International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 23s, 2025
https://www.theaspd.com/ijes.php

Fatal Oleander Cardenolide Toxicity Presenting With Bradyarrhythmia And Hyperkalemia: A Case Report

Dr Velmurugan P[1], Dr T. V. Ramakrishnan[2*], Dr. Kaarunesh A[3]
[1]MD emergency medicine, Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research Institute, Porur, Chennai, India
[2]MD Anesthesiology , Head of department of emergency medicine, Sri Ramachandra Institute of Higher Education and Research Institute, Porur, Chennai, India
[3]MD Emergency medicine, Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research Institute, Porur, Chennai, India

Abstract
Oleander (Nerium oleander) contains cardenolide cardiac glycosides that produce a digoxin-like toxidrome. We report a fatal ingestion presenting to a tertiary emergency department. A 54-year-old woman arrived two hours after ingesting crushed oleander leaves mixed with food. She complained of vomiting and breathlessness and was drowsy but arousable. Initial examination showed tachypnoea (34/min) with bilateral crepitations, blood pressure 100/60 mmHg, bradycardia 48/min, and oxygen saturation 97% on room air. ABG revealed pH 7.35, HCO_3^- 13 mmol/L, and K^+ 5.39 mmol/L. ECG demonstrated sinus bradycardia with PR prolongation, intermittent non-conducted P waves suggestive of second-degree AV block, and frequent premature ventricular complexes. Management comprised atropine 1 mg IV (heart rate to 74/min), intravenous fluids, insulin 10 units IV, and 50 g activated charcoal via nasogastric tube. She underwent rapid-sequence intubation and was prepared for temporary pacing. Repeat ABG showed worsening acidemia and hyperkalemia (K^+ 5.72 mmol/L); additional insulin and 100 mEq sodium bicarbonate were administered. She developed cardiac arrest, achieved ROSC after four minutes, received temporary pacing, lignocaine infusion, dual vasopressors, and CRRT, but suffered recurrent arrests and died nine hours after ingestion. The case underscores the rapid progression of oleander cardiotoxicity and the value of early ECG and potassium-guided escalation.

Keywords: *Oleander poisoning, Cardiac glycoside toxicity, Atrioventricular block, Hyperkalemia, Digoxin-specific antibody fragments*

INTRODUCTION

Oleander – both common (Nerium oleander) and yellow (Cascabela/Thevetia peruviana) – is a ubiquitous ornamental in India; all plant parts contain cardenolide cardiac glycosides (e.g., oleandrin, thevetin) that inhibit Na^+/K^+-ATPase and produce a digoxin-like toxidrome with bradyarrhythmias, AV block, ventricular ectopy, and hyperkalemia.(1) In South Asia, deliberate self-poisoning with oleander is a persistent public-health problem; Indian reviews identify it as one of the commonest plant-based toxic exposures presenting to hospitals, particularly in southern states.(2) Hospital series from South India further describe characteristic ECG abnormalities and the need for intensive monitoring, underscoring a substantial clinical burden on emergency services.(3)Mortality among hospitalized oleander cases in the region typically ranges from ~2–10%, driven by malignant dysrhythmias and shock.(4)

For emergency physicians in low- and middle-income countries (LMICs), several challenges complicate care. First, confirmatory laboratory testing is limited: digoxin immunoassays often read 'positive' from cross-reactivity with plant glycosides but the values poorly reflect body burden and become uninterpretable after antidote, necessitating reliance on serial ECGs and electrolytes for risk stratification.(1) Second, the only therapy with consistent evidence for rapid reversal of life-threatening plant glycoside cardiotoxicity – digoxin-specific antibody fragments (dsFab) – is costly and frequently unavailable in LMIC settings; even where indicated, access delays are common.(5, 6) Third, severe bradyarrhythmias may require temporary transvenous pacing, but pacing capacity is uneven outside tertiary centers; inter-hospital transfers of unstable patients carry significant risk – limitations highlighted in regional experience from South Asia.(6) Finally, evidence for adjuncts such as multidose activated charcoal, atropine/isoprenaline, and class IB antiarrhythmics is heterogeneous, and protocols vary across institutions, adding operational uncertainty in busy emergency departments.(6)

Against this backdrop, we report a fatal oleander ingestion managed in the Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research (SRIHER), Chennai.

Case Report

A 54-year-old woman was brought to the emergency department with an alleged history of ingestion of crushed Nerium oleander leaves mixed with food at her residence. The ingestion had occurred approximately two hours prior to presentation. On arrival she reported vomiting and shortness of breath. She denied chest pain, palpitations, profuse sweating, giddiness, or loss of consciousness, and had no abdominal pain or loose stools. There was no history of previous suicide attempts, and she had no known comorbid illnesses.

On primary survey, the patient was drowsy but arousable. The airway was patent. She was tachypnoeic with a respiratory rate of 34/min and an oxygen saturation of 97% on room air; bilateral chest crepitations were present. All peripheral pulses were palpable, regular, and of good volume. Blood pressure measured 100/60 mmHg, and the heart rate was 48/min; capillary refill time was <2 seconds and normal heart sounds (S1, S2) were audible. Neurologically, the Glasgow Coma Scale score was E3V4M6 (13/15); pupils were bilaterally equal and reactive to light, ~2 mm. Capillary blood glucose was 96 mg/dL. Temperature was 98.1 °F, and there were no external injuries.Initial arterial blood gas analysis showed pH 7.35, pCO_2 23 mmHg, pO_2 112 mmHg, HCO_3^- 13 mmol/L, Na^+ 133 mmol/L, K^+ 5.39 mmol/L, haemoglobin 12 g/dL, SpO_2 96.5%, glucose 311 mg/dL, and lactate 7.80 mmol/L. Serum creatinine measured 1.5 mg/dL and troponin I was 0.232 ng/mL. Two-dimensional echocardiography demonstrated global left-ventricular dysfunction.The initial 12-lead ECG demonstrated a bradyarrhythmia consistent with sinus bradycardia (rate approximately 45–50/min) with AV-nodal conduction delay (prolonged PR) and intermittent non-conducted P waves suggestive of second-degree AV block. There were frequent premature ventricular complexes (including bigeminal beats) and nonspecific ST–T changes without acute ST-segment elevation. A subsequent rhythm strip again showed persistent bradycardia with recurrent ventricular ectopics and brief runs of ventricular escape/ectopy.

In the emergency room, intravenous access was secured, and 1 mg of atropine was administered intravenously. Following atropine, the heart rate improved to 74/min and the blood pressure to 110/70 mmHg. Intravenous normal saline was started at 75 mL/h. Human Actrapid insulin 10 units IV was given, and 50 g of activated charcoal was administered via a Ryle's tube. Because of worsening work of breathing, the team planned airway protection and temporary pacing (TPI). The patient was intubated for impending respiratory failure with a 7.5-mm endotracheal tube, fixed at 19 cm at the incisors after confirmation of position by five-point auscultation. Medications used for rapid sequence induction were fentanyl 100 µg IV, etomidate 20 mg IV, and vecuronium 8 mg IV.

A repeat ABG revealed pH 7.27, pCO_2 33 mmHg, pO_2 102 mmHg, HCO_3^- 15 mmol/L, Na^+ 134 mmol/L, K^+ 5.72 mmol/L, haemoglobin 12 g/dL, SpO_2 96.5%, glucose 396 mg/dL, and lactate 6.32 mmol/L. Additional treatments in the ER included another 10 units of Human Actrapid IV and 100 mEq of sodium bicarbonate IV. Approximately one hour after arrival, the patient suffered a cardiac arrest while being shifted to the catheterization laboratory; return of spontaneous circulation was achieved after four minutes of cardiopulmonary resuscitation. A temporary pacemaker was then placed.During subsequent management, the patient developed frequent ventricular ectopic beats and was started on an intravenous lignocaine infusion at 60 mg/h. She required dual vasopressor support and continuous renal replacement therapy was initiated. Despite these measures, she experienced recurrent cardiac arrests and was declared dead approximately nine hours after ingestion.

DISCUSSION

Oleander (Nerium oleander) contains a suite of cardenolide cardiac glycosides (notably oleandrin), which inhibit the Na^+/K^+-ATPase pump, increase intracellular sodium and secondarily intracellular calcium, and thereby produce vagotonia, AV-nodal conduction delay, and ventricular dysrhythmias. Human poisoning is well described across South Asia, where deliberate self-poisoning with oleander or its close relative Cascabelathevetia (yellow oleander) remains a significant clinical problem.(7)

In this patient, early gastrointestinal symptoms (vomiting) and dyspnea were followed by bradyarrhythmia with AV-nodal delay, frequent ventricular ectopy, and evolving metabolic derangement – findings entirely compatible with glycoside cardiotoxicity. Conduction system disease (sinus bradycardia, PR prolongation, and 2° AV block) is typical; premature ventricular complexes, bigeminy, and ventricular escapes/ectopy are common and may alternate with bradyarrhythmias as vagotonia and triggered activity co-exist. Echo-documented global LV dysfunction has been reported in severe cases and probably reflects a combination of toxin-mediated contractile impairment, dysrhythmia-related stunning, and hypoperfusion.(6, 8)

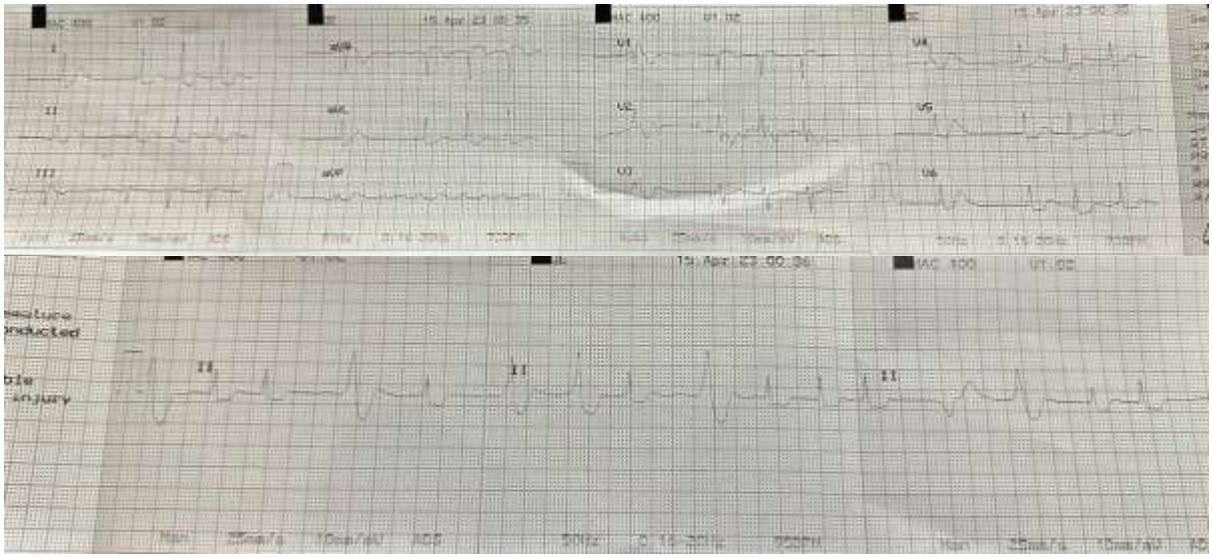

Figure 1: ECG in Oleander Cardenolide Toxicity – Sinus Bradycardia with PR Prolongation, Intermittent 2° AV Block, and Frequent PVCs

Serum potassium is both a pathophysiologic marker and a prognostic indicator. Oleander glycosides drive extracellular potassium elevation by pump inhibition; across digoxin-like poisonings, higher K^+ correlates with more severe toxicity and worse outcomes. Reviews and cohort studies in South Asia link hyperkalemia – often operationalized as $K^+ > 5.5$ mmol/L – to serious arrhythmias and increased mortality. In our case, potassium was already elevated on arrival (5.39 mmol/L) and rose to 5.72 mmol/L alongside bradyarrhythmias – consistent with a high-risk trajectory.(6, 9)Diagnosis in plant glycoside poisoning is primarily clinical and ECG-based. A practical adjunct is the hospital digoxin immunoassay – which frequently reads positive in oleander exposures because plant cardenolides cross-react variably across platforms. The assay can support the diagnosis when digoxin therapy is not being taken, but numeric values do not reflect true body burden and become uninterpretable after antibody fragments are administered. These analytical pitfalls are well documented in bench and clinical studies.(10, 11)

Decontamination with activated charcoal is reasonable when patients present early and can be protected from aspiration. Randomized data specific to yellow oleander are mixed: an earlier single-blind Sri Lankan trial suggested mortality and arrhythmia reduction with multidose activated charcoal (MDAC),(12) whereas a later, larger Lancet trial of routine single- or multiple-dose charcoal in acute self-poisoning (including a substantial oleander subset) showed no overall mortality benefit to routine MDAC.(13) Contemporary syntheses conclude that charcoal is safe, single-dose administration within a few hours is sensible, and the case for MDAC remains context-dependent.(9) In this case, administration of 50 g charcoal ~2 h post-ingestion aligned with these practices.

Supportive care remains the cornerstone. Atropine is recommended for symptomatic bradycardia; if bradyarrhythmias persist, isoprenaline infusions and temporary pacing are commonly used in severe cases, although neither has proven survival benefit and both have practical risks. Observational series from Sri Lanka and other endemic regions describe atropine/isoprenaline as initial therapy with pacing reserved for non-responders;(6, 14) however, pacing can be technically challenging in an irritable, glycoside-toxic myocardium and may itself provoke dysrhythmias.(1, 15) Our patient's heart rate rose with atropine, but she later progressed to cardiac arrest and ultimately required temporary transvenous pacing, consistent with severe conduction toxicity.

Antidotal therapy with digoxin-specific antibody fragments (dsFab; DigiFab®/Digibind®) is strongly supported for life-threatening arrhythmias, hemodynamic instability, or significant hyperkalemia in cardiac glycoside poisoning, including plant cardenolides. An RCT in yellow oleander demonstrated rapid reversal of bradyarrhythmias and hyperkalemia after dsFab,(5) and modern reviews recommend empiric, titrated dosing to clinical effect because assay-based calculations under-estimate plant glycoside burden and post-Fab measurements are unreliable.(16) International guidance (AHA 2023) considers dsFab reasonable in plant glycoside poisoning with severe features. In many endemic settings, however, cost and availability are limiting – an operational reality that likely influenced our patient's course.(17)

Management of ventricular dysrhythmias in glycoside toxicity favors class IB agents (lidocaine or phenytoin), which suppress ventricular automaticity and delayed afterdepolarizations without further depressing AV

conduction. Case series, reviews, and guideline summaries support their use as bridge therapy, ideally alongside dsFab where available.(18) Our patient developed frequent ventricular ectopy and appropriately received a lignocaine infusion (60 mg/h), reflecting these recommendations. Electrical cardioversion for glycoside-related tachyarrhythmias is often ineffective or destabilizing and is generally reserved for per-arrest situations.(19)

The role of intravenous calcium for hyperkalemia in cardiac glycoside toxicity has been controversial ('stone-heart' concern). A human cohort from a tertiary center (Levine et al.) found no increase in malignant arrhythmias or mortality with calcium use in digoxin-toxic patients;(20) nevertheless, many references still advise caution and prioritize dsFab when available. When life-threatening hyperkalemia is present and Fab is unavailable or delayed, contemporary literature supports that calcium need not be withheld on the basis of historical concerns alone. In this case, hyperkalemia was moderate and managed with insulin–dextrose and sodium bicarbonate; no intravenous calcium was documented.Renal replacement therapies do not clear cardiac glycosides effectively because of their large volume of distribution and tissue binding; however, dialysis (or CRRT) may be indicated for concomitant renal failure and to control severe hyperkalemia. Thus, CRRT in this patient would be expected to aid potassium control and acid–base management rather than toxin removal.(2)

Two additional features of this case merit emphasis. First, the lactate trajectory (7.8 → 6.32 mmol/L) and evolving acidosis likely reflected hypoperfusion from bradyarrhythmias and early cardiogenic compromise, mirrored by global LV dysfunction on echocardiography. Cardiogenic shock is a recognized mode of death in severe oleander poisoning, and global hypokinesis has been described in observational cohorts. Second, the mild troponin rise (0.232 ng/mL) should be interpreted as demand ischemia or toxin-mediated injury rather than acute coronary occlusion: nonspecific ST–T changes are common in glycoside poisonings and do not necessarily imply infarction.(8)Second, the patient presented two hours after ingestion, when gastrointestinal decontamination is still plausible. Nevertheless, the subsequent deterioration – with arrest approximately one hour after arrival and death nine hours post-ingestion – illustrates how rapidly plant glycoside toxicity can evolve despite early atropine, charcoal, airway protection, and pacing. In endemic settings, mortality of symptomatic yellow oleander poisoning has been reported between ~5–10% depending on access to antidote and critical care.(21)From a systems perspective, the case illustrates several challenges highlighted in regional literature: (i) transfers for pacing or higher-level care can be hazardous in unstable patients; (ii) antidote scarcity drives reliance on measures with unproven mortality benefit; and (iii) hyperkalemia is a readily available risk marker that can guide triage and early escalation. Studies from Sri Lanka and India have advocated for rapid ECG- and potassium-based risk stratification, continuous monitoring, and early consideration of dsFab where feasible.(1, 7)

CONCLUSION

This case highlights the fulminant course of oleander cardenolide toxicity, manifesting with early gastrointestinal symptoms, rapidly progressive bradyarrhythmia, hyperkalemia, and refractory shock despite prompt resuscitation, decontamination, airway protection, antiarrhythmic therapy, and temporary pacing. It underscores that serial ECGs and serum potassium remain the most practical bedside markers for risk stratification and escalation in emergency settings. The outcome also reflects real-world constraints in low-resource environments, where access to digoxin-specific antibody fragments – the only definitive antidote – may be limited or delayed. Strengthening antidote availability, standardizing ED protocols, and community prevention efforts are essential to improve survival in plant glycoside poisonings in India.

REFERENCES

1. Roberts DM, Gallapatthy G, Dunuwille A, Chan BS. Pharmacological treatment of cardiac glycoside poisoning. Br J Clin Pharmacol. 2016;81(3):488-95.
2. Pillay VV, Sasidharan A. Oleander and Datura Poisoning: An Update. Indian J Crit Care Med. 2019;23(Suppl 4):S250-s5.
3. Karthik G, Iyadurai R, Ralph R, Prakash V, Abhilash KPP, Sathyendra S, et al. Acute oleander poisoning: A study of clinical profile from a tertiary care center in South India. J Family Med Prim Care. 2020;9(1):136-40.
4. Sacco MA, Gualtieri S, Princi A, Tarallo AP, Verrina MC, Tarda L, et al. Human Deaths Related to Oleander Poisoning: A Review of the Literature. Toxins (Basel). 2025;17(3).
5. Eddleston M, Rajapakse S, Rajakanthan, Jayalath S, Sjöström L, Santharaj W, et al. Anti-digoxin Fab fragments in cardiotoxicity induced by ingestion of yellow oleander: a randomised controlled trial. The Lancet. 2000;355(9208):967-72.
6. Rajapakse S. Management of yellow oleander poisoning. Clin Toxicol (Phila). 2009;47(3):206-12.
7. Bandara V, Weinstein SA, White J, Eddleston M. A review of the natural history, toxinology, diagnosis and clinical management of Nerium oleander (common oleander) and Thevetia peruviana (yellow oleander) poisoning. Toxicon. 2010;56(3):273-81.
8. Zamani J, Aslani A. Cardiac findings in acute yellow oleander poisoning. J Cardiovasc Dis Res. 2010;1(1):27-8.
9. Thomas VV, Newbigging N, Johnson J, Kalimuthu M, Gunasekaran K, Iyadurai R. Review of Current Evidence in Management of Oleander Poisoning. Current Medical Issues. 2022;20(2).

10. Dasgupta A, Welsh KJ, Hwang SA, Johnson M, Actor JK. Bidirectional (negative/positive) interference of oleandrin and oleander extract on a relatively new Loci digoxin assay using Vista 1500 analyzer. J Clin Lab Anal. 2014;28(1):16-20.
11. Dasgupta A, Datta P. Rapid detection of oleander poisoning using digoxin immunoassays: comparison of five assays. Ther Drug Monit. 2004;26(6):658-63.
12. de Silva HA, Fonseka MM, Pathmeswaran A, Alahakone DG, Ratnatilake GA, Gunatilake SB, et al. Multiple-dose activated charcoal for treatment of yellow oleander poisoning: a single-blind, randomised, placebo-controlled trial. Lancet. 2003;361(9373):1935-8.
13. Eddleston M, Juszczak E, Buckley NA, Senarathna L, Mohamed F, Dissanayake W, et al. Multiple-dose activated charcoal in acute self-poisoning: a randomised controlled trial. Lancet. 2008;371(9612):579-87.
14. Fonseka MM, Seneviratne SL, de Silva CE, Gunatilake SB, de Silva HJ. Yellow oleander poisoning in Sri Lanka: outcome in a secondary care hospital. Hum Exp Toxicol. 2002;21(6):293-5.
15. Eddleston M, Warrell DA. Management of acute yellow oleander poisoning. QJM: An International Journal of Medicine. 1999;92(9):483-5.
16. Chan BS, Buckley NA. Digoxin-specific antibody fragments in the treatment of digoxin toxicity. Clin Toxicol (Phila). 2014;52(8):824-36.
17. Beaulieu J, St-Onge M. Antidote use for cardiac arrest or hemodynamic instability due to cardiac glycoside poisoning: A narrative review. Resusc Plus. 2024;19:100690.
18. Alvarado-Alvarado JA, Salas-Villela RA, Reyes-Guerrero JA, Ezquerra-Osorio A. Phenytoin as treatment for bidirectional ventricular tachycardia in a patient with anterior myocardial infarction and digoxin toxicity. Arch Cardiol Mex. 2022;92(4):550-2.
19. Regina AC, Rehman R, Hai O. Cardiac Glycoside and Digoxin Toxicity. StatPearls. Treasure Island (FL): StatPearls Publishing Copyright © 2025, StatPearls Publishing LLC.; 2025.
20. Levine M, Nikkanen H, Pallin DJ. The effects of intravenous calcium in patients with digoxin toxicity. J Emerg Med. 2011;40(1):41-6.
21. Gawarammana I, Mohamed F, Bowe SJ, Rathnathilake A, Narangoda SK, Azher S, et al. Fructose-1, 6-diphosphate (FDP) as a novel antidote for yellow oleander-induced cardiac toxicity: A randomized controlled double blind study. BMC Emergency Medicine. 2010;10(1):15.