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Case Report

Suicide tree poisoning

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ABSTRACT

Poisoning accounts to 0.33-7.6% paediatric emergency admissions, which are accidental or unintentional. Yellow oleander, an ornamental plant with a scientific name "Thevetia peruviana". All the parts of the tree are poisonous hence, known as "Suicide Tree". Trees are easily accessible and swallowing of seeds which are the most toxic, is the preferred mode of intentional poisoning in South India. Ingestion of one seed is equivalent to 100 digoxin tablets. The phytotoxins of the plant can cause local gastric irritation and severe cardiotoxic manifested as arrhythmias, conduction defects which if left untreated is fatal. Management includes gastric decontamination, specific antidotes anti-digoxin Fab and Fructose 1,6 diphosphate administration, cardioversion and cardiovascular supportive care. Here, authors report an adolescent female presenting with sinus bradycardia following intentional consumption of the yellow oleander seeds.

Keywords: Cardiotoxic, Yellow oleander

INTRODUCTION

Poisoning represents one of the most common medical emergencies. Hospital based data shows that poisoning constitutes 0.33-7.6% of total hospital admissions in children. Unintentional or accidental exposure makes up 80-90% in infants to preschool children, whereas older children and adolescents have higher rates of intentional or suicidal poisonings.[1] India, being an agrarian society, most common poisoning includes pesticides, insecticides, plants, snake bite. Authors report a case of adolescent with intentional consumption of Yellow oleander, which is widely grown in villages and easily accessible, and requires specialized cardiac care at times.

Cascabela thevetia (syn: *Thevetia peruviana*), also known as "suicide tree", is an evergreen tropical shrub belonging to Apocyanaceae family widely grown as an ornamental tree at gardens and at religious sites for offerings. It is named after French monk, Andre Thevet. Vernacular names of the tree are given below.

Table 1: Vernacular names of yellow oleander.

Language	Names
Kannada	Kadukasi, kanagile,Kharjahar
Telugu	Pachaganneru, Kastooripatte
Tamil	Pachaiyalari, Sivappu, Sevvarali, Aatrulari
Malayalam	ManjaArali, Kanaveeram
Marathi	Bitti, Pilvalakanhera, Kanher
Sanskrit	Divyapushpa, Pita-karavira, Ashvaghna
Hindi	Karavira, Kaner
Assamese	Diflee, Sammulhimar
Manipuri	Utonglei
Bengali	Karabi, Karbbe

All the parts of tree like flowers, leaves, fruits are poisonous, but seeds are most toxic. They produce gastric and cardiotoxic effects. In South India and in Sri Lanka swallowing the seeds of Thevetiaperuvianais one of the preferred methods for suicides in villages where they are

grown in abundance. Children have easy access to this plant in gardens or hedgerows. They play with and taste the bright yellow flowers and the conspicuous green fruit accidentally. Case fatality rate is around 10%.[3]

Figure 1: Yellow oleander fruit.

The leaves are spirally arranged, linear and about 13 to 15 cm in length. Flowers are bright yellow and funnel-shaped with 5 petals spirally twisted. The fruits are somewhat globular, slightly fleshy and have a diameter of 4 to 5 cm. Fruits are green in colour and become black on ripening. Each fruit contains a nut which is longitudinally and transversely divided.

CASE REPOET

A 16year old girl was hospitalised with history of multiple episodes of vomiting following consumption of yellow oleander fruit 10 hours prior to admission. On admission child had bradycardia and hyperkalemia. Other systemic examination was normal.

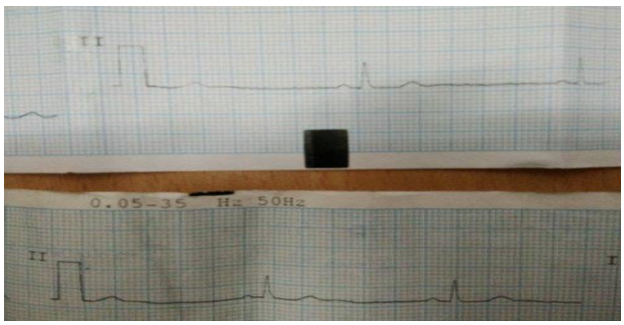

Figure 2: ECG of child.

Gastric lavage was given and supportive treatment in form of potassium free intravenous fluids, gastric acid suppressant was initiated. ECG showed sinus bradycardia. Blood investigations showed anemia, hyperkalemia and normocalcemia.

Child was regularly monitored in PICU with continuous ECG monitor, serum potassium. Child was treated for sinus bradycardia with multiple Atropine boluses (0.1-0.2mg/kg) to maintain normal heart rate. Sinus bradycardia resolved on day 5. No other symptoms during the hospital stay.

DISCUSSION

Mechanism of action

Yellow oleander contains phytotoxins like cardenolides (thevetinA,thevetin B), peruvoside, neriifolin, thevetoxin and ruvoside.[4] Seeds are most dangerous of all parts. Ingestion of an oleander seed is equal to that of swallowing 100 digoxin tablets in one container.[5]These cardenolides are not destroyed by drying or heating. They have negative chronotropic, positive inotropic and cross reactivity. This includes increased vagotonia and inhibition of sodium-potassium ATPase pump. Symptoms of oleander poisoning usually appears after 4 hours following ingestion.

Clinical features

Clinical features of acute yellow oleander poisoning are local irritation of mucous membranes and mouth followed by nausea, vomiting and giddiness, severe diarrhoea, abdominal pain, dilated pupils and occasionally convulsions. Cardiovascular manifestations include sinus bradycardia with sino-atrial-block, first and seconddegree heart block, junctional rhythms, A-V block, atrial and ventricular ectopic beats, and ventricular fibrillation. Hyperkalemia, conduction block and ventricular ectopics indicate serious toxicity. Continuous ECG monitoring for atleast 24 hours is necessary to detect arrhythmias.[6,7] ECG changes include: sinus bradycardia, inversion of T waves, P-R prolongation, A-V dissociation, ventricular tachycardia and ultimately ventricular fibrillation.[8] Conduction block and sinus bradycardia may persist for 5 days after ingestion. Patients usually recover from these if no underlying cardiovascular pathology exist. Fatal period is 6 days.

Diagnosis

Remaining parts of the ingested plant (fruit, flower, branches with leaves) and gastric contents are useful for botanical identification. Biochemical screening by Digoxin immunoassay can be done, if available.[9]

Treatment

Immediate decontamination is done by gastric lavage with multiple dose activated charcoal.Special concern must be given to a patient with bradycardia before emesis is induced because of the possibility of a vagal reaction and worsening of the bradycardia.(10)Bradycardia is treated with atropine infusion(0.6mg/hr) or bolus (0.2-0.6mg/kg) and temporary pacemaker.Treatment of coexisting metabolic abnormalities- hyperkalemia, hypokalemia, hypomagnesemia should be done. Ventricular arrythmiais treated with intravenous lignocaine or phenytoin. Use of intravenous calcium for

treatment of hyperkalemia is contraindicated. Specific antidotes like digoxin specific antibodies and FDP (Fructose 1,6 diphosphate) are usually not available and cost prohibitive. Ventricular fibrillation is treated with cardioversion in absence of anti-digoxin Fab.[5,6,9]

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