



# ACUTE CARDIAC TOXICITY OF NERIUM OLEANDER/INDICUM POISONING-A CASE REPORT

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## ABSTRACT

Nerium oleander (Apocynaceae), a Mediterranean-native evergreen ornamental plant, is grown globally, especially in mild temperate and subtropical regions. It contains a number of cardiac glycosides at varying amounts that all have the same lethal effect. We report a case of a 33-year-old male who was admitted in the SRM emergency hospital with multiple episodes of vomiting, giddiness and light-headedness, drowsiness and palpitation after ingestion of 10 leaves of Oleander leaves. Electrocardiogram revealed that T wave is reduced in V4-V5 and poor ECHO window. Patient was given one dose of 0.6 mg atropine and thiamine 200mg intravenously and theophylline 2cc intramuscularly. After a day patient bradycardia was settled. After 2 days patient was normalised and get discharged from the hospital.

## INTRODUCTION

Nerium oleander (Apocynaceae), a Mediterranean-native evergreen ornamental plant, is grown globally, especially in mild temperate and subtropical regions. The shrub may be cultivated in conservatories in Central and Western Europe, although it is not frost-tolerant. Since ancient times, people have realised that oleander may be used as a therapeutic substance, as well as a rodenticide and an insecticide [1]. However, the whole plant, including its sap, is poisonous since it contains a number of cardiac glycosides (such as oleandrin, oleandrigenin, desacetyloleandrin, glucosyloleandrin, gentiobiosyloleandrin, nerigoside, odorosides, and oleasides) at varying amounts that all have the same lethal effect [2]. A lethal dose of 5 to 15 leaves of oleandrin is described for adults. Symptoms of oleander poisoning include gastrointestinal symptoms (nausea, vomiting, abdominal pain, diarrhoea), neurological symptoms (tremor, drowsiness, ataxia), and cardiovascular symptoms (sinus bradycardia, atrioventricular (AV) block, fibrillation) if any part of the plant is ingested. It has been stated that treatment concepts include decontamination by stomach lavage and charcoal, correction of electrolyte imbalance and bradycardia, and delivery of digoxin-specific Fab antibodies [3,4]. Oleandrin, with a chemical formula of C<sub>32</sub>H<sub>48</sub>O<sub>9</sub>, has been widely recognised for its potential cardiotoxic effects dating back to ancient times. Cyanogenic glycosides (CGs) are bioactive compounds that are present in both plants and amphibians. They have a broad distribution in the natural world and possess the ability to affect cardiovascular function [5]. These cardiac glycosides exert their effects through the following mechanism: inhibition of the Na-K ATPase enzyme, which leads to an increase in the intracellular concentration of Na<sup>+</sup> and a decrease in the intracellular concentration of K<sup>+</sup>. This has an impact on the Na<sup>+</sup>/Ca<sup>++</sup> exchange channels, leading to an increase in the

concentration of  $\text{Ca}^{++}$  ions inside the cell, which in turn causes a positive inotropic effect due to the higher force of concentration. The suppression of the  $\text{Na}^{+}/\text{K}^{+}$  ATPase pump, on the other hand, has an effect on the flow of potassium ions inside of cells, which may result in hyperkalaemia. In cases of acute poisoning caused by cardiac glycosides, the severity of toxicity is correlated with the degree of hyperkalaemia that has developed. In South Asian nations (Sri Lanka, India), there have been several reports of accidental human exposure to the oleander and purposeful intake of natural formulations for medical reasons. However, the use of oleander simultaneously as a medicinal medicine and as a natural treatment might lead to an improper use of this plant, and it is more likely to generate an increase in the number of instances in which people get poisoned or pass away as a result of their exposure to oleander.

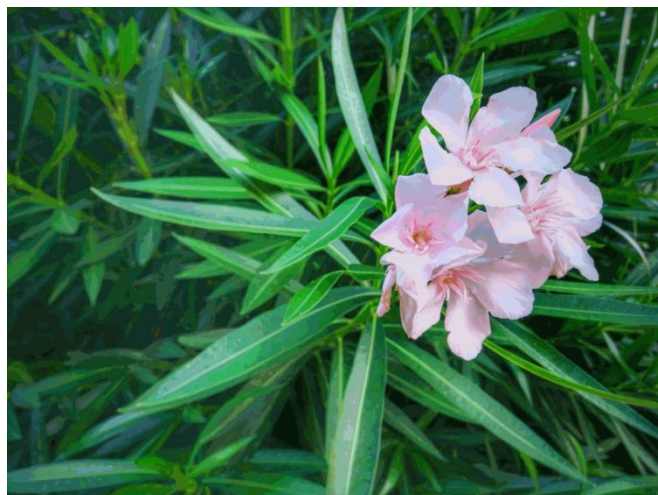


fig1: Oleander leaves.

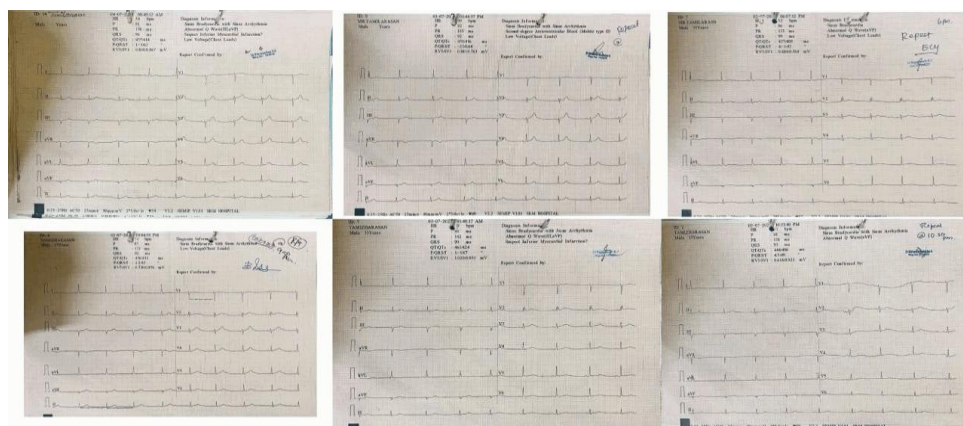
## CASE REPORT

A 33-year-old male was admitted in the SRM emergency hospital with multiple episodes of vomiting, giddiness and light-headedness, drowsiness and palpitation after ingestion of 10 leaves of Oleander. History of similar poisoning attempt of rat poison 3 months back. The patient was alcoholic for past 10 years. No other previous significant history noted. On initial examination, the blood pressure was 120/80 mmHg with irregular pulse rate of 40-46/min. O<sub>2</sub> saturation was normal 98% on room air. He was looking toxic due to excessive vomiting. Other general physical parameters were normal. His chest and lungs were clear to auscultation and percussion. Cardiovascular examination revealed an irregular rhythm. For every 3 hrs ECG was taken for 2 days then after 3 days for 6 times ECG was taken. Electrocardiogram revealed that T wave is reduced in V<sub>4</sub>-V<sub>5</sub> and poor ECHO window. Prolonged PR intervals with varying degree AV blocks and normal QRS duration. Patient was shifted to normal ward, all his routine investigations were sent (cbc, lft, rft, ionic calcium and magnesium were normal). Initially serum potassium level was on higher side (6 meq/l) which was corrected with calcium gluconate and insulin glucose drip. Serum potassium was repeated that was normal subsequently. Gastric lavage was done with activated charcoal. The patient was given one dose of 0.6 mg atropine intravenously and thiamine 200mg intravenously and theophylline 2cc intramuscularly. patient had persistent bradycardia heart rate 40-46 beats/min. Inj atropine 0.6 mg was repeated to this patient multiple times but patient heart rate was persistent 40-46 beat/min, hence patient was started on atropine drip 0.6

mg/hour. After a day patient bradycardia was settled. Atropine drip was stopped. Patient heart rate return to normal after 3 days and ECG was normalised. Till through all the treatment patient heart rate was monitor every single minute. After 2 days patient was normalised and get discharged from the hospital.

## DISCUSSION

Most plants, like foxglove and oleander, have been found to have cardiac glycosides. These are oleandrin, oleandroside, nerioside, digitoxigenin, thevetin, and thevetoxin [6]. Oleander also irritates the nasal membranes, which makes the area around the mouth burn and makes you salivate more. It also has effects on the brain and nervous system, like confusion, dizziness, drowsiness, weakness, vision problems, and mydriasis. The most serious side effects of oleander poisoning are cardiac abnormalities, including various ventricular dysrhythmias, tachyarrhythmias, bradycardia, and heart block. Electrocardiography often reveals an increased PR interval, a decreased QRS-T interval, and T wave flattening or inversion. The empirical basis for the therapy of oleander poisoning is the treatment of digitalis-glycoside toxicity, and it consists of providing the patient with hemodynamic support. Depending on the severity of the bradycardia, this may include the administration of atropine; the use of phenytoin or lidocaine hydrochloride to manage dysrhythmias; the insertion of a temporary venous pacemaker or the administration of electrical counter shock and digoxin-specific Fab antibody fragments [7]. Emesis, another kind of therapy, is used in the hope of facilitating the expulsion of the poisonous chemical from the stomach. Before inducing emesis in a patient who has bradycardia, special attention must be paid to the patient since there is a risk of a vagal response, which might cause the bradycardia to become even more severe. There are a variety of binding agents in the gut, and depending on the kind of glycoside that is there, it is possible that some of the glycoside will be bound. These agents should, in theory, be more successful at absorbing less polar glycosides like digitoxin than they are at absorbing more polar glycosides like digoxin (for example, cholestyramine resin and colestipol). One example of a less polar glycoside is digoxin [8]. Activated charcoal has been shown to be useful in preventing further absorption of the cardiac glycosides by interruption of the enterohepatic circulation of the glycoside so it was introduced in our patient.



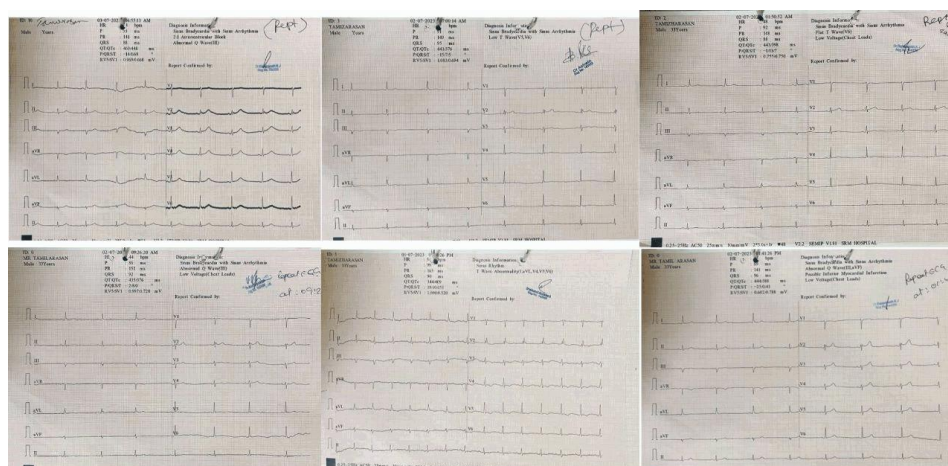


fig2: ECG of the patient.

## CONCLUSION

In prior research, the amount of oleander leaf that was discovered to be fatal on a mathematical scale was roughly 4 grams [9]. These plants are also utilized for medicinal purposes, although whether or not oleander is therapeutically active or dangerous depends on the amount or concentration. Physicians should possess knowledge and provide guidance regarding the lethal properties of oleander, as well as its global availability, in order to raise awareness about the potential dangers associated with oleander leaves.

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