Severe Catatonia Presenting with Seizure, Autonomic Storm and Reversible Cardiomyopathy: A Diagnostic Challenge in the Intensive Care Unit

Richa Lohani[1*], Nitin Garg[2], Shobhit Garg[3]

[1]Department of Critical Care Medicine, Max Super Speciality Hospital, Dehradun, Uttarakhand
[2]Department of Neurology, Max Super Speciality Hospital, Dehradun, Uttarakhand
[3]Department of Psychiatry, Max Super Speciality Hospital, Dehradun, Uttarakhand

Correspondence:

Richa Lohani

E-mail: drricha.patersonkelly.lohani@gmail.com

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Abstract:

Catatonia is a complex neuropsychiatric syndrome that may present with a variety of motor, behavioural, and autonomic abnormalities. Severe catatonia as presented in this case may mimic organic, infectious or toxicological emergencies leading to delays in diagnosis. We describe a 37-year-old woman with major depressive disorder receiving multiple antidepressants who presented with profuse sweating, involuntary movements, and generalised stiffening. She developed a seizure with subsequent respiratory compromise requiring intubation. Initial evaluation revealed autonomic instability, severe left ventricular dysfunction, high anion gap metabolic acidosis, and bilateral pneumonitis on imaging. A comprehensive workup for infectious encephalitis, autoimmune disease, serotonin syndrome, toxic ingestion, and structural brain pathology was negative. Despite empirical antibiotics and steroids, her neurological status as assessed by the Glasgow Coma Scale (GCS), remained poor with no eye opening, or motor response, and an unassessable verbal response due to intubation (E1VtM1). After organic causes were excluded, a trial of lorazepam and olanzapine was initiated with gradual and sustained neurological improvement, confirming the diagnosis of catatonia. She was successfully extubated on Intensive Care Unit (ICU) Day 11 and discharged from the ICU in stable condition. Catatonia may present with autonomic dysregulation and critical illness, mimicking life-threatening medical conditions. Early recognition and a lorazepam challenge are essential to prevent unnecessary delays in treatment.

Key words: Catatonia, Autonomic Dysfunction, Lorazepam Challenge, Depression, ICU, Encephalopathy.

Introduction

The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines catatonia as the presence of three or more of the following — catalepsy, waxy flexibility, stupor, agitation, mutism, negativism, posturing, mannerisms, stereotypies, grimacing, echolalia, and echopraxia.[1] Catatonia in the Intensive Care Unit (ICU) is often misdiagnosed mainly because of the non-specific symptoms and resemblance with sepsis and other sepsis mimics, such as thyroid storm, as well as a lack of familiarity with the syndrome. The approach in such cases is often based on a search for an organic cause. There is a lack of literature on catatonia and its management in the ICU, with very few case series describing its occurrence in the ICU and the need for awareness among intensivists.[2,3]

Case Report

A 37-year-old woman with major depressive disorder receiving bupropion, escitalopram and cariprazine presented to the emergency department with profuse sweating, involuntary body movement and generalised stiffening for 30 minutes. She had been in a low mood for two days prior to presentation.

On examination, she had sinus tachycardia with a pulse rate of 150 beats per minute (bpm), hypertension (blood pressure [BP] 190/100 mmHg), tachypnoea, and diaphoresis. Systemic examination revealed bilateral crepitations in the chest, laboured breathing, and oxygen saturation of 90% on room air. The patient developed a seizure with a threatened airway. Antiepileptics were administered, and she was intubated in the emergency department in view of a poor Glasgow Coma Scale (GCS) score and impending airway compromise. Initial arterial blood gases showed high anion gap metabolic acidosis with a lactate level of 15 mmol/L, likely secondary to ongoing seizure activity.

Her past history was significant for infertility treatment for six to seven years, and a history of laparoscopic cholecystectomy one month prior. She had also been experiencing a low mood for the preceding two days. There was a history of antidepressant dose modification 15 days prior to presentation. She had been prescribed bupropion, escitalopram and cariprazine as prescribed by her psychiatrist.

Initial differentials included unknown drug ingestion, serotonin syndrome, encephalitis or meningitis, and stroke. The patient was shifted to the ICU for further management. On arrival at the ICU, she developed hypotension. Point-of-care ultrasound (POCUS) revealed severe left ventricular (LV) dysfunction and a plethoric inferior vena cava (IVC). Vasopressors were initiated, and invasive positive pressure ventilation (IPPV) was continued with fentanyl sedation. She was started on empirical antibiotics. A comprehensive panel of tests and imaging studies was planned to rule out the differential diagnoses. A psychiatry consultation was obtained, and all the antidepressants were withheld.

Initial investigations showed a total leukocyte count of 27,000/µL with neutrophilic predominance, and normal liver and kidney function tests. Magnetic resonance imaging (MRI) of the brain with contrast was normal, with no focal parenchymal or intracranial abnormalities. Lumbar puncture was performed, which was normal, and the cerebrospinal fluid (CSF) encephalitis panel was negative. A tropical fever workup was negative. A urine drug assay to rule out poisoning or overdose was normal. Creatine kinase was 787 IU/L, making serotonin syndrome less likely.

High-resolution computed tomography (HRCT) of the thorax showed patchy and confluent areas of ground-glass opacities and consolidative changes in both upper and lower lobes. Based on these findings, antibiotic coverage was broadened to include atypical organisms. Mild elevation of cardiac biomarkers was noted, and two-dimensional (2D) echocardiography revealed poor LV function with global hypokinesia and Grade II diastolic dysfunction. Digoxin was added in view of tachycardia and poor LV function, and a diuretic infusion was started.

Ultrasound of the abdomen was performed to identify an alternative source of sepsis and showed Grade II fatty liver. Blood, tracheal, and urine cultures were negative. Serum ammonia levels and thyroid profile were within normal limits. The Venereal Disease Research Laboratory (VDRL) test was negative.

On ICU Day 2, the patient's haemodynamics improved. Autonomic dysfunction was noted in the form of pinpoint pupils, sweating, and labile blood pressure. However, in view of a persistently poor GCS, electroencephalography (EEG) was performed and was normal. Bronchoscopy was done, and bronchoalveolar lavage (BAL) samples were obtained, which were sterile.

The patient was empirically started on pulse steroid therapy, as there had been no improvement in GCS over the preceding 72 hours. However, she remained unconscious and unresponsive for the next three days despite steroids. The anti-N-methyl-D-aspartate (NMDA) receptor antibody panel was negative.

Since all organic and metabolic causes had been ruled out, the patient was started on intravenous lorazepam, amantadine, and olanzapine, considering the possibility of catatonia.

During the following days, the patient remained unresponsive with a GCS of E1VtM1 no eye opening, an unassessable verbal response due to intubation, and no motor response while receiving mechanical ventilation

and supportive ICU care. Repeat echocardiography showed improvement in LV function.

On ICU Day 7, the patient developed left lung collapse associated with hypoxaemia. Bronchoscopy and lavage were performed and showed growth of *Acinetobacter baumannii*, for which minocycline was added. A plan for tracheostomy was discussed with family members in view of persistently poor GCS and ventilator-associated pneumonia.

However, over the next 48 hours, her GCS improved. On ICU Day 10, the patient underwent a spontaneous breathing trial, which she tolerated well. Extubation was deferred for a further 24 hours due to upper airway oedema. A short course of dexamethasone was administered, and the patient was successfully extubated on ICU Day 11. She required non-invasive ventilation (NIV) support for the next 48 hours due to basal atelectasis and obesity. Lorazepam and olanzapine were gradually tapered. As she showed neurological recovery with lorazepam and olanzapine therapy, the diagnosis of catatonia was confirmed.

After a successful swallowing assessment, she was started on oral feeding. The patient was shifted to the ward in stable condition after four days.

Discussion

In mechanically ventilated patients, the application of diagnostic tools such as the Bush–Francis Catatonia Rating Scale is often not feasible, which may hinder timely recognition of catatonia. In this case, the patient presented primarily with non-specific features of autonomic dysfunction, leading to a diagnostic delay and a subsequent delay in initiating targeted therapy.[4] After ruling out the common causes of altered mental state, including serotonin syndrome, the diagnosis of catatonia was considered and was treated accordingly. This highlights the importance of considering catatonia as a potential differential diagnosis in critically ill patients who present with clinical features resembling sepsis, especially when the clinical course is atypical or unresponsive to standard management.

Conclusion

This case illustrates the diagnostic challenge of severe catatonia presenting with autonomic instability, seizures, and reversible cardiomyopathy in the intensive care setting. The overlap with infectious and metabolic emergencies can delay diagnosis and treatment. A high index of suspicion, early psychiatric involvement, and a timely lorazepam challenge are crucial for accurate diagnosis and effective management, potentially reducing morbidity and ICU stay.

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References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th Edition. Arlington, VA: American Psychiatric Association; 2013. p. 119–21.
2. Saddawi-Konefka D, Berg SM, Nejad SH, *et al.* Catatonia in the ICU: an important and underdiagnosed cause of altered mental status. A case series and review of the literature. *Crit Care Med.* 2014;42(3):e234–e241.
3. Rizos DV, Peritogiannis V, Gkogkos C. Catatonia in the intensive care unit. *Gen Hosp Psychiatry.* 2011;33(1):e1–e2.
4. Gupta R, Saigal S, Joshi R, *et al.* Unrecognized catatonia as a cause for delayed weaning in Intensive Care Unit. *Indian J Crit Care Med.* 2015;19(11):693–4.