Bilateral anterior ischemic optic neuropathy and secondary angle-closure glaucoma in a patient with COVID-19 infection

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Abstract

A 57-year-old male known case of diabetes mellitus presented with gradually bilateral decreased vision accompanied by ocular pain two weeks after SARS-CoV-2 infection. Ophthalmic examination and imaging were indicative of bilateral anterior ischemic optic neuropathy and secondary angle-closure glaucoma associated with increased choroidal thickness and hypercoagulable state following COVID-19 infection.

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a highly contagious enveloped RNA virus that caused a global pandemic in December 2019. Although Coronavirus Disease 2019 (COVID- 19) was primarily found to affect the respiratory and gastrointestinal system further studies revealed the multiorgan organ invasion of the virus[1]. Although the spectrum of ocular manifestations is not completely defined several reports from anterior segment involvement such as conjunctival hyperemia, chemosis, increased secretions, anterior uveitis to retinitis, choroiditis, and optic neuropathy are available[2,3]. The SARS -CoV-2 infection may be asymptomatic or paucisymptomatic with mild to moderate presentation of disease and some individuals may present severe to critical disease. Therefore, ophthalmologists may encounter asymptomatic or paucisymptomatic individuals who presented ophthalmic manifestation of COVID-19 infection.

Herein, we present a case of a 57-year-old male with bilateral non-arteritic anterior ischemic optic neuropathy (NAION) and secondary angle-closure glaucoma (SACG) due to choroidal hyperemia in a patient with confirmed SARS-Cov2 infection.

Case description

A 57-year-old male with a past medical history of controlled diabetes mellitus (DM) and no prior ophthalmic examination was referred due to gradually bilateral vision loss six days before the presentation which was accompanied by ocular pain in recent days. Eighteen days before the onset of visual symptoms, the patient was diagnosed with SARS-CoV-2 by a positive polymerase chain reaction (PCR) diagnostic test and symptoms of high fever, persistent cough, and shortness of breath. The patient was admitted for two weeks and received systemic Dexamethasone and Remdesivir. At presentation, his viral and systemic symptoms were resolved. There was no history of headache, scalp tenderness, fever, weight loss, and muscle weakness.

On examination, the best-corrected visual acuity (BCVA) was light perception for the right eye (RE) and no light perception for the left eye (LE). Examination of the pupils revealed nonreactive pupils to light therefore the relative afferent pupillary defect was not assessable. Extraocular movements were normal. Intraocular pressure was measured 28 mmHg for the RE and 42 mmHg for the LE. Slit-lamp examination findings indicated bilateral shallow anterior chambers (360@ peripheral anterior synechiae (PAS) was found on gonioscopic examination), corneal microcystic edema, and nuclear sclerosis 1+ cataract in both eyes.

Dilated fundoscopic examination revealed bilateral optic disc swelling. Optic discs were small-sized and the cup disc ratio was 0.5 in the RE and 0.7 in the LE. Also, an intraretinal macular hemorrhage in the RE was seen (figures 1-A, 2-A). Papillary edema was documented in retinal nerve fiber layer optical coherence tomography (RNFL OCT) (figures 1-B, 2-B). Analysis of blood tests including complete blood test (CBC), erythrocyte sedimentation rate (ESR), and C- reactive protein (CRP) was not remarkable. Increased choroidal thickness was noted in enhanced depth imaging OCT (EDI-OCT), especially in the LE (figures 1-C, 2-C). In fluorescein angiography, no sign of ischemia or peripheral vasculitis was detected (figures 1-D, 2-D). Indocyanine green angiography confirmed severe choroidal ischemia (figures 1-E, 2-E). No pathologic finding was reported in brain magnetic resonance imaging (MRI). The patient was admitted and after medical IOP reduction, peripheral laser iridotomy was done. Intravenous methylprednisolone (one gr daily) for three days was prescribed. In follow-up examination there was no improvement in visual acuity, IOP was controlled with topical medication and optic disc swelling was diminished.

Discussion

Ocular manifestations of SARS-Cov2 in humans are not fully recognized. Previous reports indicated ophthalmic presentations from conjunctivitis and anterior uveitis to retinitis and optic neuritis[3].

As shown in previous studies there is an association between SARS-Cov2 infection and hypercoagulability. The SARS-Cov2 binds to the host cells by angiotensin-converting enzyme (ACE)-2 receptor(R). ACE-2Rs are present in high density in the heart, lungs, arteries, and veins. So these organs are more vulnerable to the invasion of the pathogen. SARS-Cov2 causes endothelial dysfunction by binding to ACE-2Rs in retinal and choroidal microvasculature and leads to tissue ischemia, edema, and pre-coagulation state. Besides that, the systemic inflammatory response and cytokines release activates the coagulation cascade which results in venous and arterial thromboembolic complications [2].

Nonarteritic anterior ischemic optic neuropathy (NAION) is caused by infarction of the short posterior ciliary arteries that supply the anterior portion of the optic nerve head. Although the exact pathophysiology of the condition is not recognized some risk factors are noted in the literature including diabetes mellitus, hypercholesterolemia, smoking, anemia, and hypercoagulable state[4].

In our patient with a history of diabetes mellitus as a risk factor for AION and a history of COVID-19 infection, it seems that the hypercoagulable state and hypoxia caused by COVID-19 infection predisposed him to NAION.

COVID-19 infection has the potential to cause damage to the choroid, retina, and optic nerve. Increased choroidal thickness, features of pachychoroid and abnormal dilation of Haller's layer vessels are reported in patients with COVID-19 infection in previous research[5]. As discussed above it seems that a disturbance in autoregulation of the RAAS system caused by the binding of SARS-Cov2 to ACE-2R results in endothelial dysfunction, increased vascular permeability, dilatation of retinal and choroidal vessels. Changes in the retinal and optic nerve head microvasculature have been identified in previous research. A decrease in vessel density (VD) in the superficial and deep capillary plexus, as well as an increase in inner disc small vessel VD which can be associated with optic disc hyperemia and edema, have been documented [6-8]. These alterations suggest that COVID-19 infection puts patients at risk for retinal and optic nerve disorders.

In our patient, EDI-OCT revealed increased choroidal thickness. It is supposed that the thickening of the choroid led to anterior rotation of the ciliary body and resulted in a shallow anterior chamber. So that aqueous misdirection occurred and secondary angle-closure glaucoma developed.

NAION is caused by hypoperfusion of the optic nerve. Our patient presented with bilateral NAION in the setting of COVID- 19 infection. Hypoxemia and the hypercoagulable state as the sequence of COVID-19 infection may result in NAION in this patient. Also, increased choroidal thickness in the setting of COVID-19 infection may cause anterior rotation of the ciliary body and secondary angle-closure glaucoma consequently. There are three possible mechanisms for ocular complications in our patients: vascular endothelial damage, hypercoagulable state, and hypoxemia. At present, it is not possible to determine a causal or coincidental

relationship between NAION, choroidal hypoperfusion, and secondary angle-closure glaucoma that occurred in described patient and COVID-19 infection. But the purpose of this report is to shed light on the potential and multiplicity of ophthalmic presentation in a patient with COVID-19 infection.

Declaration of patient consent

Researchers ensure that they have obtained informed written consent from the patient for the publication of clinical examinations and retinal imaging.

Conflicts of interest

There are no conflicts of interest.

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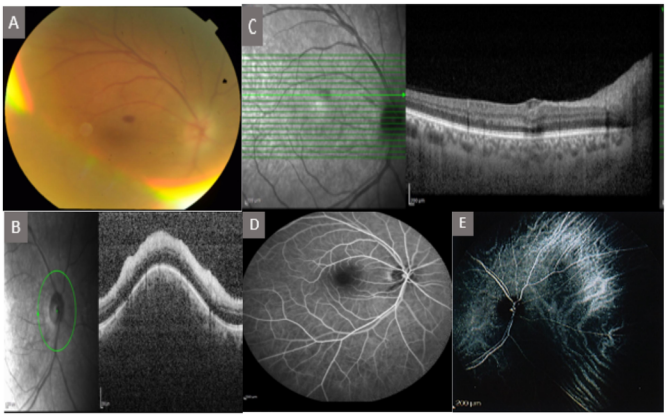
Figure legends

Figure 1. Multi-modal imaging of the RE. **A. *Color fundus photography:*** optic disc swelling and macular hemorrhage.**B.** *Retinal nerve fiber layer OCT:* papillary edema. **C: *Enhanced depth imaging OCT:* increased choroidal thickness. D: *Fluorescein angiography:*** Arteriovenous phase: No evidence of areas of ischemia or peripheral vasculitis. **E:*Indocyanine green angiography:*** Early-phase: wedge-shaped hypocyanescent areas indicative of choroidal ischemia.

Figure 2. Multi-modal imaging of the LE. **A. *Color fundus photography:*** optic disc swelling **B.***Retinal nerve fiber layer OCT:* papillary edema.**C: *Enhanced depth imaging OCT:* increased choroidal thickness. D: *Fluorescein angiography:*** Arteriovenous phase: No evidence of areas of ischemia or peripheral vasculitis. **E:*Indocyanine green angiography:*** Early-phase: patchy hypocyanescent areas indicative of choroidal ischemia.



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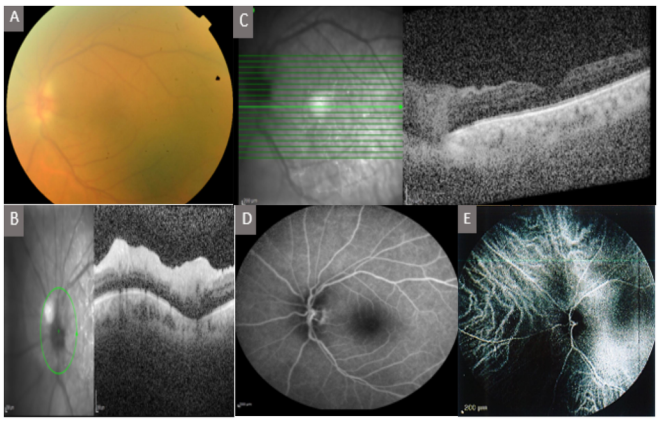

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