

## Acute Cerebellar Strokes with Anoxic Brain Injury After a Cardiopulmonary Arrest in SARS-CoV-2 Patient

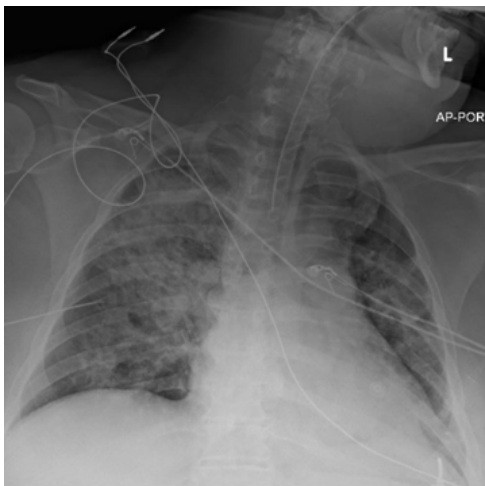
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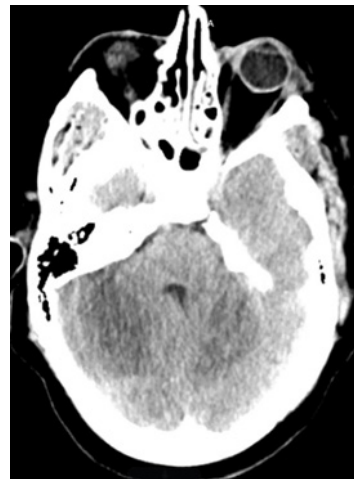
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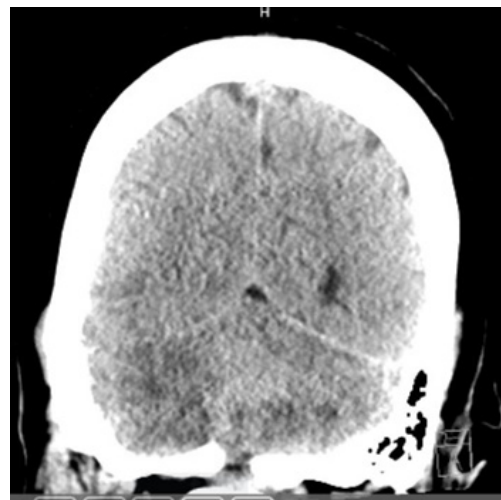
**Figure 1.** PA chest radiograph showing bilateral pulmonary infiltrates.



**Figure 2.** CT scan head showed diffuse low attenuation involving the bilateral basal ganglia, occipital lobes and cerebellar hemispheres.



**Figure 3.** CT scan head showed diffuse low attenuation involving the bilateral basal ganglia, occipital lobes and cerebellar hemispheres.



**Figure 4.** CT scan head showed diffuse low attenuation involving the bilateral basal ganglia, occipital lobes and cerebellar hemispheres.

Neurological complications from novel coronavirus is becoming more common.<sup>1,2</sup> These patients usually have primary pulmonary problem of acute lung injury. Presentation in the form of encephalitis, meningitis, Guillain-Barre syndrome and seizures are noted. It is also noted that SARS-CoV-2 has predilection for brain stem leading to patient not feeling extensive pulmonary injury. Here we share another neurological presentation.

A 65-year-old women of a history diabetes, active SARS-CoV-2 infection and mild dementia. She was at her home in quarantined state. Emergency services were called for unresponsive state. On arrival patient was apneic and in cardiac arrest. It took 10 minutes for ROSC after CPR and multiple doses of epinephrine. She was intubated in the emergency department. Her GCS was 3T at this that stage. Investigations showed lactic acidosis, profound hypoxemia and bilateral pulmonary infiltrates. (**Figure 1**) CT scan head showed diffuse low attenuation involving the bilateral basal ganglia, occipital lobes and cerebellar hemispheres concerning for diffuse anoxic injury probably related to posterior circulation limitation. There was no early cerebral edema. (**Figure 2-4**) She had no cough, gag and conjunctival reflexes. The pupillary light reflex was absent with fixed 2 mm pupils bilateral. With pupillometry there was minimal response with intact Neurological Pupil index™ (Neuroptics NPI -200 Pupillometer, Laguna Hills, CA). There was no breath triggering in spontaneous mode trial even with serum lactate of 11.7 mmol/l (0.5-2.0 mmol/l). MRI was not possible due to hemodynamic instability. Based upon presentation, severe brain injury and severe SARS-CoV-2 pulmonary involvement, patient was made comfort care.

This patient was thought to have secondary cardiac arrest from severe pulmonary disease. It is known that patient donot sense the impending worsening with SARS-CoV-2 progressive disease. Since the onset of this pandemic, multiple reports have demonstrated neuroinvasion by human respiratory coronaviruses. SARS-CoV-2 facilitates the formation of venous and arterial thromboembolic events through multiple associated pathophysiological mechanisms. The presence of inflammatory hyper-response, hypoxia, and disseminated intravascular coagulation create the necessary conditions for the development of stroke. Subclinical hypoxia has been reported as an event that is part of the initial phases, including asymptomatic.<sup>3</sup> Hypercoagulability could be the substrate for ischemic cerebrovascular events, and very high numbers of ischemic phenomena have been reported in critically ill SARS-CoV-2 patients of up to 31%.<sup>4</sup> The evolution of rapid cerebellar infarction with sudden death forcing to investigate the characteristics of the invasion of the coronavirus in the brain stem, especially in the centers that control breathing. This opens a new alert scenario for patients diagnosed with coronavirus infection.

## REFERENCES

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