

Case Report
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Case Report: Covid-19 and Catatonia

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Introduction

Catatonia is psychomotor syndrome associated with a range of psychiatric and medical illnesses [1]. It was first described in 1874 by German psychiatrist called Karl Kahlbaum. Historically, catatonia has been related to schizophrenia only, but with time, it become evident that many medical conditions might cause it, reach up to 25% of cases. Moreover, these medical conditions are widely various including metabolic, autoimmune, inflammatory, infectious, and neoplastic conditions [1, 2]. However, Due to this low level of awareness catatonia among general medical doctors is likely to be underdiagnosed which subject the patient to increased risk of morbidity and a potentially fatal outcome [3]. In view of infectious cause for catatonia, the pathogenesis is thought to be due to either a direct toxic effect of pathogen or an immune response [1, 4]. Despite that Coronavirus disease 2019 (COVID-19) known to cause severe acute respiratory symptoms, surprisingly, there have been reports of neurologic symptoms [5]. By the way, the presence of catatonia denotes the severity of the underlying illness [6]. According to Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), catatonia is associated with a mental disorder and is diagnosed when the clinical picture is dominated by at least three of the following: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, agitation, stereotypy, grimacing, echolalia, or echopraxia [3]. As catatonia is treatable, timely diagnosis and treatment significantly improves patient outcomes. Patients suffering from catatonia are at high risk of developing medical complications such as dehydration, malnutrition, deep vein thrombosis, pulmonary embolism, pressure ulcers, contractures, constipation, urinary tract infections, and aspiration pneumonia [3, 7]. The relative prevalence and diagnostic significance of catatonic signs differ among studies and patient populations, but there is general agreement that catatonia occurs in 9%-17% of patients with acute psychiatric illnesses and that retarded catatonia is the more frequently observed subtype [8, 6].

The syndrome is grouped under three forms of presentation: retarded (characterised by mutism, rigidity, immobility, negativism, catalepsy, posturing and echophenomena), excited (with restlessness, impulsivity and aggressively presenting as the main symptoms) and malignant catatonia, a life-threatening form of

which fever and autonomic instability are the warning signs [5, 9].

Here we present a case report of COVID-19 patient with pre-existing psychiatric illness presented with catatonia.

Case

Mrs N, a 39 years old lady, employed, married and having three children, without significant past medical, substance use or personal or family psychiatry history, apart of having one episode of changing in her behaviors and depressed mood eight months prior to her presentation with COVID-19, which kept at that time on antipsychotic, olanzapine 10 mg at night daily, improved dramatically in few days then discharge, patient stopped taking olanzapine since discharge and was functioning well with no persistent symptoms. Follow up appointment in psychiatry OPD given after 2 weeks of discharge but patient didn't show up.

Patient presented to emergency department on 21/09/2020, with history of sore throat and mild dehydration due to poor oral intake for 5 days prior to ED visit, her vitals were normal but tested positive for COVID-19 at that time. Intravenous fluid given along with analgesia and discharged after few hours of stabilization and advised to quarantine at home.

Four days later, she re-presented to emergency department for depressed mood, anhedonia and poor oral intake. She was afebrile 37.5c, pulse rate of 88/min with normal BP 132/76, oxygen saturation was 95% on room air. And other vital signs were within normal limits. A chest x-ray was performed, which showed no acute cardiopulmonary abnormalities. Blood work up done and showed normal inflammatory markers, blood glucose was normal, as were serum electrolyte, C - reactive protein, creatinine kinase and lactic acid. Serum drug screen and pregnancy test were negative. Liver and kidney functions showed no abnormalities and her leukocyte and neutrophil counts were within normal limits. A COVID-19 nasopharyngeal swab polymerase chain reaction test was advised along with lumbar puncture to rule out central nervous system involvement, but her husband refused despite thorough counselling. She underwent CT brain and revealed no significant abnormalities apart of broad based extensive

calcified bony structure noted along with left side of anterior interhemispheric fissure and along cerebral falx in the sitting of very prominent calcification measuring about 2.4×1.1 cm, and the same finding was seen in previous CT brain done 8 months back with no changes. After consultation of neurology, found that these finding not significant and she needs lumbar puncture to rule out CNS involvement of COVID-19 but her husband again refused.

As per history from her husband, patient reported anxiety and insomnia due to her concern about COVID-19. later, she was observed acting strangely for one week prior her first visit to emergency department, and noted to be isolated, lying in her bed most of the time with staring look toward the ceiling, not talking as usual with poor sleep and oral intake. He also reported that patient was suspicious towards her colleagues at work recently and expressing fears that they are trying to infect her with COVID-19 intentionally. Mini-mental state examination revealed poorly kempt lady with poor hygiene status, lying abnormally in lateral position and staring to the ceiling with fixed gaze and mute though she was conscious. Stat dose of lorazepam 2 mg IM given with impression of catatonia. Patient reassessed after 1 hour of given the injection and started to talk, mixed relevant and irrelevant and expressed her fear that one of her colleague, who was retired since long time wanted to fire her and infect her with COVID-19. She also revealed that she is pregnant despite negative pregnancy test done in previous visit. She denied auditory or visual hallucinations at that time. Mrs N was admitted that evening to the COVID ward for further workup and kept on lorazepam 1 mg twice daily and olanzapine 5 mg resumed.

The psychiatry consultation-liaison team was consulted the next day. On that day, Mrs N was noted to be alert, oriented to time, place and person but irritable and agitated, wanted to go back home and refusing food. Stat dose of lorazepam 2 mg given, and she calmed down. Few hours later, patient observed not moving, lying in one position, and not responding to commands, was staring blankly at the ceiling, another dose of lorazepam given, and she slept. She was re-assessed and found responding to call by opening her eyes only, but no verbal response and she kept at the same left lateral position without moving. Her vital signs remain normal since admission, neurological examination done and found to be mute and retarded but opening her eyes when calling, neck rigidity present along with marked rigidity all over her limbs, more in lower limbs and having brisk reflexes all over and waxy flexibility. Neurology consulted at that time to rule out central nervous system involvement so advised to do full autoimmune workup including ammonia, ferritin, ceruloplasmin, anti-neutrophil cytoplasmic antibody, antiphospholipid syndrome, limbi encephalitis screen, paraneoplastic syndrome, ANA and autoimmune thyroid, which all came negative. EEG and MRI brain also advised but due to COVID-19 pandemic and hospital policies, couldn't do it to minimize exposure and transmission, so CT brain done instead and revealed no changes comparing to previous one. Referral to dietitian and nasopharyngeal tube advised but her husband refused, so kept on intravenous fluid as she was dehydrated. Dose of lorazepam increased to 2 mg twice daily.

Two days later, patient started to respond to voice, conversing and answering questions and moving in her bed, she verbalized having dyspnoea and back pain so analgesia given but she was still refusing oral feed but accepting sips of water.

At day six of admission, she was reviewed by neurology again and autoimmune workup showed no significant abnormalities. Neurological examination done and showed improvement in her

rigidity but still having brisk reflexes. Patient was able to open her eyes spontaneously, obeying commands and minimally moving out of her bed and accepting some food.

Two days later, patient showed improvement, remains non-rigid, conversing better and eating and drinking adequately with persuasion. But she looks sad and crying on and off and asking to see her family. COVID-19 team was following her daily during her hospital stay, her vitals and chest examination were normal, and patient didn't show any respiratory symptoms or spiking fever.

At day 10 of admission, de-isolation done, and patient prepared to be shifted to psychiatry ward. A day after, patient re-evaluated by psychiatry team, patient was communicative and expressing well herself and she gave details about what happen prior current illness. She revealed that she was psychologically well and turned back to work until gets infected with COVID-19, where her worries started and gradually isolated and her function impaired. Patient kept in the psychiatry ward two more days with marked improvement in her mood and behaviors noted by medical staff, family, and doctors. During her hospital stay, patient kept on lorazepam 2 mg twice daily and olanzapine resumed and gradually increased to 15 mg once daily. Patient accepting medications after thorough psychoeducational counseling about her illness and treatment of choice. Then , she was discharged home with same medications with follow up appointment after two weeks.

Fortunately, patient and her husband came to psychiatry outpatient clinic after two weeks, her status was stable, and she started to take care of herself, spending time with her family with no abnormal behaviors noted. Her sleep and appetite were gradually improved. So kept on same dose of olanzapine and lorazepam was tapered down gradually and slowly in next visits.

Discussion

According to Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), catatonia is diagnosed when at least three symptoms or signs present. In our case, patient presented with mutism, negativism and posturing along with dehydration as result of poor oral intake, So diagnosis of catatonia was made [10]. In addition to that, Benzodiazepines are the mainstay of the treatment of catatonia and are helpful as a diagnostic probe. A positive Lorazepam Challenge Test validates the diagnosis of catatonia, which actually occurred in our patient as she responded well after lorazepam 1 mg intramuscular injection challenge, which supported the diagnosis of catatonia as she started to move and talk. However, her speech mixed relevant and irrelevant [7]. Moreover, she expressed persecutory delusion that workmate against, these psychotic symptoms preceded by anxiety symptoms might indicate that her catatonia might be caused by relapse of her previous psychiatric episode, which was eight months back with different presentation [11]. It well known that catatonia signs and symptoms might fluctuate between retarded and excited subtypes. However in our care patient sometime, her catatonia signs alleviated and sometimes become prominent over first 4 days during admission [12]. It know that mainstay of initial treatment of catatonia caused by psychotic illness with antipsychotic and benzodiazepines with possibility of initialing ECT in case of poor response to initial measures .So, the need of starting antipsychotic was indicated on her case which olanzapine 5 mg , as she showed good repose on it in past psychotic episode along with continued on lorazepam oral tablet as regular doses two time a day. More importantly, central nervous system involvement rolled out despite Lumbar puncture was not done [12]. However ,other possible causes such as hypoactive delirium , was less likely as patient has

no fluctuation on level consciousness .By the way, neuroleptic malignant syndrome were not considered as patient was not in any antipsychotic medications before the onset of her symptoms ,in addition to that , no other supportive clinical features of it.

Conclusion

Catatonia is psychomotor syndrome associated with a range of psychiatric and medical illnesses, and it is associated with multiple life-threatening complications if not treated [1, 13]. Catatonia is heterogeneous in presentation and cause. In this case, report, we describe the first case to our knowledge of catatonia associated with acute COVID-19 [4]. In our case, the cause could multifactorial either due to direct or indirect effect of covid-19. We support that catatonia to the growing list of neuropsychiatric phenomena observed in patients with COVID-19 which also reported by new recent emerging case reports [14]. Our case report, highly suggest to increase the awareness of catatonia among medical doctors for early detection and management which warrant better outcome.

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