Case Report

Unrecognized catatonia as a cause for delayed weaning in Intensive Care Unit

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Abstract

The cause of altered sensorium in critical care settings includes metabolic derangements, drug and toxin overdose, central nervous system infections, neurodegenerative disorders, vascular events, hypo-perfusion states, and septic encephalopathy. Here, we present a case of an elderly woman who presented to us with altered sensorium with respiratory failure requiring mechanical ventilation. Her metabolic parameters, imaging, and cerebrospinal fluid study were all normal despite that she continued to remain in altered sensorium and had an unrecognized behavioral state that delayed her weaning.

Keywords: Altered sensorium, catatonia, Intensive Care Unit, weaning

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Introduction

Altered behavior is a common occurrence in critically ill patients, and has diverse etiologies. The situations are further complicated in elderly, those with underlying co-morbidities and in individuals on mechanical ventilation. We describe an elderly woman, who developed sepsis and respiratory failure, required mechanical ventilation, and had an unrecognized behavioral state that delayed her weaning.

Case Report

A 68-year-old woman with previous hypothyroidism and bipolar disorder was on multiple medications (sodium valproate, clozapine, risperidone, and trihexyphenidyl) for two decades. A day prior to admission, she became drowsy, without any limb weakness. She was hypothermic, had pulse of 50/min, blood pressure of 90/60 mmHg, respiratory rates of 24/min, and her bilateral plantars were mute. She had crepitations over right lower lung fields. On laboratory evaluation, she had pancytopenia and normal renal and liver function tests. Chest radiograph revealed right lower and middle lobe consolidation. She was started on crystalloids, antibiotics, and hydrocortisone for sepsis due to right lower zone pneumonia. Her previous drugs were withheld given the possibility of drug-induced pancytopenia due to either clozapine or valproate. Her thyroid functions were done, and her thyroid-stimulating hormone was 22.86 mIU/L, with normal FT4 and FT3 levels. Her anti-thyroid peroxidase antibodies were negative. Her thyroxine dose was escalated to 100 mcg/day. On 3rd day, her respiratory distress worsened and became more drowsy. She was intubated and initiated on mechanical ventilation, and vasopressors were started. By day 7, sedatives were tapered, her infection was brought under control, and vasopressors were stopped. However, she remained lethargic, and had poor respiratory efforts, for which percutaneous tracheostomy was performed on D10. Magnetic resonance imaging brain, cerebrospinal

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fluid examination, total creatine kinase levels, and electroencephalography were normal. She persisted to be lethargic, with a fixed gaze, limited blinking, fixed flexor posturing, and muscle tone was increased in all limbs. A psychiatry opinion was taken, and provisional diagnosis of catatonia was made. Lorazepam test was performed, the rigidity of her limbs improved. She was initiated on intravenous (IV) lorazepam, i.e. 2 mg IV q8h. Over next 5 days, she became fully conscious. By day 24, she could be weaned off ventilator. As her neuromuscular power improved, tracheostomy tube was removed and stoma was strapped. The patient was discharged after 35 days of hospital stay, and is doing well 6 months later.

Discussion

Catatonia is a neuropsychiatric syndrome of altered mental status and characteristic psychomotor findings. Clinically, catatonia is characterized by excited or withdrawn features, though patients manifest some of both types during the course of their illness.[1] Its exact prevalence within the Intensive Care Unit (ICU) is unknown, with one report on a small number of patients suggesting it may affect up to 4% of critically ill patients.[2]

In our case, there were various risk factors for catatonia such as old age, affective disorder, withdrawal of antipsychotics, and chronic medical illness. Other likely differentials in this case with altered sensorium can be metabolic disorders, i.e., myxedema coma, hepatic encephalopathy due to valproate, central nervous system infections, neurodegenerative disorders, persistent vegetative states, serotonin syndrome, autoimmune encephalopathy, septic encephalopathy, or seizures. Negative tests and positive response to benzodiazepine makes catatonia most likely etiology in this case.

The underlying pathophysiologic mechanism for catatonia is likely heterogeneous. It has been proposed that basal ganglia thalamo-cortical circuit dysregulation with resulting changes in neurotransmitter function is a potential precipitant factor.[3] Catatonic patients have lower gamma-aminobutyric acid A (GABA A) activity in the orbito-frontal cortex than do healthy and psychiatric control patients.[4] Benzodiazepines, which are GABA A agonists, are first-line therapy for catatonia.[5]

The modified Bush-Francis Catatonia Rating Scale (BFCRS) is a 23-item rating scale that provides a standardized schema for clinical examination.[6] We used an algorithmic approach to the diagnosis and management of catatonia in the ICU as proposed by Saddawi-Konefka.[7] A trial of 1–2 mg of IV lorazepam can produce immediate, impressive reversal of catatonic features. This is known as the "lorazepam test" and is associated with reduced catatonia scores on the BFCRS by 60% within 10 min and response rates of 60–80% within hours or days.[8]

Despite the lack of prospective randomized evidence, benzodiazepines are the gold standard in the treatment of catatonia.[9] IV lorazepam is commonly used, starting with doses of 2 mg daily, and titrating occasionally to total doses of 20–30 mg daily. Other treatment options include amantadine, zolpidem, memantine, valproate, and phenobarbital.[9-11] Electroconvulsive therapy, the most successful treatment for catatonia, is usually reserved for patients with benzodiazepine-resistant catatonia.[12]

Conclusion

Timely recognition and treatment of catatonia in the critically ill patients with altered mental status is essential, given the substantial morbidity and mortality associated with the condition.

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Conflicts of interest

There are no conflicts of interest.

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