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Case Report

Transverse Myelitis as a Rare Neurological Complication of Coronavirus Disease 2019: A Case Report and Literature Review

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Abstract

The novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is primarily a respiratory virus. However, an increasing number of neurologic complications associated with this virus have been reported, e.g., transverse myelitis (TM). We report a case of a 39-year-old man admitted to Namazi Hospital affiliated with Shiraz University of Medical Sciences, Shiraz, Iran. In December 2020, the patient was infected with Coronavirus Disease 2019 (COVID-19). During hospitalization, the patient suffered from sudden onset of paraplegia, and urinary retention, and had a T6-T7 sensory level. TM was diagnosed and an extensive workup was performed to rule out other etiologies. Eventually, para-infectious TM associated with COVID-19 was concluded. The patient received pulse methylprednisolone therapy of 1 g/day for 10 consecutive days followed by seven sessions of plasma exchange without a favorable response. The patient then underwent regular physical rehabilitation and tapering oral administration of prednisolone 1 mg/Kg. As a result, weakness in the lower extremities improved slightly after six months. Overall, we suspect a correlation between COVID-19 and TM, however, further studies are required to substantiate the association.

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Keywords ● Transverse myelitis ● COVID-19 ● SARS-CoV-2

What's Known

- A few studies have reported an association of para- and post-infectious transverse myelitis (TM) with coronavirus disease 2019 (COVID-19). However, there is no correlation between the severity of COVID-19-related symptoms and the severity of TM.
- Patients with TM should be evaluated for COVID-19 even without its associated symptoms.

What's New

- COVID-19-associated myelitis causes severe functional disability and does not respond favorably to aggressive medical treatment.
- Further studies are required to substantiate the association between TM and COVID-19, identify prognostic factors, and develop new treatment options.

Introduction

Transverse myelitis (TM) is a rare acute or subacute inflammatory disorder characterized by the dysfunction of spinal cord motor, sensory, and autonomic pathways. TM may occur due to various etiologies, including infections, autoimmune disorders, and demyelinating diseases.[1] Following the outbreak of coronavirus disease 2019 (COVID-19), caused by the novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), several studies have detailed common manifestations and outcomes associated with the disease. However, the main focus has been on respiratory complications, while increasing evidence indicates SARS-CoV-2 infection is not restricted to the respiratory system and can involve other organs. In particular, there are growing concerns about neurological complications due to COVID-19. It has been reported that neurological symptoms/signs have been present in up to one-third of the COVID-19 patients.[2, 3] A

wide range of neurological disorders associated with COVID-19 has been identified, including headache, stroke, ataxia, seizures, decreased level of consciousness, cerebral venous sinus thrombosis, and neuropathy.[2-5]

We present a complex and rare case of acute transverse myelitis (ATM) following SARS-CoV-2 infection as well as its associated demographic, clinical, radiological, and outcome variables. We also reviewed previous studies reporting a correlation between the severity of TM and COVID-19. The findings of our study may help researchers to gain a better understanding of the pathophysiology of TM in the context of COVID-19 and to develop new treatment options.

Case Presentation

In December 2020, a 39-year-old man was presented to the emergency department of Namazi Hospital affiliated with Shiraz University of Medical Sciences, Shiraz, Iran. The patient complained of dyspnea and exertional chest pain several hours before admission. He suffered from bone pain, malaise, and headache for 12 days before admission. His respiratory symptoms were mild and improved using supportive care and over-the-counter drugs. However, just hours prior to admission, he developed dyspnea and exertional chest pain, which led to his hospitalization. He was not vaccinated against COVID-19, and his family history in terms of any neurological disorders was negative. Upon arrival at the hospital, the patient was awake, oriented, and his vital signs were normal except for a respiratory rate of 20 breaths/min with 93% oxygen saturation (SpO_2) on room air. A few hours after admission, he developed numbness and paresthesia in his feet. Over the following 24 hours, these symptoms progressed to severe weakness in both lower extremities, leading to the inability to walk. The patient also developed urinary retention and bowel constipation. Neurological examination revealed paraplegia with a Medical Research Council (MRC) score of 0/5, absent lower limbs deep tendon reflexes, and mute plantar reflexes on both sides. Furthermore, sensory system examination was noticeable for a sensory level at vertebrae T6 and T7 for pinprick sensation and impaired proprioceptive sensation of the lower limbs. Otherwise, the neurologic examination was unremarkable.

Due to the presence of flu-like and respiratory symptoms, a high-resolution computed tomography (HRCT) of the lungs and oropharyngeal/nasopharyngeal (NP/OP) real-time reverse-transcriptase-polymerase chain reaction (RT-PCR) swab test (Roche, Penzberg, Germany) for SARS-CoV-2 were requested. HRCT results revealed diffuse ground-glass opacification (GGO) with interlobular septal and peribronchial thickening in both lungs, suggestive of COVID-19 (figures 1A and 1B).

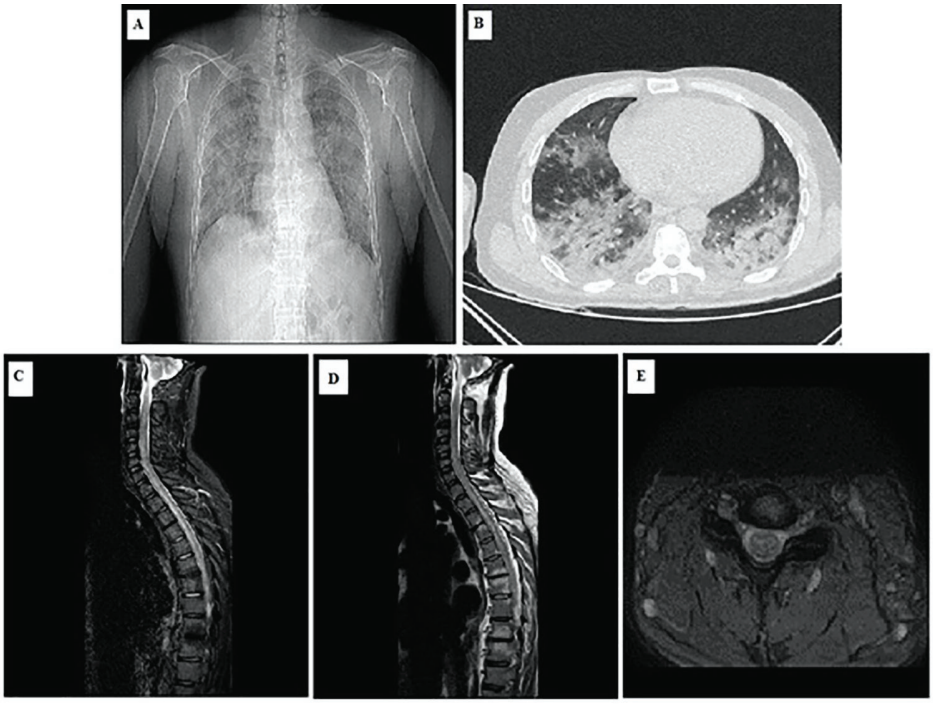

Figure 1: Chest X-ray, lung high-resolution computed tomography (HRCT), and cervicothoracic spine magnetic resonance imaging (MRI) illustrates the patient with COVID-19-associated myelitis. (A): Chest X-ray, (B): Lung HRCT scan show bilateral ground-glass opacity (GGO) and consolidation in all lung lobes consistent with COVID-19 pneumonia, (C): Sagittal short tau inversion recovery (STIR), (D): Sagittal T2-weighted, and (E): Axial T2 weighted images show longitudinally extensive high signal intensity lesion in the cervical and thoracic spinal cord consistent with transverse myelitis.

Moreover, SARS-CoV-2 viral nucleic acid was detected in the NP/OP samples. Cervicothoracic magnetic resonance imaging (MRI), in T2-weighted and short-tau inversion recovery (STIR) sequences, showed evidence of a longitudinal extensive hyperintense lesion of the spinal cord at the C2-T12 level, suggestive of TM with no gadolinium enhancement (figures 1C, 1D, and 1E). Brain MRI results were unremarkable.

The results of the initial laboratory tests are shown in table 1. Enzyme-linked immunosorbent assay was negative in serum analysis of anti-aquaporin-4 IgG antibody (AQP4-Ab), known as NMO antibody, and myelin oligodendrocyte glycoprotein (MOG). Cerebrospinal fluid (CSF) was negative for cytology, the oligoclonal band (OCB), SARS-CoV-2, varicella-zoster, herpes simplex, Epstein-Barr, influenza, tuberculosis, and brucella PCR. Besides, vasculitis profile, viral markers (hepatitis B surface antigen, hepatitis C virus antibody, human immunodeficiency virus), paraneoplastic, and autoimmune antibody panel were negative.

During hospitalization, the patient received a loading dose of remdesivir 200 mg (Darou Pakhsh, Iran) followed by intravenous (IV) administration of remdesivir 100 mg once daily for five days, as well as an IV dose of 8 mg dexamethasone (Iran Hormone, Iran) once daily. After developing TM, the latter was replaced by pulse methylprednisolone (MTP) therapy (Exir, Iran) of 1 g/day for 10 consecutive days. Moreover, due to severe myelitis attacks and non-response to treatment, the latter was discontinued and followed by seven plasmapheresis sessions. Although COVID-19 symptoms were resolved after five days, no significant improvement in the lower extremity weakness was observed. The patient was discharged with an MRC score of 1/5 in the lower limbs and a sensory level at T10. After regular physical rehabilitation and tapering oral administration of prednisolone 1 mg/Kg (Iran Hormone, Iran) over six months, lower extremity weakness improved slightly and an MRC score of 2/5 was achieved. Written informed consent was obtained from the patient to publish his anonymized information in this case report.

Discussion

A complex and rare case of TM following SARS-CoV-2 infection is reported. Given the significance of potential neurological complications of viral infections, it is essential to compile such reported cases to better understand various aspects of neurological complications due to COVID-19. Major symptoms associated with COVID-19 are pneumonia, fever, fatigue, and shortness of breath. However, SARS-CoV-2 has the potential to affect a variety of extrapulmonary organs, which may cause neurological complications.[2-5] SARS-CoV-2 virus can directly compromise the central nervous system (CNS) through infection, binding to angiotensin-converting enzyme 2 (ACE2), hypoxia, and damage to the immune system.[4]

Shiers and colleagues demonstrated the expression of ACE2 receptors in the spinal cord and reported that SARS-CoV-2 receptors are expressed in human dorsal root ganglia at the lumbar and thoracic levels.[6] Thus, COVID-19 can affect the CNS by targeting spinal cord neurons, indicating that SARS-CoV-2 is involved in ATM by binding to the cell surface of ACE2 receptors in the spinal cord neurons. This in turn leads the immune system to downregulate ACE2 expression, which could trigger inflammatory responses mediated by SARS-CoV-2 infection.[7] Another hypothesis for ATM following viral infection is that the immune system targets the infectious particles. As a result, the immune system attacks the central and peripheral nervous systems due to structural similarities between viral components and neuronal receptors.[8]

Table 1: Laboratory test results of the patient in the present case report

Laboratory test		Result	Normal range
WBC (/μL)		7.9×10^3	$4.8\text{-}10.8\times10^3$
Platelet (/μL)		313×10^3	$150\text{-}400\times10^3$
AST (u/L)		77	4-36
ALT (u/L)		77	4-36
Ferritin (ng/ml)		378	18-270
LDH (u/L)		505	200-480
ESR (mm/hr)		91	0-30
CRP (mg/L)		150	0-6
CSF analysis	White cell (/mm^3)	3 with 100% lymphocyte	Up to 5 with 100% lymphocyte
	Protein (mg/dL)	20	15-45
	Sugar (mg/dL)	57	50-80 (two-thirds of blood glucose)
	LDH (u/L)	33	<40

WBC: White blood cell; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; LDH: Lactate dehydrogenase; ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein; CSF: Cerebrospinal fluid

Table 2: An overview of clinical features and outcomes of the previous and present care reports in patients diagnosed with transverse myelitis and COVID-19

Case report	Sample size	Age (years) and sex	COVID-19 clinical symptoms*	Severity of COVID-19 infection**	Confirmatory COVID-19 results			TM clinical symptoms	TM Tempo***	Interval between COVID-19 and TM (days)	TM MRI features	Significant laboratories data	Treatment		Outcome
					NP/OP PCR	Spiral chest CT	CSF PCR						COVID-19	TM	
Rodríguez de Antonio et al.[11]	One	40/F	-	-	+	NM	NM	Numbness and hypoesthesia in lower limbs and perineum, mild urination urgency, a moderate deficit of vibratory sensitivity in the ankles and knees	NM	NM	A central 7×4 mm non-expansile T2-weighted hyperintense signal in the T5-T6 level	OCB-, NMO-, MOG-, Vasculitis-, ACE-, CSF lymphocytic pleocytosis (20 cells/μL), with normal proteins (36 mg/dL)	-	IV MTP for five days	Complete recovery of the bladder and mild recovery of sensory function
Munz et al.[12]	One	60/M	Respiratory	Moderate	+	+	-	Bladder dysfunction, progressive weakness of the lower limbs, hypesthesia below the T9 level, moderate spastic paraparesis	Acute	8	Patchy hyperintensities of the thoracic myelon at Th9-10 and Th3-5 level	OCB-, NMO-, MOG-, autoimmune panel-, CSF lymphocytic pleocytosis (16 cells/μL) with elevated protein level (79 mg/dL)	NM	IV aciclovir and ceftriaxone, IV MTP (100 mg/d) ×5 days	Significant recovery with minor disability
Durrani et al.[13]	One	24/M	Non-respiratory	Moderate	+	+	NM	Areflexia in the lower limbs with bilateral lower-extremity paraplegia and overflow urinary incontinence	NM	12	Non-enhancing T2-weighted hyperintense signal abnormality spanning from the 7th through the 12th thoracic level	NMO-, OCB-, vasculitis-, autoimmune panel-, CSF lymphocytic pleocytosis with normal protein	NM	IV MTP	Marked improve-ment
GÜLER et al.[14]	One	14/F	-	-	+	-	NA	Right-sided hemiplegia	NM	0	A contrast-enhancing lesion causing expansion at the C2-C5 level	Vasculitis-, NMO-, OCB-, and CSF revealed no cell with increased protein (262 mg/dL)	-	IVIG (400 mg/Kg/day for five days), MTP (30 mg/Kg/day for seven days)	Significant recovery
Baghba-nian et al.[15]	One	53/F	NM	NM	+	+	-	Radicular low back pain and transient urinary incontinence, asymmetrical paraparesis 3/5 and 0/5 in the right-sided and left-sided lower limbs, respectively, with sensory level at T11-T12	Subacute	14	Longitudinally extensive hyperintensity in the T8-T10 cord segments	OCB-, NMO-, MOG-, CSF lymphocytic pleocytosis (13 cells/μLl) with normal protein	NM	Plasmapheresis	Marked recovery

Fumery et al.[16]	One	38/F	Respiratory and non-respiratory	Mild	+	NM	-	Weakness of the lower limbs (MRC 4/5), hypoesthesia, and bladder dysfunction	Subacute	9	T2 extensive hyper signal involving predominantly the grey matter of the cervical and thoracic regions of the spinal cord with no gad enhancement	Vasculitis-, NMO-, MOG-, OCB-, CSF lymphocytic pleocytosis (337 cells/µL) with elevated protein (78 mg/dL)	NM	IV MTP 8 grams	Significant recovery
Chow et al.[17]	One	60/M	Respiratory and non-respiratory	Mild	+	+	NM	Urinary retention and constipation with progressive lower limbs weakness and gait impairment	Acute	16	A long segment of T2 hyperintensity in the spinal cord from T7 to T10 without contrast enhancement	NMO-, MOG-, autoimmune panel-, elevated CSF protein	NM	IV MTP 3 grams	Completely resolved
Shahali et al.[18]	One	63/M	Respiratory and non-respiratory	Moderate	+	+	NM	Sudden weakness of both lower limbs with loss of sensation below the chest in association with constipation and urinary retention	Hyper-acute	4	An extensive increased T2 signal in the central gray matter and dorsal columns from C7 to T12 with a linear enhancement in the mid- and low-thoracic cord	NMO-, MOG-, OCB-, Vasculitis-, ACE-, CSF lymphocytic pleocytosis (96 cells/µL) with increased CSF protein (128 mg/dL)	Hydroxychloro-quine Azithromycin ritonavir	IV MTP 3 grams, IVIG 25 grams/day for three days	Complete recovery
Kaur et al.[19]	One	3/F	-	-	+	-	-	Progressive flaccid quadriparesis with loss of sensation and neurogenic respiratory failure requiring intubation	Acute	21	Swelling of the cervical spinal cord with T2-hyperintense edema involving most of the transverse aspect of the spinal cord extending from the lower medulla to the mid-thoracic level with no contrast enhancement.	NMO-, MOG-, autoimmune panel-, Vasculitis-, CSF PMN pleocytosis (42 cells/µL) with elevated protein (58 mg/dL)	NM	IV MTP (30 mg/kg/d) for 5 days, IVIG 2 gram/Kg, Plasmapheresis for seven sessions, Rituximab 375 mg/m^2 for four doses	Severe disability
Present case	One	39/M	Respiratory and non-respiratory	Moderate	+	+	-	Numbness and paresthesia on feet that progressed to severe weakness in both lower extremities and loss of sensation to the level of the chest with urinary retention and constipation	Acute	12	A longitudinal extensive hyperintense lesion at the level of C2-T12 of the spinal cord with no gadolinium enhancement	NMO-, MOG-, OCB-, autoimmune panel-, vasculitis-, CSF normocellular with a normal protein level	Remdesivir Dexamethasone	IV MTP 10 grams, Plasmapheresis for seven sessions	Slight recovery with severe disability

TM: Transverse myelitis; COVID-19: Coronavirus disease 2019; F: Female; M: Male; NP/OP: Nasopharyngeal and oropharyngeal; PCR: Polymerase chain reaction; CT: Computed tomography; CSF: Cerebrospinal fluid; NM: Not mentioned; NA: Not applied; OCB: Oligoclonal band; NMO: Anti-aquaporin-4 antibody; MOG: Anti-myelin oligodendrocyte glycoprotein; IV: Intravenous; MTP: Methylprednisolone; T: Thoracic; IVIG: IV immunoglobulin; PMN: Polymorphonuclear; *Non-respiratory: fever, headache, malaise, neurological symptoms, decreased level of consciousness, gastrointestinal symptom. **Severity of COVID-19 infection: mild (no need for hospital admission), moderate (hospital admission with no need of mechanical ventilation), severe (need mechanical ventilation). ***TM Tempo: hyperacute (2-3 hours), acute (<48 hours), subacute (48 hours to 30 days).

The other proposed mechanism for ATM caused by COVID-19 is the elevation of the pro-inflammatory cytokines and interleukins (IL), such as IL-1 and IL-6, which can induce CNS immune responses and provoke inflammatory diseases of the CNS.[9, 10]

Several studies have linked SARS-CoV-2 infection to the incidence of ATM as a neurological complication. To the best of our knowledge, thus far, only nine cases of TM associated with SARS-CoV-2 have been reported.[11-19] In this review of previous studies, to exclude other differential diagnoses, we only included confirmed cases of COVID-19 (positive PCR test using NP/OP or CSF specimens) with TM and a complete workup (table 2). Interestingly, similar to our case report, the patients in previous studies were all tested negative for SARS-CoV-2-PCR-CSF, and none had severe COVID-19 symptoms. Three studies reported no respiratory or non-respiratory symptoms related to COVID-19.[11, 14, 19] Overall, there is no correlation between the severity of COVID-19 symptoms and the severity of TM. In the context of COVID-19, these findings imply that para- or post-infectious mechanisms play a much more prominent role in the pathogenesis of TM than direct invasion. Nonetheless, further studies are required to substantiate these findings.

In line with our case report, Annunziata and colleagues suggested that the combination of severe motor, sensory, and sphincteric dysfunctions at the time of ATM presentation is indicative of poor treatment outcome.[20] Given the potential poor prognostic factors, aggressive treatment is recommended with concomitant rather than individual administration of methylprednisolone IV and plasma exchange, or IV immunoglobulin (IVIg). In the case of elevated IL-6 and severe ATM-related disability, anti-IL-6 drugs may have a potential benefit if no favorable response to pulse MTP and plasma exchange is observed.[10] However, in contrast with our findings, previous studies have reported a favorable response to treatment in patients with COVID-19-associated ATM despite poor prognostic factors.[12, 13, 17, 18]

The COVID-19 pandemic is causing an increasing number of associated neurological complications, which at times result in severe functional impairment. Neurologists should therefore be familiar with such complications, underlying pathogenic mechanisms, and prognostic factors to implement prompt and appropriate treatment to reduce morbidity and mortality. Moreover, in all TM patients, COVID-19 should be included in the diagnostic workup (e.g., vasculitis, anti-NMO antibody, anti-MOG antibody, viral markers, tuberculosis, and brucellosis).

As the main limitation of the study, we did not measure the serum concentration of IL-6. In addition, some of the reviewed studies lacked information on the progression of TM, the interval between COVID-19 and TM attack, the status of SARS-CoV-2-PCR at the onset of TM, and long-term clinical follow-up.

Conclusion

SARS-CoV-2 infection should be evaluated in all TM patients regardless of a history of COVID-19-related respiratory or non-respiratory symptoms. Further observational studies with larger samples and long-term follow-up are required to substantiate the association between TM and COVID-19, identify prognostic factors, and develop new treatment options (e.g., anti-IL-6 drugs in COVID-19-associated TM).

Author's Contribution

A.A.F, M.P and F.K.A: Material preparation, data collection, and analysis, E.H, A.A.F, M.P, F.K.A, V.R.O and M.N: writing the first draft of the manuscript, E.H, V.R.O and M.N: critical revision of the manuscript. All authors contributed to the study's conception and design and commented on the last versions of the manuscript and read and approved the final manuscript and provided an agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Conflict of Interest: None declared.

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