Case Report

Acute transverse myelitis associated with SARS-CoV-2: A Case-Report

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1. Introduction

We describe the third reported case of transverse myelitis in a patient with the onset of coronavirus disease 2019 (Covid-19), the disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Although it is a well-known fact now that strains of SARS-CoV-2 can cause neurological manifestations such as anosmia and dysgeusia, recent literature has found complex neurological disease associations such as Guillain–Barré syndrome and early-onset large-vessel strokes (Xydakis et al., 2020; Oxley et al., 2020; Toscano et al., 2020). It has become apparent that Covid-19 causes an inflammatory cascade that results in multiple organ system being affected. In this case, inflammatory complications affecting the myelin in spinal cord occurred without the classic Covid-19 symptoms.

2. Case

On April 3, 2020, a 61-year-old female presented with a chief complaint of generalized weakness. She stated that her symptoms initially started with rhinorrhea and chills a week ago. She was suspicious that she had coronavirus because she had been exposed to several symptomatic coworkers potentially with Covid-19, so she was constantly checking her temperature; however, she was afebrile throughout. Three days afterward, she started also developing numbness and tingling in her hands and feet. Over the next 48 hours, this progressed to severe weakness in her lower extremities bilaterally. She began having difficulty with ambulation and standing, further stating that the numbness had ascended to the level of her abdomen and with associated symptoms of constipation and difficulty voiding.

Initial labs revealed a positive nasopharyngeal swab for SARS-CoV-2, serum lymphocytopenia, and mild elevation of her proinflammatory markers. On physical exam, she was noted to have bilateral upper extremity weakness graded 4/5, and bilateral lower extremity weakness graded 3/5. Notably, she also had decreased ankle reflexes, with pathological extensor plantar responses bilaterally. Magnetic Resonance Imaging (MRI) with gadolinium of the cervico-thoraco-lumbar spine revealed extensive intramedullary disease throughout the entire length of the cervical spinal cord, with an ill-defined patchy hyperintense signal on the T2-weighted images with mild enlargement of the caliber of the cord without pathological contrast enhancement. Cerebrospinal fluid (CSF) analysis revealed elevated protein and albumin with a white-cell count of one per cubic millimeter, which were mature appearing lymphocytes on cytology. The autoimmune encephalopathy panel was negative as well as a real-time polymerase-chain-reaction assay of the CSF for SARS-CoV-2. Results of studies and additional laboratory findings are shown in Table 1. Electromyography findings were consistent with a distal and motor, axonal-loss predominant, polyneuropathy impacting the lower extremities with evidence of ongoing active demyelination. There was sparing of all sensory nerves tested. No evidence of demyelination was found.

The patient received a five-day course of methylprednisolone with no improvement in her symptoms. Her weakness continued to worsen at which point she underwent five sessions of plasmapheresis, with mild improvements. Prior to the plasma exchange therapy, she had repeated nasopharyngeal testing for SARS-CoV-2 that tested negative. She also had a repeat lumbar puncture done for further CSF analysis which again showed no pleocytosis, elevated protein, and negative cultures (Table 1). She did not develop any respiratory decompensation throughout her hospitalization and subsequently was transitioned to inpatient rehabilitation.

Unfortunately, the patient is still undergoing inpatient physical rehabilitation, requiring an intensive degree of interdisciplinary therapies. She is suffering paraplegia, neurogenic bladder, and subsequent impairments in mobility and the ability to complete activities of daily living. However, she has significant improvement in her sensation and fewer muscle spasms are appreciated. She continues to receive 3 h of therapy daily, five days per week.

3. Discussion

Inflammation of the spinal cord, otherwise known as transverse myelitis, has been well documented as a result of viral infections, bacterial infections, and immune system disorders (Transverse Myelitis Fact, 2020). There is no cure for transverse myelitis. Typical treatments are
These treatments include corticosteroid and other therapies that suppress the immune system, such as plasmapheresis if there is poor response to initial treatment. Most patients partially recover within three months to two years after initial diagnosis. Some degree of disability may remain, but physical therapy has been shown to improve outcomes. Scattered reports since the 1980s have suggested certain coronavirus genotypes, which include SARS-CoV in 2003, have neurotropic properties, and sometimes neurologically devastating results such as encephalomyelitis, Guillain-Barre, seizure, and loss of neurofunctional status (Bohmwald et al., 2018). The first case of SARS-CoV-2 causing acute myelitis was published in March 2020 from a center in Wuhan, China where the

![Fig. 1. Magnetic Resonance Imaging of cervical spine. (A) Short Tau Inversion Recovery sequence sagittal plane view showing extensive ill-defined patchy hyperintense signal noted throughout the central aspect of the spinal cord. (B) T2-weighted axial cut (arrow) indicating mild enlargement of the caliber of the spinal cord and hyperintense signal without pathologic contrast enhancement.](image)

only aimed at preventing or minimizing permanent neurological deficits. These treatments include corticosteroid and other therapies that suppress the immune system, such as plasmapheresis if there is poor response to initial treatment. Most patients partially recover within three months to two years after initial diagnosis. Some degree of disability may remain, but physical therapy has been shown to improve outcomes. Scattered reports since the 1980s have suggested certain coronavirus genotypes, which include SARS-CoV in 2003, have neurotropic properties, and sometimes neurologically devastating results such as encephalomyelitis, Guillain-Barre, seizure, and loss of neurofunctional status (Bohmwald et al., 2018). The first case of SARS-CoV-2 causing acute myelitis was published in March 2020 from a center in Wuhan, China where the

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**Table 1**
Pertinent laboratory results including nasopharyngeal, serum, and cerebrospinal fluid studies over the patient’s hospital course.

| Encephalopathy/Autoimmune Evaluation Patel, CSF (4/23/20) | Spinal Fluid (4/14/20) |
|-----------------------------------------------------------|------------------------|
| AMPA-R Ab CBA, CSF Negative | Tube # 1 Spinal Fluid |
| AmphiPhysin Ab, CSF Negative titer <1:2 | Protein 87 |
| LGI-1-IgG CBA, CSF Negative | Appearance clear Protein 87 |
| AGNA-1, CSF Negative titer <1:2 | Color colorless Oligo bands negative |
| mGluR1 Ab IFA, CSF Negative | WBC 1 |
| ANNA-1, CSF Negative titer <1:2 | LD <25 RBC 0 |
| NMDD-A R Ab CBA, CSF Negative titer <1:2 | Culture no growth IgG Index 0.7 |
| Reflex Added None | Glucose 79 Protein 153 |
| ANNA-2, CSF Negative titer <1:2 | VDRL nonreactive |
| PCA-T, CSF Negative titer <1:2 | NPC-1, CSF Negative |
| ANNA-3, CSF Negative titer <1:2 | MOG Antibody Titer, CSF |
| PCA-2, CSF Negative titer <1:2 | TNP titer <1:2 |
| CASPR2-IgG CBA, CSF Negative | Spinal Fluid (4/23/20) |
| CRMP-S-IgG, CSF Negative titer <1:2 | Appearance clear Protein 87 |
| MOG Ab W/REFL TITER, CSF Negative | Color colorless |
| 0PFX Ab-IFA, CSF Negative | WBC 1 |
| MOG Ab CBA, CSF Negative | RBC 0 |
| GABA-B R Ab CBA, CSF Negative | IgG Index 0.7 |
| MOG Ab Titer, CSF Negative | Glucose 79 Protein 153 |
| GAD65 Ab Assay, CSF 0.00 nmol/L <0.02 | VDRL nonreactive |

| CBC (4/3/20) | CMP (4/3/20) |
|-------------|-------------|
| Auto WBC 11.3 | Sodium 134 |
| RBC 4.67 | Potassium 4.2 |
| Hemoglobin 14.1 | Chloride 102 |
| Hematocrit 42.1 | Calcium 9.4 |
| MCV 90 | Procalcitonin <0.05 |
| MCHC 33.5 | Total CK 205 |
| RDW 12.5 | Procalcitonin <0.05 |
| Platelets 240 | LDH 259 |
| Neutrophils Absolute 9.9 | Ferritin 109 |
| Absolute Imn Granulocytes 0.1 | D-dimer 311 |
| Lymphocytes Absolute 0.5 | Total Protein 7.2 |
| Monocytes Absolute 0.8 | Basophils Absolute 0 |
| Eosinophils Absolute 0 | Basophils Absolute Not detected |

**Viral tests (4/3/20)** | **Viral tests (4/16/20)** |
|------------------|------------------|
| SARS-COV-2 RNA | detected | SARS-COV-2 RNA | Not detected |

| Immune markers (4/3/20) | Immune markers (4/3/20) |
|------------------------|------------------------|
| C-Reactive Protein <0.5 | Total CK 205 |
| Total Bilirubin 0.5 | Procalcitonin <0.05 |
| Bilirubin, Direct 0.1 | LDH 259 |
| Troponin <0.03 | Ferritin 109 |

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outbreak first began, and the second case being reported April 2020 from Harvard (Zhao et al., 2020; Sarma and Bilello, 2020). Some ways by which our case differs is that our patient was afebrile throughout her presentation and course, never developed flaccid paralysis or respiratory distress, and symptoms progressed to urinary/fecal incontinence. This is important to highlight the atypical presentations of Covid-19. It is also worth noting that the first case and the recently reported cases of Guillain–Barré syndrome due to SARS-CoV-2, have all had negative polymerase-chain-reaction assay of the CSF for SARS-CoV-2 (Toscano et al., 2020). Although the CSF studies in this patient indicated a pathology such as atypical Guillain–Barré syndrome, the MRI studies revealed the true pathological process at hand (Fig. 1). One proposed mechanism for the neurologic disease has been the presence of ACE2 receptors that are expressed on the membrane of spinal cord neurons (Zhao et al., 2020). There is a need for more evidence to understand the pathogenesis and subsequent neurological sequelae of active or recent infections with SARS-CoV-2.

4. Conclusion

This case demonstrates the importance of keeping a broad differential at a time when the body of evidence regarding Covid-19 continues to grow. While we learn more about the pandemic caused by SARS-CoV-2, we must be vigilant of the critical neurological illnesses that could be affecting our patients.

Consent

Patient provided written consent for the case study per the institutions Research Compliance Office.

Acknowledgements

None.

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