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Case report

Acute myelitis and SARS-CoV-2 infection. A new etiology of myelitis?

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A B S T R A C T

The etiological agent of coronavirus disease-19 (COVID-19), SARS-coronavirus-2 (SARS-CoV-2), emerged in Wuhan, China, and quickly spread worldwide leading the World Health Organization (WHO) to recognize it not only as a pandemic but also as an important threat to public health. Beyond respiratory symptoms, new neurological manifestations are being identified such as headache, ageusia, anosmia, encephalitis or acute cerebrovascular disease. Here we report the case of an acute transverse myelitis (TM) in a patient with SARS-CoV-2 infection detected by the nasopharyngeal swab technique but not in cerebrospinal fluid (CSF) analysis. Anti-herpes simplex virus (HSV) 1 and varicella-zoster IgM antibodies were not detected in serum samples and spinal and brain magnetic resonance imaging (MRI) showed no abnormal findings. This case remarks that COVID-19 nervous system damage could be caused by immune-mediated mechanisms.

1. Introduction

Coronavirus disease-19 (COVID-19) is caused by SARS-CoV-2 infection. This novel virus emerged in Wuhan, China. It spread rapidly around the globe, becoming a threat to public health around the world and a pandemic as recognized by the World Health Organization (WHO). Usually, it is defined by a severe acute respiratory syndrome that leads to high morbidity and mortality. Lately, new clinical manifestations have been reported. The following case presents a patient with SARS-CoV-2 infection and neurological manifestations in the form of transverse myelitis (TM).

2. Case presentation

A 50-year-old man was admitted to the emergency department with dysesthesia at lower limbs and genital area that evolved to loss of strength and inability to maintain stable standing position and urinary incontinence of progressive establishment throughout the last 3 days. He also referred dysthermic sensation for the last 4 days, as well as mild low back pain, asthenia and occasional coughing episodes without respiratory distress.

Measured blood oxygen saturation was 98%. Hemodynamic stability was properly assessed. Neurological examination showed isochoria, normoreactive pupils and preserved cranial nerves function, no stiff neck nor other meningeal signs - but marked hypoesthesia with a T6 metameric level. Muscular balance: loss of strength in lower limbs 2/5, keeping upper limbs unaffected 5/5. Normoreflexia in the upper limbs and hyperreflexia in the lower limbs. Plantar response reflexes were equivocal bilaterally.

The patient was evaluated by a neurologist and admitted to Internal Medicine with suspected diagnosis of acute transverse myelitis and respiratory symptoms in the context of SARS-CoV-2 pandemic.

Labs showed an increase in inflammatory markers (C-reactive protein, lactate dehydrogenase, ferritin). Chest X-ray ruled out pneumonia. Anti-herpes simplex virus (HSV) 1 and varicella-zoster IgM antibodies were not detected in serum samples. Cervico-thoracic-lumber and brain magnetic resonance imaging (MRI) showed no abnormal findings except for mild herniation of two intervertebral discs (C5-C6) (Fig. 1). Electromyography (EMG) showed no neurogenic change. Cerebrospinal fluid (CSF) analysis showed no pleocytosis nor proteinorrachy. Both CSF culture and reverse transcription polymerase chain reaction (RT-PCR) were negative for bacteria and virus, including SARS-CoV-2. However, RT-PCR for SARS-CoV-2 performed in a nasopharyngeal swab sample showed positive.

There were no other remarkable alterations in respiratory function. Given the laboratory test results, we initiated treatment on Hydroxychloroquine plus Lopinavir/Ritonavir, Dexamethasone
Baig AM, Khaleeq A, Ali U, Syeda H. Evidence of the COVID-19 virus targeting his nervous system: a case report. J Clin Neurosci. 2020;80:280-281.

Infectious TM due to other etiologies is usually provoked by parainfectious mechanisms, e.g., autoimmune response. SARS-CoV-2 infection may be responsible for the development of a systemic inflammatory response and take part in the inflammatory cascade and cytokine release syndrome. It is still debatable whether myelitis occurs directly from the viral infection or as an autoimmune sequel [7].

The incidence of acute myelitis associated with COVID-19 infection is unknown. Four case reports of similar cases were reported up to the current date of writing this case linking COVID-19 to acute myelitis as a neurological complication [8–11]. Also, a case of possible atypical demyelinating event of the CNS and an acute necrotizing myelitis following COVID-19 has been published [12–13].

4. Conclusion

Although the pathogenic potential and mechanism of SARS-CoV-2 are unknown in detail, COVID-19 may affect the CNS and have various acute or delayed neurological complications, so we must be alert to the presence of neurological symptoms in these patients.

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