A CLINICAL STUDY OF 100 CASES OF ACUTE OLEANDER SEED POISONING IN KANYAKUMARI GOVERNMENT MEDICAL COLLEGE HOSPITAL

Sankar Selvaraj[1], Prince Sreekumar Pius[2], Suresh Kumar[3], Benitta Mary Redleene[4]

[1]Senior Assistant Professor, Department of General Medicine, Kanyakumari Government Medical College Hospital.
[2]Professor, Department of General Medicine, Kanyakumari Government Medical College Hospital.
[3]Postgraduate, Department of General Medicine, Kanyakumari Government Medical College Hospital.
[4]Postgraduate, Department of General Medicine, Kanyakumari Government Medical College Hospital.

ABSTRACT

BACKGROUND

Yellow oleander (Cascabela thevetia) is a poisonous plant that is widely found in India. All parts of the C. thevetia plant are toxic to humans as they contain cardiac glycosides.

MATERIALS AND METHODS

100 patients with alleged history of yellow oleander seed poisoning who came to Kanyakumari Government Medical College Hospital during the period of 2013-16 were enrolled in this study. Patients presenting with multiple poison consumption and those with previous history of heart disease were excluded from the study. A detailed history of the number of seeds consumed, the time of consumption, detailed clinical assessment, routine blood investigations and a 12-lead ECG were recorded. ECG was recorded at the time of admission and every 12th hourly to detect any cardiac arrhythmias.

RESULTS

Most symptomatic patients had conduction defects affecting the SA node, the AV node or both. Patients showing cardiac arrhythmias had significantly higher mean serum potassium concentrations ranging from 4.5-5.2 mEq/L. Yellow oleander seed poisoning is common among young females (56%). There is a poor correlation between the number of seeds ingested and the severity of cardiotoxicity. Arrhythmias has occurred after ingestion of one or two seeds; some patients are asymptomatic even after consuming five or more seeds without requiring specialised treatments. This could be explained on the basis that crushed seeds are more dangerous than whole seeds.

CONCLUSION

Most of these young previously healthy patients had conduction defects affecting the SA or AV nodes. Relatively, few had the atrial tachyarrhythmias or ventricular ectopic beats that are typical of digoxin poisoning. Yellow oleander induced arrhythmias were associated with high serum potassium levels when compared to patient without arrhythmias.

KEYWORDS

Yellow Oleander, Cascabela Thevetia, Cardiac Arrhythmias.

HOW TO CITE THIS ARTICLE: Selvaraj S, Pius PS, Kumar S, et al. A clinical study of 100 cases of acute oleander seed poisoning in Kanyakumari government medical college hospital. J. Evid. Based Med. Healthc. 2016; 3(87), 4726-4728. DOI: 10.18410/jebmh/2016/996

BACKGROUND

The yellow oleander (Thevetia peruviana or Cascabela thevetia) belongs to the family Apocynaceae.[(1)] It has funnel-shaped yellow flowers and green fruits containing one seed. It is found commonly in the tropics and subtropics including Nepal and India. A number of cardenolides have been identified in the bark, leaves, seeds and fruits of yellow oleander of which Thevetin A and Thevetin B are most common. These cardenolides[(2,3)] are structurally similar to those derived from Digitalis purpurea.[(4)]

Financial or Other, Competing Interest: None.
Submission 11-10-2016, Peer Review 12-10-2016,
Acceptance 21-10-2016, Published 28-10-2016.
Corresponding Author:
Dr. P. Suresh Kumar,
Room No 44, CRRI Hostel, Kanyakumari Medical College,
Kanyakumari, Tamil Nadu.
E-mail: suresh_kumardr@yahoo.co.in
DOI: 10.18410/jebmh/2016/996

Ingestion of seeds causes severe problem due to higher content of glycosides than the other parts of the plant. The percentage of glycosides existed in the seeds is 4.80, in the leaf is 0.70, in the latex is 0.045 and in the fruit of yellow oleander is 0.036, respectively. The cardiac glycosides inhibit the transmembrane Na+ /K+ ATPase pump[(2)] and this action produces increased intercellular concentrations of Ca++ and Na+ producing cardiotoxicity.

OBJECTIVES

1. To study the significant dose of poisoning.
2. To study the cardiovascular manifestations of poisoning.
3. To find the electrolyte disturbances associated with oleander seed poisoning.

MATERIALS AND METHODS

Study was performed in Kanyakumari Government Medical College Hospital, Asaripallam, between January 2013 to June

2016 on 100 cases who presented with acute yellow oleander seed poisoning. A detailed history of the number of seeds consumed, time of consumption, time of presentation to hospital and any form of treatment given before coming to hospital were documented. Routine blood investigations were done in all the patients with high attention paid towards serum potassium levels. Patients presenting with multiple poison consumption and those with previous history of heart disease were excluded from the study. All patients received gastric lavage immediately at the time of presentation and gastric decontamination was achieved using activated charcoal and supportive treatment was initiated. Patients were monitored for electrolyte imbalance and a continuous cardiac monitoring was done to recognise cardiac arrhythmias and ECG was recorded on admission and every 12th hourly. Patients developing bradyarrhythmia were managed with atropine and isoprenaline.

DISCUSSION

Acute Yellow Oleander Seed poisoning cause primary GIT and cardiotoxic effects. GIT effects include nausea, vomiting, abdominal colic and diarrhoea. With severe poisoning, vomiting maybe persistent.[5,6] Cardiac arrhythmias such as bradycardia or an irregular pulse are maybe seen. Changes in the ST segment in ECG and conduction blocks are noted with increasing severity. The time course for the progression and resolution of cardiotoxicity is variable. The exact mechanism is not known, but is found to be related to the absorption kinetics from the seed. Some patients were asymptomatic for 24 hours while others had mild toxicity for 72 hours before developing severe toxicity. Up to 40% of patients with severe cardiotoxicity may revert to sinus rhythm after several hours without specific treatment. Cardiac arrest occurs with severe poisoning usually due to VF, which is often resistant to electrical cardioversion. VPCs and tachyarrhythmias are rare. CNS effects are rare and they include weakness, dizziness, confusion or coma. Other effects include hyperkalaemia, renal and hepatic abnormalities. The severity of toxicity is determined by clinical grading of toxicity.[7,8] 1) Asymptomatic - no abnormalities. 2) Mild - predominant GIT symptoms with flattening or inversion of the T wave and depression of the ST segment. Moderate - First-degree heart block, sinus bradycardia, sick sinus syndrome. Severe - Second or third-degree heart block, ventricular fibrillation, death.

Normal ECG	31%
Sinus Bradycardia	27%
First-Degree Heart Block	22%
Second-Degree Heart Block	5%
Complete Heart Block	4%
Ventricular Premature Complexes	3%
Bundle-Branch Block	3%
Atrial Fibrillation	1%
Sinus Arrest/SA Exit Block	2%
Junctional Rhythm	2%

Table 1.1. Various ECG Manifestations in Acute Oleander Seed Poisoning in KGMCH

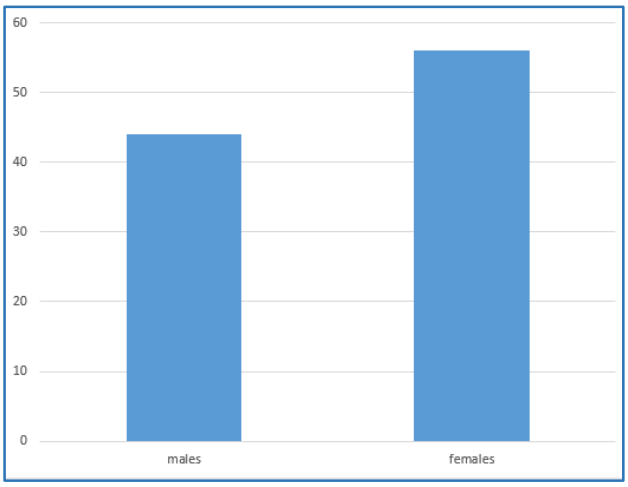

Figure 1.1. Percentage of Males and Females Who Came to KGMCH with Oleander Seed Poisoning During 2013-16

ECG Change	Average Serum Potassium (mEq/L)
Normal ECG	4.2
Sinus Bradycardia	4.5
First-Degree AV Block	4.6
Second-Degree AV Block	4.9
Complete Heart Block	5.2
VPCs	4.9

Table 1.2. Association of Cardiac Arrhythmias With Serum Potassium Level in Yellow Oleander Seed Poisoning

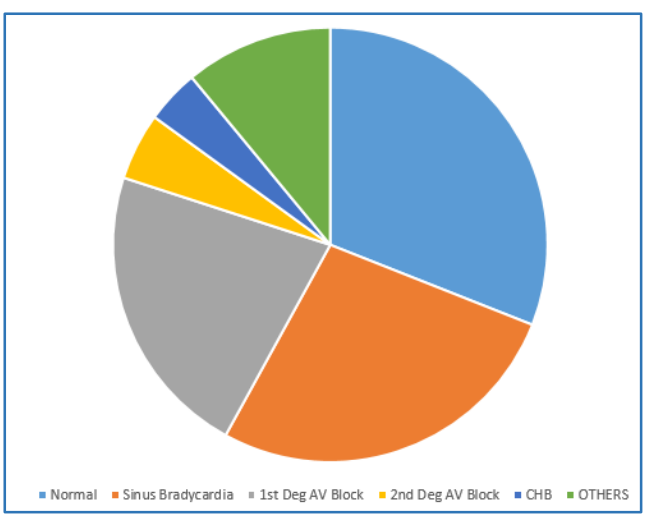

Figure 1.2. Various ECG Changes in Yellow Oleander Seed Poisoning

RESULTS

There was only a poor correlation between the quantity of yellow oleander seed consumption and the severity of cardiac illness. A normal ECG was found in 31% of people with acute yellow oleander seed poisoning. Most of the patients had Sinus Atrial Node and/or Atrioventricular Node block of which sinus bradycardia is the most common arrhythmia (27%) as shown in Table 1.1 and Figure 1.2.

Acute yellow oleander seed poisoning is most common among females (56%) when compared to males as shown

in Figure 1.1. Patients showing cardiac arrhythmias had significantly higher mean serum potassium concentrations ranging from 4.5-5.2 mEq/L as shown in Table 1.2.

CONCLUSION

Most of these young previously healthy patients had conduction defects affecting the SA or AV node. Relatively, very few had the atrial or ventricular tachyarrhythmias or ventricular ectopic beats that are typical of digoxin toxicity.[9] Serious yellow oleander-induced arrhythmias were associated with hyperkalaemia, but no dose dependent toxicity is proven.

REFERENCES

1. Watt MW, Breyer-Brandwijk MG. The medicinal and poisonous plants of southern and eastern Africa. Edinburgh, E&S Livingstone 1962;107-109.
2. Smith TW, Antman EM, Friedman PL. Digitalis glycosides: mechanisms and manifestations of toxicity (parts I-III) Prog Cardiovasc Dis 1984;26(5):413-458.
3. Parikh CK. Parikh's textbook of medical jurisprudence and toxicology. 4th edn. Bombay: Medical Publication 1989:912-914.
4. Hoff man RS. Non-pharmacological cardioactive steroids. J Tox Clin Toxicol 2002;40(3):285-286.
5. Eddleston M, Ariaratnam CA, Meyer WP, et al. Epidemic of self-poisoning with seeds of the yellow oleander tree (Thevetia peruviana) in northern Sri Lanka. Tropical Medicine & International health 1999;4(4):266-273.
6. Eddleston M, Ariaratnam CA, Sjostrom L, et al. Acute yellow oleander (Thevetia peruviana) poisoning: cardiac arrhythmias, electrolyte disturbances, and serum cardiac glycoside concentrations on presentation to hospital. Heart 2000;83(3):301-306.
7. Roberts DM, Wijayaweera K, Eddleston M. Yellow oleander poisoning. Anuradhapura Medical J 2005;4;12-17.
8. Fonseka MM, Seneviratne SL, de Silva CE, et al. Yellow oleander poisoning in Sri Lanka: outcome in a secondary care hospital. Hum Exp Toxicol 2002;21(6):293-295.
9. Langford SD, Boor PJ. Oleander toxicity: an examination of the human and animal toxic exposures. Toxicology 1996;109(1):1-13.