

Neuro-Ophthalmology

The visual pathway from the retina may be divided into seven levels:

- 1- Optic nerve: is a tract of the brain surrounded by meninges. It is formed by the axons of the ganglion cells of the retina. Its total length is 5 cm.
- 2- Optic chiasma: in which there is decussation of the fibers coming from the nasal retinae. Below which lies the pituitary gland.
- 3- Optic tract: traveling posterolaterally from the angle of the chiasma to reach the lateral geniculate body and then to the visual cortex.
- 4- Lateral geniculate body: provides a relay station for retinal axons synapsing with neurons of the optic radiation.
- 5- Optic radiation: arises in the lateral geniculate body and carries visual impulses to the occipital cortex.
- 6- The striate cortex: lies on the medial aspect of the occipital lobe.
- 7- The prestriate cortex: the area surrounding the striate cortex. It is concerned with visual memory. Lesion of this area causes visual agnosia (inability to recognize things by sight) and inability to judge distances.

Optic atrophy: may be primary or secondary

- 1- primary optic atrophy ;occurs without antecedent swelling of the optic nerve head e.g. following retrobulbar neuritis, compressive lesions such as tumors or aneurysms, hereditary optic neuropathies, and toxic and nutritional optic neuropathies.

Signs

Pale, flat disc with clearly delineated margins, with reduction in the number of small blood vessels on the disc surface.

2-secondary optic atrophy: is preceded by swelling of the optic disc e.g. following papilloedema. Anterior ischaemic optic neuropathy and papillitis.

Signs:

White or dirty grey, slightly elevated disc with poorly delineated margins and reduction in the number of small blood vessels on the surface of the optic disc surface.

Optic neuritis

Is an inflammatory, infective or demyelinating process affecting the optic nerve.

Ophthalmoscopic classification:

1-retrobulbar neuritis in which the optic disc appearance is normal.it is the most frequent type in adults and is frequently associated with multiple sclerosis.

2- papillitis: characterized by hyperaemia and oedema of the optic disc,which may be associated with parapapillary flame shaped haemorrhages. It is the most frequent type of optic neuritis in children.

3-neuroretinitis: characterized by papillitis in association with inflammation of the retinal nerve fiber layer. It is the least common type of optic neuritis and most frequently associated with viral infections. It is never a manifestation of demyelination.

Aetiological classification:

1- demyelination, the most common.

2- Parainfectious, may follow viral infection or immunization.

3- Infectious, may be sinus-related or associated with cat-scratch fever, syphilis,lyme disease and herpes zoster.

4- Autoimmune associated with autoimmune diseases.

Demyelination: is a pathological process by which myelinated nerve fibers lose their myeline sheath. The myelin is phagocytosed by microglia and macrophages and subsequent to which astrocytes lay down fibrous tissue. Demyelination will disrupt nervous conduction within the white matter tracts within the brain, the brain stem, and the spinal cord.

Demyelinating optic neuritis:

-presentation: sub acute monocular visual impairment, discomfort in or around the eye exacerbated by eye movements.

-signs: *visual acuity between 6/18 and 6/60 rarely no light perception.

*the optic disc is normal in the majority of cases.

*dyschromatopsia(impaired colour vision) is universal and worse than would be expected for that level of visual impairment.

-visual field defects.

-MRI: shows periventricular plaques of demyelination.

-course: recovery typically begins within 2-3 weeks and continues over a period of 6 months.

-prognosis: 75% of patients recover visual acuity of 6/9 or better.

-treatment: indicated when VA within the first week is worse than 6/12 specially when the disease is bilateral or when the patient has poor vision in the fellow eye.

The drug used is methylprednisolone sodium succinate 1 gram daily for 3 days followed by oral prednisolone 1 mg/k.g. daily for 11 days .

Oral prednisolone alone is contraindicated because they double the recurrence rate.
Intramuscular interferon beta-1a is beneficial in reducing the development of demyelination.

Non-arteritic anterior ischaemic optic neuropathy:

Pathogenesis: partial or total infarction of the optic nerve head due to occlusion of the short posterior ciliary arteries.

Predisposing factors: hypertension, diabetes mellitus, hypercholesterolaemia, collagen vascular diseases, antiphospholipid antibody syndrome, sudden hypotensive events and cataract surgery.

Clinical features:

Age of presentation is between 45-65 years with sudden painless monocular visual loss on awakening suggesting that nocturnal hypotension may play an important role.

Signs: * V.A. is slightly reduced.

Visual field defect is typically inferior.

dyschromatopsia.

optic disc is pale and edematous.

Fluorescein angiography: in late stages shows unequal choroidal filling. Special investigations: serological tests, fasting lipid profile and blood glucose.

Prognosis: no definitive treatment. Underlying conditions should be treated. In most patients there is no further loss of vision and the disease never recurs in the same eye.

Arteritic ant. Ischaemic optic neuropathy:

It is caused by giant cell arteritis. It typically affects patients older than 65 years and has a predilection for medium sizes and large arteries, particularly the superficial temporal, and the posterior ciliary arteries.

The four most important diagnostic criteria of giant cell arteritis are;

1 -jaw claudication. 2-neck pain.

3-c-reactive protein > 2.45 mg/dl 4-ESR >47 mm/hour.

Clinical features: presentation is with sudden, profound unilateral visual loss accompanied by periocular pain. Most cases occur within few weeks of the onset of giant cell arteritis.

Signs: *pale and a swollen optic disc.

* over 1-2 months severe optic atrophy occurs.

Fluorescein angiography: shows severe hypoperfusion of the choroid. Prognosis: very poor.

Treatment is to prevent blindness of the fellow eye. Intravenous methylprednisolone 1gm/day daily for 3 days together with oral prednisolone 80 mg daily then gradually tapered. Depending on the symptoms, the level of the C-reactive protein and ESR.

Papilloedema: is a swelling of the optic nerve head, secondary to raised intracranial pressure.

Clinical features: in early stages there is hyperaemia and mild elevation with indistinct margins of the optic disc but VA is normal in late stages there is marked elevation with visual loss and even optic atrophy with severe loss of vision may occur.

Third nerve palsy:

Signs: *ptosis due to weakness of the levator palpebrae superioris.

*abduction of the eye in the primary position due to the unopposed action of the lateral rectus.

* dilated pupil due to parasympathetic palsy with defective accommodation.

Causes of isolated third nerve palsy: 1-idiopathic.

2-vascular disease e.g. hypertension and D.M.. 3-trauma.

4-aneurysm of the posterior communicating artery. 5-miscellaneous causes: tumours, syphilis and vasculitis.

Treatment:

1- non-surgical by using prisms, uniocular occlusion to avoid diplopia if ptosis is partial or recovering. And botulinum toxin injection into the uninvolved lateral rectus to prevent its contracture.

2- surgical treatment: considered after 6 months.