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A CHILD WITH ALTERED SENSORIUM, HYPERGLYCEMIA, AND ELEVATED TROPONINS

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□ **Abstract—Background: Diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) are potentially life-threatening complications of diabetes mellitus. Although DKA and HHS share similar features, they are distinct clinical entities requiring different treatment measures. Objective: This case illustrates that the clinical distinction between these two entities can be difficult at times, especially in children who can present with an overlapping picture. Case Report: We report an interesting case of a 12-year-old whose initial presentation of diabetes was a mixed picture of hyperosmolar DKA and HHS coma complicated by myocardial strain and acute renal insufficiency. The myocardial strain resolved completely with resolution of the metabolic abnormalities. Conclusions: Emergency physicians should be cognizant of varied presentations of hyperglycemic emergencies in children to initiate appropriate management for better outcomes. Published by Elsevier Inc.**

□ **Keywords—hyperglycemia; troponin; altered sensorium; children**

INTRODUCTION

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are life-threatening complications of diabetes mellitus (DM). Even in the presence of well-defined diagnostic criteria, clear distinction between the two entities can be challenging. We describe the case of a young child with a combined picture of DKA and HHS presenting in altered sensorium with elevated troponins. We will review the diagnostic criteria for DKA and HHS and the management of these hyperglycemic emergencies, along with discussion of risk factors and clinical features of cerebral edema and the nature of acute myocardial injury in association with DM in children.

CASE REPORT

A previously healthy 12-year-old Caucasian male presented to the emergency department (ED) with the chief complaint of altered mental status. He had 4-day history of nonbloody, nonbilious vomiting with one to two episodes per day. There was progressive deterioration in his sensorium during a period of 12 h. On the day of admission, he appeared obtunded and was unable to ambulate, so the parents brought him to the ED. There was no history of fever, abdominal pain, diarrhea, headache, seizures, recent weight change, trauma, or any other systemic symptoms. There was no history of witnessed ingestions. Review of symptoms was pertinent for polydipsia, polyuria, and increased fatigability during the previous 2 weeks. Medical and surgical history were significant only for repair of cleft palate. Family history was positive for type 1 DM in a paternal uncle and hypothyroidism in the mother.

On examination, he was ill-appearing, cachectic (weight 29.5 kg, < 5th percentile) and lethargic with minimal response to verbal stimuli. His temperature was 37.2°C, heart rate 162 beats/min, respiratory rate 27 breaths/min, blood

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pressure 103/65 mm Hg, and pulse oximetry 96% in room air. He had a Kussmaul breathing pattern and appeared severely dehydrated with pasty oral mucosa. He had sunken eyes with equal and reactive pupils. His lungs were clear and he had marked tachycardia with normal heart tones. The abdomen was soft and nondistended without any masses or organomegaly. His capillary refill was prolonged. There were no rashes or acanthosis nigricans noted. He had no focal neurologic deficits.

The patient's blood sugar was critically high on bedside testing. New onset DKA was suspected and i.v. access was established. Initial laboratory studies included a capillary blood gas that showed metabolic acidosis with a pH of 7.21, PCO_2 28 mm Hg, PO_2 75.2 mm Hg, bicarbonate 12 mmol/L, and lactate of 2.9. White blood cell count was elevated at 24,000/mm^3 with a left shift; hemoglobin 16.8 g/dL and hematocrit 52.6% indicating significant hemoconcentration. Serum glucose was critically elevated at 1603 mg/dL. Serum electrolytes revealed hypernatremia, metabolic acidosis, and prerenal azotemia with sodium of 147 mmol/L (corrected serum sodium 171 mmol/L), potassium 4.8 mmol/L, chloride 98 mmol/L, bicarbonate 11 mmol/L, blood urea nitrogen (BUN) 92 mg/dL, and creatinine 4.2 mg/dL. His serum osmolality was significantly elevated at 452 mOsm/kg and his serum B-hydroxybutyrate level was 11.63 mg/dL. Urine analysis revealed glucose > 1000 mg/dL and 1+ ketones. His urine drug screen was negative and computed tomography of the head was negative for edema, hemorrhage, mass effect, or any other acute process.

Given this patient's clinical picture with severe hyperglycemia, hyperosmolarity, severe dehydration with only relatively mild metabolic acidosis and ketoacidosis, the patient was diagnosed with having a mixed picture of HHS and DKA.

Routine lead II electrocardiogram (ECG) showed ST changes prompting a 12-lead ECG (Figure 1), which showed diffuse ST changes and T-wave inversion in both limb and precordial leads suggestive of a strain pattern. Subsequently troponin I levels were obtained that were elevated at 1.230 ng/mL (normal high < 0.057 ng/mL).

Treatment was initiated with a 20 mL/kg fluid bolus of 0.9% NaCl. In view of the extremely elevated blood sugar and hyperosmolarity, insulin infusion was started at a rate of 0.05 U/kg/h and the patient was transferred to the pediatric intensive care unit. Infusion of 0.45% NaCl with potassium was continued at maintenance plus 10% deficit, which was gradually corrected over 48 h in view of the hyperosmolality. Urine output remained > 1 mL/kg/h. Serum glucose decreased at approximately 100 mg/dL/h accompanied by a gradual decrease in serum osmolality. In the initial 48 h, despite improvement in his biochemical parameters, there was no notable improvement in his mental status. Magnetic resonance imaging scan of his brain was performed and reported normal.

In view of elevated troponin and ECG changes, an echocardiogram was done, which showed normal cardiac anatomy and function. The ECG changes and elevated troponin were believed to be secondary to metabolic abnormalities rather than an acute coronary syndrome and no additional intervention was recommended.

Other laboratory studies, including thyroxine, thyroid-stimulating hormone, insulin, and islet cell antibodies were normal. He had low C-peptide levels with elevated glutamic acid decarboxylase antibody levels consistent with type 1 DM. His hemoglobin A1C was elevated at 12.2 %.

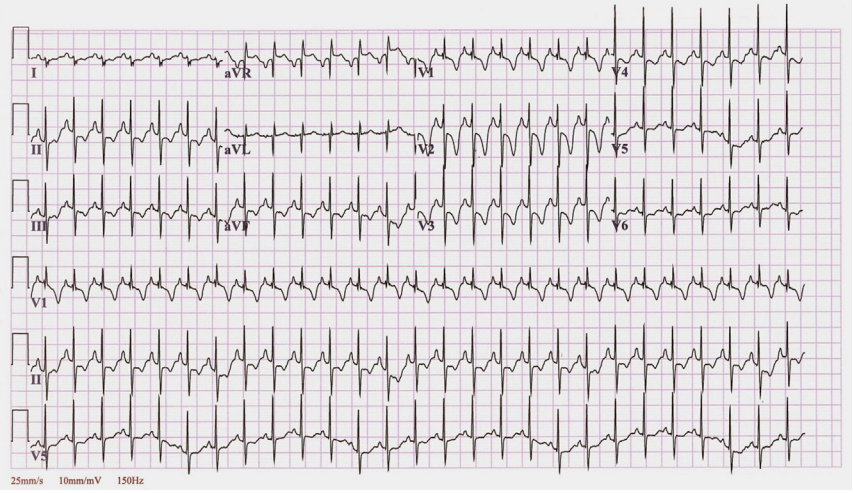

Figure 1. Electrocardiogram at presentation suggestive of myocardial strain pattern: Sinus tachycardia with ST-segment depression and T-wave inversion seen in II, III, aVF and V2-V4 and T-wave inversion in V5-6 with no pathologic Q-waves or ST-segment elevation.