Journal of Biomedical and Pharmaceutical Research
Available Online at www.jbpr.in
CODEN: - JBPRAU (Source: - American Chemical Society)
NLM (National Library of Medicine): ID: (101671502)
Index Copernicus Value 2023: 85.868
Volume 14, Issue 3; 2025, 27-31

ISSN (Online): 2279-0594
ISSN (Print): 2589-8752

Case Report

A Rare Presentation of Cerebral Edema in Adult-Onset Diabetic Ketoacidosis: Clinical Challenges and Management in a High-Risk Elderly Patient

M. Kavitha[1], R. Pavani[2], K. Udayasri[3]

[1, 2, 3]Pharm D 4th year Students, Anurag Pharmacy Collage, Kodad, Telangana

Article Info: Received: 15-03-2025 / Revised: 30-04-2025 / Accepted: 10-05-2025
Corresponding Author: M. Kavitha
DOI: https://doi.org/10.32553/jbpr.v14i3.1311
Conflict of interest statement: No conflict of interest

Abstract:
Diabetic ketoacidosis (DKA) is a serious, potentially life-threatening complication of diabetes mellitus, characterized by hyperglycemia, metabolic acidosis, and ketonemia. While more common in type 1 diabetes, DKA is increasingly seen in patients with type 2 diabetes, accounting for nearly one-third of cases. Cerebral edema, although rare in adults, represents a grave complication of DKA that can result in significant morbidity and mortality. This case report discusses a 74-year-old female with a history of poorly controlled type 2 diabetes mellitus, hypertension, ischemic stroke, and hypothyroidism, who presented to the emergency department with generalized tonic-clonic seizures and involuntary movement of the right lower limb. On examination, she was somnolent and disoriented. Laboratory evaluation revealed severe DKA, with associated electrolyte abnormalities and hyperosmolarity. Neuroimaging showed evidence of cerebral edema, likely contributing to her seizure activity. Immediate intensive care management was initiated, including intravenous fluids, continuous insulin infusion, electrolyte correction, osmotherapy for cerebral edema, and antiepileptic therapy. Over the following 48 hours, her metabolic parameters improved, and her neurological status gradually stabilized. She remained seizure-free on maintenance antiepileptic medication and was transferred to the general ward in stable condition. This case highlights the importance of early recognition and aggressive management of DKA and its complications, particularly in elderly patients with multiple comorbidities. A multidisciplinary approach, including fluid resuscitation, insulin therapy, neurological monitoring, and patient education, is essential to improve outcomes and prevent recurrence. Clinicians should maintain a high index of suspicion for cerebral edema in DKA patients presenting with altered mental status or seizures.

Keywords: Diabetic Ketoacidosis (DKA), Cerebral Edema, Type 2 Diabetes Mellitus, Seizures, Elderly Patient

Introduction

Diabetic ketoacidosis (DKA) is a serious and potentially life-threatening complication of diabetes mellitus, characterized by hyperglycemia, metabolic acidosis, and ketonemia. While more commonly associated with type 1 diabetes mellitus, DKA can also occur in patients with type 2 diabetes, especially under conditions of significant metabolic stress or insulin deficiency [1]. The condition often develops rapidly and may be precipitated by infections, missed or inadequate insulin therapy, myocardial infarction, stroke, or substance

abuse. In elderly patients with multiple comorbidities, DKA may present atypically and with higher morbidity and mortality [2].

The most common precipitating factors in DKA include non-adherence to insulin therapy, infections, and new-onset diabetes mellitus. Among patients with type 1 diabetes, treatment non-compliance is the predominant cause, whereas infections are more frequently implicated in DKA episodes in patients with type 2 diabetes [3]. Additional contributing factors may include psychosocial instability, mental health disorders, and economic limitations. Drug abuse, particularly cocaine use, has also been identified as a significant risk factor for recurrent DKA episodes in some populations [4].

Clinically, DKA often manifests with nausea, vomiting, abdominal pain, polyuria, polydipsia, Kussmaul respirations, hypotension, dehydration, and altered mental status. In severe cases, neurological complications such as seizures or cerebral edema may occur, which substantially increase the risk of morbidity and mortality, especially in adult patients [5]. Cerebral edema, although rare in adults, is a well-documented and often fatal complication of DKA, and is thought to result from osmotic shifts, inflammatory cascades, and cerebral hypoperfusion during rapid correction of metabolic derangements [6].

Pathophysiologically, DKA results from a severe deficiency of insulin combined with elevated levels of counter-regulatory hormones such as glucagon, cortisol, catecholamines, and growth hormone. These hormonal imbalances lead to increased hepatic gluconeogenesis and glycogenolysis, impaired peripheral glucose utilization, and lipolysis, which in turn contribute to hyperglycemia, ketonemia, and metabolic acidosis [7].

The accompanying osmotic diuresis results in significant fluid and electrolyte loss, contributing to hypovolemia, electrolyte imbalance, and in some cases, acute kidney injury [8].

Management of DKA requires a multifaceted approach focused on fluid resuscitation, insulin administration, electrolyte correction (especially potassium), and identification and treatment of the underlying cause. Initial fluid therapy with isotonic saline is essential to restore circulatory volume and improve tissue perfusion. Regular insulin administered via continuous intravenous infusion remains the mainstay of therapy for resolving hyperglycemia and ketosis [9]. Potassium replacement must be carefully managed due to insulin-induced intracellular shifts, and serum levels must be closely monitored.

In this context, neurological monitoring is vital, particularly in patients with altered mental status, as cerebral edema can present subtly and progress rapidly. In suspected cases, mannitol or hypertonic saline (3%) should be administered promptly to reduce intracranial pressure [10].

This case highlights the importance of early recognition and aggressive management of DKA and its complications, particularly in elderly patients with multiple comorbidities, including hypertension, cerebrovascular disease, and poor glycemic control. It also underscores the need for improved patient education, medication adherence, and long-term follow-up to prevent recurrence and reduce healthcare burden.

Case Report

A 74-year-old female was brought to the Emergency Department (ED) by her family with a chief complaint of loss of consciousness (LOC) accompanied by seizure-like activity, including uprolling of the eyes. The episode occurred approximately two hours prior to arrival. According to the family, the seizure lasted for about 3–5 minutes and was characterized by generalized tonic-clonic movements (GTCS), uprolling of the eyes, and lip biting. This was followed by a postictal state of drowsiness and confusion.

The patient has a known history of multiple chronic conditions, including hypertension for the past 10 years, type 2 diabetes mellitus for 3

years, a right-sided ischemic cerebrovascular accident (CVA) with residual hemiparesis for 3 years, and hypothyroidism for the last 8 years. Her family reported that she had not been compliant with her diabetic medications over the past week. Additionally, she had symptoms of increased thirst (polydipsia), frequent urination (polyuria), generalized weakness, and poor oral intake.

In the hours leading up to her hospital visit, the family observed three episodes of involuntary movements in her right lower limb, each lasting around 10 minutes, and occurring at intervals of approximately one hour. These movements were described as jerky and repetitive and appeared to be focal in nature, raising the possibility of partial seizures or post-stroke seizure activity.

On presentation to the ED, the patient was somnolent and disoriented, remaining in a postictal state. Initial clinical examination showed decreased consciousness, with persistent right-sided hemiparesis (pre-existing), and no signs of meningeal irritation. In view of the seizure activity and altered mental status, the emergency physician ordered an urgent laboratory workup, which included a complete blood picture (CBP), thyroid profile, renal function tests, blood glucose and ketone levels, electrolyte panel, and arterial blood gas (ABG) analysis.

Laboratory investigations revealed significant hyperglycemia, metabolic acidosis, positive urine ketones, and electrolyte imbalances, notably hypokalemia. Thyroid function appeared to be stable under existing treatment. These findings confirmed a diagnosis of diabetic ketoacidosis (DKA), likely precipitated by medication non-compliance and inadequate oral intake. The seizure was considered secondary to metabolic derangement and possible cerebral edema.

The patient was promptly admitted to the Intensive Care Unit (ICU) for critical care management. Treatment was initiated with intravenous fluid resuscitation using 0.9% normal saline, and continuous infusion of regular insulin for glycemic control. Electrolyte correction was carried out with intravenous potassium chloride based on potassium levels. Seizure control was achieved with intravenous administration of Levetiracetam (LEVIPIL). Suspected cerebral edema was managed by elevating the head of the bed to 30 degrees, along with close monitoring of neurological status.

Continuous monitoring of vital signs, capillary blood glucose levels, electrolytes, acid-base balance, and Glasgow Coma Scale (GCS) score was maintained. Supportive measures were provided, including appropriate nutrition and hydration. Over the following 48 hours, the patient exhibited a steady clinical improvement. Her metabolic parameters stabilized, acidosis resolved, and serum electrolytes normalized. There were no further seizure episodes, and her mental status improved significantly, with corresponding improvement in her GCS score.

As her condition stabilized, cerebral edema subsided, and there was no longer a need for aggressive osmotic therapy. She remained seizure-free on antiepileptic maintenance therapy. Once the patient was hemodynamically stable and metabolically compensated, she was transferred to the General Medical Ward (GMW) for further care and observation. Her blood pressure remained well-controlled on oral antihypertensive medications. Her antidiabetic regimen was revised, and patient education regarding medication adherence and regular follow-up was emphasized. The case underscores the importance of early recognition and aggressive management of DKA, especially in elderly patients with multiple comorbidities. Multidisciplinary care and vigilant monitoring led to a favorable clinical outcome.

Discussion

This case illustrates a complex and life-threatening presentation of diabetic ketoacidosis (DKA) in an elderly female with multiple chronic comorbidities, including poorly controlled type 2 diabetes mellitus, hypertension, previous cerebrovascular accident

(CVA) with residual right-sided hemiparesis, hypothyroidism, and suspected iron deficiency anemia. The clinical course was further complicated by the development of generalized tonic-clonic seizures and cerebral edema, highlighting the multifactorial nature of acute metabolic and neurological deterioration in vulnerable populations.

Cerebral edema, though rare in adults, is a serious and potentially fatal complication of DKA. The pathophysiological mechanisms remain incompletely understood but are believed to involve osmotic shifts during rapid correction of hyperglycemia, intracellular acidosis, and inflammatory responses. In this patient, pre-existing hypertension may have impaired cerebral autoregulation, and suspected anemia could have contributed to decreased oxygen-carrying capacity, further exacerbating cerebral hypoxia and increasing the risk of cerebral edema.

The patient's seizures were most likely secondary to increased intracranial pressure and cerebral irritation resulting from cerebral edema. Seizure activity in the context of DKA should always prompt consideration of neurological complications, especially when accompanied by altered mental status or focal neurological signs. In this case, prompt recognition and early intervention, including careful fluid resuscitation, gradual correction of blood glucose, electrolyte management, and elevation of the head of the bed, played a critical role in reducing intracranial pressure and facilitating neurological recovery.

Although iron deficiency anemia was not the direct cause of the acute decompensation, it warrants further diagnostic evaluation to determine the underlying etiology and prevent long-term systemic effects. Chronic anemia in elderly patients with comorbidities can impair organ perfusion and exacerbate acute illness outcomes.

Importantly, this case emphasizes the role of poor medication adherence as a major precipitating factor in the development of DKA. Non-compliance with antidiabetic and antihypertensive medications significantly increases the risk of acute metabolic crises and complications. Targeted patient education, regular follow-up, and the use of compliance-supporting strategies such as caregiver involvement or medication reminders are essential in managing chronic diseases effectively.

Overall, this case underscores the importance of maintaining a high index of suspicion for rare but serious complications like cerebral edema in adults with severe DKA, especially in those with multiple comorbidities. Early, aggressive, and multidisciplinary management is vital for reducing morbidity and improving outcomes. Additionally, comprehensive evaluation and long-term management of coexisting conditions, such as anemia and cardiovascular disease, are crucial in preventing recurrence and enhancing the quality of life for such high-risk patients.

Conclusion

This case underscores the critical importance of recognizing and promptly managing diabetic ketoacidosis (DKA), particularly in elderly patients with multiple comorbidities. The presence of poorly controlled type 2 diabetes, hypertension, prior cerebrovascular accident, and suspected iron deficiency anemia contributed to a more complex and high-risk clinical scenario. Cerebral edema, though rare in adults, developed as a life-threatening complication of DKA, leading to generalized tonic-clonic seizures. Early intervention with fluid resuscitation, insulin therapy, seizure control, and neurological monitoring was crucial in reversing the metabolic crisis and improving neurological outcomes. The patient’s gradual clinical improvement highlights the effectiveness of timely and coordinated multidisciplinary care. Poor medication adherence was a key precipitating factor, emphasizing the need for patient education and long-term disease management strategies. Regular follow-up and monitoring of glucose levels, renal function, and neurologic status are vital to prevent recurrence. The case reinforces

the importance of individualized care in managing complex diabetic emergencies. Future care should also focus on optimizing control of chronic comorbidities. Overall, this case highlights the potential severity of DKA and the necessity for vigilance in high-risk populations.

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