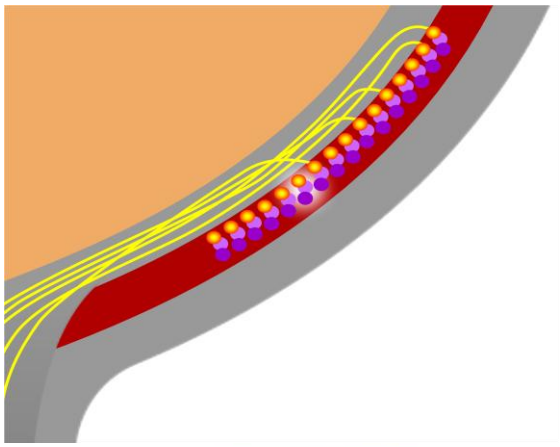
2. Evaluate the nerve head, classifying it as normal, or abnormal, because pt may have cupping of disc with normal IOP.

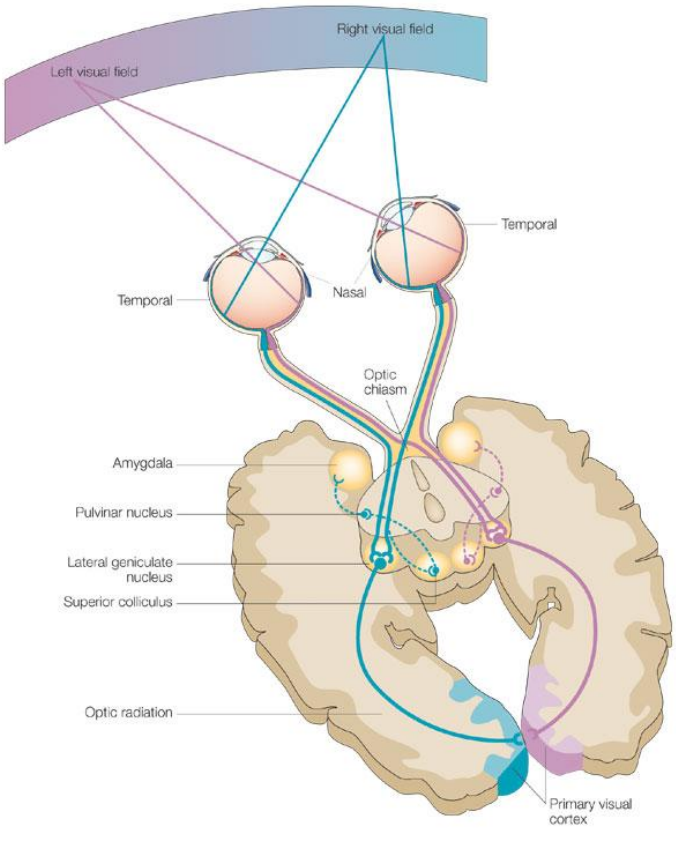
3. Evaluate the clarity of the lens (e.g. Cataract).

4. Evaluate the function and appearance of the macula (especially in elderly).

The visual pathway:







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- Light enters the eye via the refractive media, namely the cornea, anterior chamber, lens, and vitreous, and stimulates the retina posteriorly.
- Light stimulates the photoreceptors, ie., the rods and cones. Through a series of other retinal nerve cells, the end result is that the RGC is stimulated. The RGC sends its axon, or fiber, in the nerve fiber layer to the optic disc and then down the optic nerve.
- From the optic nerve, about half of the fibers cross over at the chiasm to the opposite optic tract, and the other half remain on the same side. The fibers in the optic tract synapse in the lateral geniculate nucleus of the thalamus. Neurons in the lateral geniculate nucleus then project to the occipital lobe, to the primary visual cortex. From there, there is further processing with projections to other cells in the visual cortex and elsewhere, resulting in conscious visual perception.
- Now that we know how visual information is normally transmitted to the brain, what happens with a disease like glaucoma.

Glaucoma:

- progressive **optic neuropathies**, that have in common characteristic morphological changes at the optic nerve head and retinal fiber layer in the absence of other ocular disease or congenital anomalies. Progressive retinal ganglion cell death and visual field loss are associated with these changes.
- A **major cause of blindness**.
- Damage is **irreversible**.
- Effective treatment is available.

Types of Glaucoma:

1. **Acute** Glaucoma (**angle closure** glaucoma).
2. **Chronic** Glaucoma (**open angle** glaucoma).
3. Congenital Glaucoma.

Chronic Glaucoma:

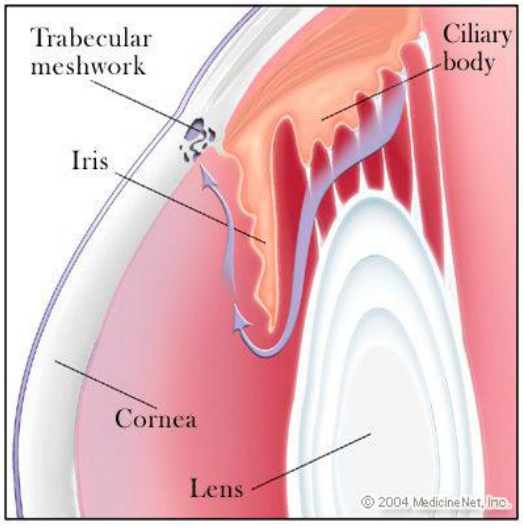
A. Primary open angle glaucoma (POAG):

Definition:

- POAG is a progressive, bilateral optic nerve damage with a typical pattern of nerve fiber bundle visual field loss, increased intraocular pressure (IOP > 21 mmHg*) that is not caused by another systemic or ocular disease, and open anterior chamber angle (AC angles).

Etiology & Epidemiology:

- Elevated IOP results from mechanical resistance to aqueous outflow that may be caused by an abnormality in the trabecular meshwork or collapse of Schlemm's canal.



***Normal IOP: 10 – 21 mmHg**

- **Angle structures:**

1. **Schlemm's canal.**
2. **Trabecular meshwork.**
3. **Sclera spure.**
4. **Iris root .**
5. **Ciliary body.**

- **Aqueous physiology: ciliary processes produce the aqueous humor that fills the posterior and anterior chambers.**
  The aqueous humor darins from the eye through trabecular meshwork, the aqueous filters through the meshwork into Sclemm's canal and then to the collector channels and veins.

- Primary open angle glaucoma (POAG) is the second leading cause of blindness in the USA and the single most important cause of blindness in African Americans.
- **Risk factors for POAG include:**
  1. Family history.
  2. Age, > 60 years old.
  3. Myopia.
  4. Black race.
  5. Diabetes.
  6. Hypertension.
  7. Cardiovascular diseases.

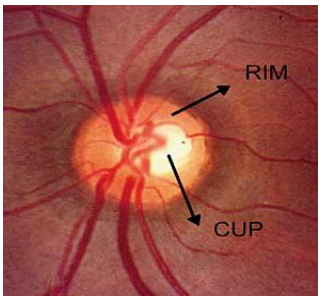
Symptoms:

- POAG is **asymptomatic.** Patients only notice decreased vision or constricted visual fields in the late stage of the disease.

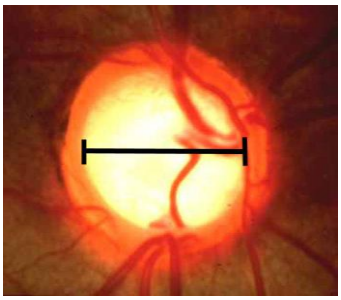
Signs:

- The hallmarks of POAG are:
  1. Elevated IOP.
  2. Optic nerve cupping (normal cupping= 0.3 and 2% had disc cup ratio more than 0.7).
  3. Characteristic visual field defects.
  4. Open AC angle.
  5. Decreased visual acuity in advanced disease.

it's a diagnosis of exclusion and that you need 2 out of 3 first signs.



Normal optic disc



Cupping

Evaluation:

- **History:**
  1. Systemic diseases.
  2. Previous eye injuries or surgeries.
  3. Family history of glaucoma.

- **Eye examination:**
  1. Central thickness of cornea.
  2. Tonometry (IOP & AC depth).
  3. Gonioscopy (open angle).
  4. Iris & lens.
  5. Ophthalmoscopy (optic nerve cupping, disc hemorrhage, nerve fiber layer appearance)
  6. Visual field.

- **Glaucoma affects the peripheral vision (no effect on macula).**
- **There are 2 direction of fibers of retina: horizontal & vertical line. In glaucoma the horizontal is damaged, particularly the superior and inferior temporal fiber which causing elongated disc damage.**
- **The vertical fiber are involved in neurological diseases.**

Management:

- The primary goal of POAG therapy is to halt or delay progression of the disease in order to prevent the patient from developing blindness.
- The effect of treatment is temporary.
- Therapy is administered in a stepwise fashion beginning with medicine, laser and finally surgery.
- The choice and timing f treatment depend on the degree of optic nerve damage, level of IOP control, progression of disease, patient age, and compliance.
- Options are as follow
  1. **Observation**
  2. **Medical:** ocular hypotensive agents: beta-blocker, prostaglandin analogues, alpha-adrenergic agonist, carbonic anhydrase inhibitors.
  3. **Laser:** trabeculoplasty to increase trabecular meshwork outflow, or to apply laser at the limbus to destroy ciliary processes and reduce aqueous production (cyclophotocoagulation).
  4. **Surgical:** The aim of the surgery is to lower the IOP by creating an alternate pathway for aqueous to escape from the eye. The most common procedure is trabeclectomy. Another option is glaucoma drainage implant.

B. Secondary open angle glaucoma:

- Refers to open angle glaucoma with elevated IOP caused by a variety of ocular or systemic disorder.

Subtypes:

- **Pseudoexfoliation glaucoma.**
- **Pigmentary glaucoma.**
- **Drug induced glaucoma:** is most commonly steroid-related, topical administration of steroid for 4-6 weeks may increase the IOP.
- **Uveitic glaucoma:** due to aqueous outflow obstruction or increased aqueous viscosity.
- **Lens-induced glaucoma:** retained lens material after surgery or trauma, or lens protein leaking from hypermature cataract and clogging the trabecular meshwork.
- **Traumatic glaucoma.**
- **Intraocular tumors.**

Symptoms:

- Depending on the underlying etiology and rapidity of IOP rise, patient are symptomatic or they may have pain, photophobia, decreased vision, redness or even systemic symptoms.

Signs:

- The signs of secondary open-angle glaucoma are the same as for POAG. Other specific finding depend upon the underlying etiology.

Evaluation & management:

- AS POAG.

C. Normal (low) Tension Glaucoma:

- Is a form of open angle glaucoma in which the IOP remains in the normal range (<22mmHg), but optic nerve damage still occurs.

Etiology:

- The etiology is unknown but theories regarding the mechanism include:
  1. Nocturnal systemic hypotension.
  2. Autoimmune.
  3. Vasospasm.
  4. Prior hemodynamic crisis.

Symptoms:

- Asymptomatic, patient only notice decreased vision or constricted visual field in the late stage of the disease.

Signs:

- Normal IOP.
- Optic nerve cupping.
- Visual field defect.
- Open AC angle.

- **In normal tension glaucoma, disc splinter hemorrhages are more common and visual field scotomas tend to be denser.**
- **Normal angle glaucoma is a diagnosis of exclusion.**

Evaluation:

- **History:** include information regarding autoimmune and vasospastic diseases, hypertension, and the use of Beta-blockers.
- **Examination:**

1. Central thickness of cornea.
2. Tonometry (IOP & AC depth).
3. Gonioscopy (open angle).
4. Iris & lens.
5. Ophthalmoscopy (optic nerve cupping, disc hemorrhage, nerve fiber layer appearance)
6. Visual field.
7. Color vision
8. Lab test: CBC, ESR, …
9. Neuroimaging.
10. Cardiovascular evaluation.

Management:

- As POAG.