Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Case report

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

Daniel Águila-Gordo [a], José Manuel Flores-Barragán [b], Ferran Ferragut-Lloret [c], Jaime Portela-Gutierrez [c], Beatriz LaRosa-Salas [d], Lourdes Porras-Leal [d], José Carlos Villa Guzmán [c]

[a] Cardiology Department, University Hospital of Ciudad Real, Obispo Torija s/n, 13005, Ciudad Real, Spain
[b] Neurology Service of Hospital General de Ciudad Real, Spain
[c] Oncology Service of Hospital General de Ciudad Real, Spain
[d] Internal Medicine of Hospital General de Ciudad Real, Spain

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ABSTRACT

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 thread 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-Coronavirus-2 (SARS-CoV-2) infection. This novel virus emerged in Wuhan, China. It spread rapidly around the globe, becoming a thread 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 maintin 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 inflamatory 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-Coronavirus-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

E-mail address: danielaguilagordo@gmail.com (D. Águila-Gordo)

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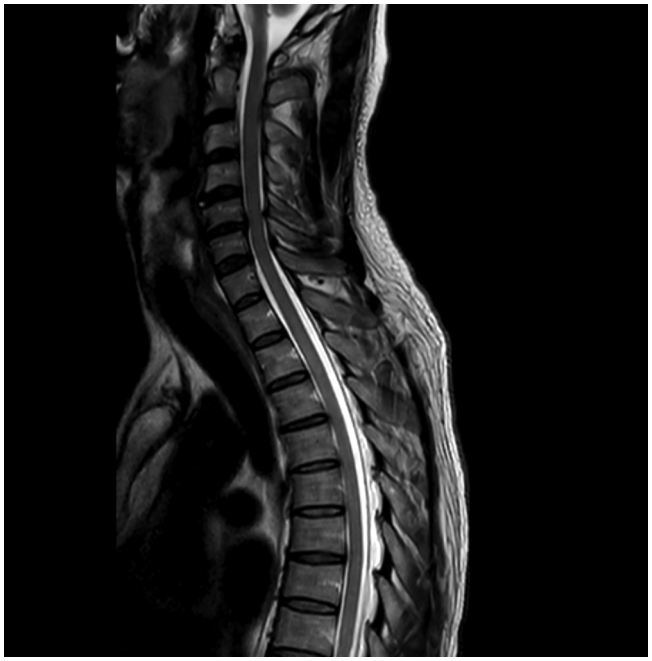

Fig. 1. Sagittal T2-weighted scan shows mild discoarthrosic changes at the C5-C6 level with obliteration of the subarachnoid space without pathological repercussion.

and immunoglobulins. Evolution was favorable and our patient gradually recovered his sensitivity.

3. Discussion

The pathogenesis of transverse myelitis (TM) is varied. Its etiologies include a wide spectrum of entities that involves systemic autoimmune diseases, recurrent autoimmune diseases of the central nervous system and infectious causes. When the original cause is unknown it is called idiopathic TM. However, it has been estimated that between 30 and 60% of idiopathic cases are the consequence of some previous respiratory, gastrointestinal or systemic disease.

In infectious TM the injury may follow two mechanisms: either direct microbial invasion of the central nervous system, or the systemic response to this infection. In the latter, the immune system mistakenly attacks the patient's own tissue, causing inflammation and damage to myelin within the spinal cord. Some of the main infectious agents involved are: enterovirus, varicella zoster virus, herpes virus, type 1 human T-cell leukemia virus, Zika virus [1,2]. . .

SARS-CoV-2 might be one of these infectious agents as well. Albeit many features of its pathophysiological pathways are still unknown, it seems that neurological manifestations are a matter of fact [3–5]. The central nervous system, as well as the lungs, heart, kidneys or intestines, express ACE2 receptors, target of SARS-CoV-2 [4].

The case we present shows SARS-CoV-2 as a possible etiology of TM, using a pathogenic mechanism already described for other virus. Genetic material and viral proteins can be detected in tissue samples from the nervous system, such as CSF, suggesting this virus can directly invade the nervous system and cause damage. Nevertheless, this mechanism is not very likely in our case because SARS-CoV-2 RNA was not detected in CSF. There are papers that highlight a positive RT-PCR test for SARS-CoV-2 in CSF in the context of encephalitis, which is not a common finding in the rest of COVID-19 neurological manifestations [5,6]. This highly suggests that there should be other explanations for neurological symptoms in the context of COVID-19.

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 mielitis following COVID-19 has been published [12–13].

4. Conclusion

Although the pathogenic potential and mechanism of SAR-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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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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