Introduction

Diabetic ketoacidosis (DKA) is a common presentation of insulin-dependent diabetes mellitus (IDDM), and it is the most common reason for pediatric intensive care unit (PICU) admission in children with IDDM. Complications can be related to the disease itself, or to the management, mainly due to fast correction of the hyperglycemia. Common complications include cerebral edema, electrolyte disturbances and hypoglycemia. Acute respiratory failure might occur and can be due to various factors, including myocardial dysfunction.

Case presentation

A 5-year-old, previously healthy Jordanian girl presented to the emergency department (ED) of a private hospital with history of nausea, vomiting and decreased activity. The parents noticed polyuria, polydipsia and weight loss for 2 months. She was found to have severe DKA with a pH of 6.9. She was given 300 mL (10 mL/kg) of intravenous (IV) normal saline bolus, and then insulin drip and hydration were started. After that she was transferred to our hospital.

On arrival to our ED, her initial blood gas values were as follows: pH 6.92, CO₂ 18.2, HCO₃ 3.7 mmol/L and base deficit of 27.8 mmol/L. Her first blood glucose reading was 20.1 mmol/L. On physical examination, her Glasgow Coma Scale (GCS) was 10/15. She was tachypneic with a saturation of 100% while breathing via a non-rebreather mask. She was tachycardic (heart rate (HR) = 130 s) with normal blood pressure and good peripheral perfusion. Her abdomen was soft, non-distended with normal bowel sounds. Genital examination showed vulvovaginitis. Her weight was 30 kg, and her height was 123 cm. She had no previous medical issues and was not taking any medications. She had no known family history of endocrine or cardiac diseases.

She was admitted to PICU and started on the DKA treatment protocol. Insulin infusion was started at a rate of 0.1 U/kg/h and IV fluids composed of dextrose 5% with normal saline and potassium phosphate at a total rate of 100 mL/h, which is around 150% of her maintenance rate. Due to the decreased level of consciousness, head computed tomography (CT) scan was done and showed signs of increased intracranial pressure (Image 1). Mannitol (1 g/kg) was given and she received hypertonic saline (5 mL/kg) and repeated as needed, targeting a sodium level of 150–160 mmol/L. She was given phenobarbitone for seizure prophylaxis. Her total fluid rate was reduced to the maintenance rate of 70 mL/h. Blood and urine cultures were done and piperacillin/tazobactam was started. Her electrolyte levels are shown in Table 1. Her osmolality was 296 mOsm/kg.
On day 2 of admission, the patient was persistently tachycardic (HR = 130–180) despite the fluid resuscitation and the positive fluid balance. She was restless with mottled skin and delayed capillary refill time. She was having respiratory distress with desaturation. She was changed from regular nasal cannula to high flow nasal cannula and ended up with intubation. Chest X-ray images showed pulmonary edema with bilateral pleural effusion (Image 2).

After intubation, her blood pressure started to drop, with mottled skin and cold extremities. Norepinephrine infusion was started to maintain her mean arterial pressure and perfusion. Echocardiogram was done and showed depressed left and right ventricular systolic functions. Ejection fraction was 33.5%. Her brain natriuretic peptide (BNP) level was 18,717 ng/L and troponin level was 0.091 µg/L (normal level is <0.014). Central venous pressure was 13–14 mmH2O through the right internal jugular venous catheter. Milrinone infusion was started at 0.3 µg/kg/min and then increased to 0.5 µg/kg/min. 12-lead electrocardiogram (ECG) was done and showed sinus tachycardia (Image 3).

CT scan of the head was repeated on day 3 of admission, which showed resolution of the brain edema (Image 4). On day 5 of admission, norepinephrine was weaned off, and she was extubated to nasal cannula. On day 6, echocardiogram was repeated, which showed improved systolic function. Milrinone was stopped. Blood and urine cultures were both negative. On day 7, she was discharged from the PICU to the pediatric ward.

Discussion

Myocardial dysfunction is not commonly reported with DKA, and the underlying pathology is not very clear. Multiple mechanisms might play a role. Acidosis can cause cardiac contractile dysfunction. It might cause injury on the cellular level and lead to myocardial dysfunction.

It is possible that the myocardial dysfunction is secondary to lung pathology. The pulmonary edema might be due to fluid overload; however, our patient received initially 1.5 times her maintenance rate, which was then reduced to the maintenance rate on day 1 of admission, which is in accordance with the current recommendations for the management of DKA. On the contrary, the pulmonary edema might be related to severe ketoacidosis, regardless of the fluid administration.

The possibility of infectious myocarditis cannot be excluded. However, in patients as young as our patient, it is expected to cause a more fulminant disease. We think it is less likely the cause of the fast and complete recovery of the heart function. Autoimmunity could be another additional mechanism. It is reported that severe DKA initiates the
synthesis of autoantibodies to cardiac antigens, which can lead to the development of cardiomyopathy in young patients with DKA.\textsuperscript{7}

Electrolyte abnormalities can also contribute to the cardiac dysfunction. In one case report, DKA with severe hypophosphatemia was associated with respiratory failure and cardiac arrest in a 14-year-old patient.\textsuperscript{8} Our patient had initially a low level of phosphate, which was corrected by adding potassium phosphate to the fluids.

We are aware of two pediatric patients who had DKA complicated with myocardial dysfunction. One is a 12-year-old boy who had severe hyperosmolarity and experienced an acute myocardial infarction likely secondary to alterations in regional blood flow and a hypercoagulable state.\textsuperscript{9} Our patient’s osmolality was 296 mOsm/kg, and the ECG did not show signs of acute infarction.

In another case, a 9-year-old girl with DKA developed myocardial dysfunction, elevated troponin level and ECG changes.\textsuperscript{10} She was first treated as a case of septic shock. She received two normal saline doses of 20 mL/kg antibiotics and two doses of methylprednisolone prior to confirming the diagnosis of DKA. It is possible that fluid overload played a major role in developing pulmonary edema in this patient. Our patient received a bolus of 10 mL/kg and then started the fluid management as per the DKA protocol.

Other cases are reported in adults for myocardial necrosis\textsuperscript{11,12} and ECG changes associated with DKA.\textsuperscript{13} In a recent report, two adult patients (53- and 57-year-old) with DKA had elevated troponin levels in the absence of coronary artery disease.\textsuperscript{14} Another report presented a middle-aged patient with uncontrolled diabetes. The patient had elevated troponin I level and ST elevations on electrocardiography with no angiographic evidence of occlusive coronary artery disease.\textsuperscript{15}

There are studies that link elevated troponin I levels and poor outcomes among adult diabetic patients with ketoacidosis.\textsuperscript{16} The significance of the elevated level of troponin I in our patient is not clear.

**Conclusion**

The incidence of myocardial dysfunction in DKA is not known, but it has been described in several reports, mostly in adults. Further studies are needed to address this aspect, especially in children. Early screening of cardiac function in cases of severe DKA may be warranted. It might help in early detection and hence reducing the morbidity of this serious complication. More studies are needed to determine the potential relevance that troponin I could have in these patients for the development of cardiovascular complications in the future.

**Declaration of conflicting interests**

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**Ethical approval**

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Informed consent
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