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Delayed onset catatonia after COVID-19

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Abstract

COVID-19 has many complications that are associated with this infection. Neuropsychiatric symptoms are common and can present with symptoms documented both during acute COVID-19 infection and developing after the resolution of respiratory symptoms. Patients have presented with a variety of symptoms such as anosmia, seizures, cognitive and attention deficits, new or progression of existing anxiety, depression, psychosis, and rarely catatonia. Although rare, catatonia and each of its subtypes have now been reported as complications of COVID-19 and therefore, should be considered known to occur in both during the acute and postinfectious states. Diagnosis of catatonia in the context of COVID-19 should be considered when work-up for more common medical causes of encephalopathy are negative. There have been cases documented in the literature of patients presenting to the hospital with catatonia during COVID-19 infection. However, we present a case of akinetic catatonia in setting of COVID-19 infection and premorbid serious mental illness that was diagnosed and treated on an outpatient basis with close collaboration between primary care and psychiatry.

Keywords:
Catatonia
Outpatient
Coronavirus
Encephalopathy

Objective

To alert both inpatient and outpatient clinicians to keep a high index of suspicion for catatonia when consulted for abnormal behavior or altered mental status in COVID-19 patients especially when work-up for more common medical causes of encephalopathy are negative.

Case report

A 51-year-old male with remote history of bipolar disorder treated for mania and depression over 20 years ago but no known psychiatric symptoms or care since then, presented to an outside emergency department for acute, progressive altered mental status. He was diagnosed with COVID-19 approximately three weeks before current presentation but at that time he was largely asymptomatic and did not require additional interventions. During the time between the COVID-19 diagnosis and his presentation to the emergency department, patient’s wife reported that patient became very lethargic, less communicative/interactive, and behaving strangely to the point where he would not recognize her. Because of the acuity of symptoms without clear etiology, he was admitted to hospital on the neurology service. Upon admission, patient’s vitals were unremarkable and a comprehensive work-up is listed below (Table 1).

His COVID-19 PCR tested was repeated and was still positive. Additionally, a CT angiogram of the head and neck and MRI of the brain was performed to work up his acute encephalopathy, which were unremarkable. Lumbar puncture revealed a clear and colorless fluid that was normal for all tests which included autoimmune panel, protein, cell count, cytology and culture; this work up did not yield any explanation for the etiology of his encephalopathy (Table 2). His urine drug test was negative for all substances; heavy metal screen which included copper, lead, mercury, cadmium and arsenic were all negative. Psychiatry was consulted while patient was admitted and felt that this was an organic etiology and not an underlying psychiatric one. An EEG was done that showed nonspecific slowing consistent with encephalopathy. He was treated supportively in the hospital for COVID encephalopathy and was discharged a week later.

He was seen for follow up with his primary care physician (PCP) shortly after his discharge. The patient was noted to be lethargic, minimally communicative, with a slow gait and minimally interactive. Differential diagnosis at this point was for encephalopathy versus catatonia. Due to the severity of the vegetative symptoms, the patient was urgently referred to both in-system outpatient neurology and outpatient psychiatry consultation service. Neurology did not believe this was long-COVID syndrome or encephalopathy or any organic neurological process. Outpatient repeat MRI and EEG were subsequently obtained which were both unremarkable. However, given concern for COVID induced catatonia, neurology also advocated for a thorough psychiatric evaluation.

At the advice of psychiatry and prior to his initial appointment with the psychiatrist, the PCP prescribed a short-term prescription of oral lorazepam 2 mg daily. The patient’s wife stated the lorazepam elicited an immediate change (more mobile and interactive) about 1-hour after taking it which lasted most of the day and during those lucid moments, he would share extensive persecutory delusions. Upon initial evaluation,
the consulting psychiatrist conducted a Bush–Francis catatonia rating scale and patient’s score was 17 with prominent akinetic catatonia symptoms: hypoactivity, withdrawal, mutism, stereotypy, mild waxy flexibility, and amimia. As patient was medically stable and family very involved, it was decided hospitalization was not necessary and outpatient treatment was continued with close monitoring by family and providers. Although initially responsive to oral lorazepam 1 mg every 8 h, efficacy waned and unable to further titrate dosage due to oversedation concerns, therefore outpatient ECT was pursued. Patient received a total of 8 bitemporal acute ECT treatment sessions and responded well. At his most recent outpatient psychiatry follow-up, patient’s catatonia was completely resolved although some paranoia, anxiety, and depression remained. He was weaned off lorazepam and started on risperidone, and is now back to his baseline.

Discussion

Catatonia is a neuropsychiatric syndrome characterized by abnormal movements, behaviors, and social withdrawal. When catatonic symptoms present, the cause is likely psychiatric, but many medical etiologies such as autoimmune, metabolic, and infectious, such as COVID-19 can lead to the development of catatonia (Burrow et al., 2021). COVID-19 catatonia is a rare neuropsychiatric complication of COVID-19. Catatonia is often linked to psychiatric disorders, but it also is underrecognized cause of altered mental status in medically ill patients (Caan et al., 2020). The etiology behind COVID-19 associated neuropsychiatric symptoms appear to be systemic inflammation, but still largely unclear. The virus may enter the central nervous system from the nares through the cribriform plate or via hematogenous spread across the blood-brain barrier (Territo et al., 2021; Spiegel, 2022). Given the morbidity and mortality associated with catatonia, it has been emphasized that inpatient providers should keep a high index of suspicion for catatonia when consulted for abnormal behavior or altered mental status in COVID-19 patients (Scheiner et al., 2021), however it is equally important that outpatient providers also keep a high index of suspicion as catatonia is often underrecognized and misdiagnosed and that collaboration between primary care and psychiatry is vital. New-onset catatonia with or without pre-existing psychiatric illness and concurrent COVID-19 can be safely and effectively managed with lorazepam and/or ECTs (Sakhardande et al., 2022). The vast majority of case reports so far reported catatonic symptoms related to COVID-19 infection that was diagnosed and acutely managed in an inpatient setting, and in general there is limited data on the prevalence and treatment of catatonia in the outpatient setting (Solmi et al., 2018). We present a rare phenomenon of a patient presenting with delayed-onset catatonia after COVID-19 infection (Zain et al., 2021) that was diagnosed and treated in an outpatient setting.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Table 1

| Variable          | Result            | Reference range |
|-------------------|-------------------|-----------------|
| COVID19 PCR       | Positive          | Negative        |
| BMP               | NL, creatinine at baseline | N/A |
| Hepatic Function  | Notable for elevated total | Total Bilirubin: 0.3–1.2 mg/dL. |
| Panel             | bilirubin of 1.8 with a direct of 0.6 and indirect of 1.2 mg/dL. | Direct: 0–0.3 mg/dL. |
| ALT elevated at 86 U/L. | Indirect: 0.1–1.0 mg/dL. |
| CBC               | NL, except platelet count of 533,000 | ALT: 10–49 U/L. |
| TSH               | 2.78 uIU/mL       | Platelets: 150,000–450,000 /ul. |
| Free T4           |                  |                 |
| ESR               | 18 mm/hr          | 0–23 mm/hr      |
| CRP               | <4 mg/L           | 0–10 mg/L       |
| LDH               | 201 U/L           | 120–246 U/L     |
| Ethanol level     | Negative          | Negative        |
| Acetaminophen level | < 10 ug/mL         | 10–20ug/mL     |
| Salicyrate level  | <3 mg/dL          | 15–30 mg/dL     |
| Ammonia level, blood | 27 umol/L        | 8–32 umol/L    |
| Venous blood gas  | WNL               | N/A             |

Table 2

| Variable                  | Result                | Reference Range     |
|---------------------------|-----------------------|---------------------|
| CSF appearance and color  | Clear and colorless   | N/A                 |
| CSF nucleated cells       | 2 /ul                 | 0–5 /ul             |
| CSF Red blood cells       | 10 /ul                | 0–1 /ul             |
| CSF protein               | 90 mg/dL              | 15–45 mg/dL         |
| CSF glucose               | 69 mg/dL              | 40–70 mg/dL         |
| CSF, Cytology             | Negative for malignancy | Negative            |
| Meningitis/Encephalitis CSF panel by PCR | Negative | Negative |
| Oligoclonal bands, CSF   | 0                     | <2 bands            |
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