Title

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CLINICAL VIGNETTE

A Case of Digoxin Toxicity

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Presentation:

An 89-year-old female with paroxysmal atrial fibrillation, chronic kidney disease, hypertension, diastolic heart failure, and hypothyroidism, presented to the Emergency Department from a nursing home with confusion. She had recently been hospitalized 1 month prior for failure to thrive, manifested by increasing weakness and anorexia. These symptoms were attributed to worsening arthritis and hypothyroidism. During hospitalization she was initiated on bumetanide, and she was discharged to a skilled nursing facility for physical therapy.

One week prior to presentation she followed up with her primary doctor with complaints of gastrointestinal upset. Upon presentation to the Emergency Department she denied recent falls, lightheadedness, syncope, chest pain, or shortness of breath. Medications in the nursing home included digoxin (0.25 mg oral daily), bumetanide, vitamin D, lisinopril, iron sulfate, and omega-3 fish oil. Physical exam was notable for a bradycardic rate in the 30s, blood pressure of 110/60, grade II systolic ejection murmur, and bilateral lower extremity ulcers. Laboratory abnormalities included creatinine of 2.0 (0.5-1.3), urea nitrogen of 108 mg/dL (7-23), with estimated GFR 23, digoxin level of 4.2 ng/mL (0.5-1.1), TSH of 39.6 mcIU/mL (0.3-4.7), and normal potassium and magnesium levels. Urinalysis revealed evidence of a urinary tract infection.

Hospital Course:

In the Emergency Department the patient's heart rate dropped to 30 with the development of complete heart block (see Figure 1). The patient failed to respond to atropoine, received 2 vials of Digibind, and she was started on a dopamine drip. Her AV conduction improved to Mobitz type I second-degree AV block (see Figure 2).

She was admitted to the ICU for telemetry monitoring. Over the next few hours telemetry revealed sinus bradycardia with first-degree AV block (see Figure 3), 2:1 AV block (see Figure 4), complete heart block (see Figure 5), and frequent bigeminy. Her acute kidney injury was attributed to prerenal azotemia, likely due to overdiuresis, and her kidney function improved with gentle IV fluid hydration. Her urinary tract infection was treated with piperacillin/tazobactam. Dopamine support was weaned on hospital day 5 and the patient was eventually discharged from the hospital without requiring a pacemaker.

Figures:

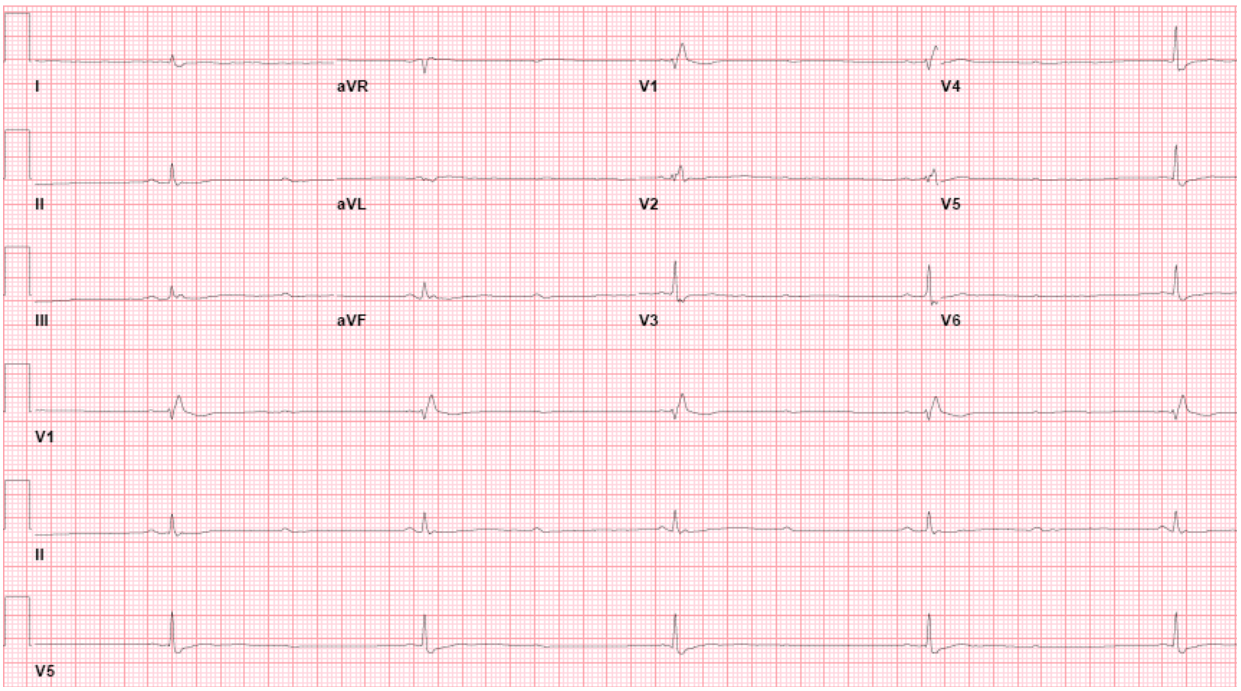

Figure 1: Electrocardiogram demonstrating complete heart block.

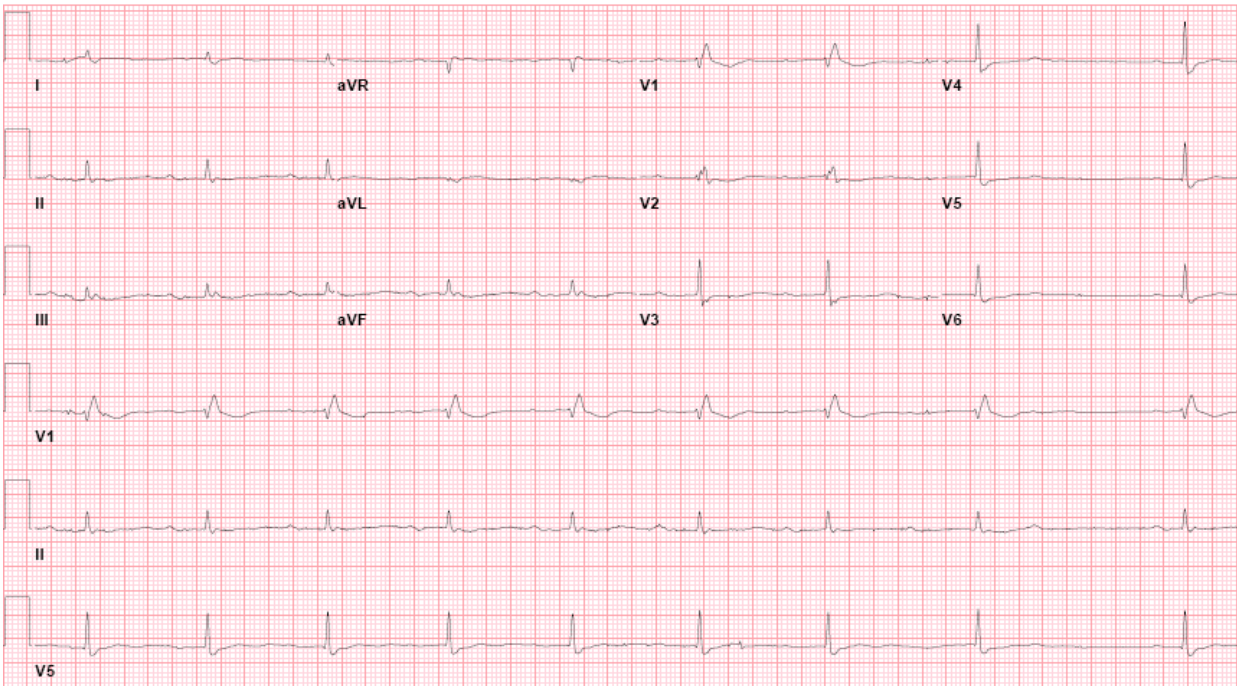

Figure 2: Electrocardiogram demonstrating Mobitz I second-degree AV block.

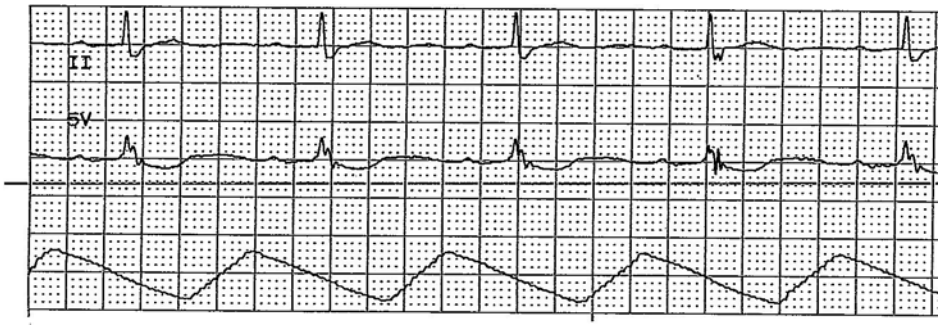

Figure 3: Telemetry strip demonstrating first-degree AV block. Note the scooped appearance in lead V5 consistent with digoxin effect.

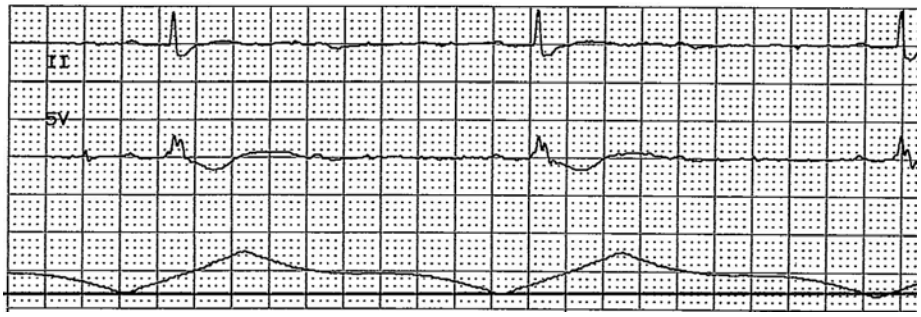

Figure 4: Telemetry strip demonstrating 2:1 AV block.

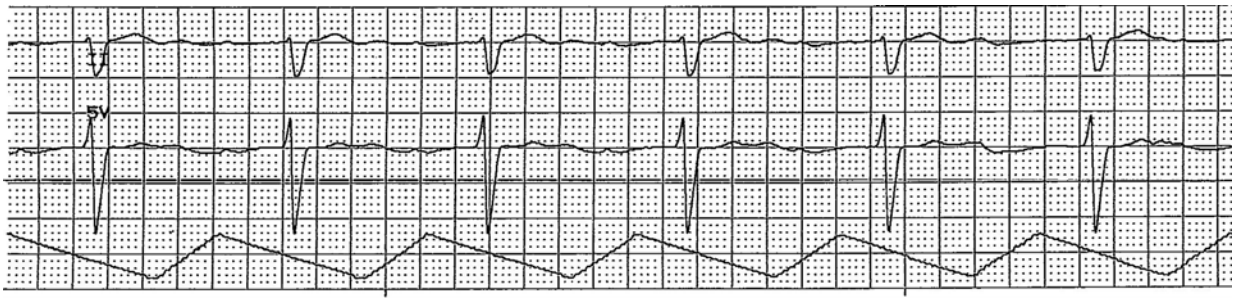

Figure 5: Telemetry strip demonstrating complete heart block. Note the change in QRS morphology consistent with ventricular escape rhythm.

Discussion:

Digoxin inhibits the sodium-potassium adenosine triphosphatase pump, causing a rise in intracellular calcium and sodium levels. This mechanism is responsible for both the inotropic and arrhythmogenic properties of digoxin. Since digoxin increases the automaticity of Purkinje fibers but slows conduction through the AV node, the combination of increased automaticity and decreased conduction (eg, paroxysmal atrial tachycardia with 2:1 block, accelerated junctional rhythm, or bidirectional ventricular tachycardia) is highly suggestive of toxicity. It should be noted however, that digoxin toxicity may cause any dysrhythmia except rapidly conducted atrial fibrillation[1].

Extracardiac manifestions of digoxin toxicity are nonspecific and common. Fatigue and anorexia followed by gastrointestinal symptoms of nausea and vomiting. Neurologic complaints of headaches and

confusion may appear. A more specific but less common symptom is visual changes, in particular halos around bright objects and increased shades of green and yellow[2,3]. Some believe that Van Gogh's work was influenced by digoxin toxicity[4].

Unfortunately there is no clear relationship between the serum digoxin level and digoxin toxicity. This is partly due to the fact that toxicity is related to intracellular levels, not serum levels. Worsening renal function, electrolyte abnormalities (particularly hypokalemia, hypomagnesemia, or hypercalcemia), pulmonary disease, and hypothyroidism have all been associated with toxicity[2,3]. For example, a patient with normal digoxin levels (0.5-2 ng/mL) but impaired renal function or severe hypokalemia may have cardiotoxicity while a patient with high digoxin levels and no renal or electrolyte disturbances may be asymptomatic[3].

In cases of cardiotoxicity due to chronic ingestion (as in our patient), treatment options include withdrawal of the medication, correction of electrolyte abnormalities, atropine, temporary pacing, and digoxin Fab fragments[3]. Digoxin-specific antibody fragments avidly bind digoxin and are excreted renally. Indications for digoxin Fab fragments include potentially life-threatening cardiotoxicity including complete heart block, severe bradycardia unresponsive to atropine, and severe ventricular dysrhythmias. Each vial of digoxin Fab fragments binds approximately 0.5 mg of digoxin. Since most digoxin radioimmunoassays measure total digoxin levels rather than free digoxin levels, serum digoxin levels are no longer useful after Fab administration. Digoxin levels may stay elevated for as long as a week in patients with chronic kidney disease and poor urinary excretion[5].

In conclusion, this case demonstrates a classic presentation of digoxin toxicity. Multiple risk factors for digoxin toxicity including age, impaired renal function, and hypothyroidism were present. The worsening renal function from diuretic therapy with continued digoxin at 0.25 mg was likely the cause of digoxin toxicitiy which first manifested by increasing weakness, anorexia, and gastrointestinal upset. The patient then developed confusion and eventually complete heart block requiring administration of digoxin Fab fragments. Clinicians should be mindful that chronic digoxin toxicity is believed to occur in 4-10% of patients on digoxin, but only suspected in 0.25% of cases[6].

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