Acute monocular vision loss as presenting symptom of delayed stroke from internal carotid occlusion in COVID-19

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Covid-19 infection has been declared a pandemic by the World Health Organization. We present a unique case of a middle-aged gentleman, who recovered from asymptomatic Covid-19 infection and presented again with delayed stroke. He had vision loss secondary to internal carotid artery occlusion in the absence of neurological symptoms. To the best of our knowledge, this is the first case that describes cerebrovascular stroke due to delayed large vessel occlusion secondary to Covid-19 infection presenting as monocular vision loss.

Key words: Covid-19, internal carotid artery occlusion, monocular vision loss

Coronavirus disease 2019 is a form of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that has been declared a pandemic by the World Health Organization (WHO). Large vessel occlusion has been increasingly seen in such patients and has been postulated that a hypercoagulable state along with endothelial injury following massive inflammatory response could be a potential contributor.1,2 Ophthalmic manifestations have been limited to case reports with active Covid-19 infection. To the best of our knowledge, this is the first case report of stroke from delayed occlusion of an internal carotid artery (ICA) presenting as acute monocular vision loss.

Case Report

A 53-year-old gentleman, presented with sudden onset loss of vision in the left eye (OS) for 1 day. He denied any visual disturbances in the right eye (OD). There was no history of diplopia, headache, weakness in limbs, sensory changes, facial weakness, or slurred speech. He seldom took alcohol. He gave a history of fever with cough 4 weeks ago not associated with changes in smell, taste, or confusion. He was found to be sputum reverse transcriptase-polymerase chain reaction (RT-PCR) positive and was isolated and treated under a physician with favipiravir 1800 mg BD for 8 days, prednisolone 40 mg and then tapered every 2 days on outpatient basis.

After 4 weeks from the first respiratory symptom, he presented to our clinic, with a vision of perception of hand movements close to face in OS and 6/6, N6 in OD. He had a left relative afferent pupillary defect (grade 4). Extraocular muscle movements were full, free, and painless. Slit-lamp examination was unremarkable. Dilated fundus evaluation revealed attached retinae in both eyes (OU) and normal disc in OD. He had disc edema without plaques, retinal whitening, or hemorrhages in OS [Fig. 1a]. Visual perimetry of the right eye was within normal limits and could not be performed on the left eye due to poor vision. Rest neurologic examination was unremarkable.

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The patient was admitted to the neurology clinic and evaluated further. Systemic evaluation for thrombotic aetiologies including blood pressure, complete blood count, packed cell volume, erythrocyte sedimentation rate, C reactive protein, serum homocysteine, Mantoux test, coagulation profile, lipid profile, and liver function tests were unremarkable. Magnetic resonance imaging (MRI) of the brain did not reveal hemorrhage or acute ischemic stroke. Magnetic resonance angiography (MRA) revealed a loss of blood flow in the left ICA and complete thrombosis of the left external carotid artery. The circulation of the left middle cerebral artery and anterior cerebral artery was supplied from the right ICA. Surprisingly, the brain matter does not show any kind of flow deficits or intensity change on diffusion-weighted scan. The flow void in the ICA as well as in the left ophthalmic artery was not visualized, and hence, the diagnosis of the left ophthalmic artery in addition was made [Fig. 1b]. This explained the lack of neurological deficits in our patient with only vision loss as presenting symptom.

The patient was immediately started on low molecular weight heparin (LMWH) (enoxaparin 40 mg intravenous 12 hourly); anti-platelet (acetylsalicylic acid 100 mg and clopidogrel 75 mg) and statin (atorvastatin 40 mg) once daily. He remained afebrile with no new ophthalmologic or neurologic symptoms. His oxygen saturation remained >95% on room air throughout his admission. At one week, his computed tomography angiography (CTA) revealed recanalization of the left internal ophthalmic artery [Fig. 2]. Oral antiplatelet and statin were continued after discharge. Further review is awaited.

**Discussion**

SARS-COV-2 is a novel, enveloped, positive-sense, single-stranded RNA virus that causes respiratory, enteric, hepatic, and neurologic diseases. In addition to common cardiovascular comorbidities, in the elderly COVID-19 positive population, mechanisms for ischemic stroke in infected patients of all age groups include hypercoagulability from proinflammatory state, infection-induced disseminated intravascular coagulation, embolism from virus-related cardiac injury, and prolonged hospitalized state. Critically ill COVID-19 patients had increased proinflammatory cytokines, including interleukin 2 and tumor necrosis factor-α.

Recent studies of Covid-19 patients revealed the possibility of large vessel occlusion in the relatively asymptomatic patient with low D-Dimer levels. When initiating anticoagulation in such patients for secondary stroke prevention, LMWH is often preferred due to consistent immediate therapeutic levels, antiinflammatory properties, and less frequent monitoring of coagulation parameters.

Our patient, during active infection with respiratory symptoms, had low D-dimer levels (212.8) and did not receive anticoagulants or antiplatelets. He was started on LMWH and antiplatelet drug immediately when he presented with a delayed stroke from ICA thrombosis.

Reported spectrum of ocular manifestations in active Covid-19 infection includes eyelid dermatitis, conjunctivitis, central retinal artery and vein occlusion, optic neuropathy, occipital cortical infarct, and acute macular neuroretinopathy. It has been speculated that thromboembolism may be more common in the second half of the COVID-19 disease course (Day 10 onwards) when a systemic inflammatory response reaction has been demonstrated to predominate. The cause for monococular vision loss can be attributed to delayed occlusion of the ICA in our patient.

The inflammatory and coagulopathic complications of COVID-19 may not directly correlate with the severity of respiratory symptoms. The timing of these events may be unpredictable and unrelated to peak COVID-19 disease severity.

To date, there have been a handful of reports identifying ICA occlusion in relation to COVID-19 infection and interestingly all of them had associated neurological symptoms and the authors do not deny the role of underlying prothrombotic mechanisms. In contrast, in our patient, there was no neurological involvement except monococular vision loss.

**Conclusion**

We report a case of a patient recently recovered from Covid-19, representing again after 4 weeks with acute monocular visual loss and delayed stroke from ICA thrombosis, in absence of traditional risk factors for cardiovascular diseases or underlying coagulopathies. While it not possible to prove a direct causative relationship, it appears that this event was due to COVID-19-induced delayed hypercoagulability. Our case illustrates the need for a high level of suspicion for delayed stroke involving large artery in patients presenting with acute visual loss and no neurologic symptoms.

**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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