Case Report

Deliberate Self Harm due to Ingestion of Oleander Seeds Presenting as Cardiac Toxicity

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Abstract

Background : Kaner, an ornamental shrub seen all over the world. All parts of the plant contain cardiac glycosides, so are toxic and present with gastrointestinal and cardiac symptoms. We present the case of a young male presented with dizziness along with nausea and vomiting after 24 hours of self ingestion of oleander seeds.

Key words : Oleander, Kaner, Poisoning, Self Harm, Atropine.

Kaner *(Cascabela thevetia)* is an ornamental small shrub commonly seen all over the world. It is one of the most common poisonous plant containing non digitalis cardiac glycosides ie, neriifolin, thevetins A and B. All parts of the plant contains these glycosides and can be isolated from it.

We report a case of a young male with deliberate ingestion of oleander seeds presenting with gastrointestinal and cardiac symptoms and was managed promptly with good outcome.

Case Report

A 34-year-old male was admitted in emergency with complaints of nausea, vomiting and dizziness 24 hours after self ingestion of 8 seeds of yellow oleander (Cascabela thevetia) commonly called kaner, after having some family dispute. Initial examination revealed vitals as followed, pulse rate 42/min which was irregular with blood pressure 100/60 mmHg. He was having vomiting which caused dehydration and he looked toxic. Rest of the general & physical examination were normal. Irregular rhythm with S1S2 were heard on cardiovascular examination. Inverted P wave was seen in inferior lead in Electrocardiograph and PR interval was prolonged (0.30s) QRS duration normal & with AV blocks (Fig 1). Complete blood count revealed Hb 13.4 gm/dl, leucocyte count 8.6 x 10^9/L, platelet count 224 x 10^9/L. Liver function test revealed mildly elevated enzymes (SGOT 67U/L, SGPT 78U/L, GGT 72U/L, ALP 66U/L). Renal function test revealed no abnormality (urea 32mg/dl, creatinine 1.0 mg/dl, Na 138 mmol/l, K 4.2 mmol/l). He was managed with supportive measures including 0.6 mg intravenous atropine twice a day along with intravenous fluids. He was discharged after 3 days of admission with sinus node dysfunction however asymptomatic (Fig 2).

Editor's Comment :

- Oleander plant is ubiquitous in the environment making it freely available.
- Physicians should be aware of its poisoning to diagnose it early.

Discussion

Cardiac glycosides containing plants include foxglove and oleander having oleandrin, oleandroside, nerioside, digitoxigenin, thevetin and thevetoxin[1]. Oleander ingestion produces more gastrointestinal effects when compared to Digoxin with symptoms including nausea, vomiting, bloody diarrhoea, mucosal irritation. Mydriasis, confusion, weakness, dizziness, drowsiness & vision disturbances are few cranial manifestations[2]. Most serious side effects are ECG abnormalities which include prolonged PR interval, decreased QRS-T interval & flattened or inverted Twave with cardiac abnormality including ventricular dysahythmias, heart block, tachyarrythmias[1,3].

Patients with Oleander poisoning are managed empirically with hemodynamic support. Supportive management includes administration of atropine for severe bradycardia, phenytoin or lidocaine for dysrhythmias or Digoxin-specific Fab antibody fragments (Digibind)[4]. Removal of toxic substance from stomach can be done by emesis however, stimulation of vagus nerve can worsen the bradycardia, so should be observed. Studies have shown that gastric lavage by activated charcoal can prevent further

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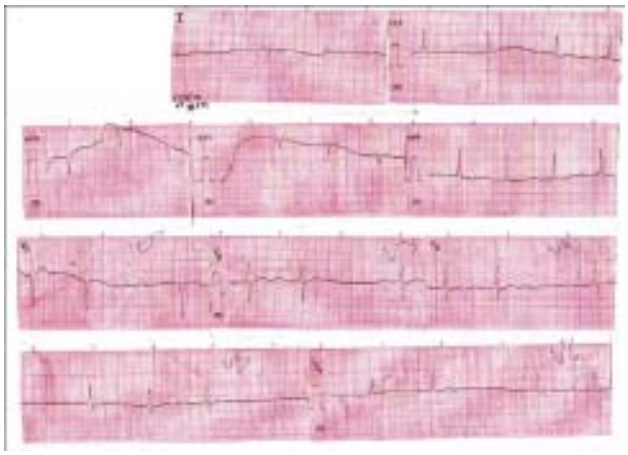

Fig 1 — Electrocardiograph revealed inverted P wave in inferior lead and prolonged PR interval (0.28 s) with

absorption of cardiac glycosides, however our patient presented after 24 hours of ingestion so it was not done.[5]

Conclusion

Ingestion of even a small amount of oleander can be fatal. Lethal dose of oleander leaf was calculated approximately 4 gm by Osterloh and associates[1]. Due to its wide availability throughout the world Physicians should be aware of lethal properties of oleander.

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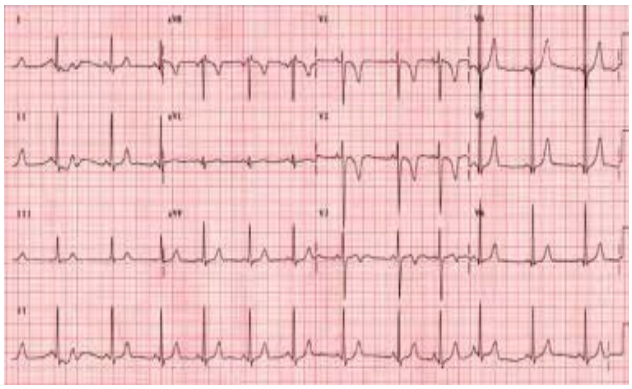

Fig 2 — Electrocardiograph revealing sinus node dysfunction

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