

# Diabetic Ketoacidosis with Extreme Hyponatremia in a 4-Year-Old Girl

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## Abstract

A 4-year-old girl admitted with altered mental status, new-onset diabetes mellitus, and diabetic ketoacidosis (DKA) had a rapid rise in serum sodium from 158 mEq/L (corrected sodium 165 mEq/L) at the admission to 204 mEq/L within 18 hours of admission despite standard fluid and insulin therapy recommended for the treatment of DKA. During her illness, she developed arterial and deep vein thrombosis (DVT), bloodstream infection with *Candida* species, and extensive skin blistering and denudation. The child needed mechanical ventilation, insulin infusion, careful fluid titration to bring down the sodium gradually, and low-molecular weight heparin for her DVT. She had a prolonged Intensive Care Unit and hospital stay but recovered completely without any neurological sequelae.

**Keywords:** Deep vein thrombosis, diabetic ketoacidosis, extreme hyponatremia, skin blisters

## INTRODUCTION

The most common sodium change observed in diabetic ketoacidosis (DKA) is hyponatremia with corrected sodium being in the eunatremic range.<sup>[1,2]</sup> Although mild hyponatremia can be seen in 30% of patients presenting with DKA, extremely severe hyponatremia in the range of 200 mEq/L in DKA has never been reported before. We present a case of severe DKA in a 4-year-old girl with a serum sodium of 204 mEq/L with intact neurological recovery.

## CASE REPORT

A 4-year 4-month-old girl was referred to our hospital for DKA with rapidly rising serum sodium with a history of fever, vomiting, and altered sensorium. The child also had symptoms of polyuria, nocturia and polydipsia for a few weeks prior to admission. Her blood glucose was >500 mg/dL, and arterial blood gas showed pH 7.03, pCO<sub>2</sub> 16.2 mmHg, HCO<sub>3</sub> 4.3 mEq/L, and a base deficit of -27, with a serum sodium of 158 mEq/L (corrected serum Na of 165 mEq/L). The management of DKA was initiated according to the standard protocol. However, serum sodium continued to rise and reached 189 mEq/L.

On admission, the child was drowsy with severe dehydration, had acidotic breathing and was in hypotensive shock with

blood pressure (BP) of 70/50 mm Hg. On the central nervous system examination, she had a Glasgow Coma scale (GCS) of 7/15, hypertonia with brisk reflexes in all the four limbs.

She was given 10 ml/kg of normal saline fluid bolus and vasoactive drug infusions to support her BP. Her dehydration was estimated to be 9%, and she was started on an appropriate volume of fluid given as 0.45 NS. Insulin infusion was continued. The free water correction calculated at 4 ml/kg/percent of dehydration was also started to correct her hyponatremia. In a view of her low GCS, she was mechanically ventilated. The right femoral central venous catheter and left radial arterial line were secured for hemodynamic monitoring. She was started on intravenous (IV) ceftriaxone.

Investigations revealed Hb 12.9 g%, white blood cell of 25,300/mm<sup>3</sup> with polymorphs of 64%, and platelets 529,000 per microliter. Blood urea nitrogen and serum creatinine were 28 mg/dl and 0.83 mg/dl, respectively. Serum

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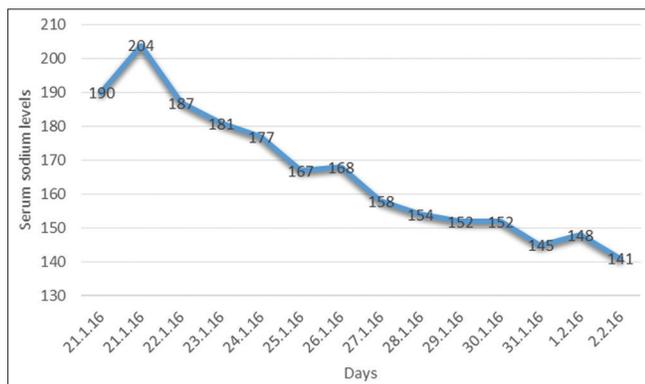
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glutamate oxaloacetate transaminase was 10 U/L, serum glutamate pyruvate transaminase was 10 U/L, serum lactate was 1.02 mmol/L (Normal range [N] – 0.9–1.6 mmol/L), and serum ketones were 3.2 mmol/L (N – 0.05–0.29 mmol/L). The urine ketones were 1+. C-peptide level was 0.08 ng/mL (N – 0.08–3.1 ng/mL), and HbA1c was 16.2% (N – 4%–6%). Despite the free water correction serum sodium rose to 204 mEq/L over the next 6 h. Her serum osmolality was 428 mosm/L, and urine osmolality was 268 mosm/L.

Vasopressin infusion was started for suspected coexistent diabetes insipidus. However, the urine output fell drastically with vasopressin, and so it was omitted. In addition to the standard ongoing fluid management of DKA, the free water correction was done using Madias formula by hourly enteral administration to lower her serum sodium gradually by 12 mEq/24 h.<sup>[3]</sup> The urine output >4 ml/kg/h was replaced hourly by an equal volume of 0.33 DNS intravenously. Serum sodium was monitored two hourly, and fluids titrated to permit a steady fall in serum sodium of 0.5 mEq/h.

She had two episodes of generalized tonic–clonic convulsions which were controlled with phenytoin. In view of the possibility of a coexisting viral encephalitis, IV acyclovir was added empirically. On day 3 of admission, the child developed thrombus in the left radial artery. The arterial line was removed, and the patient was started on low-molecular-weight (LMW) heparin in the therapeutic dosage. Later, she developed gangrenous change of the tip of the left index finger. She also developed extensive superficial and deep venous thrombosis with Doppler suggestive of thrombosis in the right external iliac vein, common femoral, proximal superficial, and deep femoral vein. Her prothrombin time was 12.6 s with international normalized ratio 1.26, partial thromboplastin time 36 s, serum fibrinogen of 211 mg/dL (N – 150–400 mg/dL), and D-dimer of 8749 microgram/L (N – <500 µg/L). The femoral catheter was removed, and right internal jugular venous catheter was placed. The inotropes and vasopressors were gradually tapered and omitted. The two-dimensional echocardiogram and magnetic resonance imaging brain were normal. Her acidosis gradually resolved. Serum sodium returned to normal over 7 days falling at a steady rate of 10–12 mEq/day [Figure 1].



**Figure 1:** Serum sodium levels over the first 2 weeks

On day 4 of admission, she developed large blisters and denudation of skin over the buttocks, groin, right forearm, and right lower limb [Figure 2].

On day 6, she developed fever with a progressive fall in platelet count to 8000/µL. Blood and urine culture grew *Candida albicans* and *tropicalis* and was treated with fluconazole as per sensitivity report. Acyclovir was omitted. The blisters gradually resolved. The child was extubated on day 10 and put on high-flow humidified nasal cannula from which she was gradually weaned off.

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. LMW heparin was continued till the resolution of arterial and venous thrombosis. She was positive for tissue transglutaminase, but endomyesial, antiglutamic acid decarboxylase, and islet-cell antibody titers were negative. She was advised a gluten-free diet. At 1 month after admission, she was neurologically normal, her diabetes under control with complete healing of her skin and total resolution of the arterial and venous thrombosis.

## DISCUSSION

Anecdotal reports of hyponatremia in adolescent children with DKA have been attributed to large intake of carbonated drinks or the use of herbal products.<sup>[4–6]</sup> Our patient had no history of such intake.

None of these patients was as young as our patient or had such extreme hyponatremia. Due to the early onset of DKA, extreme hyponatremia, and polyuria, Wolfram syndrome with diabetes insipidus, diabetes mellitus, and optic atrophy was considered as a possibility. However, the absence of optic atrophy as well as the total disappearance of polyuria after recovery made the diagnosis unlikely.

The extreme hyponatremia was primarily due to large electrolyte-free water losses associated with osmotic diuresis. This may have got aggravated during therapy because of the



**Figure 2:** Blisters on forearm

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.

Deep vein thrombosis associated with femoral vein catheterization has been reported in diabetics, and cannulation of central veins should preferably be avoided. In our patient, the unstable hemodynamic state necessitated both arterial and femoral vein cannulation. The extremely hyperosmolar state resulting from hyperglycemia with added hyponatremia was probably responsible for the arterial as well as the extensive deep and superficial vein thrombosis in our child.<sup>[7,8]</sup> Fatal transverse sinus thrombosis has been described in a 2-week-old baby with a serum sodium >200 mEq/L.<sup>[9]</sup> The current recommendations for DKA do not support the use of prophylactic anticoagulation.

Blistering has been reported with acyclovir use, and in our patient, new blisters stopped appearing after omission of acyclovir.<sup>[10]</sup>

Extreme hyponatremia 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 10 days using a pathophysiological-based fluid management.

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#### Conflicts of interest

There are no conflicts of interest.

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