Low Tension Glaucoma

On April 23, 1988 a 74-year-old white female was seen in the clinic for a glaucoma evaluation. She had been previously seen by her optometrist for a routine eye exam with a complaint of light sensitivity. He had noted suspicious cupping of the optic nerves with low intraocular pressures. She was taking Synthroid, Oretic, Lanoxin and Donnatal for her hypertension, tachycardia, stomach problems and thyroid removal. Neither the patient or her family had a history of any eye disease.

Physical exam. Rest corrected acuity was 20/40 OD and 20/20 OS. The patient's pupils were equal, round and responded to light with no afferent pupillary defect. Motility was full and Amsler grid was negative OD with the bottom right corner missing OS. Slit lamp exam revealed 2+ staphlococcal lid disease, corneas showed Hudson-Stahl lines OU, the anterior chambers were deep and quiet and lenses showed minimal opacification. Using applation tonometry the intraocular pressure was 22mmHg OU prior to dilation. A dilated fundus exam revealed discs to have prominent cupping. There was no apparent nasal rim tissue OD with sloping temporal rim tissue and a nerve fiber layer hemorrhage at the disc margin at 10:30. The left disc also had prominent cupping with no rim tissue from 12 to 2 and 4 to 6, with thin sloping rim tissue from 2 to 4. Vessels and periphery were unremarkable. Some drusen and atrophic pigment change was noted in the maculas bilaterally. Octopus visual fields showed the right eye having nasal constriction with overall depression. The left eye had a similar finding with an inferior arcuate scotoma as well. The patient was diagnosed as having low-tension glaucoma and was put on Betoptic 0.5% q12h OU.

On three-month follow-up examination the patients acuities were 20/30 OU and pressures were 17mmHg. Visual fields revealed dense field loss nasally with central islands remaining and some depressed, but intact, temporal field OU. Pilopine ointment was added qhs OU and a follow-up visit was scheduled to determine if Propine should be used in addition or if an argon laser trabeculectomy was necessary.

Low tension glaucoma was first described in 1885 by von Graefe as an eye with a typical glaucomatous disc, field changes and open angles with pressures in the statistically normal range. This definition still holds true today and LTG is said to have a prevalence of 25-30% of all glaucomas. This fact leads to much discussion as to whether primary open angle glaucoma and low tension glaucoma are one and the same disease or two separate diseases separated by a pressure level. To answer that question and discover the etiology of all glaucomas.
would help to solve the therapeutic dilemma of how to manage the patient with low tension glaucoma.

Theories as to the etiology of or contributing factors to low tension glaucoma are numerous. Most accepted is the ischemia theory which is considered 'true low tension glaucoma' and is thought to be due to multiple insults to the nerve head over time. This glaucoma is progressive and is caused by decreased vascular profusion with intraocular pressure accelerating neuronal loss. The ischemia can be due to the diseased large feeding vessels that determine the disc perfusion pressure or of the smaller vessels within the disc. Primary open angle glaucoma is primarily due to the high pressure in the eye causing damage to the optic nerve, this is where the 'two diseases' differ most. Migraines, which are considered an ischemic disorder are reported most often in LTG patients compared to normal and POAG patients. This fact helps to provide evidence supporting the ischemic theory.

Another type of low tension glaucoma is due to a single insult to the nerve head. These patients have histories of sudden and profound systemic hypotension and includes 33% of LTG patients compared to 12% of POAG patients. The visual field loss in these patients is non-progressive and is usually not treated. The systemic problems can be the result of bleeding ulcers, trauma, acute blood loss, myocardial infarction and antihypertensive medication. Previously increased intraocular pressures due to corticosteroids, uveitis or angle closure could also mimic low tension glaucoma.

Low tension glaucoma has also been associated with a high incidence of systemic disease. These diseases can include chronic vascular disease, vascular hypotension and hypertension, carotid artery disease, low ophthalmic artery pressure, elevated blood lipids and a hypercoagulable blood state. Drugs, nutritional factors and decreased physical activity can also be associated with LTG. It was also noted that the mean difference in blood pressure between lying and standing were significantly greater in patients with LTG compared with POAG and normal tension patients (-6.0 vs -1.2 vs -1.5mmHg). This indicates that postural hypotension could play a role in the pathogenesis of LTG.

Anatomical weakness is another possible explanation for the glaucomatous changes in some patients. The lamina cribosa connective tissue could be defective consequently damaging neurons as they pass through the nerve head. Other patients optic nerve heads could be more susceptible to an injury by intraocular pressure, the disc resilience being sufficiently low enough to permit disc and visual field defects at "normal" pressure levels.
It is estimated that about one-third of LTG patients have abnormal aqueous humor dynamics. The hyposecretion theory involves patients with an impaired facility of outflow and a decreased aqueous secretion rate. It is thought that many of these patients have high diurnal variations.

A thorough examination of the low tension glaucoma patient is necessary and a suggested exam could include the following. An ocular history should be taken with particular reference to previously noted high IOP's, long term use of topical or systemic steroids, ocular trauma or ocular inflammatory disease. The complete eye exam should include a careful evaluation of the disc for congenital or acquired abnormalities which give field defects that may mimic those found in glaucoma (ex: optic pits, drusen, peripapillary scars), ocularvascular disease evidence (disc hemorrhages, disc margin notching, hypertensive and diabetic retinopathy), a visual field exam with emphasis on detection of neurological defects and diurnal IOP measurements. A careful medical history should be taken noting cardiac disease (acute myocardial infarction, congestive heart failure, angina), peripheralvascular disease, diabetes mellitus, cerebrovascular disease, transient ischemic attacks, hemodynamic crises, antihypertensive andhypotensive therapy. A medical evaluation could be done including aglucose tolerance test, complete blood count, cardiac evaluation, ECG, blood pressure, temporal artery pulses and tenderness. A neurologicalevaluation is another option in which carotid pulses and bruits,ophthalmodynamometry, ocular motility disturbances, exophthalmometry,skull and optic foramen x-ray films would be checked. If any of theabove is abnormal a CT scan is suggested.

It is important to do a careful exam because many of these patientsfall into categories other than low tension glaucoma. These include:
- large diurnal variation
- impaired outflow facility causing intermittent high IOP's
- other causes of retinal damage (vascular, trauma, congenital,inflammatory degenerative, unknown)
- intracranial neoplasm (usually anterior chiasmal area)

The differences between primary open angle glaucoma andlow tension glaucoma is an important topic in current research. A rational approach to therapy might be determined if thepathophysiology of the disease and the factors that interact in theproduction of the clinical characterisitics and the progression aredetermined. Differences between the nerve head appearance and visualfield changes are most often studied, but other differences have beennoted. LTG tends to be more common and severe in females whereas POAGis most common in males. Myopia is more common in LTG patients thanthe general population (22.1%), but this percentage is not as high asPOAG patients. There also tends to be a higher percentage of
unilateral cases compared with POAG. Most other characteristics between the two glaucoma types are similar other than the pressure difference.

Ophthalmoscopy in stereo is most important in evaluation of a glaucomatous disc. Viewing the disc binocularly one can predict glaucomatous defects in the nerve head with an accuracy of 90% compared to 40% with a direct ophthalmoscope. Disc pallor, cupping (size, depth, position, shape), rim tissue, vessel displacement and asymmetry between eyes should be noted on the nerve head. It has been stated that there is greater thinning on the neuroretinal rim on the disc in the inferior and inferotemporal region in low tension glaucoma patients. The cup tends to slope broadly toward the inferotemporal rim as opposed to a steep drop in primary open angle glaucoma patients. Nerve fiber layer hemorrhages are said to be seen more often in low tension glaucoma patients. One study found that 20.5% of LTG patients have hemorrhages compared to 4.2% of POAG patients. These peripapillary hemorrhages are most often seen between 4 and 6 o'clock. Using fluorescein angiography there tends to be a localized and consistent area of hypofluorescence in area 13 (inferotemporally) without any other vascular abnormalities.

Visual field evaluation is equally important. Defects to look for include blind spot enlargement, isolated paracentral scotomas within the arcuated Bjerrum region, arcuate scotomas, nasal depression, nasal step, peripheral constriction and temporal step. It has been stated that visual field defects are closer to fixation and steeper sloping with a greater depth in LTG patients compared to POAG patients. The densest scotoma is usually found in the superior hemifield. The visual field loss is usually of sudden onset and the optic disc cupping tends to be out of proportion to the visual field loss. It has also been noted that there is twice as much loss of sensitivity in the spared hemifield in POAG patients compared with LTG patients.

Other in office tests should include: gonioscopy, biomicroscopy and color vision. Provocative tests and contrast sensitivity functions could also be performed. The provocative tests are used to produce excessive elevations of IOP in glaucomatous eyes. The tests are, however, rarely conclusive, potentially hazardous and commonly directed towards the type of glaucoma under suspicion. The test of choice to detect open angle glaucoma is the water drinking test and a rise of 9mmHg is definitive evidence of the disease. Lower spatial frequency losses can also be found in glaucoma patients whose Snellen acuity is normal. Spatial contrast sensitivity testing can be done using video display systems or simple photographic plates. This type of testing has a predictive value of 75% for glaucomatous visual field loss.
Management of the low tension glaucoma patient is usually difficult and frustrating. Medical treatment is first attempted and then some type of surgery if the former is not successful. The intraocular pressure is lowered to try to prevent further visual field loss. A reasonable goal is to obtain pressures between 9 and 12 mmHg. Close cooperation between the eye care practitioner and internist is essential. Marked decreases in blood pressure should be avoided and all disease should be treated, especially anemias and other hemodynamic crises. Medical treatment is similar to the POAG patient and is insufficient in most cases. It is said to lower the pressure by approximately 2.5mmHg in the past and more recently by 4.0mmHg.\textsuperscript{16} If medical therapy is not helpful, an argon laser trabeculoplasty is suggested. This procedure can lower the pressure by 4-5mmHg and stops the the visual loss progression in 73\% of patients.\textsuperscript{16} Other last resort surgeries include a full thickness sclerectomy, a thermal sclerostomy or trabeculectomy with a very thin scleral flap and wide sclerectomy excision. These surgeries will lower pressures by about 11, but complications are high and include: cataracts, choroidal detachment and flat anterior chamber.\textsuperscript{16}


