Retinal manifestations of ophthalmic artery occlusion with ischemic stroke in a young patient with COVID-19

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COVID-19-associated coagulopathy (CAC) has led to an increase in the incidence of large vessel stroke and cryptogenic shock. We present a case of a 30-year-old COVID-19-positive patient who developed an internal carotid artery (ICA) thrombosis, which led to ischemic stroke, aphasia, and unilateral blindness. Ophthalmic artery occlusion (OAO) was found to be the cause of vision loss. We thereby aim to highlight the detailed ophthalmic manifestations of OAO with features of posterior ciliary artery occlusion (PCAO) in this patient with proven ICA thrombosis.

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Coronavirus disease 2019 (COVID-19) is caused by infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1] Large vessel occlusion occurs secondary to COVID-19 more so in younger age group patients.[1-3] We report a case of a young Indian male patient who developed a deadly cerebrovascular stroke due to COVID-19 infection and severe vision loss due to its thrombotic complications.

Case Report

A 30-year-old male patient presented to our hospital with a history of diminution of vision in the left eye for 3 months. Ten months back during the pandemic, the patient had a fever for 2 weeks for which the patient was on symptomatic treatment at home. One night, he had an episode of vomiting and headache, and the next morning he was found unconscious. He was rushed to the hospital emergency department where he tested positive for COVID-19 through the Xpert test and was admitted. There was no known positive systemic history. On admission, his National Institute of Health Stroke score(NISS) was 19. He was mute with global aphasia. There was facial asymmetry with a deviation of the angle of the mouth to the left. Central Nervous System (CNS) examination

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showed reduced muscle tone and power of both limbs of the right side and reduced deep tendon reflexes. He was diagnosed with ischemic stroke with right hemiparesis, right upper motor neuron (UMN) facial palsy, and global aphasia. Non-contrast computed tomography (NCCT) scan of the brain revealed a middle cerebral artery (MCA) territory infarct [Fig. 1] with MCA hyperdense sign (arrow in Fig. 1). Extensive workup for stroke revealed lymphocytosis, neutrophilia, increased prothrombin time (13.6 s), hyper-homocysteinemia, and hyperlipidemia. CT-angiography of the head and neck revealed occlusion of the internal carotid artery (ICA) cut off from its origin. The carotid Doppler revealed a left ICA thrombosis. The patient was started on anti-hypertensive, anti-epileptic, dual anti-platelet therapy, and lipid-lowering agents.

After 3 months, he first noticed a diminution of vision in the left eye. On examination, he was now oriented to time, place, and person. He had a hemiplegic gait and unclear speech. His best-corrected visual acuity (BCVA) was 6/6, N6 in the right eye (RE) and 1/60, <N36 in the left eye (LE). The intraocular pressure was 12 and 13 mmHg in RE and LE, respectively. A grade 4 rapid afferent pupillary defect (RAPD) was present in the left eye. The left eye fundus examination revealed a pale disc with collaterals at the disc, severe arterial narrowing with sclerosed inferotemporal artery along with chorioretinal atrophy at the macula and in all the quadrants in the mid-periphery [Fig. 2]. Optical coherence tomography (OCT) of the macula revealed foveal thinning in the LE along with total loss of retinal architecture [Fig. 3]. Fundus fluorescein angiography (FFA) revealed a delayed arm to retina time (25 s) with a delayed patchy filling of the choroid, completed at 52 s. There was late arterial filling (50 s) and the cilioretinal artery also did not show filling until late phases. Areas of hyper fluorescence were noted in patches of chorioretinal atrophy s/o window defects [Fig. 4]. OCTA showed loss of superficial and deep vascular vessels with distorted choriocapillaris architecture [Fig. 5]. Hence, the diagnosis of LE old posterior ciliary artery occlusion (PCAO) along with Central Retinal Artery occlusion (CRAO) involving cilioretinal artery secondary to ICA thrombosis was made.

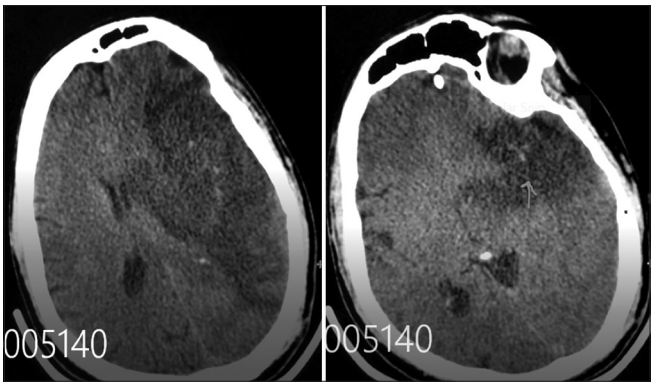

Figure 1: NCCT of the brain showing ischemia in the left temporoparietal lobes with MCS hyperdense sign (red arrow)

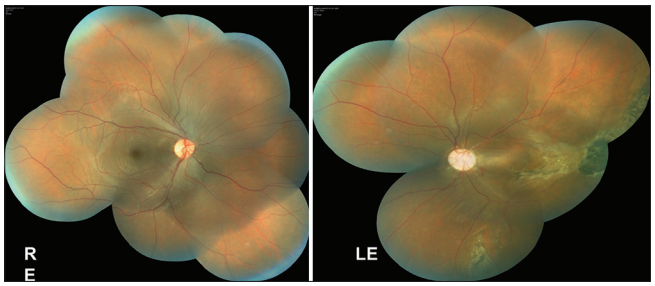

Figure 2: Color fundus picture showing a normal fundus in the RE. In the LE, there is optic atrophy, sclerosed vessels, and chorioretinal atrophy in the periphery

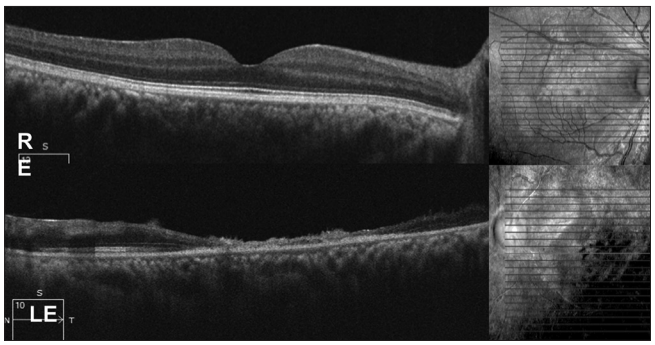

Figure 3: OCT frames showing a normal foveal contour in the RE and foveal thinning in the LE

Discussion

COVID-19 has been reported to cause ischemic stroke and large vessel occlusion in young patients more than usual.[1] Reported rates of arterial thrombosis range from 2.8% to 3.8%.[4] Mechanisms involved include increased hypercoagulability from a pro-inflammatory state, infection-induced Disseminated Intravascular Coagulation (DIC), and embolus from the virus-related cardiac injury.[5-7]

ICA is the main vessel trunk from which the ocular blood supply arises. The ophthalmic artery (OA) is a branch of the supra-clinoid segment of ICA, which gives central retinal artery (CRA), the short and long posterior ciliary arteries (PCA), and the anterior ciliary arteries. PCA supplies the optic nerve head, the outer 130 mm of the retina, and the retinal pigment epithelium (RPE).[7] CRA supplies the inner retinal layers and contributes to the vision. Left MCA territory occlusion lead to right-sided hemiparesis and UMN facial nerve palsy, also due to Broca's and Wernicke's area supplied by the same led to global aphasia. In the eye, OA occlusion leads to PCAO and CRAO and hence loss of vision.

Acute PCAO leads to optic disc edema and ischemic retina and later on leads to the development of optic atrophy and the white opacity of the fundus seen during the acute phase resolves in approximately 2 to 3 weeks, and the involved part of the fundus assumes a grayish, granular, and depigmented appearance.[7] Cherry red spot is a feature of acute CRAO, which in old cases is seen as a loss of retinal architecture with gross thinning of retinal layers, thereby, explaining the fundus appearance in the present case.

Acharya *et al*.[8] have reported the first isolated case of CRAO in a 60-year-old patient following COVID-19, owing to its hyper-coagulable nature; however, a known site of occlusion could not be demonstrated. In another report by Murchison *et al*.,[9] they described a patient in his fifth decade with a CRAO secondary to COVID-19 due to luminal narrowing of the ICA.[9] However, that patient did not develop any neurological

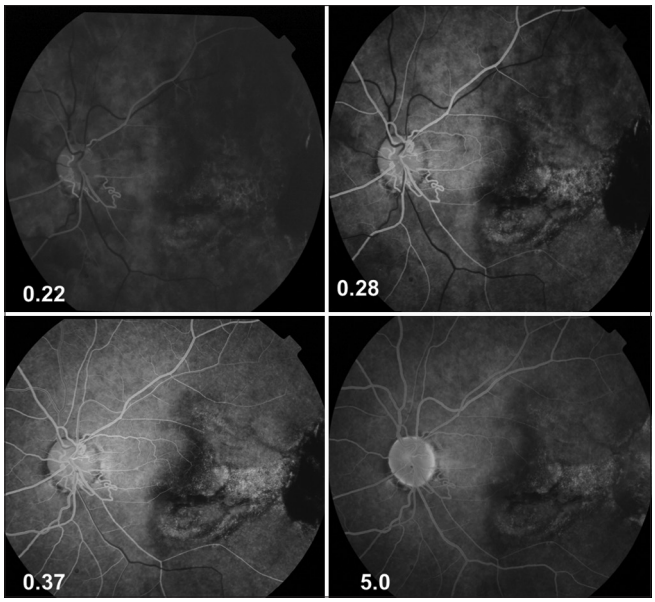

Figure 4: FFA of the LE showing a delayed arm to retina time, arterial filling, and AV transit tie, patchy and late choroidal filling

complications and the authors could not conclusively explain the reason for the absence of OAO.

This is a case of post-COVID-19 ICA thrombus leading to OA occlusion along with MCA thrombosis leading to hemiplegia and global aphasia. To the best of our knowledge, there is no such reported case in the literature.

Conclusion

An association between thrombosis and COVID-19 has been established and there is a dawning possibility of retinal vasculature involvement. With regular ophthalmic care still being sparing available, there is a need for a more rigorous retinal evaluation of these patients especially in patients with multiple risk factors. Moreover, because such a situation can arise in apparently healthy and relatively young adults as well, thus a need for retinal evaluation and further studies into the incidence of arterial disease is needed.

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Conflicts of interest

There are no conflicts of interest.

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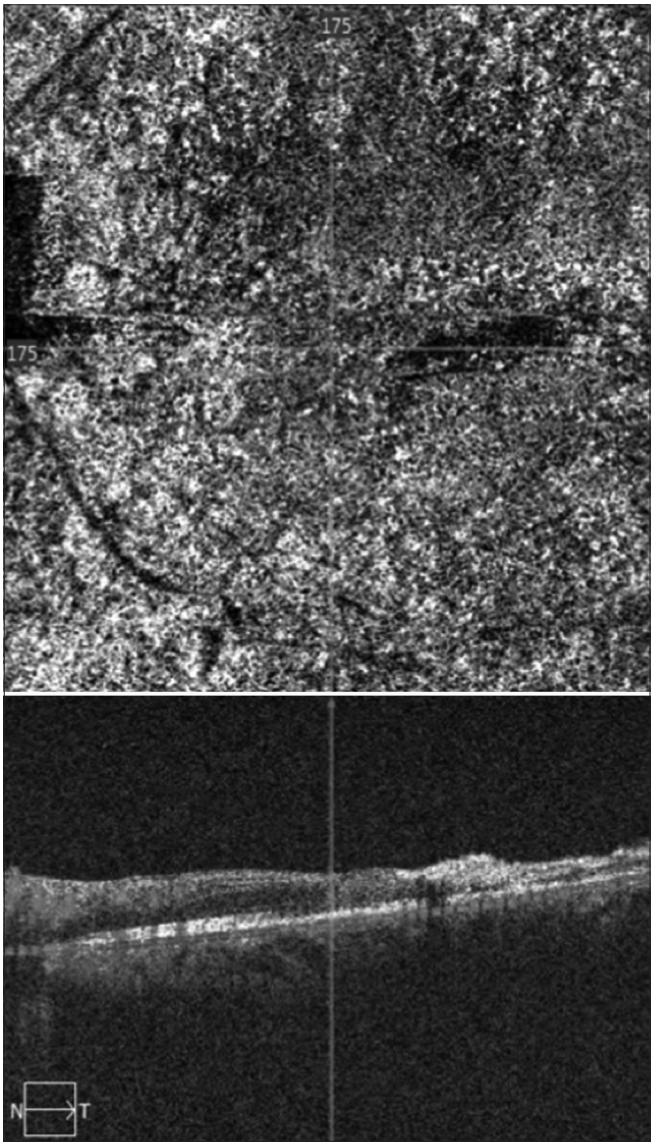

Figure 5: Distorted choriocapillaris architecture on the OCTA, the slab of choriocapillaris