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Delirium Secondary to Diabetic Ketoacidosis Initially Misdiagnosed as Benzodiazepine Withdrawal: A Case Report

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Abstract

Background: Delirium is an acute neuropsychiatric syndrome characterized by disturbances in attention, awareness, and cognition with a fluctuating course, most often secondary to an underlying medical condition. Diabetic ketoacidosis (DKA) is a metabolic emergency that may present with prominent neuropsychiatric manifestations, sometimes mimicking primary psychiatric disorders or substance withdrawal syndromes, leading to diagnostic error. **Case Presentation:** We report the case of a 58-year-old woman with a 12-year history of type 2 diabetes mellitus who presented with acute psychomotor agitation, insomnia, and incoherent speech evolving over three days. The clinical context included abrupt discontinuation of lorazepam (2 mg/day for approximately 18 months), last taken approximately 72 hours prior to admission, without prior documented withdrawal episode or known dependence features. Initial evaluation led to suspicion of benzodiazepine withdrawal delirium. However, physical examination revealed dehydration, tachypnea, and systemic stress signs. Laboratory investigations showed hyperglycemia (2.55 g/L), ketonuria (3+), glycosuria, HbA1c of 12.1%, leukocytosis, and elevated C-reactive protein. Serum electrolytes and renal function were not fully available at presentation. Arterial blood gas analysis, serum bicarbonate, anion gap calculation, and serum β-hydroxybutyrate were also unavailable. Despite these limitations, a working diagnosis of DKA was retained based on clinical and urinary criteria. Treatment with intravenous fluids and insulin led to rapid improvement within 48 hours and full cognitive recovery by day four. **Conclusion:** This case highlights a diagnostic pitfall where metabolic delirium mimics benzodiazepine withdrawal. It underscores the necessity of systematic somatic evaluation in all acute confusional states, particularly in resource-limited settings, to avoid delayed diagnosis and prevent complications.

Keywords

Delirium, Diabetic Ketoacidosis, Benzodiazepine Withdrawal, Acute Confusional State, Diagnostic Error

1. Introduction

Delirium is a common and serious neuropsychiatric syndrome characterized by an acute onset, fluctuating course, inattention, and global cognitive dysfunction. It is associated with increased morbidity, mortality, prolonged hospitalization, and long-term cognitive decline, particularly in older adults and patients with chronic medical conditions [1] [2].

Diabetic ketoacidosis (DKA) is an acute and potentially life-threatening complication of diabetes mellitus, defined by hyperglycemia, ketosis, and metabolic acidosis. Neurological manifestations such as confusion, agitation, and decreased level of consciousness are frequently observed in severe cases and are mediated by osmotic shifts, dehydration, electrolyte disturbances, and inflammatory pathways [3] [4].

Benzodiazepine withdrawal is also a recognized cause of delirium, particularly after abrupt cessation in patients receiving long-term therapy. Clinical manifestations may include agitation, tremor, autonomic instability, anxiety, and confusion, creating significant diagnostic overlap with metabolic causes of delirium [5].

This overlap may result in anchoring bias, particularly in psychiatric settings where substance-related etiologies are initially prioritized. We report a case illustrating this diagnostic challenge, where DKA-related delirium was initially misdiagnosed as benzodiazepine withdrawal.

2. Case Presentation

A 58-year-old woman with a 12-year history of type 2 diabetes mellitus was admitted for acute behavioral disturbance characterized by psychomotor agitation, insomnia, and incoherent speech evolving over three days.

Collateral history revealed abrupt discontinuation of lorazepam, prescribed at 2 mg/day for approximately 18 months for anxiety symptoms. There was no documented history of dose escalation, misuse, or prior withdrawal episode. The last dose was taken approximately 72 hours before admission.

2.1. Psychiatric Examination

On admission, the patient exhibited marked disorientation in time, place, and situation. She was unable to correctly identify the date or location and demonstrated impaired recognition of family members, suggesting significant perceptual and cognitive dysfunction. Immediate memory was severely impaired, with inability to retain simple information beyond short intervals.

Speech was incoherent, with frequent derailment, tangentiality, and incomplete sentences. Psychomotor behavior fluctuated between agitation and periods of re-

duced motor activity. Attention was globally impaired, with inability to maintain focus during clinical interaction. Based on this presentation, a provisional diagnosis of benzodiazepine withdrawal delirium was initially considered.

2.2. Physical Examination

Vital signs revealed tachycardia at 110 beats per minute and tachypnea at 28 breaths per minute. Blood pressure was 140/85 mmHg and temperature was 37.9˚C. Clinical examination showed clear signs of dehydration, including dry mucous membranes and decreased skin turgor.

Neurological assessment revealed fluctuating consciousness with preserved arousability. No focal neurological deficit was identified. The presence of tachypnea and dehydration raised early suspicion of metabolic decompensation.

2.3. Laboratory Findings

Laboratory evaluation showed marked hyperglycemia at 2.55 g/L and significant ketonuria (3+) with glycosuria. HbA1c was elevated at 12.1%, indicating chronic poor glycemic control. Leukocytosis and elevated C-reactive protein (18 mg/L) suggested an inflammatory response. Proteinuria was also noted, consistent with dehydration-related renal stress.

Arterial blood gas analysis, serum bicarbonate, anion gap calculation, serum β-hydroxybutyrate, as well as complete electrolyte and renal function panels were not fully available at presentation due to resource limitations. Despite this, the combination of hyperglycemia, ketonuria, dehydration, and clinical presentation supported a working diagnosis of diabetic ketoacidosis.

2.4. Clinical Rationale for Diagnosis

Although full biochemical confirmation was unavailable, diabetic ketoacidosis was considered the most probable diagnosis based on significant hyperglycemia (2.55 g/L), marked ketonuria (3+), clinical dehydration and tachypnea, rapid response to insulin and fluid therapy, and absence of alternative toxic or neurological explanation. These findings were considered sufficient in a resource-limited context to justify immediate treatment rather than delayed confirmatory testing.

2.5. Clinical Timeline

The clinical course began approximately three days prior to admission with progressive insomnia, increasing psychomotor agitation, and incoherent speech. On the day of admission, the patient was initially evaluated in a psychiatric context, and benzodiazepine withdrawal was suspected due to the history of abrupt lorazepam discontinuation. Within the first hours of hospitalization, physical examination revealed signs suggestive of a metabolic disorder, including tachypnea and dehydration. During the first 24 hours, laboratory investigations demonstrated significant hyperglycemia and ketonuria, leading to the diagnosis of diabetic ketoacidosis and initiation of intravenous fluid resuscitation and insulin therapy.

Over the subsequent 24 to 48 hours, there was a marked reduction in agitation and progressive improvement in orientation and cognitive function. By the fourth day of hospitalization, the patient had achieved full recovery of baseline mental status with complete resolution of delirium.

2.6. Etiological Investigation of DKA

An etiological assessment was performed to identify precipitating factors. No clear infectious focus was identified clinically, although inflammatory markers were mildly elevated. The patient reported reduced oral intake due to agitation and insomnia in the days preceding admission. There was also suspicion of suboptimal adherence to antidiabetic treatment during the same period.

No corticosteroid use, sympathomimetic exposure, or other hyperglycemic drugs were identified. The most plausible contributors were dehydration, reduced oral intake, and possible treatment non-adherence in a background of poorly controlled diabetes.

2.7. Treatment and Outcome

Treatment consisted of intravenous isotonic fluid resuscitation and continuous insulin infusion, following standard DKA management principles. Electrolytes were monitored and corrected as permitted by available resources.

Clinical evolution was rapidly favorable. Psychomotor agitation decreased within 48 hours, with progressive restoration of orientation and cognitive clarity. By day four, the patient had fully recovered baseline mental status with no residual confusion.

3. Discussion

Delirium is a medical emergency requiring immediate identification of underlying causes. Failure to recognize metabolic etiologies may lead to inappropriate psychiatric management and delayed life-saving interventions [2] [6].

DKA is increasingly recognized as a cause of neuropsychiatric syndromes, including delirium and acute confusional states. Hyperosmolarity, metabolic acidosis, electrolyte imbalance, and systemic inflammation contribute to neuronal dysfunction and acute brain failure [3] [4] [7] [8] [9].

In this case, diagnostic difficulty was amplified by the presence of abrupt benzodiazepine discontinuation, which strongly biased initial clinical reasoning toward withdrawal delirium, illustrating a classical anchoring bias.

3.1. Diagnostic Challenges

The clinical overlap between metabolic delirium and benzodiazepine withdrawal delirium is substantial. However, DKA is more commonly associated with dehydration, tachypnea, and metabolic abnormalities, whereas benzodiazepine withdrawal is characterized by tremor, marked autonomic hyperactivity, and anxiety. The main differential features between DKA-related delirium and benzodiazepine

withdrawal delirium are summarized in Table 1.

Table 1. Differential diagnosis between DKA-related delirium and benzodiazepine withdrawal.

Feature	DKA (Metabolic Delirium)	Benzodiazepine Withdrawal
Onset	Progressive	Rapid after discontinuation
Context	Diabetes, metabolic stress	Drug cessation
Consciousness	Fluctuating	Hypervigilance
Neurological signs	Confusion, possible coma	Tremor, anxiety
Respiratory pattern	Kussmaul breathing	Normal
Autonomic signs	Moderate	Marked
Blood glucose	Elevated	Normal
Ketones	Positive	Negative
Acidosis	Present	Absent
Biology	Electrolyte imbalance	Usually normal
Treatment	Insulin + fluids	Benzodiazepines
Outcome	Rapid if treated	Variable

3.2. Clinical Reasoning and Bias

This case illustrates anchoring bias, emphasizing the importance of prioritizing reversible medical causes before psychiatric diagnoses.

3.3. Clinical Implications

Systematic evaluation of delirium must include vital signs, metabolic screening, medication history, and physical examination. International guidelines emphasize delirium as a medical emergency requiring urgent etiological assessment [7].

3.4. Management Considerations

DKA-related delirium resolves with correction of metabolic abnormalities. Psychotropic medications should not be used without addressing the underlying cause.

3.5. Limitations

Limitations include absence of arterial blood gas, serum ketones, electrolyte panels, and standardized delirium assessment tools.

3.6. Differential Diagnosis

Other causes include hypoglycemia, hyperosmolar state, CNS infections, electrolyte disorders, and intoxications.

4. Conclusion

DKA-related delirium may mimic psychiatric disorders and substance withdrawal

syndromes. Delirium should always be considered of organic origin until proven otherwise.

Ethics Statement

Written informed consent was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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