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Steroid Induced Glaucoma and Cataract in Case of Juvenile Allergies

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Abstract

Steroids are commonly used in the treatment of ocular allergies due to their potent anti-inflammatory effects. However, their misuse can lead to several serious complications, especially when used inappropriately or for extended periods. Some potential issues include: Increased intraocular pressure (IOP) leading to steroid induced glaucoma, Cataract formation, Delayed wound healing, Infection risk, and Dry eye syndrome. A 17-year-old male presented in eye opd with complains of diminution of vision in both the eyes for 4 months. Patient had been using topical eye drops containing dexamethasone along with ciprofloxacin for 4 years without follow up with the doctor for itching and redness in eyes. On examination his best corrected visual acuity was 5/60 and 2/60 for the right and left eye respectively. His intraocular pressure was 32 mm of hg and 36 mm of hg for the right eye and left eye respectively measured using Goldman applanation tonometer. On examination posterior subcapsular cataract was present and advanced glaucomatous cupping was seen in both the eyes. Patient underwent cataract extraction along with trabeculectomy in both the eyes.

1. Introduction

Cataract and glaucoma have been the leading causes of blindness worldwide. Age-related cataracts are responsible for 51% of world blindness, or approximately 20 million people [1]. The increasing use of corticosteroids in various medical conditions also increases the steroid-related complications of ocular concern, particularly steroid-related cataract and glaucoma, constituting the global burden of visual impairment worldwide. Steroid use is the fourth leading risk factor for secondary cataract and accounts for 4.7% of all cataract extractions [2].

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2. Case Report

A 17-year-old male resident of Rampur, District Shimla Himachal Pradesh India who is student of class 10th presented to Ophthalmology OPD at Indira Gandhi Government Medical College at Shimla Himachal Pradesh India with chief complain of Diminution of vision in both eyes for 4 months.

2.1 History of the present illness

The patient was apparently well 4 months back when he started complaining of diminution of vision both eyes which was insidious in onset and progressed over time. Diminution of vision was more for left eye.

There was no history of glare, polyopia, coloured halos, watering, pain, headache, any prior history of trauma, previous eye surgery, spectacle use and no other eye complaints.

2.2 Past medical history

There is history of redness and itching both eyes four years back for which patient was taken for consultation at regional hospital Rampur by his father. According to the patient's father his visual acuity was normal, and he was prescribed topical medication. Patient used the medication for four years for similar complain and had used over 50 vials in due course of time by purchasing the medication from the medical store without follow up with the doctor. Topical medication procured from the patient consists of Ciprofloxacin and Dexamethasone combination.

There is no history of Diabetes mellitus, hypertension, asthma, joint pains, tuberculosis.

There is no significant family history.

He is non-smoker and non-alcoholic, follows a vegetarian diet, enjoys sound sleep, and has normal bowel and bladder habits.

His general physical examination and systemic examination was normal and vitals were in normal limit.

3. Ocular Examination

Patient had normal head posture with parallel visual axis and symmetrical facial features.

His best corrected visual acuity for right eye was 5/60 and for left eye best corrected visual acuity was 2/60 using snellen's chart. Ocular movements were normal in all cardinal positions.

Eyelid and adnexa were grossly normal. The cornea of both eyes were normal in shape, surface, size, sheen and transparency. Anterior chamber depth was grade 4 according to the Van-herick grading system. Normal pattern of iris present in both eyes. Right eye pupil was single, central both direct and indirect reflexes were present while in left eye single mid-dilated pupil was

present with Relative afferent pupillary defect (RAPD) grade-3. Posterior subcapsular cataract was seen while examining the lenses in both eyes. Intraocular pressure in the right and left eye was 32 mm of hg and 36 mm of hg respectively recorded with Goldman applanation tonometer. Anterior chamber angle was grade 4 in both eyes with shaffer system of grading.

Fundus was examined optic cup disc ratio for the right and left eye was 0.8:1 and 0.9:1 respectively. Neuroretinal rim was circumferentially thin and nasalization of vessels and bayoneting was present in both the eyes. The foveal reflex was dull in both the eyes.

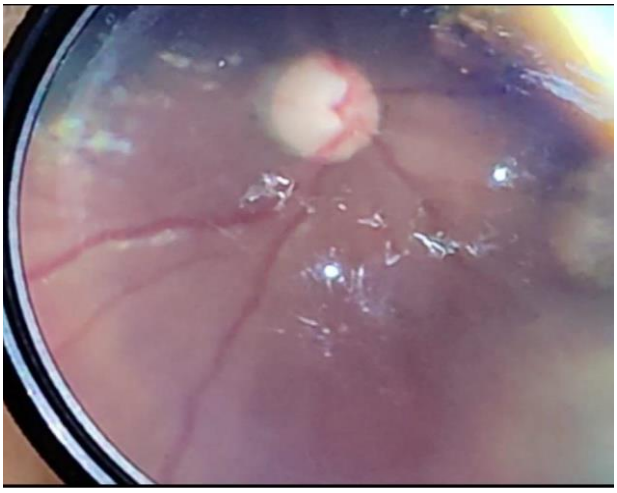

FIG. 1. **Left eye fundus image showing advanced glaucomatous cupping with cup disc ratio of 0.9:1.**

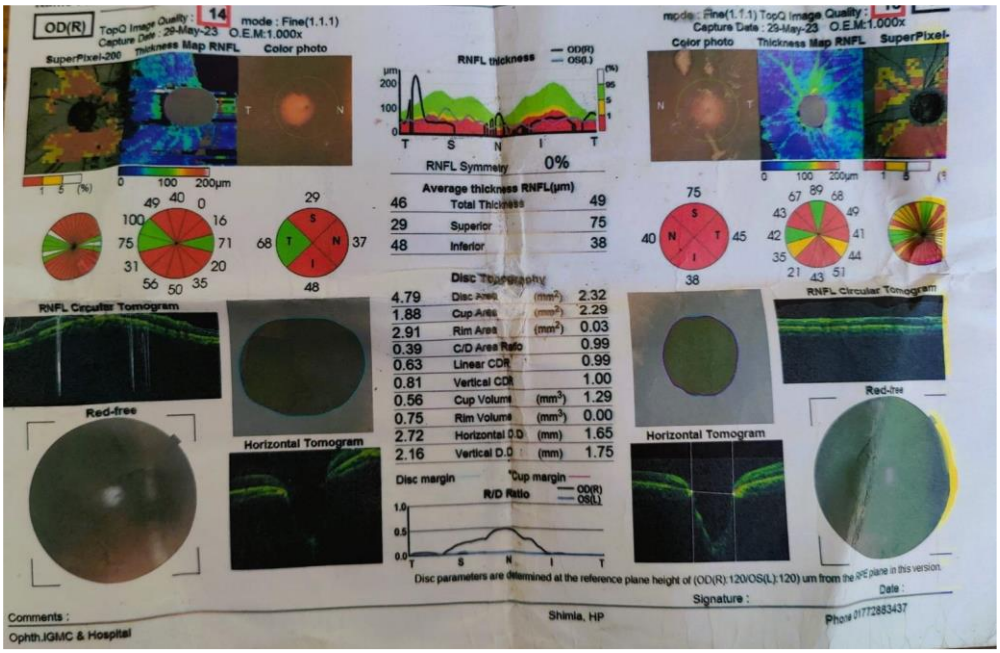

FIG. 2. **OCT image of both the eyes showing Increased cup disc ratio in both eyes and retinal nerve fiber layer (RNFL) loss in superior, nasal and inferior quadrant in right eye and super, nasal, inferior and temporal RNFL loss in left eye. OCT image has poor reliability factor because of hazy media because of posterior sub capsular cataract.**

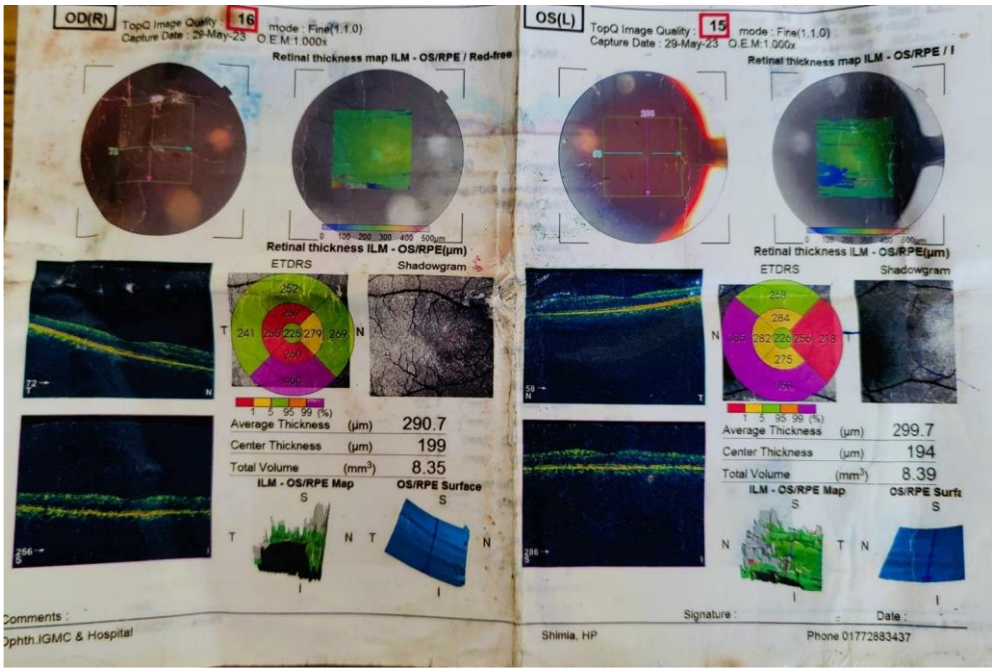

FIG. 3. **Visual field testing was advised to the patient but because of the presence of cataract the visual field charting was not reliable.**

4. Summary

A 17-year-old male presented with diminution of vision in both eyes for 4 months with history of chronic instillation of eye drops containing corticosteroid. On examination RAPD was present in left eye, IOP was 32 mm of hg for right eye and 36 mm of hg for left eye and posterior sub capsular cataract both eyes. Fundus examination showed advanced glaucomatous cupping in both the eyes.

5. Clinical Diagnosis

Diagnosis of secondary open angle glaucoma with posterior sub capsular cataract both eyes was made secondary to corticosteroid use.

6. Management

Topical eye drop containing steroid was immediately stopped. Patient was put on oral acetazolamide tablet 250 mg thrice a day along with topical anti-glaucoma drugs- combination of brimonidine tartrate with timolol maleate eye drops twice a day and prostaglandin analogue at bed time. Patient was followed up after 2 weeks and his intraocular pressure was 20 mm of hg and 24 mm of hg for right and left eye respectively. Patient was planned for combined extraction (cataract extraction with trabeculectomy) as the patient was of low socio-economic status and couldn't bear the cost of medication and resides in rural setting so patient may loss to follow up.

Post operatively patient's best corrected visual acuity was 6/24 in both the eyes and intraocular pressure was 12 mm of hg and 14 mm of hg in right and left eye respectively.

7. Discussion

Steroid-induced glaucoma is a form of secondary open-angle glaucoma occurring as an adverse effect of corticosteroid therapy [3]. It is usually associated with topical steroid use, but it may develop with oral, intravenous, inhaled, and periocular steroid administration by causing a decrease in aqueous outflow facility. The association of steroids and glaucoma was first established in 1950 when systemic administration of the adrenocorticotrophin hormone was shown to increase intraocular pressure (IOP) [4].

Corticosteroids cause elevation of the IOP by decreasing the facility of aqueous outflow [5,6]. Steroid specific receptors on the trabecular meshwork cells may play a role in the development of steroid-induced glaucoma [7]. Recent research has elucidated the possible role of genetic influences in the pathophysiology [8].

The main mechanism of action of steroids that is responsible for glaucoma is their membrane stabilizing action [9]. Hyaluronidase sensitive glycosaminoglycans (mucopolysaccharides) are normally present in the aqueous outflow system. These glycosaminoglycans in the polymerized form may undergo hydration producing a "biologic edema". Hence, these are constantly degraded by the hyaluronidase within the lysosomes of the goniocytes. The steroids stabilize the lysosomal membrane of the goniocytes and thus lead to an accumulation of polymerized glycosaminoglycans in the trabecular meshwork, producing an increased outflow resistance [10,11]. Glucocorticoid administration increases expression of collagen, elastin, and fibronectin within the trabecular meshwork and induces expression of sialoglycoprotein [12]. Another mechanism proposed is that steroids inhibit phagocytosis by the endothelial cells lining the trabecular meshwork. This leads to an accumulation of debris within the meshwork [13]. There is also extracellular deposition of fingerprint like material. Steroid use decreases expression of extracellular proteinases including fibrinolytic enzymes and stromolysin. A decrease in the synthesis of prostaglandins by corticosteroids, that regulate aqueous facility has also been proposed as one of the mechanisms leading to increase in IOP [14].

While the mechanism behind steroid induced cataracts is elusive, the most plausible hypothesis involves non-enzymatic formation of Schiff base 8 intermediates. The Schiff base intermediates are formed between the steroid C-20 ketone group and nucleophilic groups. After the formation of Schiff base intermediates, Heyns rearrangement of the adjacent C-21 hydroxyl group occurs which results in stable anime-substituted adducts [15]. The type of cataract that is typically steroid-induced is a posterior sub capsular cataract, which is formed in the posterior portion pf lens [16]. A mean daily dose of ICS exceeding 1000mcg over a period of 12 months was associated with a substantial increase in the risk of cataract [17].

8. Conflicts of Interest

There are no conflicts of interest.

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