O P H T H **A L M** O L O G Y T E A M



History Taking, Examination

OBJECTIVES:

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Color index: Important | Notes | Book | Extra

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The Visual Pathway & Importance of the Eye

- Visual Pathway:
 - Brief idea of the mechanism of vision: light → transparent cornea → anterior chamber → pupil → lens (convex-shaped) → clear vitreous → retina → stimulation of photoreceptors → horizontal, amacrine and bipolar cells (1st order neuron) → retinal ganglion cells (2nd order neuron) → optic nerve → decussation of fibers in the chiasm → optic tract → lateral geniculate body (3rd order neuron nucleus) → primary visual cortex (3rd order neuron axons) → secondary visual cortex.
 - In order to have the cornea properly clear, it has to have proper lubrication by the tears distributed by the eyelid.
 - The pupils control the amount of light entering the fundus by dilatation & constriction.
 - The function of the lens is to refract light.
 - The convex lens will converge the light to fall on the retina (specifically on the fovea).
 - 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 retinal ganglion cell (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.
 - Phototransduction: by photoreceptors (rods and cones).
 - Cons are responsible for color vision and central visual field & are present mainly in the macula.
 - Rods are responsible for night vision and peripheral visual field & are distributed on the periphery of the retina.
 - Image processing: by horizontal, bipolar, amacrine and RGCs.
 - Output to optic nerve: via RGCs and nerve fiber layer.
 - 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.







• Importance of eyes:

- **O** Diagnostically and functionally, it is the most important square inch of the body surface.
- O The eye is so intimately connected with the rest of the body (vascular and neurological connections) that it reveals enormous amount of general information.
- O Eye is the only part of the body where blood vessels and central nervous system tissues can be viewed directly.
- O 90% of our information reaches our brain via sight.
- O Unfortunately, of all the parts of the body, the eye is the most vulnerable to minor injury.

• Neurological connections:

- The 12 cranial nerves provide us with a large part of our information about the brain. Of these, the eye examination evaluates CN II, III, IV, V, VI, VII, VIII (7 CNs).
 - CN 3 (oculomotor nerve) from its name is responsible for the movement of the eye.
 - CN 4 (trochlear nerve) innervates superior oblique muscle (SO4).
 - CN 5 (trigeminal nerve) the ophthalmic branch is responsible for the sensation of the globe, eyelids & forehead.
 - CN 6 (abducent) innervates lateral rectus muscle.
 - CN 7 (facial nerve) is responsible for closure of the eyelid; in facial nerve palsy, patient is unable to close the eye.
 - CN 8 (vestibulocochlear nerve); the vestibular part is responsible for controlling the eye movement.
- In addition, it provides information about the autonomic pathways (sympathetic/parasympathetic) by looking for pupillary reaction: constriction (para-sympathetic) and dilation (sympathetic).
- The retina and optic nerve are physical extensions of the brain.
- The best-known connection between the brain and the eye is the optic nerve.
- The visual pathway, which extends from front to back across the brain can be studied easily and safely using perimeter. It can differentiate accurately between lesions of the temporal, parietal, and occipital lobes.
- In addition, the optic nerve has important clinical relationships to the pituitary gland (the optic nerve decussation at the chiasm is on top of the pituitary stalk, so any pathology in the pituitary will affect the visual field), the middle ventricles, the venous sinuses, the meningeal and bony structures of base of the skull (patients with meningitis, venous sinus congestion or skull base fractures can manifest with







- The normal optic nerve head has distinct margins, a pinkish rim and, usually, a central, pale, cup.
- The central retinal artery and vein enter the globe slightly nasally in the optic nerve head, referred to ophthalmoscopically as the optic disc.
- In optic disc edema there is unclear disc margin.

| Optic disc edema (papilledema) | Optic atrophy | |
|--|--|--|
| The optic nerve has the diagnostically useful capability of swelling (congested veins + disc) with ↑ ICP (papilledema). Papilledema is the term given to bilateral disc swelling associated with raised intracranial pressure (ICP), accelerated hypertension and optic disc ischemia. Visual loss is uncommon with the papilledema of hypertension and/or raised intracranial pressure. | The optic nerve could be visibly pale (optic atrophy) (white yellowish) normally it's (orangey red) when its nerve fibers are damaged at any point from retina to LGB. Any pathology in the visual pathway behind the LGB will not manifest in the optic nerve; for example, if someone has primary cortex infarction, it will not cause optic nerve atrophy. the patient will be blind but optic nerve will be normal. A pale optic disc represents a loss of nerve fibers at the optic nerve head. The vision is usually reduced, and color vision affected. On examination, the usual vascularity of the disc is lost. Comparison of the two eyes is of great help in unilateral cases, as the contrast makes pallor much easier to see. A relative afferent pupillary defect will usually be present (RAPD+). | |
| You have to differentiate between the two pictures! | | |
| | | |

- The study of CN III, IV, V, VI can evaluate the brainstem, cavernous sinus & apex of orbit (these areas are in front of each other & any pathology in each of these can affect the cranial nerves that innervate the eye, so pt presents w/ eye manifestation).
- The nerves that go through the cavernous sinus are oculomotor nerve, trochlear nerve, 2 divisions of trigeminal nerve (ophthalmic & maxillary branches) & abducent nerve as well as sympathetic fibers around the internal carotid artery innervating the eye & forehead.



- If someone has cavernous sinus thrombosis or cavernous carotid fistula, pt will have multiple cranial neuropathies & restricted eye movements.

Cavernous Sinus Components Mnemonic: O TOM CAT



- A unilateral dilated pupil after a head injury can occur due to pressure on pupil constrictor fibers of CN III.
 - Brain Herniation, hemorrhage, aneurysm of posterior communicating artery can compress CN III.

- What investigations would you do if you have a patient with 3rd nerve palsy with dilated pupil? order a CT **ANGIO** or MR**A (angio)**.

- How do we know if CN IV is involved in addition to CN III? patient will not be able to **look down** (damaged CN III) and eyes won't intort (CN IV damaged).

| Fibers | Innervation | Function |
|---|---|--|
| Somatic motor (general somatic efferent) | Supplies four of the six extraocular muscles of the eye <u>and</u> the levator palpebrae superioris muscle of the upper eyelid. | controlling the muscles responsible for the precise movement of the eyes for visual tracking or fixation on an object. |
| Visceral motor (general visceral efferent) | Parasympathetic innervation of the constrictor pupillae and ciliary muscles. | Involved in the pupillary light and accommodation reflexes. |

- CN VI is involved in mastoid infection (petrous ridge). Patients with inner ear infection or abscess
 collection may present with ear pain and 6th nerve palsy.
 - Commonest cause of abducens nerve palsy? TRAUMA. Why? because it has a very long course and each course is perpendicular to each other.
- CN VII is involved in parotid gland & inner ear disease. Some patients with parotid tumor or infection can present with facial palsy.
- CN VIII is involved in nystagmus.
- Focal brain lesions like vascular occlusions, hemorrhage, or neoplasm.
- **Diffuse brain lesions like** infections, demyelinating disorders \rightarrow nerve damage.

Third cranial nerve, oculomotor nerve:

- O Supplies all the muscles of the eye EXCEPT lateral rectus and superior oblique.
- O Paralysis will lead to ptosis and squint (eye deviated out and little down) & pupil might be affected.
- O It has two fibers:
 - Inside the nerve: deep fibers responsible for the muscles.
 - Surface of the nerve (periphery): superficial parasympathetic fibers.
- O Muscles only paralysis is always due to <u>medical</u> reasons. Example: If CN III is affected due to ischemia because of DM or HTN, the fibers supplying the muscles will be affected.

6

- O If there is a tumor compressing the periphery it will affect both peripheral and central part of the nerve. There will be **muscles paralysis & pupil dilation** (loss of parasympathetic activity \rightarrow unopposed sympathetic). It is an emergency situation. (surgical)
- O The difference between medical & surgical third nerve palsy is pupil affection.

SAQ

Question: What are the structures in the cavernous sinus? 1- In the wall: THREE nerves • Superior & inferior divisions of CN III (Oculomotor nerve) • CN IV (Trochlear nerve) • Ophthalmic and maxillary divisions of CN V (Trigeminal nerve) 2- Inside the cavernous sinus: ONE artery + ONE nerve

o Carotid artery.

CN VI (Abducens nerve)

"هذى أهميتها لما يجيلك سؤال عن cavernous sinus thrombosis

Vascular connections:

O Venous flow disorder:

- O Cavernous sinus thrombosis (occlusion).
 - Cavernous sinus is the venous drainage of the eye. The eye will be bulging, injected & congested sometimes with paralysis because the nerves are in the cavernous sinus.
- O Carotid cavernous fistula (orbital congestion).
 - The eye will be bulging, injected, congested, same as cavernous sinus thrombosis. The <u>difference</u> is that the eye is pulsating (bruit).
 - A fistula may develop between the internal carotid artery, in the cavernous sinus, and the cavernous sinus itself. As a result, the orbital veins are exposed to a high intravascular pressure.
 - Signs & symptoms:
 - The eye is proptosed and the conjunctival veins are dilated and engorged.
 - A bruit may be heard with a stethoscope over the eye, in time with the radial pulse.
 - Eye movements are reduced because of extra-ocular muscle engorgement.
 - **Increased IOP** secondary to increased pressure in the veins draining the eye.
 - Management:
 - The fistula can be closed by embolizing and thrombosing the affected vascular segment.
 - In cavernous sinus thrombosis and carotid cavernous fistula patients usually present with unilateral proptosis, unilateral redness, swelling that can be severe to the point that the eye is closed & orbital congestion.
 - Arterial emboli can reach the retina from carotid artery, heart valves, subacute endocarditis and traumatic bone fracture.
- O HTN.
- O Systemic vasculitis:
 - Polyarteritis nodosa (PAN), SLE.
 - Both PAN & SLE can cause scleritis (scleritis is covered in detail in red eye lecture).







spot

- O **Temporal arteritis**, also known as **giant cell arteritis (GCA).** "very important and you have to know it in detail.
 - This is an autoimmune vasculitis occurring in patients generally over the age of 60.
 - It affects the ophthalmic artery (not retinal).
 - Whitening of the retinal vessels and hemorrhage.
 - Signs & symptoms:
 - Visual symptoms: irreversible and usually sudden loss of vision, diplopia.
 - Polymyalgia rheumatica symptoms: headache, pain in the shoulders and hips, malaise.
 - Others: jaw claudication (pain on chewing) & scalp/temporal tenderness (eg. on combing).
 - Fever and constitutional symptoms.
 - Diagnosis:
 - Elevated erythrocyte sedimentation rate (ESR) & C-reactive protein (CRP) (e.g. ESR = 100 mm/h).
 - **Temporal artery biopsy**. If you miss the diagnosis the patient will have loss of vision in the other eye.
 - **Management**: IV Steroids to protect the other eye. **Steroids will not reverse the visual loss** but can prevent the fellow eye being affected.

Extra from 435 team

- Carotid-cavernous fistula (orbital congestion) has the same clinical presentation (proptotic, retinal edema) of cavernous sinus except that it has increased IOP, and thrill + pulsating **eves (Bruit)** orbital congestion.

- Came in the exam: central retinal artery occlusion/ history: of multiple bone fractures, what is the diagnosis? Retinal central artery fat embolism.

- Case: a 60-year-old pt w/ heart disease and using penicillin injections (meaning that he has bacterial endocarditis and might have a possibility of embolism manifestations leading to central retinal artery occlusion)

- Best initial investigation in temporal arteritis is ESR, followed by C-reactive protein, then Biopsy (a negative biopsy does not rule out the dx due to the focal and segmental nature of the infiltrates).

- When a patient complains of any changes in vision, rule out DM first!

• Hematological disorders:

O All types can manifest in the fundus. For example, in sickle cell retinopathy the sickle cells will cause vascular occlusion \rightarrow ischemia in the retina and inter retinal hemorrhage.

O Sickle cell retinopathy:

- Signs:
 - Tortuous veins.
 - Peripheral hemorrhages.
 - Capillary non-perfusion.
 - Pigmented spots on the retina.
 - New vessel formation, classically in a 'sea fan' pattern, which may occur as a result of peripheral retinal artery occlusion which may cause vitreous hemorrhage and traction retinal detachment.

This may require treatment with laser photocoagulation and vitrectomy.

• Metabolic disorders:

- O Almost all metabolic disorders can affect the eye:
- Diabetes Mellitus: diabetic retinopathy (proliferative & non-proliferative), cataract, refractive error caused by hyperglycemia or ophthalmoplegia due to diabetic vasculopathy (it can cause cranial nerve palsies such as 3rd ,4th or 6th cranial nerve palsy).
 - Diabetic retinopathy is the most common of irreversible blindness in people below 50 years of age.
- Wilson's disease: copper accumulation in the body, deficiency of α-ceruloplasmin.
 - Copper accumulation in the cornea will cause Kayser-Fleischer ring, corneal ring: a green ring around the cornea in the periphery).
- **Hypo**-parathyroidism \rightarrow cataract.
- Thyroid eye diseases:
 - Hyperthyroidism: Graves' disease.
 - The patient may sometimes complain of:
 - A red painful eye (associated with globe exposure caused by proptosis).
 - If the redness is limited to part of the eye only, it may indicate active inflammation in the adjacent muscle.
 - Double vision.
 - Reduced visual acuity (sometimes associated with optic neuropathy).
 - On examination:
 - Bilateral exophthalmos (also known as proptosis which is protrusion of the eye from the orbit).
 - Lid retraction (upper part of sclera is seen) and lid lag.
 - The conjunctiva may be chemosed (oedematous swelling of the conjunctiva) and the eye injected over the muscle insertions.
 - There may be **restricted eye movements** or squint (also termed restrictive thyroid myopathy, exophthalmic ophthalmoplegia).
 - The inferior rectus is the most commonly affected; Its movement becomes restricted and there is mechanical limitation of the eye in upgaze.
 - Involvement of the medial rectus causes mechanical limitation of abduction, thereby mimicking a sixth nerve palsy.
 - In severe cases, there will be so much pressure in the orbit that leads to optic neuropathy.
 - Compressive optic neuropathy due to compression and ischemia of the optic nerve by the thickened muscles. This leads to field loss and may cause blindness.
 - The commonest cause of unilateral & bilateral proptosis is thyrotoxicosis.
 - Investigations:
 - A CT or MRI scan shows enlargement of the rectus muscle.
 - Thyroid function test
 - Treatment:



- Corneal exposure and optic nerve compression require urgent treatment with systemic steroids, radiotherapy or surgical orbital decompression.

• Infections:

O Syphilis, toxoplasmosis & rubella.

- Syphilis and Rubella also can cause retinitis (inflammation of the retina).
- Congenital syphilis causes interstitial keratitis which is any vascular keratitis that affects the corneal stroma without epithelial involvement.

O Toxoplasmosis

- The retina is the principal structure involved, with secondary inflammation occurring in the choroid (retinochoroiditis).
- An active lesion is often located at the posterior pole, appearing as a creamy focus of inflammatory cells.
- Inflammatory cells cause a vitreous haze, and the anterior chamber may also show evidence of inflammation.
- Thus, the patient may complain of hazy vision and floaters, and the eye may be red and painful.
- The clinical appearance is usually diagnostic.





• Mucocutaneous disorders:

- O Steven-Johnson Syndrome (SJS), pemphigus.
 - Ulceration in the mucous membranes in reaction to chemicals like drugs and medications or infection.
 - Eyelid and conjunctival ulceration.
 - Scar tissue formation between eye and eyelid.
- Loss of goblet cells occurs in most forms of dry eye, but particularly in cicatricial conjunctival disorders such as erythema multiforme (Stevens–Johnson syndrome). In this there is an acute episode of inflammation causing macular 'target' lesions on the skin and discharging lesions on the eye, mouth and vulva. In the eye this causes conjunctival shrinkage with adhesions forming between the globe and the conjunctiva (symblepharon).





10

- Elastic tissue disorders:
 - Pseudoxanthoma elasticum: degeneration of the retina in which the patient can develop neovascularization.
 - Sub-retinal neovascular membranes may also grow through elongated cracks in Bruch's membrane called angioid streaks.
- Allergy:
 - Vernal keratoconjunctivitis (VKC)
 - Also called (spring catarrah) causes cobblestone papillae. حساسية الرمد الربيعي Also called (spring catarrah) causes cobblestone papillae. د الربيعي area dusty a is it وجيزان، " فتجيلهم area dusty a is it و allergy have children the of most مشهورة في نجران وجيزان، " من steroids، للأسف معظمهم بيروحوا يشتروا spring catarrh vernal أو catarrh vernal هذا الموضوع مهم جدا ولازم تذاكروا في الـ glaucoma. و الـ steroids الـ steroids الماها و injections و areations الماها الموضوع مهم جدا ولازم تذاكروا في الـ steroids. و injections و inhaler "even.
 - Causes chronic allergic reaction in the eye: chronic mucous discharge, chronic ulceration of the conjunctiva and chronic rubbing of the eye leading to corneal pathology and astigmatism.
 - Ig-E mediated; it often affects boys with a history of atopy.
 - It is usually seasonal but may be present all year long & may become chronic.
 - Signs & symptoms:
 - Itchiness.
 - Photophobia.
 - Lacrimation.
 - Papillary conjunctivitis on the upper tarsal plate (papillae may coalesce to form giant cobblestones).
 - Limbal follicles and white spots.
 - Punctate lesions on the corneal epithelium.

Chromosomal abnormalities:

- Trisomy: 13, 15, 18 & 21.
 - Basically, patient can have eye defects or cataract develop in their eyes.

• Eye poisoning:

- The eye is a delicate indicator of poisoning:
 - Morphine addict (opiate intoxication): leads to miotic pupil.

Constricted pinpoint pupil = morphine overdose or pontine hemorrhage.

Dilated pupil= cocaine or amphetamine.

They have the same presentation the only difference is **hyperpyrexia with pontine hemorrhage**.

- Lead poisoning, vitamin A intoxication (example for acne treatment) lead to papilledema.
 - After lead poisoning is treated or after taking methanol, patient can manifest with optic atrophy.

