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CASE REPORT

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Myelin-oligodendrocyte glycoprotein antibody-associated optic neuritis following SARS-CoV-2 pneumonia: A case report

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Abstract

We report a case of a 60-year-old man with optic neuritis following severe acute respiratory syndrome coronavirus 2 pneumonia. He tested positive for serum myelin-oligodendrocyte glycoprotein IgG antibody and was treated with intravenous methylprednisolone (1 g/d) for 5 days followed by oral prednisolone taper. Six weeks after treatment, his symptoms resolved.

KEYWORDS
COVID-19, demyelinating disease, myelin-oligodendrocyte glycoprotein, optic neuritis, SARS-CoV-2

1 | INTRODUCTION

Aside from the direct infection of the respiratory system, cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-related extrapulmonary manifestations are being increasingly reported. Recently, there have been some case reports of optic neuritis following SARS-CoV-2 infection; at least, three were found to be related to myelin-oligodendrocyte glycoprotein antibody-associated disease (MOGAD).[1–3]

2 | CASE REPORT

An otherwise healthy 60-year-old man presented with an acute onset of right-eye visual loss and pain upon eye movement for 5 days. Six weeks before symptom onset, he was diagnosed with SARS-CoV-2 pneumonia, confirmed by the detection of SARS-CoV-2 on real-time reverse transcription-polymerase chain reaction (RT-PCR) using a nasopharyngeal swab. At the time, his symptoms included fever, cough, and dyspnea for 3 days. His chest radiograph showed ground-glass opacity in both basal lungs. He was admitted to another hospital and received oral favipiravir (3600 mg/d) on day 1 and 1600 mg/d from day 2 to 5 along with prednisolone (60 mg/d). Fourteen days after admission, he was discharged without any residual symptoms or visual abnormalities.

At our hospital, the patient had normal vital signs with a SpO2 level of 97%. His best-corrected visual acuity was 20/200 in the right eye and 20/30 in the left eye. The patient had a relative afferent pupillary defect (RAPD) in his right eye, and his ocular motility was limited by pain. Fundoscopic examination showed swelling in the right optic disc. Other general and neurological examinations were normal. Laboratory findings including complete blood count, blood chemistry, and renal and liver function were all unremarkable. Serology tests for human immunodeficiency virus and hepatitis B and C virus, Venereal Disease Research Laboratory test, and *Treponema pallidum* hemagglutination test were all negative.

Brain magnetic resonance imaging (MRI) (Figure 1) indicated right optic neuritis. Lumbar puncture was performed and revealed a colorless cerebrospinal fluid (CSF) with opening and closing pressures of 11 and 8 cmH_2O, respectively. The CSF glucose and protein levels were 66.58 mg/dL and 38.44 mg/dL, respectively. The total CSF cell count was 3 cells/mm^3 of red blood cells, and no white blood cells were detected. Nasopharyngeal RT-PCR SARS-CoV-2 and serum SARS-CoV-2 IgM were negative, whereas SARS-CoV-2 IgG was positive (immunochromatographic assay). Tests for serum and CSF aquaporin 4 IgG and oligoclonal bands were negative.

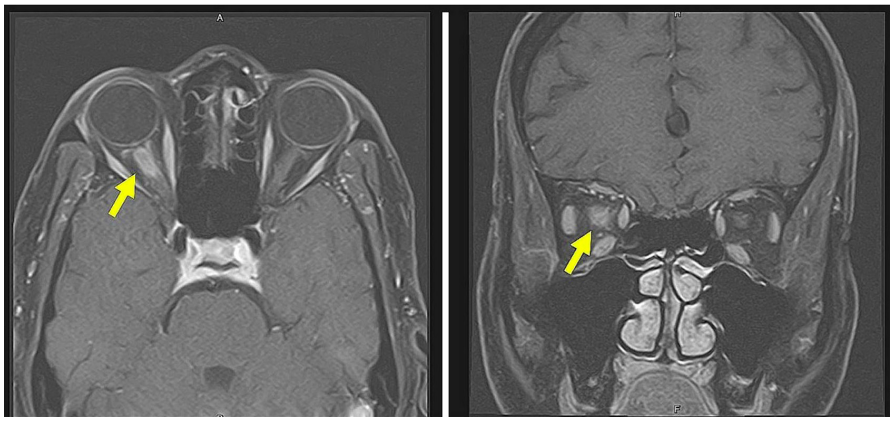

FIGURE 1 Axial and Coronal T-1-weighted post-gadolinium brain MRI show abnormal enhancement and enlargement in the anterior intraorbital part of right optic nerve (arrow), indicative of right optic neuritis

Myelin-oligodendrocyte glycoprotein (MOG) IgG was detected with the serum titer of 1:200, using cell-based assays.

The patient was treated with intravenous methylprednisolone (1 g/d) for 5 days, followed by a regimen of oral prednisolone taper (starting with prednisolone 60 mg/d). Eight days after treatment, ocular pain and visual acuity improved significantly to the level of 20/70. Outpatient follow-up 6 weeks later revealed complete recovery of the affected eye.

3 | DISCUSSION

Recently, several cases of inflammatory neurological disorders were reported to be associated with SARS-CoV-2 infection. However, in most cases, SARS-CoV-2 is not detected in the CSF.[4] This suggests that the etiology is less likely to be a direct viral infection but a concomitant post-viral immune-mediated response.

Here, we report a case of MOGAD following SARS-CoV-2 infection. The patient had an acute visual loss with pain upon eye movement and disc swelling with RAPD positivity in the right eye, prompting a diagnosis of optic neuritis. His brain MRI findings were compatible with MOG antibody-associated optic neuritis with involvement of the anterior part of the right optic nerve and marked swelling. MOG IgG antibodies were detected in his serum, confirming the diagnosis of MOGAD.

Although we did not test for CSF RT-PCR SARS-CoV-2 due to unavailability of laboratory testing, we postulate that post-viral mediated immune response rather than primary viral infection led to MOGAD based on the following: first, the onset of optic neuritis in this patient was 6 weeks after SARS-CoV-2 infection, which is within the onset period of diseases caused by cell-mediated immunity (CMI).[5] Second, CMI is likely to be triggered by an immune response to SARS-CoV-2 infection, which cross-reacts with MOG in the optic nerve. Finally, the etiological link between SARS-CoV-2 infection and CMI response has been considered and supported by several studies. Similar cases have reported the onset of optic neuritis after SARS-CoV-2 infection ranging from 2 to 6 weeks and all patients were successfully treated with corticosteroids.[1–3]

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CONFLICT OF INTEREST

Authors declare no Conflict of Interest for this article.

ETHICAL APPROVAL AND CONSENT TO PARTICIPATE

The present report was approved by the Ethics Committee of Buriram Hospital. The patient gave informed consent.

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