Original Article

Evaluation and Management of Steroid Induced Glaucoma in Vernal Keratoconjunctivitis Patients

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Purpose: To study the behaviour and management of steroid induced glaucoma in vernal keratoconjunctivitis (VKC) patients in our setup.

Material and Methods: Sixteen patients of VKC with steroid induced glaucoma were enrolled in this study presented to Al Shifa Trust Eye Hospital during year 2005. Clinical signs and symptoms, management and outcome of these patients was observed for up to six months.

Results: With steroid induced glaucoma the pressure elevation is gradual. Therefore, like primary open angle glaucoma, very few symptoms exist. Cases in which intraocular pressure did not normalize upon cessation of steroids, needed medical and surgical intervention.

Conclusion: In VKC patients, most effective drug, steroid should be carefully administered, and only for brief periods, to avoid secondary development of glaucoma. All patients who use chronic corticosteroid medication in any capacity should have a full ophthalmologic evaluation during the course of treatment.

Vernal Keratoconjunctivitis is a bilateral chronic inflammation of the conjunctiva. The disease affects children between three to sixteen years of age, though it may appear earlier than that and continue into adulthood. In the majority of the cases, symptoms resolve at puberty. Although the name vernal suggests a seasonal spring time occurrence, frequently the disease persists throughout the year[1].

Intense itching, irritation, burning and photophobia (sensitivity to light) are the main symptoms of the disease. Corneal involvement leads to complaints of reduced vision. The disease is characterized by giant flat topped papillae of the upper tarsal conjunctiva leading to the clinical picture of cobblestones. The limbal form is characterized by conjunctival hyperemia, papillae at the corneoscleral (limbal) border and Tranta's dots. The diagnosis is generally based on the signs and the symptoms of the disease. Antiallergic drops, steroids and ocular hygiene are the main stay of treatment for vernal catarrh patients.

Steroid induced intraocular pressure (IOP) elevation typically occurs within a few weeks of beginning steroid therapy. In the majority of cases, the IOP lowers spontaneously to the baseline within a few weeks to months upon stopping the steroid. In rare instances the IOP remains elevated. The study of steroid induced glaucoma in vernal keratoconjunctivitis patients is important for two reasons. Many patients who receive glucocorticoid therapy are susceptible to the development of ocular hypertension,

which if unrecognized can lead to glaucomatous optic neuropathy and the irreversible loss of vision. In addition, it helps us in evaluating the efficacy of different treatment options available for the management of steroid induced glaucoma [2].

MATERIALS AND METHODS

A prospective study was carried out on vernal keratoconjunctivitis patients with steroid induced glaucoma, coming to glaucoma department (Al-Shifa Trust Eye Hospital) and Shifa College of Medicine in the year 2005. All cases were seen and dealt by the same surgeon. Evaluation of the patient included detailed history, visual acuity, intraocular pressure measurement, slit lamp examination of the anterior segment, gonioscopy, dilated fundus examination for optic disc evaluation, and Humphrey's visual field analysis (30-2) of both eyes.

Diagnosis of vernal conjunctivitis was based on the typical history and characteristic signs and symptoms. Associated glaucoma was labeled when the patient had elevated intraocular pressure with typical optic disc cupping and corresponding visual field defects. Signs and symptoms of steroid induced glaucoma were noted and compared with those of primary open angle glaucoma.

Patients with persistently elevated pressures and ongoing disc damage were treated first medically .Beta blockers, carbonic anhydrate inhibitors (topical and systemic), prostaglandin analogues, and sympathomimetics were used variably in patients depending upon the target pressure and the affordability by the patient. Those with maximal tolerable medical treatment and intraocular pressures greater than the target pressures were surgically treated. Trabeculectomy with mitomycin C (limbal based flap, 0.01% mitomycin applied for 2 minutes at the scleral bed before making the internal window and paracentesis) remained the surgical procedure of choice in all. Patients were then followed for up to six months and postoperative complications were noted.

Patients who had settled intraocular pressures after discontinuing steroid treatment alone, and without any supportive treatment were followed on three monthly basis, and intraocular pressure and optic disc cupping was noted. In suspicious cases visual field examination was also repeated to monitor the progression of glaucomatous optic neuropathy.

RESULTS

There were 16 patients and all had bilateral disease (32 eyes). Among them 4 (25%) were females and 12 (75%) were males. Visual acuity at presention in six patients was 6/6 to 6/12 in the better eye), in 6 patients was 6/18 to 6/24 in the better eye and in 4 patients was less than 6/60 in the better eye. All patients had open angle glaucoma on gonioscopy.

No pain was reported, perhaps due to the gradual rise in pressure. Decreased vision was attributed to the associated corneal changes. None of them were aware of the associated disease process unless told. At presentation 4 patients (25%) had IOP between 20 and 30mmHg while 12 patients (75%) had IOP greater than 35mmHg. Four patients (25%) had early glaucomatous damage (cupping less than 0.7) and 12 (75%) had advanced glaucomatous damage (cupping more than 0.7). When steroids were discontinued there was return of IOP to normal in 4 patients (25%) while in the rest 12 (75%) patients there was no change in IOP when steroids were stopped.

In patients with initial IOP greater than 35 mm Hg the IOP remained above the target level despite combination treatment (systemic acetazolamide, topical beta blocker, sympathomimetics / prostaglandins analogue).

Trabeculectomy with Mitomycin C was performed in 12 (75%) patients (all uncontrolled on medical treatment). IOP on first postoperative day was 6-8 mm Hg in all 12 patients. Three patients had preservation of their postoperative visual acuity, 7 had 1 to 2 line improvement as the pressure was lowered and 2 patients showed one to two lines falls in visual acuity.

One case had failed bleb and redo surgery was performed. One case had persistent ocular hypotony, one had thinning of the bleb and conjunctival grafting was done. Six had associated cataract formation and needed surgery for those. Ten patients had successful intraocular pressure control at six months

DISCUSSION

Vernal keartoconjunctivitis is an allergic eye disease that especially affects young boys. The most common symptoms are itching, photophobia, burning and tearing. The most common signs are giant papillae, superficial keratitis, and conjunctival hyperemia.

The clinical management of VKC requires a swift diagnosis, correct therapy, and evaluation of the prognosis. The diagnosis is generally based on the

signs and symptoms of the disease, but in difficult cases can be aided by conjunctival scrapings, demonstrating the presence of infiltrating eosinophils[3]. (Eye 2004 -18, 345-351).

During this study we examined and counseled the children. Patients had visited different clinics during the course of the disease. Steroids were used without counseling about the disease. Patients continued to use the steroids, because of the immediate relief in symptoms[4]. They had no awareness of the steroid associated complications. While on topical steroids, they were visited different clinicians, but steroid related rise in intraocular pressure was missed. Patients that came to us with advanced steroid induced glaucoma (irreversible) and had been on this treatment for many years. Hence injudicious use of steroids and improper examination in between lead to steroid induced complications.

In vernal keratoconjunctivitis patients, therapeutic options are many. In most cases topical therapy should be chosen on the basis of the severity of the disease. The most effective drug steroids, should however be carefully administered, and only for brief periods, to avoid secondary development of glaucoma. A 2% solution of cyclosporine in olive oil or in castor oil should be considered as an alternative[5].

The long term prognosis of patients is generally good; however 6% of patients develop corneal damage, cataract or glaucoma[3]. (Eye 2004; 18: 345-351).

Glucocorticoid therapy can cause elevated intraocular pressure in many susceptible individuals, who are often referred to as "steroid responders". Approximately 40% of the general population can develop IOP elevation (>5mm Hg) after topical ocular administration of a potent glucocorticoid for 4-6 weeks. A smaller percentage of these individuals (4-6%) experience a large increase in intraocular pressure (>15 mmHg). This IOP elevation usually progresses over the course of weeks to months of therapy and generally reverses after discontinuation of the glucocorticoid administration, although there are reports of irreversible IOP elevation[6]. Most of the patients in the study group did not complain of pain, headache, or halos around light with rise in intraocular pressure. One likely explanation could be the gradual rise in pressure that occurs in steroid induced glaucoma; therefore, like primary open angle glaucoma very few symptoms exist[6]. Few of them were aware of the decrease in vision, but they and the clinician they visited mistook it to be due to the corneal involvement in vernal catarrh.

Clark reported many features of steroid induced glaucoma that mimic primary open angle glaucoma (POAG).

Exact pathophysiology of steroid induced glaucoma is unknown. It is known that steroid induced IOP elevation is secondary to increased resistance to aqueous outflow. Some evidence indicates that the defect could be increased accumulation of glycosaminoglycan or trabecular meshwork-inducible glucocorticoid response (TIGR) protein, which could mechanically obstruct the outflow. Other evidence points toward corticosteroid induced cytoskeletal changes that could inhibit pinocytosis of aqueous humor or inhibit the clearing of glycosaminoglycans, resulting in accumulation of this substance. Glucocoticoid induced ocular hypertension can occur with a wide variety of routes of administration, including oral, topical, intraocular, periocular, nasal or inhalation. The propensity to induce ocular hypertension is dependent on the potency of the anti-inflammatory glucocorticoid, the frequency of administration, the dose and the duration of the treatment. If unrecognized, this steroid induced ocular hypertension can lead to secondary open angle glaucoma that in many ways mimics POAG[5]. Although the glucocorticoid induced ocular hypertension is generally reversible upon discontinuation of steroid therapy, the glaucomatous optic neuropathy is irreversible. It was found that when glaucoma did not reverse on cessation of steroids treatment, medical management was not sufficient in most cases to achieve the target pressures and surgical treatment was generally needed.

Features	**Steroid glaucoma**	**POAG**
Open angle	+	+
Reversibility of elevated IOP	+	-
Increased outflow resistance	+	+
Genetic component	+	+
Morphologic changes in trabecular meshwork including ECM deposition in trabecular	+	+

meshwork		

Trabeculectomy with antimetabolites was chosen as the standard treatment (mitomycin C was used in all). Indications for the use of anti-metabolite included younger age and conjunctival congestion in most of the patients. It remained an effective method of treatment in advanced steroid induced glaucoma.

Long term follow up proved trabeculectomy with MMC as an effective method for control of intraocular pressure in vernal catarrh patients. Improvement in vision postoperatively in seven patients was due to the resolution of corneal oedema seen on presentation due to raised intraocular pressure.

CONCLUSION

Individual counseling backed up by patient information leaflets is critical in breaking the cycle of inadequate treatment of VKC.

Treatment in VKC should be titrated against the severity of the disease, those with milder symptoms and no corneal involvement may be given mast cell stabilizers. These must be used three to four times daily, even when there are no symptoms to stabilize the mast cells and to prevent the release of histamine. They are of no value when symptoms occur because their effect is not immediate. If used well, they can limit or stop the use of steroids[7]. They do not have any of the side effects of steroids and can be used for prolonged periods.

Patients on topical corticosteroid therapy should receive follow up care at regular intervals by an ophthalmologist to monitor the ocular condition and intraocular pressures. Steroid induced IOP elevation typically occurs within 2 to 6 weeks of beginning of steroid treatment. Drugs that have the potential of inducing glaucoma should only be used if truly indicated; if drugs must be used IOP must be monitored closely. Untreated glaucoma can lead to permanent visual damage and blindness.

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