

Central serous chorioretinopathy in a COVID-19-positive patient

Pradeep Kumar Panigrahi

Department of Ophthalmology, Institute of Medical Sciences and SUM Hospital, Siksha O Anusandhan (Deemed to Be) University, Bhubaneswar, Odisha, India

Abstract

A 37-year-old healthy male presented with sudden loss of vision in the left eye (LE) 1 week following starting oral steroids for COVID-19 complications. Best-corrected visual acuity in the left eye (LE) was 20/80, N12. Clinical and imaging tests led to a diagnosis of central serous chorioretinopathy in the LE. There was gradual resolution of neurosensory detachment following the discontinuation of steroid therapy. Two months following initial presentation, best-corrected visual acuity in the LE had improved to 20/20, N6.

Keywords: Central serous chorioretinopathy, COVID-19, oral steroids

Address for correspondence: Dr Pradeep Kumar Panigrahi, Associate Professor, Department of Ophthalmology, Institute of Medical Sciences and SUM Hospital, Siksha O Anusandhan (Deemed to Be) University, 8-Kalinga Nagar, Bhubaneswar 751 003, Odisha, India.

E-mail: doc.pkp25@gmail.com

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INTRODUCTION

Medical community has been greatly challenged by severe acute respiratory syndrome corona virus-2 (SARS-CoV-2) disease (COVID-19) pandemic. Although most diagnostic and therapeutic efforts have been mainly aimed at treating respiratory complications, several ocular manifestations of the disease have recently emerged.^[1,2] We report an interesting case of central serous chorioretinopathy (CSC) developing in a COVID-19-positive patient.

CASE REPORT

A 37-year-old male presented with sudden-onset painless loss of vision in the left eye (LE) of 10 days duration. The patient gave a history of fever, sore throat, cough, diarrhoea and myalgia 3 weeks back. There was a contact

history with a COVID-19-positive relative in the family. Reverse transcription-polymerase chain reaction test for nasopharyngeal sample tested positive for SARS-CoV-2. The patient underwent high-resolution computed tomography chest 6 days following the start of the symptoms which showed multiple ground-glass opacities in bilateral lung fields suggestive of atypical viral pneumonia. Blood investigation reports were erythrocyte sedimentation rate (ESR) 80 (0–25) mm at the end of the first hour, serum ferritin 200 (10–160) ng/mL, serum lactate dehydrogenase 420.8 (313–618) U/L, D-dimer 400 (0–500) ng/mL, interleukin-6 28.2 (0–6.4) pg/mL and C-reactive protein 85.04 (0–6) mg/L. At this stage, the patient was started on oral steroids. Complete list of medications taken by the patient is mentioned in Table 1. The patient developed painless loss of vision in the LE 1 week after starting oral corticosteroids. There was no history of any vision loss before this episode.

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Table 1: Medications taken by the patient during the course of the disease

| Name of the drug | Route | Dosage | Duration (days) |
|--------------------------------|-------|----------------------|-----------------|
| Doxycycline | Oral | 100 mg (twice-daily) | 5 |
| Azithromycin | Oral | 500 mg | 5 |
| Ivermectin | Oral | 12 mg | 5 |
| Vitamin C | Oral | 1 g | 7 |
| Montelukast/ Levocetirizine | Oral | 10 mg/5 mg | 7 |
| Pantoprazole | Oral | 40 mg | 21 |
| Acetylcysteine | Oral | 600 mg | 7 |
| Deflazacort | Oral | 12 mg | 7 |
| Deflazacort | Oral | 6 mg | 10 |

On examination, best-corrected visual acuity (BCVA) was 20/20, N6 in the right eye (RE) and 20/80, N12 in the LE. The anterior segment was normal bilaterally. Intraocular pressure was 14 and 15 mm Hg in the RE and LE, respectively. The pupillary reaction was normal in both eyes. Fundus examination of the RE showed clear media, normal disc and pigmentary changes in the macular area (Figure 1a). Funduscopy of the LE revealed an elevated area of neurosensory retinal detachment in the macular area (Figure 1b). We diagnosed it as a case of CSC in the LE. Spectral-domain optical coherence tomography (OCT) scan of the RE showed retinal pigment epithelial detachment (PED) temporal to foveal centre (Figure 2a). Neurosensory detachment involving the foveal centre was noted on OCT scans of the LE (Figure 2b).

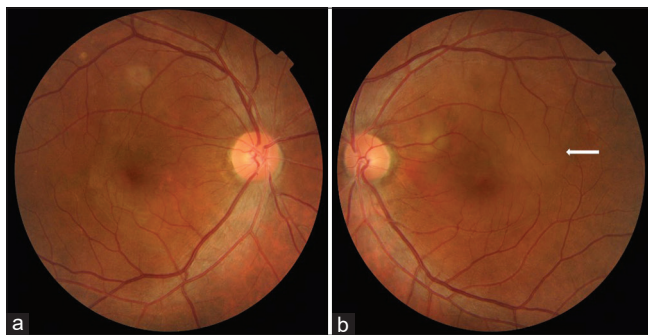


Figure 1: Colour fundus photograph of the right eye showing pigmentary changes in the macular area (a). Colour fundus photograph of the LE showing neurosensory detachment in macular area (white arrow) (b) LE = Left eye

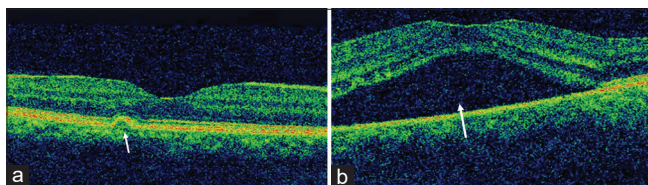


Figure 2: OCT image of the right eye showing small retinal pigment epithelial detachment temporal to foveal centre (white arrow) (a). OCT scan of the left eye showing neurosensory detachment involving the foveal centre (white arrow) (b) OCT = Optical coherence tomography

Fundus fluorescein angiography showed 1 and 2 points of inkblot pattern leakage in the RE and LE, respectively. All leakage points were more than 500 μ away from the foveal centre. There was increase in both size and intensity of the lesions as the angiography progressed. Multiple areas of PED, which increased in intensity without increase in size as angiography progressed, were noted bilaterally (Figure 3). At the time of ocular presentation, blood investigations report was ESR 40 mm, serum ferritin 60 ng/mL, D-dimer 399.4 ng/mL, interleukin-6 1.18 pg/mL and C-reactive protein 1.80 mg/L. The patient was given the option of undergoing focal laser to the leaks. Pros and cons of the procedure were discussed with the patient. The patient did not wish to undergo any intervention at that point of time. Because all inflammatory biomarkers were within the normal limits, we had a discussion with the patient's physician to discontinue oral steroids and the patient was asked to come for review after 1 month.

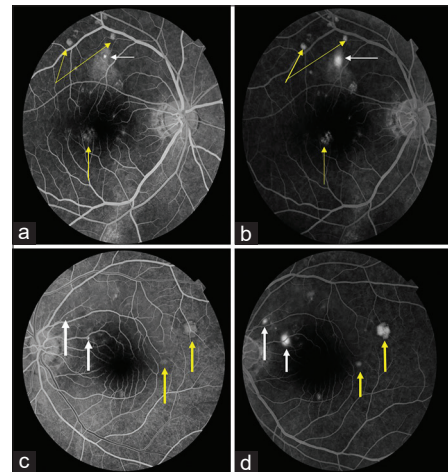


Figure 3: Early-phase FFA image of the right eye showing a single ink blot pattern of leakage (white arrow) with multiple PED (yellow arrows) (a). Late-phase FFA image showing increasing size and intensity of leakage (white arrow) and increasing intensity of PED (yellow arrows) (b). Early-phase FFA image of the left eye showing 2 ink blot pattern leakage point (white arrows) and multiple PED (yellow arrows) (c). Late-phase FFA image showing increasing size and intensity of leakage points (white arrows) and increasing intensity of PED (yellow arrows) (d) FFA = Fundus fluorescein angiography; PED = Pigment epithelial detachment

One month following presentation, BCVA in the LE had improved to 20/40, N8. OCT scans of the LE showed that the height of the neurosensory detachment had reduced. We decided to observe and asked the patient for a follow-up after 1 month. On the last follow-up, 2 months following the presentation BCVA in both the eyes was 20/20, N6. OCT scans of the LE showed complete resolution of subretinal fluid. OCT scans of the RE showed a slight increase in the height of PED, but

the patient did not complain of any blurring of vision or metamorphopsia (Figure 4). The patient has been asked to come on regular follow-ups and advised immediate ophthalmic consultation following any vision loss or metamorphopsia in either eye in future.

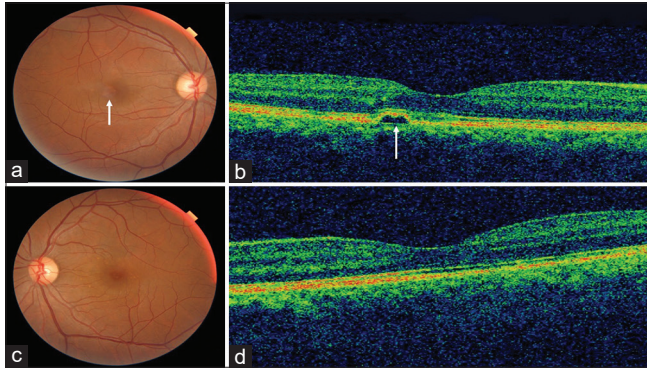


Figure 4: Colour fundus photograph of the right eye showing a PED (white arrow) temporal to foveal centre (a). OCT image of the right eye showing slight increase in height of PED (white arrow) with absence of any subretinal fluid (b). Colour fundus photograph of the left eye showing resolved exudative detachment at foveal centre (c). OCT image of the left eye showing complete resolution of neurosensory detachment (d)
OCT = Optical coherence tomography; PED = Pigment epithelial detachment

DISCUSSION

CSC is an idiopathic disease characterised by neurosensory retinal detachment with or without associated retinal PED, angiographic leakage at the level of retinal pigment epithelium (RPE) and choroidal hyperpermeability.^[3] The pathophysiology of CSC is still uncertain. Postulates for the mechanism include choroidal hyperpermeability and dysfunctional RPE pump mechanism.^[4] CSC has been associated with many risk factors such as male sex, Type-A personality, gastro-oesophageal reflux, pregnancy and use of psychotropic medications. The association of CSC and the above-mentioned risk factors remain controversial. Perhaps, the most widely accepted association is with the intake of corticosteroids.

Corticosteroids in any route can either cause or aggravate CSC. Steroids when used topically for dermatological conditions, intra-articular, intravenous, intramuscular, oral, intranasal and inhalation are all associated with CSC.^[5] Corticosteroid's intake can be associated with an increased choroidal thickness which can lead to CSC.^[6] Systemic steroid intake can also be associated with the development of systemic hypertension, which can lead to choroidal vascular dysregulation and increased choroidal hydrostatic pressure resulting in CSC.^[7]

Systemic corticosteroids are routinely being used to manage the pro-inflammatory complications arising out of COVID-19 infection.^[8] In our case, the patient tested positive for COVID-19 infection. The patient was started on systemic steroids after the elevation of inflammatory biomarkers. Visual symptoms started after the use of systemic steroids and there was the prompt resolution of neurosensory detachment following discontinuation of steroid therapy. The association of COVID-19 infection in our case seems more coincidental rather than being causal. CSC has been reported in a 42-year-old female COVID-19-positive patient who was treated with oral and inhalational steroids.^[9] CSC resolved following the discontinuation of steroids. Occurrence of CSC was documented in a 27-year-old female who was positive for COVID-19 and had been treated with oral steroids.^[10]

As the COVID-19 pandemic continues, increasing number of patients will be requiring systemic corticosteroid therapy. Ophthalmic manifestations can develop both when the patient is undergoing treatment and even after recovery from the disease. Physicians treating patients should be aware of such complications and should counsel patients to immediately approach their ophthalmologists if any visual symptoms develop.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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