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Concurrent catatonia and COVID-19 infection – An experiential account of challenges and management of cases from a tertiary care psychiatric hospital in India

ARTICLE INFO

Keywords
COVID
Catatonia
Neuropsychiatric
Benzodiazepines
Electroconvulsive therapy
ECT

ABSTRACT

Catatonia has been reported as one among many neuropsychiatric manifestations associated with COVID-19 infection. Catatonia and COVID-19 co-occurrence remain clinical concerns, often posing challenges pertaining to diagnosis, and especially management. Limited information is available regarding the appropriate approaches to the management of catatonia in COVID-19 infection, particularly with reference to the safety and efficacy of benzodiazepines and Electro-convulsive therapy (ECT). We present our experience of five patients with catatonia consequent to heterogeneous underlying causes and concurrent COVID-19 infection, who received care at the psychiatric COVID unit of our tertiary care psychiatric hospital. An interesting observation included varying underlying causes for catatonia and the potential role that COVID-19 infection may have played in the manifestation of catatonia. In our experience, 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. However, critical to the same is the need to implement modified protocols that integrate pre-emptive evaluation for COVID-19 disease and proactive monitoring of its relevant clinical parameters, thereby permitting judicious and timely implementation of catatonia-specific treatment options.

1. Introduction

Catatonia is a complex syndrome of specific psychomotor abnormalities occurring in the context of various medical and neuropsychiatric illnesses (Padhy et al., 2014). Untreated catatonia is associated with significant risks of mortality and morbidity, secondary to medical complications, such as malnutrition, infections, contractures, metabolic disturbances, and severe presentations such as malignant catatonia (Padhy et al., 2014).

Catatonia has been previously associated with viral influenza (encephalitis lethargica) (Scheiner et al., 2021). Similarly, new-onset catatonia with other psychiatric symptoms or worsening of previous psychiatric disorders with COVID (Coronavirus disease) – 19 infection has been reported (Torrico et al., 2021; Zandifar and Badrfam, 2021). Proposed mechanisms include inflammation, stress, and psychological factors (Scheiner et al., 2021). Catatonia, in particular, has been reported either as a primary manifestation, co-occurrence, or sequelae of COVID-19 (Amouri et al., 2021; Caan et al., 2020; Mulder et al., 2021) with unfavorable outcomes in most. For management of catatonia, irrespective of cause, parenteral benzodiazepines and/or Electroconvulsive therapy (ECT) are primary treatment options (Rasmussen et al., 2016). However, providing these interventions in the background of COVID-19 infection poses several challenges (Gouse et al., 2020). Benzodiazepines increase the risk of respiratory depression while general anesthesia for ECT increases the risk of aerosol-based spread, in addition to inherent risks in medically ill/unstable patients (Surve et al., 2021).

In this paper, we share our experience of five patients who were admitted to the Psychiatric Covid Unit (PCU) (Navin et al., 2021) at the National Institute of Mental Health and Neuro Sciences (NIMHANS) which is a tertiary care psychiatric hospital in Southern India. Our set-up was unique, wherein medical management for COVID-19 was provided concurrently with treatment for catatonia by the psychiatry team. Transfers to multi-specialty hospitals were made only when severe COVID-19 infection was noted or if progressive clinical deterioration occurred despite relevant treatment. Requisite medical care and evaluation were provided for all patients as per the Indian Council of Medical Research (ICMR) COVID management guidelines (Ministry of Health and Family Welfare India, 2021). These included monitoring of vitals 6th hourly, including oxygen saturation, respiratory parameters, and assessment of oral intake and clinical status by a nurse and on-duty medical officer (resident in Psychiatry). All patients received oral multivitamin and zinc tablets (100 mg per day). Oral Ivermectin up to 24 mg per day for 5 days/ Oral Doxycycline 200 mg per day for 5 days/oral Azithromycin 500 mg per day for 5 days either alone or in combination were given as per existing universal recommendations prevalent at the time, to prevent secondary infections. Baseline assessment of blood parameters (denoted as COVID workup henceforth) included hemogram, renal and liver function tests, serum electrolytes, D-dimer, Lactate Dehydrogenase, Serum Ferritin, and Serum C-reactive protein, repeated 3–5 days later or as clinically warranted, if continued inpatient care was deemed necessary. Additional clinical investigations were undertaken, based on the patient’s medical history or clinical findings.

A summary and comparison of clinical variables and investigations of the 5 cases are presented in Table 1 and Table 2, respectively.

Case 1. A 21-year-old single male, presented in October 2020 with a 2-month history characterized by mutism, withdrawal, staring, posturing, urinary incontinence, autonomic instability, immobility, rigidity, negativism, waxy flexibility, and postural instability with symptomatic worsening over the preceding 4 days. The Bush Francis Catatonia Rating
The COVID RT-PCR (Reverse Transcription Polymerase Chain Reaction) test was positive and hence admitted to PCU for evaluation and management of catatonia. COVID workup did not reveal any abnormality. His vitals were within normal limits throughout inpatient care (except for intermittent tachycardia-between 100 and 110/min). The patient was initiated on oral Lorazepam but switched to intravenous Lorazepam (6 mg daily) in three divided doses due to inadequate response (BFCRS =17). Bitemporal ECTs, administered thrice weekly were commenced further following non-response to lorazepam (BFCRS =9). A delayed response to ECT (improvement after the 10th ECT session) was observed, wherein followi wherein, following course of 13 ECTs, all catatonic symptoms resolved, except for rigidity (BFCRS =0). Lorazepam per oral (p.o.) 2 mg daily was continued due to worsening of catatonia on attempted cessation, and subsequently discontinued after a month. Serum and Cerebrospinal fluid (CSF) anti-N-methyl-D-aspartate (NMDA) and Voltage-Gated Potassium Channel (VGKC) antibodies were negative. CSF cytology and biochemistry were unremarkable. Fungal, Treponemal, and Cryptococcal infections were ruled out. Antinuclear Antibody (ANA), Antineutrophil cytoplasmic antibody (ANCA), Anti-Thyroid peroxidase (TPO) antibody, Paraneoplastic profiles were negative. Given persistent rigidity and gait instability, a neurology opinion was obtained and a diagnosis of atypical Parkinson’s disease was considered. Amantadine 50 mg and Selegiline 10 mg were initiated along with gait training. PLA2G6 homozygous gene mutation (R741Q) identified, suggested Early onset Parkinson’s disease (EOPD). At the time of discharge, there were no catatonic features and patient has shown improvement in motor symptoms with the above antiparkinsonian medications. However, gait instability persisted during follow-ups.

Case 2. A 26-year-old gentleman, with nicotine dependence and a history of psychotic depression in his mother, presented with a 10-year illness suggestive of Paranoid Schizophrenia, having failed trial of Risperidone. At admission in January 2021, he had stupor, mutism, staring, posturing, and grimacing suggestive of catatonia. The above developed over a one-month history preceding presentation to hospital. He was non-adherent to medications. There was no prior history of catatonia. BFCRS at presentation was 10. Whilst catatonic signs improved significantly with the lorazepam challenge (2 mg IV), inpatient care was planned for the management of residual catatonia and psychotic illness. RT-PCR for COVID-19 was positive, with no respiratory or systemic symptoms noted. Lorazepam p.o. (6 mg daily in divided doses) resulted in the resolution of catatonia over 2 days (BFCRS =0). Vitals remained within normal limits. Antipsychotic medications were reintiated (Amisulpride up to 600 mg and Injection Fluhenphazine Decanoate 25 mg fortnightly administered depot) with Lorazepam tapered to 4 mg and discharged with advice for home isolation. No side effects to treatment with above were noted. The patient did not follow up with our services subsequently, and hence further clinical details were unavailable.

Case 3. A 38-year-old woman presented in May 2021, with an abrupt onset illness of 4 days duration, with fearfulness, irrelevant talk, intermittent outbursts of laughter, elementary auditory hallucination, mutism, posturing, staring, withdrawal, immobility, and automatic obedience in the absence of any prior history of catatonia, or psychiatric illness. COVID exposure was present (immediate family member). The patient was diagnosed with Acute transient psychotic disorder with catatonia (baseline BFCRS score of 8).

RT-PCR test was positive. No respiratory or systemic abnormalities were noted. She was admitted to PCU and commenced on oral Olanzapine 10 mg daily, and Lorazepam 4 mg daily (divided doses). Metoprolol 25 mg daily was commenced following cardiology consultation for tachycardia and cardiac 2D-Echocardiogram was normal. Catatonia resolved over a week (BFCRS =0). Throughout inpatient care, vital parameters remained within normal limits, except for persistent tachycardia. COVID workup did not reveal any abnormality. Lorazepam was tapered and stopped and the patient discharged on Olanzapine 10 mg per day.

On follow-up, two months later, the patient did not have a recurrence of psychotic or catatonic symptoms. However, extrapyramidal signs were noted and Trihexyphenidyl 2 mg p.o. per day was initiated.

Case 4. A 24-year-old male presented with 6 years of episodic illness suggestive of schizoaffective disorder with a catatonic presentation in the previous two episodes and poor inter-episodic recovery. Valproate 1 gm per day and Quetiapine 50 mg per day advised previously was discontinued by the patient, one year before the current presentation.

He presented in March 2021 with 2 weeks of immobility, mutism, staring, posturing, grimacing, mannerism, negativism, withdrawal, impulsivity, and ambidexterity, and was diagnosed with Schizo-affective disorder with catatonia (BFCRS baseline score 18). Intravenous lorazepam (8 mg in divided doses) was associated with improvement in food intake. Subsequently, it was replaced with oral lorazepam (8 mg daily) and Aripiprazole p.o. titrated to 20 mg. As inadequate response to lorazepam was noted, Bifrontal (BF) ECTs were commenced. Following the first ECT, he was identified as COVID-19 positive. Over the subsequent 2 weeks, no upper respiratory signs were noted and vital parameters remained within normal limits. COVID workup at baseline and on repeat testing revealed no abnormality. Cross titration of Tab Risperidone (up to 8 mg) with Tab Aripiprazole was undertaken given persisting psychotic and mood symptoms. Valproate p.o. 750 mg per day was added as a mood stabilizer. Despite the same, residual mutism, staring, grimacing, and impulsivity persisted (BFCRS score 6) and ECTs were commenced. A course of 5 BF-ECTs contributed to improvement in catatonia and mood symptoms. At discharge (BFCRS score of 2), he was advised daily dosage of Valproate 750 mg, Risperidone 8 mg, Trihexyphenidyl 2 mg, and lorazepam 2 mg.

On follow-up after 2 weeks, he was readmitted for worsening catatonia with immobility, mutism, staring, grimacing, ambidexterity (BFCRS score of 7). Oral Lorazepam up to 6 mg and 9 BF ECTs were administered that improved both catatonic (BFCRS =0) and psychiatric symptoms. Other medications were continued and the patient was discharged for outpatient-based treatment.

Case 5. A 35-year-old lady, with hypothyroidism and schizoaffective disorder-depressive type (failed trials of Risperidone, Olanzapine, Amisulpride, Aripiprazole, Chlorpromazine, and multiple antidepres-
saint medications) of 15 years duration had adequate improvement with a previous trial of Clozapine 300 mg; however, it had to be stopped due to excess sedation, weight gain, and constipation. A combination of Quetiapine (800 mg) and Sertraline (150 mg) had benefitted to some extent in the past. She had received ECT (3 courses of 5–6 ECTs) in the past for suicidality. No history of catatonia was noted in the past. In the background of medication non-adherence, the patient presented in September 2020, with a 1-month duration of depressive symptoms and 4 days of stupor, negativism, mutism, posturing, staring, and withdrawal (BFCRS score was 18). In the emergency ward, catatonia responded to intravenous lorazepam (6 mg daily), however, negativism and withdrawal persisted for which inpatient care was planned. The RT-PCR test was positive, requiring admission to PCU. Sertraline (up to 200 mg) and Quetiapine (up to 600 mg) were initiated. A concurrent course of 7 Bitemporal ECTs (twice weekly with first 3 ECTs in the PCU with precautions) contributed to gradual improvement in catatonic (BFCRS - 0) and depressive symptoms. Parenteral lorazepam was switched to oral lorazepam 6 mg daily and gradually tapered and stopped over 2 weeks. No respiratory or systemic features of COVID-19 were noted. Vital parameters remained stable throughout the inpatient stay. COVID workup at baseline and on repeating on the 5th day did not reveal any abnormality. After completing treatment in the PCU, the patient was transferred to the inpatient psychiatry ward and subsequently discharged with significant improvement.
2. Discussion and conclusions

In this paper, we describe case summaries of five patients, who presented with catatonia and COVID-19 infection and underwent inpatient treatment in our PCU.

Of the five, one (Case 3) had new-onset catatonia; in the absence of any family or prior psychiatric history. COVID-19 infection is likely to have precipitated psychosis with catatonia, but a heightened sensitivity to atypical neuroleptics at follow-up suggests the additional possibility of persisting COVID-19 related neuropsychiatric sequelae. Another patient (Case 1) had an underlying genetic condition (EOPD) presenting with catatonia. Given that catatonia is not a known symptom of EOPD (Getz and Levin, 2017), the concurrent diagnosis of acute COVID-19 infection highlights how the interplay between COVID-19 and genetic susceptibility to neurological disorders may influence clinical presentations. Of the remaining three, one individual (Case 4) had a prior history of catatonia, while the other two (Cases 2 and 5) developed catatonia for the first time despite having a long-standing psychiatric illness. Gene-environment interactions influencing COVID-19 disease severity have been identified, and loci corresponding to lung,

### Table 1
Clinical characteristics of patients with COVID-19 and catatonia.

| Case   | Prior Psychiatric Diagnosis | Final Psychiatric diagnosis | Family history of catatonia/psychiatric illness | Prior history of catatonia | ILI symptoms | Covid Related metabolic derangement | Route of Administration of Lorazepam | Highest dose of Lorazepam given | Worsening of respiratory parameters and saturation with Lorazepam | Chest imaging | BFCRS (at baseline) | ECT administered | Adverse events during or after ECT |
|--------|----------------------------|-----------------------------|-----------------------------------------------|---------------------------|--------------|------------------------------------|-------------------------------------|-------------------------------|----------------------------------|----------------|-----------------|----------------|-------------------|
| 1      | Nil                        | Organic catatonia with Young-onset Parkinson’s disease | No                                             | No                        | No           | IV                                 | Oral                                | 6 mg                          | No                               | HRCT Chest- Few patchy areas of GGO in the superior segment of the left lower lobe (CO-RADS-6, CTSS-1/25) | 22                        | Yes             | None            |  |
| 2      | Schizophrenia              | Schizophrenia               | Yes (Psychotic depression in mother)           | No                        | No           | No                                 | Oral                                | 6 mg                          | No                               | Chest imaging not done           | 10                         | No              | None            |  |
| 3      | Nil                        | Schizophrenia               | No                                             | No                        | No           | Nil                                | Oral                                | 4 mg                          | No                               | HRCT chest - Peripheral patchy GGOs in bilateral lung fields (CO-RADS-6, CTSS-6/25) | 8 mg                        | Yes             | None            |  |
| 4      | Schizophrenia              | Acute and transient psychotic disorder with Catatonia | No                                             | Yes                       | No           | Nil                                | IV                                 | 8 mg                          | No                               | Chest X-ray- No abnormality detected | 18                         | Yes             | None            |  |
| 5      | Schizoaffective disorder   | Schizoaffective disorder   | No                                             | No                        | No           | Nil                                | IV                                 | 6 mg                          | No                               | Chest X-ray- No abnormality detected | 18                         | Yes             | None            |  |

ILI-Influenza like illness, CO-RADS: COVID-19 reporting and data system, CTSS- Chest CT severity score, GGO-Ground glass opacities, HRCT- High resolution Computed Tomography.

### Table 2
Blood investigations performed during inpatient care.

| Parameters                  | Case 1 | Case 2 | Case 3 | Case 4 | Case 5 |
|-----------------------------|--------|--------|--------|--------|--------|
| Hemogram                    | WNL    | WNL    | Elevated Total WBC count - 13, 900/uL*, Neutrophil-50.6%| Total WBC count – 5900/uL, Neutrophil -35.4%, Lymphocytes -49.1%* | WNL |
| Neutrophil-Lymphocyte Ratio | 2.54   | 3.22   | 1.19   | 0.72   | 2.09   |
| Renal function test         | WNL    | WNL    | WNL    | WNL    | WNL    |
| Liver function test         | WNL    | WNL    | WNL    | WNL    | WNL    |
| Serum Electrolytes          | WNL    | WNL    | WNL    | WNL    | WNL    |
| D-Dimer (<500ngFEU/ml)      | 355    | NA     | 263    | 354    | 150    |
| Serum Ferritin (10-150 ug/L) | 29     | NA     | 131    | 168*   | 25     |
| Serum C reactive protein (<5 mg/L) | 3     | NA     | 3      | 5      | NA     |
| Lactate Dehydrogenase       | 204    | NA     | 258*   | 245*   | 176    |
| (135–225 U/L)              | 279*   | NA     | 264*   | NA     | NA     |

(NA-not available, WNL- Within normal limits, * indicates abnormal investigation findings)
autoimmune, and inflammatory diseases were noted. This once again highlights how COVID-19 infection contributes to accelerating the development of, or altering the course (Quincozes-Santos et al., 2021) of pre-existing neuropsychiatric disorders. This might also explain the variable response to treatment for catatonia and psychiatric disorders seen in our patients.

Other than Case 2, all other patients were treated with antibiotics such as Doxycycline, Azithromycin, or Ivermectin in monotherapy/combination. Anti-inflammatory and immunomodulatory properties of these medications (Ali et al., 2021; Echeverría-Esnal and Grau, 2021) may have affected the course of catatonia, but we have inadequate evidence in this study to comment upon the same, and further studies are required to clarify this relationship. All patients were asymptomatic for the COVID-19 related systemic manifestations. However, whether this observation is incidental would be difficult to determine. The primary catatonia presentation may have led to an earlier presentation to the hospital thereby permitting timely treatment. Additionally, prior studies have noted that those with pre-existing psychiatric disease have lower severity of COVID-19 infection as well as lower mortality from same (Canal-Rivero et al., 2021; Drătciu and Boland, 2021; Moga et al., 2021).

Among our patients, the most common catatonic signs noted were posturing, immobility, mutism, staring, and withdrawal, similar to another study from our center examining catatonic syndromes (Subramaniam et al., 2019). Three individuals required ECTs because of inadequate response to Lorazepam. Higher scores on BFCRS were associated with the need for ECT and slower resolution of symptoms in this case series, as has been reported previously (Raveendranathan et al., 2012). While it may be argued that lorazepam doses administered were lower, adopting a cautious approach and initiating ECTs early can offset the potential risks of using high-dose benzodiazepines. Also, lorazepam is a safer choice among benzodiazepines in those with COVID-19 infection, because of the absence of liver metabolism and therefore a decreased risk of prolonged action or drug-drug interactions as occurs with other drugs of this class (Shah et al., 2021). ECT administration in the presence of COVID-19 infection requires a coordinated approach that can ensure safety of both, the patient and the treating team, details of which have been elaborated on in a previous publication from our center (Canal-Rivero et al., 2021; Drătciu and Boland, 2021; Moga et al., 2021).

3. Discussion

Patient confidentiality has been maintained. Additionally, exemption has been granted by the institutional ethics committee for preparing this case series using information available exclusively from clinical records stored at the hospital.

3. Disclosure

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Kasturi Atmaram Sakhardane, Harsh Pathak, Jayant Mahadevan, Krishna Prasad Muliyala, Sydney Moirangthem, Venkata Senthil Kumar Reddi 1
1 Correspondence to: Department of Psychiatry, National Institute of Mental Health and Neuro Sciences (NIMHANS), Bangalore, Karnataka, India
* Department of Psychiatry, National Institute of Mental Health and Neuro Sciences (NIMHANS), Bangalore, Karnataka, India
560029, India.
E-mail address: sreddi@nimbhans.ac.in (V.S.K. Reddi).