



Rare Poisoning With Yellow Oleander: A Case Report

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ABSTRACT

All parts of the yellow oleander are toxic, but the roots and seeds are more toxic compared to other parts. Yellow oleander plants are more toxic than Nerium oleander. [1] Yellow oleander is an ornamental plant that belongs to the family Apocynaceae and is native to Mediterranean regions of Europe and Asia. It is cultivated in tropical and subtropical areas of the world. Introduction Yellow oleander, with a scientific name “Thevetia peruviana [3],” is known as the “Suicide Tree” because all parts of the tree are poisonous. The usual fatal dose of yellow oleander is 8 to 10 seeds, and the roots are around 15 to 20 grams. The most common manifestations associated with yellow oleander poisoning include bradycardia with AV block, hypertension, lethargy, dizziness, and GI distress. Convulsions, electrolyte disturbances, hypertension, and coma have been reported.

Management includes gastric decontamination, specific antidotes anti-digoxin Fab and Fructose 1, 6 diphosphate administration, cardioversion, and cardiovascular supportive care. [2, 3] We came across a case of suicidal poisoning with pila kaner, an uncommon occurrence even for a major referral hospital such as ours, Rajindra Hospital Patiala (Government Medical College Patiala).

Case Report

A 32-year-old married woman consumed a few flowers of yellow oleander following a quarrel with her husband. She reported to the hospital after 3-4 hours with vomiting, abdominal pain, and diarrhea. She was a healthy-looking lady with a pulse rate of 46 beats/min, regular, and blood pressure of 111/77 mmHg.

She was in significant respiratory distress, and there was some epigastric tenderness. ECG revealed sinus bradycardia with ST segment depression. Other laboratory

parameters, including blood counts, serum electrolytes, blood sugar, and blood urea, were within normal limits. Immediate gastric lavage, continuous ECG monitoring, and symptomatic treatment with intravenous fluids, H2 blockers, and antacids were given. She expired within a few hours of treatment, and the body was shifted to the Rajindra hospital mortuary. On doing the postmortem of the deceased, the following findings were seen in this case: there were no external marks of injury seen on the body of the individual, cyanosed lips as well as peripheral cyanosis of fingers and toes, all organs were congested, gross and histopathology of the heart revealed myocardial edema,

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Fig 1: Showing oleander seeds



Fig 2: Showing Oleander plant

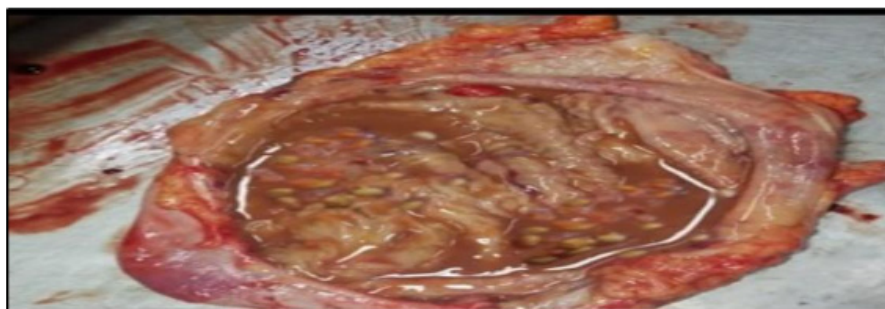


Fig 3: Showing Oleander seeds in stomach

subendocardial hemorrhages, and perivascular hemorrhages which were suggestive of yellow oleander poisoning. The stomach showed yellowish seeds and was congested. The viscera was sent to the chemical examiner Punjab, and subsequently, the cause of death in this case was given as oleander glycoside (NERIN) poisoning, which was found positive in the stomach and its contents and parts of the liver, spleen, both kidneys, and the blood sample sent.

Discussion

The toxicity is caused by “oleandrin” and “digitoxigenin,” which are toxic cardiac glycosides. Although the toxicity symptoms may be quite similar, current literature bears a far higher number of reported cases of yellow oleander poisoning than the number of common oleander cases reported. Electrolyte disturbances are common in oleander poisoning. As the cardiac glycosides inhibit Na^+/K^+ -ATPase, the extracellular potassium concentration increases. Hyperkalemia correlates with serum cardiac glycoside levels and is a marker of serious toxicity with a poor prognosis. Hypokalemia and hypomagnesaemia potentiate cardiac toxicity and should be treated. Multidose activated charcoal decreases the absorption of cardiac glycosides and should be considered in patients presenting early.

Antidigoxin antibody (Fab) cross-reacts with oleander glycosides and should be administered in patients with serious arrhythmias or hyperkalemia. [4]

Although some of these glycosides have therapeutic properties, William Withering, in his classic account from 1795, described treating dropsy with foxglove. Exposure to these plants in toxic amounts may induce cardiotoxic effects as well as gastrointestinal symptoms. The cardiac glycosides in oleander produce more gastrointestinal effects than those in digoxin, and the symptoms range from nausea and vomiting to cramping and bloody diarrhea. In addition, oleander may cause irritation to the mucosal membranes, resulting in burning around the mouth and increased salivation. Confusion, dizziness, drowsiness, weakness, visual disturbances, and mydriasis are central nervous system manifestations of toxicity. [3]

The systemic toxicity mechanism is similar to digitalis toxicity, and patients may suffer from non-specific symptoms such as nausea, vomiting, and weakness. Tremor and ataxia can also be seen, but reported cases usually presented with arrhythmias and acute deterioration. The causes of death are generally cardiogenic toxicity and electrolyte imbalances. Arrhythmias, atrioventricular blocks, and even ST segment elevated myocardial infarction could

be presented. Hypercalcemia is the main electrolyte problem, but magnesium and calcium imbalances should also be considered. [5] Osterloh and associates calculated the lethal oleander leaf dose of their patient at approximately 4 grams. [6]

Conclusion

In conclusion, as oleander is grown throughout the warm climates of India, with large sections of highway medians, practicing physicians should have a thorough understanding of the potentially lethal properties of oleander and its widespread availability across the country. To ensure safety and efficacy, we should promote education and awareness programs for the public, healthcare workers, and practitioners of all medical specialties, as well as engage policymakers. Strengthening regulatory and legal systems is essential to establish and enforce standardized guidelines. Additionally, we need to focus on future directives related to clinical practice, education, training, research, and regulation in this domain.

References

- Pillay VV. Modern medical toxicology. 4th ed. New Delhi: Jaypee Brothers Medical Publishers Ltd.; 2013.
- Rajakpse S. Management of yellow oleander poisoning. Clin Toxicol. 2009;47(3):206-12.
- Eddleston M, Ariaratnam CA, Meyer PW, et al. Self-poisoning with seeds of the yellow oleander tree. Lancet. 1999;354(9189):266-73.
- Bandara V, Weinstein SA, White J, Eddleston M. A review of the natural history, toxicology, diagnosis, and clinical management of *Nerium oleander* (common oleander) and *Thevetia peruviana* (yellow oleander) poisoning. Toxicol. 2010;56(3):273-81. doi: 10.1016/j.toxicol.2010.03.026.
- Khan I, Kant C, Sanwaria A, Meena SR. Acute cardiac toxicity of *Nerium oleander/indicum* (Kaner) poisoning. Heart Views. 2010;11(3):115-6.
- Osterloh J, Herold S, Pond S. Oleander interference in the digoxin radioimmunoassay in a fatal ingestion. JAMA. 1982;247(12):1596-7.