

Neurophthalmology

Optic neuropathies

Clinical features of optic nerve disease

The optic nerve is the exit site for all retinal nerve fibers. The papillomacular bundle contains the small calibre nerve fibers which subserve the cone system of the fovea

.Diminished visual acuity

Visual field defects which can be of various types depending on the underlying lesion

(Diminished pupillary light reactions (Afferent pupillary conduction defect

Impairment of color vision

Diminished light brightness sensitivity

Diminished contrast sensitivity

Classification of optic neuropathies

Optic neuritis

is an acute or subacute inflammatory or demyelinating disorder of the optic nerve

Ophthalmoscopic classification

1.Retrobulbar neuritis is characterized by a normal optic nerve head and retinal nerve fibre layer. This is the most common type in adults and is frequently associated with demyelination MS

2.Papillitis is characterized by disc swelling, obliteration of the physiological cup and post.vitreous cells may be seen. In some cases there may also be haemorrhages on and around the optic disc. It is the most common type in children, although it can also occur in .adults

3.Neuroretinitis characterized by optic papillitis and a macular star figure composed of .hard exudates. It is the least common and is rarely associated with demyelination

.most frequently associated with viral inf. & cat-scratch fever

sometimes syphilis & lyme disease

Aetiological classification

Demyelinating ,is the most common

. Parainfectious ,may follow viral inf or immunization

Infectious, may be sinus related or associated with cat-scratch fever, syphilis, Lyme disease and cryptococcal meningitis in patients with AIDS

OPTIC NEURITIS

Clinical features

Presentation is with an acute onset of monocular visual loss which is frequently associated with periocular discomfort made worse on moving the eye. There may also be frontal headache and tenderness of the globe

Ophthalmoscopy is normal in most cases, although a few show papillitis. There is also diminished visual acuity and other features of optic nerve dysfunction. The impairment of colour vision is typically worse than would be expected at that level of visual acuity

Visual acuity impairment becomes maximal after 1-2 weeks and is usually between 6/18 and 6/60, although, rarely, it may fall to no light perception. Recovery takes 4-6 weeks, although it may be slower in some patients

Prognosis is excellent in approximately 75% of patients with recovery of visual acuity to 6/9 or better, even if visual acuity was reduced to no light perception during the attack. However, despite return of visual acuity other parameters of visual function (e.g. colour vision, contrast sensitivity, light brightness appreciation) often remain abnormal. A mild afferent pupillary conduction defect may persist and mild optic atrophy ensue

Treatment

When the presenting visual loss is mild, treatment is probably not beneficial. However, when visual acuity within the first week of symptom onset is worse than 6/12, treatment may speed up recovery by about 2 weeks

The therapeutic regimen consists of intravenous methylprednisolone sodium succinate (250 mg 6-hourly) for 3 days followed by oral prednisone (1 mg/kg daily) for 11 days. Oral prednisone alone is of no benefit

The long-term benefits of treatment on the final visual acuity are uncertain

Other types of optic neuropathies

2-Ischaemic optic neuropathy is caused by microvascular occlusion of the prelaminar or laminar portion of the optic nerve head. The main types are arteritic, non-arteritic and autoimmune

3-Hereditary optic neuropathy may be autosomal dominant, recessive, X-linked recessive or inherited mitochondrially

4- Toxic optic neuropathy may be nutritional or drug induced.

5- Granulomatous optic neuropathy can occur in sarcoidosis

Arteritic anterior ischaemic optic neuropathy

AION is a segmental or generalized infarction within the prelaminar or laminar portion of the optic nerve, caused by occlusion of the short posterior ciliary arteries. It may be associated with giant cell arteritis (GCA) and a wide variety of vascular diseases

CLINICAL FEATURES OF ARTERITIC AION

Presentation is typically with unocular, sudden and profound loss of vision which may be accompanied by periocular pain and preceded by transient visual obscurations and flashing lights. AION usually occurs within the first few weeks of the onset of GCA and is extremely rare after 9 months have elapsed - hence the need to start steroid treatment as soon as possible. Although simultaneous bilateral involvement is rare, about 65% of untreated patients become blind in both eyes within a few weeks

Ophthalmoscopy during the acute stage shows a pale and swollen optic nerve head which may be surrounded by small splinter shaped haemorrhages. Within 1-2 months, the swelling gradually resolves and the entire optic disc becomes atrophic

(Visual acuity is profoundly impaired (HM-noPL

Prognosis is very poor because visual loss is usually permanent

TREATMENT OF ARTERITIC AION

The main treatment is to prevent blindness of the fellow eye

Immediate treatment is with intravenous hydrocortisone 250 mg together with oral prednisone 80 mg daily

Subsequent treatment is as follows

.After 3 days the dose is reduced to 60 mg for 3 days and then 40 mg for 4 days

The daily dose is then reduced by 5 mg weekly until 10 mg is reached

.Maintenance daily therapy is 10 mg for 12 months

Papilloedema

is defined as swelling of the optic nerve head secondary to raised intracranial pressure .it is
.nearly always bilateral

All other causes of disc oedema not associated with raised ICP are referred to as (disc swelling)

.Early papilloedema may be difficult to diagnose with certainty

.Visual symptoms are absent and visual acuity normal

Established papilloedema

Transient visual obscurations in one or both eyes, lasting few seconds, often on standing,
may be present

Visual acuity is normal or reduced

Optic discs showing hyperaemia ,, elevation of the surface, partial obscuration of the small
traversing blood vessels and obliteration of the cup

The disc margin is indistinct and may be surrounded by peripapillary flame shaped
haemorrhages , venous engorgement, cotton-wool spots

Longstanding (vintage) papilloedema

Visual acuity is variable with constricted visual fields

Optic discs showing marked elevation with a champagne cork-like appearance with no
exudates, cotton-wool spots or haemorrhages

Atrophic papilloedema

Visual acuity is severely impaired

Optic discs are white with indistinct margins and slightly elevated

(secondary optic atrophy)

Optic atrophy

Optic atrophy is an important sign of advanced optic nerve disease which can be primary or
.secondary

. Foster-kennedy syndrome = optic atrophy in one eye & disc edema in the other

Primary optic atrophy

Is caused by lesions affecting the visual pathways from the retrolaminar portion of O N to
. the lateral geniculate body

Unilateral atrophy ---- O N lesion

. Bilateral atrophy ----- chiasm & optic tract lesion

Causes

Following retrobulbar neuritis

Compressive lesions as T & aneurysms

Hereditary optic neuropathy

. Toxic &neutritional optic neuropathies

Disc appearance

. White ,flat disc with clearly delineated margins

. Reduction in no. of blood vessels crossing the disc

.Attenuation of peripapillary blood vessels and thinning of retinal nerve fiber layer

Secondary optic atrophy

. Is preceded by swelling of the O N head

. Causes ; papilloedema ,AION & papillitis

Disc appearance ; is variable

. White slightly raised disc with poorly delineated margins

. Reduction in no. of blood vessels crossing the disc