

Case Report

Predominant Neurological Manifestations of Severe Digoxin Toxicity in an Older Patient

Vania Chica Londoño^{1*}, MD; Sandra Isabel Suárez Velandia¹, MD

ABSTRACT

An 81-year-old woman with a history of atrial fibrillation treated with digoxin was admitted for influenza A infection. During hospitalization, she developed progressive somnolence without focal neurological deficits. Initial evaluation, including brain imaging, showed no acute abnormalities. Laboratory studies revealed a markedly elevated serum digoxin concentration of 7.5 ng/mL, consistent with severe digoxin toxicity. Renal function was also closely monitored because of its potential contribution to impaired drug clearance. Electrocardiography showed atrial fibrillation without specific signs of digoxin toxicity. Digoxin therapy was immediately discontinued, and treatment with digoxin immune Fab was initiated. Following administration of the antidote, the patient exhibited rapid and complete neurological recovery over the subsequent 24 to 48 hours, with normalization of mental status. This case emphasizes the importance of maintaining a high index of suspicion for digoxin toxicity in older patients, particularly in the setting of acute infection or unexplained neurological deterioration during hospitalization. Early recognition and prompt antidotal therapy are essential. (*Iranian Heart Journal* 2026; 27(4): 87-90)

KEYWORDS: digoxin toxicity; digoxin immune Fab; neurological manifestations; atrial fibrillation

¹ Clinical Pharmacology Service, Marqués de Valdecilla University Hospital, Santander, Spain.

* **Corresponding Author:** Vania Chica Londoño, MD; Clinical Pharmacology Service, Marqués de Valdecilla University Hospital, Santander, Spain.

Email: vaniacristina.chica@scsalud.es

Tel: +34 942 20 25

Received: June 10, 2025

Accepted: July 30, 2026

Digoxin toxicity is a potentially life-threatening condition characterized by a narrow therapeutic index. In older patients, clinical manifestations are frequently nonspecific, and neurological symptoms may predominate, leading to diagnostic delay and misattribution to more common causes such as infectious or metabolic encephalopathy.¹⁻⁴

Case Presentation

An 81-year-old woman with a medical history of hypertension, type 2 diabetes mellitus, atrial fibrillation treated with digoxin, hypothyroidism, and a previous

ischemic stroke presented to the emergency department with a 4-day history of progressive dyspnea, dry cough, and pleuritic chest pain. She denied ischemic chest pain and palpitations.

On admission, the patient was hemodynamically stable, with a blood pressure of 130/78 mm Hg, heart rate of 85 beats/min with an irregular rhythm, respiratory rate of 22 breaths/min, and temperature of 37.7°C. Pulmonary auscultation revealed bibasal crackles. She was alert, oriented, and had no focal neurological deficits. An electrocardiogram (ECG) on admission demonstrated atrial

fibrillation with a controlled ventricular response and no conduction abnormalities or electrocardiographic findings suggestive of digoxin toxicity. Initial laboratory investigations demonstrated elevated inflammatory markers, and a nasopharyngeal swab tested positive for influenza A virus. Chest radiography showed findings consistent with fluid overload.

The patient's chronic medication regimen is detailed in Table 1. Notably, she was receiving digoxin (0.25 mg daily) for atrial fibrillation, together with furosemide, olmesartan, dabigatran, and several other medications, reflecting significant polypharmacy.

The patient was initially managed with oseltamivir, diuretics, nebulizations, and supportive care. During hospitalization, she developed progressive clinical deterioration with worsening respiratory status and acute neurological impairment characterized by somnolence, reduced verbal response, and inability to follow commands, without focal neurological signs. Brain computed tomography showed no acute abnormalities

Table 1. Chronic Medication at Hospital Admission

Medication	Dose and Regimen
Mirabegron	50 mg orally once daily
Bromazepam	1.5 mg orally at bedtime
Citalopram	20 mg orally once daily (morning)
Digoxin	0.25 mg orally once daily
Dapagliflozin	10 mg orally once daily
Vildagliptin/Metformin	50 mg/1000 mg, 2 tablets orally once daily
Levothyroxine	100 µg orally once daily before breakfast
Furosemide	40 mg orally once daily
Olmesartan medoxomil	20 mg orally once daily
Dabigatran	150 mg orally twice daily
Paracetamol (acetaminophen)	1000 mg orally every 12 hours
Omeprazole	20 mg orally once daily
Nitroglycerin transdermal patch	5 mg applied once daily
Cyanocobalamin (vitamin B12)	1 mg/2 mL, subcutaneous injection every 30 days

Treatment and Outcome

Given the unexplained neurological deterioration, further evaluation was performed. Laboratory tests showed a mild decline in renal function (estimated glomerular filtration rate of 51 mL/min/1.73 m²), interpreted as functional impairment in the context of acute illness rather than established chronic kidney disease progression (Table 2). In the absence of structural neurological abnormalities and considering possible drug-related toxicity, serum digoxin levels were measured and found to be markedly elevated at 7.5 ng/mL, consistent with severe digoxin toxicity in the setting of acute illness, reduced clearance, and polypharmacy (Table 2). Digoxin was immediately discontinued, and treatment with digoxin immune Fab was initiated. Based on the patient's body weight and serum digoxin concentration, the estimated neutralizing dose was 4 vials. However, only 2 vials were available at the treating institution and were therefore administered. Despite receiving a partial antidote dose, the patient showed rapid clinical improvement, with recovery of consciousness within 24 hours and progressive resolution of neurological symptoms. Respiratory status improved, and complete neurological recovery was achieved within 1 week.

Table 2. Laboratory Findings at Hospital Admission and at the Time of Digoxin Toxicity

Parameter	Admission	Toxicity Episode
Serum digoxin, ng/mL	–	7.5
Creatinine, mg/dL	0.53	1.05
Estimated glomerular filtration rate, mL/min/1.73 m ²	>90	51
Sodium, mEq/L	139	138
Potassium, mEq/L	3.7	4.5
NT-proBNP, pg/mL	1804	1888
C-reactive protein, mg/dL	2.0	–
Glucose, mg/dL	198	–

She subsequently completed treatment for influenza A and was transferred to a lower-complexity facility for rehabilitation. The

overall clinical course, diagnostic evaluation, treatment, and outcome are summarized in Figure 1.

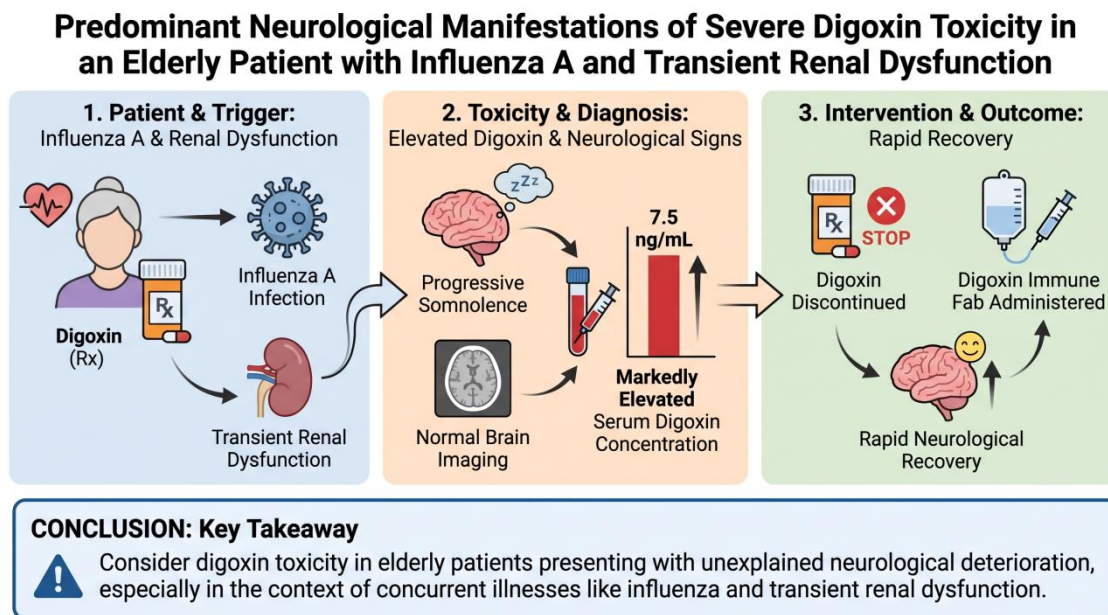


Figure 1. Clinical summary of a patient presenting with severe digoxin toxicity

DISCUSSION

This case illustrates the diagnostic complexity of digoxin toxicity in older patients, particularly when neurological manifestations predominate and mimic infectious or metabolic encephalopathy. The presence of influenza A infection and acute systemic deterioration initially directed clinical reasoning toward a primary infectious and cardiopulmonary process, delaying consideration of drug toxicity. Neurological manifestations of digoxin toxicity, including confusion, somnolence, and decreased level of consciousness, are well described in older adults, although they are less emphasized than cardiac arrhythmias.^{1–3} In this patient, the absence of focal neurological deficits and normal brain imaging further contributed to diagnostic uncertainty.

Older patients are particularly susceptible to digoxin toxicity because of age-related pharmacokinetic changes, decreased renal

clearance, and frequent polypharmacy. In the present case, the patient was receiving multiple chronic medications, reflecting a substantial medication burden. Furthermore, she was prescribed digoxin at a daily dose of 0.25 mg, which may increase the risk of toxicity in older adults, particularly during episodes of acute illness. Even modest reductions in renal function, such as the mild decrease observed in this case (estimated glomerular filtration rate, 51 mL/min/1.73 m²), may significantly impair drug elimination and promote drug accumulation.^{2–4}

Infections such as influenza A may act as precipitating factors by inducing systemic inflammation, dehydration, and transient renal dysfunction, thereby increasing susceptibility to digoxin accumulation. The DIGITOX study has also highlighted that digoxin intoxication remains a relevant cause of hospital admissions among older polymedicated patients.⁵ Early recognition is essential, as serum digoxin measurement and

prompt administration of digoxin immune Fab remain the cornerstone of treatment in severe cases, leading to rapid reversal of toxicity and favorable outcomes.^{2,3,6}

In the present case, the estimated neutralizing dose of digoxin immune Fab was four vials. Nevertheless, only 2 vials were available and administered. Despite partial neutralization, the patient experienced prompt neurological recovery and favorable clinical evolution. This observation is consistent with previous reports indicating that administration of a partial dose may still provide substantial clinical benefit in selected patients when complete neutralization is not feasible.⁷

CONCLUSIONS

This case highlights the importance of considering digoxin toxicity in older patients presenting with unexplained neurological deterioration, particularly in the setting of acute systemic illness and mild renal function decline. Early identification and treatment with digoxin immune Fab are critical for rapid clinical recovery and favorable prognosis.

Conflict of Interest

The authors declare no conflicts of interest and no financial or commercial relationships that could be considered a potential conflict of interest related to this manuscript.

Ethical Considerations

Informed consent was obtained from the patient for publication of this case report and accompanying clinical data. According to institutional policy, ethics committee approval was not required for the publication of a single case report.

Acknowledgements

The authors thank the health care professionals involved in the patient's care

REFERENCES

1. Ehle M, Patel C, Giugliano RP. Digoxin: clinical highlights and toxicity management. *Med Clin North Am.* 2016; 100(3):587–603. doi:10.1016/j.mcna.2015.12.007
2. Hack JB, Woody JH, Lewis DE. Diagnosis and management of digoxin toxicity: a narrative review and consensus. *Am J Med.* 2023;136(9):1035–1044. doi:10.1016/j.amjmed.2023.04.012
3. Chan BS, Isbister GK, Kirkpatrick CM, Duffull SB. Drug-specific treatment of digoxin toxicity. *Clin Toxicol (Phila).* 2016; 54(9):749–756. doi:10.1080/15563650.2016.1214274
4. Smith TW. Digitalis toxicity: epidemiology and clinical features. *J Am Coll Cardiol.* 1985; 5(5 Suppl A):43A–50A. doi:10.1016/S0735-1097(85)80476-9
5. Supervía A, et al. Intoxication by digoxin in Spanish hospital emergency departments: DIGITOX study. *Emergencias.* 2023; 35(5):328–334.
6. Antman EM, Wenger TL, Butler VP Jr, Haber E, Smith TW. Treatment of life-threatening digitalis intoxication with digoxin-specific Fab antibody fragments. *N Engl J Med.* 1976; 294(15):797–800. doi:10.1056/NEJM19760408294150
7. 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 Dec 1;30(6):395–401. doi: 10.1097/MEJ.0000000000001065