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Acute Transverse Myelitis as a Neurologic Complication in an Asymptomatic COVID-19 patient

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Abstract
We report a case of a female presenting with sudden onset of bilateral lower extremity weakness and hyperesthesia and was managed as case of acute transverse myelitis. Diagnostics such as CSF analysis, autoimmune and infectious workups were unremarkable. However, patient tested positive for COVID-19 incidentally since she didn't present any COVID-19-related pulmonary or systemic symptoms. Thoracolumbosacral spine Magnetic resonance imaging revealed longitudinally extensive transverse myelitis. She was then administered with intravenous methylprednisolone pulse therapy and underwent extensive physical rehabilitation with marked improvement during outpatient follow-up.

Keywords: Asymptomatic Patient, Covid-19, Transverse Myelitis, Case Report

1. Introduction

Although the COVID-19 virus is notorious for causing pulmonary symptoms, various neurological manifestations have been reported to include vascular disorders, Guillain-Barre syndrome, encephalopathy, and myopathy, to name a few (Ahmed et al., 2022; Gulati et al., 2022; Gupta et al., 2020). Moreover, there are still few case reports on acute transverse myelitis following COVID-19 infection (Qazi et al., 2021). Nonetheless, there are still no available reports regarding COVID-19 confirmed patients who only presented with neurologic manifestation, specifically acute transverse myelitis. Herein we present a case of acute transverse myelitis associated with COVID-19 in an asymptomatic patient.

2. Case Presentation

A 25-year old female, left-handed, fully vaccinated but not yet boosted, without known co-morbidities came in to emergency room department for sudden onset bilateral lower extremity weakness of 1 day duration. She noted

severe lower back pain after feeling a "pop" in the back during surfing. Progressive bilateral lower extremity weakness was felt hours after and was associated with hyperesthesia and bladder distention. Due to absence of any COVID-related symptoms, no Reverse Transcriptase-Polymerase Chain Reaction (RT-PCR) nor Rapid antigen test was requested and she was subsequently admitted to a non-COVID room.

Upon assessment, patient had stable vital signs and did not present with fever, cough, dyspnea and other COVID-associated symptoms. She only had hypogastric distention due to urinary retention. Therefore, foley catheter was inserted. She had otherwise normal vital signs upon arrival at the emergency room. On neurologic examination, patient had intact mental status examination and cranial nerves. There were no visual impairments or relative afferent pupillary defect. Motor strength revealed 5/5 on upper limbs while the lower limbs had 2/5 and 4/5 for flexors and extensors, respectively. Hyperesthesia was noted starting at the L1 level bilaterally. Hyperreflexia at 4+ was noted on lower extremities due to presence of unsustained ankle clonus bilaterally. Since she was asymptomatic for COVID-19, and fully vaccinated with booster, she was then admitted to non-COVID room, amidst the hospital's loose restrictions at that time.

Initial blood workup revealed unremarkable findings, as shown in Table 1.

Table 1: Routine blood examinations

	Result	Reference
Hemoglobin	13.8	12.5-16.0 g/dL
Hematocrit	41	37-47%
Red blood cells (RBC)	4.6	4.5-5.5 $x10^{12}$/L
Mean corpuscular hemoglobin concentration (MCHC)	33	32-36 g/dl
Mean corpuscular hemoglobin (MCH)	30	27 – 31 pg
Mean corpuscular volume (MCV)	90	78 – 100 fL
Red cell distribution width (RDW)	13.2	11.5-15.0 %
White blood cell count (WBC)	8.75	5.0-10.0 $x10^{9}$/L
Neutrophils	67	37-72 %
Lymphocytes	23	20-50 %
Monocytes	9	2-9 %
Eosinophils	1	0-4 %
Basophils	0	0-1 %
Platelet	248	150-440 $x10^{9}$/L

Moreover, only modest elevations in C-Reactive Protein and D-dimer were noted (Table 2).

Table 2: Complete metabolic profile

	Result	Reference
Sodium	137	136 – 145 mmol/L
Potassium	4.1	3.5 – 5.1 mmol/L
Magnesium	0.84	0.66 – 1.07
Ionized Calcium	1.07	1.18 – 1.30
Creatinine	52.8	49 – 90 umol/L
Aspartate aminotransferase (AST)	17	5 – 34 Iu/L
Alanine transaminase (ALT)	12	5 – 55 Iu/L
Erythrocyte sedimentation rate (ESR)	6	0 – 20
C-Reactive Protein (CRP)	14	0 – 5
Procalcitonin	< 0.05	<0.5 ng/mL
Lactate Dehydrogenase (LDH)	191	125 - 220
Ferritin	135.59	4.63 – 204

D-dimer	412	0 – 400

She also underwent lumbar puncture with normal opening and closing pressures, as well as Cerebrospinal fluid (CSF) analysis within normal limits (Table 3).

Table 3: Cerebrospinal fluid (CSF) analysis

	Result	Reference
Color	Colorless	Colorless
Turbidity	Clear	Clear
Pellicle	Negative	Negative
Total protein	445 g/L	150-450 g/L
CSF Sugar	88.38 mg/dl	50-80 mg/dl
% sugar	52.6%	50-80%
White blood cell (WBC)	2 x 10^9/L	0
Red blood cell (RBC)	1 x 10^9/L	0
Total cell count	3 x 10^9/L	0

Magnetic resonance imaging of whole spine revealed non-expansile signal abnormality involving lower thoracic spinal cord extending from T8 level down to conus tip. There was no evidence of focal disc herniation, spinal canal narrowing or cord compression at any level (Figure 1).

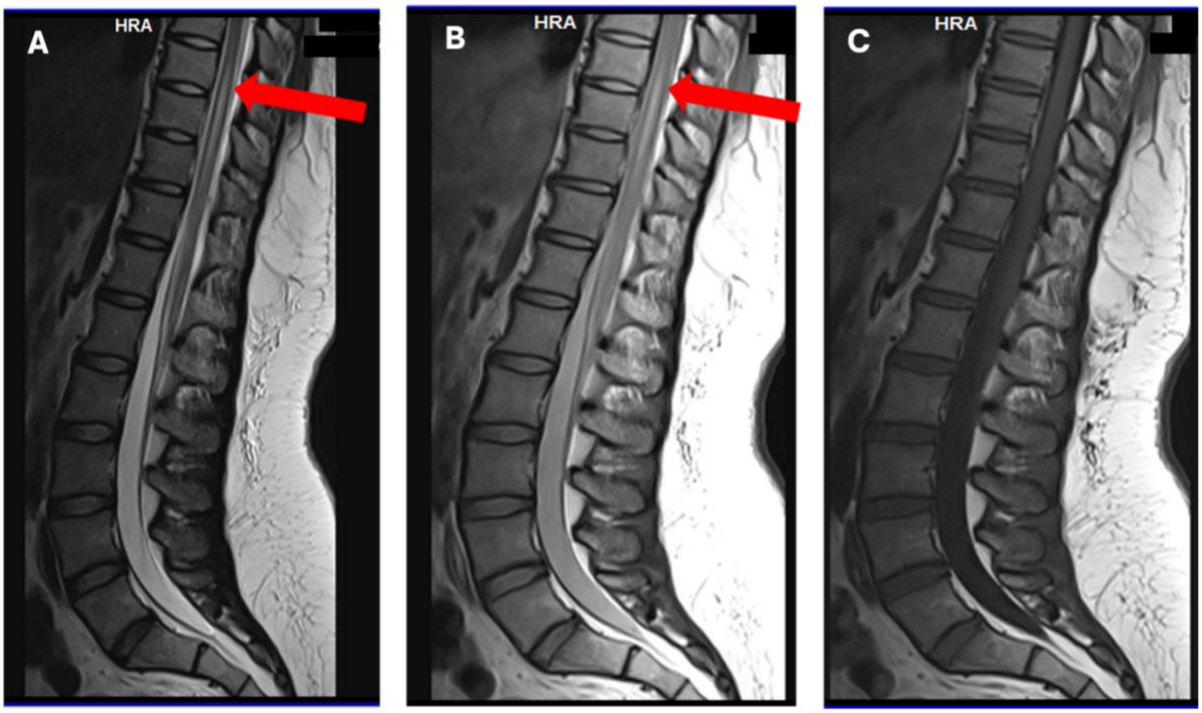

Figure 1: Thoracolumbosacral spine MRI sagittal view A) T2-weighted B) STIR/Short Tau Inversion Recovery C) T1- weighted

*Red arrows point to the longitudinally extensive hyperintense lesion starting at T8 level extending downwards

Cerebrospinal fluid analysis was also done which revealed unremarkable findings as well. CSF oligoclonal bands and IgG were negative. Moreover, both serum and CSF Anti-Aquaporin 4 antibody were negative (Table 4).

Table 4: Cerebrospinal fluid (CSF) analysis and Immunology Panel

	Result	Reference
CSF CALAS	Negative	Not detected

CSF TB Gene Xpert	Negative	Not detected
CSF Oligoclonal panel	Negative for overt CSF oligoclonal or monoclonal paraproteins	Not detected
CSF Culture/Sensitivity	No growth after 5 days of incubation	Not detected
CSF IgG	3.88 mg/dL	0.480-5.86 mg/dL
CSF Anti-Aquaporin 4 Antibody	Negative	Negative
Serum Anti-Aquaporin 4 Antibody	Negative	Negative
Cryptococcus neoformans CSF PCR	Not detected	Not detected
Varicella Zoster Virus CSF PCR	Not detected	Not detected
Human Parvovirus CSF PCR	Not detected	Not detected
Human Herpes Virus 6 CSF PCR	Not detected	Not detected
Herpes Simplex Virus 6 CSF PCR	Not detected	Not detected
Herpes Simplex Virus 1 & 2 CSF PCR	Not detected	Not detected
Enterovirus CSF PCR	Not detected	Not detected
Cytomegalovirus CSF PCR	Not detected	Not detected
Streptococcus pneumoniae CSF PCR	Not detected	Not detected
Streptococcus agalactiae CSF PCR	Not detected	Not detected
Neisseria meningitides CSF PCR	Not detected	Not detected
Listeria monocytogenes CSF PCR	Not detected	Not detected
Hemophilus influenza CSF PCR	Not detected	Not detected
Escherichia coli K1 CSF PCR	Not detected	Not detected

She was now managed as case of acute transverse myelitis, and intravenous methylprednisolone pulse therapy was immediately started, to be completed for 5 days. Since there is still no absolute explanation regarding the etiology of the disease, COVID-19 RT PCR was requested. Incidentally, it turned out positive with associated low cycle threshold or CT value. She was then transferred to COVID facility where she was diagnosed as mild case only of COVID-19. Pulse therapy was completed for 5 days wherein patient had gradual improvements of motor weakness and sensory deficits. She was advised to complete her 10-day quarantine at home. Moreover, foley catheter was removed prior to discharge as patient regained her bladder control. Rehabilitation was initiated during admission and was continued at home during her recovery for COVID-19 infection.

Patient was able to follow up two weeks after discharge and was reported to have significant improvement in motor strength of both lower limbs as she was able to ambulate independently again.

3. Discussion

Transverse myelitis is defined by an acute or subacute inflammation resulting in spinal cord dysfunction including motor weakness, sensory deficit, or autonomic impairments manifesting below the level of lesion. Moreover, different etiologies have been described such as post-infectious, immune-mediated, neoplastic, etc. (Beh et al., 2013). Given the current health landscape brought about by the COVID-19 pandemic, the virus-associated transverse myelitis has come to our attention as one of the possible neurologic complications.

There have been different pathophysiologic mechanisms proposed regarding the development of transverse myelitis in association with the COVID-19 virus. It has been studied that coronaviruses possess neurotropism, neuroinvasiveness and neurovirulence (Bauer et al., 2022). One possible mechanism is the direct invasion of the virus to spinal cord neurons. Another is the presence of angiotensin-converting enzyme 2 (ACE2), which acts as the primary receptor of the virus, on the spinal cord neuronal membranes. Furthermore, an immune-mediated injury would likely produce autoantibodies through molecular mimicry. Lastly, the virus can induce cytokine storms, which increases cytokine levels and activates complement, macrophages, T cells and endothelial cells (Beh et al., 2013; Lingas, 2022; Qazi et al., 2021; Schulte et al., 2021)

Majority of the COVID 19-associated acute transverse myelitis reported before was able to meet the criteria presented by the Transverse Myelitis Consortium Working group such as clinical evidence of motor or sensory symptoms bilaterally, or autonomic dysfunction due to a spinal cord lesion, and MRI confirmation (Román et al., 2021). Our patient presented with paraparesis, sensory level deficit, and bladder dysfunction, and with MRI-confirmed images of a longitudinally extensive lesion.

There are still no literature claiming transverse myelitis as the initial and lone presentation in an asymptomatic COVID-19 patient. There are some data on patients with transverse myelitis who have mild to moderate symptoms (Lingas, 2022; Qazi et al., 2021), or even after being clinically recovered from it (Quiles et al., 2022). Due to absence of other causation, we diagnosed this case as a post-COVID-19 acute transverse myelitis in patient without any signs and symptoms of COVID-19. Although some cases have described the appearance of transverse myelitis only after several weeks, at most up to 6 weeks post-COVID-19 infection (Ahmed et al., 2022).

High dose methylprednisolone pulse therapy was administered to our patient, and even to the reported cases as such (Qazi et al., 2021), with marked neurologic improvements afterwards. Apart from the medical therapy, our patient also underwent extensive rehabilitation at home, which also aided in her faster recovery. It has been shown that rehabilitation of post-transverse myelitis patients could help in generating the process of central plasticity and sensorimotor reprogramming (Vasconcelos et al., 2021).

4. Conclusion

Post-COVID-19 acute transverse myelitis is a rare neurologic complication that physicians especially neurologists must be wary of. Early recognition, workup, and management are needed to be able to preserve patient's functional capabilities. Furthermore, we must be vigilant that rare neurologic manifestations exist and can also present in either a COVID-19 confirmed symptomatic or asymptomatic patient.

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