

## COVID-19-induced longitudinal extensive myelitis

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Clinical manifestations of coronavirus disease 2019 (COVID-19) in the spinal cord, such as longitudinal extensive myelitis (LEM), are rare.<sup>[1,2]</sup> The virus can cause neurological manifestations via the ACE-2 receptors. Moreover, systemic inflammation triggered by the COVID-19 infection may lead to the development of myelitis.<sup>[3]</sup>

A 39-year-old female suffering from weakness and severe pain in the lower limbs and urinary retention was admitted to the emergency department. The patient's complaints had started as numbness and dysesthesia in the right leg and gradually worsened within three to four days. The patient had been diagnosed with COVID-19 15 days ago and fully recovered from moderate respiratory symptoms, such as cough and dyspnea, by favipiravir treatment (8 g for five days). The patient's medical and family history for neurological disorders were unremarkable. The initial neurological examination revealed loss of muscular strength in the right lower limb 1/5 and left lower limb 2/5, hyperreflexia in the lower limbs, and generalized allodynia. All modalities of sensation were diminished at the T10 segmental level. The nasopharyngeal and oropharyngeal swabs were positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by polymerase chain reaction. Serum laboratory test results revealed a mildly elevated white blood cell count ( $14.00 \text{ K/mm}^3$ ) and lymphopenia ( $0.7 \times 10^9 /\text{L}$ ). The other tests were negative for nutritional deficiencies and autoimmune

diseases. The evaluation of the cerebrospinal fluid (CSF) showed pleocytosis (white cell count  $110/\text{mm}^3$ ) with mostly polymorphonuclear leukocytes (90%), elevated protein (67 mg/dL), and normal glucose (78 mg/dL; concurrent serum glucose level was 96 mg/dL). The CSF immunoglobulin (Ig) G index was normal (0.6), and oligoclonal bands, neuromyelitis optica IgG, and antimyelin oligodendrocyte glycoprotein antibodies were not present in the CSF. The CSF culture was negative for bacteria and SARS-CoV-2, including EBV and HSV. Paraneoplastic and limbic encephalitis panels of CSF were also negative. Cerebrospinal fluid polymerase chain reaction for the COVID-19 virus was not performed. Serology for SARS-CoV2 in the CSF demonstrated infection [anti-SARS-CoV-2 IgG: 5.47 (negative<1)]. Electromyography displayed normal motor and sensory nerve conduction velocity in the upper and lower limbs. Cervical and thoracic magnetic resonance imaging (MRI) demonstrated a long segment of T2 hyperintensities within the central cord extending from C2 to T10 vertebrae (Figure 1). The patient was treated with intravenous (IV) methylprednisolone 1 g/day for five days. At the end of the fifth day, ceftriaxone (4 g/d) and acyclovir (2250 mg/d) were added to the treatment. The patient had no improvements, and clinical symptoms continued to progress. The patient received five sessions of plasma exchange, and additionally, IV methylprednisolone 1 g was administered for another

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**Figure 1.** (a) Longitudinal magnetic resonance imaging sequence of cervical and (b) thoracic extensive myelitis. The T2-weighted sequences demonstrate the long segment of T2 signal elevation in the spinal cord center from C2 to T10.

10 days. The patient was transferred to an inpatient rehabilitation facility.

The clinical manifestation of LEM induced by COVID-19 observed during infection has been reported in two case studies.<sup>[1,4]</sup> The World Health Organization defined the associations between SARS-CoV-2 and myelitis/myelopathy based on the presence of anti-SARS-CoV-2 IgG in the CSF and classified it as a “confirmed” case.<sup>[5]</sup> To the best of our knowledge, this case is one of three previously reported serologically confirmed cases.<sup>[1,4]</sup> Unlike other cases in the literature, which had clinical improvements after treatment, no significant improvement was observed in our patient, which can be explained by the patient’s advanced clinical findings.

In conclusion, the manifestation of LEM during a COVID-19 infection is exceedingly rare and atypical. Early recognition and management of this infrequent clinical condition is considerable in terms of preventing chronic disabling sequelae.

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