A case report on concurrent catatonia in a man with COVID-19

Abstract:

Introduction: Coronavirus disease 2019, the disease caused by SARS-CoV-2, has contributed to considerable morbidity and mortality in 2020. While respiratory symptoms are predominant in COVID-19, there have been reports of neurologic sequel including central nervous symptoms such as catatonia, which is a complex syndrome of bizarre motor behavior, impaired volition, and vegetative abnormalities.

Here we present a case of catatonia as the first symptom in a patient with Covid-19, and his challenging treatment.

Case: We describe a case of concurrent COVID-19 and catatonia in a 43-year old man without any prior psychiatric history, who was hospitalized with loss of consciousness. His symptoms of catatonia improved with the administration of ECT.

Discussion: We highlight the importance of the consideration of catatonia as a possible cause of loss of consciousness in medically ill patients. we investigate the various possible mechanisms of Covid-19 involvement in the development of catatonia and the methods of dealing with a patient with Covid-19 and psychological symptoms admitted in the ICU.

Conclusion: Not only catatonia can co-occur with COVID-19, but it may be the first and only Feature presented in the context of this infection, so it deserves special attention especially in unconscious patients. In medically-ill patients with Covid-19 and catatonia symptoms for which benzodiazepines can't be administered, ETC use can be considered as a low-complicated and rapid method.

Keywords: Coronavirus disease 2019; SARS-CoV-2; COVID-19; COVID-catatonia; Electroconvulsive therapy

Introduction:

Catatonia is a psychomotor and potentially lethal syndrome marked by a distinct constellation of motor and behavioral disturbances (1).

Prevalence rates of catatonia vary depending on catatonia concepts criteria but in mixed inpatient populations of psychiatric institutions, catatonia appears to have a prevalence of 10–25%. Up to 40 different signs and symptoms have been associated with catatonia. These signs may be summarized in 4 groups: pure motor signs (e. g. posturing, rigor, immobility), disturbances of volition (e. g. ambitendence, negativism, automatic obedience), inability to suppress complex motor activities (e. g. stereotypies, rituals, echophenomena), and autonomic instability (e. g. tachycardia, hyperthermia) (2). Approximately 25% of Catatonia cases are precipitated by psychiatric, metabolic, autoimmune, inflammatory, infectious, and neoplastic conditions (1, 3, 4). The vast majority of morbidity and mortality of catatonia is readily avoidable and requires a simultaneous search for contributing factors. Nearly all cases are likely to respond to treatment with benzodiazepines and/or electroconvulsive therapy (ECT) (5).

The most recent pandemic of coronavirus infection is coronavirus disease (COVID-19) that is caused by SARS-CoV2 (6). A recent review identified 124 cases of catatonia comorbid with infections, and 38% of them had viral infections (7). SARS-CoV2 also has neuroinvasive capabilities. A study that specifically investigated this issue documented that one-quarter of the hospitalized patients with a confirmed diagnosis of severe acute respiratory syndrome from coronavirus2 infection had some manifestations of CNS involvement (8). The most common symptoms of COVID-19 illness are fever, cough, and fatigue. Appropriately, the initial focus in Covid-19 pandemic is on respiratory emergencies, but emerging cardiovascular events, inflammatory and neuropsychiatric problems are other concerns (5, 6).

The potential neuropsychiatric mechanisms of COVID-19 are many: from direct viral encephalitis to cytokine dysregulation, immune cell transmigration, post-infectious autoimmunity, effects of immunomodulatory treatments, hypoxic brain injury, and posttraumatic stress from a near suffocation event compounded by required social isolation (5).

As mentioned, altered mental status is common in patients admitted to hospital with severe infections; among which are COVID- 19 patients who experience respiratory failure while developing delirium and other neurological and neuropsychiatric complications, however, there are only a few case reports about the incidence of catatonia alongside the COVID-19 infection (3, 9). Thus in this study, we have demonstrated the case of a young patient with catatonic features as a first complaint and positive covid-19 infection and Urosepsis at the same time.

Case:

A 43-year-old vegetarian man without a significant past medical, substance use, personal, or family psychiatric history, presented to the emergency department for a Stupor. At that visit, he was noted to have a temperature of 37 °C; blood pressure 96/65 mmHg, heart rate 89 bpm, and oxygen saturation was 97% on room air. The psychiatry consultation-liaison team was consulted. In the physical examination, the patient exhibited psychiatric signs including immobility, mutism, and negativism. He was unresponsive to the voice and just had a small muscle contraction and no withdrawal with a painful stimulant.

The first-line blood tests were requested. In the initial CBC test the patient had WBC=9200 cells/mcL (with normal range of 4500-11000 cells/mcl) with lymphocyte=15%, neutrophil=80%, monocyte=3% and Eosinophil=2%. Hb was 10.3 g/dL (NL=12-18) with RBC count= 2.77 million cells/mcL (NL= 4.2-5.3), MCV=107 fL (NL=80-109), MCH=38 pg (NL=27-32), and Plt =115000/mcL (NL=150-450) and patient had ESR=15 mm/hr (NL<13) and CRP=39 mg/L (NL<10), and BS was 87 mg/dL (NL<140). The VBG, Serum electrolyte levels, renal and hepatic function tests were normal.

In Urine analysis, the patient had WBC= many with few bacteria and RBC=1-2/hpf. Serum B12 level was 216 ng/L(NL=1100-1200) which described megaloblastic anemia.

The drug screen test was negative. He underwent a head CT-scan without contrast which showed no acute intracranial abnormality. He then underwent a lumbar puncture, and his cerebrospinal fluid was unremarkable with no polymorphonuclear leukocytes or organisms were seen on gram stain.

The abdominopelvic sonographic evaluation was normal. Chest x-ray showed reticulonodular changes in lung parenchyma with open costophrenic angles and normal heart size and mediastinum. he could have COVID 19, so the COVID-19 serum PCR along with the blood, and urine culture were done. Amp Ciprofloxacin 400 iv BD was started and the patient was admitted to the ICU.

On the second day of hospitalization, the patient's serum WBC raised to 12700 cells/mcL with Hb=10.3 g/dL, MCV=104 fL, MCH=37.6 pg with ESR=20 mm/hr and CRP=47 mg/L. Coagulation and thyroid test results showed no abnormality also serum viral markers were negative. Amp meropenem 1 gr iv TDS and amp vancomycin 1 gr iv stat then BD started and Ciprofloxacin was discontinued.

Till 2020.8.19 Patients WBC count was reached 16300 cells/mcL with Hb=10.3 g/dL, MCV=104 fL and Plt= 246000/mcL, also ESR and CRP were raised to 45 mm/hr and 70 mg/L respectively. The serum COVID-19 PCR test was positive and the blood culture test showed the growth of Pseudomonas aeruginosa. The

chest CT scan showed diffuse ground-glass opacifications in both lungs with normal mediastinum and heart size which was demonstrated to be related to COVID-19 infection. In the next consultation, Meropenem was discontinued and antibiotics were changed in favor of test results to amp cefepime 2 gr iv BD, amp aciclovir 200mg q4h, and vancomycin was continued. A significant drop in WBC count and inflammatory markers was observed in the next days; (to WBC=11500 cells/mcL and CRP=53 mg/L on the 7th day of hospitalization). The final antibiotics combine were amp ciprofloxacin 400 iv BD and amp cefepime 2 gr iv BD.

Regardless of improvement in Blood test components in two weeks of admission, the patient's mental status was still altered and no improvement was seen in his symptoms. His temperature was 37.2°C and his heart rate was 107 bpm; oxygen saturation was 96% on room air; other vital signs were within normal limits. The Bush-Francis Catatonia Rating Scale (BFCRS) was administered. The Bush-Francis Catatonia Screening Instrument (BFCSI) comprises the first 14 items of the 23-item BFCRS. The BFCSI score is reported as a tally of the number of items present in items 1-14, while the BFCRS is reported as the total severity score of all 23 items.

His initial BFCSI/BFCRS score was 15/21, indicating the presence of catatonia, and also met DSM-5 criteria for Catatonic Disorder. It was decided to give him electroconvulsive (ECT) therapy. On hospital day 14, patient got his first ECT session and soon after that, he opened his eyes but with no response to any foreign stimulant. It lasted for 30 minutes and the patient went back to a stupor state again. The second ECT was given in two days but the patient showed no change in his mental status. The third ECT was done two days later and this time patient became conscious and was noted to be alert and oriented to person, place, and time, and verbally communicated for at least 6 hours, and then went back to the previous state again. The patient got the ECT session for the fourth time with a diagnosis of schizoaffective disorder with sepsis and COVID-19 infection with catatonic features. Afterward, he was reassessed by the Psychiatry service. Within two days, he was discharged from the hospital with normal orientation and full remission of catatonic features, and olanzapine 5mg per night was prescribed. The patient went to the clinic the week after complaining of nothing but fatigue and loss of appetite.

Discussion:

We describe a case of catatonia in a young man with COVID-19, who responded favorably to repeated electroconvulsive (ECT) therapy. Catatonia is a psychomotor syndrome associated with a range of psychiatric and medical illnesses and can entail increased, decreased, and abnormal psychomotor activity. Catatonia can manifest itself with prominent motor abnormalities, mutism, withdrawal, or acute worsening of symptoms after exposure to a neuroleptic, etc. (3) Furthermore, it has been observed in about 10% of acutely ill psychiatry patients, only a minority of whom have schizophrenia. Catatonia has been associated with over 100 medical conditions, including diffuse cerebral dysfunction, and numerous neurotransmitter dysregulation(4, 10). Some patients with COVID-19 will possibly show nonspecific neurological symptoms, such as confusion and headache. A few patients with COVID-19 showed more specific neurological manifestations, such as seizure, cerebrovascular problems, or catatonia. In an early COVID- 19 manifestation, these symptoms might be observed in patients who will later develop the typical symptoms. Neurological symptoms in Covid-19 patients include one case of catatonia (11)and mutism (12)and a Case of Concurrent Delirium and Catatonia (3). To our knowledge, we describe the first case of COVID-19 associated with co-occurring Urosepsis and catatonia.

In Marissa P et al. and Jamelleh Amouri et al. study, the patients didn't have a known psychiatric history same as our study case, but in the first case, he appeared to have psychotic symptoms before his hospitalization, including delusions and auditory hallucinations. The other difference was in the use of contributive medications by the first patient such as azithromycin and baclofen, which are associated with catatonia, yet it was not the same in our case.

In such patients resolving the underlying cause can make a big difference in their situation. Iatrogenic and environmental factors such as prolonged mechanical ventilation, use of sedatives, immobilization, and social isolation can increase the risk of delirium, same as our patient who was living alone; so we can't ignore the effect of these factors. There is still a lack of knowledge in the mechanism of action of the sars-cove2 virus in the central nervous system. The pathogenesis may be through a direct toxic effect, an immune response, disinhibition or activation of excitatory receptors as well as disturbance of excitatory neurotransmitter uptake/clearance or interference with ion homeostasis, which is hard to determine by the neuroimaging studies (eg. CT-scan, MRI) because of the unchanged brain parenchyma and normal features of the CNS in all known modulations, an example of which is our case and Jamelleh Amouri et al. with the normal brain CT-scan. The only way for approving the causation of COVID-19 in such a scenario is by studying the CSF by PCR or using immunohistochemistry like Jan Mulder et al. study. They detected IgG autoantibodies against mouse brain neuronal proteins in serum and CSF at

admission and immune reactivity was observed in all brain regions investigated (cerebral cortex, hippocampal formation, thalamus, caudate-putamen). (3, 4, 7)
Due to the lack of facilities to examine the cerebrospinal fluid for COVID-19 infection, it is impossible to prove 100% of the causality of SARS-COV19 in the development of catatonia symptoms in our case. Although most psychiatric diagnoses consider as new by the notifying psychiatrist or neuropsychiatrist, we cannot exclude the possibility that these were undiagnosed before the patient developed COVID-19. Therefore, it is very likely that our patient had undiagnosed bipolar or schizoaffective disorder before infection with Covid-19, and developed catatonia at the same time as the infection. However, in the current pandemic, the co-occurrence of viral infection and catatonia in someone with no catatonia or any other psychiatric records raises the suspicion of a cause-and-effect relationship between the two events.

Catatonia is an emergent state requiring acute treatment and the catatonic signs typically resolve dramatically and completely with benzodiazepine and ECT therapy (5, 10). ECT is an essential psychiatric service that provides lifesaving treatment for severe mental illnesses if given in a timely fashion, for which there is no viable alternative. However, ECT service is often not prioritized in hospitals (13), as our patient condition with COVID-19 infection and urosepsis, along with a loss of consciousness, made the decision very difficult and led to a delay in the procedure. In comparison Marissa. P case was treated initially with 1 mg of intravenous lorazepam 3 times daily for catching a normal state of consciousness Which was not possible in our patient due to stupor status (4). Also, in another case, the symptoms of both delirium and catatonia in presence of COVID-19 infection improved with the administration of low-dose lorazepam (3).

Although there are other inherent challenges of an ECT service in a COVID-19 environment. ECT requires general anesthesia which involves close contact with patient oral and airway secretions and actively encouraging the patient to cough in the recovery room after ECT (13) Which is not possible in critically ill patients admitted to the ICU ward. There were also other concerns about other differential diagnoses such as non-convulsive status epilepticus in the presence of COVID-19 and encephalopathy, when the patient responded to the third ECT session, it was ruled out.
To confirm the interconnection between COVID- 19 and new acute neuropsychiatric complications detailed prospective longitudinal studies are required. Further research is also needed to clarify the basic mechanisms of catatonia in COVID-19, and explore the barriers of accessing an alternative treatment for catatonia in critically ill patients who cannot tolerate IV benzodiazepines.

Conclusion:

This case highlights the challenges of the management of catatonia with ECT therapy in an individual with COVID-19 infection and the development of catatonic symptoms alongside high pro-inflammatory markers such as CRP which is associated with worse clinical outcomes in COVID-19.

In the medically ill population, Catatonia is an under-recognized cause of altered mental status and there is increasing evidence suggesting that catatonia can occur in up to one-third of critically ill patients. Therefore it is crucial to keep catatonia on the differential diagnosis of altered mental status in patients with COVID-19, and that the formal evaluation for catatonia be performed when it is suspected.

Data availability: The data that support the findings of this study are available from the corresponding author, borna, s., upon reasonable request.

Conflicts of Interest: The authors declare that there is no conflict of interest regarding the publication of this article.

Funding Statement: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Patient consent: no written consent has been obtained from the patients as there is no patient identifiable data included in this case report.

References:

1. Gouse BM, Spears WE, Archibald AN, Montalvo CJB, behavior,, immunity. Catatonia in a hospitalized patient with COVID-19 and proposed immune-mediated mechanism. 2020.
2. Walther S, Strik WJCs. Catatonia. 2016;21(4):341-8.
3. Amouri J, Andrews PS, Heckers S, Ely EW, Wilson JEJP. A Case of Concurrent Delirium and Catatonia in a Woman with COVID-19.
4. Caan MP, Lim CT, Howard MJP. A case of catatonia in a man with COVID-19. 2020.
5. Cooper JJ, Ross DAJBP. COVID-19 Catatonia—Would We Even Know? 2020;88(5):e19.
6. Asadi-Pooya AA, Simani LJJotNS. Central nervous system manifestations of COVID-19: A systematic review. 2020:116832.
7. Mulder J, Feresiadou A, Fallmar D, Frithiof R, Virhammar J, Rasmusson A, et al. Autoimmune Encephalitis Presenting with Acute Excited Catatonia in a 40-Year-Old Male Patient with Covid-19. 2020.
8. Mao L, Wang M, Chen S, He Q, Chang J, Hong C, et al. Neurological manifestations of hospitalized patients with COVID-19 in Wuhan, China: a retrospective case series study. 2020.
9. Varatharaj A, Thomas N, Ellul MA, Davies NW, Pollak TA, Tenorio EL, et al. Neurological and neuropsychiatric complications of COVID-19 in 153 patients: a UK-wide surveillance study. 2020;7(10):875-82.
10. Rosebush PI, Mazurek MFJSb. Catatonia and its treatment. 2010;36(2):239-42.
11. Rogers JP, Chesney E, Oliver D, Pollak TA, McGuire P, Fusar-Poli P, et al. Psychiatric and neuropsychiatric presentations associated with severe coronavirus infections: a systematic review and meta-analysis with comparison to the COVID-19 pandemic. 2020.
12. Pilotto A, Odolini S, Masciocchi S, Comelli A, Volonghi I, Pezzini A, et al. Steroid-Responsive Encephalitis in Coronavirus.
13. Tor PC, Phu AH, Koh DS, Mok YMJTJoE. ECT in a time of COVID-19. 2020.