
Investigación Clínica
ISSN: 0535-5133
Universidad del Zulia

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Investigación Clínica, vol. 59, no. 2, 2018, April-June, pp. 155-160
Universidad del Zulia

DOI: https://doi.org/10.22209/IC.v59n2a05

Available in: https://www.redalyc.org/articulo.oa?id=372960175006

Invest Clin 59(2): 155 - 160, 2018 https//doi.org/10.22209/IC.v59n2a05

# Diabetic ketoacidosis complicated by supraventricular tachycardia in a young adult: a case report.

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**Key words:** diabetic ketoacidosis; supraventricular tachycardia; cardiovascular alterations.

**Abstract.** Diabetic ketoacidosis (DKA) is one of the most common serious metabolic complications of diabetes in adolescents and young adults. Complications are due to metabolic acidosis, electrolyte abnormalities, acute renal failure and respiratory distress. Serum electrolyte abnormalities are common in DKA. Such serum electrolyte alterations, including acidosis, have been associated with the development of cardiac arrhythmias. We report the case of a 29-year-old man whose parents have diabetes mellitus. His illness began six months earlier with a weight loss of 15 kg. He did not seek for medical attention. The patient presented in a family medical unit with drowsiness and dehydration. On examination, his blood pressure was 90/60 mmHg, pulse rate 216 beats/minute, respiratory rate 32 cycles/minute, and temperature 36.5°C. His random blood glucose was 458 mg/dL with ketonuria. His electrocardiogram showed supraventricular tachycardia. He was referred to a second level hospital where he was admitted into the Intensive Care Unit (ICU) after three hours of stabilization. The patient was discharged in good condition 11 days after his admission to the hospital and was to continue treatment with glargine insulin (1 UI/kg) at home. He was also advised on healthy nutrition, blood glucose target of 150 mg/dL and follow-up in the Outpatient Department of Cardiology and Internal Medicine.

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## Cetoacidosis diabética complicada con taquicardia supraventricular en un joven adulto.

*Invest Clin 2018; 59 (2): 155 - 160*

**Resumen.** La cetoacidosis diabética (CAD) es una de las más comunes complicaciones serias de la diabetes en adolescentes y adultos jóvenes. Las complicaciones son debidas a la acidosis metabólica, anormalidades electrolíticas, falla aguda renal y distrés respiratorio. Las alteraciones hidroelectrolíticas son muy comunes en la CAD. Estas alteraciones, junto con la acidosis, han sido asociadas al desarrollo de arritmias. Se reporta el caso de un paciente de 29 años de edad con antecedente de padres diabéticos. Su condición comenzó 6 meses antes con pérdida de peso de aproximadamente 15 kg, astenia y adinamia, sin haber recibido ninguna atención médica. El paciente fue recibido en su Unidad de Medicina Familiar somnoliento y deshidratado, con TA: 90/60, FR: 32x', FC: 216x', T: 36.5GC, glucosa de 458 mg/dL y cetonuria. Su electrocardiograma mostró taquicardia supraventricular. Trasladado a un segundo nivel de atención en hospital, el paciente fue inmediatamente transferido al Área de Reanimación e ingresado a la Unidad de Cuidados Intensivos posterior a 3 horas de estabilización. El paciente fue egresado en buenas condiciones 11 días después de su admisión al hospital con tratamiento a base de insulina glargina (1 UI/kg), y medidas higiénico-dietéticas, glucosa de 150 mg/dL, con seguimiento como paciente externo en los Servicios de Cardiología y Medicina Interna.

**Palabras clave:** cetoacidosis diabética; taquicardia supraventricular; alteraciones cardiovasculares.

*Recibido: 30-08-2017 Aceptado: 15-02-2018*

### INTRODUCTION

Diabetic ketoacidosis (DKA) is one of the most common and serious metabolic complications of diabetes mellitus in adolescents and young adults. It may be the first manifestation of a new-onset diabetes mellitus (1, 2). In 2009, there were approximately 140,000 hospitalizations for DKA, costing more than 1 million dollars in hospitalizations per year in the United States of America (3). Fifty-six percent of patients are between 18 and 44 years-old. The prognosis of the DKA worsens in the extremes of life and in the presence of a coma state, hypotension and serious comorbidities (4). In adults with DKA, the mortality at global and national levels is between 5% and 10% (5). DKA is usually accompanied by severe dehydration, catecholamine release and alterations in the potassium homeostasis. The most frequent causes of DKA are infections and poor adherence to treatment (6). The complications of DKA are alterations due to metabolic acidosis, electrolyte abnormalities, acute renal failure and respiratory distress. Although electrolyte abnormalities are common in DKA, cardiac arrhythmias are rare. The cardiovascular abnormalities in DKA are due to adverse effects of the acidosis and arrhythmias caused by electrolyte disturbances. In models of myocardial cells, the repolarization can be inhibited by the acidosis. The action potential represents a delicate balance between ions, which can be disturbed by the acidosis. The underlying

mechanisms that bring about the changes in the action potential are poorly understood (7). The effects of acidosis on the heart depend on the pH level. These effects can be an increment in the release of catecholamine, which translates into increased inotropism, chronotropism, cardiac output and peripheral vascular resistance. When pH is less than 7.2, H+ ions have a direct depressing action on the heart and it may be associated with decreased cardiac contractility and a predisposition to cardiac arrhythmias, all of which may contribute to hemodynamic instability (8). The acidosis is the key clinical feature found in DKA that can precipitate arrhythmias. Orchard and Cingolani (9), describe the arrhythmogenic consequences of the acidosis such as the re-entry and early repolarization. Youssef and Farid (10) demonstrated how the ketosis may cause arrhythmias due to alterations in driving the action potential. On the other hand, a study conducted by Süfke *et al*. (11), observed ventricular and supraventricular arrhythmias in 12 patients (10 with DKA and 2 with hyperglycemic hyperosmolar state). The autonomic dysfunction resulting from the high levels of glucose was believed to be the root cause of the arrhythmias (11, 12).

The potassium deficit is one of the most important electrolyte abnormalities found in the DKA, which could lead to severe arrhythmias. In 2014, Wolfsdorf *et al*. (13) concluded that a cardiac monitor should be used for continuous electrocardiographic monitoring to assess T waves for evidence of hyper or hypokalemia in patients with DKA. We can also find other electrolyte abnormalities such as hypophosphatemia that can precipitate cardiac arrhythmias (14). The greater part of these arrhythmias is by re-entry, polymorphic atrial fibrillation (AF), ventricular tachycardia (VT) and ventricular fibrillation (VF), premature atrial contractions, atrial tachycardia with or without atrioventricular (AV) block, supraventricular tachycardia (SVT) and premature ventricular contraction (15). SVT in the DKA can be due to different abnormalities in the formation of the cardiac electrical impulse (16). DKA associated with cardiac complications, such as the SVT, is rarely seen in clinical practice. There are three cases reported in the world's literature: Thomas *et al*. with two cases in 2007 (16) and Faruqi *et al*. with one case in 2015 (1). SVT is probably a result of the electrolyte and ketoacidosis abnormalities. However, a multifactorial etiology resulting in the development of SVT is very possible (1).

This Case Report is important and relevant with the potential of raising the alertness of physicians to improve care and prognosis in patients with DKA. The key lesson of this Case Report is to perform electrocardiogram to all patients with DKA in order to detect and treat cardiac arrhythmias such as SVT which may occur as a complication of metabolic and electrolyte disturbances which are present in these patients.

## CASE PRESENTATION

We report the case of a 29-year-old man who presented with a history of weight loss for six months. Over this period of 6 months, he lost 15 kg. He has a positive history of diabetes mellitus in both parents. He did not seek for medical attention. Five days before his admission to our hospital, he presented with diaphoresis, dyspnea, thirst and palpitations. When his symptoms worsened he decided to go seek for medical attention. The attending physician found him drowsy, dehydrated, with acidotic breathing. His vital signs (VS) were: BP: 90/60, respiratory rate 32 cycles/minute, pulse rate 216/minute, and temperature 36.5°C. His random blood glucose was 458 mg/dL, with ketonuria. The treatment started with crystalloids with a double way (Hartmann and 0.9% saline solution, 1 liter of each solution), sodium bicarbonate (222.5 mEq) and insulin infusion (100 UI of insulin in 100 mL of 0.9% saline solution to 4 mL/min). The patient was sent over to the second level 3 h later with a diagnosis of severe DKA. Upon entering the

Regional General Hospital # 36 (RGH) the patient was directed to the Triage Area and immediately transferred to the Reanimation Area. His vital signs at the time of entering were BP 90/60mmHg, respiratory rate 18 cycles/minute, pulse rate 213/minute, and temperature 36.7°C. Therapy continues with the hydration by double way with Hartmann solutions. The ECG was performed, detecting SVT (Fig. 1).

Vagal maneuvers (carotid massage) were performed in an attempt to revert the SVT for 5 minutes, without positive results. We decided for the administration of adenosine 6 mg IV in bolus, reverting after a few seconds to sinus rhythm with HR 101x' (Fig. 2).

The same fluid therapy and insulin infusion were continued without the administration of bicarbonate. Initial arterial gasometry (AG) showed metabolic acidosis (pH: 7.40, $PC0_2$:15, $p0_2$: 89, $HC0_3$: 9.3, BE: –15.5, $S0_2$: 97%). The patient was transferred to the Intensive Care Unit (ICU) after 3 hours of stabilization in the Reanimation Area. His laboratory results before beginning treatment in the Emergency Services were: glucose: 126 mg/dL, Ca: 6.2 mg/dL (8.4-10-2 mg/dL), K: 2.9 mmol/L (3.5-5.3 mmol/L). In his second day of stay in ICU, he suffered again a SVT event which required for electrical cardioversion. The serum potassium level was 3.2 mmol/L at that time. On the third day of his admission to the ICU, he presented the following laboratory results: Ca: 7.8 mg/dL, P: 1.8 mg/dL (2.5-4.5), K: 4.0 mmol/L. Five days after his admission to ICU his care was taken over by the Internal Medicine team. At this time his acid-base balance was normal. Subsequently, his blood glucose was well controlled and there was no further SVT event. He was reviewed by the Cardiology team and discharged home on glargine insulin (1 UI/kg). He was counseled on diet and was to maintain a blood glucose of 150 mg/dL, with follow-up in the Outpatient Department of Cardiology and Internal Medicine.

## DISCUSSION

In clinical practice, co-existence of DKA and SVT is rare. Thomas *et al*. (16), reported two cases of type 1 diabetes (T1D)

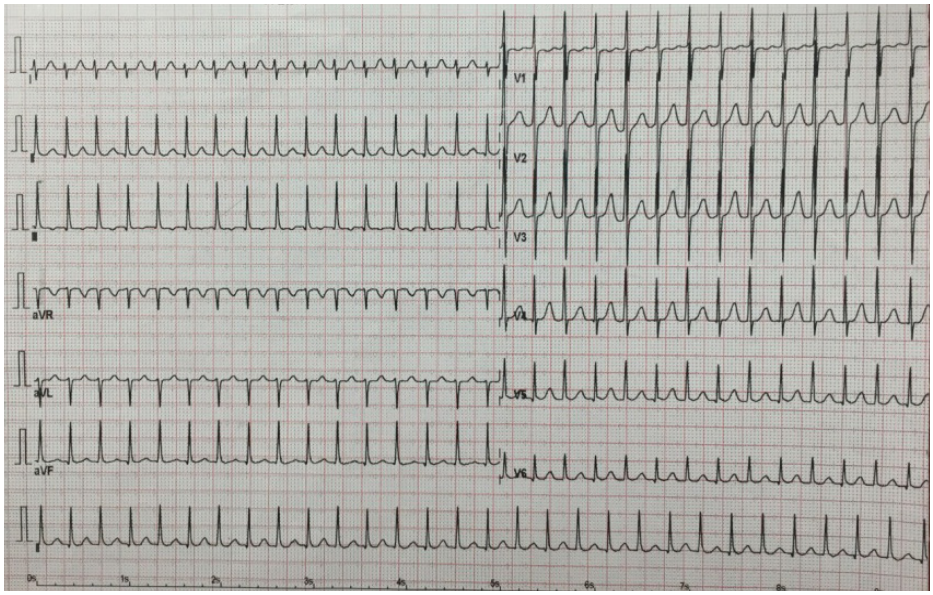


Fig. 1. An electrocardiogram of a 29-year-old man with DKA on arrival at the Emergency Services, showing supraventricular tachycardia (SVT).

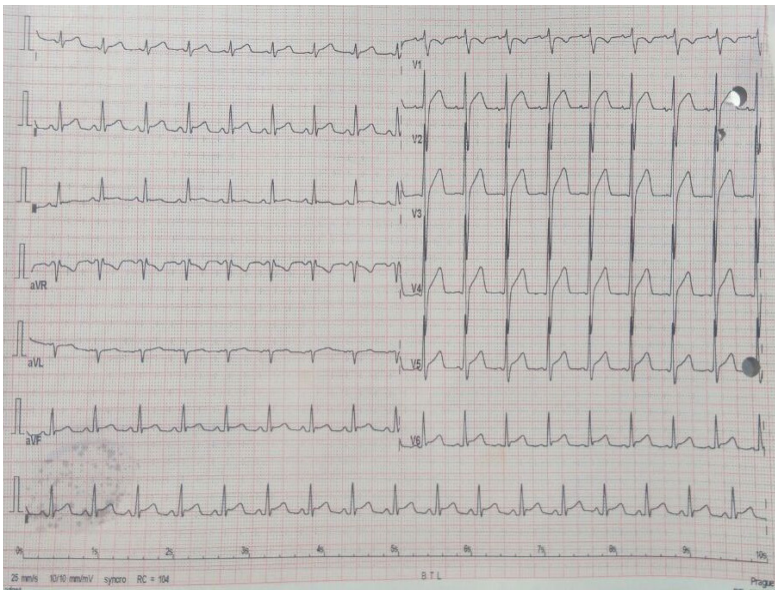


Fig. 2. An electrocardiogram of a 29-year-old man with DKA and SVT, following administration of adenosine and subsequent conversion to sinus tachycardia.

complicated by DKA associated with SVT. Both patients were girls aged 13 and 14 years, respectively. In the case of the first patient, the SVT was jugulated with vagal maneuvers, while the second patient, arrhythmia reverted initially with the treatment of the DKA, becoming a refractory arrhythmia, reversing finally with three increased doses of adenosine at 3mg, 6mg and 12 mg, respectively. In the case reported by Faruqi *et al.*(1), she was 12-year-old girl with T1D and DKA, presenting during her hospital care an event of SVT that reversed with the administration of 6 mg of adenosine, reaching 60 bpm, evidencing an AF, reversing the AF with electrical cardioversion by low spending data. Two of the three cases presented initially pH less than 7.0, bicarbonate less than 10 mEq, and potassium within normal ranges (one case in the Thomas report and another in the Faruqi report). Our case presented normal pH, bicarbonate of 9.3 and hypokalemia. At the time of presenting his second SVT event of that required electrical cardioversion, he had hypokalemia, hypocalcemia and hypophosphatemia, as Shen and Braude (14) showed in their article, hypophosphatemia is another electrolyte disturbance that can precipitate cardiac arrhythmias. Following correction of the electrolyte and acid-base disturbances, he did not have new SVT events.

The metabolic acidosis, dehydration and the electrolyte disturbances associated with DKA may produce alterations in all organs including the myocardium. It produces a lower threshold in the myocyte for the precipitation of arrhythmias such as the SVT, as it was described by Crampin *et al.* (7). Despite all the above mentioned, questions remain: what is the importance of acidosis in precipitating SVT in the DKA?, what is the role of the electrolyte abnormalities on the emergence of the SVT in the DKA?, Why not all patients that present DKA trigger events of SVT?. Is there any relation between gender and age for the appearance of SVT in patients with an event of DKA? and, do the SVT coexist or depend on each other? Because all these, we advocate performing an electrocardiogram in all patients with DKA as recommended by Wolfsdorf *et al.* (13). A review of reports and experiences of other medical centers as well as a large scale is suggested

in order to find answers to the questions. At this moment, there is no consensus of the relationship that may exist between DKA and SVT on one hand and acid-base and electrolyte disturbances in the genesis of the SVT in patients with DKA. Therefore, the questioning continues, is there coexistence or dependency between DK and the SVT?

## ACKNOWLEDGEMENTS

We would like to thank to Guillaume Marc Jackie Leriche for supporting us to translate this manuscript.

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