CASE REPORT

A rare diabetes ketoacidosis in combined severe hyponatremic hyperosmolality in a new-onset Asian adolescent with type I diabetes

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SUMMARY

A 13-year-old Asian boy presented with an 8 h history of lethargy and vomiting. He had a 3-week history of polyuria, polydipsia and a 6 kg weight loss over a period of 1 month. Fluid intake prior to admission was over 6 L of sports drinks and cola per day. Initial biochemical findings were as follows: plasma glucose 1351 mg/dL, serum sodium 154 mEq/L, serum osmolality 425 mOsm/L, arterial blood pH 6.96 and urine ketone of 3+. He was treated with intensive fluid resuscitation and an insulin infusion. He completely recovered without any neurological deficits. Severe hyponatremia is rare in diabetic ketoacidosis (DKA) but was exhibited in this case. Excess intake of carbonated carbohydrate-rich beverages may exacerbate the initial severe presentation of type I diabetes mellitus (T1DM). To the best of our knowledge, this is the first case of an Asian child with DKA combined with severe hyponatremic hyperosmolality at onset of T1DM.

BACKGROUND

Diabetic ketoacidosis (DKA) is the leading cause of morbidity and mortality in children with type I diabetes (T1DM).¹ It is diagnosed in up to 25% of children following a new diagnosis of T1DM.

A hyperosmolar state also presents in patients with hyperglycaemic diabetes due to increased glucose production and limited glucose utilisation. A hyperglycaemic hyperosmolar state usually leads to hyponatremia or eunatremia. Although mild hyponatremia could be present in patients who also exhibit as severely dehydrated DKA, the severe degree of hyponatremia is rarely presented.

There are few case reports presenting DKA combined with severe hyperosmolar hyponatremic states. It is suggested that the occurrence of hyponatremia at new-onset T1DM with DKA may be attributed to an excess consumption of carbohydrate-rich beverages to relieve thirst.² Similar cases are more likely to increase in number secondary to increased incidence of childhood obesity and increased intake of sugar-rich drinks.

We report a rare case of an Asian boy who presented with DKA complicated by severe hyperosmolar hyponatremia at onset T1DM.

CASE PRESENTATION

A previously healthy 13-year-old Korean boy was brought to our emergency department for lethargy of 8 h duration and vomiting for 2 days. He had a 3-week history of increasing polyuria and

polydipsia, and weight loss of 6 kg over the previous month. He was an avid soccer player and participated in the school soccer match for 3 days prior to his admission. His average daily fluid intake was over 6 L of isotonic sports drinks and cola. There was no parental history of diabetes. At presentation he was drowsy and responsive to pain but responded poorly to verbal stimuli. His height was 170 cm (>75th centile) and his weight was 48 kg (<50th centile). His body mass index was 16.6 kg/m². He had a blood pressure of 100/60 mm Hg, heart rate 120 bpm, respiration rate 24 breaths/min and a temperature 36.4°C. He was severely dehydrated with poor skin turgor and marked delay in capillary refill >4s.

INVESTIGATIONS

Initial biochemical findings were as follows: plasma glucose level 1351 mg/dL, serum sodium level 154 mEq/L, serum osmolality 425 mOsm/L, serum potassium 4.6 mEq/L, TCO₂ 5.1 mEq/L, blood urea nitrogen 64.7 mg/dL, serum creatinine 2.6 mg/dL, HbA1c 12.5%, c-peptide 0.08 ng/mL and insulin at 2.6 µU/mL. His arterial blood gas analysis showed a pH at 6.96, PCO₂ 20 and PO₂ 96. Ketone bodies in his serum and urine were strongly positive. The findings of brain MRI revealed a normal range without any evidence of cerebral oedema.

DIFFERENTIAL DIAGNOSIS

Given an abnormally high serum sodium and osmolality in the presence of high blood glucose and severe dehydration, central diabetes insipidus combined with diabetes mellitus could be considered as the diagnosis.

TREATMENT

After initial fluid resuscitation with 2 L of normal saline, a continuous insulin infusion was begun. The patient was rehydrated via fluid therapy on the basis of a calculated free water deficit. Clinical and biochemical monitoring were carefully performed during his intensive treatment.

OUTCOME AND FOLLOW-UP

Plasma glucose concentration improved steadily after the initial fluid therapy, whereas serum sodium and osmolality values decreased slowly over 72 h (figure 1). The patient showed an alert and normal response to verbal stimuli after 48 h of management. All other laboratory values were

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Figure 1 Change of serum glucose level (blue-line) and sodium concentration (red-line) during the first 5 days of treatment. With intensive fluid therapy and continuous insulin infusion, the hyperglycaemia and hyponatremia gradually improved.

within normal ranges. He completely recovered without any neurological deficit. After a 16-day hospital stay, he was discharged 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). In a clinical survey,³ a combined diagnosis of DKA and HHS (DKA-HHS) was noted in 30% of adults with DKA and/or HHS subjects with diabetes. According to the diagnostic criteria for the DKA-HHS definition, such as pH ≤ 7.30 , $\text{HCO}_3^- \leq 15$ mmol/L and serum osmolality ≥ 330 mOsm/L, our patient could also be diagnosed as DKA-HHS. Conversely, our case was different from other DKA-HHS cases in that associated marked hypernatremic hyperosmolality was present.

There have been few reports of combined DKA and hypernatremic hyperosmolality. One recent report² attributed the occurrence of hypernatremia at new-onset T1DM in patients with DKA to a huge daily intake of carbonated carbohydrate beverages. In another report⁴ a DKA child presented with severe hypernatremia and hyperosmolality due to herbal product ingestion. In our case, as the patient had played school soccer games for 3 days before admission, he consumed a high volume of sports drinks and cola daily to quench his thirst. These drinks usually contain large amounts of sugar, high sodium and have high carbonate content. The patient's history of high fluid consumption of carbonated, carbohydrate and sodium-rich content may have exacerbated a more severe presentation at the onset of T1DM. Worsening of hyperglycaemia aggravates the osmotic diuresis with increase in loss of free water. Replenished oral intake of high sugar/high sodium fluid is likely to result in a hypernatremic hyperosmolality state.

Similar cases of the combination of DKA/HHS with hypernatremia may become more frequent with wider use of sugar-rich drinks at the onset of DKA. Also, the incidence of childhood

obesity and T2DM is increasing worldwide. So the evaluation of the type of fluid and quantity of intake is needed to manage affected cases at new-onset DM.

A major severe complication of DKA is cerebral oedema. Hyponatremia can cause severe cellular dehydration within the central nervous system, which may result in an altered level of consciousness. Furthermore, a too rapid correction of serum sodium can precipitate cerebral oedema with the potential complications of seizures, cerebral herniation and death.⁵ In our case, hypernatremic dehydration was successfully corrected over 48–72 h without neurological complications.

To the best of our knowledge, this is the first case of an Asian child with severe hypernatremia at new-onset of T1DM with DKA. Ingestion of larger amounts of fluid with carbonated carbohydrate-rich content appeared to precipitate a more severe metabolic derangement in regard to DKA.

Learning points

- High fluid consumption of carbonated, carbohydrate-rich content may exacerbate more severe metabolic derangement in diabetic ketoacidosis (DKA) at new-onset type-I diabetes mellitus (T1DM) in patients.
- The type and amount of fluid consumption need to be evaluated in sick patients presenting with DKA at new-onset of DM.
- Fluid therapy for DKA with hypernatremic hyperosmolality should be achieved gradually to avoid cerebral oedema with continuous careful monitoring.

Contributors DHK was involved in treating the patient. YHJ gave clinical support in the care of the patient.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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