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Unusual Post COVID-19 Presentation With Tetra Ventricular Hydrocephalus: A Case Report

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Abstract:

Objective: COVID 19 infection is suggested as one of the causes for hydrocephalus (HCP) of unknown etiology. COVID-19 infection may present with a range of neurologic symptoms given viral neurotropic and neuroinvasive properties. Post-infectious hydrocephalus is a severe complication as a potential sequela of COVID-19 infection.

Methods: We identified a patient with a history of recent COVID-19 infection who presented with chronic progressive headaches with nausea, vomiting, and blurry vision over two weeks.

Results: Neurological examination showed bilateral papilledema. Head CT scan showed tetraventricular enlargement and marked fourth ventricular dilation. Cine MRI showed fourth ventricular turbulent CSF flow. The patient underwent external ventricular drain (EVD) placement and exploratory suboccipital craniotomy, which revealed a sub-arachnoid web that was microsurgically resected. Reconstituted CSF flow resolved the patient's symptoms and prevented complications.

Discussion: Fourth ventricular outlet obstruction is a rare cause of tetraventricular hydrocephalus. In most cases, it is associated with a history of inflammatory conditions or hemorrhage. In our case, history of recent COVID-19 infection and normal imaging prior to COVID-19 make COVID-19 the most probable explanation for HCP. We suggest considering COVID-19 infection in the differential diagnosis of HCP of unclear etiology

Practical implication: A pearl of wisdom for the practicing clinician: “Consider COVID-19 as the cause of Hydrocephalus of unclear etiology”.

Introduction:

Although the respiratory system is the primary target of the Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2), it affects some patients' central nervous system (CNS). Various proposed mechanisms support the neurotropic and neuro-invasive tendency of SARS-CoV-2, resulting in numerous neurologic manifestations, either from direct invasion of the CNS or critical illness and systemic infection¹. HCP is one of the rare but well-documented post-infectious complications. Herein, we present a case of HCP as a potential complication of COVID 19 infection. We suggest that post-COVID 19 complications can be considered in the differential diagnosis of HCP with unclear etiology.

Case description:

A 36-year-old African American male presented with chronic progressive headaches over two weeks. At first, headaches were intermittent and relieved by Acetaminophen; over time, headaches became constant and associated with nausea, vomiting, blurry vision, and intermittent horizontal diplopia. Symptoms worsened with exercise and straining, improved with lying flat. The patient denied any previous history of headaches.

Past medical history was significant for hypertension and recent Covid 19 infection (PCR positive) three months ago, with mild symptoms. No neurological workup or hospitalization was required. He was quarantined and then returned to usual activities.

He was alert and oriented; vital signs and general examination were unremarkable, while neurological examination showed bilateral papilledema (Grade 2) and shuffling gait. Compared to previous normal CT and MR imaging of the head three years ago, the head CT scan during this admission showed severely enlarged all four ventricles with no clear ventricular outflow obstruction, likely communicating. Brain MRI (Stealth protocol) with and without contrast showed tetra ventricular hydrocephalus possibly related to CSF outflow obstruction around the foramen magnum without any abnormal parenchymal or meningeal enhancement (Figure 1). The patient was admitted to the neurocritical care unit with a neurosurgical consultation and was kept under a close external ventricular drain (EVD) watch. To clarify possible obstruction, Cine MRI (CSF flow study) demonstrated turbulent fourth ventricle CSF flow suggesting probable outflow obstruction.

On admission day two, the patient became bradycardic and hypoxic, with apneic changes requiring EVD placement. Initial opening CSF pressure was 30 mm of Hg. CSF studies showed elevated cell count (WBCs 123, segmented neutrophils 61, lymphocytes 36, RBCs 5.6K, and disintegrated cells) with normal protein and glucose. CSF COVID-19 PCR, Gramstain/culture and meningitis panel, were negative. Serology was negative for cytomegalovirus and HIV.

Later, the patient underwent suboccipital craniectomy to explore and decompress the foramen magnum area, which revealed the fourth ventricular outflow tract subarachnoid web (proven with biopsy). Microsurgical resection of the web was performed (Figure 1).

The patient reported symptoms improvement, and the neuro-exam remained stable. Post-op head CT and MRI scans showed improvement in hydrocephalus but still moderately dilated ventriculomegaly. On post-op day two, EVD was removed (last recorded ICP 37). The patient was later discharged with close follow-up.

Discussion:

Hydrocephalus is a rare infectious complication that can occur at any point during the disease course. Herein, we described a previously healthy patient who developed symptomatic tetra ventricular HCP three months after having a COVID-19 infection.

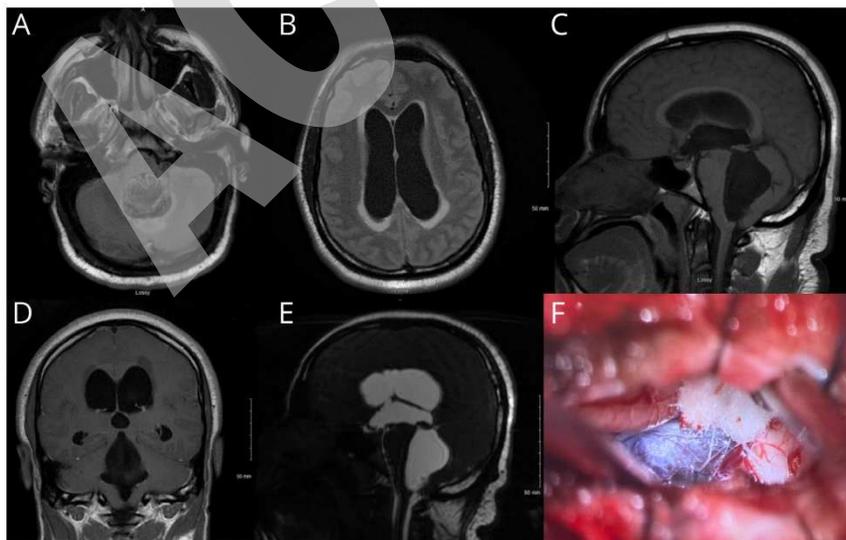
The occlusion of the fourth ventricular outlet is a rare but well-known cause of tetra ventricular hydrocephalus. In most cases, the etiology is attributed to previous parenchymal insults, including infections (such as toxoplasmosis, cysticercosis), inflammation, hemorrhage, neoplasms, Arnold-Chiari malformation, or arachnoid cysts². Arachnoid webs are abnormal formations of the arachnoid membrane in the subarachnoid space that prevents longitudinal CSF movement³.

The pathophysiologic mechanism of increased ICP and hydrocephalus is still unclear; a couple of proposed mechanisms could explain CNS invasion in COVID19. Recent studies have shown that interaction of SARS-CoV-2 viral spike proteins with angiotensin-converting enzyme 2 (ACE2) receptors on the olfactory epithelium leads to potential transsynaptic retrograde transmission via the olfactory nerve into the CNS^{4,6}. Similarly, an abundance of these receptors in the choroid plexus of the lateral ventricles could be another pathway^{4,5}. This proposed interaction between the virus and the choroid plexus could potentially alter the CSF flow dynamics, resulting in an arachnoid web formation^{4,5}. Another proposed mechanism is bloodstream viral spike protein infecting cerebral endothelium, which results in blood-brain-barrier dysfunction and viral CNS invasion^{4,6}.

Conclusion:

We presented a symptomatic tetra ventricular HCP case caused by an arachnoid web successfully treated with microsurgical fenestration surgery. Although it cannot be certain that arachnoid web and HCP occurred secondary to COVID 19 infection, the temporal correlation, given the recent viral infection and normal prior imaging, supports a causal relationship and further studies are needed to understand this association. Nonetheless, recent COVID 19 infection should be kept in the differential diagnosis by the treating physician for HCP of unclear etiology.

Figure 1: MRI showing HCP and intraoperative picture demonstrating dense arachnoid web (A) (B) Selected imaged of axial sections of T2 FLAIR images of the brain demonstrating enlarged fourth and lateral ventricles with peri-ventricular trans-ependymal edema suggestive of active hydrocephalus. (C) Sagittal T1- W FLAIR demonstrating marked lateral, third, and fourth ventriculomegaly possibly related to cerebrospinal fluid (CSF) outflow obstruction around the foramen magnum (D) T1 FLAIR post-contrast scan with no abnormal parenchymal or meningeal enhancement (E) Sagittal Fiesta showing tetra ventricular hydrocephalus (F) Dense arachnoid web at the level of the cerebello-medullary junction (4th ventricular outlet).



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