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Editorial

The well tempered crystal ball in normal tension glaucoma

Patients with chronic glaucoma are being referred at lower intraocular pressures (IOPs) than was the case in the 1980s,¹ probably as a result of an enhanced awareness among optometrists of the changes in the optic nerve head in early glaucoma² and the increasing use of routine visual field screeners.³ As the proportion of glaucoma patients designated as “normal tension” in our clinics approaches that of the prevalence studies, there is a greater need for accurate data on the natural history of the disease subtype(s) if we are to make logical decisions on therapeutic interventions.

As many patients with normal tension glaucoma (NTG) do not appear to deteriorate significantly over quite long periods of follow up, even when untreated,⁴ the identification of risk factors for progression in an individual may aid the decision making process concerning whether to treat, and if “yes”, how aggressively. Such data would appear particularly useful in the case of the relatively early case with unocular field loss which naturally presents a challenge to the ophthalmologist in his role as “clairvoyant” and adviser. The paper from Fontana and colleagues in this issue of the *BJO* (p 1002) helps clear some of the mists of the crystal ball into which we must all gaze and generates a thirst for more knowledge on the efficacy of early intervention in NTG.

The results of their clinic based cohort study indicate that there is about a 40% chance of developing a field defect within 5 years in the second eye of a patient who presents with NTG with unocular field loss. Their study also helps us to paint a picture of the patient at increased risk, at least in terms of some ocular risk factors—that is, a poor field score in the first eye and a relatively small neuroretinal rim area (NRA) in the “normal field eye”. Although not examined in the study, one might predict that nerve fibre layer examination would also be of benefit as has been demonstrated to be the case when predicting field loss in ocular hypertension.⁵

Most ophthalmologists cannot (yet!) readily acquire a measurement of NRA in their clinic. As visual field defects often occur relatively late in the excavation process in NTG, careful clinical examination utilising optic disc height measurement⁶ to judge the relative size of the disc, and therefore its natural cup size, and a knowledge of normal morphology can help to identify those already significantly compromised. Indeed, if the other disc and nerve fibre layer are unequivocally normal, it is worth reconsidering the diagnosis, particularly in the older patient.⁷

A measurable variable not shown to be a predictive factor in the Moorfields study was IOP. Should we be surprised by this? We are not given data on the subtype profiles of the NTGs in the study. Could the female/male ratio at 6:4 indicate a predominance of the “focal ischaemic” NTG subtype, with their increased incidence of vasospasm,⁸ or is the presence of marked asymmetry a sign of IOP independence? The analysed IOP was, of course, *measured* IOP, presumably with a Goldmann tonometer. Eyes with NTG tend to have thinner corneas (and therefore lower “measured than true” IOPs), when compared with normal eyes or those with primary open angle glaucoma.⁹ It would therefore be interesting to perform a repeat analysis with IOP corrected for corneal thickness.

Other studies have identified systemic risk factors for progressive field loss in NTG, in particular nocturnal hypotension¹⁰ and non-use of calcium channel blockers.^{3,11} It is interesting and perhaps relevant to many treated NTG patients that nocturnal hypotension has been associated recently with the use of β blocking eye drops.¹² It would therefore seem especially important to exclude this in patients with early NTG, remembering that it might be induced iatrogenically, either by tablets or eye drops.¹² Ambulatory blood pressure monitoring can usually be arranged with your local cardiology department and is well tolerated by most patients.

Fontana and colleagues close by suggesting that treatment for the normal field eye may be started at diagnosis in the scenario of the at risk individual. Recent literature appears to favour the treatment of progressive NTG in order to prevent further field loss,^{13,14} whether it be by medical or surgical intervention. Studies aimed at preventing field loss in the, as yet, “unaffected” eye should be our next step forward. Until evidence of a significant long term advantage is established from carefully controlled trials, it might be wise not to subject a normally sighted eye (and its owner) to therapy which may have significant side effects, unless the disc has clear cut signs of excavation and the fellow eye has moderate to advanced disease.

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