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Hypokalemia with ECG Abnormalities Mimicking Acute Coronary Syndrome in a Diabetic Ketoacidotic Patient

Diyabetik Ketoasidozlu Hastada Akut Koroner Sendromu Taklit Eden EKG Değişikliği ile Seyreden Hipokalemi

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ABSTRACT

Diabetic ketoacidosis is an extremely serious complication of diabetes mellitus. It arises because of a complex disturbance in glucose metabolism. There is usually a precipitating cause such as sepsis or myocardial infarction. If not recognised and appropriately treated, it can have devastating consequences. This is a case report of a patient with severe diabetic ketoacidosis and interesting electrocardiographic (ECG) findings. The initial electro-cardiographic findings were suggestive of an acute coronary syndrome. The ECG changes normalised remarkably following management of the diabetic ketoacidosis. In this presentation we wished to point out the challenges of differential diagnosis of ECG abnormalities in diabetic ketoacidosis with hypocalemia.

Keywords: *Diabetic ketoacidosis, ECG abnormalities, hypokalemia*

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ÖZET

Diyabetik ketoasidoz diyabetin son derece ciddi bir komplikasyonudur. Glukoz metabolizmasında kompleks bir rahatsızlık nedeniyle ortaya çıkar. Genellikle sepsis veya miyokard infarktüsü gibi presipite eden bir neden bulunur. Tanınması ve uygun şekilde tedavi edilmemesi halinde yıkıcı sonuçlar doğurabilmektedir. Bu, ciddi diyabetik ketoasidozu ve elektrokardiyografik (EKG) değişiklikleri olan vaka takdimidir. Başlangıç EKG bulguları akut koroner sendromu düşündürmektedir. Bu bulgular, diyabetik ketoasidoz tedavisi ile gerilemiştir. Bu sunum ile diyabetik ketoasidozlu hastada hipokalemi ile birlikte görülen EKG bulgularının ayırıcı tanısında yaşanan zorluklar vurgulandı.

Anahtar Kelimeler: *Diyabetik ketosidoz, EKG değişikliği, hipokalemi*

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INTRODUCTION

Diabetic ketoacidosis (DKA) occurs in 10 to 70% of patients with type 1 diabetes mellitus (Type 1 DM) and carries a significant risk of mortality, mostly due to cerebral edema (1). Other potential complications of DKA include hypokalemia, hypophosphatemia, hypoglycemia, intracerebral and peripheral venous thrombosis, mucormycosis, rhabdomyolysis, acute pancreatitis, acute renal failure and sepsis (2).

We describe our experience of treating a patient presenting with DKA and hypokalemia as the first manifestation of (Type 1 DM).

CASE PRESENTATION

An 18-year-old woman without a significant past medical history presented with a two day history of fatique, weakness, nausea and vomiting. She did not t of complain chest pain. The patient denied use of any medications (prescription or nonprescription) or any illicit substances. The patient had a family history of diabetes mellitus type 2 on the maternal side.

On presentation, the patient appeared in mild distress secondary to her stated abdominal pain. Body mass index on admission was 27.1 (weight 91 kilograms), vital signs were within normal limits, and the patient appeared hipovolemic. On physical examination; the tongue was dry, cardiovascular system and abdominal examination were normal, pretibial edema was not found. Initial laboratory studies revealed a high anion gap metabolic acidosis (arterial pH 7.07, arterial PCO_2 10.9 mmHg, 3.1 mmol/L, serum anion gap 29) and hyperglycemia (serum glucose 476 mg/dL, normal range: 70-100 mg/dL). The patient was found to have ketonemia, ketonuria, leukocytosis (white blood cell: 20800/ml, range:4500-1000/ml) and hyponatremia (serum sodium level was 128 mmol/l, range:135-145 mmol/l) Additional data, including serum chloride, serum potassium, magnesium, phosphorus, liver functions, lipid fractionation, serum troponin, creatine kinase- MB, plain chest radiography were within normal limits. The patient was thought to be in a diabetic ketoacidotic state and was started on isotonic saline infusions and intravenous insulin, to which she responded well with rapid resolution of the acidosis. Following insulin therapy, the potassium level was decreased (potassium: 2.1 mmol/l, range: 3.5-5mmol/l). At the time, the ECG pattern indicated acute coronary syndrome (Figure 1) D1-D3 and V1-V6 ST segment depression and T negativity. Then the patient was takento the cardiology intensive care unit and was started on acute coronary syndrome medication with anticoagulant therapy and followed for cardiac enzyme and chest pain. However the patient had no chest pain and serum troponin and CK-MB level did not increase during follow up. After 1 week of her hospitalization she had no symptoms, and with diabetic ketoasidosis management, ECG findings (Figure 2) and electrolyte abnormalities were improved and medication for acute coronary syndrome was stopped. ECG abnormalities were related to the diabetic ketoasidosis and electrolyte imbalances, and the patient was not considered to be having a myocardial infarction. She remained normoglycemic for the remainder of her hospitalization stay. Hemoglobin A1C was 11.07% (4.4%-6.4%), C peptide was 0.4 ng/mL (1.1-4.4 ng/mL), Anti-GAD antibody 37.5U/ml(0-1.0 U/ml).

DISCUSSION

Here we present a case of hyperglycemic ketoacidosis with ECG abnormalities. DKA may lead to multiple organ dysfunctions

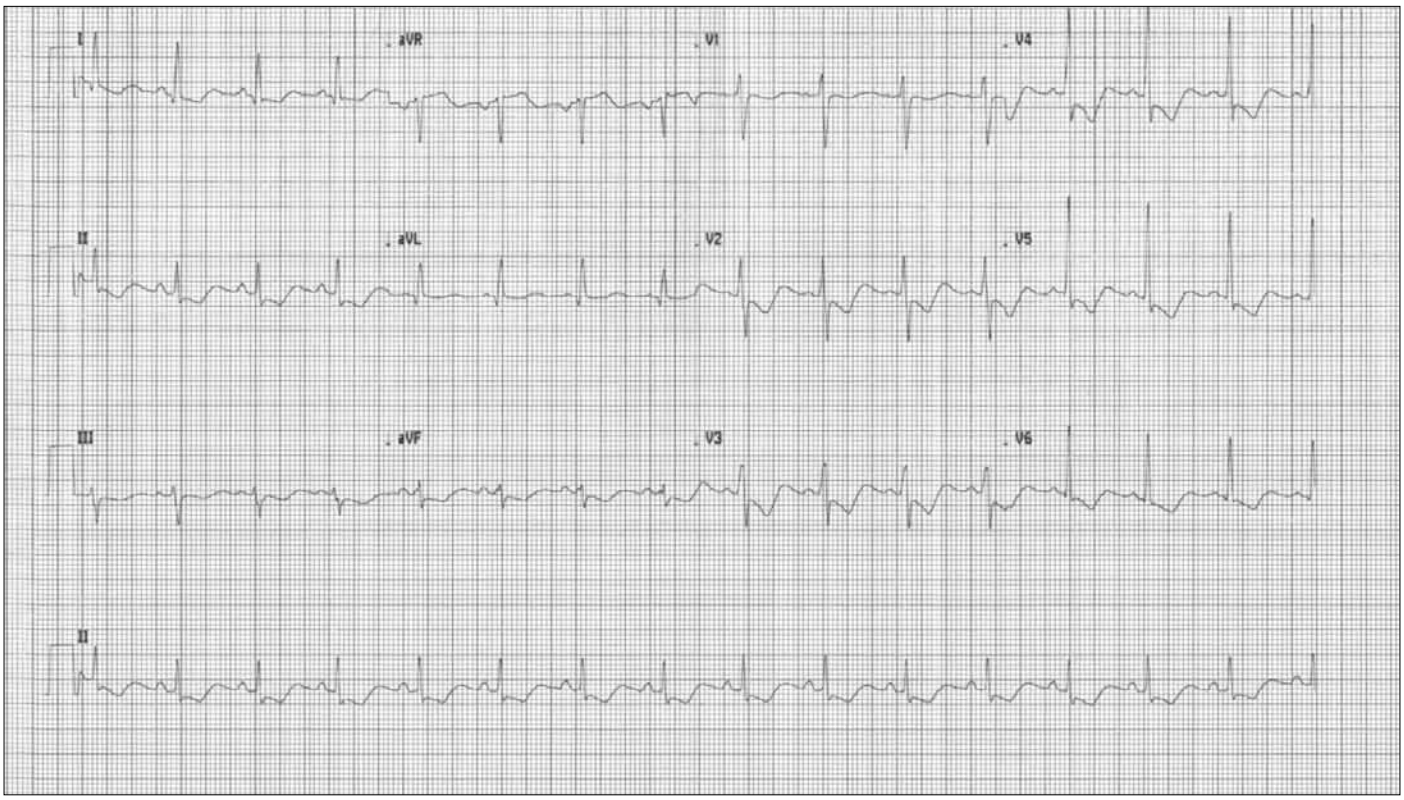

Figure 1. ECG findings at initial visit

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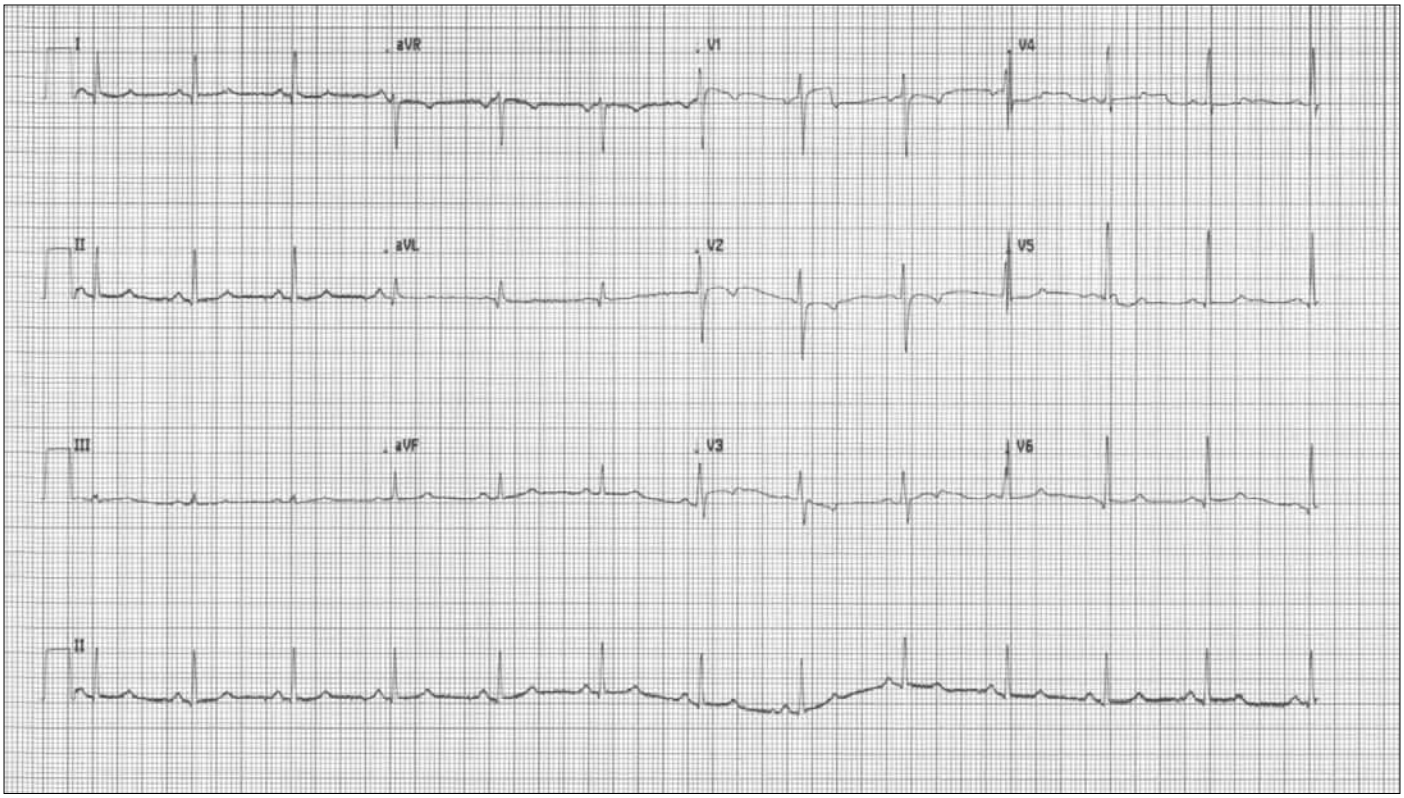

Figure 2. ECG findings after diabetic ketoacidosis treatment

including ECG abnormalities (3). Precipitating causes such as sepsis or myocardial infarction may lead to diabetic ketoacidosis in diabetic patients (4).

In our literature search, there are many states mimicking acute coronary syndrome. In a study from Taiwan 25 patients with pheochromocytoma had an abnormal ECG that suggested an acute coronary syndrome, but all of them had normal coronary arteries (5). The other reported state was cerebral arteiovenous malformation. ST elevations have been shown in precordial leads but no coronary lesion was found (6). Also, ECG abnormalities like acute coronary syndrome have been shown in subarachnoid hemorrhage (7).

The patient described in our case report had severe emesis, hyperglycemia, dehydration with hypokalemia, and persistent acidosis caused by diabetic ketoacidosis.

For differential diagnosis of ECG abnormalities in these cases, troponin levels can be accepted as valuable markers. In this case we had seen troponın I levels within normal limits. A life threatening situation such as myocardial infarction, should not be overlooked so, in order to monitor the patient status, we followed her in the coronary intensive care unit. The patient's hyperglycemia was resistant to insulin infusion and at first it was considered to be due to myocardial infarction, also other possible causes of insulin resistance such as infection, medication, electrolyte imbalances were evalauted.

In the presented case, higher doses of insulin were used to provide euglycemia. Prolonged profound ketoacidosis with emesis and insulin infusions can lead to severe hypokalemia. There have been only occasional reports of hypokalemia causing electrocardiographic changes closely resembling those of acute myocardial infarction.

It is therefore very important to detect changes in serum potassium levels early on in DKA in order to prevent the above mentioned complications. However, symptoms of even severe hypokalemia may mimic symptoms of the underlying disease and therefore may not be recognized in a critically ill patient.

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