CASE STUDY

ACUTE TRANSVERSE MYELITIS AS A NEUROLOGICAL COMPLICATION OF COVID-19: A CASE REPORT

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ABSTRACT

We aim to report a COVID-19-related case of acute myelitis that has not been associated with any other viral infections. A 23-year-old student was admitted to the hospital within a month from the time of loss of smell and taste with features of acute-onset non-compressive myelitis with paresthesia on both sides from the Th9 level. Complex neurological, clinical, laboratory, and neuroimaging examination was performed within 24 hours of admission. MRI of the spine showed a segment of increased T2 signal in the center of the spinal cord at Th11-Th12. Elevated protein level and lymphocytic pleocytosis were detected in the cerebrospinal fluid. A serologic blood test for SARS-CoV-2 showed recent infection. PCR for other viral infections was negative. The patient was treated with injectable steroids and showed full recovery. Specific neurological features of acute myelitis associated with COVID-19 were reported, described, and analyzed. Patient was treated and recovered.

KEY WORDS: COVID-19, SARS-CoV-2, myelitis, complication, infectious diseases, spinal cord

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INTRODUCTION

More and more data and evidence that confirm damage to the central nervous system (CNS) in the context of acute infection and post-infectious complications of COVID-19 have been reported [1-3]. It is essential to recognize the neurological manifestations and complications of COVID-19, some of which can progress rapidly and require urgent intervention.

COVID-19 DEFINITIONS

COVID-19 is a highly contagious respiratory disease in humans, caused by a new coronavirus called SARS-CoV-2. It was first identified in Wuhan city of China, in December 2019. [4] On February 11, 2020, the World Health Organization (WHO) announced an official name for the disease caused by the novel coronavirus – COVID-19. In this abbreviation, 'CO' stands for 'corona,' 'VI' for 'virus,' and 'D' for disease. Formerly, this disease was referred to as "2019 novel coronavirus" or "2019-nCoV." [5]

COVID-19 PANDEMIC HEALTH IMPACT

On March, 11, 2020, the WHO declared the ongoing outbreak of the respiratory disease, named Coronavirus disease 2019 (COVID-19), to be a global pandemic, pointing to over 3 million cases and 207,973 deaths in 213 countries and territories. [6] The morbidity and mortality of the global community due to this disease is increasing dramatically. As of January 11, 2021, authorities in 218 countries and territories around the world have reported over 90.8 million COVID-19 cases and 1.94 million deaths since China reported its first cases to the WHO in December, 2020. [7]

COVID-19 ECONOMIC IMPACT

The COVID-19 infection has not only become a public health crisis but has also affected the global economy. The pandemic caused the largest global recession in history, with more than a third of the global population at the time being placed on lockdown. [8]

The quarantines and lockdowns that are needed to fight the COVID-19's spread are freezing the economies with unprecedented force and speed. Significant economic impact has already occurred across the globe due to reduced productivity, premature deaths, business closures, workplace absenteeism, trade disruption, decimation of the tourism industry, and reduction in productivity. It has created a negative supply shock, with manufacturing productive activity slowing down due to global supply chain disruptions and closures of factories. [9] Worldwide, 660 million workers lost their jobs, 38 million have filed for unemployment insurance during the pandemic. A record 3.28 million Americans applied for unemployment benefits, the highest number ever recorded. [10]

The COVID-19 pandemic has triggered the deepest economic recession in nearly a century, threatening health, pushing the hospital system to its capacity, disrupting economic activity, and

hurting well-being and jobs. The pandemic has created a demand shock, a supply shock, and a financial shock all at once with the global labour income losses over US$3.5 trillion. [11, 12]

COVID-19 NEUROLOGICAL MANIFESTATIONS

As a second wave of COVID-19 occurred, it has become clear that SARS-CoV-2 may affect not only the respiratory system, but multiple parts of the body, including the nervous system. Reported neurological manifestations of COVID-19 include, but are not limited to: headache, impaired consciousness, stroke, seizure, meningitis, encephalitis, necrotizing encephalopathy, Guillain-Barré syndrome, and acute demyelinating encephalomyelitis [13-16].

Neurological complications in COVID-19 infected patients have not been widely reported. Here we report the case of a patient who presented with acute-onset non-compressive myelitis and was found to be infected with COVID-19.

The purpose of this study is to report, describe, and analyze a COVID-19-related case of acute myelitis in a young adult that has not been associated with any other viral infection. Comprehensive clinical and neuroimaging analysis follows.

CASE REPORT

A 23-year-old male student was admitted to the hospital with progressive bilateral lower extremity weakness, difficulty urinating, and constipation. A month earlier, he had a loss of taste (dysgeusia) and smell (anosmia). The symptoms disappeared on their own after 14 days. The patient was not examined for SARS-CoV-2, and therefore did not receive any treatment for COVID-19. On the 20th day from the time of loss of smell and taste, he developed weakness in his legs, and began to experience difficulty urinating. Over the next 7 days, sub-febrile conditions and pain in the lumbar and thoracic spine were noted. This was accompanied by progressive weakness of the lower extremities, difficulty walking, and constipation. He turned to the Regional Clinical Center of Neurosurgery and Neurology, Uzhorod city, Ukraine.

MEDICAL HISTORY

Patient does not smoke, does not take any recreational drugs or medications. No alcohol intake. No incidents of head or spinal injury. Denies chronic diseases and any surgical interventions. There is no family history of any neurological disorder.

VITALS UPON ADMISSION

Upon admission to the department, the patient showed a normal general condition. The respiratory rate was 16 breaths per minute, oxygen saturation was 100%, and blood pressure was 120/75 mm Hg. A heart rate of 70 beats per minute, temperature 36.7 ° C.

CLINICAL EXAMINATION

Clinical neurological examination revealed decreased muscle tone, hyporeflexia, and decreased proprioception of the lower extremities. Muscle strength in the legs was: 2.5 points on the left and 3.0 points on the right. The patient also had paresthesia on both sides from the Th9 level. No pathological reflexes were found. The patient was hospitalized for further examination and treatment.

LABORATORY AND INSTRUMENTAL EXAMINATION RESULTS

Preliminary general blood tests and X-Ray of the chest did not reveal any pathological changes. MRI of the spine showed a segment of increased T2 signal in the center of the spinal cord at the Th11-Th12 level (Figure 1). MRI of the brain and orbits did not reveal any signs of inflammatory changes to suggest a relationship with the changes in MRI seen in the spine.

Cerebrospinal fluid (CSF) showed increased protein level (1.02 g / L) and lymphocytic pleocytosis (130 cells). PCR for viral infections was negative for toxoplasma, herpes simplex viruses (types 1 and 2), and cytomegalovirus. SARS-CoV-2 (PCR) cerebrospinal fluid and oligoclonal bands were negative. A serologic blood test for SARS-CoV-2 showed recent infection (presence of SARS-CoV-2 / IgG as determined by internal immunofluorescence antibody testing). Antibodies to anti-myelin-associated glycoprotein IgM and IgG to optic neuromyelitis were negative in serum.

DIAGNOSIS

Based on initial diagnostic assessment (patient history, physical exam, evaluation of the patient's chief complaint and symptoms, differential diagnosis, and diagnostic tests and treatment results) patient was diagnosed with acute transverse myelitis at the level of Th11-Th12 (KOVID-19 associated), with lower moderate paraparesis, sensitive disorders, pelvic floor dysfunction (urinary retention and constipation), and gait disorders.

DIFFERENTIAL DIAGNOSIS

The presence of transverse sensorimotor myelopathy with segmental neurological deficit necessitated a detailed neurological examination. The patient did not report significant head or spinal trauma. The progressive appearance of neurological symptoms and young age made the diagnosis of cerebrovascular accident less likely. The patient's preliminary blood count and clinical examination did not indicate a serious systemic infection. The patient's medical history does not provide further indication of any cause for the disease. Neuroimaging, including brain imaging, showed no signs of a demyelinating disease such as multiple sclerosis or Devik's optic myelitis (taking into account, among other things, the absence of antibodies to aquaporin-4). Due to the indication of recent viral infection (COVID-19) in the form of its typical manifestations, and the presence of SARS-CoV-2 IgG in the blood, we assumed the possibility of developing post-viral acute partial transverse myelitis (ARTM).

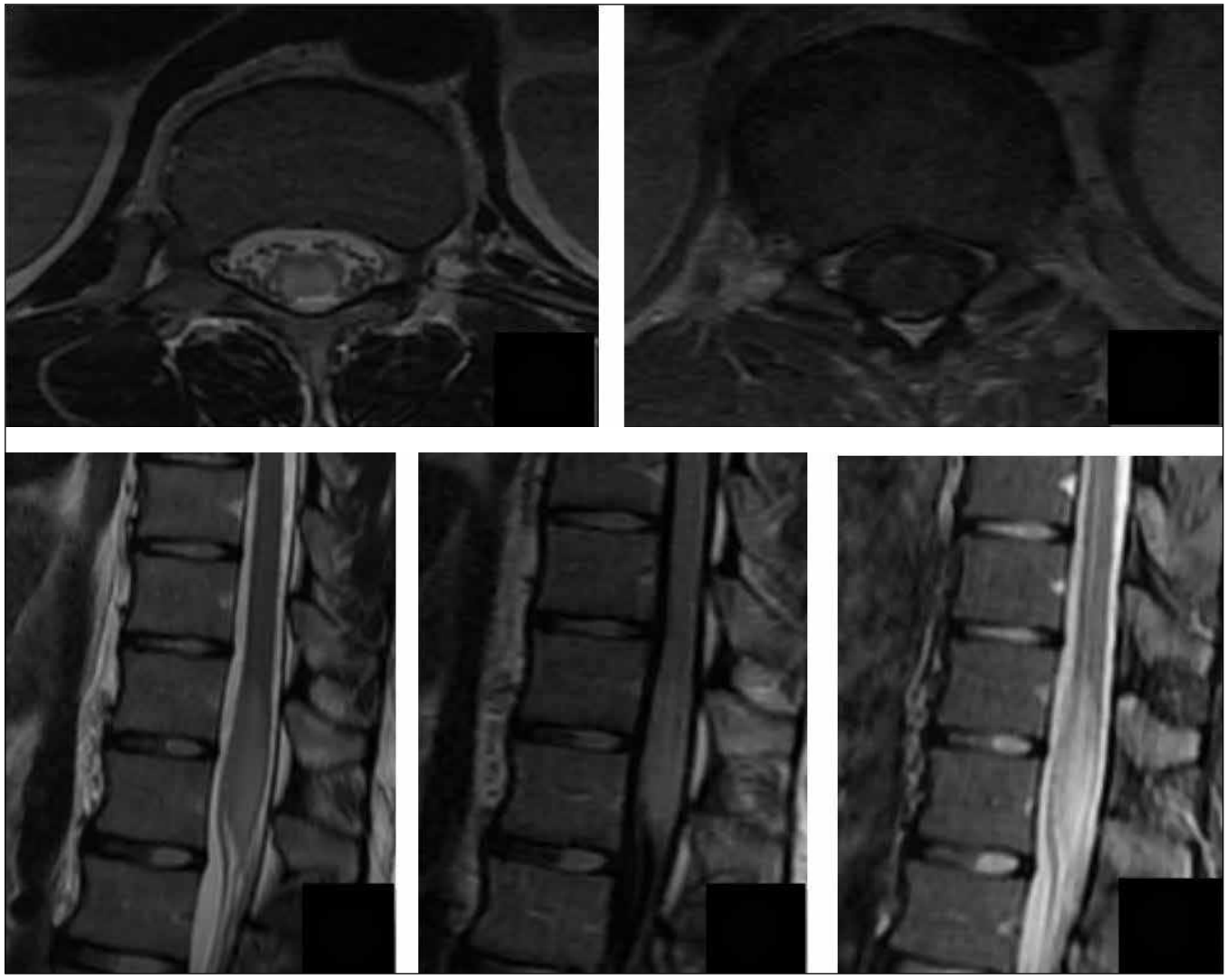

Fig 1. MRI of the patient's thoracic spine, sagittal projection (T2 FLAIR, STIR, and T2W sequences) and coronary projection (T2W).

TREATMENT

The patient was treated with methylprednisolone intravenous injections of 1 gram per day for 5 days. After that methylprednisolone was given in tablet form that was titrated, starting from 32 mg/day. No antiviral medicine, antibiotics, or immunomodulatory medicine, other than methylprednisolone, was prescribed. Neurological symptoms improved with corticosteroids treatment. The patient fully recovered motor strength in the lower extremities. Paresthesias disappeared completely and the pelvic disorders regressed. The patient was discharged home on the 8th day of hospitalization. Repeated MRI of the spinal cord was performed on day 30 and showed complete disappearance of intramedullary changes observed earlier at the Th 11-Th 12 level.

The incidence of acute myelitis associated with COVID-19 infection is not widely reported. Several reports of similar cases have been published in the literature, in which COVID-19 is associated with the onset of acute myelitis as a neurological complication. [17-21]

According to the literature, the most common symptoms associated with COVID-19 infection are fever or chills, cough, shortness of breath or difficulty breathing, fatigue, muscle or body aches, loss of taste or smell, headache, sore throat, congestion or runny nose, nausea or vomiting, and diarrhea. [2, 24]. The symptoms may appear 2-14 days after exposure to the virus. [2, 24] It is believed that anosmia and dysgeusia are due to the fact that the SARS-CoV-2 virus can directly enter the nervous system via the olfactory tract or bloodstream at a pro-inflammatory stage of the disease. [1-3]

Acute necrotizing encephalopathy has been reported following the onset of the severe acute respiratory syndrome (SARS-CoV-2) and detection of viral DNA in a nasopharyngeal swab sample. [25] In this case, the analysis of cerebrospinal fluid was not performed for technical reasons, and the diagnosis was confirmed by neuroimaging.

Viral encephalitis has been reported as another manifestation of CNS damage in the context of acute COVID-19 infection. [26] In this case, SARS-CoV-2 RNA was found

in the cerebrospinal fluid, and the brain MRI results conformed to meningitis.

Cases of Guillain-Barré syndrome associated with COVID-19 [30], as well as polyneuropathy and Miller Fisher syndrome [32] have been reported. In the indicated cases, a nasal swab for SARS-CoV-2 PCR was positive. SARS-CoV-2 was not detected in the cerebrospinal fluid.

It was suggested a hypothesis that SARS-CoV-2 can cause neural damage through hypoxic and immune-mediated pathways. [2] SARS-CoV-2 binds to ACE2 receptors, which have been described in the heart, lungs, central nervous system, and skeletal muscle. [1,2] Viral replication and increased activation of the ACE2 receptor in the CNS can induce a systemic inflammatory response leading to increased permeability of the blood-brain barrier and immune-mediated CNS inflammation. IL-6, a pro-inflammatory cytokine, is thought to mediate this response. [27-29] Increased levels of IL-6 can lead to increased production of acute-phase proteins such as CRP and fibrinogen. [27]

It has recently been highlighted that IL-6 is a potential predictor of severity, progression, and mortality from COVID-19 infection. [27-29] Elevated ferritin levels have also been associated with more severe infection and worse outcomes. Unfortunately, the determination of the level of IL-6 and ferritin in our patient was not performed.

CONCLUSIONS

Health care providers should be aware that patients with COVID-19 can present with myelitis in the acute setting and during hospitalization. We presented a case of acute partial transverse myelitis in the context of a recent COVID-19 infection. We did not find SARS-CoV-2 RNA in the cerebrospinal fluid and suggested that this case was probably associated with an immune-mediated inflammatory process, and not the direct effect of SARS-CoV-2 on the central.

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The Authors declare no conflict of interest.

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