

## Case Report

# COVID-19 associated acute transverse myelitis unresponsive to steroid therapy

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## ABSTRACT

A 63-year-old female presented to hospital with nausea, vomiting and abdominal pain. After primary evaluation, SARS-CoV-2 infection was suspected and confirmed via polymerase chain reaction (PCR) assay. She was started on broad spectrum antibiotics and remdesivir. After 12 days of hospitalization, she reported bilateral weakness and numbness of lower extremities and increased shortness of breath in the absence of fever. MRI with contrast was performed which showed intrinsic spinal cord lesion at the C7-T3 levels suggestive of transverse myelitis. She was started on IV steroids and was transferred to tertiary hospital for higher level of care. On neurological exam, there was an obvious reduction in the power of lower extremities and hyperreflexia was noted. Due to increasing weakness, MRI of cervical and thoracic spine was repeated and subsequently was started on Solu-Medrol 1-gram IV daily for 5 days. She was unresponsive to steroid therapy and refused plasmapheresis. Her course of hospitalization was complicated with acute on chronic renal failure and obstructive uropathy.

**Keywords:** COVID19, Transverse myelitis, Corticosteroids

## INTRODUCTION

SARS-CoV-2 infection has been known classically to present with fever, headache, myalgia, cough and dyspnea.<sup>1</sup> However, several complications have been reported which are believed to be caused by the host immune reactions or viral infection or by a combination.<sup>2,3</sup> Neurological complications are reported previously in the literature including headache, anosmia, dizziness, and acute encephalitis.<sup>4</sup> Among these neurological conditions, Guillain-Barré syndrome, cerebrovascular accident, and transverse myelitis (TM) are possible life-threatening complications.<sup>2,5</sup>

TM can occur sporadically as a neurological manifestation of COVID-19, which can lead to sensory,

motor, and autonomic dysfunction.<sup>6,7</sup> It is an inflammatory myelopathy which can affect people at any age with no sex predominance.<sup>8</sup> TM can present with acute weakness, sensory disturbance, urinary and intestinal dysfunction.<sup>9</sup> In this study, we report one of the rare complications of SARS-CoV-2 infection unresponsive to steroid therapy in a female with several comorbidities.

## CASE REPORT

A 63-year-old female with past medical history of diabetes mellitus, hypothyroidism, congestive heart failure, obesity hypoventilation syndrome, chronic tracheostomy, chronic renal failure, and hyperlipidemia presented to hospital with nausea, vomiting and

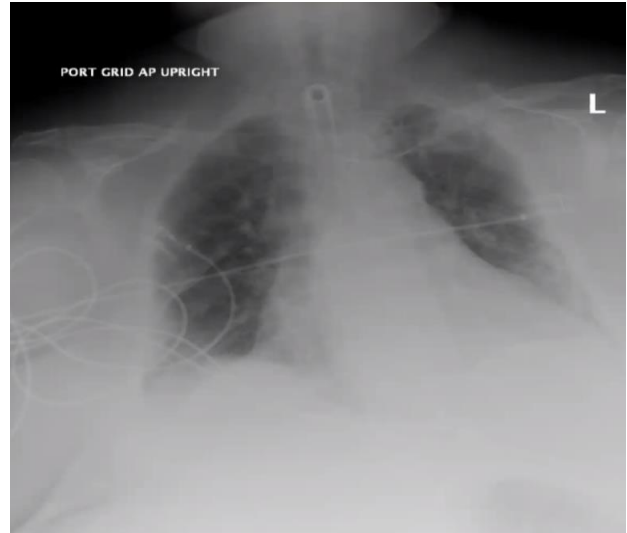
abdominal pain. After primary evaluation, SARS-CoV-2 infection was suspected and confirmed via polymerase chain reaction (PCR) assay. She was started on broad spectrum antibiotics and remdesivir. After 12 days of hospitalization, she reported bilateral weakness and numbness of lower extremities and increased shortness of breath in the absence of fever. MRI with contrast was performed which showed intrinsic spinal cord lesion at the C7- T3 levels suggestive of TM. Neuromyelitis optic IgG was negative. Under clinical impression of COVID-19 associated transverse myelitis, she was started on IV steroids. She had decreased mobility and could ambulate with a walker. She was then transferred to a tertiary hospital for higher level of care. On transfer, there was no recollection of fever, acute respiratory symptoms and headache. Her vital signs were as following: blood pressure: 146/64 mmHg, Heart rate: 53 bpm, respiratory rate: 27 per minute and oxygen saturation: 97% on trach collar. She was not in respiratory distress. On neurological exam, there was an obvious reduction in the power of lower extremities and hyperreflexia was noted. Laboratory data on admission is summarized in Table. Serological testing for hepatitis B and hepatitis C were negative.

**Table 1: Laboratory data on admission.**

Parameters	Data	Parameters	Data
<b>White blood count</b>	7.6	Sodium	131*
<b>Red blood count</b>	4.35	Potassium	4.8
<b>Hemoglobin</b>	12.3	Chloride	104
<b>Mean corpuscular volume</b>	87	Blood urea nitrogen	45*
<b>Mean corpuscular hemoglobin</b>	28.3	Creatinine	1.2*
<b>Mean corpuscular hemoglobin concentration</b>	32.7	Estimated glomerular filtration rate	45*
<b>Platelet count</b>	276	Calcium	8.5
<b>Red cell distribution width</b>	15.2	Glucose	431*
<b>Prothrombin time international ratio</b>	1.0	Ferritin	191
<b>C-reactive protein</b>	1.1*	Alkaline phosphatase	39
<b>Total protein</b>	6.6	Interleukin 6 (IL-6)	99.7*

\*Abnormal values.

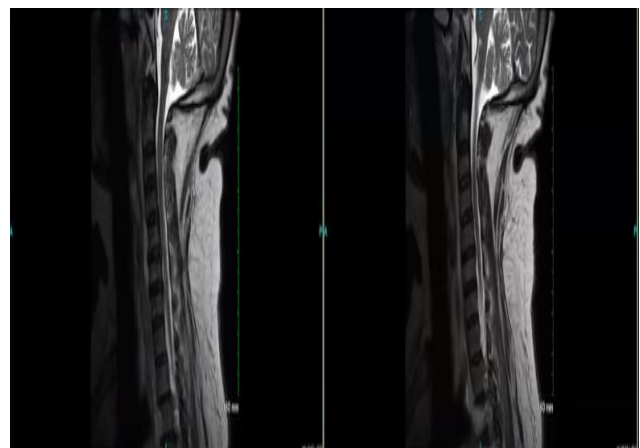
Portable upright chest film was performed which showed mild increased interstitial markings in the perihilar and basilar regions with chronicity and possibility of mild interstitial pneumonitis, mild pulmonary edema, atelectasis and scarring at the lung bases (Figure 1).



**Figure 1: Chest x-ray on admission. Portable upright chest film showed mild increased interstitial markings in the perihilar and basilar regions with chronicity and possibility of mild interstitial pneumonitis, mild pulmonary edema, atelectasis and scarring at the lung bases.**

She was transferred to ICU and a multidisciplinary team including cardiology, neurology, infectious disease, pulmonology and nephrology were involved.

Due to progressive weakness and numbness of the lower extremities, MRI of cervical and thoracic spine was repeated which showed focus of abnormal nonexpansile T2 and STIR hyperintensity in the substance of the lower cervical and upper thoracic spinal cord at the C7 and T1 levels (Figure 2). Findings reflected an area of demyelination or possibly acute transverse myelitis.



**Figure 2: Focus of abnormal non-expansile T2 in the substance of the lower cervical and upper thoracic spinal cord at the C7 and T1 levels.**

She was subsequently started on solu-medrol 1-gram IV daily for 5 days and due to uncontrolled hyperglycemia after IV steroids, high scale of insulin was adjusted.

During her course of hospitalization, renal function was continued to worsen, with the suspicion of obstructive uropathy. Her creatinine and potassium were 2.9 mg/dL and 5.9 mmol/L respectively. Renal ultrasound was performed which showed mild bilateral hydronephrosis. She received kayexalate along with insulin dextrose to medically treat hyperkalemia. Her renal function was improved by urinary catheter insertion and dialysis.

After completion of steroid therapy, no improvement was observed in the lower extremity strength. Plasmapheresis was proposed but was refused by the patient. Since more than 3 weeks had passed from initial hospitalization, the chances of any meaningful recovery from her myelitis were very poor. The patient signed leave against medical advice form and was discharged with a foley catheter.

## DISCUSSION

Transverse myelitis is acute or subacute dysfunctions of spinal cord because of inflammation.<sup>4</sup> This non-compressive heterogeneous myelopathy may result from acquired demyelinating disease or is most commonly seen after infection or vaccination due to autoimmune dysfunction.<sup>10</sup>

Our patient presented with a potential neurological manifestation of SARS-CoV-2 infection, as transverse myelitis with unresponsiveness to conventional steroid therapy. Based on MRI findings, intrinsic spinal cord lesion at the C7- T3 levels was discovered in favour of TM. The interval between the onset of COVID-19 symptoms and neurological manifestations was 12 days which is in line with previously published cases and is consistent with a post-infectious neurological complication hypothesis.<sup>11-15</sup>

CNS involvement in SARS-CoV-2 infection happens via hematogenous route or neuronal antegrade or retrograde polarization via nucleus solitaries, peripheral nerves, spinal cord, olfactory nerve and trigeminal nerve.<sup>16</sup>

Exact mechanism for COVID-19 associated neuropathogenesis is not defined, however, two fundamental mechanisms were proposed by previous studies, either independently or together.<sup>17</sup> The first hypothesized pathway is according to neurotropic and neuroinvasive characteristics of this virus, which may lead to viral invasion of the central nervous system through blood circulation, vagus nerve or nasal mucus directly via the angiotensin converting enzyme 2 (ACE2), and lead to demyelination.<sup>18,19</sup>

The second possible mechanism is post-infectious immunological dysfunction due to SARS-CoV-2.<sup>17</sup> This increased inflammatory state is exemplified by production of cytokines and chemokines leading to myelin damage.<sup>20,21</sup> Elevated IL-6 level is believed to be a possible predictor for assessment of COVID-19 severity

level, progression of disease and mortality.<sup>22</sup> IL-6 increases acute phase protein reactants such as CRP.<sup>23</sup> Our patient had increased level of IL-6 and CRP consistent with this mechanism.

Up to date, just few cases of spinal cord pathologies associated with COVID-19 have been described.<sup>24</sup> Quadriplegia, paraplegia, loss of deep reflexes, sensory disorders and urinary incontinence are the most prevalent clinical symptoms of TM which result from sensory, motor and autonomic spinal cord disorders.<sup>25</sup> Our patient presented with weakness of bilateral lower extremities and obstructive uropathy after fever was resolved. This is in contradiction with the majority of previous reported cases indicative of development of fever during COVID-19 associated TM.<sup>25,26</sup> However, similar to our findings, Ahmad et al highlighted that their case did not developed fever.<sup>7</sup> Also, our results correlate fairly well with previous studies which reported bladder dysfunction among some cases of TM.<sup>4,24,25,27</sup>

Primary evaluations of TM should concentrate on sensory, motor, and autonomic dysfunction related to spinal cord, ruling out compressive cord lesions and T2 hyperintense signal change on spinal magnetic resonance imaging.<sup>28</sup> Corticosteroids are known as the first-line choice.<sup>7</sup> There has been no evidence about efficacy of steroid therapy in COVID-19 related TM. In cases where corticosteroids are not effective, plasma exchange is suggested.<sup>7</sup> In our case, Solu-Medrol 1-gram IV was used daily for 5 days. Although, no clinical improvement was observed, the patient refused plasmapheresis.

## CONCLUSION

This study reported one of rare complications of SARS-CoV-2 infection unresponsive to steroid therapy in a female with comorbidities. Future studies investigating the prognosis of COVID-19 associated TM and efficacy of proposed treatment are warranted.

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