# CLINICAL OUTCOMES AND MANAGEMENT OF OLEANDER SEED POISONING: A CASE SERIES

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## ABSTRACT

**Background**: The oleander or Nerium oleander is a common ornamental plant known for: can result in severe toxicity that can impact several organ systems and can be fatal. This case series describes the management approaches, clinical presentation, and results of oleander poisoning cases.

**Methods**: Medical records of patients with proven oleander toxicity were collected based on clinical presentation and toxicological investigation. Data on demographics, circumstances of exposure, clinical features, laboratory findings, treatment modalities, and clinical outcomes were collected and analyzed.

**Results**: The majority of patients presented with gastrointestinal symptoms vomiting. Treatment strategies included supportive care, gastrointestinal decontamination, and therapy for comorbid conditions. Early recognition of symptoms and prompt initiation of supportive measures are crucial in improving patient outcomes.

**Conclusion**: This case series highlights the spectrum of clinical manifestations and treatment strategies for oleander poisoning. Improved awareness among healthcare providers, timely supportive care, and targeted therapeutic interventions are essential in reducing morbidity and mortality associated with oleander toxicity.

**Keywords:** oleander, cardiac glycoside, gastric lavage, anti digoxin Fab antibodies, supportive care, Toxicity.

**INTRODUCTION**:

Oleander is an ornamental evergreen shrub of the genus Nerium belonging to the family Apocynaceae, found in the tropics and subtropical countries. Currently, over fifty different varieties of oleander plants are cultivated for gardening purposes [(1)] out of which 3 types are more commonly seen in India: yellow, white, and pink. Oleander contains cardiac glycosides in its seeds, roots, leaves, flowers, fruits, branches, and stems, of which yellow is more toxic[(2)]. The cardiac glycosides in white oleander are oleandrin, folinerin, and digitoxegenin, while those in yellow oleander include thevetin A, B, thevetoxin, nerifolin, peruviside, and roveside[(3)]. Out of these components, oleandrin is the most potent, with more cardiotoxic effects [(2)]. However, the whole plant, including the sap, possesses the same lethal effect [(4)]. Self-harm from the consumption of oleander plants is common in India and Sri Lanka[(1)(5)(6)]. Yellow oleander glycosides were used in the management of atrial fibrillation in the 1930s, in India, until digoxin came [(7)]. The clinical manifestation of yellow oleander poison is the same as that of digoxin poisoning, which is vomiting, diarrhea, bradycardia, dizziness, sinusitis, and AV block[(7)]. GI symptoms include nausea, vomiting, abdominal pain, and diarrhea. Neurological symptoms include tremors, ataxia, and drowsiness. CVS includes sinus bradycardia, AV block, and atrial fibrillation [(4)]. The effect of 100 digoxin tablets is seen in one oleander seed. This works by providing a positive ionotropic effect and slowing the heart, like digoxin [(3)] by inhibiting the Na-K-ATPase of myocytes and conducting systems. The inhibition of the Na+/K+ pump results in the accumulation of $Na^+$ inside and $K^+$ outside the cell. The $Na^+$ $Ca^{2+}$ exchange pump will usually pump $Ca^{2+}$ out of the cell and $Na^+$ inside but because of high intracellular $Na^+$ levels, this exchange is stopped, which in turn will result in a high intracellular $Ca^{2+}$ level, which may be the cause of ventricular arrhythmias. These effects can eventually lead to a rise in the excitability and automaticity of cardiac cells during both the early and late depolarization stages. The patient's $K^+$ level will be elevated due to the inhibition of $Na^+$ $K^+$ ATPase. As a result of all these changes, the patient may develop arrhythmias and hypotension[(7)].

The primary assessment and management are the same as those of other poison management methods. ECG monitoring is to be done for at least 24 hours to find arrhythmias. Supportive care, like normal saline for the correction of dehydration and antiemetic for severe vomiting, can be used to correct electrolyte abnormalities; in hyperkalemia, an insulin dextrose infusion is used that causes an extracellular shift of $K^+$. Gastric decontamination processes, like emesis induction and gastric lavage, are common clinical practices. Activated charcoal is used to prevent poison absorption in the patient's stomach and induce vomiting. Atropine and orciprenaline are used for the treatment of bradyarrhythmia[(7)]. Anti digoxin Fab has been used since 2000 as an antidote universal, but its use is limited due to its availability and excessive cost[(3)].FDP is a newer antidote under trial for the treatment of oleander poisoning. However, the lack of drugs/facilities limits the direct management of oleander poisoning in developing countries[(7)]. Hence this case series highlights the clinical manifestation after ingestion of oleander seed and therapeutic management is outlined.

## CASE REPORT

**CASE 1**:

A 64-year-old male patient was admitted to the general medicine ward with an alleged history of consumption of oleander seed at his residence with the compliance of loss of consciousness. The patient consumed four oleander seeds upon mixing them with alcohol. The patient exhibited no symptoms; there was no vomiting, frothing of the mouth, headache, or abdominal pain. The patient's personal history is a mixed diet, normal bowel, and bladder habits, and being a chronic alcoholic and smoker for the past 15 years. On physical examination, the patient is drowsy, disoriented, responds to painful stimuli, is afebrile, restless, and has a breath smell of alcohol. His vital signs were as follows.

**TABLE 1:**

| | | | |
|---|---|---|---|
| BP (mmHg) | 112/84 | 140/70 | 140/70 |
| Pulse (bpm) | 102 | 86 | 88 |
| $Spo_2$ | 98% | 98% | 97% |

The patient's laboratory investigations are given in table 5. Echo screening is done it shows a normal study with an ejection fraction of 52%. The ECG shows a normal sinus rhythm.

The patient initially underwent gastric lavage and started IV fluids at 75ml/hr.Inj.Thiamine100mg OD, Inj.Ranitidine50mg BD, Inj.Atropine1cc stat given. Tablet chlordiazepoxide and tablet B complex OD were given to the patient. The patient was discharged in a stable condition after 4 days of hospital admission.

**CASE 2:**

A 23-year-old male patient was admitted to the general medicine department for 3 days with an alleged history of oleander seed poisoning. 2 numbers were crushed and taken at a nearby residence. He has the symptoms of vomiting in four episodes; the patient exhibited no other symptoms like frothing of the mouth, headache, or abdominal pain. The patient's personal history is a mixed diet, normal bowel, and bladder habits, and being a chronic alcoholic for 6 years. On physical examination, the patient is drowsy, oriented, responds to painful stimuli, is afebrile, and has a breath smell of alcohol. His vital signs were as follows: blood pressure:

**TABLE 2:**

| | | | | |
|---|---|---|---|---|
| BP(mmHg) | 120/70 | 114/70 | 110/70 | 110/70 |
| Pulse(bpm) | 108 | 102 | 90 | 90 |
| $SPO_2$ | 98% | 98% | 98% | 98% |

The patient's laboratory investigations are given in table 5. The patient initially underwent gastric lavage and started IV fluids at 75 ml/hr., Inj.Thiamine100mg OD, Inj.Ranitidine BD, Inj.Atropine 1cc stat was given to the patient. After 3 days of admission, the patient was discharged in a stable condition.

**CASE 3:**

A 48-year-old male patient was admitted to the general medicine ward with an alleged history of consumption of oleander seed near a field with complaints of vomiting. The patient consumed three oleander seeds. The patient exhibited no other symptoms like frothing of the mouth, headache or abdominal pain, chest pain, palpitation, or breathing difficulties. The patient has a known history of type 2 diabetes mellitus on treatment (T. metformin 500mg and T. glimepiride 1 mg) for the past year. The patient's personal history is a vegetarian diet and normal bowel and bladder habits. On physical examination, the patient is conscious, oriented, and afebrile. His vital signs were as follows:

**TABLE 3:**

| | | | | | |
|---|---|---|---|---|---|
| Bp(mmHg) | 110/80 | 112/76 | 128/82 | 100/80 | 100/70 |
| Pulse(bpm) | 98 | 96 | 92 | 71 | 86 |
| $SPO_2$ | 98% | 98% | 99% | 94% | 89% |

The patient's laboratory investigations are given in table 5.ECG shows normal sinus rhythm. The patient initially underwent gastric lavage and started IV fluids at 75 ml/hr. Inj. Thiamine100mg OD, Inj.Ranitidine50mgBD, Inj.Dexamethasone8mg OD given. T.Cetirizine 10mg OD. By the completion of 5th day of treatment, the patient suddenly had breathing difficulties and was referred to a tertiary care hospital.

**CASE 4:**

A 34-year-old female patient was admitted to the general medicine department for 4 days with an alleged history of consumption of 4 oleander seeds ground and mixed with food and experiencing vomiting. Other than that, the patient exhibited no other symptoms. The patient's personal history is a mixed diet and normal bowel and bladder habits. On physical examination, the patient is conscious, oriented, and afebrile. His vital signs were as follows:

**TABLE 4:**

| | | | | |
|---|---|---|---|---|
| Bp(mmHg) | 130/80 | 120/70 | 110/70 | 100/70 |
| Pulse(bpm) | 92 | 90 | 90 | 92 |
| $SPO_2$ | 97% | 97% | 97% | 97% |

The patient's laboratory investigations are given in table 5. ECG shows normal sinus rhythm. The Patient initially underwent gastric lavage and started IV fluid 100ml/hr, Inj. Ranitidine 50mg BD, Inj. Ondansetron 8mg iv BD, T. Orciprenaline 10mg TDS, Inj. Atropine 1cc Stat.

**TABLE 5:**
**LABORATORY INVESTIGATION:**

| **Parameters** | **Case 1** | **Case 2** | **Case 3** | **Case 4** |
|---|---|---|---|---|
| HB (g/dL) | 12.6 | 14.8 | 13 | 10.2 |
| RBC (1012/L) | 3.7 | 4.8 | 5.0 | 4.65 |
| WBC (109/L) | 7.5 | 6.9 | 6.6 | 6.21 |
| PLT (109/L) | 285 | 249 | 285 | 370 |
| MCHC (g/dL) | 34 | 35.9 | 32 | 33.4 |

| | | | | |
|---|---|---|---|---|
| MCH (pg) | 28 | 27.5 | 29.4 | 28.6 |
| MCV (fL) | 81 | 85 | 82 | 81 |
| RBS (mg/dL) | 96 | 98 | 100 | 85 |
| UREA (mg/dL) | 18 | 20 | 15 | 26 |
| CREATININE (mg/dL) | 0.7 | 0.8 | 0.9 | 0.7 |
| T.PROTEIN (mg/dL) | 7 | 6.5 | 6 | 6.9 |
| ALBUMIN (g/dL) | 2# | 3.6 | 4.3 | 3.7 |
| T. BILIRUBIN (mg/dL) | 0.3 | 0.3 | 0.4 | 0.6 |
| SGOT (IU/L) | 40* | 29 | 20 | 16 |
| SGPT (IU/L) | 26 | 40 | 14 | 23 |
| ALKP (IU/L) | 305* | 174 | 223 | 58 |
| Na +(mmol/L) | NA | NA | NA | 138 |
| K +(mmol/L) | NA | NA | NA | 3.8 |

**#: slightly decreased *: slightly elevated NA: not assessed**

**DISCUSSION:**

Oleander is an ornamental plant that consists of cardiac glycoside. All parts of the plants have toxic effects. The effect of 100 digoxin tablets is equal to one oleander seed. The most common effect seen in the consumption of oleander seed is bradycardia. Atropine is widely used for the treatment of bradycardia. Atropine which is a centrally acting anticholinergic increases the heart rate and antagonizes the vagomimetic effect in oleander seed poisoning.

The severity of poisoning is correlated with the number of seeds consumed ($p<0.001$), and 8–10 seeds are considered to be fatal doses, as reported by Lokesh *et al*[8] in their study, whereas in our study the patients had taken a maximum of 4 seeds and 2 seeds as a minimum, whereas Pirasteh *et al* [9]., didn't find any relation between the number of seeds consumed and its complications causing cardio toxicity.

Aparna *et al* [5]., mentioned in their study that the incidence of cardio toxicity was high in patients who consumed crushed seeds, but in contrast to this, our patients who consumed crushed seeds did not experience any cardiac events as they received initial treatment.

In their study, G. Gopalakrishnan *et al*[10], found that vomiting was predominant in most of the patients and also in patients receiving gastric lavage. Also, he states that symptoms and mortality risk were low in patients who received treatment within one hour of consumption, which aligns with our study as our patients had vomiting as the first symptom,

and gastric lavage was done within an hour, which reduced fatal symptoms and mortality risks. Our case series has no mortality because of initial treatment, which is parallel to the study conducted by Utsav Mani *et al*[8], who reported mortality as 0.1% in their study. The mortality rate has been based on time duration.

The hospital stay of patients was limited to 3-5 days due to a better prognosis, as reported by Eddleston *et al*[10]., In his study, in recent trials, anti-digoxin fab antibodies have been found to bring down the mortality of patients, as reported by Eddleston *et al*[10]., It is a universally accepted antidote for naturally occurring cardiac glycosides, due to its excessive cost and unavailability, it was not used in our study, which aligns with the study done by Utsav *et al*[3].,

In the first case, the patient has taken crushed seeds along with alcohol, as noted in a study by Gopalakrishnan *et al*[12]., who stated that consumption of seeds on an empty stomach worsens the pain, whereas alcohol co-ingestion did not alter the same. This was also stated in another study done by Mathan *et al*[1]., who reported that patients took oleander seeds along with food, and alcohol, or on an empty stomach.

Amrita *et al* [7]., in their study, say that IV calcium is not recommended in the treatment of hyperkalemia, which may lead to the accumulation of intracellular calcium and an increase in the risk of cardiac arrhythmias in these patients. In our case series, none of the patients experienced hyperkalemia; IV calcium was not given.

**CONCLUSION**:

The range of clinical symptoms and treatment measures for oleander poisoning are illustrated in this case series. To reduce morbidity and death related to oleander toxicity, we must be more aware of the risks, provide timely supportive care, implement focused therapeutic measures, and seek early management. In areas where exposure to oleanders still poses a serious threat to public health, prevention through awareness is necessary. In areas where exposure to oleanders still poses a serious threat to public health, prevention through awareness is necessary.

**Acknowledgement:**

We Sincerely express our gratitude to the department of pharmacy practice for providing internal guidance and encouragement.

**Conflict of interest:**

The authors declare no conflict of interest.

**REFERENCES:**

1. Mathan M, Bharathi G. A STUDY OF CLINICAL, BIOCHEMICAL AND ELECTROCARDIOGRAPHIC CHANGES IN OLEANDER POISONING IN A TERTIARY CARE CENTRE. Int J Acad Med Pharm. 2023;5(1):929-33
2. Okuda H, Fukushima H, Nakatsukasa T, Yamamoto K, Kaizaki-Mitsumoto A, Numazawa S, Kamijo Y. Fatal poisoning due to ingestion of boiled oleander leaf extract. Journal of forensic sciences. 2024 Jan;69(1):351-4

3. Mani, UtsavAnand& Kumar, Mukesh& Abbas, Haider& Gupta, Pranay. (2022). A case series of oleander poisoning: challenges faced by emergency physicians in developing countries. Frontiers in Emergency Medicine. 10.18502/fem.v6i4.10431

4. Beniwal, Shreya& Sharma, Akshay& Bhatia, Pranav&Kaura, Ashna&Vashisht, Rishabh& Gupta, Prakhar& Ibrahim, Abdulgafar. (2023). ACUTE CARDIAC TOXICITY OF NERIUM OLEANDER/INDICUM POISONING-A CASE REPORT Section A-Research paper ACUTE CARDIAC TOXICITY OF NERIUM OLEANDER/INDICUM POISONING-A CASE REPORT. 4914-4918. 10.48047/ecb/2023.12.10.3472023.17/07/2023
5. Aparna S, Sharmila M. Yellow oleander seed poisoning–a profile. IOSR J Dent Med Sci. 2017;16:64-71
6. Karthik G, Iyadurai R, Ralph R, Prakash V, Abhilash KP, Sathyendra S, Abraham OC, Truman C, Reginald A. Acute oleander poisoning: A study of clinical profile from a tertiary care center in South India. Journal of family medicine and primary care. 2020 Jan 1;9(1):136-40
7. Sundaram, Rajagopal& Prasad, Amrita &Kandasamy, Krishnaveni& K.A, Neha& T.D, Neethu&Ramanathan, Sambathkumar. (2016). A REVIEW ON MANAGEMENT OF COMMON OLEANDER AND YELLOW OLEANDER POISONING. WORLD JOURNAL OF PHARMACY AND PHARMACEUTICAL SCIENCES. 5. 493-503.
8. Shanmugam, Lokesh&Arunkumar.R,. (2013). A clinical study of 30 cases of Acute Yellow Oleander Poisoning. Journal of Current Trends in Clinical Medicine and Laboratory Biochemistry. 1. 29-31
9. Pirasath, Selladurai&Kanagasingam, Arulnithy. (2013). Yellow oleander poisoning in eastern province: An analysis of admission and outcome. Indian journal of medical sciences. 67. 178-83. 10.4103/0019-5359.125879.
10. Eddleston M, Ariaratnam CA, Meyer WP, Perera G, Kularatne AM, Attapattu S, Sheriff MH, Warrell DA. Epidemic of self-poisoning with seeds of the yellow oleander tree (Thevetiaperuviana) in northern Sri Lanka. Trop Med Int Health. 1999 Apr;4(4) 266-273. doi:10.1046/j.1365-3156.1999.00397.x. PMID: 10357862Anandhi, D. &Pandit, Vinay&Kadhiravan, Tamilarasu& R., Soundaravally&Raju, Knj. (2018). Cardiac arrhythmias, electrolyte abnormalities and serum cardiac glycoside concentrations in yellow oleander (Cascabelathevetia) poisoning – a prospective study. Clinical Toxicology. 57. 1-8. 10.1080/15563650.2018.1499930.
11. Gopalakrishnan SK, Kandasamy S, Isaac B, Jayasankar C, Chandru C. Oleander Toxicity–the clinical spectrum and mortality predictors: an observational study. Internet Journal of Medical Update-EJOURNAL.2017May 16;12(1):4