Hyperkalemia and Cardiac Glycosides

Justin Davis, DO and Kristopher Hart, DO

Integris Health Emergency Medicine Residency, Oklahoma City, Oklahoma

Case

A 60-year-old man with a history of hypertension, congestive heart failure, atrial fibrillation, and type II diabetes mellitus presented to the emergency department (ED) with generalized weakness. He had awoken a few hours prior with generalized weakness, three episodes of emesis, and four loose bowel movements. He attributed his GI complaints to some shellfish that he had for dinner the previous evening. He specifically denied any abdominal pain, chest pain, or shortness of breath. The patients home medications included lisinopril 20mg daily, diltiazem ER 240mg daily, coumadin 5mg daily, furosemide 80mg twice daily, spironolactone 25mg daily, potassium chloride 20mEq twice daily, digoxin 0.25mg daily, and pioglitazone 45mg daily.

VS: P 87 bpm, BP 122/48 mmHg, RR 16 bpm, SPO_2 99%, and T 97.7 F

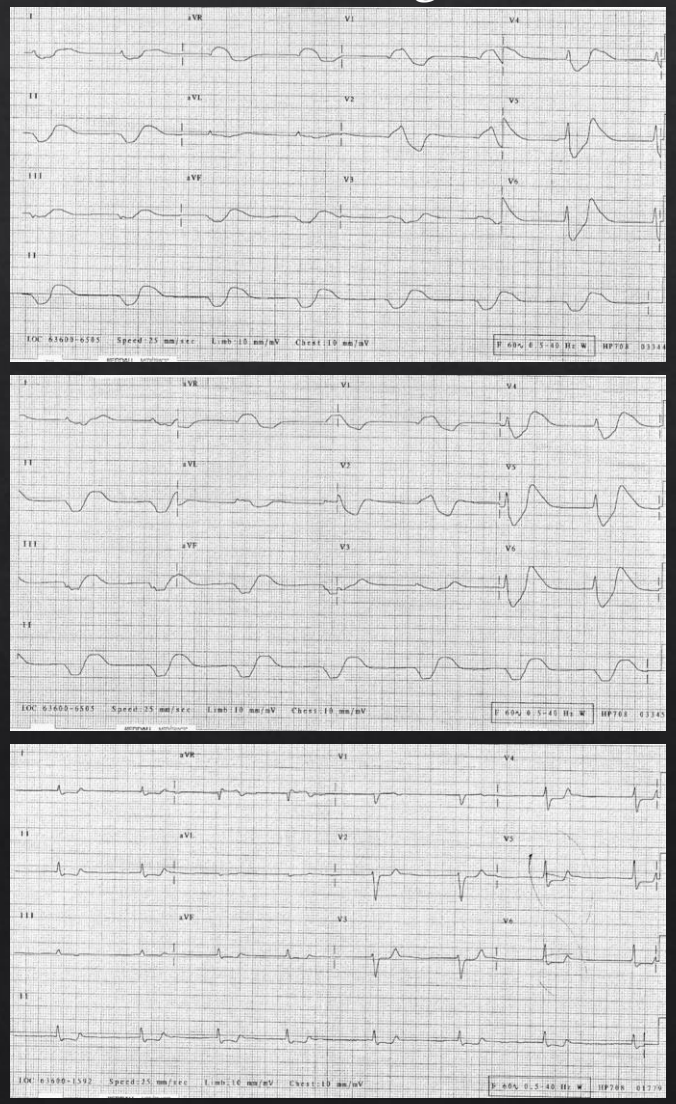
Physical examination was notable for a euvolemic-appearing patient in no acute distress with clear lungs, a grade II/VI systolic murmur at the left upper sternal border, absence of JVD, and benign nontender abdominal exam with normal bowel sounds.

An ECG was performed after initial history and physical exam (Figure 1).

Clinical Course

After obtaining the ECG, empiric therapy for hyperkalemia was initiated with 40 mg furosemide IV, 1000 mL normal saline IV bolus, 50 mEq sodium bicarbonate IV, 10 units regular insulin IV, 25 g dextrose IV, 10 mg albuterol by nebulizer, and 30 g sodium polystyrene sulfonate PO. Repeat ECG (Figure 2) after this treatment showed an increased rate of 43 beats/min, but was otherwise unchanged. The patient was noted to have remained hemodynamically stable during this time and an AP chest x-ray was obtained which showed no acute cardiopulmonary abnormality.

Electrocardiograms



Basic metabolic panel was obtained which showed a sodium of 127 mmol/L, potassium of 8.6 mmol/L, chloride of 103 mmol/L, carbon dioxide of 20 mEq/L, BUN of 56 mg/dL, creatinine of 5.1 mg/dL, and glucose of 164 mg/dL. Troponin I was <0.06 ng/mL. The patient's INR was 2.3. A serum digoxin level was resulted as 4.1 ng/mL. Given the elevation of the patient's serum digoxin level as well as significant hyperkalemia, six vials (240 mg) of digoxin immune Fab were given. The patient was noted to remain hemodynamically stable and resting comfortably in the room in no acute distress. A third ECG (Figure 3) was taken which showed atrial fibrillation at 47 beats/min. QRS duration had shortened to 126 mS. There were still diffuse ST-segment depressions with mild ST-segment elevation in aVR and the T-waves had normalized.

Discussion

This patient presented with generalized weakness, nausea, vomiting, and diarrhea which led to diagnoses of acute renal failure, hyperkalemia, and digoxin toxicity. The case highlights the importance of thoroughly exploring the precipitating cause of acute hyperkalemia in the ED, especially when the patient does not respond as expected to empiric therapy. The patient's swift improvement after digoxin immune Fab administration reinforces the importance of medication history-taking and understanding the potential effects of those medications on a patient's clinical presentation.

Digoxin directly inhibits the transmembrane sodium potassium ATPase pump, increasing concentrations of both intracellular sodium and extracellular potassium. The resultant increase in intracellular sodium has a negative feedback on another transmembrane channel, the sodium-calcium antiporter. This increases intracellular calcium concentrations and is largely responsible for the desired therapeutic effects of digoxin. Digoxin has a very narrow therapeutic index, is primarily eliminated through the kidneys (60-80%), and small increases in serum concentration can have profound toxic effects. Hyperkalemia potentiates the cardiotoxic effects of digoxin, even at therapeutic levels.

INTEGRIS