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COVID-19-associated acute necrotizing myelitis

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A 69-year-old otherwise healthy woman presented to our clinic with irradiated cervical pain, imbalance, and motor weakness and numbness in the left hand, which had been ongoing for 7 days. Eight days before the onset of these symptoms, she had fever and dry cough.

On admission, her neurologic examination showed right facial and left hand hypoesthesia, subtle left hand interosseous weakness, and general hyperreflexia.

MRI of the brain was normal, whereas spinal cord images (figure 1, A and D) showed T2-hyperintensity extending from the medulla oblongata to C7, involving most of the cord with diffuse patchy enhancing lesions, suggesting acute transverse myelitis.

Extensive diagnostic workup was performed, showing negative results in blood test for infectious, autoimmune diseases (including myelin oligodendrocyte glycoprotein and aquaporin-4 antibodies), and other potential causes such as vitamin deficits or antiphospholipid syndrome.

The patient's CSF analysis showed a traumatic puncture (75 erythrocytes/μL), mild lymphocytic pleocytosis (75 cells/μL, 98% lymphocytes), hyperproteinorraquia of 2.83 g/L, normal adenosine deaminase, and glucose results; IgG index was normal, no oligoclonal bands were present, and bacterial culture and viral multi-PCR test were also negative. The presence of neuronal surface antibodies was also ruled out. Thoracoabdominal CT scan was negative for tumor and lymphadenopathy. SARS-CoV-2 PCR was positive in nasopharyngeal swab and negative in CSF. No cytokine levels were examined neither in serum nor CSF.

Treatment with methylprednisolone 1 g IV for 5 days resulted in initial improvement. However, a few days later, her clinical condition worsened markedly: she developed sensory motor deficits in both hands and paraparesis with sphincter incontinence.

A new spinal MRI was performed (figure 1, B and E), showing transversally and caudally progression until T6 level with similar enhancement and a new area of central necrosis at the T1 level with peripheral enhancement.

Treatment with plasma exchange and other course of methylprednisolone pulses for 5 days with posterior slow oral prednisone tapering resulted in improvement of strength and motor function, until being able to walk with assistance, use electronic devices such as typing on her mobile phone or write with some difficulties, remaining left leg moderate weakness and no sphincter control. After 4 weeks from the clinical onset, she continues improving slowly and performing physical and occupational therapy.

Spinal MRI postplasmapheresis (figure 1, C and F) showed substantial decrease in myelitis extension and enhancement, but central necrosis at the C7-T1 level remained unchanged.

Acute necrotizing myelitis (ANM) is a rare inflammatory disorder of the spinal cord. Only a few cases have been associated to inflammatory diseases such as neuromyelitis optica or vasculitis, paraneoplastic mechanisms, or adverse effect of new oncologic treatments.[1–3]

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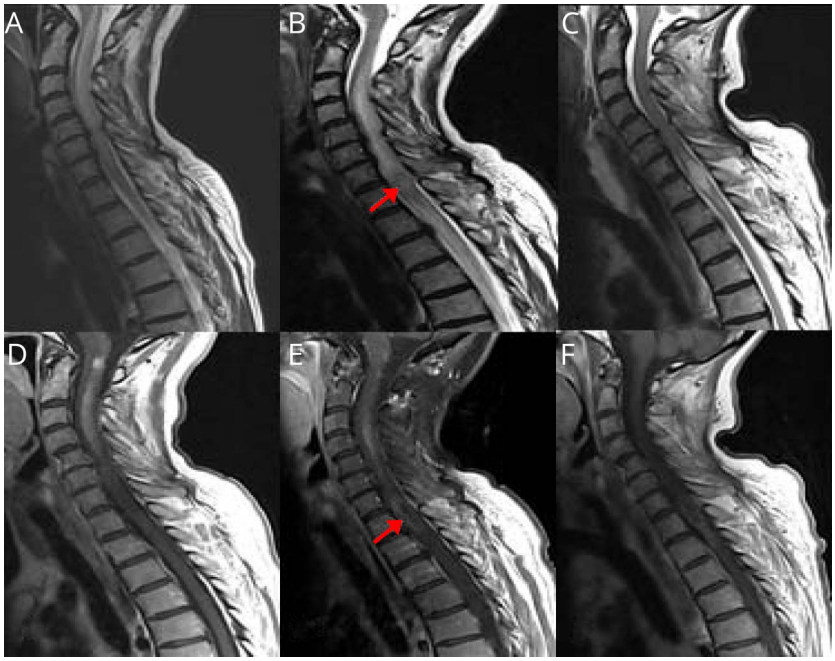
Figure Spinal MRI evolution of LETM with necrotizing area



Sagittal T2-weighted (A–C) and sagittal postcontrast T1-weighted (D–F). Baseline performed (A and D), follow-up 1 week (B and E), and plasmapheresis posttreatment (C and F). A LETM is seen in the cervical spinal cord (A) with patchy enhancement (D). Progressive swelling of the spinal cord and a new necrotizing area (B: arrow) that shows peripheral enhancement (E: arrow). Significant decreases of both myelitis extension (C) and enhancement (F) after plasmapheresis treatment, with necrosis area in evolution. LETM = longitudinal extensive transverse myelitis.

Spine MRI in ANM usually shows hypointense T1 signal and corresponding increase in T2 signal and, characteristically, hemorrhage, cavitation, and postcontrast enhancement could also be seen.

The exact pathogenesis of ANM remains obscure, and analogously to acute necrotizing encephalitis (ANE), an inflammatory response ("cytokine storm") secondary to a viral infection has been postulated as a possible cause.[4] Human coronaviruses are a group of respiratory viruses that can naturally reach the CNS in humans through hematogenous or neuronal retrograde route and could potentially be associated with neurologic symptoms.[5] A systemic cytokine production due to the SARS-CoV-2 infection has been suggested to contribute to the pathophysiology of severe coronavirus disease 2019 (COVID-19).[6] Recently, a case of ANE caused by the new coronavirus infection has been reported.[7]

In our patient, the presence of a longitudinal extensive transverse myelitis with subsequent worsening along with development of a necrotic area, associated with focal swelling, peripheral enhancement, and hypointense foci on T2 images, led us to the diagnosis of ANM in a patient COVID-19 positive. Immunomodulatory treatment such as steroids or plasmapheresis can result in neurologic improvement as the patient reported here.

Additional studies are needed to better define the potential role of human coronaviruses in the pathogenesis and the effectiveness of any therapeutic measure in the management of ANM.

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