TURKISH JOURNAL OF EMERGENCY MEDICINE

Case Report/Case Series

Access this article online

Quick Response Code:

Website: https://turkjemergmed.com/

DOI: 10.4103/tjem.tjem_275_24

Floral phantosmia and bradycardia: A unique case of digoxin toxicity in an elderly patient

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Abstract:

This case report presents a rare clinical manifestation of digoxin toxicity in a 73-year-old female with acute kidney injury, bradycardia, and unique sensory disturbances, including phantosmia (floral scent hallucinations) and photopsia (seeing sparkles of light). The patient, with a history of hypertension and atrial fibrillation, had been on digoxin for 2 weeks, raising concerns about possible digoxin intoxication. Upon admission, bradycardia, hypokalemia, and elevated serum digoxin levels confirmed toxicity. The patient's olfactory and visual hallucinations, rare symptoms in such cases, gradually resolved after stopping digoxin. This case emphasizes the importance of recognizing subtle and unusual symptoms, like changes in smell and vision, which can enhance early detection, especially in older patients, leading to quicker interventions and better patient outcomes.

Keywords:

Digoxin intoxication, drug interactions, older patients, phantosmia, renal failure

Introduction

Digoxin, a cardiac glycoside with a narrow therapeutic range, continues to be widely used for managing atrial fibrillation and heart failure. While it offers considerable therapeutic benefits, the potential for toxicity is notably high, especially in older adults who may have impaired renal function or are taking multiple medications. This case presents a rare and unusual manifestation of digoxin toxicity, emphasizing the critical need for dynamic monitoring and prompt intervention.

Case Report

A 73-year-old female presented to the nephrology department with acute kidney injury (AKI), accompanied by olfactory and visual disturbances (reporting floral scents and seeing sparkles of light), abdominal discomfort, nausea, vomiting, and bradycardia. She had a long-standing history of hypertension and atrial fibrillation and was on multiple medications, including olmesartan, amlodipine, hydrochlorothiazide, digoxin, carvedilol, omeprazole, and warfarin. Digoxin had been introduced to her treatment regimen 2 weeks prior to admission. Despite reporting adherence to a once-daily digoxin dosage of 0.25 mg for 14 days, the recent onset of mild drowsiness and psychomotor slowing raised concerns about potential unintentional overconsumption. Furthermore, she had been taking furosemide for peripheral edema in the 5 days leading up to her admission.

On examination, the patient appeared pale and in orthopnea. Vital signs revealed a blood

Submitted: 20-12-2024
Revised: 16-04-2025
Accepted: 16-04-2025
Published: 01-10-2025

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How to cite this article: Kasa M, Elezi B, Sinamati E, Spahia N, Rroji M. Floral phantosmia and bradycardia: A unique case of digoxin toxicity in an elderly patient. Turk J Emerg Med 2025;25:317-20.

pressure of 100/60 mm Hg, a heart rate of 37 beats/min, and oxygen saturation of 86% on room air. Pulmonary auscultation detected bilateral rales and peripheral edema was present. Urine output was reduced to 500 mL/day. The patient was mildly drowsy, demonstrating psychomotor slowing and delayed responses, but remained oriented (abbreviated mental test 8/10, 4AT Delirium screening tool 2/12), indicating mild cognitive slowing without significant delirium. Despite being fully aware that no flowers were present, she persistently perceived a floral scent for 5 days, a new and concerning symptom. A thorough neurological examination and a brain computed tomography scan, both arranged by the consulting neurologist, revealed no abnormalities to explain her sensory disturbance. Laboratory results revealed elevated creatinine (1.8 mg/dL), urea (55 mg/dL), hypokalemia (K ▯ 2.7 mEq/L), base excess 11 mmol/L, bicarbonate 35 mmol/L, hemoglobin 11.7 g/dL, WBC 11,000/mm^3, and CRP 1.5 mg/dL (<0.5 mg/dL). Bradycardia and clinical signs suggested digoxin toxicity, later confirmed by a level of 3.5 ng/mL (therapeutic range 0.5–2.0) 2 days after stopping digoxin. NT-proBNP was markedly elevated (11,500 pg/mL), and urine culture grew *Escherichia coli* sensitive to imipenem. Management included discontinuing digoxin, IV potassium for hypokalemia, atropine for bradycardia, and imipenem for infection. Bradycardia, sensory disturbances, nausea, and drowsiness resolved as digoxin levels normalized. The patient was discharged with normal renal function, stable vitals, and a reduced regimen of carvedilol, amlodipine, and warfarin, with digoxin permanently discontinued.

Discussion

Digoxin exerts its therapeutic effects by reducing heart rate and rhythm through increased vagal tone, which slows conduction at the atrioventricular (AV) node. In addition, it enhances myocardial contractility by inhibiting the Na+/K+-ATPase pump, resulting in elevated intracellular calcium levels, thereby improving the force of cardiac contractions.[1]

Patients presenting to emergency services with digoxin intoxication exhibit a broad spectrum of symptoms, that includes gastrointestinal (loss of appetite, nausea, vomiting, and diarrhea), neurological (headache, confusion, lethargy, trigeminal neuralgia, and other neuralgias, delirium, or hallucinations), visual (blurring, halos, scotomas, and xanthopsia), olfactory (disosmia and hyposmia) and cardiac (arrhythmia and syncope due to severe bradycardia) disturbances.[2,3] None of those are pathognomonic for digoxin toxicity.[4,5]

Around 80% of patients on digoxin experience color vision deficiencies, primarily yellow and green chromatopsias. Some patients may have abnormal electroretinograms, which typically normalize after discontinuing digoxin, indicating retinal toxicity.[6] Rare cases manifest olfactory disturbance as disosmia in association with digoxin intoxication.[2]

In our patient, the primary symptoms included olfactory hallucinations or phantosmia (such as the perception of floral scents) and visual anomalies such as photopsia (seeing light sparkles), along with abdominal discomfort, nausea, vomiting, and fatigue followed by the onset of bradycardia. She had olfactory hallucinations or phantosmia, specifically reporting the perception of floral scents despite no external stimuli.

Phantosmia refers to the false perception of smells in the absence of an odorant source.[7] This is the first documented case in the literature linking phantosmia with digoxin intoxication, marking it as a novel clinical manifestation of the condition. The presence of such olfactory hallucinations expands the known symptomatology of digoxin toxicity, particularly in older patients, and suggests that sensory disturbances, including abnormal smells, should be considered in its diagnosis.

The electrocardiogram upon arrival to the emergency department (ED) exhibited hallmark features of digoxin toxicity, including a junctional rhythm (37 bpm), reflecting sinoatrial node suppression due to enhanced vagal tone. Conduction abnormalities, including left anterior hemiblock and right bundle branch block, indicated disruption within the His-Purkinje system, likely worsened by Na+/K+-ATPase inhibition and hypokalemia. Diffuse down-sloping ST depressions with a characteristic "scooped" appearance and biphasic T waves reflected the digoxin effect on myocardial repolarization [Figure 1a].

Digoxin toxicity in this 73-year-old female patient results from multiple interrelated factors, including compromised renal function, polypharmacy, electrolyte disturbances, or just unintentional overconsumption due to mild drowsiness and psychomotor slowing. Patients over 70, like the one in this case, are at heightened risk for digoxin toxicity, even when serum

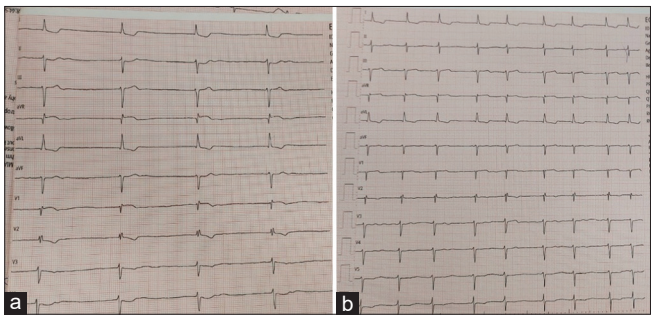

Figure 1: Electrocardiogram of the patient on the 1st (a) and the 7th day (b)

concentrations fall within the therapeutic range.[8] This increased susceptibility in older adults has been well-documented, with studies indicating a higher incidence of supratherapeutic digoxin levels despite lower daily doses (0.27 vs. 0.35 mg/day).[9] Factors contributing to this vulnerability include age-related declines in renal function, reduced lean body mass, and polypharmacy required to manage comorbidities.[10] Renal impairment, in particular, prolongs the half-life of digoxin from the typical 36–48 h to as long as 6 days, necessitating careful dose adjustments and extended dosing intervals.[11] In this case, AKI, likely precipitated by dehydration due to vomiting, was a significant contributing factor, further complicated by the concurrent use of ARBs and loop diuretics in the setting of a urinary tract infection (UTI).

Electrolyte imbalances further compound this risk, with hyperkalemia due to Na+/K+-ATPase inhibition, often being a trademark of acute digoxin toxicity. On the other hand, chronic digoxin administration alongside potassium-wasting diuretics frequently results in hypokalemia, which augments toxicity via increased binding of digoxin to Na+/K+-ATPase and reduced renal clearance. The combined action of hydrochlorothiazide and furosemide likely developed hypokalemia in this patient. This calls for immediate and continued potassium replacement therapy throughout the whole hospital stay to respond to the persistent electrolyte imbalance.[12,13] Hypomagnesemia, which often occurs under long-term diuretic therapy, also increases toxicity by depressing Na+/K+-ATPase activity.[14]

Coadministration of omeprazole further increased the risk of toxicity by increasing absorption and impairing the elimination of digoxin due to P-glycoprotein inhibition or shared CYP3A4 metabolism.[15] This constellation of factors underscores the complicated interplay between pharmacokinetics and underlying patient conditions that predispose older adults to digoxin toxicity.

Measurement of serum digoxin levels remains an important component of toxicity assessment. Whereas levels measured 6–12 h postdose most accurately reflect circulating levels, levels can be measured immediately in suspected overdose. However, relying solely on serum concentration can be very misleading, as toxicity may present even at therapeutic levels in the presence of disturbances in electrolytes such as hypokalemia, hypomagnesemia, or hypercalcemia.[3] Given this fact, clinical symptoms and other diagnostic findings should be the basis for correctly interpreting digoxin levels to identify and manage toxicity.[16]

Treatment for digoxin intoxication centers on stabilization, correcting metabolic disturbances, and lowering digoxin levels. The first step is discontinuing digoxin. Digoxin-specific immune antibody fragments (Digoxin-Fab) neutralize digoxin and resolve dysrhythmias within 30–45 min but are typically reserved for life-threatening cases, such as severe arrhythmias or hyperkalemia exceeding 5.5 mEq/L.[17] After consulting a toxicologist, Digoxin-Fab was not used due to a moderately elevated serum digoxin level (3.5 ng/mL) and resolution of bradycardia with atropine. The absence of high-degree AV block, ventricular arrhythmias, or hemodynamic collapse further supported this decision. The presence of hypokalemia (2.7 mEq/L) prompted prioritization of potassium correction to mitigate digoxin's toxic effects. While calcium use is controversial, it may help address severe hyperkalemia, and hemodialysis is ineffective in digoxin intoxication. The patient showed rapid improvement with electrolyte management and supportive care, including the resolution of conduction blocks, sinus rhythm restoration, and normalization of ST-T changes [Figure 1b]. Mild UTI or AKI in an elderly patient could theoretically cause delirium and unusual perceptions, but both were mild here, and the patient remained fully aware that the scent was unreal. Floral phantosmia resolved only after digoxin levels normalized, strongly suggesting a novel link between olfactory hallucinations and digoxin toxicity, which has not been previously documented. Moreover, visual disturbances (such as seeing sparkles of light) and other sensory alterations, although less frequently described, are well-recognized manifestations of digoxin toxicity. However, further research is needed to confirm this observation, as this is, to our knowledge, the first reported case associating floral phantosmia specifically with digoxin toxicity. However, further research is necessary to validate this observation, as this is, to our knowledge, the first reported case associating floral phantosmia specifically with digoxin toxicity.

Conclusion

This case represents the first documented case with our knowledge of digoxin intoxication presenting with phantosmia, expanding the clinical spectrum of symptoms. Digoxin toxicity is a significant concern for older patients, especially those with risk factors such as renal impairment, polypharmacy, and electrolyte imbalances. This case underscores the need for vigilant monitoring of kidney function, drug interactions, and electrolytes to prevent toxicity. Older adults are at higher risk due to reduced renal clearance and polypharmacy. Addressing these factors is essential to enhance safety and prevent life-threatening complications.

Author contributions statement

Following the case MK (lead) and ES (supporting); conceptualization MK and MRr (lead); review the literature MK, MRr, ES, BE, and NS (equal); writing the manuscript MK, MRr, NS (supporting) and

BE (equal); review the manuscript and editing the final version MR, BE (equal). Marsida Kasa and Merita Rroji contributed equally as the first author in the manuscript.

Conflicts of interest
None declared.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

Funding
None.

References

1. Vamos M, Erath JW, Hohnloser SH. Digoxin-associated mortality: A systematic review and meta-analysis of the literature. Eur Heart J 2015;36:1831-8.
2. Ishimaru T, Yokogawa H. Olfactory and gustatory disturbances caused by digitalism: A case report. Auris Nasus Laryn×2006;33:465-9.
3. Andrews P, Anseeuw K, Kotecha D, Lapostolle F, Thanacoody R. Diagnosis and practical management of digoxin toxicity: A narrative review and consensus. Eur J Emerg Med 2023;30:395-401.
4. Arbabian H, Lee HM, Graudins A. Elderly patients with suspected chronic digoxin toxicity: A comparison of clinical characteristics of patients receiving and not receiving digoxin-fab. Emerg Med Australas 2018;30:242-8.
5. Chung EK. Digitalis intoxication. Postgrad Med J 1972;48:163-79.
6. Shi L, Sun LD, Odel JG. Colored floaters as a manifestation of digoxin toxicity. Am J Ophthalmol Case Rep 2018;10:233-5.
7. Gillette B, Reid JA, Shermetaro C. Phantosmia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
8. Hack JB, Wingate S, Zolty R, Rich MW, Hauptman PJ. Expert consensus on the diagnosis and management of digoxin toxicity. Am J Med 2025;138:25-33.e14.
9. Charfi R, Ben Sassi M, Gaies E, Jebabli N, Daghfous R, Trabelsi S. Digoxin therapeutic drug monitoring: Age influence and adverse events. Tunis Med 2020;98:35-40.
10. Kirilmaz B, Duygu H, Yilmaz R. Digoxin intoxication: An old enemy in modern era. J Geriatr Cardiol 2012;9:237-42.
11. Kiley CA, Cragin DJ, Roth BJ. Omeprazole-associated digoxin toxicity. South Med J 2007;100:400-2.
12. Winter ME. Basic Clinical Pharmacokinetics. 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2004.
13. Cummings ED, Swoboda HD. Digoxin toxicity. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
14. Smith TW, Antman EM, Friedman PL, Blatt CM, Marsh JD. Magnesium status and digoxin toxicity. Br J Clin Pharmacol 1991;32:717-21.
15. Smith TW, Antman EM, Friedman PL, Blatt CM, Marsh JD. Digitalis glycosides: Mechanisms and manifestations of toxicity. Part I. Prog Cardiovasc Dis 1984;26:413-58.
16. Digiovanni-Kinsley S, Duke B, Giovane R, Paisley C. A case of digoxin toxicity due to acute renal failure. Cureus 2021;13:e17599.
17. Lavonas EJ, Akpunonu PD, Arens AM, Babu KM, Cao D, Hoffman RS, *et al*. 2023 American Heart Association focused update on the management of patients with cardiac arrest or life-threatening toxicity due to poisoning: An update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2023;148:e149-84.