

Oleander poisoning and Management: Case Report

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Date of Submission: 05-12-2020

Date of Acceptance: 20-12-2020

ABSTRACT-oleander poisoning encountered rarely in clinical practice. All parts of the oleander plant contain cardiac glycosides, including the roots, leaves and fruits. The toxic component are the two potent cardiac glycosides, oleanderin and neriine, which can be isolated from all parts of the plant(1). We report a case of oleander poisoning in an adult male presented with multiple episodes of vomiting and palpitation and drowsiness after ingestion of 8 fruits of oleander.He was responded to conventional treatment iv atropine and symptomatic management.

I. INTRODUCTION

Kaner (*Nerium oleander/indicum*) is an ornamental shrub or small, densely branched tree, 1 to 10 m tall in the Dogbane family Apocynaceae. Leaves are in pairs of three or whorled, very green, leathery, narrowly elliptic to linear entire. Flowers grow in clusters in terminal branches, each 2.5 to 5 cm, funnel-shaped with five lobes, fragrant, various colors from pink to red, white, peach, and yellow(2). its botanical name is ThevetiaPeruviana, their seeds contains highly toxic cardiac glycosides thevetinsA,B and nerifoline.

Ingestion of these plants produced clinical picture very similar to that of Digoxine poisoning including Most symptoms from oleander poisoning are cardiac and gastrointestinal in nature including vomiting, diarrhoea, dizziness, sinus bradycardia and atrio ventricular block (AV blocks) and any other arrythmias (3). They cause poisoning by inhibition of sodium-potassium pump of the heart and increased vagal tone producing positive ionotropic and chronotropic effect. (4)

II. CASE REPORT

A 25-year-old male patient was admitted in the emergency with multiple episode of vomiting and lightheadedness, drowsiness and palpitation after ingestion of 8 fruits of common oleander after 10 hours.

The patient was asmokerandalcoholic. No other previous significant history noted. On initial examination, the blood pressure was 122/80 mmHg with irregular pulse rate of 40-46/min. o2 saturation was normal 98% on room air. He was looking toxic due to excessive vomiting. Other general physical parameters were normal. Her chest and lungs were clear to auscultation and percussion. Cardiovascular examination revealed an irregular rhythm with soft S1and normal audible S2 over the cardiac apex.

Electrocardiogram revealed inverted P wave in inferior lead and prolonged PR interval (.28 s), with varying degree AV blocks and normal QRS duration. Patient was shifted to ccu, all his routine investigations were send (cbc, lft, rft, ionic calcium and magnesium were normal). Initially serum potassium level were on higher side (5.7 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 charchol.

The patient was given one dose of 0.6 mg atropine intravenously. That showed no response, 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. Patient digoxine level were send which was 0.30 (not raised). After 1day



patient heart rate responded and bradycardia settles and atropine drip was stopped. Patient heart rate return to normal after 3 days and ecg was normalised. After 3 days patient shifted to ward from ccu and after 2 days discharge from hospital.

III. DISCUSSION

Most of the plants, including foxglove and oleander, have been identified as containing cardiac glycosides and these are oleandrin, oleandroside, nerioside, digitoxigenin, thevetin, and thevetoxin.(4) The cardiac glycosides in oleander produce more gastrointestinal effects than those in digoxine because oleanders are highly irritant to gastrointestinal tract and the symptoms range from nausea and vomiting to cramping and bloody diarrhea [4,5,6]. Oleander also causes irritation to the mucosal membranes, resulting in burning around the mouth and increased salivation. It also causes neurological manifestations like Confusion, drowsiness, weakness, dizziness, visual disturbances and mydriasis [5,7]. The most serious and life threatening effects of oleander poisoning are cardiac abnormalities, including various ventricular dysrhythmias, bradycardia and heart blocks [5]. ECG often reveals an increased PR interval, a decreased QT interval and T wave flattening or inversion. The clinical manifestations are due to increased vagal tone and direct cardiac glycoside toxicity [4,5]. The treatment of oleander poisoning is empirically based on symptomatic management digitalis glycoside toxicity. Initially gastric lavage with activated charchol is effective for absorption prevention. Although, our patient was brought after 10 hours of ingestion of the toxin, but activated charcoal was still used to remove any residual toxin from the stomach. Injection Atropine for severe bradycardia and injection Phenytoin or injection Lignocaine hydrochloride can be used to control dysrhythmias. In severe cases, placing a temporary pacemaker, DC. Shock and administration of digoxin specific (Digibind) Fab antibody fragments is recommended [7]. As this patient was responded to inj atropine and supportive management hence Specific Fab antibodies were not used. Special precaution should be given to a patient with bradycardia before emesis is induced because of the possibility of a vagal stimulation and worsening of bradycardia [4]. How were serum digoxine level were measured in our case that was normal, the blood sample was collected after 10 hour of ingestion, serum digoxine level should not necessarily to corelate with toxicity in many reports have described many asymptomatic patients have toxic level and other patients with significant

toxicity have s digoxine levels are within normal therapeutic range.[8]

IV. CONCLUSION:-

Oleander poisoning can be fatal with relatively small amounts ingested. The calculated lethal oleander leaf dose was found to be approximately 4 gm in previous studies [5]. Its plants are also used for therapeutic purpose, therapeutic activity or poisonous nature of oleander depends upon the dose or concentration. Physicians should understand the potential lethal properties of oleander and its availability throughout the world, especially in India and Sri Lanka (Asian continent). Prompt recognition of the toxic symptoms and its prompt management have successful outcome.

BIBLIOGRAPHY

- Oleander poisoning: treatment with digoxinspecific Fab antibody fragments.Shumaik GM, Wu AW, Ping ACAnnEmerg Med. 1988 Jul; 17(7):732-5.[PubMed] [Ref list]
- [2]. Frohne DP, Fander HJ. A colour Atlass of poisonous plants.London: Wolfe Publishing LTD; 1984. p. 190.
- [3]. Epidemic of self-poisoning with seeds of the yellow oleander tree (Thevetiaperuviana) in northern Sri Lanka. <u>Eddleston</u> <u>M¹, Ariaratnam CA, Meyer WP, Perera</u> <u>G, Kularatne AM, Attapattu S, Sheriff</u> <u>MH, Warrell DA.</u>
- [4]. Diagnosis of oleander poisoning in livestock.<u>Galey FD¹</u>, <u>Holstege DM</u>, <u>Plumlee</u> <u>KH</u>, <u>Tor E</u>, <u>Johnson B</u>, <u>Anderson</u> <u>ML</u>, <u>Blanchard PC</u>, <u>Brown F</u>.
- [5]. Osterloh J, Herold S, Pond S (1982) Oleander interference in the digoxin radioimmunoassay in a fatal ingestion. JAMA 247: 1596-1597.
- [6]. Everist SL (1981) ApocynaceaeIn: Poisonous plants of Australia. Angus and Robertson, London. Pg no 2: 77-89.
- [7]. Shumaik GM, Wu AW, Ping AC (1988) Oleander poisoning: Treatment with digoxine specific fab antibody fragments. Ann emerg med 17: 732-735
- [8]. Bayer MJ. Recognition and management of digitalis toxicity : implications for emergency medicines. Am J Emerg Med 1991: 9:29.

DOI: 10.35629/5252-0211235236 | Impact Factor value 7.429 | ISO 9001: 2008 Certified Journal Page 236