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Case Report
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COVID-19-Associated Acute Transverse Myelitis (ATM): A Case Report

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Abstract

Coronavirus 2 is a new enveloped RNA coronavirus, first reported in Wuhan, China. The virus neurological tendencies are well established in literature and through clinical experience. Despite being a relatively rare disease, during the COVID-19 pandemic, there was a rise in the incidence of ATM worldwide. The pathophysiology behind this rise is still not well understood. Here, we present a 65 years old male with COVID-19 associated ATM. He presented with lower extremities sensorimotor weakness for 1 day. His serum microbiology was negative. During hospitalization, the patient developed urinary retention and abdominal obstruction. The patient was given 1 mg of solu-medrol IV for five days once per day, 42 mg of disflatyl twice per day, and he received IV immunoglobulins once per day for two days.

Keywords: COVID-19; Acute transverse myelitis; SARS-CoV-2

Introduction

Coronavirus Disease 2019 (COVID-19) is caused by infection with Severe Acute Respiratory Syndrome (SARS) Coronavirus 2 (SARS-CoV-2), a new type of enveloped RNA coronavirus [1]. First reported in Wuhan, China in 2019, the outbreak was soon declared as a global pandemic by the World Health Organization (WHO) in March 11th, 2020 [2]. By the end of May, 2021, there have been more than 169,118,995 laboratory confirmed cases, resulting in 3,519,175 fatalities worldwide [3]. It is well-established that SARS-CoV-2 uses a spike protein to attach to host's Angiotensin-Converting Enzyme 2 (ACE2) receptor, which is expressed on various human tissues including endothelial cells, epithelial cells and alveolar cells [4-6]. While common symptoms of the disease include Acute Respiratory Distress Syndrome (ARDS), acute cardiac injury, acute hepatic injury, acute kidney injury, and neurological manifestations such as anosmia, headaches, dizziness, hypogeusia, and loss of consciousness, have also been reported [7]. In addition, recent findings have also highlighted the association between SARS-CoV-2 and complex neurological diseases including Guillain-Barre syndrome, early-onset large-vessel strokes and acute transverse myelitis [4,7,8]. Here, we present a case of post-infectious Acute Transverse Myelitis (ATM) in a 65 years-old male patient in Saudi Arabia who presented with lower limb weakness.

Case Presentation

A 65 years old male, newly diagnosed with type 2 diabetes, presented to the ER with lower limb weakness and sensory loss for one day. The patient reported no history of trauma, shortness of neither breath, fever, neither hypogeusia nor any symptoms related to COVID-19 infection for the past three months. A nasopharyngeal swab was performed and it indicated the patient was positive for SARS-CoV-2. Upon physical examination, the patient was vitally stable, alert and oriented in person, place, and time. No abnormalities were detected on chest auscultation and abdominal palpation. Neurological exam indicated no upper extremity weakness; however, a bilateral lower extremity weakness was graded 1/5 with hyperreflexia and extensor response. Higher cortical functions, cranial nerves and cerebellar examination were all intact.

Chemistry, complete blood count, liver function test and renal function test were all normal except for a slightly increased HbA1c (6.4%). Furthermore, his serology was negative for Rheumatoid factor, Brucella Ab, Mel, and tuberculin tests Hepatitis profile, HIV and VLDR. Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) indicated no brain abnormalities. However, MRI of the cervico-thoraco-lumbar spine revealed a vertical lesion located at the distal end of the spinal cord appearing dark at T1-weighted images and bright at T2-weighted images, suggestive of a pathological process (Figure 1). No para-vertebral soft tissue abnormalities

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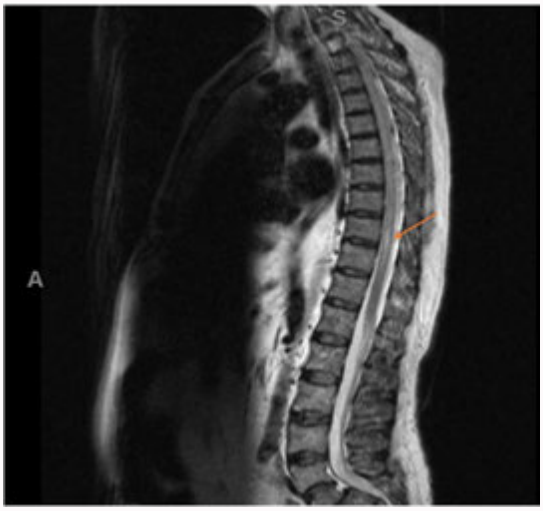

Figure 1A: There is long segment of T2 WI/STAIR hyper intense signal on the spinal cord, extending from T8-T10, with no corresponding diffusion restriction and enhancement post contrast administration.

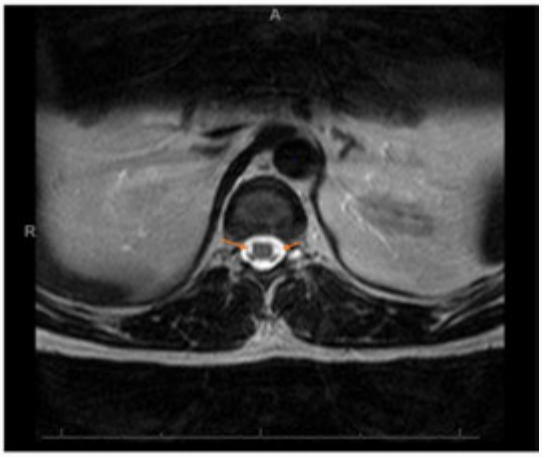

Figure 1B: The lesion shows bilateral symmetrical high signal intensity on T2 WI/STAIR images, with no diffusion restriction and enhancement post contrast administration.

were detected. Cerebrospinal Fluid (CSF) analysis revealed elevated proteins, reduced glucose, and a normal CSF culture. Lymphocytes were predominant and mildly elevated.

After three days of hospitalization, the patient developed urinary retention, abdominal distension and sever constipation. Hence, imaging for the abdomen and pelvis were ordered to rule out any cause of obstruction and the patient was catharized to relief the urinary retention. Abdomen and pelvis ultrasound showed no significant findings except a gaseous distension. CT with intravenous contrast of the abdomen and pelvis showed no masses, or significant adenopathy. The pelvis imaging came with no significant findings except for bladder catherization. Giving these findings were consistent with acute transverse myelitis, the patient was given 1 mg of solu-medrol IV for five days once per day, 50000 IU of vitamin D once per week and 600 mg of calcium thrice daily. Finally, he was given 42 mg of disflatyl twice per day. He also received IV immunoglobulins once per day for two days.

Discussion

Transverse Acute Myelitis (ATM) is rare inflammatory neurological condition characterized by inflammation of the spinal cord. Common symptoms include sensorimotor weakness as well as anatomical dysfunctions [9]. Although the incidence of ATM is estimated to be as low as 1.34 to 4.6 cases per million populations per year, a recent clinical review collected 43 COVID-19-associated ATM cases in one year [10]. Moreover, COVID-19-associated ATM was observed in 21 out of 1760 COVID-19 patients with neurologic complications as reported by a single hospital in Italy [11]. Additionally, a Wuhan retrospective observational study of 214 COVID-19 patients indicated neurological symptoms were seen in 36.4% of patients and were more prevalent in severely ill patients (45.5%) [12]. Indeed, SARS-CoV-2-associated neurological complications are well-documented in literature, with dizziness (16.8%) and headache (13.1%) being the most frequent, followed by impaired consciousness (7.5%), hypogeusia (5.6%), and hyposmia (5.1%) [12].

A recent clinical review of 43 cases of COVID-19-associated ATM, reported slightly higher male/female ratio (23 males and 20 females) with a mean age of 49 years - excluding pediatric cases (3 patients). Of all patients, 27 were quadriplegic while 15 were paraplegic. Further, MRI analysis of the spinal cord indicated a longitudinally-extensive lesion with a cervicothoracic involvement in the majority of cases, excluding children (28 out of 40). In a patient from Denmark, the spinal lesion extended from medulla oblongata to conus medullaris, making it one of the most extensive lesions reported [13].

In this article, we reported the first case of transverse acute myelitis in Saudi Arabia. The first case report in literature was reported by Kang Zhao et al. [14]. Their patient presented first with high grade fever. During the night, the fever flared up to reach 40°C coupled with weakness of the lower limbs as well as bowl and urinary incontinence. In [14] another case reported by Valiuddin et al. [8] their patient was symptomatic for COVID-19 a week before she presented with generalized weakness. Numbness developed later distally and ascended to reach the abdomen. Similarly, AlKetbi et al. [4] reported similar case presentation. The only difference between our case and the mentioned cases is that our patient was asymptomatic for COVID-19 for the last two months. Up to our knowledge, asymptomatic presentation of acute myelitis case due to COVID-19 is rare in the literature.

The neurotropic tendencies of SARS-CoV-2 are well-established [6,10]. It has been shown that the virus gains access to the CNS via various ways, such as the cribriform plate or by infecting the capillary endothelium in the cerebral circulation where the blood flow is slow. Thus, the virus is more likely to interact with ACE2 receptors [15]. The postulated mechanisms by which SARS-CoV-2 damages neuronal cells include direct invasion of the cells *via* the ACE2 receptor, retrograde axonal transport through the cribriform plate, or indirect damage to the neuronal cells by inducing a Systemic Inflammatory Response Syndrome (SIRS) [16]. Direct invasion and damage of neuronal cells is supported by the fact that SARS-CoV-2 has a higher affinity to ACE2 receptor by 10 to 20 folds when compared to its predecessor SARS-CoV-1 [15,16]. However, the majority of CNS manifestations are due to indirect damage mediated by COVID-19, coupled with a decrease in replication of the virus [16]. This hypothesis also supported by the inability to detect the virus in CSF when patients present with symptoms of ATM. SARS-CoV-2

mainly present with respiratory symptoms, but its neurological manifestations should not be ignored, for they can lead to permanent decrease in quality of life.

Conclusion

This case, in addition to many others like it, further emphasize the need to facilitate as much consideration to extra-pulmonary symptoms of COVID-19 as to the respiratory manifestations of the disease. Specifically, the neurological complications, as they might result in permanent damage to the nervous system. Meanwhile, it is likely that the incidence of ATM will continue to rise as long as this pandemic exists. Intrinsically, one must be attentive such diseases.

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