COVID-19 Causing Brain and Cervical Spine Demyelination

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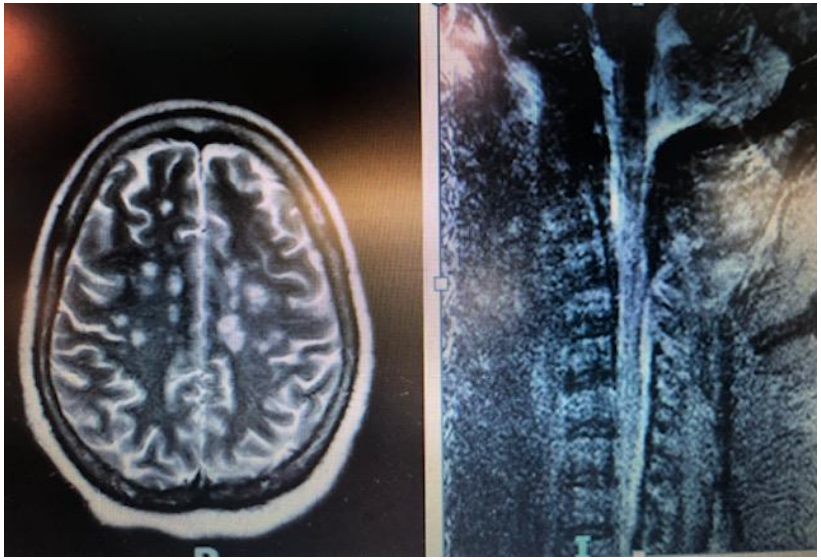

- **Abstract**: Much of the focus regarding the global pandemic of the novel coronavirus disease (COVID-19) has been on the cardiovascular, pulmonary, and hematologic complications. However, neurologic complications have arisen as an increasingly recognized area of morbidity and mortality. With increasing knowledge of COVID-19, we now understand that COVID-19 presents with various extrapulmonary manifestations with multi-organ involvement. Involvement of the central nervous system (CNS) occurs likely via transsynaptic spread or transfer across the blood-brain barrier. Hypoxia, immune-mediated injury, and vascular damage are the potential mechanisms for the CNS manifestations.
- **Case**: The patient is a 56 year old Hispanic female with a history of hypertension that presentedwith aseveral day history of cough and fever. She also complained of mild myalgias, but denied any chest pain, shortness of breath, or abdominal complaints. The patient denied any recent travel or sick contacts. She appeared well and physical examination was unremarkable except for scattered wheezes on pulmonary auscultation. Complete blood count and comprehensive metabolic panels were ordered and both were unremarkable. Chest x-ray and rapid viral panel were done, both of which were negative for acute disease. The patient was then discharged home with azithromycin and prednisone for 5 days with a diagnosis of acute bronchitis.
- The patient returned back to Emergency Department 4 days later with worsening cough and fever, and this time with severe shortness of breath. The patient endorsed that her sister, whom she has close contact with, recently tested positive for COVID-19 2 days prior. She was hypoxic to room air and required supplemental oxygen via nasal cannula while in the Emergency Department. Repeat chest x-ray revealed diffuse patchy infiltrates, markedly changed from imaging 4 days prior, and the patient was admitted to a telemetry unit with confirmed COVID-19 on PCR test several hours later. Throughout the next several days, she received hydroxycholoroquine, azithromycin, and albuterol MDI inhalers without any relief, and despite supportive management, her hypoxia continued to worsen.The patient was ultimately intubated and intermittently proned throughout a prolonged ICU stay. Upon extubation, it was noted that the pt had a profound cognitive decline and incomplete quadriplegia. MRI showed demyelination of both brain and cervical spine. While the patient did recover some motor function through rehabilitation, the demyelination persisted and the patient did not return to her baseline neurologic capacity.
- **Discussion:**The mechanism of encephalopathy in COVID-19 remains to be determined. From available studies, COVID-19 encephalopathy appears to be more common in patients with more severe disease, associated comorbidities, and evidence of multi-organ system dysfunction. This includes hypoxemia,renal and hepatic impairment, and elevated markers of systemic inflammation.
- While the exact route used by the SARS-CoV-2 (COVID-19) virus to invade the CNS has not yet been fully explored, its structure has been discovered to be taxonomically very similar to the SARS-CoV virus responsible for the 2003 outbreak. Both viruses have been found to share similar surface spike proteins that dictate tissue tropism and affinity to the ACE-2 receptor.
- Extensive research was undertaken to explore the pathogenesis and mechanism of invasion of the SARS-CoV virus into the human body following its outbreak. It was discovered that the angiotensin-converting enzyme type 2 (ACE-2) protein served as a functional receptor for the virus. This protein was subsequently discovered to be found in almost all tissue types within the human body, including the central nervous system (CNS). ACE-2 protein receptors have been found to be particularly present in the brainstem and medulla as a part of the reticular activating system involved in the regulation of the cardiovascular system. In addition, further research has established the presence of the ACE-2 protein receptor in the vascular endothelium of the CNS and glial cells. Therefore, we hypothesize that the mechanism of invasion of the SARS-CoV-2 virus into the CNS may likely be related to its interaction with the ACE-2 receptors within the CNS.