BRIEF COMMUNICATION

Short-Term Steroid-Induced Central Serous Chorio-Retinopathy in a Patient with Laser Foveal Burn

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ABSTRACT
Oral intake of steroids has been associated with central serous chorio-retinopathy (CSCR) for long time. We report a 23 years old male who had exposure to green laser pointer after which he developed laser maculopathy. The treating ophthalmologist started oral steroids with patient developing CSCR in his left eye. He was referred to our retina clinic for further evaluation. On examination his vision was 6/6 part in his right eye and 6/9 part in his left eye. His dilated fundus examination revealed small foveal scar in his right eye and dull foveal reflex in his left eye. On OCT he had laser maculopathy in the right eye and left fundus findings coincided with diagnosis of central serous chorio-retinopathy (CSCR). Patient was called for review after 3 weeks with instructions to discontinue his deltacortil tablets. His vision improved and sub retinal fluid was absorbed. Later he went back to his primary ophthalmologist who restarted deltacortil tablets and he presented to us with recurrence of CSCR.

Key Words: Maculopathy, burn, Steroids, Central Serous Chorio-retinopathy.

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INTRODUCTION
Central serous chorio-retinopathy (CSCR) is characterized by elevation of neuro sensory retina at the posterior pole. In majority of the cases, the cause of this condition remains unknown. However, CSCR has been associated with intake of steroids taken in oral, inhaled and topical form.¹ We report a 23-year old male who developed CSCR following 2 weeks intake of oral steroids.

CASE PRESENTATION
A 23-year old male had exposure to green laser pointer and complained of blurred vision. He went to see his local ophthalmologist who diagnosed laser maculopathy in his right eye and started oral Prednisolone (Deltacortil) 50 mg/day in divided doses. After 2 weeks of intake of Deltacortil tablets, the patient noticed blurred vision in his left eye. He was referred to our retina clinic for further evaluation. On examination, his vision was 6/6 partial in his right eye and 6/9 partial in his left eye. Anterior segments were unremarkable with intraocular pressures (IOP) of 12 mm Hg in each eye. His dilated fundus examination revealed small foveal scar in his right eye and dull foveal reflex in his left eye (Figure 1). Vitreous was free of any inflammatory cells. Optical coherence tomography (OCT) with macular map of 6.6 mm x 6.6 mm showed rectangular outer retinal defect, focal loss of IS-OS/ Ellipsoid Layer with intact external limiting membrane (ELM) in his right eye. The OCT of his left eye revealed presence of sub-retinal fluid over posterior pole (Figure 2). Diagnosis of laser maculopathy and central serous chorio-retinopathy was made in the right and left eye respectively. Patient was asked to discontinue Deltacortil tablets.
The patient was again reviewed after 3 weeks. His visual acuity was 6/6 partial in right eye and 6/6 in the left eye. The OCT of his right eye showed persistent outer retinal defect but left fundus showed disappearance of sub-retinal fluid (Figure 3). The patient was advised to return after 4 weeks with no treatment. The patient went back to his primary ophthalmologist who restarted Deltacortil tablets 40 mg/day. He again developed blurred vision in his left eye and came back to our retinal clinic. OCT of right eye showed persistent IS/OS loss with very mild shrinkage of focal outer retinal disruption, while left fundus showed presence of small sub-retinal fluid (Figure 4).

![Fig. 1: Fundus photographs of Right & Left Eye.](image1.png)

![Fig. 2: Optical Coherence Tomography of right and left macula with thickness map. Right Eye showed focal loss IS-OS/ Ellipsoid zone with intact external limiting membrane (ELM), left eye central serous chorioretinopathy.](image2.png)
DISCUSSION

CSCR is a self-limiting disease with spontaneous resolution occurring within 3–4 months of initial episode resulting in good visual outcome.² Observation alone with no treatment is therefore advised as the first line approach in the newly diagnosed cases. However, risk factors such as increased stress score, raised homocysteine and serum cortisol level should be addressed.³ CSCR has been associated with intake of oral steroids and also described in patients with Cushing disease, pregnancy and stress with endogenous high level of cortisol secretion.⁴ The exact role of glucocorticoids in pathogenesis of CSCR is not known but possible
mechanism includes increased capillary fragility and hyper-permeability leading to choroidal decompensation with leakage of fluid in the sub-retinal space. The exact duration of steroids intake which may result in CSCR is not known. However, the patients on chronic deltacortil therapy such as kidney transplants are at higher risk.

We report a 23-year old man taking Deltacortil for 2 weeks resulting in CSCR with fluid accumulation in sub-retinal space of his left eye. This was clearly demonstrated on OCT of his left eye. CSCR recovered after discontinuation of oral steroids and recurred with re-commencement of Deltacortil therapy.

A similar patient is described by Alkin and coworkers. They had a 54-year old male with decreased vision in his right eye diagnosed with non-arteritic anterior Ischemic optic neuropathy. He was treated with intravenous methyl prednisolone 1 gm/day for 3 days followed by oral steroids in dose of 1 mg/kg daily for 1 week. After 6 days of oral treatment with steroids, patient developed CSCR in his left eye. Grixty and Kumar published their case report of 67 years old female with biopsy proven giant cell arteritis with no visual symptoms. She was treated with intravenous methyl prednisolone for 3 consecutive days followed by 60 mg oral prednisolone daily. Five days after initiation of treatment, the patient complained of blurred vision in her left eye. Examination showed visual acuity of 6/18 with diagnosis of CSCR which was confirmed on OCT. Hardwig and colleagues have described three patients, two of whom received intramuscular corticosteroid injections and the third one received epidural corticosteroid injections 6 weeks prior to development of CSCR.

Our patient developed CSCR two weeks after taking oral steroids. Koyama and colleagues believed that the duration from the beginning of corticosteroid treatment to the onset of CSCR can range from three days to 23 years and in dose as low as 10–15 mg/day.

CONCLUSION

This case report reinforces CSCR as a potential side effect of oral intake of corticosteroids.

Conflict of Interest

Authors declared no conflict of interest.

REFERENCES


Authors’ Designation and Contribution

Nasir Ahmed Memon; Assistant Professor: Manuscript preparation.
Abdul Sami Memon; Assistant Professor: Data Analysis.
Israr Ahmed Bhutto; Associate Professor: Manuscript editing.
PS Mahar; Professor and Dean: Manuscript review.

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