

CASE REPORT

Bilateral combined central retinal artery and vein occlusion in a COVID-19 patient

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**Abstract**

To report a case of a male patient who gave history of fever, dry cough, and dyspnea, and he was tested positive for COVID-19 by reverse transcriptase-polymerase chain reaction from the nasopharynx. Presented with bilateral sudden onset of decreased vision due to combined central retinal artery and vein occlusion, the patient was treated with systemic medication including anticoagulant treatment and intravitreal bevacizumab, visual recovery was limited in spite of treatment.

Introduction

The incidence of thrombotic complications in patients with COVID-19 infection is elevated due to multiple factors and is associated with poorer outcomes.^[1,2] Hypercoagulability associated with COVID-19 has been described as a “sepsis-induced coagulopathy.”^[3] Such thrombotic events lead to retinal vascular occlusion. We report a rare case of combined retinal artery and vein occlusion in COVID-19 patient which has not been reported before.

Case Description

A 36-year-old man without any preexisting systemic comorbidities presented with fever, cough, and progressive dyspnea (SpO₂-88%). RNA reverse transcriptase-polymerase chain reaction from the nasopharynx was positive for COVID-19. After 2 days of initial symptoms, the patient developed severe breathlessness and the chest X-ray showed bilateral patchy middle and lower field pulmonary infiltrates. He was shifted to intensive care unit (ICU) for acute hypoxemic respiratory distress and received COVID-19-directed therapy including hydroxychloroquine, azithromycin, tocilizumab remdesivir, and pulse steroid therapy. He remained in the intensive care

for total 7 days and on stabilization, he was shifted from ICU to medical ward for further management, on the 13th day since the onset of COVID-19 symptoms patient complained of sudden onset of painless profound loss of vision in both eyes. On examination, both eyes had visual acuity of perception of light, pupillary reaction was ill sustained in both eyes. Anterior segment examination and ocular motility were normal. Dilated fundus examination of both eyes showed mild disk edema, attenuated arteries, and dilated veins with scattered peripapillary hemorrhages, cotton wool spots, and retinal hemorrhages in all the quadrants. Area of retinal edema with whitening noted over the posterior pole, with this diagnosis of combined retinal artery and vein occlusion was made [Figure 1a and b]. For further confirmation of diagnosis and management, the patient underwent optical coherence tomography (OCT) and fundus fluorescein angiography (FFA). FFA showed [Figure 1c and d] absence of flow in both retinal artery and retinal veins with associated large area of capillary non-perfusion over the posterior pole and retinal periphery. Vessel wall staining noted in few quadrants. OCT examination [Figure 2a and b] showed increased reflectivity in the inner retinal layers and diffuse macular edema (CMT RE-985 μ and LE-916 μ). Choroidal details could not be appreciated. The patient underwent work up for hypercoagulable state (hereditary or acquired thrombophilia

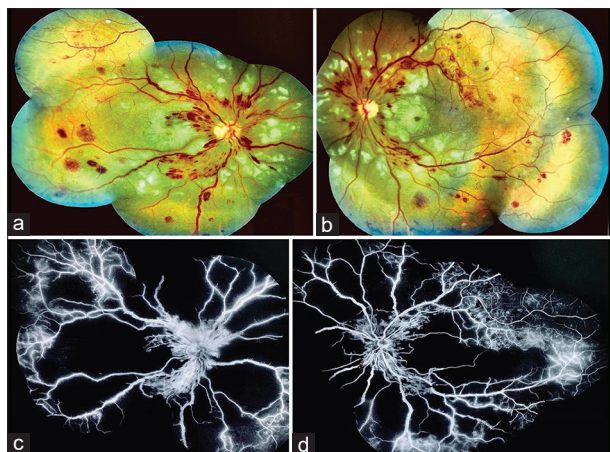


Figure 1: (a and b) Right eye and left eye fundus images showing mild disk edema, retinal hemorrhages, attenuated arteries, dilated veins, retinal edema with whitening. (c and d) Right eye and left eye fundus fluorescein angiography showing absence of dye flow in both arteries and veins with extensive areas of capillary non-perfusion

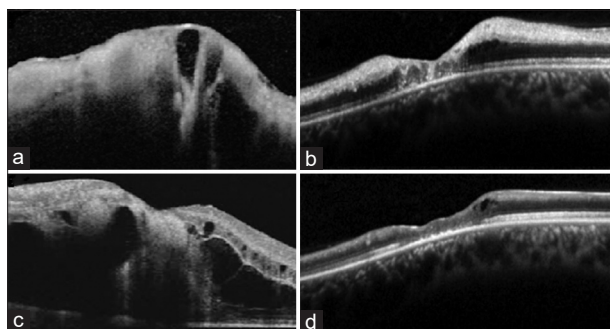


Figure 2: Pretreatment optical coherence tomography (OCT) (a and b) showing inner retinal layer thickening with gross macular edema, 8 weeks posttreatment OCT (c and d) shows retinal thinning with resolution of macular edema

factors), vasculitic syndromes, blood hyperviscosity, and other recognized systemic vascular inflammatory disorders. Laboratory work-up after the onset of visual symptoms revealed no pathological findings in blood analysis comprising blood sugar, lipid profile, homocysteinemia, anti-cardiolipin immunoglobulin (Ig)M and IgG antibodies, and screening for genetic thrombophilia (Factor V Leiden and prothrombin mutations, antithrombin III, and proteins C and S deficiencies), ANA, C-ANCA, P-ANCA, and ds DNA, but showed elevated Inflammatory markers; D-Dimer (>1050 ng/ml; normal range <255 ng/ml), serum ferritin (615.30 ng/ml; normal range 22–274 ng/ml), C-reactive protein (CRP) (13 mg/L; normal range <10 mg/L), and pro-calcitonin (0.16 ng/ml; normal ≤ 0.07 ng/ml). Coagulation profile showed; fibrinogen (546 mg/dl; normal range 200–400 mg/dl), prothrombin time (17.6 s; normal range 11.9–14.4 s), international normalized ratio (1.5; reference range 0.8–1.3), and partial thromboplastin

time (36 s; normal range 22–37 s). Hemogram showed mild lymphopenia ($0.66 \times 1000 \mu\text{l}/$) and thrombocytosis ($412 \times 1000 \mu\text{l}/$). Magnetic resonance imaging of the brain and orbits and computed tomography (CT) angiogram head and neck was normal. Transthoracic echocardiogram was unremarkable. Considering thrombotic microangiopathy as the cause for combined retinal vascular occlusion, the patient was started on anticoagulant enoxaparin 1 mg/kg twice daily therapy along with standard COVID-19 treatment protocol. The patient was treated with intravitreal anti-vascular endothelial growth factor (Bevacizumab, 1.25 mg/0.05 ml), and then, second intravitreal dose was given after 4 weeks and was asked to continue systemic medications including tapering oral steroids as advised by the physician. The patient was followed up at 1 week, 1 month, and 2 months. There was partial improvement in best corrected visual acuity in both eyes (1/60 both eyes) at the end of 2 months. At the last follow-up of 2 months, OCT of both eyes showed resolution of macular edema but associated retinal thinning [Figure 2c and d].

Discussion

There are no published reports of combined bilateral retinal artery and vein occlusion in COVID-19 patients and these patients are at risk of presenting venous and arterial thrombotic events. COVID-19 infection is associated with coagulation activation and a disproportionate systemic inflammatory response.^[4,5] Whether the coagulation cascade is directly activated by the virus or whether this is the result of local or systemic inflammation is not completely understood. The three main factors involved in the pathogenesis of coagulopathy in patients with COVID-19 are as follows: (1) Endotheliitis, which causes vasoconstriction, (2) hyperinflammation and cytokine storm which activates clotting factors, and (3) stasis and hypoxia activates coagulation mechanisms.^[6] Ocular manifestations such as anterior uveitis, retinitis, and optic neuritis have been only documented in animal models.^[7] Previous studies have reported CRAO, papillophlebitis and isolated CRVO secondary to COVID-19.^[3,8] In COVID-19 due to venous or arterial thromboembolic complications, an evaluation for hypercoagulable disorders must be considered. In our patient, an interdisciplinary exploration process and a complete thrombophilia study were performed and the only findings were consistent with a hypercoagulable state induced by the COVID-19 infection and treated accordingly with anticoagulant (enoxaparin) and intravitreal bevacizumab but with limited posttreatment visual recovery.

Conclusions

Ophthalmologists must be prepared to treat vision threatening retinal vascular occlusions in the intermediate stages of COVID-19. Evidence of venous and arterial thromboembolic events in these patients suggests that pharmacologic anticoagulation prophylaxis may benefit hospitalized patients with confirmed or highly suspected COVID-19.

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