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Original Research Article

Steroid Induced Ocular Hypertension and Glaucoma: A Brief Clinical Analysis

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Abstract

Background and Aim: Individual differences in steroid response risk play a part in the unpredictability of steroid-induced glaucoma. While topical application of this medication will result in a decrease in aqueous outflow, a rise in circulating corticosteroid may cause an increase in aqueous inflow. The current study sought to examine the etiological risk factors, length of steroid use, and clinical presentation of steroid-induced glaucoma.

Material and Methods: The present analysis is the prospective analysis done at the department of the ophthalmology, medical college & hospital. The study was performed for the period of 12 months. The study population consists of 86 patient established with steroid induced glaucoma. The demographic details of the included patients were recorded. The reasons for and length of the detailed history of steroid use were documented. The different risk factors, including primary angle glaucoma, connective tissue disorder, hypertension, diabetes mellitus, and family history of glaucoma, were also thoroughly documented. In order to rectify refractive defects in all the details, visual acuity was measured and refraction was performed in each case. Field of vision defect, frequent eyeglass replacement, and vision defect were all carefully noted.

Results: A total of 172 eyes of 86 patients were taken into account of the analysis. Steroid induced glaucoma was resolve in 34 eyes of 26 patients. The visual acutity was found to be normal in 34 eyes. Less vision was found in 16 eyes. The visual acuity was found to be defective in 86 eyes. The defect was due to change in lens in 12 eyes. In 74 eyes the reason was found due to refractive error. 50 patients' eyes were found to be normal in the current investigation, 36 eyes had tubular fields, 14 eyes had inferior arcuate defects, and 6 eyes had superior arcuate defects. IOP significantly changed between before and after the intervention.

Conclusion: Steroids should be avoided or, if necessary, delivered in lesser dosages to people who are at risk or who are susceptible. According to our study, patients who had cataract surgery were more negatively impacted by the ongoing use of topical steroids.

Keywords: Ocular hypertension, Steroid-induced glaucoma, Visual acuity, Vision.

Introduction

Steroids are routinely used following intraocular surgery and are frequently given for a variety of autoimmune and inflammatory diseases. Steroids can cause negative systemic and ocular side effects, such as cataracts, increased IOP, and glaucoma, despite having many advantages.[1] A third of people can have the steroid response form of ocular hypertension. Elevated IOP and glaucomatous optic neuropathy in the presence of corticosteroid use are considered to be symptoms of steroid-induced glaucoma. Since patients may need to continue receiving steroid treatment for their underlying problems, this iatrogenic disease is frequently challenging to control.[2]

Individual differences in steroid response risk play a part in the unpredictability of steroid-induced glaucoma. When the general population is treated to topical ocular steroids, 5% to 6% of people experience high steroid responses (IOP elevation >15 mm Hg), 29% to 36% of people experience moderate responses, and 58% to 66% of people do not experience any discernible IOP elevation.[3,4]

In 1950, when systemic treatment of adrenocorticotropin hormone (ACTH) was demonstrated to increase intraocular pressure (IOP), the association between steroids and glaucoma was first described. Armaly and Becker were the first to report an increase in IOP in response to topical steroids.[5,6] A kind of secondary open-angle glaucoma known as "steroid-induced glaucoma" was once thought to develop only following indiscriminate topical steroid use. Since then, it has been determined that other routes of delivery, including as oral, intravenous, periocular, dermatological, intravitreal, and intranasal, are to blame for the severe and irreversible loss of vision caused by the resulting glaucoma.[7] Glaucoma is currently the second most frequent reason for blindness. The most frequent worldwide cause of permanent blindness is glaucoma.[8,9]

While topical application of this medication will result in a decrease in aqueous outflow, a rise in circulating corticosteroid may cause an increase in aqueous inflow.[10,11] Since multiple studies have linked the vehicle to the agent that would elevate IOP, it is possible that the vehicle itself plays a role in this differential effect when it comes to topical medications. As a result, the current study's goal was to examine the clinical presentation, etiological risk factors, and duration and type of steroid use in steroid-induced glaucoma.

Material and Methods

The current analysis was conducted by the department of ophthalmology, the medical college, and the hospital. The study covered a year's worth of time. 86 patients who have been diagnosed with steroid-induced glaucoma make up the study population. Prior to the study's launch, the ethical committee was made aware of it and an ethical clearance certificate was obtained. Patients received information about the trial in their native tongue, and they signed an informed consent form before being admitted.

The inclusion and the exclusion criteria followed in the study were as follows:

Inclusion criteria for the study comprised patients with a history of topical or systemic steroid use, persistent increase of IOP greater than 21 mm Hg, and presence of gonioscopically open angles. Patients who had developing glaucoma, normotensive glaucoma, or narrow angle glaucoma were not included in the study.

The included patients' demographic information was noted. The reasons for and length of the detailed history of steroid use were documented. The numerous risk factors, including primary angle glaucoma, connective tissue condition family history of glaucoma, diabetes mellitus hypertension, and others, were also noted in detail. In order to rectify refractive defects in all the details, visual

acuity was measured and refraction was performed in each case. Defect in vision, frequent change in the glasses and defect in field of vision were recorded in detail.

Slit lamp technology was used during the eye examination to both rule out potential glaucoma causes and track potential lens changes. Gonioscopy was performed using a single mirror Goldmann gonioprism. The angles were graded utilising Shafer's grading methodology. Using a Goldmann application tonometer, the central corneal thickness as determined by pachymetry was compensated for when the intraocular pressure was measured. Using +90D slit lamp biomicroscopy, the cup, disc ratio was measured during the fundus examination.

All of the patients underwent a diurnal variation test (phasing), which involved taking 6 readings spaced 4 hours apart throughout the course of the day and graphing the points on the graph. Utilizing a Perkins applanation tonometer, the recording was made. For the study, valid field tests were conducted with false positive and false negative rates below 30%.

At first, unnecessary steroid use was reduced, and patients received routine monitoring. As recommended by the rheumatologist, some patients with connective tissue disorders were taking maintenance doses of steroids. Depending on the degree of glaucomatous damage, patients received individualized care. The patients were then regularly checked on and followed up with at regular intervals. For 20 participants, plasma cortisol levels were assessed at 8 a.m. and 8 p.m.

Twenty individuals had selective laser trabeculoplasty done. Selective laser trabeculoplasty was performed on glaucoma patients who continued to worsen despite receiving medicinal treatment. Patients were given non-steroidal anti-inflammatory drops and anti-glaucoma medications for 5 days following selective laser trabeculoplasty. Patients were then examined shortly after, anti-glaucoma medications were withdrawn, and an examination was conducted two weeks later. Hypertensives had their blood pressure checked, and they were then directed to a hypertension clinic. Random blood sugar readings were taken, and if they were high, a diabetologist's opinion was sought. Rheumatologists provided care for those whose connective tissue illnesses had been identified.

Results

A total of 86 patients were taken into consideration. A total of 172 eyes of 86 patients were taken into account of the analysis. Steroid induced glaucoma was resolve in 34 eyes of 26 patients. There were 44 males and 42 females included in the study. The male to female ratio was found to be almost similar. The age distribution of the patients ranged from 15 to 70 years. The predisposed patients were generally above 45 years of age. The maximum numbers of patients were between the ages of 50 to 60 years. Similar results were obtained in the studies done earlier.[12]

The visual acutity was found to be normal in 34 eyes. Less vision was found in 16 eyes. The visual acuity was found to be defective in 86 eyes. The defect was due to change in lens in 12 eyes. In 74 eyes the reason was found due to refractive error. In the present analysis in 58 eyes the average IOP was found to be 21 – 30 mm of Hg. The IOP was found to be below 20 mm of Hg in 20 eyes.

Oral steroids were administered in 56 eyes. Posterior subtenon injection was given in 6 patients, intravitreal triamcinolone was given in 4 patients, topical medication was given in 48 patients. According to studies, people who use topical medications are more likely to develop steroid-induced glaucoma.

In our study, steroid-induced glaucoma was most frequently caused by cataract surgery. There had been cataract surgery on 40 patients, 24 of whom had only had it done on one eye.

The remaining 16 patients received cataract surgery in both eyes. According to research, allergic conjunctivitis was the primary factor causing steroid-induced glaucoma.

50 patients' eyes were found to be normal in the current investigation, 36 eyes had tubular fields, 14 eyes had inferior arcuate defects, and 6 eyes had superior arcuate defects. Due to inadequate cooperation, automatic perimetry was not achievable in 10 eyes. Patients with steroid-induced glaucoma frequently develop field abnormalities like those in primary open angle glaucoma.

In the current investigation, 76 eyes had glaucoma under medical control. The administration of antiglaucoma drugs was based on IOP at presentation. Antiglaucoma drugs were either continued or terminated based on changes in the optic nerve head and subsequent IOP measurements. 24 eyes received surgical care; 14 eyes received trabeculectomy and cataract extraction, while 10 eyes received trabeculectomy only. For 36 eyes, medical treatment and then selective laser trabeculoplasty were performed. IOP significantly changed between before and after the intervention. A paired t test was used to compare the values. The test appears to be significant because the P value was below 0.001. The mean intraocular pressure (IOP) at presentation was 35.40 mm Hg, and the mean IOP at follow-up was 13.53 mm Hg.

Table 1: Comparison of IOP Before and After Treatment

Parameters	Mean	P Value
IOP on Presentation	35.40	**< 0.001**
Follow up IOP after Treatment	13.53	

Discussion

The increase in intraocular pressure is brought on by a decrease in trabecular outflow in steroid-induced secondary open angle glaucoma. When corticosteroids are used locally or systemically, IOP rises, but responses vary from person to person. Although seldom possible, an abrupt elevation in IOP within hours in connection with systemic use of steroid or adrenocorticotropic hormone can occur after initiating topical steroids.[13,14]

According to Armaly *et al*., a 4-week course of topical dexamethasone 0.1% elevates IOP by more than 6 mm Hg in about one-third of normal eyes and more than 90% of individuals with primary open-angle glaucoma. Increased intraocular pressure (IOP) occurs in 30 to 50% of patients undergoing intravitreal triamcinolone acetonide.[14] Even though topical medicines are used to treat the majority of corticosteroid-induced glaucoma patients, increasing optic nerve injury has been observed without being supported by visual field abnormalities.[15]

A strong index of suspicion is necessary for the diagnosis of steroid-induced glaucoma, and patients must be specifically questioned regarding their usage of steroid eye drops, ointments, skin preparations, and pills. Along with the duration of steroid use, the history should include any glaucoma in the family. IOP measurement, gonioscopy, and an assessment of the optic disc should all be done as part of a thorough ocular checkup. Although they are not required, fundus photos and optic disc imaging are preferred for tracking development.[16]

It is necessary to closely and frequently monitor the IOP in individuals receiving corticosteroid therapy. The dosage, mode of administration, half-life, and duration of treatment of the medicine should all be considered together with the patient's risk factors for steroid-induced rises in blood

pressure. Patients at high risk who receive intravitreal injections need to be checked out the day and the week following therapy, as well as at least monthly after the medicine is stopped.

Conclusion

Steroids should be avoided or, if necessary, used in lesser dosages in people who are at risk and those who are susceptible. According to our study, patients who had cataract surgery were more negatively impacted by the ongoing use of topical steroids. Once the eye is calm, topical steroids should be withdrawn. In contrast, post-operative patients at risk for glaucoma should ideally be provided non-steroidal anti-inflammatory medications. In these people, IOP should be checked often.

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