Case Report

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Myocardial dysfunction associated with diabetic ketoacidosis in a 5-year-old girl

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Abstract

In this study, we report a case of a 5-year-old girl with new onset of insulin-dependent diabetes mellitus, who presented with severe diabetic ketoacidosis associated with brain edema and severe myocardial dysfunction, needing intubation and inotropic support. To our knowledge, this is the youngest reported case with severe diabetic ketoacidosis complicated with myocardial dysfunction.

Keywords

Diabetes/endocrinology, critical care/emergency medicine, cardiovascular, diabetic ketoacidosis, heart failure, myocardial dysfunction

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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.[1] Acute respiratory failure might occur and can be due to various factors, including myocardial dysfunction.[2]

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, CO_2 18.2, HCO_3 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.

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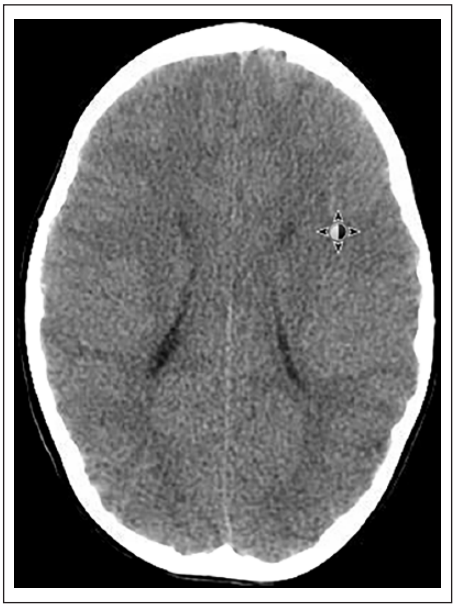

Image 1. Head CT scan on day 1. It shows effacement of brain sulci and narrowing of the lateral ventricle, suggesting increased intracranial pressure.

Table 1. Selected values of the patient's electrolytes during her stay.

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Na	135	149	153	150	140	144
K	3.2	3.0	4.6	3.3	3.2	3.8
Cl	104	124	131	112	96	104
CO_2	2	11	11	25	35	29
Ca	2.62	2.11	1.73	2.10	2.21	2.29
Mg	0.76	0.86	0.83	0.76	0.63	0.89
P	0.47	0.84	1.91	1.61	1.1	1.36

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

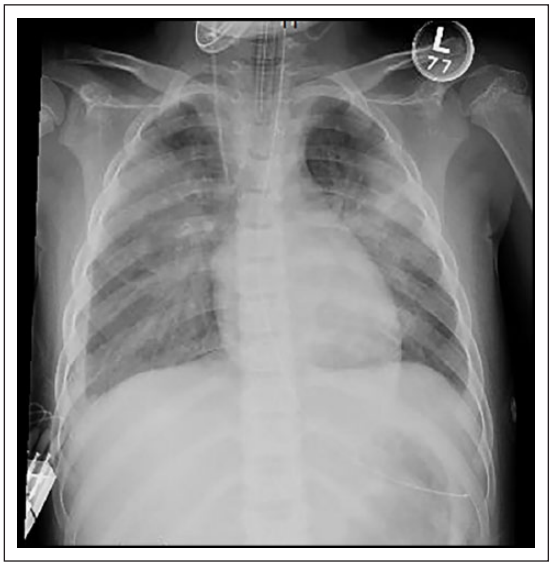

Image 2. Chest radiograph showing pulmonary edema with bilateral pleural effusion.

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.[3] It might cause injury on the cellular level and lead to myocardial dysfunction.[4]

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.[5]

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.[6] 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

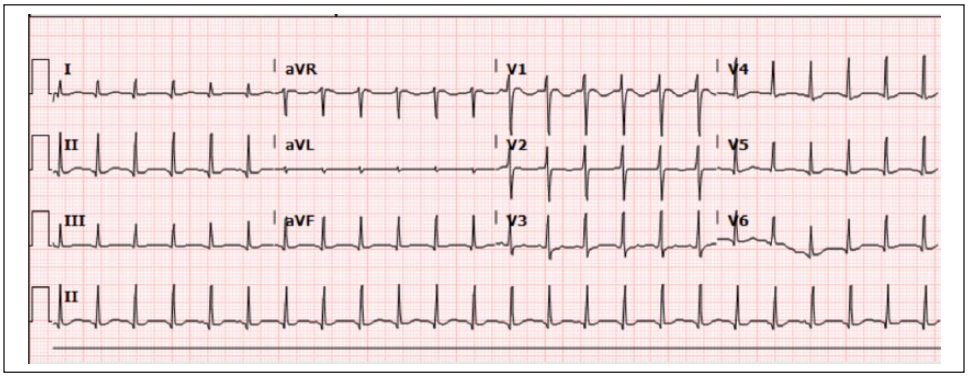

Image 3. 12-lead ECG of the patient showing sinus tachycardia.

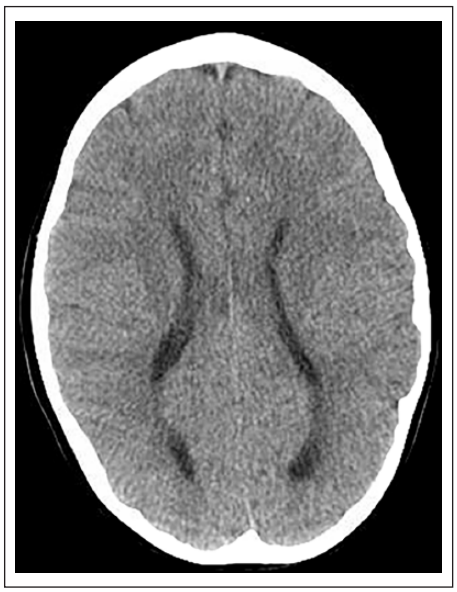

Image 4. Head CT scan on day 3 showing improvement in the brain edema.

synthesis of autoantibodies to cardiac antigens, which can lead to the development of cardiomyopathy in young patients with DKA.[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.[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.[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.[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[11,12] and ECG changes associated with DKA.[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.[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.[15]

There are studies that link elevated troponin I levels and poor outcomes among adult diabetic patients with ketoacidosis.[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

The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

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Informed consent

Written informed consent was obtained from the father, who is the patient's legally authorized representative, for anonymized patient information to be published in this article.

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References

1. Wolfsdorf JI, Allgrove J, Craig ME, et al. A consensus statement from the international society for pediatric and adolescent diabetes: diabetic ketoacidosis and hyperglycemic hyperosmolar state. *Pediatr Diabetes* 2014; 15(Suppl. 20): 154–179.
2. Konstantinov NK, Rohrscheib M, Agaba EI, et al. Respiratory failure in diabetic ketoacidosis. *World J Diabetes* 2015; 6: 1009–1023. DOI: 10.4239/wjd.v6.i8.1009.
3. Lasheen NN and Mohamed GF. Possible mechanisms of cardiac contractile dysfunction and electrical changes in ammonium chloride induced chronic metabolic acidosis in Wistar rats. *Physiol Res* 2016; 65(6): 927–940.
4. Karmazyn M. NHE-1: still a viable therapeutic target. *J Mol Cell Cardiol* 2013; 61: 77–82. DOI: 10.1016/j.yjmcc.2013.02.006.
5. Hoffman WH, Locksmith JP, Burton EM, et al. Interstitial pulmonary edema in children and adolescents with diabetic ketoacidosis. *J Diabetes Complications* 1998; 12: 314–320.
6. Cooper LT. Myocarditis. *N Engl J Med* 2009; 360: 1526–1538.
7. Hoffman WH, Sharma M, Cihakova D, et al. Cardiac antibody production to self-antigens in children and adolescents during and following the correction of severe diabetic ketoacidosis. *Autoimmunity* 2016; 49: 188–196.
8. Choi HS, Kwon A, Chae HW, et al. Respiratory failure in a diabetic ketoacidosis patient with severe hypophosphatemia. *Ann Pediatr Endocrinol Metab* 2018; 23: 103–106. DOI: 10.6065/apem.2018.23.2.103.
9. Roberts KD and Levin DL. Diabetic ketoacidosis, respiratory distress and myocardial dysfunction. *BMJ Case Rep*. Epub ahead of print 1 December 2009. DOI: 10.1136/bcr.01.2009.1530.
10. Batra AS, Acherman RJ, Wong P, et al. Acute myocardial infarction in a 12-year-old as a complication of hyperosmolar diabetic ketoacidosis. *Pediatr Crit Care Med* 2002; 3: 194–196.
11. Tretjak M, Verovnik F, Vujkovac B, et al. Severe diabetic ketoacidosis associated with acute myocardial necrosis. *Diabetes Care* 2003; 26: 2959–2960.
12. Mokuno T, Sawai Y, Oda N, et al. A case of myocarditis associated with IDDM. *Diabetes Care* 1996; 19: 374–378.
13. Kale T, Agrawal H, Pandit R, et al. T-wave inversion in diabetic ketoacidosis with normokalemia in an adolescent. *Pediatr Cardiol* 2013; 34: 1508–1510.
14. Manikkan AT. Elevated troponin I levels in diabetic ketoacidosis without obstructive coronary artery disease. *J Endocr Soc* 2018; 2(9): 1020–1023. DOI: 10.1210/js.2018-00152.
15. Odubanjo AA, Kalisetti R, Adrah R, et al. Severe myopericarditis in diabetic ketoacidosis-all troponin are not myocardial infarction. *Clin Med Insights Case Rep*. Epub ahead of print 1 January 2018. DOI: 10.1177/1179547618763356.
16. Abdo AS and Geraci SA. Significance of elevated cardiac troponin I in patients with diabetic ketoacidosis. *J Miss State Med Assoc* 2013; 54(5): 127–130.