Case Report

Diabetic ketoacidosis with sepsis complicated by central pontine myelinolysis: A case report

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ABSTRACT

Diabetic ketoacidosis (DKA) is a condition characterized by high blood sugar, elevated ketone bodies, and acidosis. Central pontine myelinolysis (CPM) is a severe demyelinating syndrome that can be life-threatening. The association between CPM and DKA is very less reported in the medical literature. In this case, a 22-year-old female presented with new-onset diabetes and developed DKA, which was managed with aggressive fluid resuscitation and sodium bicarbonate infusion. However, the patient subsequently experienced loss of consciousness and altered mental status. Further examination revealed altered serum sodium levels and positive urinary ketones. Brain magnetic resonance imaging (MRI) showed cerebral edema and abnormal signal intensity in the medulla and central pons. Supportive therapy was initiated, and over the next few weeks, the patient gradually regained consciousness and muscle strength. Follow-up after 5 months showed complete resolution of symptoms and no residual damage on MRI. The exact mechanism underlying the development of CPM while managing DKA remains unclear, although it is hypothesized to be related to a rapid hypertonic insult. This case highlights the importance of recognizing and managing this rare complication in patients with DKA.

Key words: Case reports, Central pontine, Diabetic ketoacidosis, Hypernatremia, Hypokalemia, Myelinolysis, Osmotic demyelination syndrome

Diabetic ketoacidosis (DKA) is a metabolic emergency seen in individuals with uncontrolled diabetes, characterized by hyperglycemia, elevated ketones, and metabolic acidosis [1]. Central pontine myelinolysis (CPM) is a rare and potentially devastating neurological disorder characterized by demyelination in the central pons [2]. It is often associated with rapid changes in serum sodium levels, although other risk factors and underlying conditions can contribute to its development [3]. The association between CPM and DKA is unusual, with only a few reported cases in the medical literature [4-7].

Although CPM is classically associated with rapid correction of hyponatremia, its occurrence in the setting of DKA remains uncommon and poorly understood. The coexistence of severe hyperglycemia, hypernatremia, hypokalemia, and sepsis in our patient makes this case clinically significant. We report this case to highlight the potential role of osmotic and electrolyte fluctuations during DKA management in precipitating CPM, emphasize the importance of careful electrolyte monitoring, and contribute to the limited literature regarding this rare neurological complication.

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CASE PRESENTATION

A 22-year-old female presented with a high-grade fever accompanied by chills and rigors for 5 days, along with vomiting and abdominal pain. The pain was dull aching in nature, moderate in intensity, non-radiating, and associated with multiple episodes of vomiting. There were no clear aggravating or relieving factors, and no history of abdominal distension, hematemesis, or melena. In addition, she had experienced an altered sensorium for 1 day. The patient did not have a known history of diabetes or hypertension.

Upon clinical examination, she appeared drowsy and unresponsive to stimuli. Both pupils were small and non-reactive to light. Her temperature was 102.9°F, heart rate was 120 beats/min, and blood pressure

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measured 113/70 mmHg. Tachypnea was observed, with a respiratory rate of 28 breaths/min and oxygen saturation at 98%. Blood sugar level at presentation was 811 mg/dL/examination of the chest, heart, and abdomen did not reveal any significant abnormalities.

Laboratory investigations are summarized in Table 1. Initial evaluation revealed severe hyperglycemia with elevated HbA1c, hypernatremia, hypokalemia, hyperchloremia, and mild renal dysfunction. Urine analysis demonstrated ketonuria with significant pyuria, while both blood and urine cultures were positive for bacterial infection. Arterial blood gas analysis showed metabolic derangements consistent with DKA. Hematological investigations revealed leukocytosis with neutrophilic predominance. Cerebrospinal fluid analysis showed mildly elevated adenosine deaminase and protein levels. Magnetic resonance imaging (MRI) of the brain revealed hyperintense signals on T2 and fluid-attenuated inversion recovery images in the pons and bilateral brachium pontis, suggestive of demyelination (Fig. 1). Contrast MRI was not done due to a rise in serum creatinine.

The patient was diagnosed with DKA with sepsis and possible demyelination and immediately received treatment, including 0.45% saline infusion, intravenous insulin, and appropriate antibiotics. Despite medical advice to stay, the patient was voluntarily discharged against medical advice. Four days later, she was brought back to the hospital in a state of unconsciousness and with a history of 1 day of altered sensorium. Initial vitals were notable with blood pressure of 60/40 mmHg and blood sugar levels of 37 mg/dL. After receiving intravenous adrenaline infusion and intravenous 25% dextrose infusion, vitals were corrected to 100/60 mmHg and 198 mg/dL, respectively. Other vital signs were within normal limits. On clinical examination, bilateral crepitations were heard in the respiratory system, bilateral plantar reflexes were non-responsive, and doll's eye movement was present. The Glasgow Coma Scale (GCS) score was E1V1M1. Laboratory investigations revealed elevated sodium levels, low potassium levels, and elevated chloride levels (Table 1). Arterial blood gas analysis showed a pH of 7.379 (normal range 7.35–7.45) and a pO2 of 164.2 mmHg. The patient was intubated due to a poor GCS score and to prevent aspiration.

Management included intravenous fluid administration (3 units of Ringer's lactate at a rate of 50–70 mL/h), injection of noradrenaline, appropriate antibiotics (injection piptaz 4.5 g intravenously 3 times a day, capsule doxycycline 100 mg twice daily, injection metronidazole), and steroids (injection dexamethasone 8 mg intravenously 3 times a day) for 6 days. Supportive therapy was initiated, and over the next few weeks, the patient gradually regained consciousness and muscle strength. The patient was fully recovered and subsequently discharged. Follow-up after 5 months showed complete resolution of symptoms and no residual damage on MRI.

Table 1: Laboratory investigations during first and second admission

Parameter	First admission	Second admission	Normal range
Blood glucose	811 mg/dL	37 mg/dL	70–140
HbA1c	9.2%	—	<5.7
Serum sodium	159	156	135–145
Serum potassium	1.73	2.06	3.5–5.3
Serum chloride	117	113	96–106
Serum creatinine	1.6	—	0.7–1.4
Blood urea	50	—	15–40
WBC count	13,200	—	4,000–11,000
Neutrophils	85%	—	40–70
Lymphocytes	12%	—	20–40
pH	—	7.379	7.35–7.45
pCO_2	31.99	—	35–45
pO_2	85.65	164.2	75–100
Urine ketones	Positive	—	Negative
Urine culture	Positive	—	Sterile
Blood culture	Positive	—	Sterile

WBC: White blood cell, HbA1c: Hemoglobin A1C

DISCUSSION

DKA is a severe complication of diabetes mellitus that can be life-threatening if left untreated [1]. It is a complex metabolic state characterised by hyperglycemia, acidosis, and ketonuria, which is consistent with the initial presentation of our case [7]. Osmotic demyelination syndrome, which includes both CPM and EPM, is one of the encephalopathies that result from extreme fluctuations in serum sodium concentration and plasma osmolality [8].

CPM can also develop irrespective of serum sodium levels, in various other conditions, such as diabetes mellitus, hepatocellular dysfunction, lithium toxicity, hypokalemia,

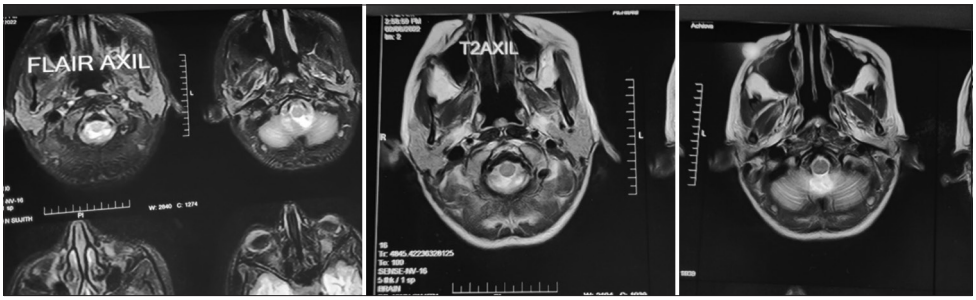

Figure 1: Magnetic resonance imaging of the brain revealed hyperintense signals on T2 and fluid-attenuated inversion recovery images in the pons and bilateral brachium pontis, suggestive of demyelination

pituitary surgery, chronic alcoholism, hypophosphatemia, chemotherapy, malnutrition, prolonged diuretic use, burns, and post-liver transplant [2-4]. Pathophysiologically, CPM is caused by disruption of the blood-brain barrier, leading to demyelination due to a rapid change in serum osmolality and a shift in the idiogenic osmolytes. Subsequent damage to the brain cells (particularly the oligodendrocytes) occurs in response to osmotic injury, resulting in the release of harmful substances and dehydration of the brain. CPM lesions are most commonly found in the pons and areas with a significant overlap of grey and white matter. Extrapontine lesions may occur in the basal ganglia, thalamus, and/or midbrain. Females are more commonly affected by CPM [2-4]. DKA causes varying degrees of derangement of fluid balance and electrolytes due to hyperglycemia-induced osmotic fluid shifts, osmotic diuresis, complications of end-organ injury, and therapies used in its management [5]. CPM is a relatively rare complication of DKA. It was originally described as a complication of rapid correction of hyponatremia. Demyelination is now believed to occur secondary to rapid osmotic shifts from changes in any osmotically active agents, including sodium, potassium, glucose, and ammonia [6].

Although hyponatremia is by far the most common finding on the presentation of CPM, recently other associations have also been observed, most notably hypokalemia. Osmotic diuresis, secondary hyperaldosteronism, and emesis in DKA all cause net depletion of body stores of potassium. Hypokalemia is also a recognised complication of the management of DKA, as insulin administration and correction of metabolic acidosis shifts potassium intracellularly [8]. Several reports have shown that underlying hypokalemia may predispose patients to more pronounced myelin damage [9]. CPM has also been reported to arise from hyperglycemia in association with concomitant acidosis, hypernatremia, and hyperosmolar syndrome. Yoon *et al.* described a case of CPM developed in the setting of relatively mild hypernatremia [9]. Most of the previous reports of CPM occurring in the setting of hyperglycemia describe concomitant ketoacidosis, abnormality in serum sodium, or occurrence after treatment of the hyperglycemic hyperosmolar state. Saini *et al*., reported a rare presentation of CPM, which was purely secondary to hyperosmolar hyperglycemia. Recently, there has been an increase in the number of reports of CPM occurring in association with other hyperosmolar states and with radiological lesions that extend beyond the pons [4]. In a pediatric case reported by Sivaswamy *et al*., there were no wide fluctuations in the sodium level, but they noted fluctuations in the osmolarity [6]. Gonzalez Caldito *et al.*, described that it is plausible that a rapid drop in osmolality in a chronic state of high osmolality led to CPM in their case and a slower correction of hyperglycemia possibly could have prevented it [3].

Notably, the patient presented with hypernatremia and hypokalemia, which is atypical for CPM. It remains unclear whether demyelination occurred due to the severity of illness, the therapies administered, or a combination of these factors. In this case, CPM may have developed during hyperglycemia correction in DKA treatment, as a result of the hyperosmolar state at the initial presentation of DKA, due to hypokalemia alone, or from a combination of these contributors. Changes in plasma osmolality, which were not recorded during either hospital stay, could have also played a role in the demyelination. Rapid correction of hyponatremia is less likely to have been the primary cause, as sodium levels remained consistently high or normal across both admissions. Continuous monitoring could potentially have prevented the acute worsening of the patient's condition, as indicated by improving sodium and potassium trends during hospitalization and their subsequent deterioration at home (Table 1). Proper supervision by physicians and hospital staff, as opposed to the patient being at home, might have mitigated the risk of further decline.

Distinguishing CPM from other conditions, such as demyelinating diseases, pontine neoplasms, and infarcts, can be challenging. Clinical features, time course of evolution, and specific radiological characteristics can help differentiate these conditions. It is important to note that CPM lesions tend to regress over time and do not require specific therapy [3]. Supportive care and regular follow-up are crucial for recovery from CPM. In some cases, treatment with steroids, plasmapheresis, or intravenous immunoglobulin has been successful [4]. Recently, there have been reports suggesting that corticosteroids may improve the outcome of hypernatremic demyelination as they protect the brain blood barrier and play a critical role in protecting against demyelination. Dexamethasone had a protective role against osmotic-induced demyelination in rats, and a Japanese study from 2007 described that five patients who received corticosteroids had better outcomes [8]. In our case, the patient was treated with dexamethasone 8 mg intravenously three times daily, which led to noticeable improvement, supporting the likelihood that the hypernatremic state contributed to her symptoms.

CONCLUSION

This case report presents a unique instance where CPM developed during the management of DKA with sepsis. Our case highlights the importance of recognizing that CPM can result from osmotic shifts not limited to serum sodium levels but also involving glucose, potassium, and ammonia. Early recognition and prompt management of CPM are essential to optimizing patient outcomes. Healthcare professionals should remain vigilant about the risk factors and potential development of CPM in DKA patients, ensuring close monitoring of electrolyte imbalances, particularly in the context of hyperosmolar and hyperglycemic states.

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