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COVID-19 Vasculitis: A Case Report with Complicated Course

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Abstract Coronavirus disease 2019 (COVID-19) is a viral infection caused by the Severe Acute Respiratory Syndrome coronavirus 2 (SARS-CoV-2), it spreads from person to person rapidly and presents with multi systemic manifestations namely respiratory symptoms, similar to prior SARS viruses. Neurologic manifestations of COVID-19 are uncommon; those include encephalopathy, strokes, and polyneuropathy. Here we report a unique neurologic complication of COVID-19 in a patient who had extensive cerebral small-vessel ischemic lesions resembling those of cerebral vasculitis. This case report supports the fact that neurologic complications from CVOID 19 could be more debilitating than the respiratory illness itself, especially for those patients who were eventually able to survive the complicated course of being on mechanical ventilation but unfortunately end up with this extensive CNS insult.

Keywords: *COVID-19, CNS, Vasculitis*

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1. Introduction

Coronavirus disease 2019 (COVID-19) has now became a world pandemic, initially reported in Wuhan, China by the end of 2019 The World Health Organization declared COVID-19 a pandemic on March 11, 2020 [1]. The virus is highly contagious. Similar to other coronaviruses, the Severe Acute Respiratory Syndrome coronavirus 2 (SARS-CoV-2) virus mainly affects the respiratory system. In symptomatic patients, the most common symptoms are fever, fatigue, cough, headache, and shortness of breath. Severely symptomatic patients may present with pneumonia, acute respiratory distress syndrome, acute myocardial infarctions, and multi organ failure. Several neurologic manifestations of COVID-19 have now been reported, which include strokes and encephalopathies. Here we report a unique neurologic complication of COVID-19 with extensive ischemic lesions resembling CNS vasculitis.

2. Case Description

We report a 66-year-old man who presented to the emergency department with a 7-day history of fatigue, fever, and dry cough. Initial blood work showed raised inflammatory markers with an elevated Creactive protein (120 mg/L), and lymphopenia. Given the wide spread of COVID 19, the patient was admitted to the isolation ward as a suspected case of COVID-19. Noncontrast chest CT showed peripheral ground-glass opacities which is characteristic of COVID-19 pneumonia. The diagnosis was confirmed by reverse transcription polymerase chain reaction detection of SARS-CoV-2 from a nasopharyngeal swab. Overnight, the patient had a high grade fever and chills. He was tachypnic with a respiratory rate of 40, oxygen saturation on room air 85 %, an arterial blood gas showed severe hypoxemia, 50 mmhg, intense oxygen therapy was initiated with a high-concentration mask (15L/min) and antibiotic therapy (ceftriaxone and doxycycline). The patient was showing signs of fatigue and accessary respiratory muscle use along with nonresponsive hypoxemia, consequently he was electively intubated and placed on mechanical ventilation to avoid sudden arrest, he was shifted to the intensive care unit for close monitoring. A central line and arterial lines were placed.

As per the COVID 19 protocol, the patient received a full course of favipiravir, intravenous steroids along with Interleukin-6 inhibitors to help get the cytokine storm under control. His oxygen requirements on the mechanical ventilation gradually reduced but multiple attempts to wean him off the ventilator had failed due to poor cognitive status. The patient was not responding to any kind of stimuli although being off sedation. Neurology team were consulted for further input from their side, and

given the chances of having a CNS insult as a result of COVID 19, MRI brain was done. It showed diffuse bilateral multiple white matter cortical and subcortical lesions that demonstrate T2/FLAIR hyperintense signal and diffusion restriction. No mass effect was noted. (Figure 1).

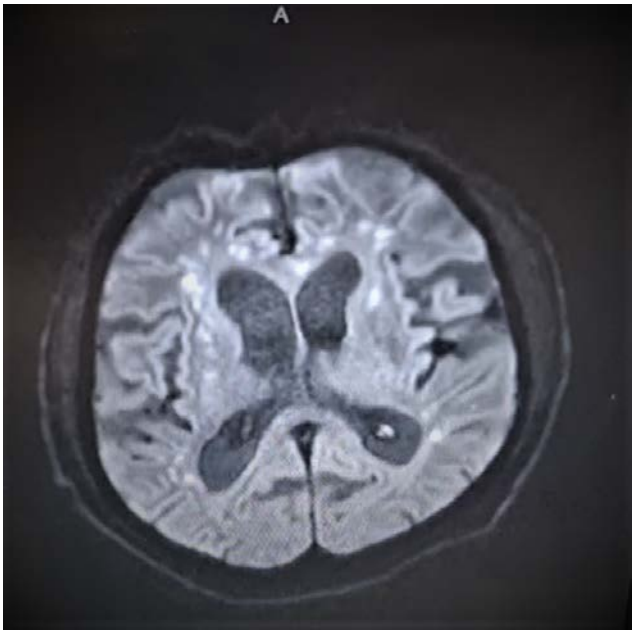

Figure 1. Diffuse bilateral multiple white matter cortical and subcortical lesions that demonstrate hyperintense signal and diffusion restriction. No mass effect was noted.

Given the overall clinical picture and suspected diagnosis of CNS vasculitis, trials with Intravenous immunoglobins and pulse steroid therapy were attempted but unfortunately with no beneficial outcome. More over due to the prolonged period of mechanical ventilation and the patient being bed bound in the intensive care unit, he sustained critical care neuropathy and myopathy. He underwent tracheostomy placement eventually as well.

Unfortunately the patient had a poor outcome, he was shifted to a long term rehab facility.

3. Discussion

Over the past period it has become more evident that COVID-19 can have CNS manifestations, yet the exact mechanism remains unclear. It is uncertain if SARS-CoV-2 directly infects the cerebral vessels, as evidence is limited and inconsistent. [2].

SARS-CoV-2 infects the host through its CoV spike glycoprotein, which binds to the angiotensin converting enzyme 2 (ACE2) receptor, which is expressed mainly in the lungs, heart, and kidney (the most frequenty targeted organs in COVID-19), but also endothelial cells. The expression of the ACE2 receptor in neurons and cerebral endothelial cells indicates a high level of invasiveness for the SARSCoV-2 in comparison with other coronaviruses (SARS and Middle East Respiratory Syndrome). [3].

In our patient unfortunately he was not responding despite multiple attempts of weaning off mechanical ventilation and sedation, although he had an element of critical care neuropathy and myopathy, but the lack of eye opening and higher brain function raised the possibility of a CNS insult in the brain. As described earlier MRI imaging of the brain showed extensive cortical and subcortical white matter lesions which raise the possibility of microembolisation but given the context that he had a normal echocardiography and no evidence of vegetations on transesophegeal echo, thus the presumed diagnosis was CNS vasculitis due to COVID 19 virus. A pro-thrombotic state was not considered a likely causative factor because the coagulation panel findings (including the antiphospholipid antigen) were normal.

Until the current date, there is no clear guidelines on the management of CNS vasculitis due to COVID-19, we tried multiple conventional therapies such as intravenous immunoglobins along with steroids but unfortunately the patients clinical condition didn't improve. We hope in the near future more robust treatment regimens would be implemented and there would be greater sum of research in that field.

4. Conclusions

This case report supports the fact that neurologic complications from CVOID-19 could be more debilitating than the respiratory illness itself, especially for those patients who were eventually able to survive the complicated course of being on mechanical ventilation but unfortunately end up with this extensive CNS insult.

List of Abbreviations

CNS : Central Nervous System
COVID-19 : Coronavirus disease 2019

Declarations

Availability of data and materials

All data are within the article.

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Authors Contribution

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Ethics Approval and Consent to Participate

Not applicable.

Competing Interests

The authors declare that they have no competing interests.

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