A Case of Post-COVID Catatonia Partially Refractory to IV Lorazepam

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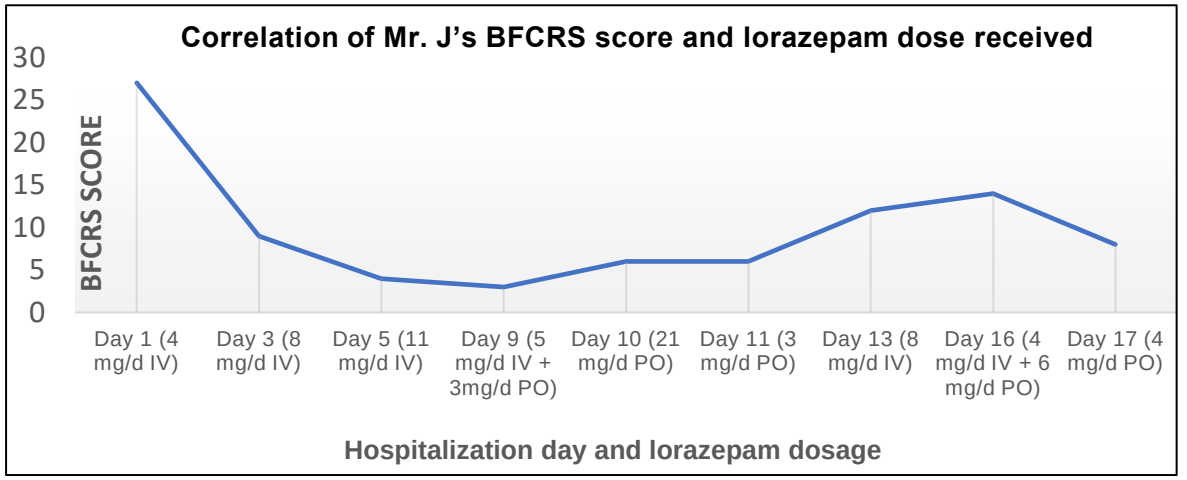
Background: COVID-19 has been associated with neuropsychiatric sequelae, including catatonia, with no consensus on the most appropriate therapeutic approach. We report a case of post-COVID catatonia refractory to standard treatments in a 22-year-old man with history of depression and mild COVID-19 infection, who presented to our Hospital in March 2021.

Discussion: catatonia associated with COVID-19 infection has been described[1-4] with lorazepam as the first line of treatment with mixed results. Electroconvulsive therapy (ECT) remains the standard of care for refractory cases[4], however, it may not be available in certain clinical situations. Catatonia in the setting of COVID-19 may be associated with an increase in pro-inflammatory mediators[2] leading to altered GABA-ergic transmission and a hypodopaminergic state. As in more typical presentations, avoiding antipsychotic agents remains an important consideration to prevent the development of malignant catatonia.

Bush-Francis Catatonia Rating Scale (BFCRS)	
Excitement	Verbigeration
Immobility/stupor	Rigidity
Combativeness	Negativism
Autonomic Abnormality	Waxy Flexibility
Impulsivity	Withdrawal
Mutism	Automatic Obedience
Staring	Mitgehen
Posturing/catalepsy	Gegenhalten
Grimacing	Ambitendency
Echopraxia/echolalia	Grasp Reflex
Stereotypy	Preservation
Mannerisms	**0-3 points per item**



Case and Clinical Course

November 2020: symptoms of clinical depression and anxiety. Partial remission with escitalopram and as needed alprazolam

February 2021: diagnosis of COVID-19 infection, with mild symptoms

Early March 2021: onset of disorganization of thoughts, behavior and speech, followed by catatonia and self-neglect. No improvement with treatment with perphenazine and quetiapine as an outpatient

Mid March 2021: patient was hospitalized, BFCRS of 27 on admission. SARS-CoV-2 PCR was positive, and he had elevation of muscle (CPK) and liver enzymes. An MRI of the brain was unremarkable. Started on intravenous (IV) lorazepam, up-titrated to 3 mg q6hrs, the maximum IV dose he tolerated. Poor oral intake was a challenge throughout the hospitalization. The patient's health care proxy did not consent to ECT

May 2021: persistent symptoms of catatonia. Repeat MRI brain and EEG were both unremarkable. Received 9 sessions of ECT

June 2021: seen as follow-up in clinic, showing full resolution of psychiatric symptoms. Lorazepam was discontinued

Late March 2021: after 17 days in the hospital, patient was discharged once transitioned to oral agents (lorazepam, memantine and bupropion) with residual symptoms of catatonia and BFCRS of 8

Proposed Pathophysiology

- Neurotropism of SARS-CoV-2 conferred by the binding of its surface glycoproteins to ACE2 enzyme present in neurons
- Direct viral encephalitis, as well as less direct effect of the infection, including inflammation, hypoxia, hypercoagulability, postinfectious auto-immunity, or effects of immunomodulatory treatments
- Alterations in GABA-ergic and dopaminergic modulation of the cortico-basal ganglia-thalamo-cortical circuit
- Exposure to proinflammatory cytokines has been associated with altered GABA-ergic transmission in the basal ganglia
- Proinflammatory mediators such as interferon-alpha have been associated with a hypodopaminergic state in the basal ganglia which is postulated to be a potential precipitating factor to the development of catatonia and neuroleptic malignant syndrome

Conclusion: Consultation-liaison psychiatrists are increasingly managing neuropsychiatric sequelae of COVID-19, including catatonia. Knowing how to readily recognize it and prevent and/or efficiently treat its complications is imperative.

References

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