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CLINICAL, BIOCHEMICAL, AND ELECTROCARDIOGRAPHIC CHANGES IN YELLOW OLEANDER (THEVETIA PERUVIANA) SEED POISONING: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Poisoning is an important public health problem and contributes significantly to morbidity and mortality worldwide. Among plant poisonings, yellow oleander (Thevetia peruviana) seed poisoning is commonly encountered in South India and is frequently used for deliberate self-harm. The seeds contain potent cardiac glycosides which mainly affect the cardiovascular system and can produce life-threatening arrhythmias and conduction abnormalities. Clinical manifestations vary from gastrointestinal symptoms to severe cardiac toxicity depending on the quantity consumed and time of presentation to the hospital. Electrocardiographic abnormalities and biochemical disturbances, especially hyperkalemia, play a major role in determining the severity and prognosis of poisoning. Hence this study was conducted to assess the clinical, biochemical and electrocardiographic changes among patients with yellow oleander seed poisoning.

Materials and Methods: A prospective observational study was conducted in the Department of General Medicine, Coimbatore Medical College Hospital, Tamil Nadu from June 2023 to July 2024. It is a prospective observational study conducted among 100 adult patients with confirmed yellow oleander seed poisoning. Patients with pre-existing cardiac disease, concurrent intake of cardiotoxic drugs, known dyselectrolytaemia, ingestion of plant parts other than seeds and paediatric patients were excluded from the study.

The subjects were enrolled after obtaining informed consent. Institutional Ethical Committee approval was obtained and a structured proforma was used to collect data regarding sociodemographic details, quantity of seeds consumed, form of ingestion, time interval between ingestion and hospital presentation, clinical manifestations, electrocardiographic changes and biochemical parameters. Serial 12-lead ECGs were recorded on admission, Day 3 and Day 5. Biochemical investigations including serum potassium, serum creatinine and serum CPK-MB were analysed. The data was collected, entered and analysed using SPSS software version 22.0. Descriptive analysis of study variables were expressed as frequencies, percentages, mean and standard deviation. Student's independent t-test, Fisher's exact test and Chi-square test were used to assess the association between variables. A p-value less than 0.05 ($p<0.05$) was considered statistically significant.

Results: The mean age was 35.43 ± 14.32 years; males predominated (78%). ECG abnormalities were detected in 46% of patients. The most common ECG change on Day 1 was sinus bradycardia (24%) followed by first-degree heart

block (16%). ECG changes showed significant resolution by Day 3 (9% abnormal) and Day 5 (1% abnormal). Serum potassium was significantly higher in patients with ECG changes (4.71 ± 0.53 vs. 3.91 ± 0.44 mEq/L; p=0.001). Consumption of ≥4 seeds was associated with 100% ECG abnormality (p=0.001). All patients survived; no mortality was recorded.

Conclusion: The study concluded that yellow oleander seed poisoning commonly produces electrocardiographic abnormalities such as sinus bradycardia and heart block. Hyperkalemia, higher number of seeds consumed and advancing age showed significant association with cardiac toxicity and ECG changes. Early hospital presentation and prompt management contributed to favourable clinical outcomes and complete recovery in most patients. Serum potassium can be used as a reliable marker for assessing severity of cardiotoxicity in yellow oleander poisoning.

Keywords: yellow oleander; Thevetia peruviana; cardiac glycoside; poisoning; ECG changes; hyperkalaemia, sinus bradycardia.

INTRODUCTION

Poisoning accounts for over one million illnesses globally per year; in India, an estimated 50,000 deaths annually are attributed to toxic exposures.[1] Among plant-based toxins, yellow oleander (Thevetia peruviana) is one of the most frequently implicated agents in South India and Sri Lanka, where it is commonly used as a means of deliberate self-harm.[2] Thevetia peruviana belongs to the Apocynaceae family and contains potent cardenolide cardiac glycosides—principally thevetin A, thevetin B, nerifolin, peruvoside, ruvoside, and thevetoxin—concentrated primarily in the seed kernel.[3] These glycosides inhibit the sarcolemmal Na+/K+ ATPase pump, raising intracellular sodium and calcium, which leads to increased automaticity, delayed after-depolarisations, and ultimately malignant arrhythmias.[4] Cardiotoxicity represents the chief cause of death, yet the spectrum of ECG changes and their temporal evolution, alongside biochemical correlates such as serum potassium and CPK-MB, are incompletely characterised in Indian cohorts.

This prospective study was undertaken to (1) document the clinical and demographic profile of yellow oleander seed poisoning in adults, (2) characterise ECG changes and their temporal resolution, and (3) correlate biochemical parameters with the occurrence of cardiac toxicity.

MATERIALS AND METHODS

This study was conducted in the Department of General Medicine, Coimbatore Medical College Hospital (CMCH), Tamil Nadu, India from June 2023 to July 2024. It is a single-centre prospective observational study conducted among adult patients with yellow oleander seed poisoning. Institutional Ethical Committee approval was obtained and written informed consent was obtained from all the study participants.

A total of 100 patients aged 18 years and above admitted with confirmed yellow oleander seed poisoning were included in the study. Patients with pre-existing cardiac disease, concurrent intake of cardiotoxic drugs, known dyselectrolytaemia, ingestion of plant parts other than seeds and paediatric patients were excluded from the study.

The subjects were evaluated using a structured proforma which included sociodemographic details, quantity of seeds consumed, form of ingestion, prior first aid received and duration between symptom onset and hospital presentation. Detailed systemic examination and vital signs were recorded. Serial 12-lead electrocardiograms were performed on admission (Day 1), Day 3 and Day 5. Continuous cardiac monitoring was carried out for the first 24 hours and echocardiography was performed for all patients.

Baseline biochemical investigations including serum creatinine, blood urea, random blood glucose, serum sodium, serum potassium and serum CPK-MB were analysed. Serum potassium levels were reassessed every six hours among patients with ECG abnormalities.

All patients received gastric lavage with normal saline, intravenous fluids and corticosteroids. Sinus bradycardia was managed with oral orciprenaline. Patients with heart rate less than 40 beats/minute were shifted to intensive care unit and treated with atropine. Digoxin-specific antibody fragments and temporary cardiac pacing were not available in the institution.

The data were collected, entered and analysed using SPSS software version 22.0. Descriptive analysis of study variables were expressed as frequencies, percentages, mean and standard deviation. Student's independent t-test, Fisher's exact test and Chi-square test were used to assess the association between variables. A p-value less than 0.05 ($p<0.05$) was considered statistically significant.

RESULTS

Demographic and clinical profile

One hundred patients were enrolled over the 8-month study period. Their demographic and clinical characteristics are summarised in Table 1. The mean age was 35.43 ± 14.32 years (range 19–78 years),

with the majority (40%) in the 21–30-year age group. Males accounted for 78% of cases. Ninety-six percent of patients had received some form of first aid (gastric lavage at a primary health centre) prior to hospital admission. Gastrointestinal symptoms (predominantly vomiting) were present in 13% of patients, and palpitations were reported by 3%. No patient exhibited neurological manifestations. Echocardiography was normal in all 100 patients. There was no in-hospital mortality. The Mean hospital stay (days) was 5.61 ± 1.25 (range 3–10).

Table 1. Demographic and Clinical Profile of Study Participants (n=100)

Characteristic	n (%)
Total patients	100
Peak age group (21–30 years)	40 (40%)
Male	78 (78%)
Female	21 (21%)
Patients receiving first aid	96 (96%)
GI symptoms (vomiting)	13 (13%)
Cardiac symptoms (palpitations)	3 (3%)
CNS symptoms	0 (0%)
ICU admission	47 (47%)
Mortality	0 (0%)

ECG findings and their temporal resolution

Forty-six patients (46%) demonstrated ECG abnormalities. The distribution of ECG changes across Day 1, Day 3, and Day 5 is shown in Table 2. On Day 1, the commonest abnormality was sinus bradycardia (24%), followed by first-degree heart block (16%). Second-degree heart block and atrial fibrillation were observed in 2% and 1% of patients, respectively. No ventricular tachycardia or ventricular fibrillation was recorded. There was marked temporal improvement: by Day 3, 91% of ECGs had normalised, and by Day 5, 99% were normal. Among patients with first-degree heart block on Day 1, 14 out of 16 had reverted to normal by Day 3. All patients with ECG changes required ICU admission (97.9% vs. 0% without ECG changes; p=0.001; Table 6).

Table 2. Serial ECG Changes on Days 1, 3, and 5 Post-Ingestion (n=100)

ECG Finding	Day 1 n (%)	Day 3 n (%)	Day 5 n (%)
Normal	54 (54%)	91 (91%)	99 (99%)
Sinus bradycardia	24 (24%)	5 (5%)	1 (1%)
First degree heart block	16 (16%)	2 (2%)	0 (0%)
Second degree heart block	2 (2%)	1 (1%)	0 (0%)
Atrial premature complexes	3 (3%)	0 (0%)	0 (0%)
Atrial fibrillation	1 (1%)	1 (1%)	0 (0%)

Biochemical correlates of cardiac toxicity

Serum potassium was significantly elevated in patients with ECG changes compared to those without (4.71 ± 0.53 vs. 3.91 ± 0.44 mEq/L; mean difference 0.796; p=0.001). Serum creatinine showed a non-significant trend towards higher values in the ECG-change group (0.93 ± 0.21 vs. 0.86 ± 0.24 mg/dL; p=0.086). Serum CPK-MB did not differ significantly between groups (20.54 ± 3.82 vs. 22.15 ± 6.16 IU/L; p=0.115). These data are presented in Table 3.

Table 3. Biochemical Parameters in Relation to ECG Changes

Parameter	Group	N	Mean ± SD	Mean diff.	p value
Serum potassium (mEq/L)	ECG changes	46	4.71 ± 0.53	0.796	**0.001**
	No ECG changes	54	3.91 ± 0.44		
Serum creatinine (mg/dL)	ECG changes	46	0.93 ± 0.21	0.077	0.086
	No ECG changes	54	0.86 ± 0.24		
Serum CPK-MB (IU/L)	ECG changes	46	20.54 ± 3.82	1.607	0.115
	No ECG changes	54	22.15 ± 6.16		

Dose–response relationship: seeds consumed and ECG changes

A clear dose-response relationship was observed between the number of seeds ingested and the likelihood of ECG abnormality (Table 4). Patients who consumed 1 seed had a 34.1% risk of ECG change, rising to 100% for those who consumed 4 or more seeds (p=0.001). The mean seed count was significantly higher in patients with ECG changes (2.33 ± 1.30 vs. 1.63 ± 0.71; p=0.002).

Table 4. Number of Seeds Consumed vs. ECG Change Occurrence

No. of seeds	ECG changes n (%)	No ECG changes n (%)	Total n (%)	p value
1	14 (34.1%)	27 (65.9%)	41 (100%)	**0.001**
2	17 (45.9%)	20 (54.1%)	37 (100%)	
3	5 (41.7%)	7 (58.3%)	12 (100%)	
4	7 (100%)	0 (0%)	7 (100%)	
5	2 (100%)	0 (0%)	2 (100%)	
6	1 (100%)	0 (0%)	1 (100%)	

Age group and ECG changes

Patients older than 60 years showed a 100% rate of ECG abnormality, and the association between advancing age group and ECG changes was statistically significant (p=0.022; Table 5). Sex did not significantly influence ECG change rates (males 48.7% vs. females 38.1%; p=0.094).

Table 5. Age Group vs. ECG Change Occurrence

Age group (years)	ECG changes n (%)	No ECG changes n (%)	Total n (%)	p value
≤20	2 (28.6%)	5 (71.4%)	7 (100%)	**0.022**
21–30	17 (42.5%)	23 (57.5%)	40 (100%)	
31–40	11 (50%)	11 (50%)	22 (100%)	
41–50	7 (50%)	7 (50%)	14 (100%)	
51–60	4 (33.3%)	8 (66.7%)	12 (100%)	
>60	5 (100%)	0 (0%)	5 (100%)	

ICU stay and outcome

All 46 patients with ECG changes required ICU admission compared to none (0%) of the 54 patients without ECG changes (p=0.001; Table 6). Mean hospital stay was 5.61 ± 1.25 days. All 100 patients were discharged with a normal ECG; there was zero in-hospital mortality.

Table 6. ICU Stay vs. ECG Change Occurrence

ICU stay	ECG changes n (%)	No ECG changes n (%)	Total n (%)	p value
Yes	46 (97.9%)	1 (2.1%)	47 (100%)	**0.001**
No	0 (0%)	53 (100%)	53 (100%)	

DISCUSSION

This prospective study of 100 adults with yellow oleander seed poisoning provides a comprehensive characterisation of the clinical, ECG, and biochemical profile of this condition in a South Indian tertiary-care setting. The salient findings are: (1) ECG abnormalities occurred in 46% of patients with sinus bradycardia and first-degree heart block as the predominant patterns; (2) cardiac toxicity showed rapid temporal resolution, with 91% normalisation by Day 3; (3) serum potassium and seed quantity were the strongest biochemical and exposure-related predictors of ECG change; and (4) zero mortality was recorded with conservative pharmacological management.

The demographic profile—predominantly young males (mean age 35.43 years, peak 21–30 years)—is consistent with published literature from Sri Lanka and South India.[2,5] Fonseka et al. reported a mean age of 34.8 years from a Sri Lankan series, though with female preponderance[5]; our male-to-female ratio of 3.7:1 likely reflects regional sociocultural differences in suicidal behaviour patterns.

The mechanism underlying oleander cardiotoxicity is well-established. Cardenolide glycosides competitively inhibit the Na+/K+ ATPase pump on the cardiac myocyte membrane, causing intracellular sodium accumulation.[4,6] This reduces calcium extrusion via the Na+/Ca2+ exchanger, producing intracellular hypercalcaemia, which enhances automaticity through delayed after-depolarisations and an inward transient current—the primary electrophysiological mechanism of arrhythmogenesis.[6,7] The resultant bradyarrhythmias (sinus bradycardia 24%, first-degree heart block 16%) mirror the "digitalis-like" ECG pattern described in earlier reviews.[3,8] The absence of ventricular fibrillation or tachycardia in our cohort may reflect the relatively low seed dose consumed (mean 1.9 seeds; 78% ≤2 seeds) and the prompt institution of gastric lavage in 96% of patients.

The dose–response relationship observed between seed count and ECG toxicity (p=0.001)—with 100% ECG abnormality for ≥4 seeds—corroborates earlier work by Sreeharan et al. and Saravanapavananthan et al., who identified a similar threshold in Sri Lankan cohorts.[9,10] The estimated lethal dose of 8–10 Thevetia peruviana seeds in adults[11] contextualises our finding: the majority of patients consumed sub-lethal quantities, explaining the zero mortality.

Serum potassium emerged as the single most significant biochemical discriminator between patients with and without ECG changes (4.71 vs. 3.91 mEq/L; p=0.001). The pathophysiology is direct: Na+/K+ ATPase inhibition impairs cellular potassium re-uptake, causing extracellular hyperkalaemia that further depresses the resting membrane potential and potentiates arrhythmogenesis.[4,6] Bismuth et al. similarly identified hyperkalaemia as a marker of severe acute oleander poisoning.[7] The lack of CPK-MB

elevation despite significant bradyarrhythmias is consistent with the predominantly electrophysiological (rather than myocardial necrotic) nature of oleander cardiotoxicity, confirming previous observations.[3]

The rapid ECG normalisation by Day 3 (91% normal) parallels the known pharmacokinetics of thevetin glycosides, whose elimination half-life approximates 72–96 hours for peruvoside and ruvoside.[3] All patients were discharged with a normal ECG, supporting a 5-day monitoring protocol as adequate for most cases. These findings align with Zamani et al.[8], who reported 40% bradyarrhythmia incidence and comparable resolution timelines.

The absence of digoxin-specific antibody fragments and temporary pacing facilities at our institution precluded their use; however, no patient required pacing and all survived. This underscores that with adequate gastric decontamination and careful pharmacological rate management, outcomes can be favourable even in resource-limited settings.

CONCLUSION

Yellow oleander seed poisoning predominantly affects young adults and produces ECG changes—chiefly sinus bradycardia and first-degree heart block—in 46% of cases, with rapid resolution by Day 3. Hyperkalemia and consumption of ≥4 seeds are strong independent predictors of cardiac toxicity. Serum potassium is a practical, widely available biomarker for risk stratification in emergency settings. Conservative management with gastric decontamination, electrolyte correction, and careful use of atropine or orciprenaline yielded zero mortality in this cohort. These findings have direct implications for triage, monitoring duration, and clinical management protocols in regions where oleander poisoning is endemic.

Limitation:

Serum thevetin glycoside assay and digoxin-specific antibody fragments were not available in the institution. Hence the results cannot be generalised to the entire population.

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Conflict Of Interest: Nil

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