



INTRODUCTION

 COVID-19 is a disease caused by a novel Coronavirus (SARS-CoV-2) originating in Wuhan, China around December 2019. On March 11, 2020 it was declared a pandemic by the World Health Organization (WHO) as the number of infections rapidly grew around the world. Currently, cases have described COVID-19 involvement in respiratory, gastrointestinal, neurological, and ocular tissues. Respiratory droplets and direct contact are the most probable transmission routes, although aerosolized sneezes and coughs may also contaminate the immediate surroundings.[1] •COVID-19 most commonly causes respiratory symptoms such as dyspnea and cough which may progress to pneumonia and ARDS in a subset of patients, however recent studies have demonstrated neurological manifestations such as dizziness, headache, hypogeusia, hyposmia, neuralgia, impaired consciousness, acute CVD, ataxia, and seizure.[2][3] SARS-CoV-2 binds to the angiotensin-converting enzyme 2 (ACE2) receptor in human cells which is hypothesized to allow CNS access by ACE2 receptor expressing glial cells, neurons, endothelial and arterial smooth muscle cells in the brain.[4] A recent case of meningitis/encephalitis with SARS-CoV-2 RNA detected in the CSF directly supports the neuroinvasive ability of the virus.[5] In addition, SARS-CoV-2 has a similar viral structure and receptorbinding domain to SARS-CoV, a more well studied virus that demonstrated olfactory bulb infection with transneuronal spread in mice leading to neuronal loss.[4] Considering these characteristics, it is reasonable to conclude that SARS-CoV-2 shows neurotropism that can manifest neurological symptoms in an infected individual.

Recently, there have been case reports of neuro-ophthalmological manifestations in association with COVID-19 such as diplopia, Miller-Fisher syndrome, ophthalmoplegia, and polyneuritis cranialis. There is currently a limited number of publications that have been able to elaborate the neurological impact of COVID-19 on ocular tissues. To the best of our knowledge this is the first case describing acute strabismus decompensation following COVID-19 infection.

CASE PRESENTATION

A 27-year-old female presents to the emergency room with acute onset horizontal binocular diplopia and a history of COVID-19 infection 1-2 weeks prior. She has a history of longstanding intermittent right esotropia, myopia, and well-treated amblyopia in the right eye with excellent best corrected visual acuity. She states that before her COVID-19 infection she only had double vision after extended reading and typically sees clearly. Now she has constant double vision which started acutely earlier that day and worsened over the several hours. Her only other symptom at the time was increased fatigue. She was afebrile and denies having any respiratory or GI symptoms. During examination of eye tracking, there was a left esotropia when looking left and a right esotropia when looking right. Labs showed a normal WBC count, C-Reactive Protein, and ESR. The CT angiogram of the head with and without contrast, chest X-ray, and MRI of brain without contrast showed no abnormalities.

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CASE PRESENTATION (cont.)

She was discharged with a Medrol Dosepak for 6th nerve palsy with follow up as needed. Around 3 weeks later, she presented to the optometrist with horizontal diplopia. She states the diplopia has been stable and constant since her visit to the emergency department 3 weeks prior. On physical examination, her visual acuity was 20/20 in both eyes with a corrective lens. Motility was full to ductions and versions. An alternating esotropia greater than 45 prism diopters was found with the coveruncover test. Confrontational visual fields were full to count fingers. Pupils were equal, round, and reactive to light with no afferent pupillary defect. Intraocular eye pressure was measured to be 18 mmHg in both eyes via Goldmann tonometry. Slit lamp and dilated fundoscopic exam was unremarkable. She was diagnosed with alternating esotropia after decompensation of an intermittent esotropia in the right eye. Three weeks later during follow up, she reports a gradual and small improvement of the diplopia in her near vision. Her distance vision, however, is still perceived to be doubled with little to no improvement.

RESULTS & DISCUSSION

•The present case illustrates an acute strabismus decompensation as a neurological manifestation of COVID-19 infection. Based on our scientific literature review, this is the first reported case of decompensated strabismus as a direct result of COVID-19 infection. Strabismus is an anomaly of ocular alignment found in 2-4% of the population and is responsible for complications such as diplopia and amblyopia. Many cases are congenital; however, instances of acute strabismus may be seen in association with viral infections. •There are few studies on the spectrum of neuro-ophthalmologic manifestations that occur in association with COVID-19. Consuelo Gutiérrez-Ortiz et al. presented 2 cases of COVID-19, one with Miller-fisher syndrome and another with polyneuritis cranialis. The author hypothesized that these manifestations may be due to an aberrant immune response to COVID-19.[7] Marc Dinkin et al. described 2 cases of COVID-19 after presenting with diplopia and ophthalmoparesis.[6] It was hypothesized in one case to be a direct neurological infection, because there were neurologic manifestations within a few days of COVID-19 onset. In both cases, it was stated that acute cranial neuropathy should prompt consideration for COVID-19.[6][7] Overall, these publications may share similarities to our case with respect to the possible pathophysiological pathway, though the exact mechanism has yet to be thoroughly explored. •SARS-CoV-2 is an emerging strain of virus causing infections on a pandemic scale. Its neurological effects are poorly understood and may have a long-term impact that signifies a burden to the patient and the health care system. Understanding the neurological manifestations on ocular tissues may help in the recognition and diagnosis of the disease while promoting the need for targeted therapeutics. Further studies are needed to understand the pathogenesis of neurological manifestations due to COVID-19.

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COVID-19 has neurotropic effects which can result in decompensation for patients with strabismus. Understanding the neurological manifestations of the disease may help in the recognition and diagnosis while promoting the need for targeted therapeutics. This is the first reported case we are aware of with decompensated strabismus due to COVID-19.

CONCLUSION

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