Journal of Investigative Medicine High Impact Case Reports
Volume 8: 1–3

DOI: 10.1177/2324709620976018
journals.sagepub.com/home/hic
SAGE

COVID-19-Induced Acute Bilateral Optic Neuritis

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Abstract

A 44-year-old male patient with no past medical history presented 2 weeks after seropositive coronavirus disease 2019 (COVID-19) infection with vision problems suggestive of optic neuritis. Radiological testing showed findings suspicious for acute bilateral optic neuritis. The patient had also anti-MOG antibodies. Whether this was an optic neuritis due to COVID-19, MOG antibody disease, or an activation of MOG antibody disease by COVID-19 is discussed in this case.

Keywords

bilateral optic neuritis, COVID-19

Introduction

Bilateral acute optic neuritis is a rare phenomenon, particularly in individuals without known systemic inflammatory or autoimmune disorders. Optic neuritis in adults is usually unilateral and commonly linked to multiple sclerosis (MS). It usually occurs as sudden onset of visual loss associated with pain on eye movement and progress in its course to reach its maximum deficit in a week.[1] Diagnosis is usually clinical based on history and examination findings. Brain and orbital imaging such as magnetic resonance imaging (MRI) help in the diagnosis in most cases. After confirmation, treatment is usually straightforward.

Case Presentation

A 44-year-old Hispanic male patient with no past medical history presented from clinic with bilateral eye pain and vision loss. Two weeks prior to onset of eye and vision symptoms, he had tested positive for coronavirus disease 2019 (COVID-19) virus by nasopharyngeal polymerase chain reaction (PCR) after developing symptoms of shortness of breath and cough. He was quarantined at his home and treated symptomatically with improvement; he never required hospitalization for his respiratory illness nor medications including hydroxychloroquine. One week prior to his admission, he reported developing pain in his right eye that had progressed to his left eye along with worsening bilateral blurring of vision to the extent of complete vision loss. He denied any family history of any neurological or immunological diseases. On formal visual fields examination, a global vision loss was noted in the right eye with acuity 20/200 along with relative afferent pupil area defect. Left eye's vision was with a superior arcuate visual field defect and 20/30 acuity. Laboratory findings including complete blood count, comprehensive metabolic panel, urine analysis, and drug screen were all negative and/or unremarkable. Rheumatoid factor was 8.6 IU/mL (8.6-11.9 IU/mL), erythrocyte sedimentation rate 37 mm/h (0-15 mm/h). Brain MRI showed enhancement in the right more than the left optic nerve suggestive of optic neuritis although no other abnormalities were noted in brain, cervical, or thoracic spine (Figures 1-3). Lumbar puncture was done with nonspecific findings of colorless cerebrospinal fluid (CSF), white blood cell count of 3 cells/μL (0-5 cells/μL), red blood cell count of 6.0 cells/μL, albumin 23.3 mg/dL (8-42 mg/dL), glucose of 88 mg/dL (40-70 mg/dL), and total protein 50 mg/dL (12-60 mg/dL) with negative gram stain. No oligoclonal bands were detected. CSF cytology showed lymphocytes 90%. Myelin oligodendrocyte glycoprotein (MOG) was detected with a titer of 1:160 (<1:10). Viral panel including Epstein–Barr virus, HIV, and cytomegalovirus was negative. Lyme disease PCR was not detected. Vitamin B_{12} level was 724 pg/mL (239-93 pg/mL). Immunological panel including IgG, ANA, JO-1, Anti-NMO ab, SS-A, and SS-B were also negative. Nasopharyngeal severe acute respiratory syndrome virus RNA PCR and serum severe acute respiratory syndrome

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Received October 6, 2020. Revised October 24, 2020. Accepted October 31, 2020.

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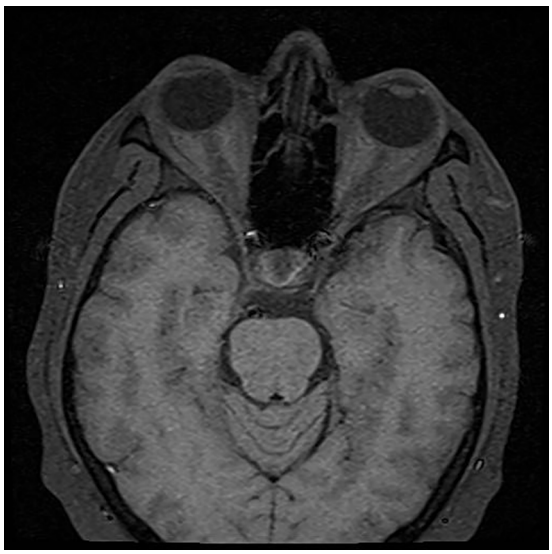

Figure 1. Axial orbital magnetic resonance imaging pre gadolinium contrast showing right optic nerve has a slightly ill-defined appearance more than left.

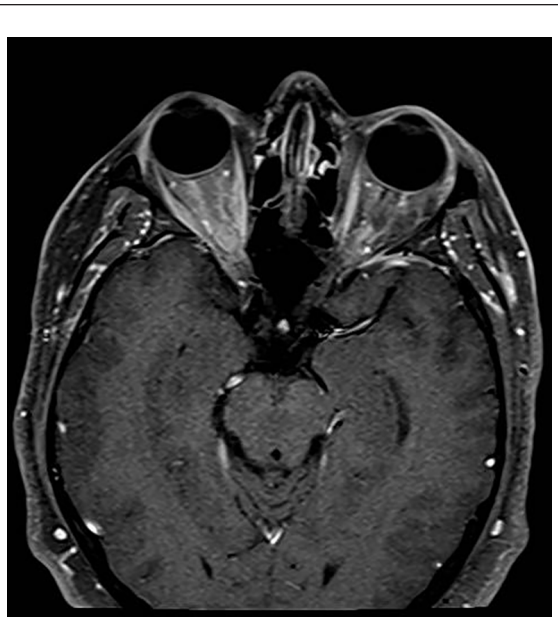

Figure 2. Axial orbital magnetic resonance imaging post gadolinium contrast showing enhancement of the right optic nerve. Lesser degrees of enhancement are seen in the left optic nerve, particularly in the more proximal segment. Optic chiasm has a normal appearance. Globes are normal in appearance. Extraocular muscles are symmetric and within normal limits.

coronavirus 2 IgG antibodies were detected. The patient was started on methylprednisolone 1 g every 24 hours for a total of 5 days/doses. Within 48 hours, his vision and eye pain had

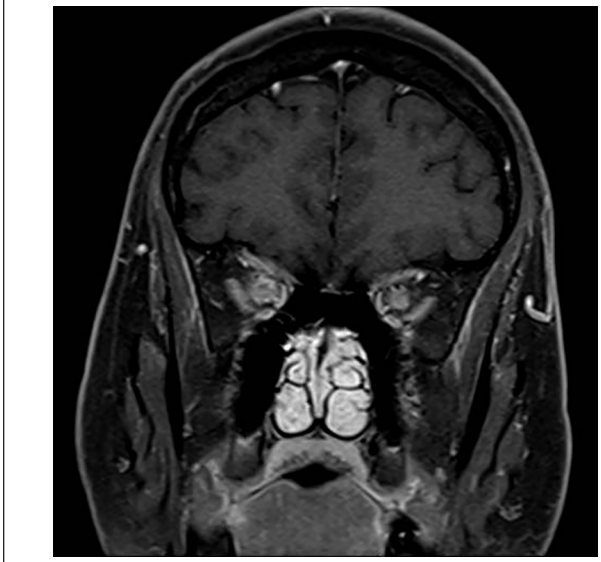

Figure 3. Coronal orbital magnetic resonance imaging post gadolinium contrast showing right optic nerve has a slightly ill-defined appearance more than left.

shown significant improvement. By day of discharge, our patient had complete restoration of vision in the left eye with remarkable but not complete vision recovery in the right eye. He was discharged on a tapering dose of prednisone over 4 weeks with planned follow-up with ophthalmology and neurology.

Discussion

Optic neuritis is defined as an inflammatory, demyelinating condition that causes acute, usually monocular, visual loss. It is highly associated with MS, and it is the presenting feature in 15% to 20% of these patients and occurs in 50% of them at some time during their illness.[2] It is believed that the demyelination in optic neuritis is immune-mediated, but the exact targeted antigens remain unknown. Systemic T-cell activation is identified at symptom onset and precedes changes in the CSF.[3] T-cell activation leads to the release of cytokines and other inflammatory agents. B-cell activation against myelin basic protein is not seen in peripheral blood but can be demonstrated in the CSF of patients with optic neuritis.[4]

In general, optic neuritis is a clinical diagnosis based on the history and examination findings. Because important findings on funduscopic examination help differentiate typical from atypical cases of optic neuritis, an ophthalmologic examination can be considered in the clinical evaluation. MRI of the brain and orbits with gadolinium contrast provide a better assessment of the diagnosis in most cases. Further diagnostic testing, such as lumbar puncture, is done to exclude other causes of visual loss in atypical cases.

However, around 60% to 80% of patients with acute optic neuritis have nonspecific findings in the CSF such as lymphocytes and elevated proteins.[5]

Myelin oligodendrocyte glycoprotein antibodies were originally thought to be involved in MS, but subsequent studies found it to be a distinct disease called myelin oligodendrocyte glycoprotein antibody disorder (MOGAD), which can be seen with optic neuritis, acute disseminated encephalomyelitis, and transverse myelitis. Neuromyelitis optica (NMO), which is among the neuromyelitis spectrum disorders (NMOSD), is suspected when NMO antibodies (aquaporin-4 autoantibodies) are present and are pathologically classified as astrocytopathies.[6] Optic nerve is more commonly affected in MOGAD disease and more likely to have bilateral involvement than in NMOSD.[7] Despite presence of MOG Abs, this patient, by exclusion, is believed to have acute bilateral optic neuritis triggered by COVID-19 infection.

Once the diagnosis of optic neuritis is made, treatment is usually straightforward with intravenous methylprednisolone (typically 1 g per day for 3 days) often followed by oral prednisone (1 mg per kg per day for 11 days), with subsequent tapering over a period of 4 days.[2] Treatment is associated with a more rapid recovery of vision by 2 to 3 weeks with no impact on long-term visual function. Therefore, a follow-up with neurology and ophthalmology after discharge is recommended to reach a final diagnosis in cases like this.

Our case sheds light on an interesting and rare finding for presentation of COVID-19 infection with optic neuritis, which is usually associated with MS. Despite excellent response to high-dose steroids and the high suspicion for this case to be induced by COVID-19 infection, we recommended our patient to follow-up with outpatient neurology clinic to rule out other possible diagnoses such as MS as well as rarer conditions such as MOGAD and NMOSD.

Conclusion

Our patient described in this clinical vignette has signs and symptoms typical of acute bilateral demyelinating optic neuritis on his presentation. His evaluation with brain and orbital MRI along with lumbar puncture to determine the possibility of MS or other autoimmune disease was negative as well as other laboratory assessment. Therefore, we believe his infection with COVID-19 virus has triggered his immune system to present these findings. COVID-19 virus infection is causing a huge impact in the world as pandemic; its extent as clinical disease is still poorly understood and explained. Therefore, we share this interesting presentation to the world to add to the vast ways in which COVID-19 infection can present.

Acknowledgments

We would like to thank Charles M. McClain III, MD, for his assistance with providing the images for this article.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

Verbal informed consent was obtained from the patient for their anonymized information to be published in this article.

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