LETTER TO THE EDITOR

Hyperkalemic Cardiac Arrest in a Patient with Diabetic Ketoacidosis

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Abstract

Aim: To highlight the occurrence of cardiac arrest due to hyperkalemia in diabetic ketoacidosis (DKA).

Background: Diabetic ketoacidosis is a commonly encountered condition. These patients can have normal or mildly elevated levels of potassium. Our patient had severe hyperkalemia due to DKA resulting in cardiac arrest. Her high potassium diet and use of angiotensin receptor blocker along with acute kidney injury (AKI) would have also contributed to hyperkalemia.

Case description: A 58-year-old female, known case of diabetes mellitus on insulin therapy and hypertension on telmisartan, presented with nausea, vomiting, and abdominal pain. She was diagnosed to have DKA with AKI precipitated by missed insulin and urinary tract infection. She was also on high potassium diet. Her electrocardiogram showed sinus bradycardia with prolonged QRS interval. Her potassium levels were elevated. She soon went into asystole and cardiac arrest and was resuscitated. Diabetic ketoacidosis protocols were followed along with antibiotics, and the patient improved.

Conclusion: Severe hyperkalemia in DKA is uncommon, and this hyperkalemia resulting in cardiac arrest is an unreported scenario. Potassium correction along with DKA management protocol forms the mainstay of treatment.

Clinical significance: Mild to moderate elevation in serum potassium occurs frequently in DKA. However, severe hyperkalemia is uncommon and is likely to be the result of insulin deficiency, acidosis, hyperosmolality, severe dehydration, and renal potassium retention. Such elevated level of potassium requires urgent correction in order to prevent cardiac arrest.

Keywords: Cardiac arrest, Diabetes, Diabetic ketoacidosis, Hyperkalemia, Insulin, Urinary tract infection.

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Dear Sir,

A 58-year-old female was brought at night to the emergency department with complaints of recurrent vomiting, nausea, and diffuse abdominal pain since morning. She was a diabetic on insulin therapy and hypertensive on telmisartan (40 mg once daily) for the past 10 years. She had changed her diet plan to only fruits (pomegranate, bananas, apple, orange, and watermelon), salad (cucumber, carrots, lettuce, and olives), tender coconut water, and soup (chicken, mushroom, tomato) for the past 5 days. During this period she stopped insulin but continued telmisartan.

On presentation, she was conscious, oriented, and dehydrated. Her pulse was 60 minute and blood pressure 100/60 mm Hg. She had Kussmaul breathing with respiratory rate of 32 minute (saturation 94% room air). She had diffuse abdominal pain with mild guarding (no rigidity). Other systemic examinations were normal.

Her blood glucose level was 412 mg/dL, and arterial blood gas (ABG) report revealed pH 7.04, pCO_2 15 mm Hg, HCO_3 10 mEq/L, and anion gap of 19 mEq/L. Sinus bradycardia with prolonged QRS interval was seen on her electrocardiogram (ECG). Her blood investigations showed hemoconcentration (hemoglobin 17.2 g/dL and hematocrit 51%), leukocytosis (WBC 12,400 cells/mm^3 with neutrophils 80% and lymphocytes 20%), mildly elevated renal parameters (urea 74 mg/dL and creatinine 1.85 mg/dL), mild hyponatremia (129 mEq/L), and hyperkalemia (8.5 mEq/L). Her liver functions, calcium, magnesium, and ammonia were normal. Serum amylase was mildly elevated (100 U/L), but lipase was normal. Her ECG (on cardiac monitor) suddenly showed asystole, and patient went into cardiac arrest. Cardiopulmonary resuscitation was carried out, and the patient was put on mechanical ventilation. A single dose of intravenous sodium bicarbonate (50 mEq) was given along with multiple doses of intravenous 10% calcium gluconate (10 mL every 30 minutes for 2 hours) and salbutamol nebulization (10 mg every 2 hours for 4 hours). Urine ketones were positive (4+), with pus cells (20–25 HPF) and no hematuria. Serum β-hydroxybutyrate was elevated (4 mg/dL). Intravenous ceftriaxone was started. Insulin infusion and fluid management was carried out according to diabetic ketoacidosis (DKA) protocol. Echocardiography, ultrasound abdomen, and chest X-ray were normal. Her repeat electrolytes (after 4 hours) showed sodium 131 mEq/L, potassium 6.7 mEq/L, and bicarbonate 16 mEq/L. Her repeat ECG (after 2 hours) was normal. After about 8 hours, the patient became conscious and was obeying commands, with stable vitals and ECG. Her blood glucose levels were 200 mg/dL, sodium 134 mEq/L, potassium 4.2 mEq/L, bicarbonate 23 mEq/L, and pH of 7.37 on ABG. Serum β-hydroxybutyrate reduced to 2 mg/dL. She was extubated, and

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oral diet was started. Insulin infusion was stopped and changed to subcutaneous insulin (basal bolus regimen). Her urine culture grew *Escherichia coli*, and intravenous ceftriaxone was continued (as per antibiotic sensitivity report). Her coronary angiogram was normal. She was discharged on day 5 of admission and educated not to skip insulin and avoid high-potassium diet. Telmisartan was stopped and changed to oral amlodipine (5 mg once daily). On review after 1 week, her vitals were stable, with normal electrolytes, renal functions, and blood glucose levels.

Diabetic ketoacidosis is an acute life-threatening condition precipitated by inadequate insulin, infection, and infarction. The diagnosis requires hyperglycemia (blood glucose 250–600 mg/dL), ketosis (elevated plasma ketones), and metabolic acidosis (pH 6.8–7.3, bicarbonate <15 mEq/L, high anion gap).[1]

In DKA, the potassium levels may be normal or mildly elevated. However, severe hyperkalemia is rare. In DKA, the lack of insulin produces hyperglycemia and also causes the shift of potassium from the intracellular to extracellular space by reducing Na^+ K^+ ATPase activity. Since there is no proper metabolism of glucose, ketones get produced and the pH decreases with increase in H^+. The body tries to compensate by exchanging the intracellular K^+ for extracellular H^+, thereby increasing serum potassium levels. Moreover, due to acute kidney injury, there will be reduced renal excretion of potassium.[2] Telmisartan, an angiotensin receptor blocker, can cause hyperkalemia by inhibition of renin-angiotensin-aldosterone axis leading to decreased potassium excretion.[3]

Our patient was a diabetic on insulin therapy, which she stopped for few days. She also had urinary tract infection (UTI). Due to missed insulin and UTI, she developed DKA. She was found to have hyperkalemia as a part of DKA, which was aggravated by her potassium rich diet, acute kidney injury and use of telmisartan. The aftermath was hyperkalemia-induced cardiac arrest. With less than a handful of cases of severe hyperkalemia in DKA being observed, this scenario is uncommon.[4–8] And the occurrence of cardiac arrest, to the best of our knowledge, has not been reported yet. Patients should be well educated regarding consequences of skipping their antidiabetic medications. Also, they should consult their doctor before starting a diet plan.

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