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Lady Stone Heart?

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Background: Digoxin's inhibition of the sodium/potassium ATPase pump contributes to its therapeutic and toxic effects. The presence of hyperkalemia is predictive of mortality in acute digoxin toxicity cases, and the treatment of hyperkalemia with intravenous (IV) calcium in this setting is controversial.

Case Report: A 71-year-old female with a history of atrial fibrillation, pulmonary hypertension, and coronary artery disease presented to the ED after syncope. Her vital signs were normal and examination demonstrated generalized weakness, altered mental status, tremors, and abdominal pain. She was found to have an acute kidney injury [BUN 64 mg/dL, SCr 1.43 mg/dL] and hyperkalemia [6.1 mEq/L]. ECG demonstrated T wave inversions and ST depressions, and an elevated troponin [0.105 ng/mL]. Head CT was unremarkable. She received insulin IV, kayexelate PO, and calcium gluconate IV for the hyperkalemia. Within minutes of receiving calcium, the patient had a transient episode of cardiac arrest that resolved without intervention, and was subsequently found to have an elevated digoxin level [4.3 ng/mL]. Repeat ECG showed worsening ST depressions but down trending troponin [0.086 ng/mL] and no chest pain. She received digoxin immune fab therapy and symptoms of digoxin toxicity resolved. There were no other complications during her hospitalization and she was discharged home when her renal function improved.

Discussion: The treatment of hyperkalemia with calcium IV in digoxin toxicity may result in a state of myocardial tetany or "stone heart" due to elevated intracellular calcium levels in the cardiac myocytes. Animal studies appear to disprove the "stone heart" theory. Historically, the few human case reports of "stone heart" were incomplete and the largest retrospective study to date did not find an increase in dysrhythmias or mortality when calcium was given for hyperkalemia in chronic digoxin toxicity cases. Our case suggests that calcium induced cardiac arrest in the setting of digoxin toxicity. The exogenous calcium administration likely worsened the digoxin toxicity's cytoplasmic hypercalcemia, which is associated with dysrhythmias.

Conclusion: This case highlights the need for caution when giving calcium IV in patients with digoxin toxicity who do not have ECG findings suggestive of hyperkalemia.