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Resolution Of Treatment Resistant Catatonia: A Case Report

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Abstract

This is the case of a 25-year-old female past psychiatric history of Schizoaffective Disorder Bipolar Type, several psychiatric hospitalizations for psychiatric decompensation secondary to medication non-adherence, history of polysubstance abuse, history of physical and sexual trauma, past medical history significant for Hepatitis C, who presented to the emergency department with alcohol intoxication and features of catatonia. She exhibited echolalia, posturing, mutism, alogia, and stereotyped repetitive movements. She also displayed marked disorganization and purposeless hand movements along with intermittent episodes of agitation and aggression with expression of nonsensical phrases.

Introduction

This case illustrates the importance of identifying and treating Catatonia, as well as utilizing the different treatment resistant options available when faced with a Catatonia that proves resistant to first line treatment options such as benzodiazepines, to ensure that patients are being treated with the highest quality of care **[1, 2, 3, 4, 5]**.

Case Presentation

This patient was brought to the Emergency Department due to alcohol intoxication after being found wandering in the streets. Psychiatric evaluation in the emergency department revealed evidence consistent with a catatonic state exhibited by echolalia, posturing, mutism, alogia, and stereotyped repetitive movements. She demonstrated marked disorganization such as bizarrely walking around in circles, purposeless hand movements and episodes of agitation with expression of non-sensical phrases. She was markedly confused and could not elucidate situational details. She was not oriented to time, claiming the year was 2019 without knowing the month or day. She knew her name but geographically could only say she "was in a hospital". Vital signs in the emergency department were BP of 119/71 mm Hg, temperature of 98 F, and respiratory rate of 16 per minute. Pulse was slightly elevated at 110 per minute which decreased into the normal range within a few hours. A urine drug screen was negative for common substances tested (marijuana, cocaine, PCP, benzodiazepines, opioids) and blood alcohol was below 10.1 units**.** CT head exam without contrast was conducted which was negative for any acute abnormalities. Intermittently, she was both verbally and physically abusive, which required intermittent administered doses of Lorazepam 2mg intramuscular and Haloperidol 5mg intramuscular.

Once patient was medically cleared from the emergency department, she was transferred to the inpatient adult psychiatric unit and started on 2mg of Lorazepam PO TID, Benztropine 1mg PO BID due to EPS symptoms from intramuscular medications administered in the emergency department, Haloperidol 5mg PO BID and Trazodone 100mg PO BID. Haloperidol was substituted with Quetiapine once the patient was less agitated, which was subsequently titrated to 350mg PO HS and Depakote was added, which was eventually increased to 500mg PO BID.

Despite 16 days of Lorazepam 2mg PO TID, the patient's catatonic symptoms still did not resolve. The patient continued to exhibit resistant symptoms such as echolalia, posturing, stereotyped repetitive movements, poverty of speech with increased latency of response, thought blocking, despite having had some improvement in the intermittent episodes of mutism, alogia, and intermittent episodes of unprovoked agitation. In view of this poor response, decision was made to start Memantine 10mg oral once daily for treatment resistant Catatonia.

After one day of Memantine administration, the patient's Catatonia significantly improved. She was able to verbally converse appropriately, take care of her activities of daily living (showering, brushing her teeth, feeding herself) and was able to express her thoughts and concerns with increased frequency and volume of communication. She was no longer agitated, aggressive, or combative. She still maintained a degree of disorganization and poor insight and judgment into her schizoaffective disorder, but over the rest of her stay in the hospital, a gradual reduction of the more marked symptoms of her Catatonia continued, as evidenced by decreased and resolution

of her posturing, echolalia, and stereotyped hand and physical movements. The patient's organization improved enough for her to express concerns of having untreated Hepatitis C, which the patient was evaluated and initiated on treatment for as per recommendations from the infectious disease consulting specialist. After 27 days of inpatient psychiatric hospitalization, the patient was eventually discharged to a long-term state psychiatric facility for further evaluation and plan of care management. The patients' medications on discharge were Memantine 10mg PO once daily, Lorazepam 2mg PO once daily, Quetiapine 350mg PO once nightly Depakote 500mg PO BID, Atenolol 25mg PO once daily and Trazadone 200mg PO once nightly.

Discussion

Catatonia is a complex neuropsychiatric syndrome characterized by a cluster of psychomotor disturbances including immobility, mutism, staring, rigidity, negativism, and posturing **[1]**. It can occur in the context of various psychiatric, neurological, and general medical illnesses, most commonly mood disorders and schizophrenia **[1]**. Early identification is essential due to the high risk of medical complications such as malnutrition, dehydration, deep vein thrombosis, and, in some cases, death **[1]**. The Bush-Francis Catatonia Rating Scale (BFCRS) remains a valuable tool for assessing the severity of catatonia and guiding treatment response **[1]**.

The mainstay of initial treatment involves the administration of Benzodiazepines, particularly Lorazepam, which serves both diagnostic and therapeutic purposes **[1]**. Most cases of acute catatonia respond to low-dose lorazepam (1–2 mg), often with striking improvement within hours **[1]**. If catatonic symptoms persist despite adequate trials of lorazepam, electroconvulsive therapy (ECT) is considered the next-line and highly effective intervention **[2]**. However, a subset of patients— particularly those with chronic catatonia or schizophrenia—may exhibit resistance to both lorazepam and ECT, a clinical presentation termed treatment-resistant catatonia (TRC) **[2, 3, 4, 5]**.

Recognition of TRC is critical, as continued immobility or mutism without effective treatment significantly increases morbidity **[1, 2, 3, 4]**. Evidence for managing TRC is limited but growing, based largely on case reports and small case series. Emerging treatments target glutamatergic hyperactivity, a hypothesized mechanism underlying TRC, especially in the context of GABAergic and dopaminergic hypoactivity **[5]**.

Several N-methyl-D-aspartate (NMDA) receptor antagonists, such as Amantadine, Memantine, and Ketamine, have shown promising results in this population **[5]**. Case reports have documented resolution of catatonic symptoms with memantine in lorazepam- and ECT-resistant cases of schizophrenia, suggesting its role as a viable adjunctive therapy **[2, 3, 4, 5]**. Similarly, oral and intranasal ketamine, including sublingual ketamine and Esketamine nasal spray, have been used successfully in TRC, particularly in cases with co-occurring treatment resistant depression or schizoaffective disorder. These agents may offer faster onset of action and broader accessibility in outpatient settings compared to ECT **[2, 3, 4, 5]**.

In the reviewed literature, augmentation strategies including Lorazepam combined with ECT, cautious use of antipsychotics (particularly those with partial D2 agonism like aripiprazole or lumateperone), and NMDA antagonists were employed when standard approaches failed **[5]**. The literature also emphasizes caution with traditional antipsychotics in the acute catatonic phase due to the risk of precipitating neuroleptic malignant syndrome (NMS), particularly in cases with elevated creatine kinase or autonomic instability **[1, 2, 3, 4, 5]**.

Recent case reports illustrate that maintenance therapy with Ketamine/Esketamine may help prevent catatonia relapse, offering a novel approach for patients with recurrent or chronic catatonia **[3]**. While Memantine and Amantadine have longer half-lives and are generally well tolerated, Memantine may pose a lower risk of inducing psychosis than Amantadine, making it preferable in psychosis-prone populations **[5]**.

The integration of glutamatergic-targeting agents into the treatment algorithm for catatonia, particularly in refractory cases, represents a meaningful shift in management strategy. Nonetheless, more systematic studies are needed to determine efficacy, optimal dosing, and long-term outcomes for these agents.

Conclusion

In conclusion, timely diagnosis and initiation of first-line therapy for catatonia are crucial. In cases where Benzodiazepines and ECT fail, clinicians should recognize treatment-resistant catatonia and consider NMDA antagonists such as Memantine, Amantadine, or Ketamine as adjunctive or alternative therapies **[1, 2, 3, 4, 5]**. Awareness of these emerging options is essential to improve outcomes and reduce the substantial morbidity associated with this often-underdiagnosed syndrome **[1, 2, 3, 4, 5]**.

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