

Glaucoma: an optometry case study

Mrs. BA, a 49 year old female, presented in private practice for a routine examination. Her main presenting symptom was presbyopia; having difficulty with reading fine print.

Previous ocular history: She had been prescribed spectacles in her early twenties for reading work. Distance vision is quite clear and she is currently using hobby glasses to read more comfortably.

Personal Medical History: No reported medical conditions, with no prescribed medication being taken.

Family Medical and Ocular History: No reported history of glaucoma, cataracts, unusual eye conditions, blindness, heart or blood pressure problems, or diabetes.

Ocular examination findings:

Unaided Right VA 6/7.5

Prescription: Plano – 0.25 x 180
BCVA 6/7.5 ADD +1.00

Unaided Left VA 6/7.5

Prescription: Plano – 0.25 x 180
BCVA 6/7.5 ADD +1.00

Pupils exhibit round, reactive, and brisk responses.

Slit-lamp bio-microscopy showed the cornea to be clear, excluding a small old traumatic scar in the inferior right cornea. The bulbar conjunctiva was very quiet and no notable redness present. The anterior angle was 0.3 using the Van Herrick technique while

gonioscopy showed the angle to be open with the Schwalbe's line, Schlemm's canal and all of trabecula network seen.

Ophthalmoscopy showed the maculas to be healthy however the cup/disc ratios were 0.8 in the right and 0.7 in the left with a depth of 1 dioptre. The neural retina rims exhibited thinning and the ISNT sign was disrupted.

Intra-ocular pressures were 12 mmHg in the right eye and 14 mmHg in the left eye measured by AO Non contact Tonometer at 1.00 p.m.

Visual fields exhibited superior arcuate defects in both eyes close to the fixation and relatively deep.

The patient was referred to an ophthalmologist for confirmation of diagnosis of normal tension glaucoma and to initiate medical treatment.

DIAGNOSIS

The criteria for diagnosis of normotensive glaucoma, as provided by Moorfields Hospital are:

- typical glaucomatous optic nerve damage with cupping and loss of neural retinal rim,
- progression of damage,
- an open anterior angle on gonioscopy,
- a mean intra-ocular pressure of less than 22 mmHg and no measurement greater than 24 mmHg,
- and an absence of a secondary cause.

This particular case fulfils these

criteria, given that at the time of presentation a pressure series had not been preformed thus the criteria of mean pressure could only be assumed.

Other conditions that would need to be eliminated include:

Glaucoma disorders: such as primary open angle glaucoma with large diurnal intra-ocular variation, intermittent closure glaucoma, glaucomatocyclitic crisis, steroid induced glaucoma, burnt out glaucoma, pseudoexfoliation or pigment dispersion glaucoma, glaucoma secondary to anterior segment trauma, false intra-ocular pressure readings (corneal thinness, low scleral rigidity).

Non glaucoma optic neuropathies: such as branch retinal artery or vein occlusions, physiological cupping, nerve head drusen, compressive lesions, blood shock or loss, drug induced optic atrophy, ischemic optic neuropathy.

Congenital defects: such as coloboma, pits, or tilted discs.(Gutteridge, 2000).

By careful evaluation of the signs and symptoms in each case then the majority of the differential diagnoses can be eliminated. Signs and symptoms to be evaluated include; the optic nerve head appearance, patient history, anterior segment including angle, an intra-ocular pressure series, and visual field loss evaluation.

Thus the diagnosis of normotensive glaucoma is often a diagnosis of exclusion.

“visual fields exhibited superior arcuate defects in both eyes close to fixation”

DISCUSSION

Glaucomatous changes in the optic disc, which result in visual fields changes, require treatment to endeavor to prevent progression of visual field loss. The more common sign of glaucoma being elevated intraocular pressure is affected by the anatomy of the anterior chamber and aqueous humor formation. The structures found in the anterior angle are; (anterior to posterior) Schwalbe's line, Schlemm's canals, trabecular meshwork, scleral spur and anterior boarder of ciliary body and iris. Schwalbe's Line is the termination of Descemet's membrane of the cornea. Schlemm's canal is a circumferential canal, posterior to the trabecular meshwork which traverses from Schwalbe's line to the scleral spur. The meshwork enables drainage of aqueous from the anterior chamber through to Schlemm's canal and further through collector canals to aqueous veins (Forester, 2002). These then join either deep interscleral or episcleral veins plexus or directly drain to superficial conjunctival veins. This process accounts for 70% of aqueous drainage and is thought to be the most pressure sensitive pathway, the remaining 30% is via the uveoscleral pathway. There are three zones of the meshwork; uveal meshwork, corneoscleral meshwork and cribriform meshwork which is probably the main site of resistance to aqueous flow (Forester, 2002).

The aqueous humor's main function is the supply of nutrients to and removal of metabolic waste from, the avascular tissues of the anterior eye and inflating the globe creating IOP for the normal optical function of eye. Formation of aqueous humor is thought to be mostly via active ion transport and movement of solutes across the ciliary epithelium (To C, Kong C, Chan C, Shahidullah M, Do C, 2002)

The fluid dynamics of the aqueous humor are associated with glaucoma. If there is an elevation of IOP, due to either increased production of aqueous humor or reduction of drainage this causes the effect of producing damage to the optic nerve at its exit point from the globe. The structure this damage is exhibited in is the optic disc. Evaluation of this structure causes difficulty in diagnosing the presence of glaucoma. Numerous indicators are used to evaluate the optic neuropathy caused in glaucoma. Vingrys (2000) reviews these indicators and their sensitivity. Signs of glaucoma optic neuropathy include abnormal neural retinal rim configuration (the absence of ISNT sign and/or notching), abnormal cup configuration (cup size for disc size), blood vessel changes and compromises, peripapillary atrophy, retinal nerve fiber layer loss, and function loss typical of glaucoma of the eye.

Normo-tensive glaucoma is the condition where there is characteristic glaucoma optic nerve damage with a resultant visual field loss, in the presence of intra-ocular pressure averaging less than 22 mmHg, with open drainage angles and the absence of a secondary cause. This condition can be considered to have the same progression as primary open angle glaucoma without the risk factor of elevated intra-ocular pressure. Normo-tensive glaucoma can be associated with various vascular and cardio-vascular disorders. These disorders include systemic hypertension, arterial hypertension, previous haemodynamic crisis, increased blood viscosity, diabetes, migraine and other vasospastic disorders (Gutteridge 2000). Each of the above risk factors supports a theory for a vascular cause or component in the glaucomatous changes to the optic nerve in normo-tensive glaucoma rather than a mechanical or pressure theory.

A significant proportion of primary open angle glaucomas are normo-tensive glaucomas which compounds the clinical difficulty in early diagnosing of this group of glaucomas.

It is believed that normo-tensive glaucoma occurs more commonly in females, with a mean age at first diagnosis being 60-70 years of age (Gutteridge, 2000).

The prime site of pathological glaucoma changes in the optic nerve head. This neuropathy involves loss of ganglion cell axons, resulting in an enlarging cupping excavation of the nerve head with the corresponding progressive visual field loss. The nerve head is susceptible in the inferior and superior poles which results in thinning or nothing in these areas earlier on in the disease process.

Traditionally the mechanism of axon damage is thought to result by one or both of two mechanisms. Firstly by mechanical disruption of axoplasmic transport which causes distortion to the axons through the lamina cribrosa. This results in backward bowing of the lamina cribrosa, axon loss and progressive cupping. The second mechanism suggests axon transport is affected by a primary anoxia associated with high pressure decreasing optic nerve head perfusion.

More recent theories suggest baro-trauma or ischemia of the optic nerve head results in increased apoptosis of axon cells, thus loss in the number of axons, thus glaucomatous changes to the optic nerve head (Gutteridge, 2000).

Vaso-spasm or other mechanisms resulting in transient decreases in blood flow to critical parts of the optic nerve head have been thought of as possible causes of glaucomatous damage to the nerve when the intra-ocular pressure does not appear high enough to be the dominant factor in causing that damage.

Clinical signs and features outlined by Gutteridge, 2000, include the presence of increased optic disc cupping due to loss of neural retinal rim tissue. Care must be taken in assessing this cupping to allow for the size of the optic disc as larger discs have larger cups. Large studies of glaucomatous discs have shown that normo-tensive glaucoma tends to be associated with either focal ischemic cupping, where there is localized tissue loss at the superior and/or inferior poles of the disc, or senile sclerotic disc which exhibit broad shallow cupping with saucer-like cup margin, frequently with peripapillary atrophy and choroidal sclerosis.

The presence of optic disc haemorrhages is thought to be indicative of open angle glaucoma, both with high or normal pressures. It is generally agreed that haemorrhages do occur more frequently in normo-tensive glaucoma, possibly associated with focal ischemic changes in the disc.

Peripapillary atrophy is thought to occur with greater frequency in normo-tensive glaucoma. The atrophy may be ischemic in origin and thus may fit in with a ischemic theory for normo-tensive glaucoma (Gutteridge, 2000).

There are thought to be some differences between the visual field defects of primary open angle glaucoma and normo-tensive glaucoma. These are considered to be that the defect is closer to fixation, within 5 degrees of fixation in over 90 percent of normo-tensive glaucoma and with a steeper slope to the defect (Gutteridge, 2000). Also the superior hemifield is more likely to be involved by a factor of 2 to 4 times.

This corresponds to the prevalence of higher frequency of inferior disc and neural retinal rim damage seen in normo-tensive glaucoma. It should be noted however that superior field defects are common in primary open angle glaucoma.

Although many clinical signs indicate that glaucomatous cupping and visual field loss could be due to normo-tensive glaucoma, there are no signs that are specific to normo-tensive glaucoma or that cannot be found in a patient with primary open angle glaucoma. Thus while there is considerable overlap with risk factors in both conditions, signs such as infero-temporal thinning of the neural retinal rim, disc haemorrhage in the infero-temporal quadrant of the disc, detectable nerve fiber layer defects and superior arcuate visual field defects, relatively close to fixation, have the greatest probability of a causal association with normo-tensive glaucoma (Gutteridge, 2000).

MANAGEMENT

Treatment of glaucoma can be either medical or surgical. In normo-tensive glaucoma a reduction of 30 percent would be considered a reasonable initial target pressure after treatment.

Sack (2000) outlined that drugs can be broadly broken into two groups. Those drugs that lower intra-ocular pressure and those drugs that offer the additional benefit of neuroprotection, independent of intra-ocular pressure reduction.

Drugs in the first group include;

Betaxolol, a beta-1 selective agent thus reducing aqueous production and also thought to increase ocular blood flow to the ocular nerve head, due to vasodilatation. Studies suggest this may have a neuro-protective effect against visual field deterioration.

Brimonidine, an alpha-2 adrenergic agonist thus reducing aqueous production also with some neuroprotective effects.

Dorzolamide, a carbonic anhydrase inhibitor, which also increases ocular blood flow increasing perfusion.

Latanaprost, a prostaglandin inhibitor, which reduces intra-ocular pressure via uveoscleral outflow which has the advantage of not being pressure driven thus can be very effective in reducing intraocular pressure in normo-tensive patients.

Neuroprotective drugs are suggested to have an action by increasing ocular blood flow or inhibiting excitatory neurotransmitters. This group includes:

Oral Calcium channel inhibitors, which cause vasodilatation. Care is needed with their use as they also reduce blood pressure thus reducing the blood flow to the optic nerve head. They are also considered to have neuroprotective effect outside increased blood flow by inhibiting enzymatic cascades, which may result in ganglion cell death.

Mementine, which blocks the activity of glutamate and is considered to have the potential to slow visual field deterioration.

Recent papers have suggested that reducing the intraocular pressure by 30 percent does provide benefits. This amount of reduction might not be achieved with monotherapy using prostaglandin analogues only so combination therapy with drugs such as brimonidine or dorzolamide may be necessary (Hoyng PF, Kitazawa Y, 2002). These treatment therapies can be especially be beneficial to specific patient profiles. This differentiation between profiles in response to treatments suggests there maybe different factors that contribute to optic neuropathy in different subgroups of normo-tensive glaucoma (Anderson DR, Drance SM, Schulzer M. 2003)

OUTCOME

On referral the ophthalmologist confirmed the diagnosis of normo-tensive glaucoma and started treatment binocularly with Xalatan (Latanaprost 0.005%) 1 gutt at night with the aim of reducing the pressure to at least 30 percent of the mean current pressure. Review of pressures and fields were performed and as adequate reduction of pressure could not be obtained the patient was referred on to a glaucoma specialist. Current treatment is Travatan 1 gutt at night.

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