Case Report

Diabetic Ketoacidosis with Extreme Hypernatremia in a 13-Year-Old Girl

Dr Vandana Yadav[1], Dr Jaswir Singh[1]

Author's Affiliation:
1- Department of Pediatrics Govt. Medical College/ Rajindra Hospital Patiala 147001, India

Correspondence:
Dr. Vandana Yadav Department of Pediatrics Govt. Medical College/ Rajindra Hospital Patiala 147001, India Email: vandanay19@gmail.com

Received on: 30-May-19

Accepted for Publication: 31-Dec-19

Abstract:

Diabetic Ketoacidosis commonly presents with hyponatremia, but hypernatremia is a rare entity. A 13-year-old girl admitted with altered mental status, known case of type I diabetes mellitus and diabetic ketoacidosis (DKA) had a rapid rise in serum sodium from 155mEq/L (corrected sodium 162mEq/L) at the admission to 193mEq/L within 36 hours of admission despite standard fluid and insulin therapy recommended for the treatment of DKA. The patient was in uncompensated shock. During her illness, renal functions and GCS further deteriorated. The child was kept on non-invasive ventilation with continuous monitoring of vitals and urine output, started on insulin infusion, careful fluid titration to bring down the sodium and glucose gradually and maintain tissue perfusion. She had a prolonged Intensive Care Unit and hospital stay but recovered completely without any neurological sequelae.

Keywords: diabetic ketoacidosis, extreme hypernatremia

INTRODUCTION

In the increasingly expanding population, Diabetes Mellitus accounts for 20-50% of new-onset diabetic patients in young adult population [1]. And Diabetic Ketoacidosis (DKA) continues to be the most severe medical emergency requiring admission to Intensive Care Unit (ICU).The most common sodium change observed in diabetic ketoacidosis (DKA) is hyponatremia with corrected sodium being in the eunatremic range[2,3].Although mild hypernatremia can be seen in 30% of patients presenting with DKA, extremely severe hypernatremia in the range of 190 mEq/L in DKA has been reported very rarely. Here, we present a case of severe DKA in a 13-year-old girl with a serum sodium of 193mEq/L with intact neurological recovery.

CASE REPORT

A 13-year old girl known case of type I diabetes mellitus presented with a history of fever, vomiting, pain abdomen, difficulty in breathing and altered sensorium. The child also had symptoms of polyuria, nocturia and polydipsia for a few weeks prior to admission.

On presentation, the child was drowsy with severe dehydration, had acidotic breathing and was in hypotensive shock with blood pressure (BP) of 70/50 mm Hg, respiratory rate of 25 breaths per minute, heart rate of 134 beats/minute, pulse oximetry of 97% on ambient air, and temperature of 97.6 Fahrenheit. She had a Glasgow Coma scale (GCS) of 9/15 with slightly increased tone with normal reflexes in all the four limbs.

Her blood glucose was >500 mg/dL, and arterial blood gas showed pH 7.03, $pCO_2$36 mmHg, HCO_3 4.3 mEq/L, and a base deficit of -27, with a serum sodium of 155mEq/L (corrected serum Na of 162mEq/L). The

management of DKA was initiated according to the standard protocol. However, serum sodium continued to rise and reached 171mEq/L.

She was given 20 ml/kg of normal saline fluid bolus and vasoactive drug infusions to support her BP. Oxygen was started with mask to correct hypoxia. She was catheterized to monitor urine output. Her dehydration was estimated to be 8%, and she was started on an appropriate volume of fluid given as 0.45 NS. Insulin infusion was started. The free water correction calculated at 4 ml/kg/percent of dehydration was also started to correct her hypernatremia. She was started on intravenous (IV) ceftriaxone.

Investigations revealed Hb 11.9 g%, white blood cell of 19,800/mm^3 with polymorphs of 90%, and platelets 341,000 per microliter. Blood urea nitrogen and serum creatinine were 68 mg/dl and 1.7 mg/dl, respectively. Serum glutamate oxaloacetate transaminase was 12 U/L, serum glutamate pyruvate transaminase was 15 U/L, serum lactate was 1.02 mmol/L (Normal range [N] – 0.9–1.6 mmol/L). The urine ketones were 3+. HbA1c was 13.2% (N – 4%–6%). Despite the free water correction serum sodium rose to 193mEq/L over the next 26 hrs. Her serum osmolality was 356mosm/L, and urine osmolarity was 228 mosm/L.

To minimize the risk of cerebral edema, in addition to the standard on going fluid management of DKA, the free water correction was done slowly to lower her serum sodium gradually by 12 mEq/24 h[4]. The urine output >4 ml/kg/h was replaced hourly by an equal volume of 0.2 DNS intravenously. Serum sodium was monitored two hourly, and fluids titrated to permit a steady fall in serum sodium of 0.5 mEq/h.

0.45% NS then changed to D5 with 0.45%NS when RBS was below 250mg/dl. Insulin infusion was continued. Sodium was further increasing with declining Potassium levels from 3.9mEq/L till 1.2mEq/l correction with KCl was given up to 40mEq/l. Shock was improving till that time there were no signs of arrythmias during hypokalemia, ECG was normal. While on treatment GCS declined to 7/15, because patient was maintaining airway she was not intubated and kept on high flow oxygen under continuous monitoring. On fundus examination, there was slight blurring of nasal margins. Osmotherapy was a confusion because RFTs were deranged-Mannitol was a contraindication, due to hypernatremia-Hypertonic saline was a challenge. Serum Osmolality calculated was 356 mosmol/L and renal functions were deranged so hypertonic saline was given as an osmotic agent.

The inotropes and vasopressors were gradually tapered and omitted. Her acidosis gradually resolved. Serum sodium returned to normal over 6 days falling at a steady rate of 10–12 mEq/day [Figure 1].

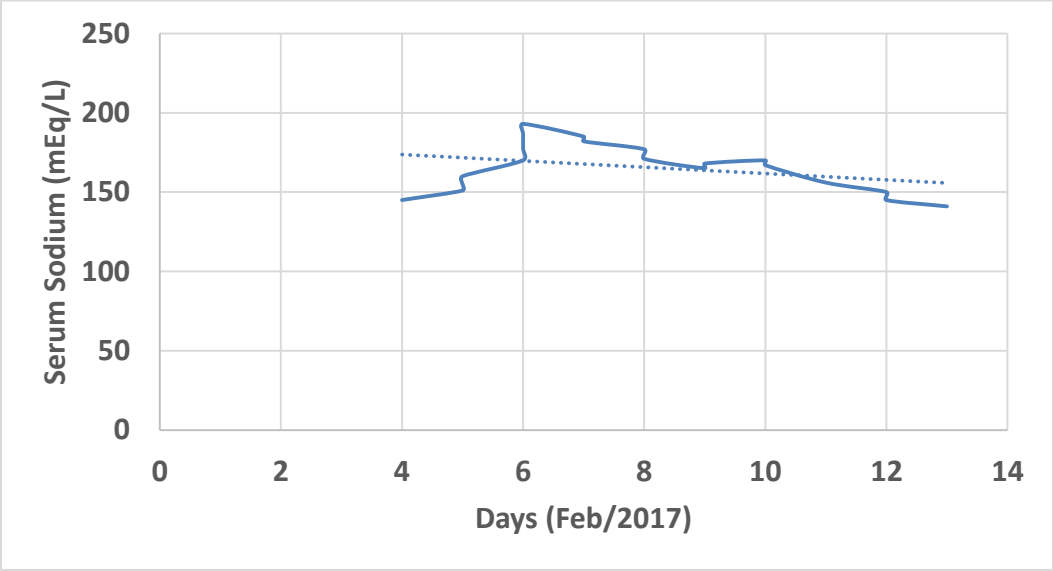

Figure 1 Serum sodium levels over 10 days

Steady sodium correction with target decline of 10–12 mEq/day was achieved by titrating the tonicity of the maintenance fluid, free water deficit correction, and replacing the excessive urinary losses.

Insulin infusion and glucose content of the IV fluid was titrated with a target blood sugar of 150–200 mg/dL. GCS improved to 12/15. Oxygen was omitted on day 5th of treatment. Insulin was changed to subcutaneous route on day 7. She was positive for tissue transglutaminase, but endomysial, antiglutamic acid decarboxylase, and islet-cell antibody titers were negative. She was advised a gluten-free diet. The two-dimensional echocardiogram was normal. Magnetic resonance imaging brain were normal. At 1 month after admission, she was neurologically normal, her diabetes under control.

DISCUSSION

Electrolyte disturbances are common in patients with diabetes and may be the result of an altered distribution of electrolytes related to hyperglycemia-induced osmotic fluid shifts or of total body deficits brought about by osmotic diuresis[1].

Patients with DKA commonly present with hyponatremia on admission to the hospital. Uncontrolled plasma glucose causes increase in plasma tonicity, creating a driving force for the movement of water from intracellular space to extracellular space, which dilutes the extracellular concentration of sodium. In addition, secretion of vasopressin limits water loss via kidney, which all leads to hyponatremia [1, 5], while hypernatremia in DKA occurs from hypotonic renal losses, which is water excretion in excess of sodium and potassium, due to glycosuria-induced osmotic diuresis and inappropriate water replacement [5]. As per consensus statement guidelines, a correction factor of 1.6 mg per deciliter is added to the measured plasma sodium concentration for each 100 mg per deciliter of glucose above 100 mg per deciliter to account for the dilution effect of glucose. Corrected serum sodium provides a handy tool in monitoring and management during acute hyperglycemic crisis [1, 5].

Anecdotal reports of hypernatremia in adolescent children with DKA have been attributed to large intake of carbonated drinks or the use of herbal products[6,7,8]. Our patient had no history of such intake.

The extreme hypernatremia was primarily due to large electrolyte-free water losses associated with osmotic diuresis. This may have got aggravated during therapy because of the large amount of sodium she received in her fluid management according to the standard DKA protocol and the failure to replace the large urinary free water losses.

Extreme hypernatremia is a catastrophic condition that is known to be associated with death or severe neurological sequelae in survivors. The intact neurological survival was probably related to the very gradual reduction in serum sodium to normal levels over almost 8 days using a pathophysiological-based fluid management.

Three cases have been reported till this time with Hypernatremia Hyperosmolarity DKA:

1) A 13 year old Asian boy in November 2014, in Korea [9]
2) A 4 year old girl in September 2017, in Mumbai Maharashtra[10]
3) 44 year old female in April 2018,inNew York USA[11]

REFERENCES

1. J. R. Ingelfinger, B. F. Palmer, and D. J. Clegg, "Electrolyte and Acid–Base Disturbances in Patients with Diabetes Mellitus," The New England Journal of Medicine, vol. 373, no. 6, pp. 548–559, 2015. View at Publisher · View at Google Scholar
2. Wolfsdorf J, Glaser N, Sperling MA. American Diabetes Association. Diabetic ketoacidosis in infants, children, and adolescents: A consensus statement from the American Diabetes Association. Diabetes Care. 2006;29:1150–9. [PubMed]
3. Frier BM, Steer CR, Baird JD, Bloomfield S. Misleading plasma electrolytes in diabetic children with severe hyperlipidaemia. Arch Dis Child. 1980;55:771–5. [PMC free article] [PubMed]
4. Barsoum NR, Levine BS. Current prescriptions for the correction of hyponatraemia and hypernatraemia: Are they too simple? Nephrol Dial Transplant. 2002;17:1176–80. [PubMed]
5. G. Liamis, E. Liberopoulos, F. Barkas, and M. Elisaf, "Diabetes mellitus and electrolyte disorders," World Journal of Clinical Cases, vol. 2, no. 10, pp. 488–496, 2014. View at Publisher · View at Google Scholar
6. McDonnell CM, Pedreira CC, Vadamalayan B, Cameron FJ, Werther GA. Diabetic ketoacidosis, hyperosmolarity and hypernatremia: Are high-carbohydrate drinks worsening initial presentation? Pediatr Diabetes. 2005;6:90–4. [PubMed]
7. Vanelli M, Chiari G, Ghizzoni L, Capuano C, Bonetti L, Costi G, et al. Ketoacidosis and hyperosmolarity as first symptoms of type 1 diabetes mellitus following ingestion of high-carbohydrate-containing fluids. J Pediatr Endocrinol Metab. 1999;12:691–4. [PubMed]
8. Bhowmick SK, Hundley OT, Rettig KR. Severe hypernatremia and hyperosmolality exacerbated by an herbal preparation in a patient with diabetic ketoacidosis. Clin Pediatr (Phila) 2007;46:831–4. [PubMed]
9. Kim HJ, Kim DH, Jun YH. A rare diabetes ketoacidosis in combined severe hypernatremic hyperosmolarity in a new-onset Asian adolescent with type I diabetes. BMJ Case Rep. doi: 10.1136/bcr-2014-208016
10. Arya MK, Ahmed SM, Shah KN. Diabetic Ketoacidosis with Extreme Hypernatremia in a 4-Year-Old Girl.Indian J Crit Care Med. 2017 Sep; 21(9): 610–612. doi: 10.4103/ijccm.IJCCM_226_17
11. Kumar V, Nanavati SM,Melki G. A Case of Diabetic Ketoacidosis Presenting with Hypernatremia, Hyperosmolarity, and Altered Sensorium. https://doi.org/10.1155/2018/4806598