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

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ABSTRACT

Introduction: The POAG is more commonly seen in elders between 5th and 7th decade. The prevalence of POAG and ocular hypertension is more in patients suffering from Graves ophthalmic disease than the normal individuals. Present study was done to find the association between hypothyroidism and glaucoma.

Material and methods: A Prospective clinical study was carried out in a tertiary eye care hospital in a period from May 2016to April 2017. Out of 50 patients of thyroidimbalance 20 patients were taken into study. Patients were followed after 15 days, 1 month, 3 months and 6 months. Different Parameters were used like IOP, visual field defects, fundus changes and treatment given.

Results: Out of 50 patients with thyroid imbalance, 20 patients (4%) met the inclusion criteria and out of these 15 were females and 5 were males; Mean age at referral was 55 years (range 23 – 77 years). 5 patients had ocular hypertension without field defects or optic nerve head changes. 5 patients had POAG with a cup disc ratio >0.5, typical glaucomatous visual field defects and pressures ranging from 16 to 27 Hg with medications. There was even a slightly higher IOP in upgaze.

Conclusion: Thyroid related ophthalmopathy is usually a self-limiting condition, although it can take several years to abate. The aim is to preserve vision until it does so. For a good outcome, care of the cornea and the optic nerve are equally important, along with optimal general health.

Keywords: Thyroid, Graves Ophthalmopathy, POAG, Ocular Hypertension

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.[1-6] 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.[7,8] 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 diagonosed 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 applanation 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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Total no. of patients	Patients of POAG	Patients with ocular hypertension	Pts without ocular findings
50	20	5	25

Table-1: Total no. of patients = 50

Total no. of selected patients	Male	Female	Patients with optic nerve head changes	Patients with field changes
20	5	15	5	5

Table-2: Total no: of selected patients=20

Patient age in years	Sex	IOP mmHg	Optic nerve head changes	Visual field
74	F	18/24	RE: glaucomatous(G) LE: normal	RE:temporal defects LE:Normal
54	F	26/26	BE: G 0.4-0.5	BE:Nervefibre bundle defects
64	F	19/27	BE:G 0.5-0.6	BE:CC scotoma
48	F	23/22	BE:G C:D ratio 0.5	BE:temporalscotoma
60	M	21/22	BE:G C:D ratio 0.6-0.7	BE:annular ring

Table-3: Patients with graves ophthalmopathy (GO) and POAG (total = 5 patients)

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 visuals 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.[9] 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 up gaze 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[6] 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 ofthe 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.

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