Acute Vision Loss in a Patient with COVID-19

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ABSTRACT

To date, there have been reports of neurologic manifestations in COVID-19 patients including ischemic strokes, Guillain-Barre Syndrome and anosmia. In this case report, we describe a patient who presented with dysosmia, dysgeusia, along with monocular peripheral vision loss after being diagnosed with COVID-19.

KEYWORDS: vision loss, SARS-CoV-2, COVID-19, neurologic, ophthalmology

INTRODUCTION

As of June 6, 2020, there are more than 6,500,000 cases of SARS-CoV-2 worldwide.[1] To date, there have been several reports of neurologic manifestations in these patients including ischemic strokes, Guillain-Barre Syndrome and anosmia.[2-4] Wu et al described a case series involving 38 patients with COVID-19, where 12 patients had ocular signs of epiphora, conjunctival congestion and chemosis.[5] We report a patient who presented with dysosmia, dysgeusia along with monocular peripheral vision loss after being diagnosed with COVID-19.

CASE PRESENTATION

A woman in her 50s with a history of hypertension, hyperlipidemia, and headaches presented to the hospital with fever, chills, and cough one week after she tested positive for SARS-CoV-2. She reported acute, painless right eye monocular visual disturbance, described as a white cloud and blurriness involving most of her right eye, sparing the superior nasal aspect. She denied any left eye visual disturbances. She denied any other ocular symptoms such as flashers, floaters, or diplopia. She denied any jaw claudication, scalp tenderness, unintentional weight loss. Other neurological symptoms included dysgeusia, dysosmia, right ear hypoascusis, and subjective right hemiparesis. She was not taking any medications at home.

On the day of admission, her neurological exam was remarkable for severe right eye vision loss. She was unable to visualize or count fingers in the right temporal field and inferior nasal field. The left eye exam was normal. Relative afferent pupillary defect was absent. There was no tenderness to the palpation of the temporal area. The following day, she reported fifty percent improvement in her vision. Her vision in the far periphery of the right eye was blurry but she was able to count fingers in all fields. Visual acuity was 20/70. The dilated fundoscopic exam was normal. Ocular pressures were normal. There was no evidence of optic disc edema, Hollenhorst plaque, retinal whitening. or hemorrhages.

Her laboratory values were normal including CBC, BMP and ESR. Her CRP was 7, and d dimer was 206 ng/ml. LDL was elevated at 131. Initial MRI of the brain without gadolinium did not reveal any intraparenchymal or cranial nerve abnormalities, though it was notable for a partially empty sella turcica. MRI of the orbits, face, and neck with and without gadolinium revealed no area of abnormal enhancement. The optic nerves, chiasm, and optic tracts appeared normal. CT angiography showed no significant carotid disease. Her vision spontaneously improved during her hospitalization, and she was discharged home on aspirin and atorvastatin. She was advised to follow up in the Ophthalmology clinic in one month.

DISCUSSION

The clinical spectrum of illness due to COVID-19 continues to evolve. Acute vision loss is a medical emergency and can occur over a few seconds or minutes to a few days. Vision may become blurry, cloudy, entirely or partially absent, or affected by flashes or floaters. Acute vision loss is usually painless but may also be associated with ocular pain, redness and headache. Most cases of visual loss can be diagnosed by history and physical examination alone.

Common causes of acute vision loss include Central Retinal Artery Occlusion, Central Retinal Vein Occlusion (CRVO), Retinal Detachment, Optic Neuropathy, and Inflammatory conditions such as Giant Cell Arteritis (GCA). CRVO was unlikely due to the absence of retinal hemorrhages and cotton wool spots on the fundoscopic exam. Given the history of peripheral monocular vision loss, transient Branch Retinal Artery Occlusion (BRAO) was considered a possibility, although there was no evidence of retinal whitening or edema.

Given her normal ophthalmologic exam, Posterior Ischemic Optic Neuropathy (PION) was considered to be more likely. There are three different types of PION: arteritic,

non-arteritic, and surgical. The capillary plexuses supplying the posterior part of the optic nerve are vulnerable to hypoperfusion and ischemia. Vision typically recovers if circulation is restored before axonal death, as observed in our case. Arteritic PION is usually due to GCA, which was unlikely given normal ESR, CRP, and the absence of classic symptoms such as jaw claudication and scalp tenderness. Our patient likely had non-arteritic PION due to small vessel disease that is usually linked to systemic illness. Given the MRI evidence of a partially empty sella, idiopathic intracranial hypertension, or pseudotumor cerebri, was also considered a possibility for transient visual loss. She did not undergo a lumbar puncture to measure intrathecal pressure as her ocular symptoms had improved and she denied any headache symptoms.

Previous strains of coronavirus seem to invade the CNS mostly through the hematogenous route but also can invade through the cribriform plate and the conjunctiva.[5,6] The pathophysiology in our case is unclear. One of the mechanisms could involve inflammation associated with COVID-19 itself, although her CRP and ESR were normal.[7] Another mechanism could be related to the thromboembolic phenomenon and occlusion of small capillaries feeding the optic nerve, although our patient's d dimer was normal. Magro et al showed that there might be a microvascular injury syndrome mediated by activation of complement pathways and an associated procoagulant state that may also be at play in these patients.[8]

Our patient's symptoms were early in the course of her illness and could be useful in triaging patients. A thorough neurologic exam is essential in all patients diagnosed with COVID-19. This case illuminates a broader spectrum of COVID-19-related symptomatology and emphasizes the need for clinicians to be aware of the various clinical manifestations associated with this infection.

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