Case Report

Oleander (Kaner) Poisoning

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Abstract

Nerium oleanders are potentially lethal plants after ingestion. Poisoning by these plants is a common toxicological emergency in tropical and subtropical parts of world. All parts of these plants are toxic which contain cardiac glycosides resulting into mostly in arrythmias. We present a case of oleander aqueous root extract poisoning manifested by vomiting,altererd sensorium and severe bradycardia.

Keywords: Bradycardia, Cardiac glycosides, *Nerium oleander*

Introduction

Kaner (*Nerium oleander/indicum*) is an ornamental shrub a small, densly branched tree, 1 to 10 m tall in the Dogbane family **Apocynaceae** genus **Nerium.** Oleander grows well in warm subtropical regions, where it is extensively used as an ornamental plant in landscapes, in parks, and along roadsides. 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.[1]

The common oleander is one of most poisonous plants that have been shown to contain cardiac glycosides. All parts of the oleander plant contains cardiac glycosides, including the roots. . The toxic component are the two potent cardiac glycosides, oleanderin and neriine, which can be isolated from all parts of the plant, Both are very similar to the toxin of Foxglove[4] Both have positive inotropic, negative chronotropic, and cross reactivity. This includes direct glycoside poisoning of the sodium-potassium pump of the heart and increased vagotonia. Most symptoms from oleander poisoning are cardiac and gastrointestinal in nature and appear four hours after the ingestion.

Case Report

A middle aged male was admitted in ICCU with vomiting, loose motions and altered level of consciousness 30 min after ingestion of half glass of oleander aqueous root extract as a folk remedy.

On examination his pulse was 40/min regular, B.P. was 110/70 mm Hg , he was drowsy .respiratory rate was 26/min,spo2 was 90% pupils were normal sized reacting to light .on auscultation of lungs there were bilateral crepitations. Cardiovascular examination was within normal limits except bradycardia Except drowsiness his.Central nervous system examination did not reveal any abnormality. On investigation his blood counts, liver function and renal function tests and serum electrolytes were within normal limit. Serum Digoxin level was 3ng/ml (non-toxic level 0.8-2.4ng/ml) .His ECG showed bradycardia with heart rate 52/min.(Fig1) Subsequently the rate normalised after receiving inj Atropine.(Fig2) Patient also received treatment in the form of gastric lavage and antibiotics. He became fully conscious after 6-8 hours and discharged after 4 days.

Discussion

The oleander is most prevalent, and alluring flowers make it a particular hazard for accidental ingestion[2] The plant also has shown toxicologic importance for accidents when used in folk remedy for variety of conditions including dermatitis, ring worm, abortifacients, tumors; when adults unknowingly eat parts of the plant, or food that has come into contact with the plant, such as in homicides or suicides.

Oleander has been identified as containing cardiac glycosides and these are oleandrin, oleandroside, nerioside, digitoxigenin, thevetin, and thevetoxin.[3] 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. Also, it causes irritation to the mucosal membranes, resulting in burning around the mouth and increased salivation. Confusion, dizziness, drowsiness, weakness, visual disturbances, mydriasis , seizures and coma are central nervous system manifestations of toxicity.

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The most serious side effects of oleander poisoning are cardiac abnormalities, including various ventricular dysrhythmias, tachyarrhythmias, bradycardia, and heart block.[2] Electrocardiography often reveals an increased PR interval, a decreased QRS-T interval, and T wave flattening or inversion. It is thought that these clinical manifestations are the result of both increased vagotonia and direct cardiac glycoside toxicity.

The treatment of oleander poisoning is empirically based on the treatment of digitalis-glycoside toxicity and consists of supporting the patient hemodynamically. This may include induced vomiting, gastric lavage, charcoal to reduce absorption of toxic compounds; administering atropine for severe bradycardia; using phenytoin or lidocaine hydrochloride to control dysrhythmias; placing a temporary venous pacemaker; or electrical counter shock and administering digoxin-specific Fab antibody fragments (Digibind).

Conclusion

It is interesting that oleander poisoning can be fatal with relatively small amounts ingested. So Practicing physicians should understand the potential lethal properties of oleander and its availability throughout the world.

References

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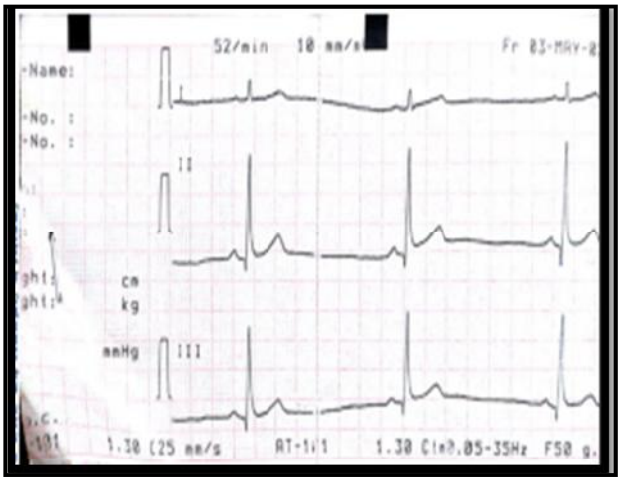

Fig 1 On admission H.R.52/min (Bradycardia)

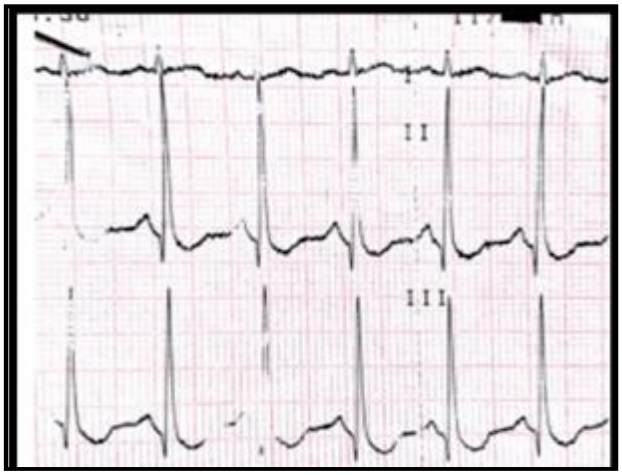

Fig 2 After treatment 0.6 mg inj. Atropin