

Two Cases of Digoxin Intoxication: Insights into ECG Findings and Visual Disturbances

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Abstract

Digoxin, a widely prescribed cardiac glycoside, is known for its therapeutic benefits in managing various cardiac conditions. However, its narrow therapeutic index poses a significant risk of toxicity. We present two cases of digoxin intoxication with pathognomonic electrocardiographic (ECG) findings and visual disturbances. The detailed analysis of ECG abnormalities and visual disturbances associated with digoxin toxicity will aid in prompt diagnosis and appropriate management, ultimately improving patient outcomes.

Keywords: Digoxin intoxication, electrocardiography, color vision disturbance

Introduction

Digoxin is a medication derived from the foxglove plant (*Digitalis purpurea*). It belongs to a class of drugs known as cardiac glycosides. Digoxin exerts its effects primarily on cardiac tissue by inhibiting the sodium-potassium ATPase pump. By inhibiting this pump, digoxin leads to an increase in intracellular calcium levels. This, in turn, enhances cardiac contractility (positive inotrope) and improves electrical conduction through the heart (negative chronotropic and decreased atrioventricular conduction). Digoxin is commonly used in the management of heart failure (HF), rate control of atrial fibrillation (AF), and atrial flutter (1-5).

The pharmacokinetics of digoxin can be influenced by various factors, which can impact the risk of toxicity. Some of these factors include renal function, drug interactions, electrolyte imbalances, age, and body weight (3).

While digoxin offers therapeutic benefits, it also carries a significant risk of toxicity. This risk arises from the narrow therapeutic index of digoxin. Digoxin toxicity can lead to

severe and even life-threatening complications, including cardiac arrhythmias, central nervous system disturbances, and visual disturbances (1-7). Therefore, recognizing the early signs and symptoms of digoxin toxicity is crucial for patient safety and optimal therapeutic outcomes.

Case Reports

Case-1

A 69-year-old female patient was admitted to the emergency department with nausea and vomiting. Medical history revealed she had coronary artery disease, low cardiac output, atrial fibrillation, and hypertension. She was on rivaroxaban, metoprolol, digoxin, valsartan, spironolactone, and furosemide. Her vital signs were: blood pressure: 116/77 mmHg, pulse rate: 47 bpm (irregular), body temperature: 36.6 °C, and oxygen saturation 97% at room air. Her physical and neurological examinations were normal other than rare fine rales on lung auscultation and minimal pretibial edema. Her ECG is shown in figure 1.

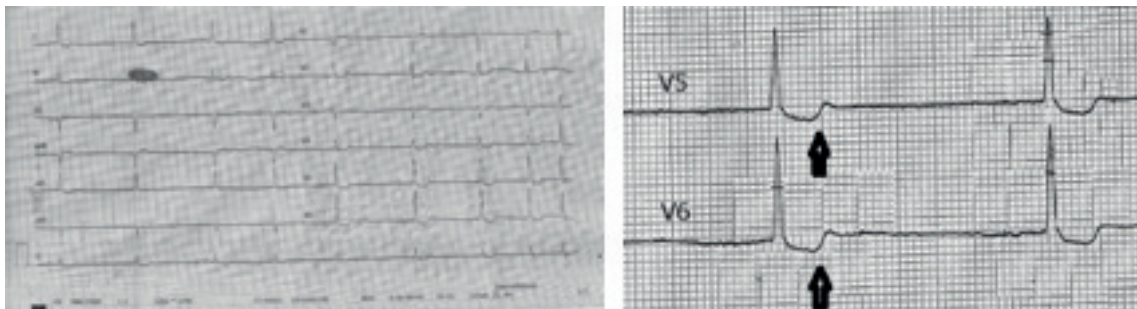


Figure 1. 12 lead ECG of the first case; atrial fibrillation with ST-segment depressions at I, AVL, V4, V5, V6. At a closer look (small figure), scooping of ST segments (Dali's mustache sign) is evident (arrows).

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Upon examining the ECG in light of the patient's history and findings, digoxin level was ordered in suspicion of digoxin intoxication. Her digoxin level was 3.31ng/ml (reference range: 0 – 2 ng/ml) and other laboratory tests (renal and hepatic function tests, Na, K, Ca, and troponin) were normal. She was admitted to the coronary care unit and digoxin was stopped. Medications other than digoxin were continued. She was monitored for possible life-threatening arrhythmias and none were observed. Nausea and vomiting were controlled with metoclopramide and ondansetron on different occasions. After the second day of hospitalization, nausea and vomiting were stopped. Digoxin levels were followed daily. By the end of the seventh day, her digoxin level was 0.81ng/ml, she was asymptomatic and discharged from the hospital.

Case-2

An 83-year-old female patient was admitted to the emergency department with nausea and vomiting, palpitations, and blurred vision. She had coronary artery disease, heart failure, diabetes mellitus, hypertension, and atrial fibrillation. She was on diltiazem, digoxin, and furosemide. Her vital signs were: blood pressure: 113/92 mmHg, pulse rate: 44 bpm (irregular), body temperature: 36°C, and oxygen saturation 98% at room air. Her physical and neurological examinations were normal. During the visual examination, she stated that she had green-red flying visions for a few days. ECG was atrial fibrillation with a slow ventricular response (Figure 2). Her digoxin level was 4.14ng/ml. At the emergency department follow-up she had an episode of hypotension and severe bradycardia (mean heart rate was 32 and blood pressure was 80/60mmHg) which responded well to 1 mg of intravenous atropine and hydration. She was admitted to the coronary care unit and followed up closely. After admission, she received controlled intravenous hydration, and metoclopramide for nausea and her medications other than digoxin were continued. No life-threatening arrhythmias were observed during the follow-



Figure 2. ECG of the second case: Atrial fibrillation with slow ventricular response and non-specific ST segment and T wave changes.

up. Digoxin levels decreased gradually together with the symptoms and she was discharged after 8 days of admission.

Discussion

Although common indications of digoxin are narrowed mainly to HF and AF it is still widely prescribed. As HF and AF's incidence increases with age, digoxin is prescribed mainly to a digoxin intoxication susceptible population. Furthermore, the number of accompanying diseases and polypharmacy increases in this group of patients which also increases the risk of digoxin toxicity (4). We have presented 2 cases of digoxin intoxication with pathognomonic findings of Dali's mustache sign on ECG and colored floaters symptoms.

ECG is a rapid, powerful, non-invasive, and cheap test that helps physicians determine not only cardiac conditions but also conditions that affect myocardial electrical activity. Including electrolyte abnormalities and intoxications such as digoxin toxicity (7). In the therapeutic range, digoxin has a positive inotropic effect by inhibiting Na/K ATPase and increasing the myocardial concentration of calcium. Digoxin also affects the vagal system and decreases heart rate. In toxic doses, various ECG abnormalities including first-degree AV block, extrasystoles, supraventricular tachycardias, and other arrhythmias may be observed. As well as arrhythmias, characteristic scooping of the ST segment resembling famous painter Salvador Dali's mustache may be observed (Figure 1). Getting familiar with this pattern may help prompt recognition of digoxin intoxication (3,7).

The second case we presented was admitted to the emergency department with non-specific nausea and vomiting. After a thorough medical history and examination, a history of visual green-red colored floaters suggesting digoxin intoxication was acquired, and a laboratory test for digoxin level was ordered. Various visual disturbances related to digoxin use have been reported in the literature including snowy vision, flashing and flickering lights, flowerlike figures, green and yellow vision, and colored floaters. Many mechanisms including the central nervous system, optic nerve, and retinal photoreceptor toxicity of digoxin, were suggested as the cause of the optical effects of digoxin (6). Independent of the mechanism, visual disturbances attributed to digoxin tend to increase with age and multidrug use (6). A history of visual disturbances combined with digoxin use should prompt suspicion of digoxin toxicity.

Management of digoxin intoxication, whether chronic or acute, primarily targets the stabilization of the patient by controlling arrhythmias, CNS manifestations, and gastrointestinal symptoms, correcting electrolyte imbalances, and improving renal function. Digoxin-binding antibodies were reported to reduce the mortality of digoxin intoxication from 20 – 30% to 5-8%. However, there are no clinical studies demonstrating a net clinical benefit.

Current indications for digoxin-binding antibody use are; life-threatening arrhythmias, cardiac arrest, hyperkalemia with serum potassium level of more than 5 mmol/L, end-organ dysfunction, or a digoxin level of more than 10ng/ml (acute intoxication), or 6 ng/ml (chronic intoxication) (2-4,7). Our cases were chronic intoxications and digoxin-binding antibodies were not indicated. They were managed symptomatically and responded well to cessation of digoxin.

Conclusion

Digoxin intoxication is a life-threatening condition that may easily be overlooked. Getting familiar with Dali's mustache ECG sign and colored floater symptoms may prompt a rapid diagnosis.

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