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Digoxin Toxicity and Acute Renal Failure in a 75 Year-Old Female

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Introduction:

Digoxin toxicity can present with varying manifestations. While pathognomonic symptoms such as xanthopsia (object appearing yellow) are a board favorite it is not a required finding and is in fact not seen with most patients. Rather digoxin toxicity presents with more non-specific symptoms such as GI distress (anorexia, N/V) neurological distress (lethargy, fatigue, delirium, confusion, disorientation, weakness. [1] EKG findings are varied and include premature ventricular contractions, bradycardia, atrial tachyarrhythmias with AV block, ventricular bigeminy, junctional rhythms, various degrees of AV nodal blockade, ventricular tachycardia, and ventricular fibrillation. Although rarely seen, digoxin is one of the only causes of bidirectional ventricular tachycardia [2]. Digoxin levels should be ordered when a patient presents with any of the above symptoms, especially if they have a history or renal disease. In the following case the patient had only recently began taking digoxin a couple weeks prior for new onset a fib and had a history of chronic kidney disease secondary to polycystic kidney disease with bilateral renal transplants 12 years prior.

Case Presentation:

The patient is a 75 year old female with the past medical history of recent CVA, recently diagnosed A fib, orthostatic hypotension, diabetes, high output ileostomy secondary to ulcerative colitis and bilateral renal transplant in 2010 presenting to the emergency department for evaluation of generalized weakness, chest pain and lightheadedness. She states these symptoms have been ongoing for the last 2 weeks and increasing in severity. She describes the chest pain as a sharp and intermittent with episodes occurring for approximately 10-15 mins in the center of her chest that does not radiate. The day she presented to the ED she had PT at her home for rehab for her CVA and the therapist noted she was in a fib and hypotensive. From there she was recommended to go to the ED per her PCP. Of note she recently began taking digoxin for a fib and states that the symptoms she was complaining about began approximately one week after. She endorses having a difficult time keeping herself adequately hydrated as she loses so much volume from her ileostomy.

On presentation her BP was 124/58, Pulse 82, temp 97.9, SPo2 98% RR 18. Her physical exam yielded normal heart sounds with a regular rhythm, Lungs clear to auscultation, non tender abdomen and existing deficits from her stroke 3 months prior.

Laboratory work up revealed electrolyte disturbances of Sodium 123, Potassium 6.0, Chloride 88, BUN 84, Cr 2.63 with a baseline of 1.06 and a glucose of 237. Her initial troponin was 41.0 with a repeat of 44.0. Her BNP was 77. Her digoxin level was 4.1 with the therapeutic range of 0.8-1.5 ng/ml. Her CBC was unremarkable. Imaging consisted of a 2 view Chest X ray that was unremarkable.

ECG noted Sinus rhythm with 1st degree heart block with a PR interval of 238. Her prior ECG taken 3 months prior showed a PR interval of 178. Also noted were scooped ST segments and a shortened QT.

Calcium gluconate was given upon potassium resulting. Nephrology was contacted who recommended lowering the K by insulin, bicarb, sodium polystyrene-sorbitol as well as 80 ml/hr NSS.

Cardiology was consulted in regards to digoxin level. Dig fab was not indicated as patient was not exhibiting any toxic EKG findings. Rather it was held and digoxin level was trended until it returned to a therapeutic level. Patient was admitted to Step down as she was on insulin drip. She was discharged from the hospital 3 days later.

Nephrology assessed AKI most likely pre-renal in the setting of high output ileostomy and hyponatremia likely hypovolemic. This was further supported by findings of Urine Na <10 and Urine osm 380.

Cardio restarted her digoxin on a every other day schedule at 0.125 once therapeutic levels were achieved.

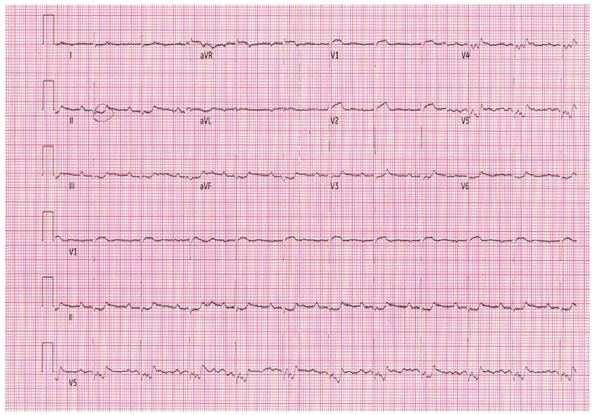
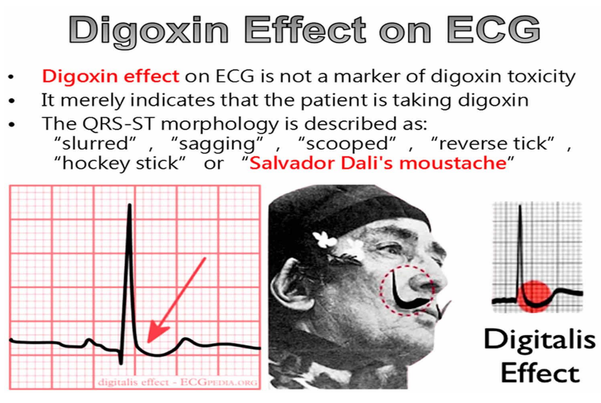


Figure 1: EKG of patient at time of presentation. Note the characteristic ST slope, shortened QT and prolonged PR interval. These are normal findings of a patient on digoxin and do not qualify as toxic indicators.



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Conclusions:

Patient's symptoms could not definitively be determined to be caused by digoxin though considering the levels, timing of symptoms to starting the medication and the fact that the symptoms subsided once the levels were decreased supports contributing to her illness. The fact she was in ARF at the time of presentation most likely also contributed to her presentation. Digoxin can often raise to toxic levels in patients with acute renal failure and in patient populations such as hers who are at risk for ARF (predisposed to prerenal syndrome secondary to high output ileostomy and renal transplant) care needs to be taken to monitor for digoxin levels, especially when first beginning the medication.

Discussion:

Digoxin toxicity is treated primarily with Fab (digoxin-specific antibody). Clinical indications for it include:

- Life-threatening arrhythmia (eg, ventricular tachycardia; ventricular fibrillation; asystole; complete heart block; Mobitz II heart block; symptomatic bradycardia) [36]
- Hyperkalemia (serum potassium >5 to 5.5 meq/L)
- Evidence of end-organ dysfunction from hypoperfusion (eg, renal failure, altered mental status) (3)

A level alone is usually not enough to necessitate Fab treatment, but some will advocate for giving Fab fragments if the serum digoxin concentration is greater than 10 ng/mL (13 nmol/L) at steady state in acute ingestions, or greater than 4 ng/mL (5.1 nmol/L) in chronic ingestions, or when an adult ingests more than 10 mg or a child more than 4 mg acutely.

Electrolyte abnormalities

Hyper and hypokalemia — Hyperkalemia accurately reflects the degree of toxicity and risk of death in **acute** digoxin intoxication. Hyperkalemia itself does not cause death, and treatment with potassium-lowering agents such as insulin and dextrose, sodium bicarbonate, or ion exchange resins does **not** reduce mortality [4].

Once treatment with digoxin-specific antibody (Fab) fragments is instituted, hyperkalemia due to cardiac glycoside toxicity is rapidly corrected as the sodium-potassium ATPase function returns and pumps potassium back into cells. Thus, aggressive treatment with potassium-lowering agents could cause significant **hypo**kalemia following antidotal therapy.

If the hyperkalemia is thought to be due to underlying renal failure, however, treatment of hyperkalemia with use of potassium-binding resins, sodium bicarbonate, or insulin/glucose may be utilized.

If a patient with digoxin poisoning is hypokalemic, potassium should be administered since hypokalemia exacerbates digitalis toxicity [1]. The need for potassium repletion is important for patients treated with Fab fragments, because this treatment leads to a further reduction in the serum potassium. Many patients with hypokalemia may have coexistent hypomagnesemia, which should be corrected as well.

Should calcium be administered for hyperkalemia? — Because hyperkalemia is not the cause of death and because excessive intracellular calcium is present in digoxin poisoning, it is not recommended to give calcium in hyperkalemic patients with recognized digoxin toxicity. In this setting, hyperkalemia is best treated with digoxin-specific antibody fragments.

References:

References available on request