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Letter to the Editor

Chronic subclinical spondylotic myelopathy exacerbated by COVID-19: A case report

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ABSTRACT

Introduction: Besides typical respiratory symptoms, the coronavirus disease 2019, also known as COVID-19, is characterized by a wide range of neurological symptoms that result from the injury of the brain and peripheral nerves. Only a few reports have described the involvement of the spinal cord among COVID-19 patients. Furthermore, little is known about the risk of individuals with chronic degenerative conditions of the spine for acute neurological complications of COVID-19.

Case presentation: Here, we describe the case of a 73-year-old man with a subclinical cervical multifocal spondylotic myelopathy that manifested neurological symptoms of spinal cord injury only some days after getting infected with SARS-CoV-2. The patient did not show any data associated with respiratory involvement and improved clinically after decompressive spinal surgery and administration of steroids.

Conclusions: This is the first reported case of an acute exacerbation of a chronic degenerative condition of the spine caused by COVID-19.

1. Introduction

The emergence of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the causative agent of the coronavirus disease 2019 (COVID-19), is a global public health crisis. Although respiratory symptoms mainly characterize this disease [1], SARS-CoV-2 has the potential to affect the nervous system, causing a wide range of neurological manifestations [2]. The spectrum of neurological findings observed in COVID-19 patients is associated with the affection of the brain and peripheral nerves. In contrast, only a few reports have described the involvement of the spinal cord [3–5]. Also, little literature exists about the risk of patients with chronic degenerative disorders of the spine for exacerbations of their underlying conditions after SARS-CoV-2 infection.

Here, we report the case of a patient with chronic silent cervical spondylotic myelopathy who manifested neurological symptoms of spinal cord injury only after getting infected with the novel SARS-CoV-2. Our report provides evidence of the potential of COVID-19 to unmask and exacerbate chronic degenerative spinal conditions.

2. Case presentation

A 73-year-old right-handed man attended our center with a history of sub-acute quadriparesis, urinary retention, and fecal constipation. Six weeks earlier, he tested positive for SARS-CoV-2 infection by real-time polymerase chain reaction (RT-PCR) in a nasopharyngeal swab sample. He presented a fever that remitted with antipyretics as his only clinical manifestation of illness. Seven days later, he developed acute urinary retention and fecal constipation, requiring admission to another hospital, vesical catheterization, and evacuating enemas. After three days, the patient developed myoclonic movements in both lower limbs that progressed to paraparesis. He received unspecified medical management and discharged with minimal clinical improvement. During the following month, he was evaluated by a gastroenterologist and a urologist, who ruled out primary disorders of the gastrointestinal and urinary tracts. Over such a period, he developed irradiated cervical pain and weakness of the upper limbs. Also, he presented a weight loss of 6 kg due to reduced food intake as a result of fecal constipation and restrictive abdominal distention. The patient was referred to our center with the clinical suspicion of an upper spinal cord injury.

His past medical history was unremarkable other than occasional alcohol consumption. He was a half-marathon runner (last running six months before symptoms onset) otherwise healthy. On admission, the patient was alert, oriented to person, place, and time. The physical examination was relevant for painful abdominal distention, which also conditioned restrictive respiratory distress. His neurological examination showed general hyperreflexia and muscle weakness of left predominance. Also, he presented the Babinski sign in the left lower limb and hyperalgesia in both left limbs. He was unable to stand and walk unaided, and his gait was ataxic. The cerebellum was not evaluable clinically due to the presence of motor deficits, and meningeal signs were negative. The patient's clinical findings were consistent with the American Spinal Injury Association (ASIA) impairment scale Grade B [6].

Due to the recent antecedent of COVID-19, we suspected of an immune-mediated neuropathy vs. acute non-traumatic myelitis. The results of the laboratory workup showed hemoglobin of 13.3 g/dL, platelets 249,000 (10^9/L), glucose 98.6 mg/dL, and normal white blood cell counts. Also, thyroid, liver, and renal function parameters, as well as the metabolic panel, lipid panel, prothrombin time (PT), partial thromboplastin time (PPT), International Normalized Ratio (INR), fibrinogen, procalcitonin, MB fraction of creatine phosphokinase (CPK-MB), serum levels of calcium, magnesium, sodium, potassium, and chloride were normal. The cerebrospinal fluid (CSF) analysis showed normal opening pressure, glucose 92 mg/dL, proteins of 34.7 mg/dL,

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normal adenosine deaminase, and no cells, oligoclonal bands, nor bacterial growth. Assessment of other acute phase reactants and tumoral markers showed erythrocyte sedimentation rate (ESR) of 12 mm/h, C-reactive protein (CRP) of 2 mg/L, rheumatoid factor (RF) 40 IU/mL, D-dimer 2.28 mg/L, carcinoembryonic antigen (CEA) 0.69 ng/mL, carbohydrate antigen CA 19-9 159.4U/mL, and alpha-fetoprotein (AFP) 1.44 ng/mL.

The thoracoabdominal computed tomography (CT) scan was relevant for bowel distention and retained excessive stool in the small intestine and colon, but negative for pneumonia, tumor, and lymphadenopathy (Fig. 1). Magnetic resonance imaging (MRI) of the brain was normal (Fig. 2). T2-weighted MRI of the spinal cord revealed enlargement of the posterior arch of the atlas (C1), and multiple anterior and posterior compressions at segments C3-C6 of the spinal cord associated with intervertebral disc herniations and yellow ligament hypertrophy (Fig. 3a). These compressions conditioned myelopathy manifested as an intramedullary hyperintensity at segments C2-C3, which was not characteristic of acute transverse myelitis (ATM; Fig. 3b). Also, stenoses at segments L1-S1 of the spine were observed (Fig. 3c). The spinal nerve roots looked normal at all levels, including the cauda equina.

Administration of methylprednisolone 1 g IV for two days caused improvement of the motor deficits. However, bowel and bladder dysfunction remained unchanged. Due to the risk of permanent serious neurological dysfunction and respiratory failure resulting from the affection of the upper spinal cord, we decided on the surgical decompression of the cervical stenoses. We performed a resection of the posterior arch of C1 and laminectomy of C3-C6 by a posterior approach without postoperative complications. For this procedure, neurosurgeons, anesthesiologists, and nurse personnel were required to wear an N95 mask and appropriate personal protective equipment (PPE). After the surgery, we continued the steroid administration for an additional three days. The patient recovered the muscle strength of his upper and lower limbs and was able to stand with assistance seven days after surgery (ASIA Grade D). Also, he presented an improvement of his intestinal motility and was able to pass stool. A postoperative MRI of the cervical spine showed no residual compressions of the spinal cord (Fig. 3d). The decompression of the lumbar canal was postponed. He was discharged with a urinary catheter and scheduled for neurological and bladder rehabilitation. The patient provided informed consent for the publication of the case report.

3. Discussion

The COVID-19 pandemic has modified the routine workflow of several areas of medicine, including spine surgery. In many centers, urgent spinal surgery has been reserved only for conditions of high risk for severe neurological sequela, and most procedures have been conducted following strict recommendations of PPE usage [7]. Elective surgeries have been canceled or postponed to preventing possible contagions. However, due to the continuously increasing number of COVID-19 cases around the world, it is highly likely that neurosurgeons would have to provide medical care to individuals with active COVID-19 and underlying disorders of the spine. Furthermore, patients with chronic degenerative spinal diseases may be at risk of acute disease exacerbations caused by the infection with SARS-CoV-2.

Here, we described the case of an elderly patient with a chronic subclinical cervical spondylotic myelopathy that developed

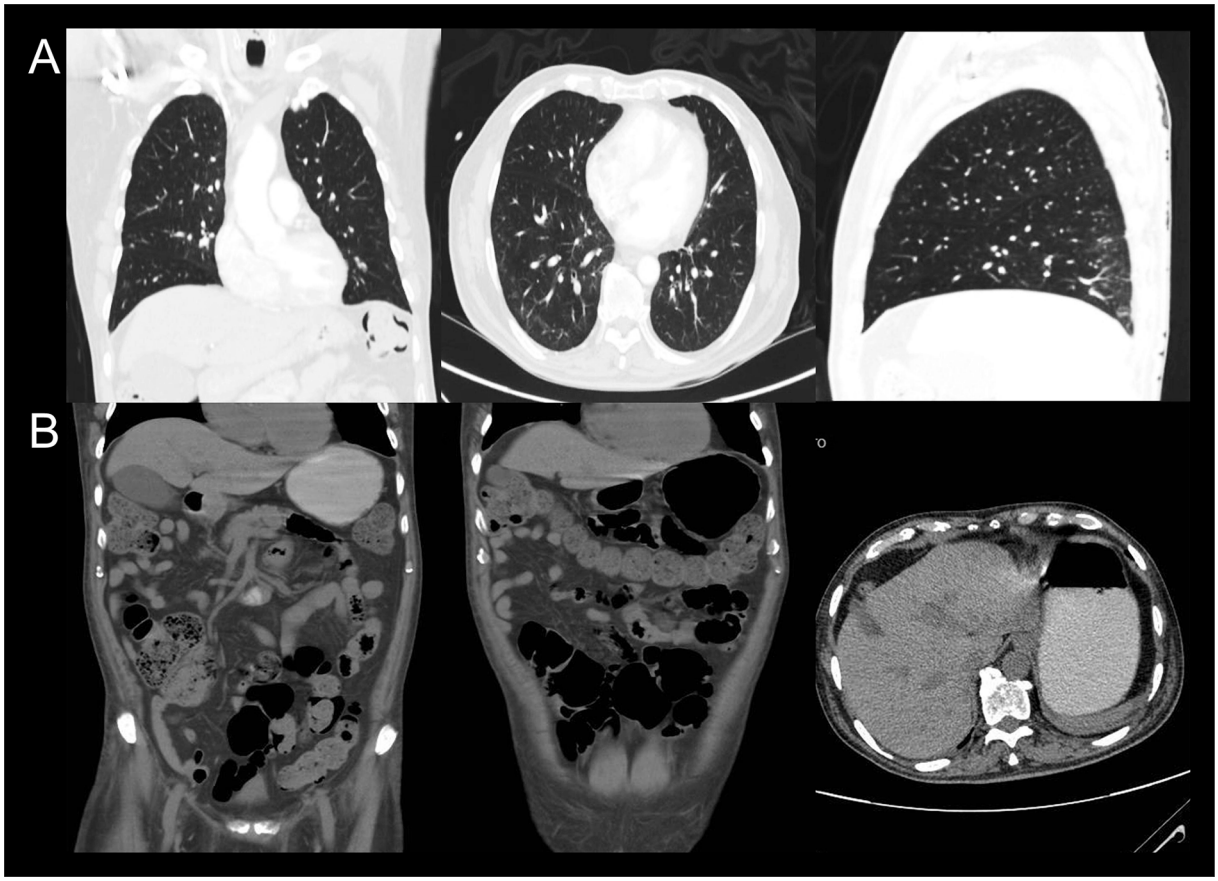

Fig. 1. Thoracoabdominal CT scan demonstrating no signs of COVID-19 pneumonia. (A) Coronal (left), transverse (middle), and sagittal (right) CT scan images of the lung that demonstrate typical characteristics of the organ. (B) Coronal (left) and transverse (right) CT scan images of the abdomen that show distention of the stomach, small intestine, colon, and even gallbladder, associated with the retention of stool due to the autonomic dysfunction of the patient.

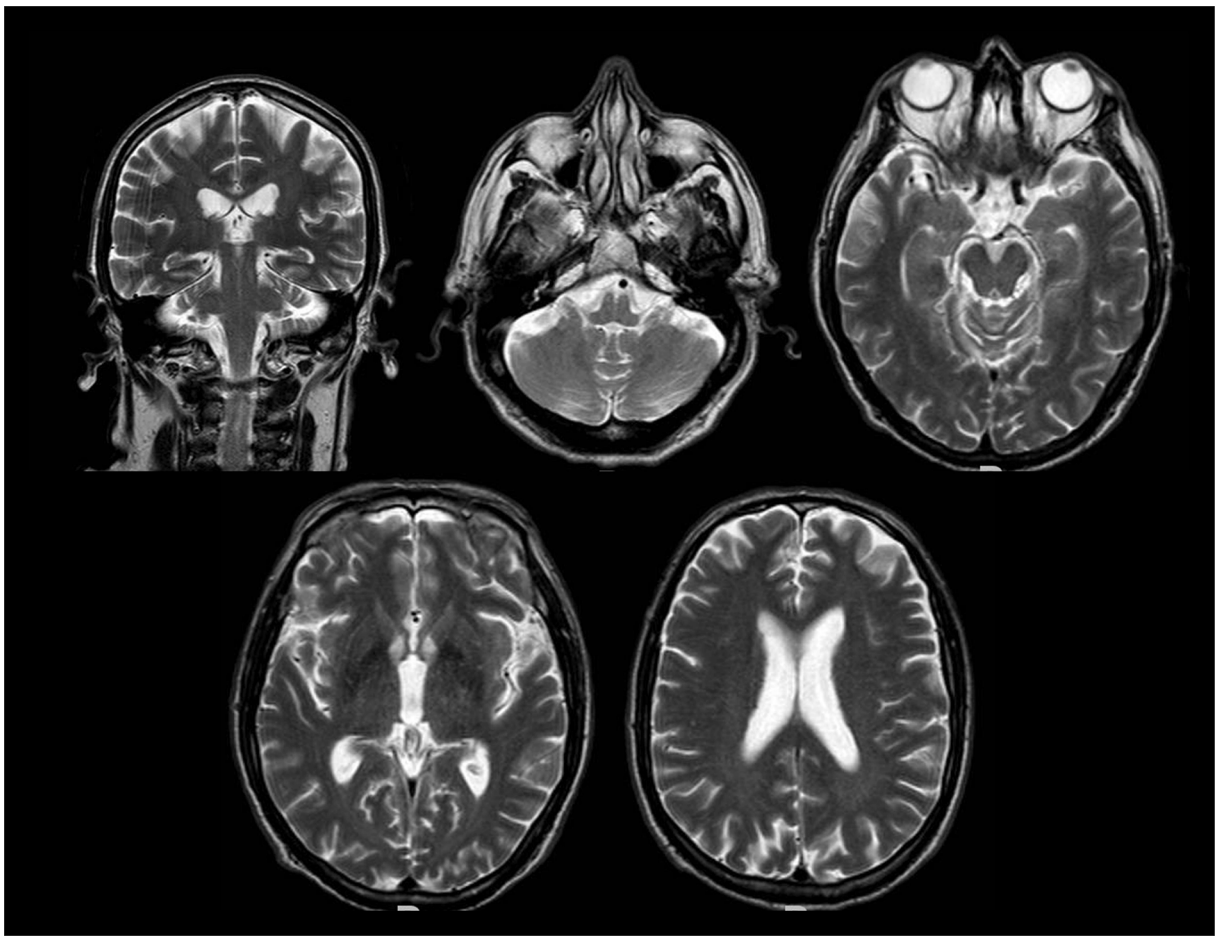

Fig. 2. Brain MRI demonstrating no focal lesions. Coronal and transverse T2-weighted images.

neurological manifestations of acute spinal cord injury some days after getting infected with the novel SARS-CoV-2. We found particularly important to communicate this case as it illustrates an acute exacerbation of a chronic subclinical degenerative process of the spine/spinal cord induced by COVID-19. In fact, our patient had stable clinical cervical myelopathy and was able to practice high-intensity sports until the SARS-CoV-2 infection precipitated the neurological decline.

This case differs from other reports of COVID-19 patients with acute spinal cord injury. Those cases have been characterized by ATM in individuals with a history of respiratory symptoms and with no demonstrated structural alterations of the spine [3–5]. Conversely, our case does not resemble a classic ATM, since the T2-weighted MRI of the spinal cord showed an intramedullary hyperintensity occupying only two cervical segments, which coincided with the sites of spondylosis, whereas ATM mostly affects the thoracic spinal cord [8]. Furthermore, we did not observe enlargement of the affected cervical segments, nor enhancement of the leptomeninges and dorsal nerve roots, which are features of parainfectious and idiopathic ATM [8]. Also, the CSF analysis of our patient was not inflammatory, although the delayed diagnostic evaluation of our patient did not allow us to analyze the CSF during the acute phase of the patient́s illness. Furthermore, our patient showed clinical improvement after treatment with steroids and decompressive surgery, even when these interventions were administered six weeks after disease onset.

Interestingly, our patient did not present respiratory symptoms. This supports a possible neurotropism for SARS-CoV-2 and suggests that direct effects driven by the virus could cause the spinal cord injury. The chronic inflammatory process induced by ischemia and mechanical compression could make the spinal cord more prone to the detrimental effects of the infection. In this regard, it has been demonstrated that the expression of the angiotensin-converting enzyme 2 (ACE2), the putative receptor for the “spike” (S) protein of SARS-CoV-2, is overregulated by inflammatory signals [9].

Finally, segments of the spinal cord under chronic compression display a range of pathological alterations, including ischemia, vascular remodeling, and endothelial dysfunction [10]. These changes contribute to a disruption of the blood-spinal cord barrier (BSCB) and increased vascular permeability, which might result in increased inflammatory infiltration to the spinal cord parenchyma. Hence, the anti-viral inflammatory response could have further exacerbated the local inflammation at sites of compressive myelopathy.

4. Conclusions

Our report provides evidence about the potential of SARS-CoV-2 to cause disease exacerbations in patients with chronic degenerative conditions of the spinal cord, such as spondylotic myelopathy.

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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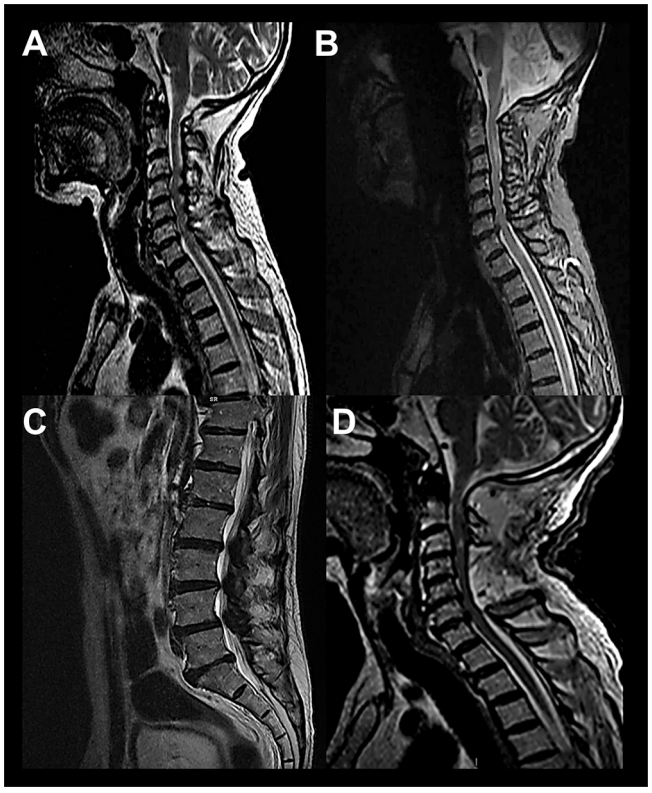

Fig. 3. MRI of the spinal cord demonstrating cervical spondylotic myelopathy and lumbar stenosis. (A) Sagittal T2-weighted contrasted image of the cervical spinal cord showing multilevel stenoses of the cervical canal and compressions to the spinal cord. Notice the enlargement of the posterior arch of the atlas. (B) Spondylotic myelopathy is revealed by a slight intramedullary hyperintensity at C2-C3 segments. (C) Sagittal T2-weighted contrasted image of the lumbar spine showing data of multilevel lumbar stenosis. (D) Postoperative sagittal T2-weighted MRI of the cervical spinal cord demonstrating decompression of the cervical canal.

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Author contribution

PG-O, JP-S, PR-M, DP-R were responsible for the patient's medical care. JC-P, FP-S, and GG-Q retrieved the clinical of the patient. JC-P drafted the manuscript. PG-O, FP-S, and GG-Q participated in the writing process of the document and revised for intellectual content. All the authors read and approved the final version of the manuscript.

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