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METHAMPHETAMINE-INDUCED TACHYDYSRHYTHMIA IN AN ADOLESCENT IN DIABETIC KETOACIDOSIS

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☐ **Abstract—Background: Diabetic ketoacidosis (DKA) is a common complication affecting patients with type 1 diabetes, and DKA is associated with dehydration and electrolyte abnormalities. Supraventricular tachycardia (SVT), although a common tachydysrhythmia in the pediatric population, remains a rare entity in patients presenting with DKA. Case Report: We describe a case of first-time SVT in an adolescent patient with DKA and recent methamphetamine abuse, with both factors likely predisposing him to develop a tachydysrhythmia. Why Should an Emergency Physician Be Aware of This?: SVT can be present in a patient who has concomitant recent stimulant intake and DKA. A trial of abortive therapy, such as adenosine, should be considered upon diagnosis.** © **2019 Elsevier Inc. All rights reserved.**

☐ **Keywords—arrhythmia; diabetic ketoacidosis; dysrhythmia; methamphetamine; supraventricular tachycardia**

INTRODUCTION

The management of diabetic ketoacidosis (DKA) is often challenging because it requires a careful balance of fluid resuscitation and insulin therapy with the risk for cerebral edema (1). Although rare, the acidosis and electrolyte derangements associated with DKA can predispose patients to dysrhythmias (2). In addition, stimulant drugs, such as cocaine or methamphetamines, are linked with cardiovascular toxicity, such as tachycardia or angina-like symptoms (3). We present a case of amphetamine-induced atrioventricular nodal re-entrant tachycardia (AVNRT) in a young man presenting to the pediatric emergency department with DKA.

CASE REPORT

A 20-year-old man with poorly controlled type 1 diabetes presented to the pediatric emergency department with hyperglycemia and sleepiness. Since his initial diagnosis at 17 years of age, he had a history of poor compliance with his medications. His insulin regimen included self-injecting glargine and lispro. Self-reported glucose measurements ranged from 300–350 mg/dL on the day of presentation. He had diffuse abdominal pain, headaches, multiple episodes of emesis, and decreased urine output. His temperature was 36.5°C, his heart rate was 160 beats/min, his respiratory rate was 16 breaths/min, his blood pressure was 94/58 mm Hg, and his oxygen saturation was 96% on room air. He was tired on examination but arousable and able to answer questions and follow commands. A 10-mL/kg normal saline bolus

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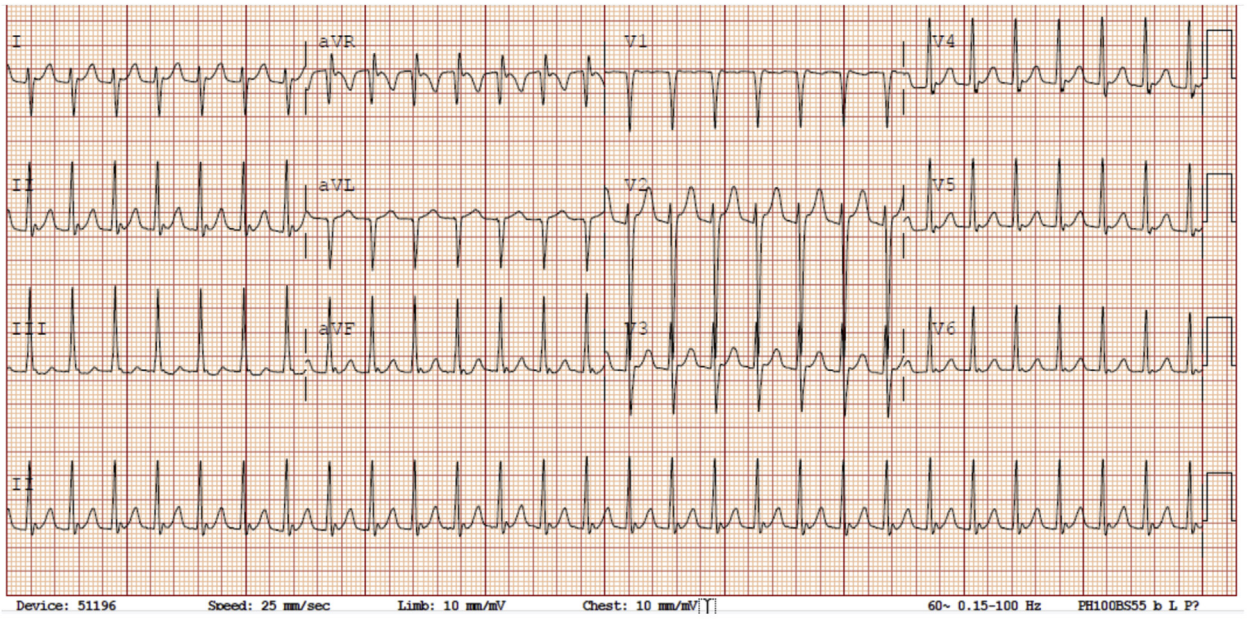

Figure 1. Initial electrocardiogram in the emergency department.

was administered for the first hour. Initial venous blood gas showed a pH of 7.12, bicarbonate of 10 mEq/L, and glucose of 432 mg/dL. Urinalysis was positive for ketones. His potassium was 4.7 mEq/L, with no other electrolyte abnormalities.

He was given an additional 10-mL/kg normal saline bolus and an insulin infusion was started at 0.1 units/kg/h. Maintenance fluids using 2 bags of differing dextrose concentrations were initiated. His second blood gas showed a pH 7.18, bicarbonate of 12 mEq/L, and glucose of 342 mg/dL. The patient was noted to be increasingly sleepy although still arousable to noxious stimuli and responsive to commands. Given his persistent tachycardia, an electrocardiogram (ECG) was performed (Figure 1). A third 10-mL/kg normal saline bolus was administered in consultation with the intensive care and endocrinology subspecialists, after which his heart rate decreased to 150 beats/min. Given his

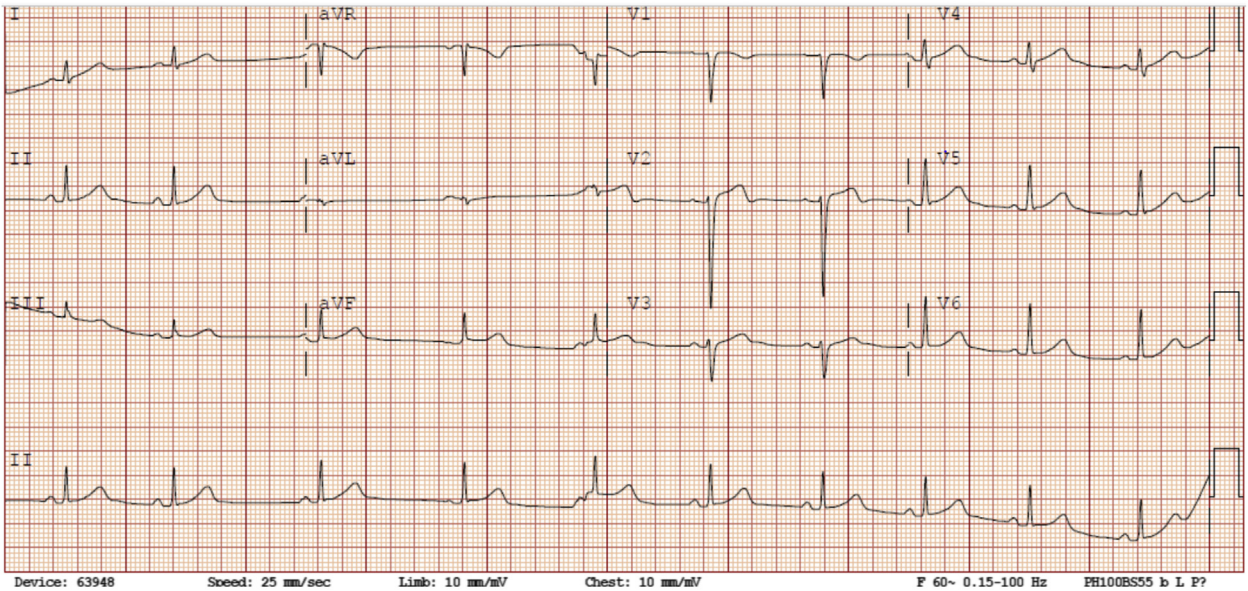

Figure 2. Follow-up electrocardiogram after spontaneous termination of supraventricular tachycardia on the inpatient ward.

history of noncompliance, the patient underwent a urine toxicology screening, which returned positive for amphetamines. He admitted to crystal methamphetamine use a few days earlier when presented with the laboratory findings.

His heart rate improved in response to fluid resuscitation and he maintained a reassuring neurologic examination, and therefore he was deemed appropriate for the general medical ward. His tachycardia was attributed to stimulant intake and ketoacidosis. After transfer to the ward, his DKA resolved and he was transitioned to subcutaneous insulin. His heart rate also dropped suddenly from 160 to 60 beats/min on telemetry. Cardiology alerted the team the next day that the initial ECG was concerning for a re-entrant supraventricular tachycardia (SVT). A follow-up ECG showed that he had reverted to normal sinus rhythm (Figure 2). Outpatient follow-up with the cardiology department was arranged before the patient was discharged from the hospital.

DISCUSSION

Our patient exhibited a narrow complex tachycardia to 160 beats/min, an RP interval (the interval between the start of the QRS complex and the start of the p wave) of 66 msec, and a constant R-R interval. In addition, the termination of the tachycardia was sudden and without obvious trigger. These findings are consistent with a re-entrant type of SVT such as AVNRT, the most common form of SVT in adolescents (4).

In 2011, the United Nations Office on Drugs and Crime estimated that about 34 million people worldwide used amphetamine-like stimulants (5). These drugs increase catecholamine secretion and inhibit reuptake, and can therefore increase the amount of atrial and ventricular ectopy (6–8). Multiple case reports have described tachydysrhythmias, including SVT and ventricular tachydysrhythmia, that resulted from methamphetamine intake (7–11).

In addition, catecholamines and electrolyte imbalances can temporarily modify the conduction time and refractory periods of the pathways to levels that may increase the likelihood of SVT (12). DKA is a particularly concerning disease state not only because of hypovolemia but also because of electrolyte derangements (13). Faruqi et al. described a 12-year-old female presenting with DKA with severe acidosis (pH 6.91) and hyperkalemia (potassium 5.2 mEq/L) (14). She developed SVT shortly after presentation and required cardioversion to restore perfusion (14). Thomas et al. reported 2 adolescent females with DKA whose SVT dysrhythmias resolved after correction of their acidosis and electrolyte abnormalities (15). Another 11-year-old male with new-onset type 1 diabetes presenting in severe DKA (pH 6.90) with hyperkalemia (potassium 6.8 mEq/L) developed SVT that was refractory to adenosine, requiring flecainide for termination (2).

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Although previous case reports have addressed patients with DKA with SVT, this is the first known case to document a patient who had concomitant substance abuse and DKA. Moreover, our patient did not have any electrolyte derangements other than his bicarbonate, and his acidosis was not severe (pH > 7.10) (16). This suggests that even this moderate degree of acidosis and dehydration in combination with stimulants may have predisposed him to tachydysrhythmia. Because the patient had minimal improvement in response to fluid resuscitation and tested positive for stimulants, the diagnosis of sinus tachycardia was incorrectly made, in retrospect. Consideration should have been given to a trial of adenosine based on close review of the ECG and toxicology screening. Ultimately, the patient was discharged after his DKA and SVT resolved with outpatient follow-up with the endocrinology and cardiology departments.

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