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Clinical Analysis of Steroid Induced Glaucoma

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Abstract
Background: Steroid use has the potential to cause the elevation of intraocular pressure (IOP) without any regard to the form of steroid. This study analyses the various clinical presentation, etiological risk factors, duration and type of steroid used in causing steroid- induced glaucoma. It helps in arriving at the diagnosis and discusses about the various management options in the treatment of steroid - induced glaucoma. The response of selective laser trabeculoplasty in treatment of steroid induced glaucoma patients is also being analysed.
Materials and Methods: This prospective study was conducted at Institute of Ophthalmology in 43 patients of established Steroid - induced glaucoma. 68 eyes of 43 patients were taken into study.
Results: Steroid induced glaucoma resolved in 17 eyes of 13 patients with their stoppage of steroids and with initiation of medical treatment. In the study group 51% were male 49% were female. Visual acuity was normal (6/6) in 25% (17 eyes) of the cases. Average IOP on presentation was between 21-30mm of Hg (42.6%) in 29 eyes. (41.2%) 14 patients (28 eyes) were on oral steroids. Diabetes mellitus was the major risk factor in this study. The most common reason for steroid induced glaucoma in this study was, following cataract surgery. Average duration of steroid in years, less than 1 year – 41.5% in 27 eyes and above 6 years (13.8%) in 9 eyes. 38 eyes had medical management of glaucoma.
Conclusion: The patients who underwent cataract surgery were more affected due to continued application of topical steroids. Post-operative patients who are prone to glaucoma should be prescribed preferably with non-steroidal anti-inflammatory drugs. Plasma cortisol does not correlate clinically in steroid induced glaucoma patients. Selective laser trabeculoplasty helps in short term control of IOP.

Keywords: steroid induced glaucoma, visual acuity, cataract surgery, plasma cortisol

Introduction

Today glaucoma is the second most common cause of blindness. Glaucoma is the most common cause of irreversible blindness in the world. The world health organisation estimates (2002) that number of people who have become blind because of glaucoma were 4.4 million (12.3% of the blind worldwide) population based studies have estimated that the prevalence of glaucoma in India to be about 11.9 million and 60.5 million in the world in 2010.

The response to long term steroid therapy whether given by the topical, systemic procedure or intraocular route and the IOP elevation can lead to glaucomatous optic atrophy and loss of vision, such a condition is called as steroid induced glaucoma1. Patients who receive corticosteroid therapy may develop IOP elevations in days, weeks, months or years after initiating treatment. The clinical picture resembles that of chronic open angle glaucoma with an open normal appearing anterior chamber angle and absence of symptoms. Approximately one third of the individuals experience moderate increase in IOP after topical steroid use. However 5-6% of normal population develops a marked increase in IOP after 4-6 weeks of topical steroid therapy. Thus 5% of the general population is considered to be "steroid responders" i.e. they may develop steroid induced glaucoma when steroid are administered. This was shown by studies conducted by Armaly and Becker [13]. Hence this study was undertaken to analyse the clinical presentation, etiological risk factors and toassess the response of selective laser trabeculoplasty in treatment of steroid induced glaucoma patients.

Materials and Methods

This was a prospective study conducted at glaucoma services at regional Institute of Ophthalmology at tertiary care hospital. The study was done in 43 patients of established steroid induced glaucoma after complete evaluation. Patients with the history of any mode of steroid use either topically or systemically and those who presented with persistent elevation of IOP more than 21mm of Hg, disc suspicious of glaucomatous change and gonioscopically diagnosed as open angle glaucoma were included in this study. Patients with normotensive glaucoma, narrow angle glaucoma, all other secondary glaucoma and developmental glaucoma were excluded. A detailed history regarding the type of steroid used, mode of use, reason for use, and duration of use was recorded. The risk factors of family history of glaucoma, primary open angle glaucoma, diabetes mellitus, hypertension, high myopia and connective tissue disorders were also asked in detail from the patients.

Patients were enquired about defective vision, defective field of vision and frequent change of glasses. Visual acuity was recorded

and refraction was done in all cases to correct refractive errors. Ocular examination of both eyes with slit lamp was done to rule out other causes of glaucoma and to know about the lens changes. Gonioscopy was done using single mirror goldmann gonioprism and angles were graded by using shaffers grading method. Intra ocular pressure was measured using goldmann applanation tonometer and corrected to the central corneal thickness as measured by pachymetry. Fundus examination was done using +90D slit lamp biomicroscopy and the cup, disc ratio was noted. Associated vascular signs like nasalisation, Bayonetting, Laminar dot sign, baring of circumciliary vessels and splinter hemorrhages near the disc were noted. Diurnal variation test (phasing) was done in all the patients by recording 6 readings each 4 hrs apart throughout the day plotting the graph connecting all the points. The recording was done using perkins applanation tonometer. Field charting was done by computer assisted static automated perimetry, (octopus 123, G1X program TOP strategy) for both the eyes. Reliable field testing with false positives and false negatives below 30 percent were taken for the study.

Unnecessary usage of steroids was curtailed initially and the patients were followed up regularly. Certain patients with connective tissue disorders were on maintenance dose of steroids as advised by the rheumatologist. Patients were treated according to the amount of glaucomatous damage. Then the patients were reviewed frequently and followed up regularly in equal intervals. Plasma cortisol levels were measured at 8 am in the morning and 8pm in the evening for 10 patients. Selective laser trabeculoplasty was performed for 10 patients. The patients who showed increased cup disc ratio even after medical therapy were chosen for selective laser trabeculoplasty. After selective laser trabeculoplasty patients were prescribed non-steroidal anti-inflammatory drops and anti-glaucoma drugs were continued for 5 days. Soon after which patients were reviewed, anti-glaucoma drugs were stopped and evaluation was done after 2 weeks.

Results

68 eyes of 43 patients were taken into study. Two patients were one eyed. One had leucomatous corneal opacity and other patient had evisceration done in one eye. Rest of the 16 eyes were not involved. Steroid induced glaucoma resolved in 17 eyes of 13 patients with their stoppage of steroids and with initiation of medical treatment.

Age distribution

Ages of the patients studied varied from 14-65 years. There was no age preponderance. Predisposed patients were generally above the age of 40 years. The maximum age of incidence was between 51-60 years. Steroid use was more in older age groups for various medical ailments. This was similar to the studies conducted earlier [44].

Sex distribution

Among the 43 patients 22 were male and 21 were female. There was no sex preponderance. In the study group 51% were male 49% were female.

Visual acuity (VA)

Visual acuity was normal (6/6) in 25% (17 eyes) of the cases. 8 eyes had vision less than 5/60 (11.8%). Among those 8 eyes, 1 eye had posterior capsular opacification, 2 eyes had severe posterior subcapsular cataract, 5 eyes had glaucomatous optic atrophy. In 6(8.8%) eyes visual acuity was defective due to lens changes and in 37 (54.4%) eyes it was due to refractive error.

Intraocular pressure (IOP) distribution

Average IOP on presentation was between 21-30mm of Hg (42.6%) in 29 eyes. 10 eyes (14.7%) the IOP was below 20mm of Hg. These patients had glaucomatous damage. They had already stopped applying steroid before presentation. IOP measured was similar to the previous studies [44].

Distribution of risk factors

5 patients were hypertensives, 4 patients were myope, 4 were case of rheumatoid arthritis, 1 patient had diabetes mellitus and rheumatoid arthritis, 3 patients had diabetes mellitus, 3 patients had diabetes mellitus and hypertension, 1 patient had primary open angle glaucoma and diabetes mellitus, 1 patient had dermatomyositis and 1 patient had polymyositis. The major risk factor in our study was Diabetes mellitus. This was similar to previous studies [44].

Type of Steroid used

The use of oral steroids was seen in (41.2%) 14 patients (28 eyes). (4.4%) 3 patients had posterior subtenon injection. (2.9%) 2 patients' intravitreal triamcinolone was given. (51.5%) 24 patients (35 eyes) were on topical medication alone. As per studies patients on topical medication are more prone for steroid induced glaucoma [42, 43].

Reason for Steroid Use

The steroid use following cataract surgery was the most common reason for steroid induced glaucoma in our study. 20 patients (41.2%) had under gone cataract surgery, of which 12 patients had undergone cataract surgery in only one eye. Rest 8 patients had undergone cataract surgery in both the eyes. As per studies done allergic conjunctivitis was the most common reason for steroid induced glaucoma [42, 43].

Duration of steroid

The Average duration of steroid use for less than 1 year –was 41.5% in 27 eyes and above 6 years was 13.8% in 9 eyes. For 2 patients one dose of intravitreal triamcinolone was given, of which one was for cystoid macular edema due to central retinal vein occlusion, and for another patient intravitreal triamcinolone was given for Diabetic Retinopathy. 3 patients were injected with posterior subtenon injection of which 2 were given for parsplanitis and 1 for retinal vasculitis.

Optic nerve head changes

The average cup disc ratio was normal 0.3 (26.5%) in 18 eyes. 5 patients had glaucomatous optic atrophy.

Field defects distribution

25eyes (36.8%) of patients in this study were found to be normal. 18 eyes had tubular field (26.8%), Inferior arcuate defects were found in 7 eyes (10.3%), Superior arcuate defects were found in 3 eyes (4.4%). In 5 eyes (7.4%) automated perimetry was not possible due to poor co-operation. Steroid induced glaucoma patients normally have field defects similar to primary open angle glaucoma [1].

Treatment

Medical management of glaucoma was done in 38 eyes. Depending upon IOP on presentation antiglaucoma medications was given. Based on optic nerve head changes and follow up IOP (done after 3 weeks), antiglaucoma medications were either continued or stopped. Surgical treatment was done for 12 eyes, cataract extraction along with trabeculectomy was done for 7 eyes, trabeculectomy alone was done for 5 eyes. Medical management followed by selective laser trabeculoplasty was carried out for 18 eyes.

Follow up IOP

The average follow up IOP was between 17 and 20 mm of Hg. In equal percentage of patients the follow up IOP was less than 12 mm of Hg. When the IOP on presentation was high (above 30), oral acetazolamide was given 250 mg BD, oral glycerol 30 ml TDS, intravenous injection of mannitol 200 ml BD, and topical medication of 0.5% BD timolol was given. Once the IOP was reduced medicines were gradually stopped or continued depending upon the glaucomatous changes.

Steroid Induced glaucoma along with cataract

14 eyes of 7 patients had post subcapsular opacity. 2 patients underwent cataract extraction with trabeculectomy. Lens changes were not compromised in of the 5 patients. Those who presented with cataract and glaucoma were all on oral steroids. Among the 7 patients, 3 patients had rheumatoid arthritis, 1 patient had rheumatoid arthritis and diabetes mellitus. 1 patient was a case of dermatomyositis, and 2 patients were bronchial asthmatics.

Maintenance on steroids

4 patients were on maintenance dose. 3 patients of rheumatoid arthritis were on maintenance dose of tablet prednisolone. 1 patient of dermatomyositis was also on maintenance dose of steroid. While 2 patients of bronchial asthma were on oral steroid only during exacerbation in winter season.

Comparison of IOP before and after treatment

There was a significant difference in IOP before and after treatment. The values were compared by paired T test. The P value was <0.001, suggesting that the test is significant. The mean IOP on presentation was 31.10 and the mean follow up IOP was 15.59.

Plasma cortisol

Plasma cortisol levels were measured for 10 patients at 8 am and 8 pm. The mean of values taken at 8 am in the morning was 13.7190. The mean of values taken at 8pm was 6.9219. All the values found at 8 am and 8 pm were within normal limits, showing that there is no effect of plasma cortisol in steroid induced glaucoma. Morning values were greater than the evening values. Morning (8am) values compared with 8 pm (evening values) by t test showed significant variation with p value (0.012).

Comparison of IOP Before and after SLT

The patients were treated medically with single drug before SLT. The aim of SLT was to provide benefit of reducing IOP similar to medication. So that patients need not apply topical medication. The mean IOP before SLT was 17.37, the mean IOP after SLT was 19.26. IOP of 18 eyes of 10 patients were compared with paired T test. P value was 0.283 suggesting that it was not significant. There by implying SLT did not bring down IOP to desired levels to prevent further disc damage.

Discussion

Steroid-induced glaucoma is an iatrogenic secondary open angle glaucoma, with decreased trabecular outflow causing a rise of intraocular pressure. An increase in IOP occurs in response to the local or systemic use of corticosteroids, but the response varies among individuals. IOP response usually takes 2 to 4 weeks after starting topical steroids, though rarely there can be an acute rise of IOP within hours in association with systemic use of steroid or adrenocorticotropic hormone (ACTH). If the ocular hypertension of a significant magnitude, which was not recognized and treated, leads to the development of subsequent glaucomatous optic neuropathy (that is, steroid-induced glaucoma). In vernal keratoconjunctivitis (VKC), steroid induced glaucoma is a common complication as patients require long-term therapy and steroids are often used to provide early relief of symptoms.

Recently, the popular use of intravitreal triamcinolone acetonide (IVTA) for subretinal fluid, macular edema, and adjunctive therapy in the treatment of choroidal neovascularization has led to an increased incidence of corticosteroid-induced ocular hypertension and glaucoma. In this study 2 patients were given intravitreal injection of triamcinolone. One for diabetic retinopathy with macular edema and the other for macular edema due to central retinal vein occlusion. Both of them under went trabeculectomy due to increased IOP on presentation, as it could not be controlled with medication.

Pre-existing POAG, or a status of a first-degree relative with POAG are important risk factors for corticosteroid-induced ocular hypertension and glaucoma. Age may be a risk factor; increased risk appears to occur in a bimodal distribution peaking first at age 6. As one progresses through adulthood age may not be a factor until late adulthood when the risk again rises. Finally, those with connective-tissue disease, type-1 diabetes mellitus, and high myopia should all be considered to be high risk, and prudent follow up and monitoring is mandatory during prolonged periods of corticosteroid use.

Evidence supports that synergistical action of several independent potential mechanisms are involved in increasing the resistance to the outflow of aqueous humor, leading to corticosteroid-induced ocular hypertension:

Reduced availability of lysosomal enzymes leads to the accumulation of polymerized glycosaminoglycans in the trabecular mesh work, there is further increase in outflow resistance due to the suppression of phagocytosis by trabecular endothelial cells resulting in accumulation of trabecular debris. Probable influence of Genetic mechanisms, with possible upregulation of myocilin, optineurin and other factors which resulted in increase in aqueous outflow resistance.

Diagnosis of steroid-induced glaucoma requires a high index of suspicion and the questioning of patients specifically about their use of steroid eye drops, ointments, skin preparations, and pills. History should also include duration of steroid use, and family history of glaucoma. Complete ocular examination should be done including measurement of IOP, gonioscopy and optic disc evaluation. Fundus photographs and optic disc imaging are desirable for documenting progression, though not mandatory.

In individuals with an IOP more than 20% above their baseline measurement, or in those for whom there is clinical or functional evidence of damage to their optic nerve during or after treatment with corticosteroids, any or all of the following may be necessary to reduce IOP.

To look into the indication for steroid use, if steroid is not necessary, to taper and stop steroids. If indicated, to reduce the concentration or dosage of the steroid, change to lesser potent steroid (e.g. fluorometholone, loteprednol, or rimexolone). Switch to a topical nonsteroidal anti-inflammatory drug (e.g. ketorolac 0.4%, diclofenac 0.1%) and start antiglaucoma therapy if required.

Obtain baseline visual fields and/or optic nerve photography or peripapillary retinal nerve fibre layer measurements, if appropriate.

If the IOP is at alarming levels (> 50 mmHg, even in the case of an optic nerve that appears healthy), surgical intervention with either a tube or a filter may be appropriate. Four patients in our study had IOP greater than 50 mm of Hg on presentation. Now their IOP is under control with medication. Among these three had advanced glaucomatous cupping and they have been advised to undergo surgery and the remaining one patient had normal fundus findings. These surgeries are required in fewer than 2% of patients receiving an intravitreal injection. Surgeons should consider a vitrectomy or the explantation of the steroid implant for patients who have received intravitreal injections or intraocular implants of a corticosteroid.

Close and regular monitoring of the IOP of patients treated with corticosteroids is required (especially those with a personal or family history of POAG or steroid-induced glaucoma). The frequency of IOP monitoring should match the patient's risk factors for steroid induced spikes in pressure as well as the medication's potency, dosage, route of administration, and half-life and the duration of treatment.

High-risk patients who receive intravitreal injections require examinations one day and one week after treatment and at least monthly follow-up examinations after the medication's cessation [41].

Conclusion

In susceptible individuals and persons with risk factors, steroids should be avoided or if required should be administered in smaller doses. As per our study patients who underwent cataract surgery were more affected due to continued application of topical steroids. Topical steroid application should be stopped once the eye is quiet. Alternatively post-operative patients who are prone to glaucoma should be prescribed preferably with non-steroidal anti-inflammatory drugs. IOP should be measured regularly in these individuals. In cases of advanced glaucomatous damage surgery will be a better option. Plasma Cortisol does not correlate clinically in steroid induced glaucoma patients. Selective laser trabeculoplasty is not effective in preventing further glaucomatous damage. Selective laser trabeculoplasty helps in short term control of IOP.

Future Scope

Selective laser trabeculoplasty can be used in short term control of IOP. It is useful in very old patients who are not amenable to surgery and for those who are not willing for surgery. Plasma Cortisol levels can be measured more frequently once in four hours to find out the association between plasma Cortisol levels and steroid induced glaucoma. It can also be carried out in larger number of patients. Steroid provocative test will help in identifying the high steroid responders in the general population. It can also be performed in patients for whom intravitreal triamcinolone has to be given. Genetic studies may throw more light on the etiopathogenesis of steroid induced glaucoma.

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