

Ischaemic optic neuropathy

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Still a relatively uncommon cause (compared with other causes) of sudden visual loss in the elderly worldwide, ischaemic optic neuropathy (ION) is, nonetheless, a well-known clinical entity, well-described in ophthalmic texts⁽¹⁾ and often highlighted in journal reports describing variations in causation and presentation, and as country reports. The visual loss that ensues is often solid and irreparable, and therein lies the tragedy.

There are three types of ION, viz: non-arteritic ION, perioperative ION and arteritic ION. All three types may present as acute anterior or posterior (retrobulbar) optic neuropathies. Patients with non-arteritic ION require a systemic evaluation; there is no treatment. Patients with perioperative ION require correction of anaemia and/or hypotension; there is no treatment and no preoperative way to prevent it. Patients with arteritic ION require immediate treatment with high-dose steroids (do not wait for a high erythrocyte sedimentation rate and temporal artery biopsy to begin treatment).

Firstly, to dismiss the rare causes: although common in Caucasians, arteritic ION caused by giant cell arteritis (temporal arteritis or cranial arteritis), is extremely uncommon in Singapore, as emphasised by Cullen and Por in this month's issue of the Singapore Medical Journal.⁽²⁾ The local medical practitioner may not need to consider this in the differential diagnosis. Perioperative ION have been reported most often after back surgery in prone position and in cardiac surgery, attributed to hypotension. Approximate rates are: after back surgery 0.1%–0.01% (Mayo Clinic); after cardiac bypass surgery 0.06% at Mayo Clinic and 0.1% at Johns Hopkins Hospital.⁽³⁾ There have been reports of non-arteritic anterior ION (NA-AION) associated with cataract extractions.⁽⁴⁾ Cullen and Por also described a case (their patient number 2), but rightly pointed out that there is "as yet no final agreement regarding a possible connection".⁽²⁾ Because cataract surgery is so commonplace, the writer is inclined to believe that the association is purely coincidental.

Non-arteritic ION is the commonest variation worldwide. This is also the Singapore experience as reported by Cullen and Por, where they observed that, of 902 new patients attending their neuro-ophthalmology clinics at the Singapore National Eye Centre and the three other main Singapore hospitals over a 18-month period between 2002 and 2004, 200 optic

neuropathies (22%) out of the 902 new patients were ischaemic. Of the non-arteritic ION, the majority were NA-AION and only 2% of the non-arteritic ION involved the posterior portion of the optic nerve, also named non-arteritic posterior ION (NA-PION).⁽²⁾

The paper by Cullen and Por addresses a major cause of sudden blindness from optic neuropathy in their Singapore experience.⁽²⁾ This is a timely call to attention to a major unmet cause of sudden visual loss in the elderly, often with disastrous consequences. A general overview⁽³⁾ of non-arteritic ION may therefore be informative.

Non-arteritic ION occurs usually in patients aged over 55 years. Men and women are equally affected, usually unilateral and usually of the anterior variety. Eye pain is rare, and pain on eye movement is very rare. Clinically, visual acuity loss is variable: 6/6 (20/20) to hand movements or worse. Colour vision usually mirrors acuity. Visual fields usually shows altitudinal or arcuate defect. Relative afferent pupillary defect always present if it is unilateral and there is no abnormality in the opposite eye. The optic disc head is usually hyperaemic, as yet unexplained, and has been attributed to reactive hyperaemia from the retinal circulation following the hypotensive event. Peripapillary flame-shaped haemorrhages are often present. The opposite disc is small with little or no cup.

With regard to causation of non-arteritic ION, underlying systemic vasculopathy is usually present but may not be known at the time of visual loss. This may include hypertension, hypercholesterolaemia, diabetes mellitus, migraine and coagulopathy (especially hyperhomocysteinaemia, anti-platelet abnormalities). Other settings are: anaemia (nonsurgical), hypotension (nonsurgical, nocturnal, Viagra) and sleep apnoea.

The risk factors are a congenitally small disc with little or no cup plus vascular compromise from the underlying systemic vasculopathy or acute blood loss or hypotension (nonsurgical). The pathogenesis is ischaemia at the level of the prelaminar/laminar portion of the optic nerve supplied by the circle of Zinn-Haller via short posterior ciliary arteries.

In the final outcome of NA-AION, about 40% improve spontaneously; visual acuity is more likely to improve than visual fields. Patients are at risk for subsequent cerebrovascular and cardiovascular events. There is no satisfactory treatment. Dilantin, steroids and surgery (optic nerve sheath fenestration) are of no

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benefit. There is a variable risk (10%–19%) for involvement of the other eye. Factors include age, vascular disease and visual acuity, and there is no prophylactic therapy.

A final word on NA-PION, uncommon as it may be. It is not as common as AION but not as rare as many believe. It is not related to optic disc morphology. The same underlying (known or unknown) systemic vascular risk factors may be present (e.g. diabetes mellitus, hypertension, hypercholesterolaemia). The prognosis is the same as for NA-AION.

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