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A case series of oleander poisoning: challenges faced by emergency physicians in developing countries

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Abstract: **Objective:** Through the reporting of this case series, we aim to establish whether a conservative approach, through managing arrhythmias and vital signs, can be reliably used as a treatment modality for oleander poisoning in developing countries.
Methods: This study is a case series of 11 patients who presented with oleander poisoning and were conservatively managed in the absence of standard antidote.
Results: All 11 patients treated with conservative approach survived. Conservative approach included use of atropine for management of symptomatic bradycardia followed by Dopamine infusion, correction of serum potassium and magnesium levels, standby defibrillation, and transvenous pacing.
Conclusion: The absence of reliable dosage of poison ingested, the lack of facilities for serum digoxin estimation, and the unavailability of digoxin fab antibodies pose challenges for the management of patients with oleander poisoning. Patients can, however, be managed conservatively following the Advanced Cardiac Life Support (ACLS) algorithm in a setting that lacks the standard treatment of this poison.

Keywords: Antibodies; Digoxin; Thevetia; Nerium; Poisoning; Self-Injurious Behavior; Suicide

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1. Introduction

Yellow Oleander (Thevetia peruviana) (Figure 1) and White Oleander / Common Oleander / Pink Oleander (Nerium oleander) are commonly found in tropical and subtropical countries around the world. They are widely cultivated as hedges on landscape plots due to their thick and sturdy growth, which acts as an impregnable barrier when planted closely in rows. Due to attractiveness of the plant, many house owners and municipalities plant it; thus, the potential for exposure amongst humans is high. All parts of the plant are toxic upon ingestion, especially the seeds (1). The cases of accidental poisoning have been reported worldwide, especially in children (2, 3). The ubiquitous presence of the plant and knowledge of its toxicity has caused it to be a common source of poison to be used for self-harm (4, 5). The Cardiac Glycosides contained in White oleander are Oleandrin, Folinerin, and Digitoxigenin. Thevetin A, Thevetin B, Thevetoxin, Nerifolin, Peruviside and Ruvoside are the cardiac glycosides in Yellow oleander (1). Seeds have the highest concentration of cardiotoxic glycosides. In our state, the flowers are sometimes used as an offering in temples and are colloquially referred to as "Kanail ka phool" i.e; The flowers of Kanail.

The cardiac glycosides contained in both yellow and common oleanders bind to the sodium- potassium ATPase pump on the extracellular phase, thus, inhibiting its action and causing a rise in intracellular sodium and calcium. This excess intracellular calcium causes increased ionotropic effect and makes the heart prone to arrythmias and hypotension (1). Patients present to the Emergency medicine department with complaints of nausea, vomiting, altered sensorium, sweating, abdominal pain and diarrhea (5, 6). Bradycardia and irregular pulse are the most common clinical signs. Organophosphate (OP) poisoning is an important differential diagnosis to be considered. The absence of typical pungent garlicky smell and the absence of circumstantial evidence of any container of organophosphate or carbamate should point towards the diagnosis of oleander poisoning. Seeds are often crushed and grinded to form a paste before consumption. Upon electrocardiography, bradycardia, dysrhythmias, conduction blocks, and ST changes will be noted, a feature not seen in early OP poisoning. Hypotension occurs late in oleander poisoning. Death occurs commonly proceeding ventricular fibrillations. Due to varying concentrations of the poison prepared for ingestion, absorption kinetics of patients, and the resolution and reappearance of cardiac arrhythmias, it is difficult to ascertain the time interval between consumption of poison and death.

2. Methods

This study is a cluster of 11 cases, who have presented to the emergency medicine department (ED) over the past 3 years. All patients with documented evidence of oleander consumption in the form of poison, brought to ED (Whatsapp images of the poison taken at home by the attendant) were included. Patients and attendants who could not pro-

Figure 1 Yellow Oleander (Thevetia peruviana)

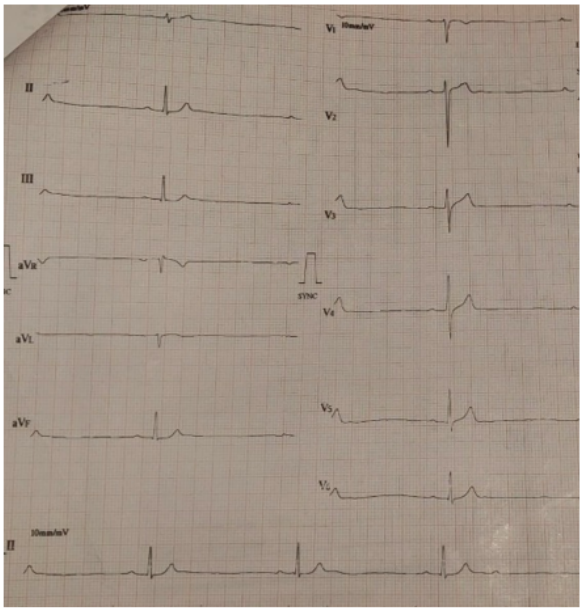

Figure 2 Sinus Bradycardia (Heart rate – 38 beats/min; Rhythm – Regular and slow; P wave – normal, one per QRS; PR interval – 0.12 to 0.2, consistent; QRS complex – normal, <0.12)

vide any positive identification of the poison consumed were excluded. Due to the unavailability of Digoxin antibodies, only supportive treatment was given to all patients. Upon admission and confirmation of oleander poisoning, the patients were given repeated doses of atropine up to 3 mg titrated as per symptomatic bradycardia. If the bradycardia did not resolve, then the patient was given Dopamine infusion at 5-20 mcg/kg/min. Fluids were given bolus and on maintenance dosage as per the presentation of the patient. After the patients were stabilized, a gastric lavage was performed with a pinch of potassium permanganate until clear solution obtained on suctioning. Medico-legal information was given to the police in all the patients. Cardiology review was performed for all patients with a 2D ECHO. The patients were placed in emergency intensive care unit (ICU) for least 48 hours or until the resolution of arrhythmia, whichever was later. During the time of discharge, a psychiatry consultation was done to address the stressor factor.

3. Results

All patients reported in this study survived. No adverse events were noted. Conservative management, using bradycardia algorithm in the Advanced Cardiac Life Support (ACLS) protocol, was followed. Due to the unavailability of serum digoxin level estimation and the lack of digoxin fab antibodies in the city, only conservative management was appropriated for the patients. The average duration of hospital stay for the patients was 3.45 days, with the average duration of Emergency ICU stay being 67 hours. The intent of all patients was suicidal and the only route of poison consumption was oral. All patients had consumed seeds. 8 of the 11 patients had crushed the seeds before consumption and 3 had consumed the seeds as whole. Two patients had consumed white oleander and upon taking detailed history, they admitted to having consumed white oleander only because it was more readily accessible to them than yellow oleander. All patients reported the presence of the oleander plant within half a kilometer of their house. The male to female ratio was 7:4 and the mean age of patients presenting was 32.63 years. The maximum age of the patients presenting was 56 years, and the minimum age was 17 years. Sinus Bradycardia (Figure 2) was the most common rhythm on presentation. 54 % of the patients had atrioventricular (AV) blocks, all of which had 1st degree AV block. Ventricular ectopics were seen in 7 patients and the electrocardiogram (ECG) of only one patient exhibited junctional rhythm. The cardiac rhythm of patients was continuously monitored on a 5 – para monitor, and a 12 lead ECG was performed every 4 hours. None of the patients underwent a ventricular fibrillation / Ventricular tachycardia. Cardiac troponin markers were studied on admission, all of which came out to be normal. Serum electrolyte was repeated every 8 hours for 3 of the patients, who showed hyperkalemia on presentation, one of which was co-morbid with Chronic kidney disease. Potassium was corrected in the patients and the scheduled dialysis was delayed till the resolution of arrhythmia. Bradycardia resolved within 8 hours of initiation of dopamine infusion and temporary pacing was not done for any patient. Five patients had recurrence of arrhythmias during the course of hospital stay, which resolved on initiation of repeat dosage of atropine followed by Dopamine infusion.

Table 1 Overview of patients with oleander poisoning

Sr.no	Sex	Age (Years)	Oleander	Arrhythmias during course of hospitalization	Duration of admission (Days)
1	F	56	Yellow	Sinus Bradycardia, First degree AV Block, Ventricular Ectopics	3
2	M	17	Yellow	Sinus Bradycardia, Ventricular Ectopics, Junctional rhythm	4
3	M	22	White	Sinus Bradycardia, First degree AV Block, Ventricular Ectopics	5
4	M	34	Yellow	Sinus Bradycardia	3
5	F	45	Yellow	Sinus Bradycardia, First degree AV Block, Ventricular Ectopics	2
6	M	42	Yellow	Sinus Bradycardia	3
7	F	36	Yellow	Sinus Bradycardia, Ventricular Ectopics	4
8	M	29	Yellow	Sinus Bradycardia, First degree AV Block	3
9	M	23	White	Sinus Bradycardia, First degree AV Block, Ventricular Ectopics	4
10	M	19	Yellow	Sinus Bradycardia, First degree AV Block, Ventricular Ectopics	3
11	F	36	Yellow	Sinus Bradycardia	4

AV: atrioventricular.

4. Discussion

The poisonous properties of the plant, in particular the seeds, are common knowledge amongst the population of the Indian subcontinent. It is supported by our study, that all the patients used the seeds of the plant as a means to cause self-harm. In 1980, reports being published in the local newspaper about the poisonous properties of the plant led to a dramatic rise in the consumption of yellow oleander as a means to cause self-harm from 1981-1983 (5, 7). All the patients in our study were aware that consumption of fruit, seeds, and the flowers can cause death. Two of our patients had witnessed the death of street animals due to consuming parts of the plant. In order to avoid a situation similar to that in Sri Lanka, we took care that the identity and the nature of poisoning of our patients were not reported in the local media.

Various studies have been published on oleander poisoning with varying mortality rates from negligible to 20% (2, 8, 9). Our study reports the mortality of 0%. It can be argued that studies in which a higher mortality rate has been reported, have mostly been from a time period when oleander poisoning had become a major public health crisis of epidemic proportions. After that decade, when suicide by oleander poisoning has not been so much in news and cases have been reported sporadically, the mortality rates have come down. This may be in part due to lack of awareness about the fatal dosage of the different parts of the plant. It has been speculated that one oleander seed has an effect equivalent to 100 digoxin tablets, but the accurate fatal dose in terms of the number of seeds is not known with figures ranging from 1-10 (4, 10). The patients in our study could not recall the number of seeds consumed but eight of them knew about crushing the seeds before consuming, a fact that has been associated with higher mortality.

Sinus Bradycardia is the most common arrhythmia associated with oleander poisoning (8). Junctional rhythm, first degree heart block, and ventricular ectopics are also very commonly seen often in the same patient over the course of hospitalization (11). The incidence of ventricular tachycardia and ventricular fibrillation has been reported to be around 0.8-14% and 0.4-4%, respectively (8, 11). The wide array of cardiac arrhythmias is often easy to manage as the patients are young and free from cardiac morbidity. Temporary transvenous pacing has been needed in 3% of the patients (12). The use of Anti Digoxin fab antibodies has been popularized since 2000 in a randomized control trial, where it was stated that its use could bring down the mortality of patients (13). Ever since, it has been universally accepted as the antidote for naturally occurring cardiac glycosides. One of the major limitations of this study was the inability to use Digoxin immune fab due to its unavailability and high cost. The lack of serum digoxin level estimation further limited our scope of using digoxin – fab antibodies. No long term effects have been noted due to oleander poisoning (14).

5. Conclusion

Based on our experience in treating oleander poisoning, we would like to suggest that the absence of reliable dosage of poison ingested, the lack of facilities for serum digoxin estimation, and the unavailability of digoxin fab antibodies pose challenges in the management of patients with oleander poisoning. Patients can, however, be conservatively managed following the ACLS algorithm in a setting that lacks the standard treatment of this poison.

6. Declarations

6.1. Acknowledgment

None.

6.2. Authors' contributions

All authors passed four criteria for authorship contribution based on recommendations of the International Committee of Medical Journal Editors.

6.3. Conflict of 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.

6.4. Funding

The study did not receive funding from any source.

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