An Overlapping Presentation of Hypoglycemia and Catatonia—A Case Report and Literature Review

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Abstract: Catatonia is a clinical syndrome characterized by a distinct constellation of psychomotor disturbances. It is known as a clinical manifestation of many medical and psychiatric conditions. Neuroglycopenia is a term that refers to a shortage of glucose in the brain resulting in the alteration of neuronal function. Catatonia has been observed in hypoglycemic states. We present a single case report of a 36-year-old male, with no known medical or psychiatric history, presenting with catatonia and hypoglycemia due to malnutrition. Catatonia and hypoglycemia may present similarly, and can present a challenge in differentiating the underlying etiology. It is unclear whether the hypoglycemia-catatonia overlap phenomenon is rare or rather underdiagnosed.

Keywords: catatonia; hypoglycemia; neuroglycopenia; inanition; med-psych; combined medicine

1. Introduction

Catatonia is a clinical syndrome characterized by a distinct constellation of psychomotor disturbances. The DSM-5 criteria include the presence of at least three of the following symptoms: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypy, agitation, grimacing, echolalia, and echopraxia [1]. Catatonia is recognized as a clinical manifestation of multiple psychiatric, neurologic, and medical conditions [2]. The exact mechanism for catatonia is not fully understood, but cingulate-basal ganglia-thalamocortical pathways dysfunction, as well as dysfunctional GABA, glutamate, serotonin, and dopamine transmissions, are implicated in the neuro-pathophysiology of catatonia [3,4]. The Bush-Francis Catatonia Rating Scale is commonly used for screening and severity rating [5].

The concurrent or underlying psychiatric diagnoses in catatonic patients include affective disorder (46%), schizophrenia (20%), schizoaffective disorder (16%), benzodiazepine withdrawal (4%), and other mental health disorders (8%). Six percent of patients presenting with catatonia suffer from a range of other medical and neurological illnesses [6].

Neuroglycopenia is a term that refers to a shortage of glucose in the central nervous system (CNS) resulting in an alteration of neuronal function. Neuroglycopenic symptoms include fatigue, change in mental status, confusion, speech impairment, seizures, and coma. During the early stage of neuroglycopenia, symptoms are subtle and may become evident only through systematic cognitive testing [7]. Catatonia has been observed in hypoglycemic states. In our literature review; using the keywords “catatonia,” “hypoglycemia” or “low blood sugar”, we found only one case report looking at the association between neuroglycopenic symptoms and catatonia [8].

Internal medicine physicians infrequently encounter patients presenting in catatonic states [9]. However, the majority of catatonic patients in general hospitals are improperly...
diagnosed on admission, due to factors such as a lack of awareness and the heterogeneous presentation of catatonia [10]. When such patients present to healthcare systems, they are typically referred to a psychiatry consultation service potentially delaying further workup. This delay in the proper evaluation and treatment of the underlying etiology may lead to increased mortality [11].

Experts agree that any case of a new-onset psychiatric illness should be evaluated for possible organic etiology, as it may be fully reversible with the correction of the underlying cause [12]. Hypoglycemia may be seen as a complication of a prolonged catatonia state, but it may in turn contribute to the presence of catatonic symptoms. The aim of the present study is to describe a rare manifestation of mixed major metabolic and psychiatric pathologies and to emphasize the importance of a comprehensive interdisciplinary clinical approach.

2. Case Report

Our patient is a 36-year-old undomiciled male, with no known medical or psychiatric history, who was found unresponsive on the floor in a shelter and brought in by ambulance to the emergency room. According to shelter staff, he had been observed acting strangely for several months before admission. He had reduced his food intake, would stare inappropriately, and made efforts to avoid face-to-face communication with others. No history of substance use was reported. The primary care doctor had previously referred the patient for psychiatric assessment, but he had not complied.

A medical workup in the emergency room revealed vital signs within the normal range (blood pressure 117/87 mmHg, temperature 36 °C, heart rate 85 beats per minute, oxygen saturation 99%). The patient was found to be cachectic and malnourished. Hyporeflexia and low muscle tone were evident in all limbs. Laboratory examinations revealed that urine toxicology and blood work were negative for drugs and ethanol. Hemoglobin was 16.3 g%; blood sugar was 45 mg/dL sodium was 149 mmol/L, potassium was 3.39 mmol/L, and blood-urea/Cr ratio of 21.6. Liver and pancreatic enzymes were within the normal range (ALT 8 U/L, AST 16 U/L, amylase 66 U/L). C-reactive protein was 0.7 mg/dL, blood pH was 7.4, protein total was 6.3 g/dL, and albumin was 3.2 g/dL. Intravenous (IV) diazepam 40mg was administered with poor response. The patient’s blood sugar and hypokalemia were corrected with the administration of IV glucose and potassium chloride. The patient’s blood sugar improved to a normal range, but serum levels of protein and albumin, as well as muscle strength, remained low.

Head computed tomography (CT) on admission followed by brain magnetic resonance imaging (MRI) and electroencephalography (EEG) later were unremarkable. The patient was alert, but withdrawn, unresponsive to commands, and exhibited stupor, negativism, mutism, and severe psychomotor retardation, and resisted during the examination. After the correction of glucose levels, he displayed posturing and waxy flexibility. Pressure ulcers (stage I) were found on his sacrum and heels. A diagnosis of catatonia was entertained. The patient was administered a total daily dose of IV diazepam 40 mg in place of the preferred IV lorazepam due to a drug shortage. No improvement was observed. A nasogastric tube was inserted and daily olanzapine 20 mg was administered. Nutritional support along with thiamine, vitamin C, vitamin B12, folic acid, and vitamin D supplementation was provided. Serial laboratory monitoring was performed due to the risk of refeeding syndrome. The patient was treated with low molecular weight heparin for deep venous thrombosis (DVT) prophylaxis. Attempts to provide the patient with physical therapy failed due to his catatonic behavior. After one week of treatment, his catatonia symptoms decreased but did not vanish. He was treated with a course of electroconvulsive (ECT) therapy with gradual improvement in his mental, physical, and nutritional status. The patient was then diagnosed with schizophrenia, catatonic type, after a detailed history from the patient and his siblings had revealed a pattern of progressively disorganized behavior, disordered thought and speech, and abnormal affect over seven months prior to admission.
3. Discussion

This case demonstrates the diagnostic challenge posed by the overlapping symptoms of hypoglycemia and catatonia. (See Table 1) Catatonia may result in prolonged starvation and a resultant hypoglycemic state. In turn, hypoglycemia may aggravate, overlap, or obscure catatonic symptoms. While decreased oral intake is typically compensated for by gluconeogenesis, it can cause hypoglycemia in the setting of malnourishment [13]. Hypoglycemia may present as a potentially life-threatening central nervous system dysfunction that may mimic catatonia [14]. Ghosh reported catatonia in the presence of low blood glucose levels and highlighted the relationship between hypoglycemia and catatonia, referring to it as a neuropsychiatric manifestation of hypoglycemia [8].

Table 1. Clinical manifestations of hypoglycemia and catatonia.

<table>
<thead>
<tr>
<th>Clinical Manifestation</th>
<th>Hypoglycemia</th>
<th>Catatonia</th>
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<tbody>
<tr>
<td>Cognitive impairment</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Immobility</td>
<td>+</td>
<td></td>
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<tr>
<td>Inability to Communicate</td>
<td>+</td>
<td></td>
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<tr>
<td>Unusual/Repetitive movements</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>Echolalia/Echopraxia</td>
<td>−</td>
<td>+</td>
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<tr>
<td>Autonomic instability</td>
<td>+</td>
<td></td>
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<tr>
<td>Refusal to eat</td>
<td>−</td>
<td>+</td>
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<tr>
<td>Seizure</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Sweating</td>
<td>+</td>
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</tbody>
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As previously mentioned, catatonia is frequently misdiagnosed or missed. It is imperative to correct hypoglycemia in the presence of catatonia and to rule out other possible etiologies of neuropsychiatric dysfunction, such as drug intoxication, infection, or hepatic failure [15]. Simultaneously, one must consider that hypoglycemia may be the result of prolonged catatonia leading to malnutrition and starvation, as was seen in our patient.

In our case, prompt administration of glucose did not reverse the catatonic state. The emergency room clinicians’ working diagnosis of unresponsiveness due to hypoglycemia delayed the prompt treatment of catatonia. However, it is possible that hypoglycemia had contributed to the prolonged catatonic state. The more classic symptoms of catatonia including mutism, awkward posturing, and waxy flexibility unfolded after IV glucose administration. It is possible that the correction of hypoglycemia may improve the response success of catatonia to treatment with benzodiazepines. We postulate that treating the patient with benzodiazepines alone was not sufficient until the underlying hypoglycemic state was corrected. [6]. Several medical, neurological, and psychiatric conditions can induce catatonia [16], making the etiological diagnosis challenging, particularly when the patient cannot cooperate with the interview. Catatonia is associated with psychiatric and medical pathology and leads to multiple complications [17].

Notably, the patient did not present with the autonomic alterations expected with severe hypoglycemia (e.g., tachycardia, tremor, sweating) [18]. This may be due to “hypoglycemia unawareness,” a state in which the autonomic warning symptoms may not be perceived due to prolonged hypoglycemia [19].

4. Summary

Failure to recognize and treat hypoglycemia may lead to sudden death, mediated by Cardiac arrhythmias and seizures [20,21]. We, therefore, suggest checking glucose levels and correcting hypoglycemia prior to the diagnosis and treatment of catatonia. Ideally, an interdisciplinary team approach should be utilized, including a psychiatry consultation and liaison service, internal medicine, and/or neurology specialists.

The true incidence of the hypoglycemia-catatonia overlap phenomenon is unknown. Further research is warranted to clarify the neurophysiological pathways of these overlap-
ping conditions and to confirm our hypothesis that treating the patient with glucose may improve the response to catatonia treatment.

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References