Salty Diabetes: Hypernatremia in Diabetic Ketoacidosis

Hyponatremia in diabetic ketoacidosis (DKA) is an expected electrolyte imbalance secondary to dilutional effect of hyperglycemia. Hypernatremia in DKA is rarer and is associated with increased morbidity and mortality. The exact etiology for the same is not clear. Several mechanisms such as renal losses secondary to glucosuria mediated osmotic diuresis, vomiting and diarrhea and inappropriate water replacement have been proposed. Treatment consists of aggressive management of DKA instead of hypernatremia, choosing a hypo-osmolar fluid, and switching to dextrose normal saline (0.45%). We present a 14-month-old girl with DKA and severe hypernatremia who responded to aggressive management of DKA, rigorous intravenous hydration and the above-mentioned strategies.

A fourteen-month-old girl presented in altered sensorium with a history of respiratory distress and fever. She had a heart rate of 172 beats per minute, respiratory rate of 38 breaths per minute, oxygen saturation of 89% on room air, normal blood pressure and Glasgow Coma Scale (GCS) of 10 (E3V3M4). Physical examination was remarkable for a thin physique, dull activity, acidic breathing and altered sensorium. At admission, fingerstick glucose was 511 mg/dL. Initial laboratory reports revealed a blood sugar of 545 mg/dL, pH of 6.9, lactate of 0.8 mmol/L, bicarbonate of 4.4 mEq/L, partial pressure of CO2 of 7.6 mmHg, suggestive of metabolic acidosis, with serum sodium 147 mEq/L (corrected sodium 153 mEq/L), potassium 5.2 mEq/L, chloride 110 mEq/L, and positive urine ketones. HbA1C level was 9%. There was no previous history of diabetes in the child.

The child was started on oxygen using a high-flow nasal cannula (HFNC) and received a 0.9% saline bolus. Three hours later, the sodium levels increased from 147 mEq/L to 154 mEq/L. Intravenous fluids were continued at 50 mL/hour with added potassium. Insulin infusion was started at 0.1 units/kg/h and was adjusted as required. Subsequently the corrected serum sodium of 160-166 mEq/L was recorded at 3 and 20 hours respectively (Table I). Fluids were changed from 0.9% to 0.45% NS once the blood glucose was close to 250 mg/dL, to initiate the correction of sodium at a rate of approximately 0.5 mEq/L/hour. Chest X-ray was suggestive of bronchopneumonia. Continuous monitoring of vitals, urinary ketones, electrolytes and blood gas was done. The child received blood transfusion in view of low hemoglobin (6.2 g/dL).

The corrected sodium decreased after 30 hours to 149 mEq/L (Table I) with a noticeable improvement in mental status. Child was weaned off HFNC on day 2. Insulin infusion was tapered and subcutaneous insulin was started at 0.4 units/kg/day once the child was able to tolerate oral intake, mental status improved, and the anion gap resolved. She was discharged after counseling regarding the risks and complications of the disease and precautions to be taken.

Electrolyte disturbances are common in patients with diabetes and may be related to the osmotic effect of glucose that causes the fluid to shift from intracellular spaces to extracellular compartment and osmotic diuresis leading to dehydration.

Hypernatremia has been described less commonly in pediatric DKA and is usually associated with excessive soft drink ingestion [1]. Our patient did not have any similar history. Hypernatremia may ensue following the loss of water during vomiting, glucose-induced osmotic diuresis and insensible losses, which add to consequential high osmolarity. Acidosis was reported as the most influential and synergistic factor with high osmolality, dismissing the role of serum ketones in DKA with altered sensorium [2].

Normal saline is the fluid of choice in DKA with low or normal serum sodium levels [3]. It will cause intravascular expansion and correct the hyperosmolar hypovolemic hypernatremia. This is based on the consideration that every litre of normal saline can, theoretically, increase serum sodium by 0.41 mEq/L per litre of normal saline administered, assuming serum sodium of 140 mEq/L and total body water of 60%. However, in patients with hypernatremia and DKA, solutions with less sodium content, such as Ringer’s lactate (RL) (130 mmol/L of sodium for every litre of the solution infused) or half normal saline (77 mmol/L of sodium for every litre of the solution infused) are more appropriate to decrease the serum sodium at an initial stage.

The rate of sodium correction is critical and suggested at 10 mEq/L per 24 hours [4]. However, the correction rate of acute hypernatremia is not as well defined as it is for acute hyponatremia. Low pH seen in DKA can cause increased proteolysis and an inability of the proteins to
function at their physiological pH dysregulating the normal function of cells [5]. It can also decrease systemic response to catecholamines, leading to hypotension, organ dysfunction, and death if left untreated. Dehydration requires rapid management in moderate to severe hypovolemic hyperosmolar hypernatremia and may be more critical to treat.

In the index case, the treatment of DKA was prioritized followed by correction of hypernatremia to prevent a rapid change in osmolarity and cerebral edema. Altered sensorium has been attributed to the hyperosmolarity of the intravascular space that develops acutely and decreases the water content in the brain. Hypernatremia also causes altered sensorium due to cellular dehydration. In this patient, both DKA and hypernatremia contributed to the alteration in sensorium. These were identified and treated timely that improved recovery.

This case report highlights the importance of understanding the management approach required for hypernatremia and DKA to prevent complications associated with these two conditions.

### REFERENCES