

A Case of Catatonia Secondary to COVID-19 in an Unlikely Candidate

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Background

Numerous neurological and psychiatric sequelae of SARS-CoV-2 have been described in the literature, ranging from relatively benign (e.g. anosmia, ageusia) to those with higher potential morbidity (e.g. delirium, encephalitis, cerebrovascular accidents, neuroleptic malignant syndrome, psychosis) (Caan, 2020; Divani, 2020; Gouse, 2020). Here we present a case of catatonia secondary to COVID-19, which to our knowledge represents only the 15th case reported thus far in the literature.

Case

A 59 year old male with no significant past medical/psychiatric history presented with worsening cough and shortness of breath 7 days after testing positive for SARS-CoV-2. Chest x-ray showed bilateral, interstitial airway opacities. He was admitted for pneumonia and treated with ceftriaxone and methylprednisolone. His complete blood count, complete metabolic panel, and blood cultures were reassuring. He maintained adequate oxygenation on room air, and his respiratory symptoms gradually resolved.

Discussion

At the onset of his catatonic symptoms, our patient's elevated IL-6 indicates central nervous system inflammation, which is thought to be a significant risk factor for the development of catatonia (Oldham, 2018). The pathogenesis of catatonia is thought to involve aberrant GABAergic, glutaminergic, and dopaminergic signaling in the cortico-basal ganglia-thalamo-cortical pathway (Gouse, 2020). In SARS-CoV-2 infection, massive proinflammatory cytokine release may disrupt dopamine synthesis, leading to the dopamine depletion state seen in catatonia. It remains unclear, however, why an individual patient develops catatonia as a rare sequela of COVID-19.

Ten days after admission, psychiatry was consulted due to acute behavioral changes. His EEG, brain MRI, and vitals were within normal limits, though interleukin-6 (IL-6), C-reactive protein (CRP), and procalcitonin were elevated. His evaluation was notable for staring, mutism, negativism, grimacing, echolalia, and echopraxia, scoring 24 on the Bush-Francis Catatonia Rating Scale (BFCRS). After a positive response to a lorazepam challenge, he was treated with scheduled lorazepam, titrated to a dose of 2mg QID. After 7 days, his BFCRS was 0 and he was discharged to home.

Our patient had no known risk factors predisposing him to a hypodopaminergic state (e.g. Parkinson's Disease, psychotic disorder, hypoferritinemia, dopamine antagonist use.) Despite his moderate respiratory symptoms, at no point was he considered critically ill. He showed no signs of sepsis and did not require supplemental oxygen or mechanical ventilation. Notably, this case illustrates that even a relatively benign course of illness can produce a cytokine storm significant enough to cause profound neuropsychiatric disruption (i.e. catatonia).

Conclusions

As the COVID-19 pandemic evolves, clinicians must be mindful that the neuropsychiatric effects of SARS-CoV-2 infection, specifically catatonia, may present after the resolution of other symptoms and can occur in patients with no identifiable risk factors. Additionally, severity of neuropsychiatric symptoms may not necessarily correlate with the overall clinical severity of the patient's illness.

References

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