

Supraventricular Tachycardia With Underlying Atrial Flutter in a Diabetic Ketoacidosis Patient

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Diabetic ketoacidosis (DKA) is one of the most common complications in adolescents and young adults with type 1 diabetes. Type 1 diabetes with childhood onset has an incidence that fluctuates from 0.1 to 57.6 per 100,000 and is on the rise (1). In children with type 1 diabetes, the incidence of DKA has been reported to be >30% (2). In a study conducted by Dabelea et al. (3), a higher prevalence of DKA was reported in patients who were younger, male, or of a minority race/ethnicity.

Complications of DKA are multiple and include electrolyte disturbances, acute kidney failure, and respiratory distress. The most serious DKA complication is cerebral edema (4). The incidence of cerebral edema in DKA patients is 1%, but the associated mortality rate is 20–25% (5–7). Although electrolyte abnormalities are common, arrhythmias are a rare complication of DKA. We report here the case of a 12-year-old girl who presented with DKA and subsequently developed supraventricular tachycardia (SVT) with an underlying atrial flutter rhythm.

Presentation

A 12-year-old girl with a known history of type 1 diabetes presented with altered mental status of 1 day's duration. The event was precipitated by the consumption of a large quantity of sugar-sweetened beverages. Her vital signs in the emergency department showed a respiratory rate of 32 breaths/min, heart rate of 204

bpm, and blood pressure of 135/115 mmHg. The patient was tachycardic with Kussmaul respirations and lethargic, and she only responded to deep painful stimuli. Although the patient was diagnosed with type 1 diabetes at the age of 10 years and had experienced previous episodes of DKA, cardiac abnormalities had never occurred before. Except for a maternal great grandmother with type 2 diabetes, her family history was noncontributory.

The patient received 2,000 mL of normal saline boluses and 2 units of regular insulin in the emergency department. Initial laboratory tests revealed a glucose concentration of >600 mg/dL, pH of 6.91, partial pressure of carbon dioxide (PCO₂) of 13.4 mmHg, partial pressure of oxygen of 337 mmHg, and bicarbonate of 2.7 mmol/L. After being transferred to the pediatric intensive care unit, additional laboratory tests revealed a glucose of 1,005 mg/dL, potassium of 5.2 mEq/L, calcium of 8.6 mg/dL, blood urea nitrogen of 37 mg/dL, C-reactive protein of 1.5 mg/dL, lactic acid level of 3.4 mmol/L, and A1C of 9.3%. Urinalysis was significant for 3+ glucose, 3+ ketones, and 1+ blood.

The patient was started on intravenous (IV) regular insulin at 0.1 unit/kg/hour. IV fluids included 0.45% normal saline with 20 mEq/L of potassium chloride and 20 mmol/L of potassium phosphate at a rate of 185 mL/hour. Electrolytes and acid-base status were constantly mon-

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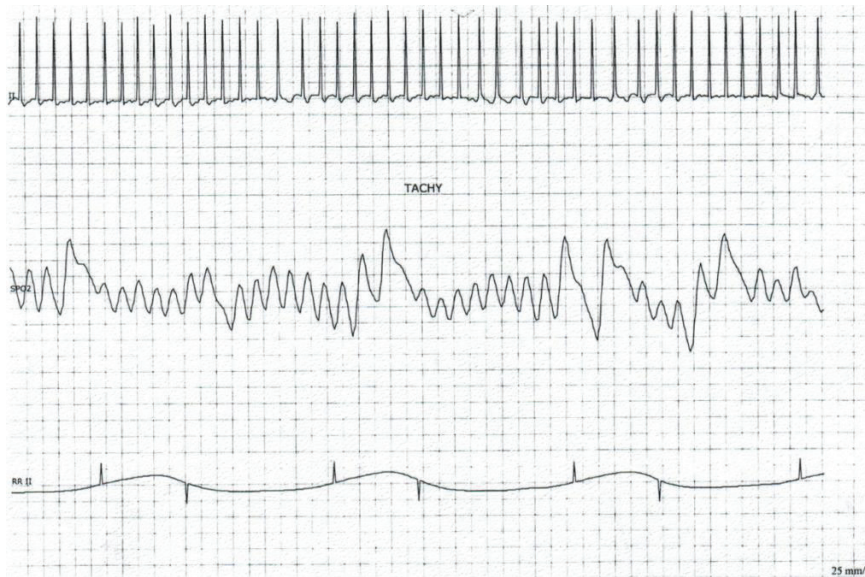
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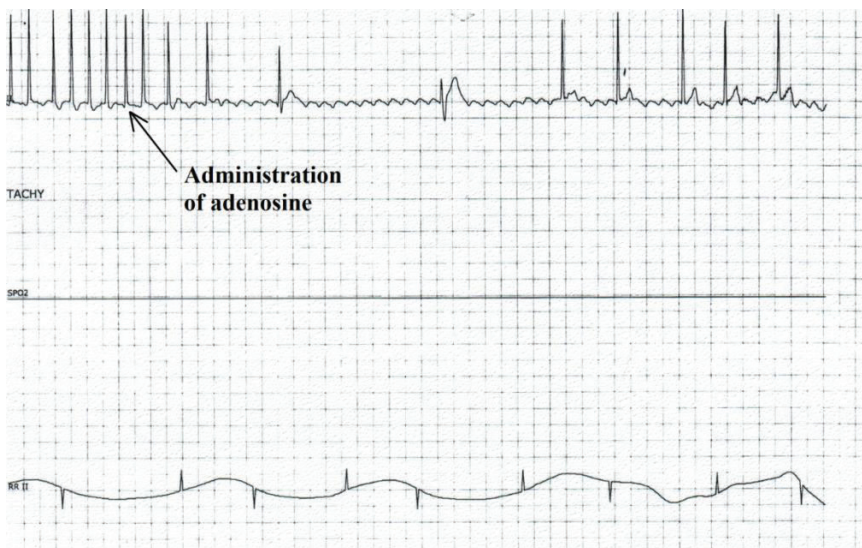
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■ **FIGURE 1.** SVT in a patient with DKA.



■ **FIGURE 2.** Atrial flutter after adenosine administration.

itored. Five hours after admission, her pH improved slightly to 7.09 with persistence of her metabolic acidosis, venous PCO_2 of 22.7 mmHg, potassium of 5.1 mEq/L, and sodium of 149 mmol/L, and her bicarbonate increased to 6.9 mmol/L.

Nearly 6 hours after admission, sudden onset of persistent tachycardia with a heart rate of ~270 bpm was noted on the monitor. Pulses were weak, and the patient's capillary refill was delayed at 3–4 seconds. Electrocardiogram (ECG) revealed the presence of a SVT (Figure 1).

Vagal maneuvers in attempts to restore sinus rhythm, including ice to the face, were unsuccessful. A single dose of 6 mg adenosine was bolused, slowed the heart rate to ~60 bpm, and revealed an underlying rhythm of atrial flutter (Figure 2). Within a few seconds, the heart rate returned to 260–280 bpm, and blood pressure could not be measured. A chest X-ray was done and was unremarkable. An echocardiogram was done but was limited because of the increased heart rate. The echocardiogram only displayed mild tricuspid regurgitation,

and the right ventricle was not well visualized.

The patient was thought to be hemodynamically unstable, and synchronized cardioversion was immediately employed (Figure 3). The patient quickly responded to 30 joules of current. Sinus rhythm was restored with a heart rate of 133 bpm. Blood pressure after cardioversion was 80/40 mmHg. Standard management of DKA continued. Blood pressure improved over the next few hours, and electrolytes showed a potassium of 4.3 mEq/L, chloride of 114 mmol/L, calcium of 9.4 mg/dL, magnesium of 2.0 mg/dL, and phosphate of 3.2 mg/dL. The patient's pH improved to 7.27.

During the next few days, the patient remained hemodynamically stable with no recurrence of arrhythmias. A repeat echocardiogram showed no underlying structural abnormalities. The patient did not require any additional treatment for the arrhythmia. At discharge, the DKA had resolved, the glucose was under control, and the patient was asymptomatic.

Questions

1. What is the role of electrolyte abnormalities in the development of SVT and atrial flutter?
2. What is the role of ketoacidosis in the precipitation of SVT and atrial flutter?

Commentary

Serious metabolic abnormalities can often occur with type 1 diabetes. One such life-threatening complication is DKA. Findings in DKA include an increased anion gap metabolic acidosis with a pH <7.3, bicarbonate <15 mmol/L, glucose concentration >200 mg/dL, and a combination of ketones in the blood and glucosuria (8).

DKA associated with cardiac complications such as SVT is rarely seen clinically. Thomas et al. (9) presented two cases of DKA associated with SVT. The two patients were both female; one was 13 years old, and the other was 14 years old. The



FIGURE 3. Restoration of sinus rhythm after synchronized cardioversion.

13-year-old patient presented with a potassium of 7.3 mEq/L, sodium of 137 mmol/L, bicarbonate of 6.8 mmol/L, pH of 6.93, and A1C of 9.5%. After 4 hours of insulin, saline, and dextrose-containing fluid therapy, SVT was noted with a heart rate of 220 bpm. Submerging the patient's face into ice water abolished the SVT (9). The 14-year-old patient presented with a pH of 7.26, potassium of 4.4 mEq/L, bicarbonate of 12.8 mmol/L, and A1C of 9%. SVT was noted on initial presentation with a heart rate of 229 bpm. A treatment regimen consisting of insulin, saline, and dextrose-containing fluids was adopted to address the DKA, which corrected the SVT. However, the SVT resumed 1 hour later, refractory to three incremental doses of adenosine (3, 6, and 12 mg). The DKA treatment regimen continued, which increased the pH to 7.36 and bicarbonate to 20 mmol/L. After a large emesis, the patient's SVT resolved (9). Beyond SVT, only a few arrhythmias in association with DKA have been reported. These include atrial fibrillation (10) and sinus tachycardia that progressed to ventricular fibrillation (11).

Interestingly, quite a few similarities such as age, sex, and blood pH can be noted between these two cases and our patient. Thus, patients pre-

senting with such a clinical picture may be more likely to develop SVT. However, this hypothesis requires further study.

Arrhythmias can potentially be associated with dangerous hyperkalemia. This acidosis-associated hyperkalemia can speed cardiac repolarization or decrease the conduction speed through the myocardium (12). However, multiple additional factors can also cause arrhythmias, including re-entry circuits, abnormal automaticity, or after-depolarizations occurring within the myocardium (12). A common cause of re-entry circuits within the myocardium is electrolyte abnormalities involving calcium and potassium (12). Because our patient presented with a serum potassium level of 5.2 mEq/L and serious acidosis, the SVT and atrial flutter observed could in part be attributed to these electrolyte abnormalities. Additionally, hypomagnesemia and hypophosphatemia have also been associated with tachyarrhythmias such as SVT (13).

Acidosis resulting from the production of ketoacids is a key clinical finding in DKA that may precipitate arrhythmias. Orchard and Cingolani (14) described how arrhythmogenic consequences of acidosis such as re-entry, pulsus alternans, and early/

delayed after-depolarizations may provoke arrhythmias. Youssef and Farid (15) discussed how ketosis may lead to a prolonged corrected QT interval and QT dispersion, thus evoking arrhythmias because of alteration in action potential conduction (15). Beyond electrolyte abnormalities and acidosis from increased serum ketones, other factors have also been implicated in the development of arrhythmias. During observation, Sufke et al. (16) witnessed supraventricular and ventricular arrhythmias in 12 patients (10 with DKA and 2 with hyperglycemic hyperosmolar syndrome). Autonomic dysfunction resulting from high glucose levels was elucidated to be the cause of these arrhythmias (16).

Although DKA can cause life-threatening arrhythmias, it is usually reversible with appropriate treatment consisting of insulin and IV fluids. During treatment, it is imperative to closely monitor patients' electrolyte and acid base status. It is recommended that electrolytes be replaced with a solution that has a tonicity $\geq 0.45\%$ saline, as was given to this patient, with the addition of potassium chloride, potassium phosphate, or potassium acetate (17). These treatment options, along with synchronized cardioversion to address the SVT, were employed in this patient, resulting in resolution of the DKA.

In conclusion, the SVT and atrial flutter observed in the reported case was most likely the result of electrolyte abnormalities and ketoacidosis. Deviations in electrolyte levels from normal reference ranges, as well as acidosis, have been associated with the development of arrhythmias owing to a variety of mechanisms such as re-entry circuits, after-depolarizations, and alterations of AP conduction among other factors. However, a multifocal etiology resulting in the onset of the arrhythmia is very possible. DKA and arrhythmias are a rare association; it would be

interesting to see other reports of a similar clinical picture.

Clinical Pearls

- Electrolyte abnormalities and ketoacidosis probably contributed to the development of SVT and atrial flutter in this case.
- Synchronized cardioversion was indicated with hemodynamic instability and failure to respond to vagal maneuvers and adenosine.

Duality of Interest

No potential conflicts of interest relevant to this article were reported.

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