Hyperosmolar Diabetic Ketoacidosis in Two Adolescents with New Onset Type 2 Diabetes Mellitus

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Abstract

We present the unique cases of two adolescent patients who presented to a pediatric tertiary care facility in the Midwest United States with newly diagnosed type 2 diabetes mellitus, complicated by the combination of diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS). Each case required intensive therapy with treatment consisting of replacement of fluids over a prolonged period of =>72 hours and careful monitoring of electrolyte response. Complications included acute renal failure, superficial thrombosis, and rhabdomyolysis. The two cases made a complete recovery without neurological sequelae. Both cases highlight the complexity of

Corrected Sodium, mmol/L	164.3	148	(136-145)
Potassium, mmol/L	5.7	4.8	(3.5-5.3)
BUN, mg/dL	35	16	(6 -23)
Creatinine, mg/dL	1.91	1.37	(0.2-0.5)
B-hydroxybutyrate , mmol/L	N/A	11.6	(0.02-0.27)
Insulin ,uIU/mL	1	5	(3-19)
Altered mental status	Yes	Yes	
Length of stay, days	14	6	

Unfortunately, on the same night, he developed a fever of 39.3 C, with rapid deterioration, and developed septic shock. He was treated with fluid resuscitation and intravenous broad-spectrum antibiotics, and required inotropes for a total of 20 hours. Blood culture reported Enterobacter Cloacae. In addition, he developed a superficial right cephalic vein thrombus. After improvement in clinical status, he was switched back to SC insulin and was transferred to the general pediatrics floor to continue his IV antibiotic course. He was discharged on metformin and SC insulin; total daily dose (TDD) 0.7 U/kg/day [2].

Materials and methods

A 12-year male with a past medical history of epilepsy and asthma presented to the ED with symptoms of fatigue and altered mental status. Patient was obese and found to have acanthosis nigricans, and elevated liver enzymes. He was clinically diagnosed with type 2 diabetes mellitus, which was later confirmed with negative GAD-65, insulin and islet cell antibodies. At presentation, he was hemodynamically stable. A head CT scan was negative for intracranial abnormalities or cerebral edema. He was noted to have hyperglycemia (serum glucose 981 mg/dl), acute kidney injury (serum creatinine 1.37 mg/dL) hypernatremia (corrected Na 148 mmol/L, measured 134 mmol/L), hyperosmolarity (serum Osmolarity 372 mOsm/kg), and acidosis (pH 7.1), consistent with both DKA and HHS (Table 1). Fluid resuscitation with 10 ml/kg 0.9% saline was given, followed by

normal saline may be used for urine replacement. In contrast, the ADA guidelines recommend 0.45– 0.9% saline according to serum osmolality, infused at a rate of 1.5 times maintenance requirements over a 48-hour timeframe (8, 9). Both guidelines agree that isotonic fluids should be re-started if at any point there is hemodynamic instability. A rapid rate of decline in serum sodium should be avoided; 0.5 mmol/L/h has been recommended for hypernatremic dehydration. Frequent sodium concentration measurements and fluid adjustments are mandatory to avoid a rapid sodium decline.

Insulin treatment is necessary to resolve ketosis in these patients and continuous insulin infusion should be started after the initial fluid bolus(es). Insulin drip adjustment is recommended to achieve a decline in serum glucose between 50-100 mg/dl per hr. However, re-assessment of fluid and renal function should be carried out before any adjustment. ISPAD guidelines recommend IV insulin of 0.05-0.1 U/kg/hr depending on degree of acidosis. ADA guidelines recommend IV insulin dose at 0.1 U/kg/hr. It should be emphasized that a drop in glucose of over 100 mg/dL/hour may precipitate shock, so close monitoring of the glucose is essential. Often, the glucose will be above the upper limit of detection on a bedside blood glucose meter or even a blood gas, making adjustment of fluids and the insulin drip much more challenging. Close communication with the laboratory to have rapid turn-around of the serum glucose measurement is key to proper management.

Here, we report two patients who presented with a mixed DKA/HHS picture. Impaired cognition was seen in both of them with no evidence of cerebral edema. We believe that the acute encephalopathy was due to the hyperosmolar state. In both patients, mental status improved after hyperosmolarity was corrected. The incidence of clinically overt cerebral edema is 0.5% to 0.9%, and the mortality rate is 21%-24%. Cerebral edema is less common in pediatric HHS cases due to less hypocapnic cerebral vasoconstriction (5). The risk of cerebral edema is higher in mixed DKA/HHS cases than in those with a classical presentation of HHS. Therefore, early vigilant monitoring for fluid replacement and mental status is essential to prevent cerebral edema. However, fluid replacement should never be withheld for fear of cerebral edema, and its importance in preventing complications cannot be emphasized enough (8).

We encountered rhabdomyolysis in ; it is one of the complications that is well documented in both DKA and HHS, but it is more common in HHS. Our patient progressed to rhabdomyolysis shortly after admission, he had depleted intracellular phosphate stores, and extreme hyperosmolar status, acidosis and hyperglycemia; all are known risk factors to develop rhabdomyolysis (10). Hence, ISPAD guidelines recommend monitoring serum CK values in children with HHS every 2-3 hours, as rhabdomyolysis can worsen acute renal failure and precipitate electrolyte abnormalities, cardiac arrhythmias, and even muscle compartment syndrome (8). The severity of dehydration may have been underestimated due to his obesity and serum hypertonicity. Therefore, early and aggressive fluid resuscitation and intensive monitoring are mandatory to prevent this complication.

The other complication we encountered in case #1 was superficial thrombosis at site of peripheral line at day 6 of admission. On admission, Enoxaparin sodium was not a viable option due to impaired renal function. Therefore, he was started on SC heparin for DVT prophylaxis. Unfortunately, he developed thrombocytopenia at day 5 of admission, so SC heparin was discontinued. It is known that diabetes is a hypercoagulable state, explained by endothelial abnormalities and smooth muscle dysfunction favoring coagulation cascade activation (11). In addition, severe dehydration and hypertonicity can activate the coagulation pathway and cause venous stasis. Deep vein thrombosis has been reported in critically ill children who required central venous catheter placement (11, 12). Thromboembolic complications occur commonly in HHS and heparin