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Extreme Hyperglycemia and Hyperosmolar State in New Onset Type 1 Diabetes: Are Sugar- and Salt-Containing Beverages at Fault?

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Introduction

The hyperglycemic hyperosmolar state (HHS) is an acute complication of diabetes mellitus generally thought to occur in individuals with type 2 diabetes mellitus (T2DM) and usually in older patients who are dependent on others for support.¹ Although HHS is infrequently encountered in children, it is associated with a mortality rate of approximately 15%.² Some reports have implicated high-carbohydrate-containing beverages in causing exaggerated hyperglycemia and hyperosmolarity, resembling the clinical and laboratory picture of HHS, in the acute presentation of type 1 diabetes mellitus (T1DM).^{3,4} With the high case fatality rates associated with HHS, patients with diabetes presenting in a similar manner require prompt identification to ensure adequate fluid replacement and gradual correction of serum osmolarity.⁵ The following cases describe 3 children seen at our institution with new onset T1DM and severe hyperglycemia, hypernatremia, and hyperosmolarity. Although these cases are not classic for HHS due to the presence of ketones,⁵ the clinical presentation and other biochemical abnormalities are comparable. Each child required a prolonged hospital stay and intensive fluid management to correct fluid and electrolyte abnormalities. The clinical and biochemical characteristics of all 3 cases are described in Table 1. The potential influence of high-carbohydrate- and high-sodium-containing beverages is discussed.

Case Reports

Case 1

A 9.6-year-old prepubertal white male presented with a week history of flu-like symptoms. Parents noted polydipsia, polyuria, and vomiting 2 days prior to admission. He drank 4 liters of soft drinks in a 14-hour period, resulting in consumption of approximately 217 g of carbohydrates and 392 mg of sodium. He was noted to have hyperglycemia, hypernatremia, hyperosmolarity, and acidosis (Table 1).

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He initially received 1 liter of normal saline and was then started on 5% dextrose in half normal saline at maintenance. He was also placed on an insulin drip at 0.1 U/kg/h and transported to our medical center. At our hospital, his serum glucose was 756 mg/dL and serum sodium was 159 mEq/L. He had acute neurologic changes, including decreased activity, impaired cognition, and incoherent speech. One dose of mannitol was administered, and a head computed tomography showed no intracranial abnormalities or cerebral edema. After 16 hours of IV fluids (IVF), he was switched to 5% dextrose in quarter normal saline at 1.5 times maintenance to correct his hypernatremia.

After his neurologic status returned to baseline, he was transferred to the pediatric floor. Serum sodium was 156 mEq/L, serum glucose was 163 mg/dL, and acidosis had resolved. He was started on subcutaneous insulin and allowed to eat but was maintained on IVF for another 24 hours. IVF were discontinued on the fourth day of admission when his serum sodium was 137 mEq/L. In total, he received 6100 mL (175 mL/kg) of IVF. At his follow-up clinic visit, he did not demonstrate any neurologic sequelae.

Case 2

A 13.2-year-old pubertal white male presented with a 5-day history of polyuria, polydipsia, and 20-pound weight loss. Prior to arriving at the hospital, he consumed 196 ounces of sugar-containing beverages, including regular soda, root beer, orange soda, and sweetened tea. This resulted in approximately 500 g of carbohydrates and 529 mg of sodium consumed in a 24-hour period. He had hyperglycemia, hypernatremia, and hyperosmolarity, but not acidosis (Table 1). He was neurologically stable.

He initially received 1 liter of normal saline and was started on 5% dextrose in half normal saline at maintenance. He was also placed on an insulin drip at 0.1 U/kg/h. Four hours later, his serum glucose was 356 mg/dL and corrected serum sodium was 148 mEq/L. He was given another liter of normal saline, was started on subcutaneous insulin, and ate a regular diet. He continued to receive IVF hydration of half normal saline at maintenance for an additional 24 hours. Fluids were discontinued when his serum sodium reached 147 mEq/L. During the hospital course, his total IVF administration was 4300 mL (110 mL/kg).

Case 3

A 15-month-old white male presented with a 1-month history of increasing polyuria and polydipsia. Parents reported that in the days prior to admission, he consumed at least 44 ounces per day of regular soda, sports drinks, and apple juice, resulting in an estimated daily intake of 451 g of carbohydrates and 56 mg of sodium. Approximately 48 hours prior to presentation, his fluid intake decreased, and he began vomiting multiple times per day. At presentation, he was hyperglycemic, hypernatremic, hyperosmolar, and acidotic (Table 1).

He initially received a 20 mL/kg bolus of normal saline, and he was transferred to our medical center. He was started on an insulin drip at 0.03 U/kg/h and normal saline at twice his maintenance rate. Because of the level of hyperosmolarity, he was switched to 5% dextrose in half normal saline 4 hours after initial IVF resuscitation. Although initially responsive, he had waxing and waning mental status, which stabilized after normalization of

his electrolytes. He spent an additional 24 hours in the intensive care unit for observation because of his mental status changes, hyperosmolarity, and young age.

After 36 hours of IVF administration, his serum sodium was 146 mEq/L. He received a total volume of 2400 mL (300 mL/kg) of IVF, and he was switched to subcutaneous insulin and ate a regular diet. After 6 days of hospitalization, he was discharged home. There was no evidence of permanent neurologic abnormalities at hospital discharge or follow-up clinic visits.

Discussion

Extreme hyperglycemia and hyperosmolarity is a potentially life-threatening complication in individuals with new onset diabetes. Although more commonly seen in T2DM, children with T1DM can also have a clinical picture similar to HHS.⁵ In this report, we describe 3 children with new onset T1DM who presented with hyperglycemia, hypernatremia, and severe hyperosmolarity after ingesting large amounts of carbohydrate containing fluids. Previous reports have described similar scenarios associated with T1DM in adolescents who had free access to high-carbohydrate-containing fluids.^{3,4} Interestingly, our third case involved a 15-month-old male who relied solely on his family for fluid replacement. Our experience demonstrates that even toddlers with new onset T1DM, who ingest sugar- and salt-containing fluids, are also at risk for developing the same degree of hyperosmolarity as seen in older children and adolescents.

In our case series, 2 patients presented in diabetic ketoacidosis (DKA), and both had acute mental status changes. It is unclear if these acute neurologic changes were due to cerebral edema or from the hyperosmolar state. Both metabolic conditions are associated with acute neurologic changes.² Hyperosmolarity may also be a harbinger for unusual complications in the initial presentation of both T1DM and T2DM, including rhabdomyolysis,² intestinal ischemia,⁶ and cerebral thrombosis.⁷ Fortunately, none of our patients developed these severe complications despite profound hyperosmolar states.

The reason for the development of extreme hyperosmolarity and hypernatremia in some patients with T1DM may be 2-fold. First, hyperglycemia in children with new onset T1DM results in an osmotic diuresis. Without adequate oral rehydration, hyperosmolarity and hypernatremia may escalate. Second, the ingestion of high-sugar- and high-salt-containing fluids may exacerbate the preexisting hyperglycemia and hyperosmolarity. Although all our patients consumed large amounts of sugar- and sodium-containing fluids prior to presenting to the hospital, the total amount of ingested sodium was much less than the dietary reference intake (DRI) of maximum sodium requirements.⁸ In contrast, the amount of carbohydrates ingested for each child was greater than the DRI maximum recommended carbohydrates for age (Table 2). Therefore, dehydration due to hyperglycemia appears to play a more significant role in the development of hyperosmolarity as compared with the actual amount of sodium ingested. Because of their hyperosmolar state, all our patients required large volumes of fluid resuscitation, which is not typically the case in new onset diabetes or in the treatment of DKA. Importantly, all our patients recovered without any permanent morbidity.

In conclusion, it is important for health care providers to be aware that children with new onset T1DM can present with clinical and laboratory findings that are similar to HHS, specifically in those patients who consume high-carbohydrate-containing beverages. Because the consumption of high-carbohydrate beverages, such as juices and soft drinks, continues to increase in popularity⁹ and can account for up to 15% of the daily calories consumed by children and adolescents,¹⁰ an extreme hyperglycemic and hyperosmolar state may be more frequently observed in patients with new onset T1DM. The initial evaluation of children with new onset diabetes is often performed by their pediatrician. Thus, in a child with new onset diabetes who presents with such a clinical picture, it is important to address hydration status and biochemical abnormalities. Based on our experience and review of the literature,^{3–5} aggressive hydration and appropriate fluid replacement may reduce complication rates and improve clinical outcomes.

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Table 1

Clinical and Biochemical Characteristics of 3 Boys at Presentation With New Onset Type 1 Diabetes Mellitus

	Case 1	Case 2	Case 3	Reference Range
Age (years)	9.6	13.2	1.25	
Weight (kg)	34.4 (75th percentile)	41.7 (20th percentile)	8.0 (10th percentile)	
Tanner Stage	Stage I pubic hair, prepubertal testes	Stage I pubic hair, 8 mL testes	Stage I pubic hair, prepubertal testes	
Urine ketones	Large	Large	Large	Negative
Bicarbonate (mg/dL)	10	21	13	22–29
pH	7.23	7.32	7.25	7.35–7.45
Sodium (mEq/L)	145	135	134	135–145
Corrected sodium (mEq/L)	169	149	173	135–145
Chloride (mEq/L)	96	96	101	95–105
Glucose (mg/dL)	1520	1024	1857	65–110
Blood urea nitrogen (mg/dL)	94	16	40	5–20
Creatinine (mg/dL)	4	0.9	1.25	0.4–1.5
Calculated serum osmolarity (mOsm/kg)	405	333	374	280–305

Table 2

Amount of Carbohydrates and Sodium Ingested by Each Boy in Comparison With the Daily Maximum Requirements of Sodium and Carbohydrate Intake Based on Age-Based Dietary Reference Intake Requirements⁸

	Carbohydrates Ingested (g)	Maximum Recommended Carbohydrate Intake (g)	Sodium Ingested (mg)	Maximum Recommended sodium Intake (mg)
Case 1	208	130	376	2200
Case 2	500	130	529	2200
Case 3	451	130	56	1500