Neuro ophthalmology

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Fifth Grade

optic nerve

3rd cranial nerve

4th cranial nerve

6th cranial nerve

The optic nerve

Is composed of 1.2 million afferent nerve fibers originating from retinal ganglion cells. Most of these fibers synapse in the lateral geniculate body the surrounding layers 1-innermost layer is the delicate vascular pia mater 2-the outer sheath composed of the arachnoid mater and the tough dura mater which is continuous with the sclera

Nearly 1/3 of the nerve fibers sub serve the central 5 degrees of the visual field.

Anatomical sub-divisions

-1.intraocular part =optic nerve head, 1 mm

2-intra-orbital portion =25mm, start to have myelination.

3-intra-canalicular =6 mm, fixed to the optic canal

-4.intra-cranial part, variable length 5-15, joins the chiasm

The optic nerve head

It is the intraocular portion of the optic nerve, usually we call it: the optic disc on examination of the disc we should comment on the 3 Cs (C C C) 1-cup 2-color 3-contour

reduced visual acuity 2-Abnormal pupillary reflex (RAPD)=AFFERENT DEFECT 3-dyschromatopsia: impaired color vision mainly red and green (simply compare the two eyes) 4-reduction in other aspects of visual function: light brightness sensitivity, Contrast sensitivity)

5-visual field defect 6- Abnormal visual evoked potential :VEP: latency (delay) & amplitude

OPTIC NEURITIS

Demyelinating optic neuritis is the most common etiology Age: around 30 (20-50) More in females' Clinical presentation: 1-subacute monocular visual loss associated with pain, esp. with eye movement, also tenderness of the globe.

2-Abnormal pupillary reflex 3-impaired color vision 4-VF abnormality ex. central scotoma or generalized depression Examination: the majority of cases shows normal optic nerve!! WHY?? BECAUSE: most of the cases involves the retrobulbar portion of the optic nerve.

INVESTIGATIONS

1-All the cases should be sent for brain MRI to check for the presence of periventricular plaques (white matter lesions) for MS (systemic demyelinating disease) 2-LP with CSF protein electrophoresis (to show oligo-clonal bands Optic Nerve Diseases According to A etiology

-Inflammatory (optic neuritis) : A-demyelinating, B-infectious and Para infectious, C-immunological. 2-Ischemic optic neuropathy (ant. or post.) 3-Heriditary optic atrophy 4-Toxic (tobacco-alcohol, drugs))

-Glaucomatous (cupping) 6-Papilloedematous(ICP) 7-traumatic 8-Compressive (tumor-aneurysm) 9-Infiltrative : ex. tumors

signs and symptoms of optic nerve diseases

-1-reduced visual acuity 2-Abnormal pupillary reflex (RAPD)=AFFERENT DEFECT 3-dyschromatopsia: impaired color vision mainly red and green (simply compare the two eyes) 4-reduction in other aspects of visual function : light brightness sensitivity, Contrast sensitivity.

Ishihara plates for color testing

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Optic neuritis

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1-All the cases should be sent for brain MRI to check for the presence of periventricular plaques (white matter lesions) for MS (systemic demyelinating disease) 2-LP with CSF protein electrophoresis (to show oligo-clonal bands)

3-VEP: prolonged latency (conduction delay) more than the reduction amplitude in 100% .

TREATMENT

1-intra-venous methyl -prednisolone sodium succinate 1 gm daily for 3 days followed by oral prednisolone 1mg / kg body wt. for 11 days, then tapered for 3 days the benefit of steroid is to speed up recovery by 2-3 weeks and delay possible future MS on short term 2-MT: immuno-modulatory treatment ex. Interferon beta

Papilledema

It is bilateral swelling of the optic nerve heads secondary to raised intracranial pressure. all other causes of disc edema in the absence of raised intracranial pressure are referred to as disc swelling

All patients with papilledema should be suspected to have an intracranial mass until proved otherwise. So neurological consultation and neuro-imaging is mandatory (MRI & MRA) If no SOL is seen, then the next step is to exclude Idiopathic Intra-cranial hypertension by doing LP & measuring the opening pressure of the CSF.

Clinical features of raised ICP: 1-early morning headache &nausea with or without vomiting. 2- horizontal diplopia caused by stretching of the 6th cranial nerve. (a false localizing sign) 3-CNS symptoms

Treatment

It is directed to the cause: whether a brain tumor or an intracranial aneurysm or an idiopathic intracranial hypertension, where the treatment is done by the neurologist & fellow up by the ophthalmologist.

Optic atrophy

It is sign of advanced optic nerve disease, which may involve any part of the optic nerve, optic chiasm, optic tract up to the lateral geniculate body.

Primary OA atrophy is not preceded by a previous swelling of the ON Signs: white flat disc with clearly delineated margins. examples of causes of primary OA 1-Compression (tumors, aneurysms) 2-hereditary optic neuropathies 3-toxic & nutritional neuropathies

secondary OA preceded by long standing swelling of the optic nerve head Signs: white, slightly raised disc with poorly delineated margins due to gliosis. Examples of secondary causes of optic atrophy: 1-ischemic optic neuropathy 2-papillitis

The 3rd CN: oculomotor nerve

The nucleus is located in the mid brain The nerve is alone along most of its course except when it enters the lateral wall of the cavernous sinus , where it is accompanied by the 4th , the 5th and the 6th CNs It supplies all the EOM except LR & SO It also carries the parasympathetic fibers that supplies the pupillary sph. & ciliary mm.

parasympathetic fibers are located superficially in the nerve and this is important in differentiating surgical from medical causes of third nerve palsy.

Causes of 3rd CN palsy

1-microvascular e.g. D.M, HPT (pathology =vasa nervorum) 2-aneurysm 3-trauma (direct or indirect) 4- tumors= uncommon 5-miscellaneous: idiopathic, inflammatory & autoimmune diseases, migraine ….

Trauma: subdural hematoma with uncal herniation

Clinical features

1- ptosis due to weakness of the lavator palpebrae superioris 2-divergant squint 3-limitation of ocular movement except lateral gaze 4-parasympathetic palsy leading to dilated pupil and defective accommodation

Management

1-in pupil involvement: the cause is usually surgical , so we have to do neuroimaging 2-in pupil sparing palsy: if the patient is old, hypertensive, diabetic,… then observation and Mx of risk factors. If there is no improvement after 6-12 weeks, then we should send for neuroimaging. BUT for a young patient or without vascular risk factors, then neuroimaging is indicated even for pupillary sparing 3rd CN palsy. 3-Sx

Fourth CN Palsy (Trochlear)

t is a crossed nerve; this means that the 4th nerve nucleus innervates the contralateral superior oblique muscle. It is a very long and slender N. Ipsilateral hypertropia Vulnerable to trauma Clinical picture (acute onset of vertical diplopia in the absence of ptosis combined with characteristic head posture).

CF of Left 4th nerve palsy include: 1-left hypertropia in the primary position 4-abnormal head posture (head tilt to the right; face turn to the right; chin depression)

Causes

1-congenital lesions are common although symptoms may not develop until decompensation occurs in adult life. 2-trauma: frequently causes bilateral 4th nerve palsy 3-microvascular are common 4- aneurysms and tumors are extremely rare. Routine neuroimaging for isolated 4th nerve palsy is not required. Management - for congenital or decompensated congenital surgery for inferior oblique mm (THE ANTAGONIST) - microvascular & or sometimes traumatic cases may recover spontaneously

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Abducent nerve palsy

The abducent nerve nucleus lies in the pons. It travels in the cranial cavity alone until it enters the cavernous sinus. increased intracranial pressure can cause stretching of the nerve and giving signs of nerve palsy (false localizing sign)

Causes: 1-microvascular 2-tumours 3- trauma 4-raised ICP 5-miscelaneous

Signs: 1- horizontal diplopia 2-convergent squint 3-limited lateral eye movement 4-ipsilateral face turn

Management of 6th CN palsy

if the cause is microvascular then the palsy usually improves after 6-12 weeks and if not, then neuroimaging should be done. -Young patients and children with palsy may need neuroimaging because of the high risk of intracranial neoplasms -surgical correction: for permanent palsy after treating the cause..