



Normal tension glaucoma

Low tension glaucoma

DESCRIPTION

Primary open-angle glaucoma (POAG) is a condition where the majority of patients show an elevation of intraocular pressure (IOP) above the normal range at some time during the course of their disease. However, up to 30 per cent of cases do not have elevated IOP and these patients are said to have normal tension glaucoma (NTG). Reports suggest that this sub-group of POAG patients is older, with twice as many females as males affected, and often a hereditary trend is present. Some patients also have systemic anomalies such as Raynaud's phenomenon, migraine, vasospasm and nocturnal hypotension, which indicates that NTG, at least in some cases, has a significant vascular component to its aetiology.

SYMPTOMS

NTG is frequently asymptomatic until late in the course of the condition, when the patient may complain of blurred vision and symptoms due to severe bilateral damage to the visual fields.

SIGNS

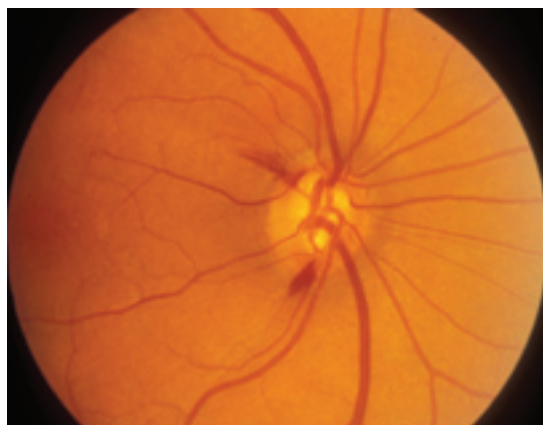
IOP remains within the normal range. The angle is open on gonioscopy with normally appearing angle structures. The optic disc and visual field changes are essentially the same as for patients with POAG and elevated IOP, (see Glaucoma – primary open angle; Glaucoma – Neuro-retinal rim assessment). Some studies have suggested, however, that NTG patients manifest focal notching of the neuroretinal rim of the optic disc more often, together with splinter or Drance haemorrhages that may be recurrent, and visual field defects that threaten fixation.

PREVALENCE

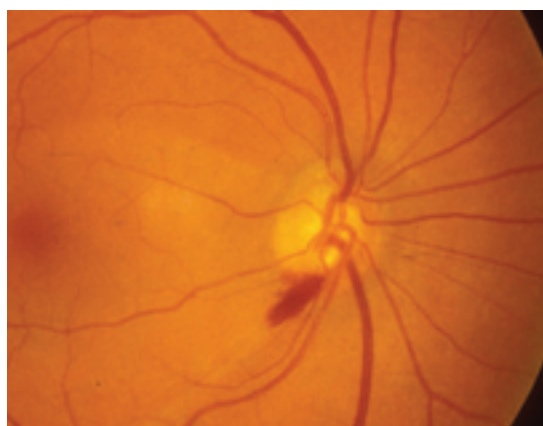
NTG accounts for up to 30 per cent of the cohort of patients in the population who have POAG.

DIFFERENTIAL DIAGNOSIS

Glaucoma – primary open angle, other secondary open-angle glaucoma conditions listed in: Glaucoma – classification. Optic atrophy from chiasmal compression and ischaemic optic neuropathy, Optic disc coloboma, Optic disc pits, Myopia, Optic nerve head drusen, Branch artery occlusion.



A 50-year-old white female with NTG. There are splinter haemorrhages and erosion of the superior temporal neural rim. Visual fields showed a dense paracentral scotoma, inferior arcuate defect and nasal step. IOP was R 12 L 12mmHg



Two years later, the condition had progressed despite medical treatment. A superior nerve fibre layer defect is evident

MANAGEMENT

Advice

A treatment regimen should be instituted for those cases where progression of the disease has been clearly demonstrated. Some studies have indicated that a proportion of NTG patients do not show progression. These patients and those who may exhibit the circulatory difficulties mentioned above should be identified.

Additional investigations

The Ocular Hypertension Treatment Study (OHTS) showed the value of measuring central corneal thickness. A thin cornea (<500microns), whether naturally occurring or post-surgical, will give an artifactually low intraocu-

lar pressure reading. In addition, a thin cornea is an independent risk factor for the development of glaucoma.

Topical medication

Treatment is aimed at lowering IOP by medical or surgical means and a reduction in IOP of the order of 30 per cent may be needed to halt progression. Neuro-protective drugs such as calcium channel blockers may be useful in patients with vasospasm. A prostaglandin agonist such as latanoprost, one drop *nocte*, may be the initial treatment of choice (see Glaucoma – primary open angle).

Review

It is also vital that the patient is monitored very closely which may involve visual field testing at threshold at, say, three monthly intervals to unequivocally establish whether the disease has progressed or not. Compliance with medications that are being taken is essential and must be stressed.

The full series of these articles will be available in the book *Posterior Eye Disease and Glaucoma A-Z* by Bruce AS, O'Day J, McKay D and Swann P. £39.99. For further information click on the Bookstore at opticianonline.net

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