

Normal tension glaucoma: recognising the signs

BY GRANT WATTERS & PROFESSOR HELEN DANESH-MEYER*

CASE HISTORY

A 77 year-old Caucasian retiree presented for a routine eye examination at her optometrist, having broken her two year-old reading spectacles. She had been on medication for elevated cholesterol and high blood pressure for 10 years, and her older sister had been previously diagnosed with glaucoma. There was no known previous history of eye disease, neurological problems or ischaemic events.

The eye examination was unremarkable with no significant distance prescription and corrected visual acuities of 6/6 in both eyes. She had no significant cataract, no afferent pupillary defect, no evidence of pigment dispersion, pseudexfoliation or anterior chamber inflammation, and had non-pigmented open angles on gonioscopy.

Fundoscopy revealed disc asymmetry with estimated cup:disc (CD) ratios of 0.75 in the right eye (RE), and 0.65 in the left eye (LE), with inferior thinning of the neuroretinal rims in both eyes. The intra-ocular pressure (IOP) as measured by non-contact tonometry was 14mmHg in the right eye and 15mmHg in the left eye. Upon further questioning the patient confirmed that she suffered from significant peripheral circulation insufficiency (Raynaud's syndrome) but not from migraines. The patient was prescribed new spectacles and asked to return for Medmont visual field testing and a morning recheck of her IOPs the following week.

Medmont glaucoma field testing was performed with excellent reliability and revealed a small dense superior scotoma close to fixation in the RE, and a mild superior arcuate defect extending superiorly from the blind spot in the LE. IOPs were 15mmHg in the RE and 15mmHg in the LE.

Based on the asymmetric discs and apparent early field defects, the patient was referred to a glaucoma subspecialist ophthalmologist for further investigation.

The ophthalmologist confirmed slightly thinner than normal corneae (RE 501 microns, LE 505 microns), open angles on gonioscopy, and described the CD ratios as 0.85RE and 0.80LE, with disc asymmetry and "some loss of neuroretinal rim" in the LE. Sequential Zeiss-Humphrey central

24-2 threshold tests confirmed early "non-specific" changes. Blood pressure was measured as normal, being 130/90.

A baseline Heidelberg HRT-II test of the optic nerve head was performed and confirmed cupping outside normal limits in 3 of 6 sectors of the disc in the RE and 2 of 6 sectors in the LE.

A therapeutic trial of Xalatan (latanoprost 0.001%) drops once a day in the RE only was initiated, with follow-up at one month. At follow-up the IOP had decreased from 14 to 11mmHg in the RE and the patient was subsequently commenced on the same treatment in the LE. At the 6 month follow up the IOPs were RE 11mmHg, LE 12mmHg and Zeiss-Humphrey fields showed no progression in the right eye and possibly slight progression in the left eye. Repeat HRT examination of the discs showed no significant change in the RE, and perhaps slight further loss of neuro-retinal rim in the temporal region of the LE. However, over the following 5 years there has been no further deterioration of IOP, visual fields or HRT assessment of the optic disc.

OPTOMETRISTS VIEW: GRANT WATTERS

As one of Optometry's most important roles is the early detection of glaucoma, it is important to remember the risk factors contributing to normal tension glaucoma (NTG). Unfortunately it is estimated that 50% of all NTG cases go undetected and NTG could contribute to up to half of all cases of open angle glaucoma. This patient exhibits many NTG risk factors, including family history of glaucoma, female gender, age, history of low blood pressure (or nocturnal BP dipping), and Raynaud's syndrome (white-cold fingers). Other risk factors for NTG are myopia, Asian ethnicity, migraine/vasospasm and smoking.

An accurate analysis of the optic disc appearance, particularly of asymmetry and non-obeyance of the ISNT rule (especially temporally and inferotemporally) is also essential but is sometimes difficult in the case of myopic patients with tilted discs. To illustrate an example of this, my most recently diagnosed patient was a 62yr old contact lens-wearing female with moderate myopia, myopic tilted discs, large cups and a

mother and brother already diagnosed with glaucoma.

Fortunately, with the current accessibility of retinal cameras a considered comparison of the two optic nerves heads is possible. It can also be argued that Optometry should also embrace pachymetry in conjunction with Goldmann IOPs to get a truer picture of an individual's thickness-corrected or "true" IOPs – not just for NTG but for all IOP measurements. I believe it can be too easy to "switch off" once we think an individual's IOPs are within the normal range and not still look carefully enough at the optic nerve head for subtle changes, let alone think about corneal thickness.

Another objective measurement of the status of the retinal nerve fibre layer can be achieved with optical coherence tomography (OCT) or GDx examination. These instruments are now used routinely in the Optometry Clinic at The University of Auckland as part of student training. Unfortunately, current cost issues with these instruments still put them out of reach of the average Optometry practice; but this is slowly changing.

The problem with NTG is that progression of field defects can be rapid; the defects tend to be close to fixation and can be dense and steep. An optic nerve head with vascular insufficiency is more easily damaged. It can also be difficult to distinguish between NTG and other forms of optic nerve disease, including anterior ischaemic optic neuropathy secondary to giant cell arteritis, and neurological disease. An assessment of disc pallor and colour vision testing can help eliminate the latter. Additionally, gonioscopy, which is now accepted as a necessary basic skill for Optometrists, is required for a diagnosis of NTG.

It can be argued that Optometrists should be proactive in screening family members of patients with glaucoma. All people with diagnosed glaucoma should be telling their family to have a check-up, but should we be offering to notify family members of the importance of a glaucoma check?

So please keep these risk factors and signs of NTG in mind and don't forget to fully check all of your contact lens patients for glaucoma at their annual visit!

OPHTHALMOLOGIST VIEW: PROFESSOR HELEN DANESH-MEYER

As one reads through the case history above it would be easy to get the impression that this is a simple case of glaucoma to diagnose. However, it actually has all the ingredients of being one of those cases that is most commonly missed: the patient has 6/6 vision in both eyes, she has 'normal' IOP, and while the cups are large there was no gross notching or loss of neuroretinal rim. This case is the wolf in sheep's clothing of glaucoma!

It reflects very strongly on the clinical acumen of the optometrist that there was an index of suspicion that precipitated further investigations. So what are the key features of this case that supported a suspicion of glaucoma?

First, the patient has a family history of glaucoma. Patients may not spontaneously proffer this information and it is important to note that it is the responsibility of the eye care professional to ask the appropriate questions. It is known that there is a genetic component to glaucoma. In fact,

if the family member is a sibling, as in this case, the risk of glaucoma is 10 times greater than the general population.

There is great debate regarding the risk factors for NTG. Some evidence points toward patients with glaucoma having more 'vasospastic' tendencies which includes Raynaud's phenomenon and migraine. Another school of thought disputes these findings. The jury is probably still out, and I personally find it useful to question patients in this regard. In my clinical experience, there certainly seems to be a tendency for patients with NTG to have a higher incidence of vasospasm.

The key findings in the optic nerve appearance that suggested early glaucoma included the asymmetry between the two eyes (albeit subtle), the loss of inferior neuroretinal rim (and consequently the ISNT rule was not obeyed), and the correlation of the inferior thinning of the neuroretinal rim to the superior paracentral visual field loss.

An important issue in patients with glaucoma, but particularly NTG, is the question of their systemic hypertensive control. If they are receiving treatment for systemic hypertension, as this patient is, then it is important to explore the possibility of nocturnal dips in blood pressure. Such events could contribute to poor perfusion of the optic nerve head and progressive visual field loss.

Once it has been decided that the patient has glaucoma and requires treatment the next question regards the target pressure. Again, the concept of target pressure in itself is controversial and you will see glaucoma specialists wrestling over this quite frequently in public. My personal view is that it is important to have a target pressure, although a target range is also acceptable. For this patient I would have a target of 10-11 mmHg. She was started on Xalatan to which she responded favourably, and over the past 5 years there has been no deterioration in her visual fields or optic nerve appearance. Research has shown that patients go blind from glaucoma most commonly for two reasons: 1) delayed diagnosis, so they present with advanced visual field loss and 2) lack of compliance with treatment. Patient education (and consequently patient compliance) can be greatly enhanced by enrolment in Glaucoma New Zealand which provides regular education for patients with glaucoma.

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Right and left optic discs