Optic Nerve Inflammation Associated with Severe Acute Respiratory Syndrome Coronavirus 2

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Optic neuritis is an inflammation of the optic nerve and has several causes. The hallmarks of clinical manifestation are pain on movement of the eyes and decreased vision. Typical optic neuritis is an idiopathic demyelinating condition that is often associated with multiple sclerosis, affects young women, is unilateral, and has a good prognosis. Atypical optic neuritis can be bilateral and results from an inflammatory, infectious, or autoimmune disorder. One subtype of atypical optic neuritis is infectious optic neuritis, which may be secondary to Lyme disease, bartonella, herpetic disease, west Nile virus, or human immunodeficiency virus [1].

Our case describes an adult male who was diagnosed with coronavirus disease 2019 (COVID-19) and high inflammatory marker expression who had transient visual loss presumed to be secondary to optic nerve involvement, associated with COVID-19 infection. Optic inflammation is a rare symptom of COVID-19, and was the only presenting symptom of the patient.

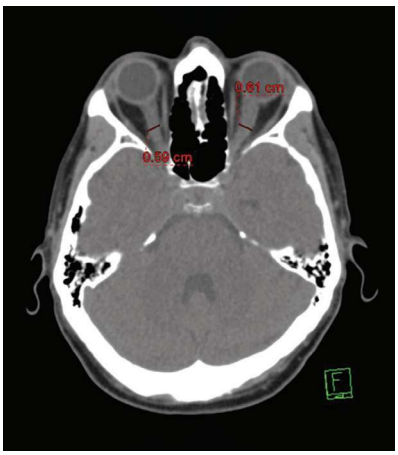
PATIENT DESCRIPTION

A 66-year-old male presented with two occasions of transient left monocular vision loss and periorbital dull pain exacerbated by eye movements. Two weeks before, he had been exposed to a carrier of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and his polymerase chain reaction (PCR) test for SARS-CoV-2 was positive. Nevertheless, he did not have respiratory symptoms. His past medical history included Crohn's disease, bronchial asthma, gout, and dry age-related macular degeneration. His daily medications included mesalamine and a budesonide/formoterol inhaler.

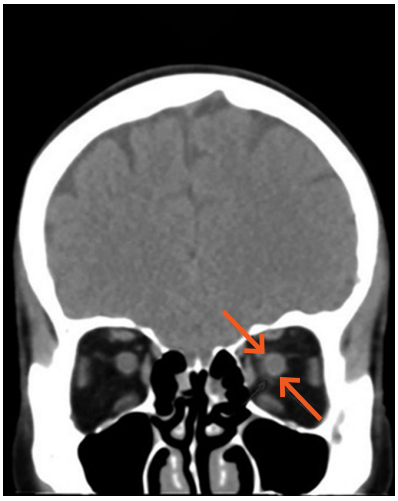
At admission, he presented with normal vital signs and no signs of respiratory distress. He denied temporal headaches, jaw claudication, or shoulder or hip girdle pain. The neurological examination was normal. The ophthalmological examination revealed negative relative afferent pupillary defect (RAPD), normal eyelids on both eyes (BE), white conjunctiva and clear cornea BE, attached retina BE, clear vitreous in left eye and floaters in the right eye, and blurring of optic disc margins in the right eye. Blood tests showed elevated inflammatory markers including C-reactive protein (3.6 mg/dl, normal < 0.5), erythrocyte sedimentation rate 70 mm/h (normal < 30), fibrinogen 700 mg/dl, (normal < 500), and thrombocytosis 500,000/μL. Computed tomography angiography of the head and neck disclosed thickening of the optic nerve bilaterally with fat enhancement around the optic nerve sheath [Figure 1]. All other neurovascular aspects were normal. Based on the clinical presentation,

Figure 1. Computed tomography angiography of the head and neck

[A] Sagittal brain computed tomography demonstrating thickening of the optic nerves (bilateral)



[B] Axial brain computed tomography demonstrating fat enhancement around the optic nerve sheath



laboratory results, and radiological findings, the patient was diagnosed with optic neuritis secondary to COVID-19. No specific treatment was prescribed at that time. At two week follow-up, the patient was asymptomatic with no vision loss or eye pain and his ophthalmic examination was without significant findings.

COMMENT

A 66-year-old male presented with optic neuritis as the presenting symptom of COVID-19. His blood tests indicated an inflammatory state, most likely induced by SARS-CoV-2 infection. Vision loss and eye pain were transient, and he recovered within 2 weeks without specific treatment.

There are limited data on optic neuritis as a manifestation of COVID-19. One study on neurological involvement among 841 hospitalized COVID-19 patients reported one female patient with vision loss, assumed to be optic neuritis. The vision loss appeared during the recovery phase (11th day from onset). She also experienced respiratory symptoms and dysgeusia [2]. Sawalha and colleagues [3] described a 44-year-old male with optic neuritis 2 weeks after he developed symptoms of shortness of breath and cough and tested positive for SARS-CoV-2. Zhou et al. [4] described a 26-year-old COVID-19 patient with optic neuritis and myelitis. Benito-Pascual and co-authors [5] described panuveitis and optic neuritis as an unusual presentation of ocular involvement in COVID-19.

We described a COVID-19 patient with clinical and radiological findings compatible with bilateral optic neuritis. The absence of positive RAPD, which could be explained by bilateral involvement, and gradual vision loss are unusual; however, the radiological findings, the inflammatory markers and the clinical presentation suggested that there was inflammation of the optic nerves. No other manifestations of COVID-19 were apparent, but there was recent exposure to a COVID-19 patient and the PCR testing was positive. The patient presented with a clinical picture suggesting atypical optic neuritis and showed spontaneous recovery; therefore, steroid treatment was not recommended. COVID-19 is characterized by robust inflammation; thus, it is reasonable to assume that optic neuritis is part of the inflammatory process induced by SARS-CoV-2 infection. Another possible mechanism is direct damage by the virus [2].

We described a 66-year-old patient who presented with transient optic nerve inflammation associated with COVID-19. This report has some limitations. First, unfortunately, due to social distancing restrictions and isolation we do not have access to some essential clinical data (optic nerve function and visual field testing) and magnetic resonance imaging results. However, the available clinical and imaging data suggested an inflammatory process that involved the optic nerve. In addition, the diagnosis of COVID-19 and the ophthalmic manifestation could be a coincidence. Our case contributes to real-time data about the plethora of COVID-19 manifestations.

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Capsule

Activating adenylyl cyclase to treat Duchenne muscular dystrophy

Duchenne muscular dystrophy (DMD) is characterized by repeated contraction-induced muscle injury that eventually leads to failure of skeletal muscle regeneration. Extraocular muscles (EOMs) are generally spared in DMD, suggesting the presence of a potential compensatory mechanism. **Taglietti** and colleagues generated a rat model of DMD and found that EOMs had increased thyroid stimulating hormone receptor (TSHR), resulting in decreased muscle stem cell senescence. Forskolin, an adenylyl cyclase activator, stimulated TSHR signaling and led to improved proliferation and reduced senescence of skeletal muscle stem cells, resulting in improved functional performance in affected rats. These findings suggest that activation of TSHR signaling may have therapeutic value in patients with DMD.

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