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Case Report

Hyperkalemia-Induced Coronary Artery Spasm and Junctional Tachycardia in Diabetic Ketoacidosis Reversed with Insulin and Saline, A Case Report

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Abstract

Rationale: Serum potassium concentration is usually elevated in the cases of diabetic ketoacidosis. Coronary artery spasm is recognized after the hematological chemical disturbance. Hyperkalemia is a rare cause of junctional tachycardia. Insulin decreases potassium levels in the blood by redistributing it into cells via increased sodium-potassium pump activity.

Patient concerns: A young housewife female patient presented to the emergency department with diabetic ketoacidosis, coronary artery spasm, and junctional tachycardia.

Diagnosis: Hyperkalemia-induced coronary artery spasm and junctional tachycardia in diabetic ketoacidosis.

Interventions: Electrocardiography, oxygenation, central venous pressure monitoring, and echocardiography.

Lessons: Electrolytes disturbance especially hyperkalemia is a significant serious metabolic problem in ketoacidosis. Hyperkalemia is a possible cause for both coronary artery spasm and junctional tachycardia in diabetic ketoacidosis.

Outcomes: Successful reversal of ketotic hyperkalemia-induced coronary artery spasm and junctional tachycardia with insulin and saline.

Keywords: Ketotic hyperkalemia; Induced junctional tachycardia; Insulin and saline; Diabetic ketoacidosis

Abbreviations: AVN: Atrioventricular node; CAS: Coronary artery spasm; DKA: Diabetic ketoacidosis; ECG: Electrocardiogram; ICU: Intensive care unit; SAN: Sinoatrial node

Introduction

The admission serum potassium concentration is usually elevated in patients with diabetic ketoacidosis (DKA) [1]. Potassium levels can fluctuate severely during the treatment of DKA because insulin decreases potassium levels in the blood by redistributing it into cells via increased sodium-potassium pump activity [2]. Despite a total body potassium deficit of ~3–5 mEq/kg of body weight, most patients with DKA have a serum potassium level at or above the upper limits of normal. In a recent series, the mean serum potassium in patients with DKA was 5.6 mEq/l, respectively. These high levels occur because of a shift of potassium from the intracellular to the extracellular space due to acidemia, insulin deficiency, and hypertonicity [1]. The primary treatment of DKA is with intravenous fluids and insulin [3]. Both insulin therapy and correction of acidosis decrease serum potassium levels by stimulating cellular potassium uptake in peripheral tissues1. A large part of the shifted extracellular potassium would have been lost in urine because of osmotic diuresis. Hypokalemia (low blood potassium concentration) often follows treatment [2]. Therefore, to prevent hypokalemia, most patients require intravenous potassium during DKA therapy. This increases the risk of dangerous irregularities in the heart rate. Therefore, continuous observation of the heart rate is recommended [2], as well as the repeated

measurement of the potassium levels and addition of potassium to the intravenous fluids once levels fall below 5.3 mmol/l. If potassium levels fall below 3.3 mmol/l, insulin administration may need to be interrupted to allow correction of the hypokalemia [4]. Replacement with intravenous potassium (two-thirds as potassium chloride [KCl] and one-third as potassium phosphate [KPO4] should be initiated as soon as the serum potassium concentration is below 5.0 mEq/L. The treatment goal is to maintain serum potassium levels within the normal range of 4–5 mEq/L[1].

Coronary artery spasm (CAS) is an intense vasoconstriction of coronary arteries that causes total or subtotal vessel occlusion [5]. CAS or smooth muscle constriction of the coronary artery is an important cause of chest pain syndromes that can lead to myocardial infarction, ventricular arrhythmias, and sudden cardiac death. About 2 % of people with angina, or chest pain and pressure, experience CAS [6]. The attacks of CAS are associated with either ST-segment elevation or depression on electrocardiogram (ECG) [7]. The injury to vessels causes spasm are recognized after chemical irritation, physical trauma, or ischemia [8]. Smoking, age and high-sensitivity C-reactive protein (hs-CRP) are significant risk factors for CAS. Calcium antagonists are an essential medical therapeutic option. Coronary angiography and provocative testing are the main diagnostic tools [5]. Coronary angiography is the gold standard for the diagnosis of variant angina. Several provocative tests for CAS are used, including ergonovine, acetylcholine, neuropeptide Y and dopamine [9].

The implicated nomenclature to recognize the type of junctional rhythms (JR) is based on their rate. They are classified as follows:

a. Junctional bradycardia: Ventricular rate <40 bpm.

b. Junction escape rhythm: Ventricular rate 40-60 bpm.

c. Accelerated junctional rhythm (AJR): Ventricular rate of 60-100 bpm.

d. Junctional tachycardia: Ventricular rate >100 bpm [10].

If there is a blockage for the sinoatrial node (SAN) electrical activity is blocked or is less than the automaticity of the atrioventricular node (AVN)/His Bundle a JR starts. Numerous conditions and medications can lead to a diseased SAN and lead to the AVN/His Bundle to take over due to the higher automaticity of the ectopic pacemaker [11-13]. Hyperkalemia, sick sinus syndrome, pericarditis, myocarditis, unstable angina, acute myocardial infarction, repair of congenital heart disease, atrial septal defect, tetralogy of Fallot, persistent left superior vena cava, adenosine, digoxin, calcium channel blockers, lithium, amitriptyline, clonidine, reserpine, inhalation anesthetics, cimetidine, isoproterenol infusion, narcotics, beta-blockers, and ivabradine implicated in causing junctional tachycardia [10,14-17].

Case Presentation

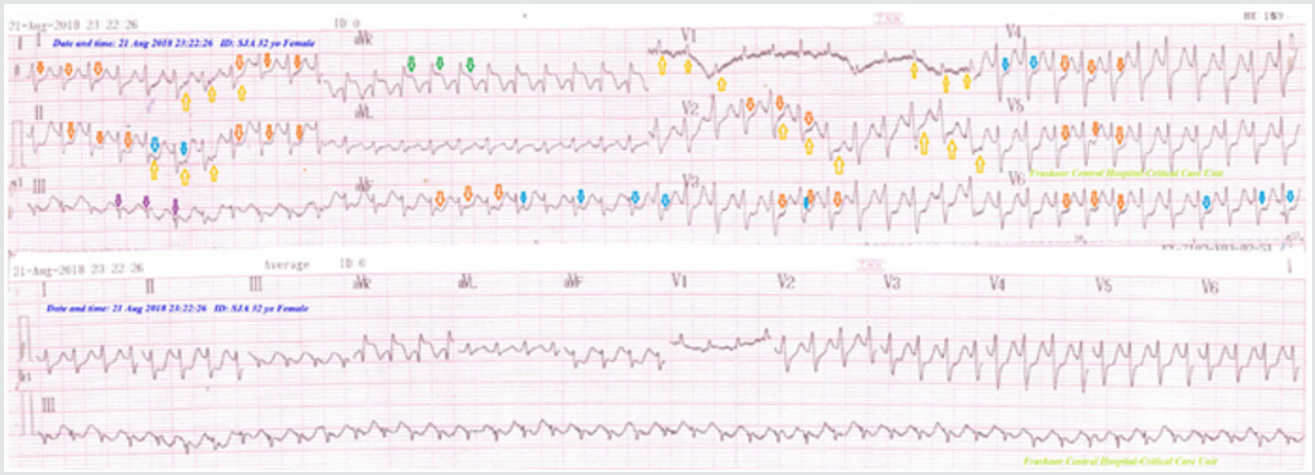

Figure 1: ECG tracings showing junctional tachycardia; "evidenced with inverted P-wave (purple arrows), retrograde P-wave (blue arrows), and absent P-wave (the remaining leads)" extensive straight ST-segment depressions in infero-anterior ECG leads (II, aVF, I, and V2-6) (red arrows), ST-segment elevation in aVR lead (green arrows), and wavy triple ECG sign of hypocalcemia in leads (gold arrows).

A 32-year-old housewife Egyptian female patient presented to the emergency department with chest pain, palpitations, and rapid breathing. The patient had a history of diabetics on long-acting insulin. She gave a history of missed insulin for 3 days. Upon examination, the patient appeared confused, sweaty, and tachypneic. His vital signs were as follows: the blood pressure of 90/70 mmHg, the heart rate of 150/minute, the temperature of 36.9°C, respiratory rate of 34/min, and the initial pulse oximetry of 94%. The patient was admitted to the intensive care unit (ICU) as diabetic ketoacidosis. The patient initially treated with act-rapid insulin (initial 8 units IVB), normal saline 0.9% (1000 ml IVB in the first hour), hourly vital signs monitoring, hourly blood glucose measurement, and hourly urinary acetone check-up. Maintenance of act-rapid insulin (0.1 u/kg; 8u/hours) was continued for about 6 hours. Another 1000 ML normal saline 0.9% was added. RBS was 568 mg/dl on admission and urine analysis (glucose ++++ acetone +++). ABG showed metabolic acidosis. Electrolytes profile showed plasma Na^+: 131 mg/dl, plasma Ka^+: 6.1 mg/dl, I Ca^{++}: 0.65 mmol/L. Urgent ECG recording showed junctional tachycardia, extensive ST-segment depressions and wavy ECG sign of hypocalcemia (Figure

1). Serial ECG tracings were done for follow up. RBS measures were steadily gradually decreased until RBS become;174 mg/dl, the disappearance of urinary acetone. IVI dextrose 5% added with adjustment of act-rapid insulin to (4 units per hour), and A 250 ml Ringer solution over 2 hours, and 250 ml Ringer solution over 2 hours. Electrolytes profile repeated within 6 hours of management showed normalized plasma Na^{+}: 141 mg/dl, plasma Ka^{+}: 5.2 mg/dl, I Ca^{++}: 1.1 mmol/L. ECG tracing on discharge was showed the disappearance of junctional tachycardia with normalization of the above ST-segment depressions and wavy ECG sign of hypocalcemia (Figure 2). Later echocardiography was normal. The patient discharged after stability on the fourth day of admission.

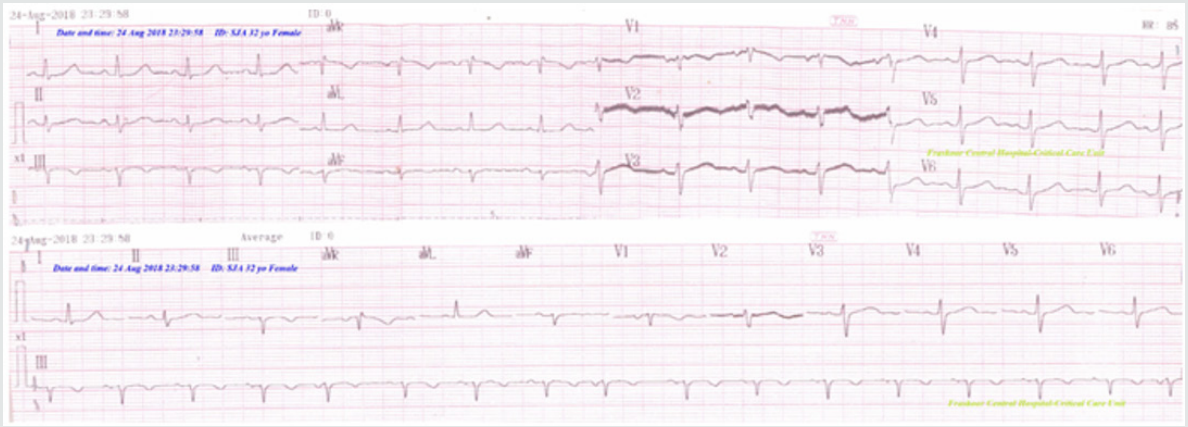

Figure 2: ECG tracings showing the disappearance of junctional tachycardia with normalization of the above ST-segment depressions and wavy triple ECG sign of hypocalcemia.

Discussion

Overview: A 32-year-old single Egyptian male patient presented with diabetic ketoacidosis, junctional tachycardia, and coronary artery spasm in the presence of marked hyperkalemia.

The primary objective: for my case study was the diagnosis of diabetic ketoacidosis, junctional tachycardia, and coronary artery spasm.

The secondary objective: for my case study was the choice management for diabetic ketoacidosis, junctional tachycardia, and coronary artery spasm in the presence of marked hyperkalemia.

Limitations of the study: There are no known limitations in the study.

I can't compare the current case with similar conditions: There are no similar or known cases with the same management for near comparison.

Study question here: How did the relationship among diabetic ketoacidosis and junctional tachycardia, elevated potassium level, coronary artery spasm, and insulin therapy?

Recommendations

a. It is recommended to widening the research in clearing the effect of diabetic ketoacidosis on potassium elevation and its role in causing junctional tachycardia and coronary artery spasm.

b. Also, it is recommended to extend the research on the impact of rapid-acting insulin therapy in the management of diabetic ketoacidosis on junctional tachycardia, coronary artery spasm, and elevated potassium level.

Conclusion

The physician should consider spontaneous recovery of junctional tachycardia, coronary artery spasm, and hyperkalemia on the initial management of diabetic ketoacidosis. So, don't hurry to treatment, but tight follow up for elevated potassium level is obligatory.

Conflicts of Interest

There are no conflicts of interest.

Acknowledgment

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