Print ISSN: 2319-2003 | Online ISSN: 2279-0780

IJBCP International Journal of Basic & Clinical Pharmacology

DOI: http://dx.doi.org/10.18203/2319-2003.ijbcp20161587

Case Report

Steroid induced central serous retinopathy following follicular unit extraction in androgenic alopecia

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Received: 22 February 2016
Accepted: 21 March 2016

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ABSTRACT

Dermatologists for various conditions and procedures commonly use corticosteroids worldwide. The development of central serous retinopathy is a lesser known complication occurring in <10% of the cases with steroid use. This case report highlights the development of central serous retinopathy after prescribing low dose of prednisolone 20 mg per day for androgenic alopecia during post-surgical follicular unit extraction (FUE) surgery follow-up that recovered spontaneously after gradual withdrawal of steroids. Therefore, awareness is required for its early detection and management as it has a potential of causing irreversible visual impairment.

Keywords: Hair transplantation surgery, Optical coherence tomography, Central serous retinopathy, Prednisolone, Hamilton anxiety rating scale

INTRODUCTION

Central serous retinopathy (CSR) is an idiopathic retinal disorder characterized by neuro-sensory detachment of the retina affecting the macula triggered by systemic steroids. Incidence of CSR is 9.9 per 1,00,000 for men and 1.7 for women having male predominance of 8:1 to 10:1 over females.[1] Racial variations in the prevalence of CSR have been reported, with high rates in Caucasians and Hispanics, higher rates in Asians and lower rates in African-Americans.[2,3] Most of these cases are asymptomatic and commonly present between the age group of 25-55 years. The acute manifestation of CSR is characterized by fluid accumulation in the sub retinal space or retinal pigment epithelium. Usually, one eye is involved, but bilateral involvement occurs in 20% of patients.[4]

The exact mechanism of CSR is not understood. It is thought to be associated with Type A personality, elevated levels of glucocorticoids levels, other factors that are associated with CSR include the usage of systemic steroids, antibiotics, antihistamines, pregnancy, autoimmune diseases, untreated hypertension, alcohol and tobacco, sildenafil, tadalafil, H. pylori infection, bone marrow and solid organ transplantation.[5-10] The clinical presentation is of a sudden onset of blurring of vision along with the complaints of micropsia (objects appear smaller), metamorphopsia (objects appear distorted), moderate to severe impairment of visual acuity, central

relative scotoma, hyperopic shift in refraction and colour and contrast sensitivity reduction.

This case report highlights the development of central serous retinopathy in a middle-aged male after 20 mg of prednisolone after post-surgical follow-up period of follicular unit extraction (FUE) surgery for androgenic alopecia. The visual acuity of the patient gradually resolved after withdrawal of offending drug within 2 weeks.[11]

CASE REPORT

A 41 years male, voluntarily underwent FUE surgery after giving his written informed consent. He presented with symptoms of sudden onset painless diminution of vision in left eye especially on fifth day during post-surgical follow-up period of FUE. His others complaints were seeing black spot or central shadow, metamorphopsia and headache. The best corrected visual acuity (BCVA) was 20/20 oculus dexter and 20/40 oculus sinister with refractive error of -1.0D Sph and -0.5D Sph respectively. There was mild hyperopic shift of left eye by+0.5D. The anterior segments of both the eyes were normal. Fundus examination of right eye was unremarkable but left eye showed sub retinal fluid temporal to the fovea (Figure 1).

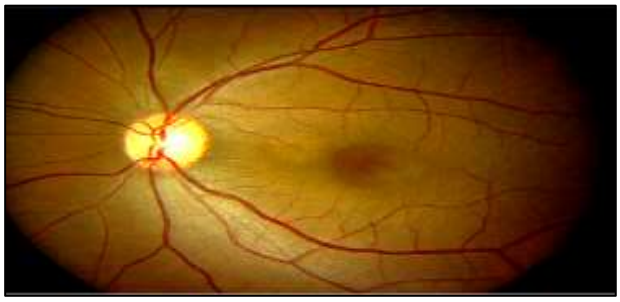

Figure 1: Sub retinal fluid underneath fovea.

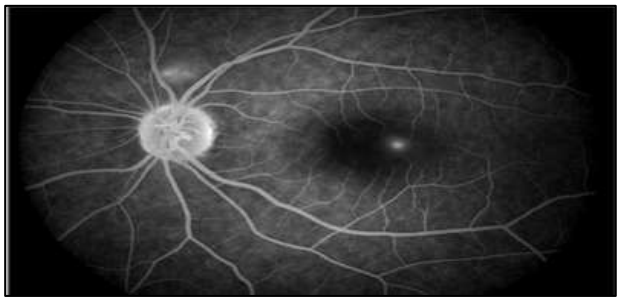

Figure 2: A focus of ink blot leakage in late phase of fluorescein angiography

The intraocular pressure at the time of examination was normal. This case did not have any known risk factors e.g., local, systemic diseases or ophthalmic conditions that could have predisposed him to CSR. There was no past or family history of any such complaints. Mental status examination showed that he was conscious, orientation to time, place and person. No history of any mood, thought disorder, perception, and higher mental status examination was within normal limits. The assessment for personality type was done on a modified version of the Jenkins activity survey, which showed a score of 70, indicating that he was not having type A personality.[12] Then patient was administered Hamilton anxiety rating scale (HAM-A) for assessment of underline anxiety where his scores were 7 on 5th day (baseline) and 9 after (2 weeks).[13] Clinical global impression-severity (CGI-S) score was 2 and 1 and clinical global impression-improvement (CGI-I) scores was 4 and 1.[14] The efficacy index was 2, indicating very much improved after withdrawal of steroid. Causality assessment on WHO-UMC scale delineates prednisolone as the "probable cause". Hence, the provisional diagnosis was of CSR. Fluorescein angiography imaging's of left eye showed pinpoint focus of early dye leakage within the region of sub retinal fluid which increased in late phase (Figure 2). Fourier domain optical tomography showed a serous detachment of macula with no break in the retinal pigment epithelium confirming the diagnosis of CSR (Figure 3).

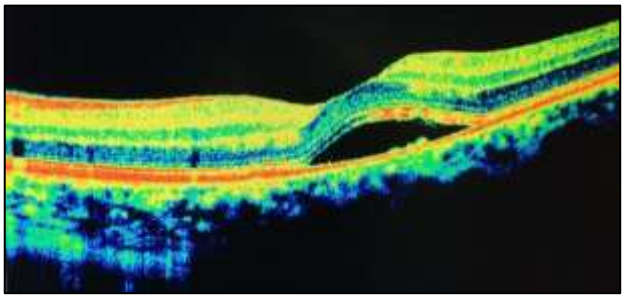

Figure 3: OCT Left eye showing serous fluid underneath fovea with intact retinal pigment epithelium.

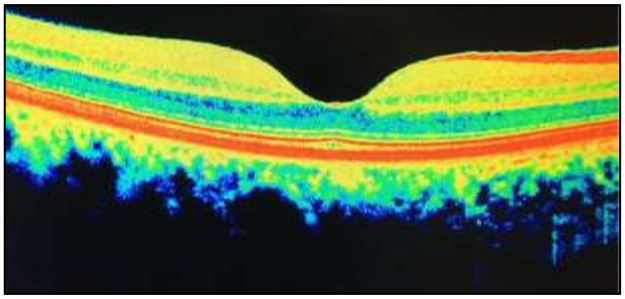

Figure 4: OCT Left eye showing regression of serous fluid with normal foveal contour.

The parafoveal temporal thickness of left eye was 368 µm while it was 229 µm for right eye His medications included 20 mg/day of prednisolone. Prednisolone was rapidly tapered off in a period of seven days. Nepafenac eye drops (0.1%) thrice daily along with antioxidant capsules containing L-arginine and vitamin E were prescribed. His vision recovered completely during the follow-up period, which was conducted initially weekly for two weeks, then at a monthly interval for three months with no further complaints. OCT was repeated at

an interval of 4 weeks of follow-up, which showed complete regression of sub retinal fluid (Figure 4).

DISCUSSION

Glucocorticoids by various routes (oral, intravenous, intranasal, inhalation, topical intra articular and epidural) are known to cause CSR probably by increasing cAMP in REP (retinal pigmented epithelial) cells changing the ionic pump function or by alternating the permeability of blood aqueous barrier and disrupting the outer blood retinal barrier.[15,17,18] They are known for causing an increase in catecholamine levels thus, making choroidal vasculature more susceptible to it (i.e., hyperpermeable choroid).[19] They can also induce hypercoagulability and increased platelet aggregation. Increased coagulation and platelet aggregation appear to explain the connection between steroid and the onset of CSR, through a focal transitory or permanent occlusion of the choroidal vasculature. The chain of events appears to be linked to an increase in plasminogen activator inhibitors-1 (PAI-1).[20,21]

In the present case, the patient was prescribed steroids following FUE surgery. Symptoms appeared on fifth day while he was on steroid. Diagnosis was based on the ophthalmoscopic findings. Fluorescein angiography and OCT helped in confirmation of CSR. His symptoms abated after the steroids were gradually withdrawn. The time course of the onset of ADR and response to steroid withdrawal probably suggest role of prednisolone as "Probable cause" on causality assessment WHO-UMC scale.[22] The preventability assessment was carried using modified Schumock and Thomton criteria, which showed that ADR was not preventable.[23]

The daily dose of steroids has greater influence in onset than the total dose. The latency time is shorter with higher doses and recurrences occur earlier when the dosage of steroids are increased further. The patients are often older in steroid induced than the patients with idiopathic type of CSR.[16] In this case, patient was aged 41 years. The visual prognosis of CSR is usually good and reversible if detected earlier and aggravating factors are withdrawn in time except in chronic, recurrent cases or in cases of bullous CSR. Most of the cases (80-90%) undergo spontaneous resorption of the subretinal fluid within three to four months in most cases of unilateral CSCR.[24] In persistent or chronic cases photodynamic therapy may be helpful.[25] Other treatment options for persisting and chronic forms include low dose aspirin, intravitreal bevacizumab, and systemic ketoconazole. Lim et al showed no positive effect of bevacizumab over control group in his study.[26] Golshahi et al in his clinical trial refuted the role of systemic ketoconazole.[27]

CONCLUSION

The physicians and surgeons commonly use corticosteroids as a treatment for various diseases and procedures. The development of central serous retinopathy occurring after FUE is a lesser known complication. This case report suggests that CSR can occur with any form of steroid administration. Early remission and recovery is usually expedient with discontinuation of exacerbating factors, including steroids. Therefore, awareness is required among specialists and patients of a potentially irreversible visual loss due to steroids if left untreated.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Raj RT, Raj R, Raj A. Steroid induced central serous retinopathy following follicular unit extraction in androgenic alopecia. Int J Basic Clin Pharmacol 2016;5:1152-5.