A Clinical Study to Evaluate a Correlation between Thyroid Problems and Glaucoma

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INTRODUCTION

Primary open angle glaucoma (POAG) is the leading cause of visual impairment and blindness in the United States and worldwide. It is higher among Africans-Americans than most other racial and ethnic groups.¹-⁷ The risk increases with increasing age. The term Graves disease includes a constellation of disorders consisting of goiter, hyperthyroidism and often associated with ophthalmopathy occasionally with infiltrative dermopathy or acropathy. The exact aetiopathogenesis of Grave’s disease is unknown but it is likely to be an autoimmune disorder. It is diagnosed in 3rd and 4th decade of life and it shows a female dominance over a male with male female ratio being 1:7.

Grave’s ophthalmopathy (GO) and Primary open angle glaucoma (POAG) show similar signs – elevated intraocular pressure (IOP) and visual field defects. Grave’s ophthalmopathy is characterized by eyelid retraction, eyelid swelling, keratitis, proptosis, restrictive myopathy, elevated IOP on upgaze, impaired visual acuity and visual field defects in severe cases. POAG is diagnosed through the classic triad of elevated IOP, open angle of anterior chamber, glaucomatous cupping of optic disc, and characteristic visual field loss. Risk factors in the development of glaucomatous optic nerve damage are the amount of elevation of the IOP, increasing age, a family history of glaucoma and black race. Patients with elevated IOP, but normal visual fields and cup/disc ratios, are classified as having ocular hypertension (OH). In patients with Graves ophthalmopathy elevated IOP in upgaze is a common finding and is explained by a tight inferior rectus muscle that blocks the episcleral aqueous outflow and causes orbital congestion. Other studies, however have failed to find a significant association between hypothyroidism and glaucoma.⁸¹⁹ So study was done to find the association between hypothyroidism and glaucoma.

MATERIAL AND METHODS

In this study all the patients referred from department of medicine and diagnosed with thyroid imbalance were taken into consideration. Included in this study were patients with a diagnosis of Grave’s orbitopathy (Based on the clinical picture, a characteristic computed tomograph scan, and supported by immunological and/or endocrinological findings) and who met one of the following criteria: the use of glaucoma medication at referral, repeated elevated IOPs (> 22 mmHg) measured by application tonometry in the standard position, glaucomatous or dysthyroid optic neuropathy (DON) visual field defects and/or glaucomatous optic disc cupping. In the patients who fulfilled the inclusion criteria, age at the time of reference, sex, race as well as family history for glaucoma were recorded. All these patients were subjected to following protocol of examination.

A detailed history regarding their complaints, the onset, duration, past history and family history of thyroid diseases and glaucoma were taken into account. Preliminary examination of uncorrected and bestcorrected visual acuity was determined with Snellen’s chart. Intraocular pressure was measured in all selected patients of thyroid with

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Shiotztonometry. Detailed slit lamp examination and fundus examination with direct and indirect ophthalmoscopy was done to rule out any other organic cause of blurred vision. Perimetry was done in all 20 selected patients of thyroid irrespective of their intraocular pressure. All patients diagnosed with POAG were followed up after 1,3 and 6 months for repeat IOP and perimetric analysis.

**STATISTICAL ANALYSIS**

Descriptive statistics like mean and percentages were used for analysis of data.

**RESULTS**

5 patients (10%) had elevated IOP labelled as ocular hypertension with non glaucomatous optic disc or visual fields changes (Table 1). Out of the 20 selected patients, 15 were female and 5 were male (Table 2). In this study concerning 20 patients, 5 patients were labelled as Grave’s ophthalmopathy with POAG showing typical field and disc changes (Table 3).

**DISCUSSION**

Wessely, in 1918, was the first to describe increased IOP on upgaze in patients with Grave’s ophthalmopathy. Since then, several other studies described the phenomenon of increased IOP on upgaze in patients with Grave’s ophthalmopathy. All the studies agreed to the mechanism of increased IOP on upgaze i.e., inelasticity of inferior rectus muscle as a result of fibrosis and thus the incapacity to relax causes compression on the globe when the antagonist pulls the eye upwards. The prevalence of ocular hypertension in the general population older than 30 yrs is 1.6%. In a recent study of Peele Cockerham K et al the 24% prevalence of ocular hypertension in 500 patients with Grave’s ophthalmopathy could be partly explained by the mechanism of looking slightly upwards during applanation tonometry. All were treated with glaucoma medications. Initially only prostaglandin analogues were started and if IOP was not controlled after 15 days beta blockers were added provided, patient was not asthmatic or a heart patient. All the patients responded well with glaucoma medications except one in which orbital decompression was planned. The IOP is determined by the rate of aqueous humour production by the ciliary body, the resistance to aqueous outflow across the trabecular meshwork and the level of episcleral venous pressure. Increased orbital pressure in Grave’s orbitopathy causes increased episcleral venous pressure which subsequently causes increased IOP. Orbital decompression decreases the IOP by reducing the intraorbital pressure and thus, the episcleral venous pressure. Along with the glaucoma medications, artificial tear drops containing carboxy methyl cellulose was also started.

**CONCLUSION**

POAG has the same prevalence in the general population as in the GO subgroup. The combination of elevated IOP and visual field defects in the GO patients may be attributed to mechanisms other than obstructed aqueous outflow in the trabecular meshwork and should be treated accordingly. The IOP should be measured in standard position and in down gaze. Visual field defects of DON (dysthyroid optic neuropathy) must not be confused with that of glaucoma. Treatment should be at two distinct levels. Both general health and eye health need to be assessed and managed independently but simultaneously. Other health problems like hypertension, elevated cholesterol and sugar intolerance must be identified and and controlled. This is especially important if steroids need to be taken orally, as they can increase blood pressure as well intraocular pressure and can predispose to diabetes. Management of the eyes involves using particular types of anti-glaucoma medications to reduce IOP and other medications such as strong lubricants to protect the anterior surface of the eye and to achieve comfort for the patient.
If anti glaucoma measures fail, orbital decompression and recession of the inferior rectus muscle may reduce the IOP.

REFERENCES


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