

Bilateral painless loss of vision in a patient with COVID-19 infection.

Introduction

COVID-19 can cause neurological symptoms in 36.4% cases with hyposmia and hypogeusia being most common. We present a patient with COVID-19 infection and bilateral vision loss.

Case presentation

A 42-year-old gentleman with diabetes presented with symmetric, bilateral, painless loss of vision for three days. No redness, flashers/floaters, diplopia, jaw claudication, headache, fevers, dyspnea, weakness, facial deformity, slurred speech, or palpitations were reported. The patient had an occasional non-productive cough. No new medications or drug intake was noted, except minimal alcohol intake. Vital signs were stable. Visual acuity was limited to the perception of hand movements at one foot. Ocular examination and fundoscopy were normal bilaterally. Laboratory parameters showed mild elevation of inflammatory markers (CRP, LDH, D-dimer, ferritin) and hemoglobin-A1c was 8%. Thiamine, cyanocobalamin and folate were normal. A toxicological workup was negative. COVID-19 was positive with RT-PCR testing but was not treated in the absence of respiratory symptoms. Brain CT and MRI/MRA did not reveal any pathology. CSF studies were unremarkable. Three days of pulsed methylprednisolone intravenously led to visual improvement to finger counting at three feet by day#3. The patient was discharged on an oral prednisone taper and the vision improved to baseline after three weeks.

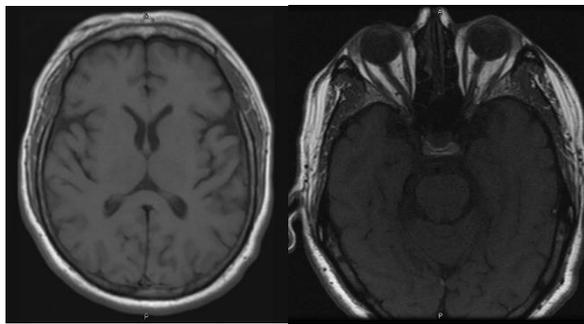
Discussion

COVID-19 associated transient visual loss with suspected optic neuritis, PRES-like syndrome and posterior ischemic optic neuropathy (PION) have been reported.

★ Initial labs on presentation

Parameter	Reference	Patient
Total white count (x10 ⁹ /L)	4-11	10.5
Absolute lymphocyte count (x10 ⁹ /L)	1-3.5	0.63
Platelets (x10 ⁹ /L)	150-400	231
D-Dimer (ng/mL)	0-211	<150
Lactate dehydrogenase (U/L)	140-271	165
C reactive protein (mg/L)	0-5	<5
Ferritin (ng/mL)	18-464	250
Thiamine (µg/L)	25-75	50
Cyanocobalamin (pg/mL)	180-914	505
Folate (ng/mL)	>2.8	>24.5

★ Normal MRI of the brain and orbits



SARS-CoV-2 may invade host cells via the ACE-2 receptor, also expressed in the endothelium and central nervous system (CNS). It has been isolated from the endotheliocytes, frontal lobe neurons, and CSF. The inflammation results in endothelial dysfunction, complement activation, and cytokine recruitment leading to a prothrombotic state.

The inflammation, coagulopathy, and microvascular dysfunction lead to vasculitis, cerebrovascular disease, and blood-brain barrier damage. Direct viral infection may lead to encephalitis and neuritis. The virus invades via the cribriform plate and gains access to olfactory neurons and may disseminate trans-synaptically.

Acute painless vision loss is a medical emergency. Etiologies may include amaurosis fugax, central retinal artery occlusion (CRAO), central retinal vein occlusion (CRVO), vitreous hemorrhage, ischemic optic neuropathies (ION), optic neuritis, toxic optic neuropathy (TON), Leber's hereditary optic neuropathy (LHON), retinal detachment, posterior cerebrovascular accidents (CVA) or transient ischemic attack (TIA). CRAO, CRVO, vitreous hemorrhage, retinal detachment, TON, LHON, CVA, and TIA were unlikely given the presentation and investigations. ION may either be anterior or posterior. Arteritic or non-arteritic anterior ION were both unlikely. PION was possible due to COVID-19 associated microvasculopathy. Optic neuritis was also probable, with suspected SARS-CoV-2 neuroinvasion leading to neuritis. The elevated inflammatory markers and the prompt response to the corticosteroids support our hypothesis.

Conclusion

Since the etiology remained indeterminate, vigilance and more research into such cases associated with COVID-19 are suggested.