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Laurel but Hardy: unintended poisoning, a case report of oleander misidentification as bay laurel

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ABSTRACT

Introduction: Nerium oleander is a toxic plant containing cardiac glycosides throughout all its parts, thereby posing severe health risks upon ingestion. The clinical manifestations of oleander poisoning closely resemble those of digoxin toxicity, encompassing a *spectrum* of gastrointestinal symptoms, neuropsychiatric disorders, and cardiac disturbances. This scientific case report describes a case of accidental intoxication resulting from the consumption of an oleander leaves infusion misidentified as bay laurel leaves.

Case report: An 84-year-old patient consumed an oleander leaves infusion, and after four hours experienced gastrointestinal symptoms. He contacted the poison control center (PCC) and was advised to go to the emergency department (ED). Upon arrival, the patient presented stable vital signs without cardiac irregularities. The PCC recommended the administration of activated charcoal, vigilant monitoring, including electrocardiography (ECG). Subsequent ECGs assessments revealed the presence of third-degree atrioventricular block; in consultation with the PCC, digoxin-specific antibodies and external pacing were necessary. The patient was discharged on the eighth day in good hemodynamic condition, and outpatient follow-up visits showed clinical stability.

Discussion: This study offers insights for the management of similar cases. The limitations of conventional assays in measuring oleander cardiac glycosides were observed, emphasizing reliance on clinical evaluation. The patient's trajectory, remaining asymptomatic despite severe ECG changes post-ingestion, underscores the importance of prolonged clinical monitoring.

1. Introduction

Nerium oleander is an ornamental shrub or small tree belonging to the *Apocynaceae* family. It is originally distributed in subtropical Asia and the Mediterranean region but is now growing widespread [1,2]. While visually appealing, all parts of the plant are poisonous, primarily due to the presence of cardiac glycosides, namely oleandrin, oleandrigenin, adynerin, digitoxigenin, folinerin, and rosagenin [1,3].

Laurus nobilis, commonly known as bay laurel, is also a ubiquitous plant in Europe and some areas of Asia. It belongs to the *Lauracee* family, and it is a staple in many Mediterranean and international cuisines. It is also used as an ingredient in some traditional herbal remedies, such as infusion herbal tea. Although it is a totally different plant, its leaves share some similarities with those of the oleander. Both leaves are leathery, dark green, have a natural sheen on the surface, and are generally elongated and oblong. However, oleander leaves tend to be narrower and longer compared to bay leaves [4].

Oleander accidental poisoning can occur through ingestion of parts of the plant (just one or a few leaves may be lethal in children), inhalation of smoke from burning oleander, or use of oleander leaves-based medical preparations that have been used to treat leprosy, malaria, venereal diseases, and induce abortion [5]. Deliberate poisoning has been reported in suicide attempts and criminal cases. The main effect of cardiotoxic glycosides is positive inotropy. Glycosides bind to the sarcolemma transmembrane ATP-ase of cardiac muscle cells (Na^+/K^+ATP-ase) and compete with K^+ ions, inactivating the enzyme

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[5]. This results in an accumulation of Na^+ and Ca^{2+} ions into the cardiac muscle cells, leading to stronger and faster heart contractions. Moreover, the increased amount of extracellular K^+ ions may lead to lethal hyperkalemia. Therefore, clinical features of oleander poisoning are similar to digoxin toxicity and include nausea and vomiting due to stimulation of the area postrema of the medulla oblongata, neuropsychic disorders, and pathological motor manifestations. Cardiotoxic glycosides are also responsible for stimulating the vagus nerve (leading to sinus bradycardia) and the phrenic nerve (leading to hyperventilation), and lethal brady- and tachyarrhythmias, including asystole and ventricular fibrillation [1,6]. The severity of the intoxication can vary based on the quantity ingested and an individual's physiological response, as well as the time of symptom onset after oleander ingestion: they can rapidly occur after drinking teas prepared with oleander leaves or roots or develop more slowly due to the ingestion of unprepared plant parts [5].

The management of oleander intoxication involves monitoring blood pressure, oxygen saturation, and electrocardiography (ECG). Initially, it is important to insert an intravenous (IV) cannula for fluid administration and to treat possible pronounced hypotension (systolic pressure < 70 mmHg) or bradycardia (< 40 bpm) through bolus doses of atropine (2–3 mg). If necessary, small boluses (0.3–0.6 mg) or an infusion (0.6 mg/h) of atropine has to be administered to maintain the heart rate around 70–80 bpm [1,5]. In addition, the administration of activated charcoal is recommended. Serum electrolytes, especially potassium and magnesium, should be measured to promptly treat any eventual hypokalemia and hypomagnesaemia. As the measurement of digoxin levels would not accurately reflect the concentration of the oleander cardiac glycosides, carrying-out this examination may be not necessary [1,7]. Indications for digoxin-specific antibodies administration include atrioventricular (AV) node and/or severe sinus node block, ventricular tachycardias, and serum potassium levels exceeding 5.5 mEq/L [1,5]. However, to date, there is still no clear consensus on the most effective regimen for the administration of this medication; it may be considered to administer 400 mg over 20 min, followed by 400–800 mg over 4–8 h through infusion to sustain a therapeutic concentration over a longer period. If digoxin-specific antibodies are unavailable, treatment of hyperkalemia with insulin plus dextrose is recommended. Additionally, temporary pacing and low-energy direct current cardioversion for severe bradycardia due to AV block and ventricular fibrillation, respectively, should be considered [1,4].

We report a clinical case of oleander leaves misidentified as bay laurel, leading to intoxication with delayed onset of symptoms. Nevertheless, due to the consistent and close support of the local poison control center (PCC), signs of clinical deterioration were promptly recognized, and the appropriate antidote and supportive therapy were administered.

2. Case report

An 84-year-old man independently contacted the regional PCC of Verona four hours after drinking an infusion of oleander leaves

Table 1
Initial medical evaluation at the emergency department.

Patient Medical History		
Demographics	84 years; height 1.7 m; weight 73.5 kg, BMI 25.4 kg/m^2	
Social history and family diseases	Retired; he lives with his wife; economically stable; former smoker; both parents passed away years ago from natural causes; he has one daughter without significant pathologies	
Past medical history	Chronic ischemic heart disease; coronary artery bypass surgery in 2009; aortic endoprosthesis for endovascular treatment of subrenal abdominal aortic aneurysm in 2010, followed by abdominoplasty for treating an incisional hernia; thromboendoarterectomy for right internal carotid artery stenosis in 2006; hypertension in pharmacological treatment; dyslipidemia; history of major gastrointestinal bleedings and intraretinal hemorrhages; gastric resection for peptic ulcer treatment in 1976	
Allergies	No known allergies	
Regular medications	Allopurinol; alfuzosin; simvastatin; esomeprazole; candesartan; enoxaparin sodium 6000 I.U.	
History of the present illness	Recent (two months before) hospitalization for upper gastrointestinal bleeding, discharged with a hemoglobin level of 9.7 mg/dL. After drinking the oleander infusion, several episodes of vomiting, and one of diarrhea, occurred; no other symptoms were reported. An episode of vomiting during first medical evaluation occurred	
Vital parameters and objective physical examination at first evaluation		
Blood pressure	140/80 mmHg	
Heart rate	55 bpm	
Respiratory rate	16 per minute	
Oxygen saturation	99% in room air	
Body temperature	37 °C	
GCS	15	
Objective physical examination	Normal in all anatomical regions	
ECG	Sinus rhythm; mild bradycardia (54 bpm); some supraventricular extrasystoles	
Laboratory tests (blood) at ED admission	**Reference interval**	
Glucose	109 mg/dL	70–100 mg/dL
$HbA1_c$	5.9%	4.0–5.6%
Hemoglobin	9.9 g/dL	13.5–17.5 g/dL
WBC	5.83 * 10^3 /μL	4.5–11 * 10^3/μL
HCT	33.6%	38.8–50%
MCV	72.0 fL	80–96 fL
Platelets	219 * 10^3 /μL	150–450 * 10^3/μL
Troponin I	5.1 ng/L	34 ng/L
Sodium (Na^+)	136.3 mmol/L	135–145 mmol/L
Potassium (K^+)	4.72 mmol/L	3.5–5.0 mmol/L
Digoxin	0.50 ng/mL	-
BNP	132 pg/mL	< 100 pg/mL
CRP	7.9 mg/L	< 10 mg/L
Creatinine	1.16 mg/dL	0.84–1.21 mg/dL
eGFR CDK-EPI	58 mL/min/1.73 m^2	> 90 mL/min/1.73 m^2

Abbreviations: BMI = Body mass index; BNP = Brain natriuretic peptide; bpm = Beats per minute; CRP = C-reactive protein; ECG = Electrocardiography; ED = Emergency Department; eGFR = Estimated glomerular filtration rate; GCS = Glasgow Coma Scale; $HbA1_c$ = Glycated hemoglobin; HCT = Hematocrit; I.U. = International units; MCV = Mean corpuscular volume; WBC = White blood count

misidentified as bay laurel, picked up from his garden. During the phone conversation, the patient referred experiencing gastrointestinal symptoms, including several episodes of vomiting, and one episode of diarrhea. In response, the PCC recommended that the patient proceed to the Emergency Department (ED) for clinical monitoring and potential treatment.

Upon arrival at the ED, the patient was in stable general condition, conscious, alert, and oriented. Cardiac objective examination revealed normal and rhythmic heart sounds, oxygen saturation of 98% in room air, and a blood pressure of 140/80 mmHg with mild bradycardia (55 bpm). An ECG showed sinus rhythm with some supraventricular extrasystoles. Objective physical examination of all body regions did not reveal any abnormalities (Table 1).

The medical team consulted the PCC, which recommended the administration of activated charcoal (40 g every four hours), supportive therapies, and close ECG monitoring. Activated charcoal was promptly administered, and initially, the patient's clinical condition remained stable, hence not requiring antidote administration. As it was not available within the hospital pharmacy, the PCC arranged for the supply of the antidote to the hospital.

A subsequent ECG conducted in the ED a few hours later revealed first-degree AV block, moderate bradycardia (heart rate of 51 bpm), left axis deviation, and frequent supraventricular extrasystoles, consistent with the digoxin-like and hyperkalemic effects of the ingested substance.

Following subsequent laboratory tests that showed an increase in potassium levels (5.3 mmol/L), a 500 mL of 5% glucose solution containing 10 units of insulin lispro was administered.

After a total of three doses of activated charcoal and approximately 15 h post-ED admission (i.e., 20 h after oleander infusion ingestion), telemetry monitoring revealed third-degree AV block, characterized by bradycardia (heart rate of 37 bpm), although the patient remained asymptomatic without any symptoms or physical signs. In consultation with the PCC, the patient was administrated the antidote (intravenous digoxin-specific antibodies) at a dose of 160 mg (four vials of 40 mg each) over a period of 30 min. Subsequently, the patient was transferred to the intensive care unit (ICU) for closer clinical monitoring.

About six hours following antidote administration, due to worsening bradycardia (heart rate of 30 bpm), external transcutaneous pacing was initiated and continued for three hours, resulting in a return to first-degree AV block. No further complications occurred, and the patient's condition remained stable. The day after, he was moved to the geriatric ward, where continuous ECG and clinical monitoring were maintained. Sinus rhythm with a tendency towards bradycardia (heart rate of 50–59 bpm) was observed in the ECG monitoring, and no arrhythmias were detected in the remaining three days of hospitalization.

Finally, the patient was discharged on the eighth day of hospitalization, exhibiting stable hemodynamic condition and presenting no symptoms (Table 2). The patient received a recommendation for outpatient cardiological follow-up. Subsequent follow-ups at 10 days, one month, and two months showed the patient to be clinically stable without new ECG abnormalities or other notable clinical signs.

3. Discussion

To the best of our knowledge, very few case reports of oleander intoxication have been published in the literature. Cases of death due to oleander intoxication have been reported both following the consumption of whole leaves [6,8], as well as after the ingestion of oleander infusion for suicidal purposes [9]. In another documented clinical case, a middle-aged woman consumed a self-prepared oleander tea for sedative purposes, leading to nausea, vomiting, and third-degree AV block [10]. Such changes occurred as soon as two hours after ingestion, differently from our patient, who experienced ECG changes several hours after ingestion. Digoxin blood levels peaked on the first day and rapidly declined over the following two days. Conversely, in our case, the maximum digoxin value was significantly lower and was observed on the fourth day of hospitalization.

Table 2
Patient's parameters at discharge.

Vital parameters and objective physical examination at discharge		
Blood pressure	110/60 mmHg	
Heart rate	60 bpm	
Respiratory rate	16 per minute	
Oxygen saturation	96% in room air	
Body temperature	36.7 °C	
GCS	15	
Objective physical examination	Normal in all anatomical regions	
ECG	Sinus rhythm	
Laboratory tests (blood) at the eighth day (discharge)	**Reference interval**	
Glucose	102 mg/dL	70–100 mg/dL
Hemoglobin	10.6 mg/dL	13.5–17.5 g/dL
WBC	5.5 * 10^3 /μL	4.5–11 * 10^3/μL
HTC	35.2%	38.8–50%
MCV	73.1 fL	80–96 fL
Platelets	231 * 10^3 /μL	150–450 * 10^3/μL
Sodium (Na^+)	136.7 mmol/L	135–145 mmol/L
Potassium (K^+)	4.44 mmol/L	3.5–5.0 mmol/L
CRP	39.0 mg/L	< 10 mg/L
Creatinine	1.32 mg/dL	0.84–1.21 mg/dL
eGFR CDK-EPI	49 mL/min/1.73 m^2	> 90 mL/min/1.73 m^2

Abbreviations: bpm = Beats per minute; CRP = C-reactive protein; ECG = Electrocardiography; eGFR = Estimated glomerular filtration rate; GCS = Glasgow Coma Scale; HCT = Hematocrit; MCV = Mean corpuscular volume; WBC = White blood count

A series of critical lessons emerges from the clinical case reported, that can significantly inform the approach to managing similar cases in the future. Firstly, the finding that a substantial elevation of digoxin levels was not detected (Table 3) reinforces the inherent limitations of conventional immunofluorescence assays in accurately quantifying blood oleander cardiac glycosides [1,7]. Therefore, the patient's therapy should be guided by clinical physical examination and instrumental diagnostics. The patient's clinical trajectory, characterized by sustained asymptomatic status despite the manifestation of ECG changes indicative of severity over 20-hour period post-ingestion, provides a considerable example of the evolving nature of toxicological manifestations. Indeed, the toxic dose of cardiac glycosides and the timing of the onset of clinical manifestations are extremely variable, and they mainly depend on individual factors (e.g., age and health status) as well as toxicological factors (e.g., the quantity and the part of plant ingested, and the concentration of glycosides) [1,2].

This emphasizes the necessity for extended monitoring and heightened vigilance to effectively capture the eventual delayed onset of critical indicators.

Moreover, this study demonstrates the effectiveness of the national antidote management system, facilitated by a collaborative synergy between regional PCCs and hospital institutions, in managing poisonings and it underlines the relevance of toxicological services in such a clinical emergency. This collective effort is a clear example of how inter-organizational cooperation can markedly enhance emergency response mechanisms, and resource allocation. Lastly, it seems that different degrees of preparedness and recognition exist between poisonings

Table 3
Blood digoxin of the patient during the hospitalization, measured using conventional immunofluorescence assays.

Day of hospital stay	Day 1	Day 2	Day 4	Day 5
Digoxin Level (ng/mL)	0.50	0.75	1.41	0.60

stemming from other natural sources, such as mushrooms, versus those resulting from plant exposures [1]. Cases of plant intoxication are not frequently referred to EDs, and, as such, the continued and often expanding utilization of plants and herbal preparations highlights the need for enhanced medical education to increase awareness among general practitioners and ED medical team. Plant poisonings are much more common in the pediatric setting, usually due to curiosity and generally not severe. In adults, ingestions are mostly for suicidal purposes, and therefore the amount consumed is different. In this particular case, however, the poisoning, although not intended for suicidal purposes, involved a significant amount of toxins that indeed led to the onset of severe clinical manifestations.

In conclusion, this study's multi-faceted insights collectively contribute to a broader understanding of toxicological emergencies. The complex interplay between detection methodologies, delayed symptomatology, collaborative networks, and the evolving landscape of plant-based product use underscores the need for a holistic and adaptable approach to clinical practice.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability

Data will be made available on request.

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