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A Case Of Steroid Induced Cataract In A Exogenous Cushing Syndrome Secondary To Oral Prednisone For Juvenile Idiopathic Arthiritis

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Abstract
Glucocorticoid therapies are extensively used in a wide range of medical conditions including respiratory, allergic, inflammatory and autoimmune diseases. Inappropriate use of therapeutic doses of glucocorticoids can lead to many adverse effects including hypothalamic pituitary axis suppression and Cushing's syndrome. Cushing's syndrome is a rare entity in children. Adrenal tumour is the common cause of this syndrome in young children, whereas, iatrogenic causes are more common among older children. In this report, we present a nine-year-old boy of exogenous Cushing syndrome due to prolonged use of oral prednisolone for JIA who also developed posterior subcapsular cataract.

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I. Introduction

Posterior subcapsular cataracts is one of the important presentations of prolonged use of oral corticosteroids. It is of major importance in pediatric age group.

Steroid induced cataracts were first described in 1975 in patients with long standing Rheumatoid arthritis, however, they were usually secondary to steroid given for long term treatment rather than the disease itself. (1) These cataracts are the posterior subcapsular types and likely to produce visual symptoms in early stage of development.

Cushing syndrome, an endocrinological disorder resulting from abnormally high plasma cortisol levels, is characterized by increased free plasma glucocorticoids level [1]. It can result from either endogenous release of excess steroids (e.g., adrenal gland tumors, adrenal hyperplasia, or pituitary adenoma) or exogenous use of corticosteroid medications such as iatrogenic Cushing syndrome (2)

Clinically, patients with Cushing syndrome present with facial plethora and edema, giving the appearance of “moon face”; accumulation of fat in the supraclavicular area and upper trunk, giving the appearance of “buffalo hump;” accumulation of fat around the waist leading to truncal obesity, which leads to skin fragility resulting in appearance of purple stria, particularly on the abdomen, arms, and upper thighs. Furthermore, it causes hirsutism, skin bruises, ecchymoses, delayed wound healing, proximal muscle wasting, arterial hypertension, hyperglycaemia, and retarded growth (3).

The development of iatrogenic CS is dependent on dose and duration of steroid therapy. Because iatrogenic CS is medication dependent, the process to reverse the excessive cortisol exposure is accomplished by tapering the dose with the aim of discontinuing the medication, if possible.

The chronic use of corticosteroids is considered a widely prescribed medication for a variety of chronic conditions. However, when it is used for a prolonged period, it can result in the presentation of the systemic effects characteristic of Cushing syndrome. Moreover, this particular group of patients is subjected to develop tertiary adrenal insufficiency following the decrease or the elimination of corticosteroids. The mechanism through which this can occur is through the negative feedback loop on the hypothalamic-pituitary-adrenal (HPA) axis, which involves mainly corticotropin hormone (CRH) and adrenocorticotropic hormone (ACTH) release. Therefore, the reactivation of CRH is a necessity for the successful reactivation of the HPA axis after the elimination of chronic corticosteroid treatment. (4)

II. Case Report

A 9-year-old boy presented to us in Ophthalmology department with complaints of diminution of vision for distance for 6 months. His father gave history of difficulty in seeing the black board in the class as the teacher

complained to him regarding the same. The diminution of vision was progressive in nature. There was no history of using glasses.

In the past medical history, the child was diagnosed with Rheumatoid arthritis (RA factor positive) at the age of 5 years and was undergoing treatment. He has been on oral corticosteroid since long time.

The child was first born at term by normal vaginal delivery and birth weight of 3 kg. there was no history of jaundice/NICU stay/fever/seizures on birth.

Physical examination revealed a chubby boy with moon face, buffalo hump, protruding abdomen, increase body hair which were the features of cushing syndrome. [figure1]

Upon asking his father told the particular body features developed after 3 years of taking treatment for JIA.

The child also had a swollen, stiff and painful joints especially knee and fingers. [figure 2] His height was 110 cm, weight was 40 kgs. His younger sibling was in good health and other family members did not have any metabolic or similar problems.

Pediatric opinion was taken for the child and the diagnosis of exogenous cushing syndrome secondary to use of oral prednisolone for JIA was made. Although the cortisol level was within normal limits on presentation.

On ocular examination, his visual acuity was counting finger 3 meters in both eyes. The intraocular pressures were 12 mmhg and 14 mmhg in right and left eye respectively on Non-Contact Tonometer. The ocular adnexa were normal. On Slit lamp examination, the anterior segment was normal except for lens which showed lenticular changes. On dilated examination with cyclopentolate, there was posterior subcapsular cataract in both eyes. [figure 3]

The retina was within normal limits both eyes.

OCT macula was done and was normal in both the eyes. Ultrasound A-scan biometry was performed. The axial length in Right eye was 23.14mm and left eye was 23.10mm. With SRK formula the intraocular lens power was measured. Right eye IOL power used was 21.00 Diopter and left eye 21.05 Diopter.

Various blood investigations including Complete blood count was done. Only RA factor was positive, rest tests were within normal limits.

The child was scheduled for cataract extraction with IOL implantation in both eyes under general anesthesia after taking the pediatric fitness. Phacoemulsification with implantation of foldable hydrophobic monocular lenses was performed in both eyes. The posterior capsular rhexis was also performed. Post surgery patient was kept in pediatric ICU under observation for 24 hours. There was no fever.

Postoperatively, eyedrop moxifloxacin 0.5% 6 times for 1 week, eyedrop Prednisolone in weekly tapering dose starting from 6 times, eyedrop Nepafenac 3 times for 1 month and eyedrop Atropine 1 % 2 times for 1 week were started.

On 1st Post operative day, on SLE, the anterior chamber was quiet and well formed, the IOL was in situ. The patient was not cooperative for visual acuity on 1st day.

On postoperative 1 week, the anterior chamber was quiet and IOL was in place. The intraocular pressure was within norma limits. Visual acuity was 20/40 in both eyes. Patient was advised to use the medications as per schedule.

On follow up after 1 month, the ocular examination was normal. The visual acuity was 20/40 in both eyes.

III. Discussion

Studies have shown that systemic steroid therapy is a risk factor for the development of cataract in humans. (5)

The child presented in this article developed iatrogenic Cushing syndrome after prolonged use of oral steroid for JIA. The child also developed one of the ocular complication of steroids in the form of Posterior subcapsular cataract.

There have been many hypotheses on how steroids induce cataract formation. Cotlier proposed that steroids gain entry into the fiber cells of the crystalline lens and then react with specific amino acid groups of lens crystalline. These alterations free protein sulfhydryl groups from the disulfide bonds leading to protein aggregation and lens opacification. (6)

The clinical presentation of the child was typical for a case of Cushing syndrome including the signs of moon face, generalized edema, and more weight in contrast to his age.

It is well established that the risk of development of CS due to exogenous administration of corticosteroids is not uncommon. It has been seen in several different settings where steroid is the mainstay of treatment, especially in chronic diseases. The development of Cushing's features depends on duration and dose of steroid administration.

It is also known that, intermittent steroid therapy, which causes less hypothalamic pituitary adrenal axis suppression, may result in a lower incidence of Cushingoid changes. However, in this case the likelihood of developing such changes remains dose and frequency dependent. This occurrence highlights the plausibility of cumulative dose effect of intermittent oral glucocorticoid administration and the risk of iatrogenic Cushings.

Prolong use of steroid is one of the common causes for development of cataracts in children.

IV. Conclusion

The use of systemic and local Corticosteroids for a long time can make us face a lot of side effects. In such conditions we recommend the patients which use Corticosteroids to make at least once a year an eye check.

Rheumatologists, Endocrinologist and family doctors must be careful for the use of Corticosteroids with the right doses and time, keeping into consideration the side effects in Ophthalmology. Any physician prescribing corticosteroids should be aware of potential ocular side effects and should advise patients accordingly. Steroids are invaluable agents in the treatment of a diverse spectrum of disorders; however, their use is not without risks. The risk/benefit ratio of corticosteroid therapy can be improved by proper use of corticosteroids.

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Figures

Figure 1.

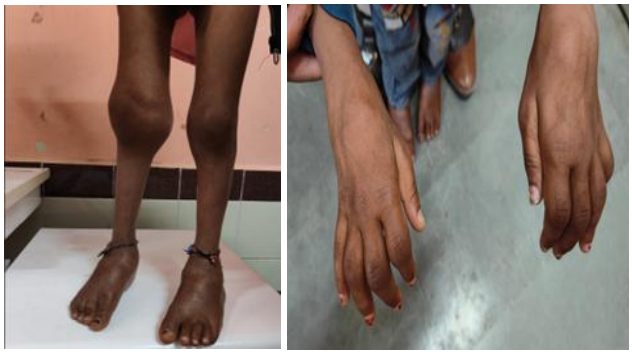

figure 2.

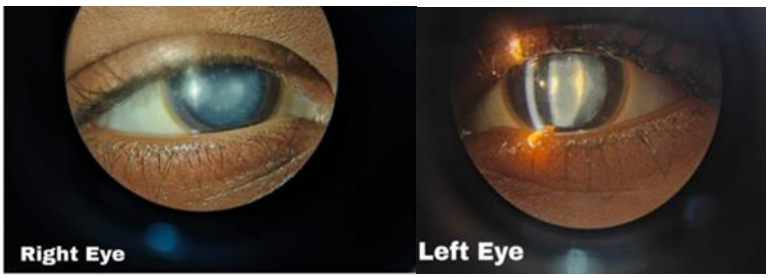

Figure 3