The Digoxin Effect

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Case:
HPI: 84 yo female w/ PMH of CAD, HTN, DM, **A-fib**, and hyperlipidemia who arrives by EMS with a c/o an occipital HA after a fall. States that she fell from a standing position while attempting to go into her closet and that she did not slip/trip and fall but does relate that she "felt a bit woozy" just prior to the incident and that she fell backwards onto the floor and hit her head on the floor. Denies LOC; relates that she remembers all events pre and post incident. Relates that she was not down very long before her husband found her and called 911. States that she has been **nauseous and has vomited several times** over the past few days but that she has not vomited since the fall occurred. Denies any CP, SOB, diaphoresis, abdominal pain, dizziness, or blurry vision. Pt is a rather poor historian but and while she is unable to recall much of her medical Hx, she does state that she is on warfarin but she does not remember why. She also remembers that a doctor has told her in the past that she has a heart murmur but she is unsure of anything more than that.

Meds - **Coumadin**, NovoLog, **Digoxin**, Nitroglycerin, KCl, Protonix, ASA, Metoprolol, Cozaar, pravastatin.

Vitals; HR 60, BP 154/86, RR 14, Spo2 98%

ROS/PE: Positive for weakness, N/V, neck and occipital HA; PE unremarkable except for borderline **sinus bradycardia at 60 bpm w/ 1st degree AV block** and a grade IV-V/VI holosystolic murmur most prominent at the L upper sternal border (Neuro exam completely normal w/ no deficits).

Labs: CBC/CMP, EKG, Cardiac markers/Coag studies, Digoxin level, TSH, UA, CT head and C-spine

Significant results: Creatinine **1.30**, PT 42.7/INR 3.9, Troponin 0.05, CT negative for ICB or Fx. Since the important labs never show up until later… ***Digoxin level - 4.1*** *(therapeutic range 0.5-0.9)*

EKG shows Sinus Bradycardia w/ 1st degree AV block, ST changes noted in V leads with ischemic changes noted in I / AVL and inferior leads, Prolonged QT interval

Clinical question: ***What effects can Digoxin toxicity play on the body and what can be your common presenting S/S and findings?***

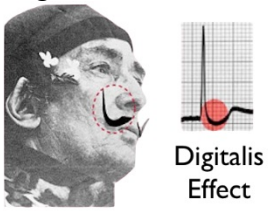
Common:
GI symptoms - Typically include nausea, vomiting, diarrhea, abdominal pain, or anorexia.
CNS symptoms - Typically include lethargy, weakness, and confusion
Cardiovascular symptoms - Typically include palpitations, syncope, arrhythmias, and dyspnea

Uncommon;
Visual Symptoms - Typically include disturbances of color vision with a tendency to perceive yellow halos around objects (xanthopsia), blurred vision, and diplopia.

Digoxin Toxicity Risk Factors
Age >55yo
Decreased renal clearance (our pt's creatinine was 1.30)
Hypo/Hyperkalemia
Concomitant use of **drugs** that decrease digoxin clearance
Hypomagnesemia
Hypercalcemia
Hypothyroidism - Recognized risk factor for developing digoxin toxicity

Digoxin Mechanism of action
Digoxin binds to a site on the extracellular aspect of the α-subunit of the **Na+/K+ ATPase pump** in the membranes of heart cells (myocytes) and decreases its function. This causes an increase in the level of sodium ions in the myocytes, which leads to a **rise in the level of intracellular calcium** ions. Increased intracellular calcium lengthens phase 4 and phase 0 of the cardiac action potential, which leads to a **decrease in heart rate**. Increased amounts of Ca2+ also leads to increased storage of calcium in the sarcoplasmic reticulum, causing a corresponding increase in the release of calcium during each action potential. This leads to **increased contractility** (the force of contraction) of the heart without increasing heart energy expenditure.

Digoxin has a characteristic effect on the ST segment in EKGs.



The morphology of the **QRS complex / ST segment is variously described as either "slurred", "sagging" or "scooped"** and resembling either a "reverse tick", "hockey stick" or "Salvador Dali's moustache"!

The most common T-wave abnormality is a biphasic T wave with an initial negative deflection and terminal positive deflection. This is usually seen in leads with a dominant R wave (e.g. **V4-6**). The first part of the T wave is typically continuous with the depressed ST segment. The terminal positive deflection may be peaked, or have a prominent U wave superimposed upon it.
Sagging ST segments are most evident in the lateral leads **V4-6, I and aVL.*

The treatment… Digibind!
If the intoxication is acute, activated charcoal "can" be used, but the standard of care of symptomatic digoxin toxicity is **digoxin immune Fab (aka Digibind)** and addressing any underlying electrolyte abnormalities. Patients who receive digibind have a **drop in serum K+** as it moves intracellularly. Some pts who have also been treated for hyperkalemia and also received digibind develop profound hypokalemia as a result, therefore, **serial K+ measurements** are necessary. After administration of digibind, serum digoxin concentrations are **usually falsely**

elevated (10-30 fold). Serum digoxin concentration can be **measured again in 3-4 days after dose is given but has been reported to be elevated for up to 10 days**, especially in pts with renal insufficiency.

Conclusion:
- Know the S/S of digoxin toxicity as they often **masquerade as vague nonspecific complaints and often do NOT include Chest pain**.
- Be able to **keep a broad differential when pts present with multifactorial problems**. Do NOT tunnel vision on the problem at hand… there may always be other factors at play.
- **Recognize the EKG effects of Digoxin** and keep a high index of suspicion with pts taking Digoxin.

References:
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