Case Report

Digoxin toxicity: A case report emphasizing the role of therapeutic drug monitoring

Johnson[1]*, Sherlin[2], Vignesh[2]

[1]Group Clinical Pharmacist, Kauvery Hospital, Trichy, Tamil Nadu
[2]Clinical Pharmacist, Heart City, Kauvery Hospital, Trichy, Tamil Nadu
*Correspondence

Abstract

Background: Digoxin has a narrow therapeutic spectrum (range), making toxicity a significant clinical concern. We report a case of an elderly patient with multiple comorbidities, including left ventricular dysfunction and atrial fibrillation, who developed digoxin toxicity, presenting with nausea, vomiting, fatigue, bradycardia and conduction abnormalities. Contributing factors included possible drug interactions, impaired renal function, and chronic therapy. Detecting the Serum digoxin levels will be helpful in confirming the diagnosis. Prompt recognition, withdrawal of digoxin, correction of electrolyte imbalance, and supportive management, which may include Digoxin Antibodies, are urgently required to achieve clinical stability and safety. This case highlights the critical role of therapeutic drug monitoring (TDM) in optimizing digoxin therapy, preventing toxicity, and guiding dose adjustments, especially in high-risk populations.

Key words: Digoxin; Bradycardia; Therapeutic drug monitoring; Digoxin antibodies.

Citation: Johnson, Sherlin, Vignesh. Digoxin toxicity: A case report emphasizing the role of therapeutic drug monitoring. Kauverian Med J. 2026;3(7):78—82.

Academic Editor: Dr. Venkita S. Suresh

ISSN: 2584-1572 (Online)

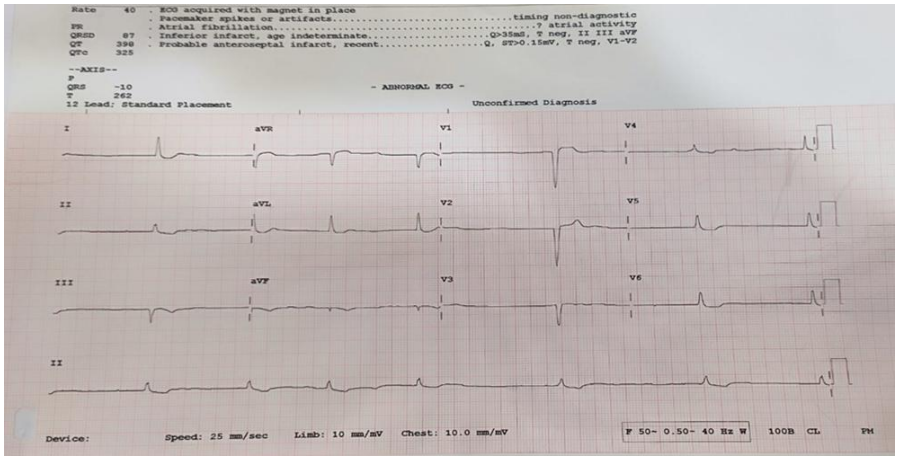
1. Introduction

Digoxin is a cardiac glycoside derived from the foxglove plant (Digitalis purpura). It has been widely used in the management of atrial fibrillation and heart failure. It inhibits the Na^+/K^+ ATPase pump, leading to an increase in intracellular sodium, which subsequently raises intracellular calcium levels through the sodium–calcium exchanger resulting in positive inotropy, improving myocardial contractility. At the same time, digoxin increases vagal tone, causing suppression of AV nodal conduction, which can manifest as severe bradycardia and varying degrees of conduction block.

2. Case Presentation

The 76-years-aged female, with a history of diabetes mellitus and hypertension, had a recent inferior wall myocardial infarction (IWI). Coronary angiography had revealed single-vessel disease with 100% proximal RCA occlusion. She underwent successful PCI to proximal–mid RCA with POBA to and thrombus aspiration on 08/01/2026. She presented a week later with breathlessness, nausea and vomiting, loss of appetite, melaena,

generalized fatigue. She was on Digoxin 0.25 mg BD, Bisoprolol + Amlodipine 5 mg/5 mg OD, Valsartan 40 mg BD, Amiodarone 100 mg BD and other routine medications.



ECG revealed severe bradycardia, with 'coving: of the ST segment" in inferior and anterior leads.

Clinical symptoms were suggestive of systemic toxicity. No immediate alternative clear cause of bradycardia identified initially.

ECG, with the classical changes described above, was suspected to be secondary to digoxin- toxicity, likely potentiated by concomitant amiodarone and Bisoprolol therapy, along with clinical features suggestive of toxicity.

3. Therapeutic Drug Monitoring (TDM) of Digoxin

Therapeutic monitoring is essential because digoxin has a narrow therapeutic index and a high risk of toxicity, especially in elderly or renally impaired patients. Therapeutic range of digoxin in heart failure is 0.5–0.9 ng/mL. Levels >2.0ng/ml are generally considered toxic, but toxicity can occur even at lower levels.

3.1. Key learnings

- Prevent toxicity in high-risk groups (elderly, renal impairment, polypharmacy)
- Aim for therapeutic benefit (HF and AF control)
- Be aware of early drug interactions (especially amiodarone)
- Adjust doses, guided by clinical changes
- Improve patient safety and avoid ICU admissions
- Suspect toxicity

3.2. When to Measure Digoxin Levels

- At steady state (after ~5–7 days of starting or dose change)
- At least 6–8 hours after the last dose (best: 12–24 hours post-dose for accuracy)
- When:

- Suspected toxicity
- Altered renal function
- Possible drug interactions
- Lack of therapeutic response

3.3. ECG Features in Digoxin Effect / Toxicity

- ST segment depression (scooped appearance or the curled moustache of artist Salvador Dalí, 'reverse tick' or 'hockey stick' shape)
- Shortened QT interval
- Flattened or inverted T waves
- Possible PR prolongation
- Arrhythmias in toxicity (AF, AV block, VT)

3.4. Important Clinical Point

- The scooped ST segment is a "digoxin effect", not always toxicity
- It can be seen even at therapeutic levels
- Toxicity is suggested when ECG changes are combined with:
  - Symptoms (nausea, confusion, visual changes)
  - Arrhythmias
  - Electrolyte imbalance (especially hypokalemia)

4. Factors Affecting Digoxin Levels

4.1. Patient Factors

- Renal impairment (increases levels)
- Age (elderly more sensitive) and body weight

4.2. Electrolytes

- Hypokalemia → increases toxicity risk
- Hyperkalemia (in toxicity) → poor prognosis

4.3. Drug Interactions

- Amiodarone, Verapamil and Quinidine Increases digoxin levels
- Rifampicin decreases digoxin level

4.4. Herbal supplements like Tejecote root and yellow oleander

- Their cardiotoxic properties mimicks digoxin-like Toxicity with prolonged consumption.

5. Discussion

This case highlights atypical and multi-system presentation beyond classical arrhythmia. The presentation included melaena, persistent nausea/vomiting, anorexia and fatigue. This emphasizes that digoxin toxicity may be present as a multisystem syndrome rather than isolated bradyarrhythmia, which can delay diagnosis.

The combination of digoxin and amiodarone is a well-known interaction, but in real-world CCU settings it is frequently continued without early dose adjustment. In this case, the concomitant use of amiodarone is highly significant, as it reduces the clearance of digoxin and thereby increases its serum concentration, predisposing the patient to toxicity. There is a critical gap in real-world practice- the continuation of multiple AV nodal blocking agents (digoxin + beta-blocker + amiodarone) without dose reassessment after acute coronary events. This case highlights how even low-dose amiodarone (100 mg BD) can precipitate toxicity in an elderly post-MI patient.

The patient was on digoxin 0.25 mg BD, which may be perceived as standard in some settings. However, in elderly, low body weight, post-infarction patient, this can become a supratherapeutic exposure, underscoring the importance of individualized dosing.

Furthermore, the patient's advanced age and possible renal impairment likely contributed to reduced drug clearance and increased susceptibility to toxic effects. thereby emphasize diagnostic importance of clinical suspicion over serum levels.

The important differential diagnoses to be considered include AV nodal ischemia secondary to inferior wall myocardial infarction, beta-blocker–induced bradycardia, sick sinus syndrome, electrolyte imbalance, and drug-induced conduction delay due to digoxin and amiodarone. Given her recent inferior wall MI and multiple rate-limiting medications, these differentials must be carefully evaluated alongside the clinical suspicion of digoxin toxicity.

Management should begin with immediate cessation of digoxin and a thorough review of all AV nodal blocking medications, including beta-blockers and amiodarone, with rationalization of therapy. The patient should be admitted to the CCU for continuous ECG monitoring. Symptomatic bradycardia should be treated with atropine, and if the patient remains hemodynamically unstable or does not respond adequately, temporary pacing should be considered. Simultaneously, all reversible factors must be corrected, including optimization of serum potassium and magnesium levels and careful assessment and improvement of renal function. In cases of severe toxicity, particularly when there are life-threatening arrhythmias, severe bradycardia or AV block, hemodynamic instability, or significant toxicity associated with hyperkalemia, treatment with digoxin-specific antibody fragments (Digoxin immune Fab) is indicated as the definitive antidotal therapy.

6. Case highlights

- High-risk but often under-recognized digoxin–amiodarone interaction
- Toxicity occurring at routine or seemingly acceptable dosing

- A multisystem presentation (GI symptoms + bradycardia + fatigue + melaena)
- Importance of distinguishing drug-induced bradycardia from post-MI conduction disease
- Need for strict medication reconciliation in post-PCI elderly patients
- Emphasis that digoxin toxicity is primarily a clinical diagnosis, not purely laboratory-based

7. Conclusion

This case emphasizes the critical importance of early recognition of digoxin toxicity in an elderly post-PCI patient, particularly in the presence of high-risk drug interactions such as amiodarone. This case reinforces the need to actively consider iatrogenic causes in the differential diagnosis of bradycardia post-MI. It underscores the need for careful dosing, vigilant therapeutic monitoring, and prompt clinical intervention to prevent life-threatening complications.

References

[1] Katzung BG, Vanderah TW. Basic and Clinical Pharmacology. 15th ed. New York: McGraw Hill; 2021.

[2] Brunton LL, Hilal-Dandan R, Knollmann BC. Goodman & Gilman's The Pharmacological Basis of Therapeutics. 14th ed. New York: McGraw Hill; 2022.

[3] Tisdale JE, Miller DA. Drug-Induced Diseases: Prevention, Detection, and Management. 3rd ed. Bethesda: American Society of Health-System Pharmacists; 2018.

[4] Rathore SS, Curtis JP, Wang Y, Bristow MR, Krumholz HM. Association of serum digoxin concentration and outcomes in patients with heart failure. JAMA. 2003;289(7):871–878.

[5] Aronson JK. Meyler's Side Effects of Drugs. 16th ed. Oxford: Elsevier; 2016