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CASE REPORT

Myelin oligodendrocyte glycoprotein-associated transverse myelitis after SARS-CoV-2 infection: A case report

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Abstract

BACKGROUND

Cases of myelin oligodendrocyte glycoprotein (MOG) antibody-related disease have a history of coronavirus disease 2019 infection or its vaccination before disease onset. Severe acute respiratory syndrome virus 2 (SARS-CoV-2) infection has been considered to be a trigger of central nervous system autoimmune diseases.

CASE SUMMARY

Here we report a 20-year male with MOG-associated transverse myelitis after a SARS-CoV-2 infection. The patient received a near-complete recovery after standard immunological treatments.

CONCLUSION

Attention should be paid to the evaluation of typical or atypical neurological symptoms that may be triggered by SARS-CoV-2 infection.

Key Words: Myelin oligodendrocyte glycoprotein antibody-associated encephalomyelitis;

Myelin oligodendrocyte glycoprotein antibody-associated disease; SARS-CoV-2; COVID-19; Case report

Core Tip: Here we present a case of myelin oligodendrocyte glycoprotein-associated disease after severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) infection. The patient received a near-complete recovery after standard immunological treatments. We suggest that attention should be paid to the evaluation of typical or atypical neurological symptoms that may be triggered by SARS-CoV-2 infection, and focus on the exclusion of coexisting autoimmune reactions or diseases.

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INTRODUCTION

Many cases of coronavirus disease 2019 (COVID-19) have been reported with co-existing neurological symptoms. Several co-exsiting autoimmune neurological diseases, including central (limbic and brainstem encephalitis, acute disseminated encephalomyelitis, and myelitis) and peripheral neurological diseases (Guillain-Barré and Miller fisher syndromes), have been reported[1]. The correlation between COVID-19 and neurological symptoms is yet not clear, with the possible mechanisms of autoimmunity and inflammation. Currently, the diagnosis standards and treatment options for COVID-related neurological diseases are ambiguous, leading to a possibly underestimated number of total cases and posing new challenges for clinical work in the neurological discipline.

Myelin oligodendrocyte glycoprotein (MOG) is a member of the immunoglobulin superfamily[2]. It is specifically expressed on the membrane surface of oligodendrocytes and the outermost layer of the myelin sheath, and its expression level usually reflects the degree of myelination. MOG has been proven to be one of the most common antigens in demyelinating diseases. MOG antibody-related disease (MOG-AD) accounts for 1.2% to 6.5% of all adult demyelinating syndromes, with acute disseminated encephalomyelitis, optic neuritis and myelitis as the common clinical symptoms[3].

In this case repot, we report a case of rapidly progressing acute myelitis presented with motor sensory disorders of both lower extremities and urinary retention after severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2) infection. The patient was further diagnosed with MOG-associated transverse myelitis (TM) after being tested positive for anti-MOG-IgG in the cerebrospinal fluid (CSF). We also reviewed published cases of MOG-AD associated with SARS-CoV-2 infection, intending to improve clinicians' understanding of MOG-AD after SARS-CoV-2.

CASE PRESENTATION

Chief complaints

A 20-year-old young male of Asian ethnicity was admitted to our hospital for lower limb weakness and sensory disturbance and urinary retention for 3 days.

History of present illness

Eight days prior to admission, the patient developed weakness and a fever with chills, and was diagnosed with COVID-19 after conformation of SARS-CoV-2 infection by a pharyngeal swab reverse transcription-polymerase chain reaction test. No other symptoms such as cough, sputum, chest tightness, or shortness of breath were present at that time. After 5 days of consistent fever, the patient began to develop symmetrical weakness in lower extremities, accompanied by symmetrical sensory impairment below the waist, inability to walk by himself, and difficulty in urination.

History of past illness

The patient was previously healthy.

Personal and family history

He had previously received the SARS-CoV-2 adenovirus vaccine before infection and had no symptoms or discomfort at the time of vaccination.

Physical examination

At administration, the patient had a fever of 38.5 °C. Neurological examination revealed pyramidal tract impairment and

sensory abnormalities, including decreased muscle strength in both legs (3/5 in medical research council scale of muscle strength rating system) and diminished tendon reflex, accompanied with hypesthesia below the T9 dermatome level [modified Rankin scale (mRS) 4 and expanded disability status scale (EDSS) 7.0]. His abdominal wall reflexes were elicited only at the left T7-T8 plane. And urinary retention resulted in his need for urinary catheterization.

Laboratory examinations

The initial laboratory tests showed an elevated level of white blood cell count (15.69 × 10^9/L), C-reactive protein (84.590 mg/L), D-dimer (1.17 mg/L, fibrinogen equivalent units), and erythrocyte sedimentation rate (52 mm/hour), with no other general examination remarkable. A lumbar puncture was therefore performed. The pressure of the cerebral spinal fluid was remarkably over 330 mmH_2O, with a raised white blood cell count (66 × 10^6/L), mononuclear cell percentage (91%), and elevated protein level (0.26 g/L). Oligoclonal bands were seen in both serum and CSF with the same pattern. A CSF test was positive for anti-MOG antibody (titration 1:32), while was negative for other antibodies, especially anti-aquaporin 4 antibodies.

Imaging examinations

A magnetic resonance imaging (MRI) scan of the spinal cord revealed an abnormal signal in the C3-C6, T7-T8 segment resembling demyelinating lesion (Figure 1), presuming a diagnosis of TM. Neither typical glassy nor solid shadows were observed in his lung from an immediate chest computerized tomography scan (Figure 2).

FINAL DIAGNOSIS

Based on this patient's recent history of SARS-CoV-2 infection, clinical presentation, laboratory findings, MRI, and CSF analysis, the diagnosis of MOG-TM was supported.

TREATMENT

Pulsed methylprednisolone therapy [1000 mg × 3 days; 500 mg × 3 days; 250 mg × 3 days; 120 mg × 3 days followed by 65 mg (1 mg/kg/day) orally] was administered by our medical team.

OUTCOME AND FOLLOW-UP

The patient responded well to steroid treatment with gradual improvements in symptoms. After a one-week of treatment, the patient's muscle strength of the lower limbs was restored (4 +/5 in medical research council scale of muscle strength rating system score). He could walk independently and was mostly self-care, despite slight sensory abnormalities and mild urinary retention, for which a catheter was still needed. At re-exam in the second week, an MRI scan showed reduced volume of the abnormal signals compared with the previous scan (Figure 3). At discharge, the mRS score was at 2, and the EDSS score was at 3.5. A prescription of a long-term oral prednisolone until further reevaluation was given, with a slow tapering plan.

DISCUSSION

With the accumulation of cases affected by the COVID-19 pandemic, studies have found that a range of neurological complications such as cerebrovascular disease, encephalitis, and myelitis could occur in addition to the respiratory and cardiovascular symptoms[4-7], which is gaining more attention in clinical practice. COVID-19 related myelitis was first reported by Zhao *et al*[8], despite a lack in CSF and spinal MRI findings.

The number of reported MOG-AD cases seems slightly higher than that in the pre-pandemic period of COVID-19, and its coincidental occurrence should not be neglected[9,10]. COVID-19-associated MOG-AD can occur in patients of all ages, but is more commonly seen in younger patients. In these patients, myelitis or TM is usually accompanied by encephalitis (acute disseminated encephalomyelitis) and optic neuritis[10,11]. Our patient presented with typical neurological symptoms including lower extremity weakness, sensory impairment and urination retention, which is consistent with the reported cases. Similar to other previously reported cases, in the cross-sectional MRI scans of this reported patient, the spinal cord lesion showed a typical "cloudy" and "H-shaped" pattern[11-14]. A recent multimodal meta-analysis exhibited that both structural and functional alterations of right superior temporal gyrus, left insula and right orbito-frontal cortex have been found in COVID-19 patients compared with healthy persons. This study prompted that the cortex is more susceptible than the white matter in brain after SARS-CoV-2 infection, which may accordingly make an explanation why patients with SARS-CoV-2 associated MOG-TM mainly manifest the dysfunction of the grey matter in spinal cord[15]. However, direct evidence especially the multimodal imaging and autopsy examination of the spinal cord demonstrating the pathophysiological link, is still lacking to date, and further clinical and preclinical studies are still needed to reveal the underlying mechanisms of MOG-TM associated with COVID-19.

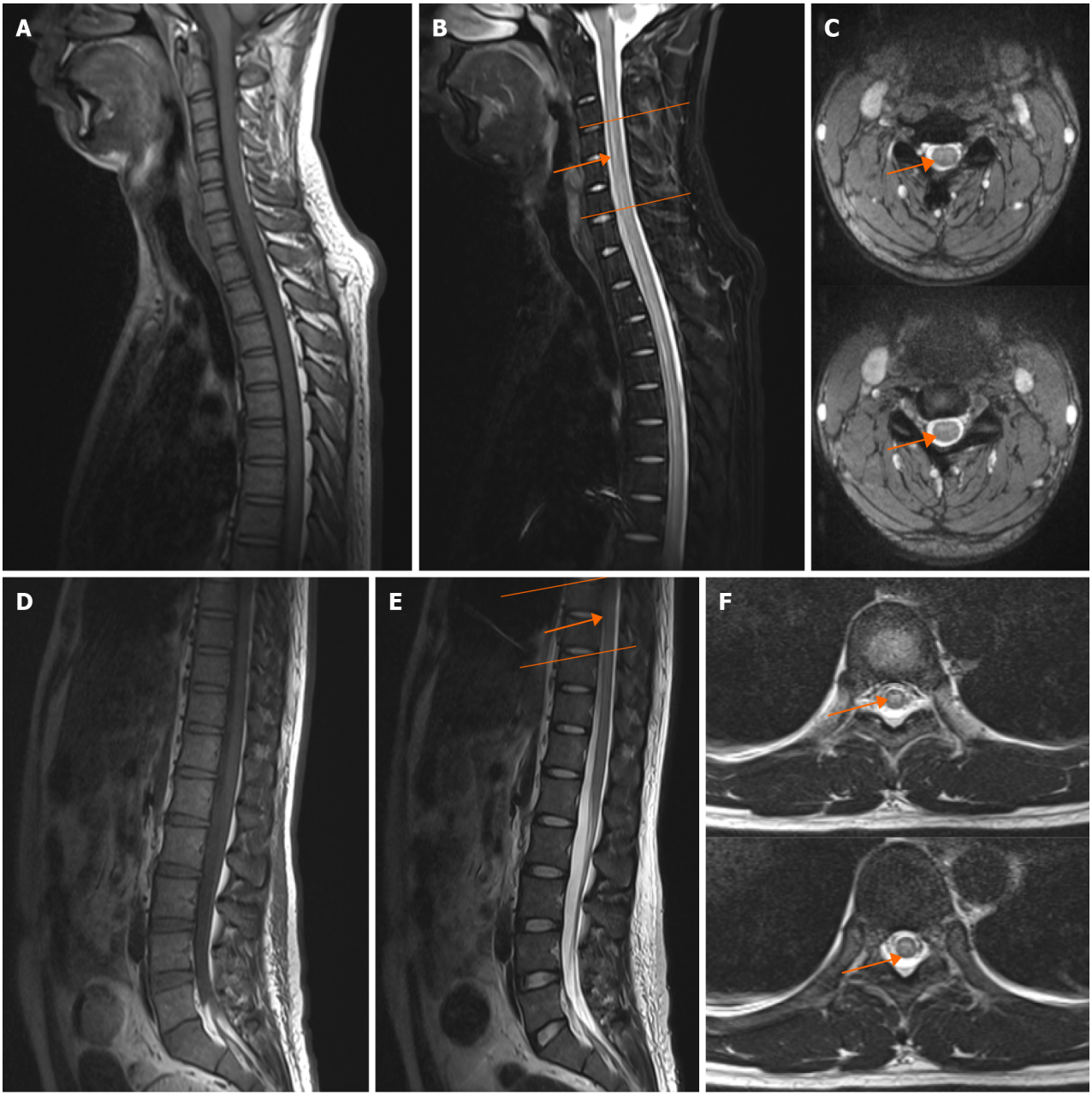

Figure 1 A magnetic resonance imaging scan of the thoracic spinal cord of the patient before treatment. A: Sagittal T1WI sequence of the cervical spine; B: The sagittal T2WI-FS sequence of the cervical spine shows hyperintensity in the long central segment of the spinal cord C3-C6 (arrow); C: Axial T2WI sequence of the cervical spine shows hyperintensity in the central spinal cord at the C3-C6 level (arrow); D: Sagittal T1WI sequence of the lumbar spine; E: Sagittal T2WI sequence of the lumbar spine shows hyperintensity from the central long segment of the T7-T8 spinal cord (arrow); F: Axial T2WI sequence of the lumbar spine shows hyperintensity at the T7-T8 level (arrow).

It is difficult to confirm the causality of SARS-CoV-2 infection and TM. Considering the characteristics of the cases available so far and the temporal relationship between SARS-CoV-2 infection and MOG-AD, it is very likely that this was an inflammatory para-infection or post-infection phenomenon, supporting the hypothesis of autoimmune process rather than viral damage directly. The homologous trimeric spike protein S in SARS-CoV-2 membrane has a receptor binding domain that recognizes angiotensin-converting enzyme 2 (ACE2) on target cells and the virus can subsequently fuse with the target cell membrane to complete viral replication and infection[16,17]. Since ACE2 receptors are present on brain glial cells and neurons, it is suggested that the nervous system is also highly susceptible to SARS-CoV-2[18]. The possible route for virus infection in the central nervous system may be through the central protrusion of the olfactory cells to the olfactory bulb or through the disruption of the blood-brain barrier by infecting vascular epithelial cells, which is further evidenced by the detection of SARS-CoV-2 genetic material in patients' CSF[19,20]. However, SARS-CoV-2 polymerase chain reaction test was only positive in the CSF of a very small number of patients presenting with encephalitis or encephalomyelitis, and is usually negative in patients presenting with simple myelitis for the time being, contradicting the idea of direct SARS-CoV-2 infection on neuron[1,9]. MOG-AD can also occur secondarily to the infection of other viruses such as herpes simplex virus and Epstein-Barr virus, suggesting that viral infections may be triggers for the development of immune-mediated responses, but is not specific to a defined pathogen[3]. Previous study identified

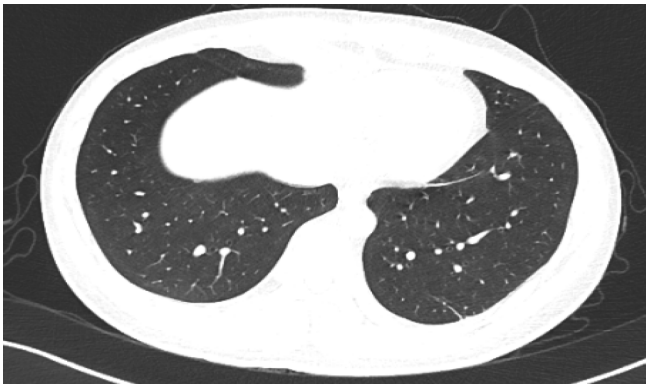

Figure 2 A chest computerized tomography scan of the patient. Chest computerized tomography showed mild interstitial lesions in both lower lungs without the typical ground glass-like or solid shadows of severe acute respiratory syndrome virus 2 infection.

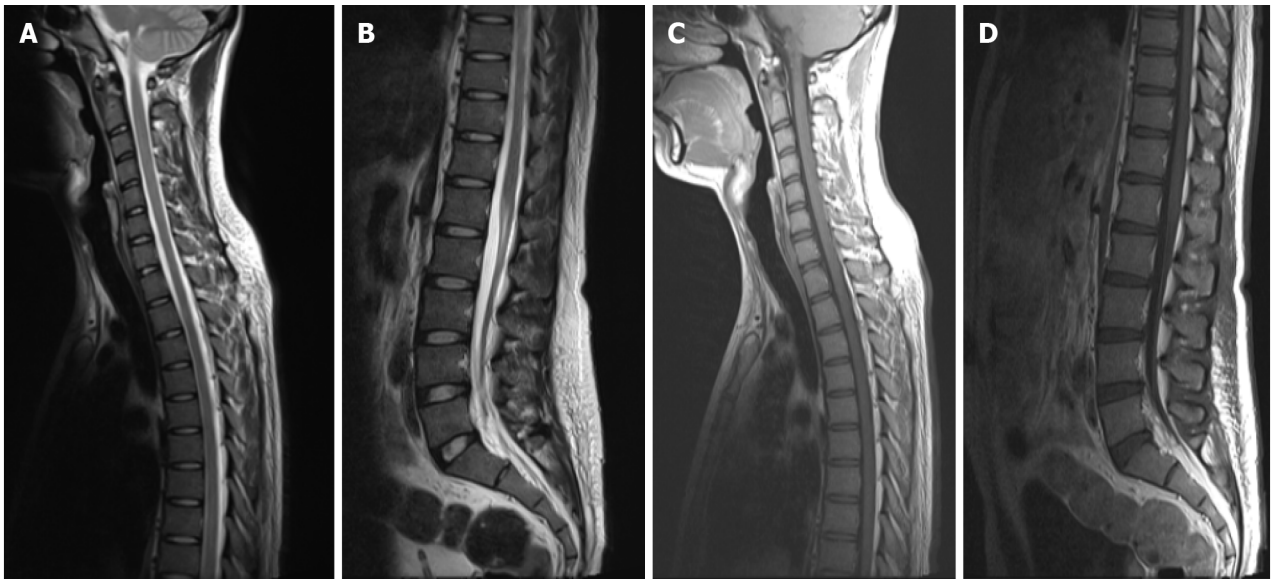

Figure 3 Magnetic resonance imaging scan of spinal cord one week after methylprednisolone treatment. A: Sagittal T2WI sequence of the cervical spine shows a reduced range of long T2 signals in C3-C6 and T7-T8 segments compared with the previous one (arrows); B: Sagittal T2WI sequence of the lumbar spine: A reduced range of long T2 signals in T7-T8 segments compared with the previous one (arrows); C: Sagittal enhancement sequence of the cervical spine: No abnormal enhancement was seen; D: Sagittal enhancement sequence of the lumbar spine: No abnormal enhancement was seen.

inflammatory markers [such as C-reactive protein, interleukin (IL)-2R, IL-6, IL-10, tumor necrosis factor-α] are significantly elevated in patients with severe COVID-19 infection, which underlie the cytokine inflammatory storm which leads to hyperpyrexia and respiratory failure[21,22]. However, in this reported case, neurological damage is much more evident than respiratory symptoms, suggesting that anti-MOG antibody activation, rather than inflammation itself, was playing a pivotal role. In addition, MOG-AD was found to be closely associated with SARS-CoV-2 vaccination. Indeed, MOG-AD after vaccination may induce more severe symptoms compared to non-vaccine related MOG-AD, represented by longer focal involvement of segments and even require multiple immunotherapy in some cases[23], while routine lab tests of CSF could be normal[23,24]. Therefore, according to the currently proposed criteria for a "probable" causal relationship between SARS-CoV-2 vaccination and neurological complications, SARS-CoV-2 infection or vaccination may lead to the process of molecular mimicry and epitope spread, which may contribute to the inflammation-associated MOG-Ig as a non-specific trigger releasing and finally result in MOG-AD[25].

As for disease regression, in this group of patients, immunotherapy with adequate steroid hormones is most commonly adopted in clinical practice, and most patients have good response to steroid treatments and are able to achieve clinical remission[1]. Immunoglobulin and plasma replacement are alternative clinical strategies. As with typical MOG-AD, patient with MOG-AD after SARS-CoV-2 infection could receive complete or near-complete recovery after treatments, and relapse cases are rare[1,11]. However, given the specificity of MOG antibodies, the current epidemiological setting of SARS-CoV-2, and the limited follow-up time, and it is recommended that such patients should be followed up for a longer period of time for assessment or to prevent relapse.

CONCLUSION

Here we present a case of MOG-AD after SARS-CoV-2 infection and a literature review of available reports. We suggest that attention should be paid to the evaluation of typical or atypical neurological symptoms that may be triggered by SARS-CoV-2 infection, and focus on the exclusion of coexisting autoimmune reactions or diseases. We suggest that SARS-CoV-2 infection may play a potential role in triggering and driving inflammation in the pathogenesis of MOG-AD, but direct pathophysiological evidence is still lacking. More prospective studies are needed to elucidate this possible association.

FOOTNOTES

Author contributions: Zheng JR and Chen XH conceptualized and designed the case report, drafted the initial manuscript and revised it; Liu ZG contributed to the conception and design of the manuscript; Chang JL supervised and coordinated the manuscript; Hu J, Lin Z, Lin KH and Lu BH were involved in patient care and data collection; All authors reviewed and approved of the manuscript.

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