



Hypernatremia in a Case of Type I Diabetes Presenting as Diabetic Ketoacidosis- Case Report

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Abstract

Diabetic ketoacidosis (DKA) is the leading cause of morbidity and mortality in patients with Type I Diabetes (T1DM). A hyperosmolar state also presents in patients presenting as DKA due to increased glucose production and limited glucose utilisation. A hyperglycaemic state will lead to hyponatremia due to osmotic diuresis. Although mild hypernatremia could be present in patients who also exhibit as severely dehydrated DKA, the severe degree of hypernatremia is rarely presented.^[1] Hereby we report a case of Type I Diabetes presenting as DKA having hypernatremia.

Keywords: Diabetic ketoacidosis, Type I Diabetes, hypernatremia.

Case Presentation

A previously healthy 38 year old female was brought to our emergency department with complaints of altered sensorium of 5 h duration and vomiting around 8-10 episodes daily for the last 2 days. She had a 3-day history of fever, intermittent type, associated with chills which had subsided on taking medications from a local clinic. She was a known case of Type I Diabetes since the last 3 years and was on treatment with Insulin (30:70 Regular and NPH) 15-0-10 units. At presentation she was unconscious and mildly responsive to deep pain. She had a blood pressure of 90/60 mm Hg, heart rate 130 bpm, respiratory rate 24 breaths/min and a temperature 36.4°C. She

was severely dehydrated with dry tongue and poor skin turgor.

Investigations

Initial biochemical findings were as follows: plasma glucose level 500 mg/dL, serum sodium level 158 mEq/L, serum osmolarity 425 mOsm/L, serum potassium 3.9 mEq/L, serum creatinine 0.7 mg/dL. Her arterial blood gas analysis showed a pH of 6.92, PCO₂ 17.8, PO₂ 74.8 and HCO₃ 3.6. Ketone bodies in her serum and urine were positive. The findings of NCCT brain revealed normal without any evidence of cerebral oedema.

Treatment

After initial fluid resuscitation with 2 L of normal saline, a continuous insulin infusion was begun until the sugars came to 250 mg/dl. After which she was started on DNS infusion with Insulin added to it. The patient was rehydrated via fluid therapy on the basis of a calculated free water deficit ($0.6 \times \text{BWt} \times [1 - (140 \div \text{Na})]$) by administration of 0.45% saline. Antibiotic coverage was put appropriately. Metabolic Acidosis was corrected by administering Bicarbonate after calculation of Bicarbonate deficit. Clinical and biochemical monitoring were regularly performed during her intensive treatment.

Outcome and Follow-up

Plasma glucose concentration improved steadily after the initial infusion therapy and came back to normal within the 1st day itself. The patient was prophylactically intubated after 3 hours of admission in view of tachypnoea, tachycardia and hypoxia. Serum sodium and osmolality values decreased slowly with the required correction and came back to normal over 96h. All other laboratory values were normal. The patient recovered completely and was discharged after 10 days with a regimen of multiple daily insulin injections.

Discussion

DKA is usually considered a complication of T1DM, while a hyperosmolar hyperglycaemic state (HHS) is normally associated with type II DM (T2DM).^[1] DKA is a rare yet potentially fatal hyperglycemic crisis that can occur in patients with both type 1 and 2 diabetes mellitus. Due to its increasing incidence and economic impact related to the treatment and associated mortality and morbidity, effective management is essential. Elements of management include making the appropriate diagnosis using current laboratory tools and clinical criteria and coordinating fluid resuscitation, insulin therapy, and electrolyte replacement through feedback obtained from

timely patient monitoring and knowledge of resolution criteria.^[2]

There have been few reports of combined DKA and hypernatremic hyperosmolality. One recent report attributed the occurrence of hypernatremia at new-onset T1DM in patients with DKA to a huge daily intake of carbonated carbohydrate beverages.^[3] In another report a DKA child presented with severe hypernatremia and hyperosmolality due to herbal product ingestion.^[4] In patients with DKA, as the glucose concentration and extracellular osmolality increases, an osmolar gradient is created that leads to the redistribution of water from the intracellular to the extracellular compartment.^[5] This initial shift of water leads to a decrease in the serum sodium concentration (1.6 mmol/L per each 5.5 mmol/L increase in glucose); thus, a hyponatremia is often seen in these patients.^[5]

Our case presented with Hypernatremia which was treated with appropriate water deficit correction and showed no other complications due to the same such as cerebral oedema.

Conclusion

There are very few such cases reported worldwide and thus physicians should be aware of it. Fluid therapy for DKA with hypernatremic hyperosmolality should be achieved steadily to avoid cerebral oedema with continuous monitoring.

References

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