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Original Article

A Study on Incidence, Clinical Profile and Outcome of Cardiac Dysrhythmias in Yellow Oleander Poisoning

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Abstract

Background: Cardiac toxicity after self-poisoning from ingestion of yellow oleander seeds is a common toxicological emergency in South India.

Objectives: The objectives of this study were to identify the various cardiac arrhythmias and electrolyte abnormalities in yellow oleander poisoning. This study was also designed to identify the clinical and biochemical parameters at presentation which can predict serious arrhythmias.

Materials and Methods: This was a observational study among 111 patients who attended our emergency department after consuming yellow oleander seeds. Clinical, biochemical, electrocardiographic, and treatment details were collected from the patients and recorded. Patients were monitored with serial electrocardiograms (ECGs).

Results: Oleander seed poison was most prevalent in the 20–40 years of age. Incidence was more among the young females. ECG abnormalities were found in majority of the individuals. More the crushed seeds consumed and delay to admission to the hospital, poorer was the outcome. All symptomatic patients had conduction defects affecting the sinus node, atrioventricular node, or both. Patients showing cardiac arrhythmias that required specific management had significantly higher serum potassium concentrations. At presentation, 21 patients had serious arrhythmias, and on follow-up, 4 developed new-onset serious arrhythmia. Sinus bradycardia (41.8%) was the most common arrhythmia followed by segment-wave changes (33%). Mortality rate was 1.9%.

Conclusions: The arrhythmias produced by this poisoning might range from sinus bradycardia to complete heart block and ventricular tachycardia. Although serum potassium correlated significantly with serious dysrhythmias, it did not predict the development of new-onset serious arrhythmia. On the whole, serious dysrhythmias were significantly associated with ingestion of crushed seeds, delay in admission, hyperkalemia.

Key words: Cardiac glycoside toxicity, Cardiotoxicity, Yellow oleander poisoning

INTRODUCTION

Oleander is an ornamental tree of the Apocynaceae family that is common throughout the tropics and subtropics. The Apocynaceae is a source of African arrow poisons and also contains many of the most beautiful but deadly tropical flowers such as yellow oleander.[1] Ingestion of oleander seeds or leaves is a common cause of accidental poisoning worldwide, particularly among children.[2] The oleander has been used for suicide, homicide, and abortion and as herbal remedies in India, Thailand, Brazil, and elsewhere.[2] The yellow oleander contains at least eight different cardiac glycosides.[3] All parts of the plant are dangerous, especially the seeds. The active principles are thevetin, thevetoxin, cerberin, and nerifolin. These are all digitalis-like glycosides. These substances act by inhibition of sodium-potassium ATPase pump on cell membranes which, in turn, increases intracellular calcium and inotropic effect.[4] Ingestion of oleander seeds results in a clinical

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picture similar to that of digoxin overdose. The release of excessive calcium results in enhanced cardiac contractions which are delayed after depolarizations and manifest clinically as after contractions, such as premature ventricular contractions. Cardiac glycosides also have vagotonic effects, resulting in bradycardia and heart blocks. Inhibition of Na^+-K^+-ATPase in skeletal muscle results in increased extracellular potassium and contributes to hyperkalemia. Severe hyperkalemia can contribute to atrioventricular (AV) block and depressed myocardial excitability. Dysrhythmias often associated with cardiac glycoside toxicity include bradydysrhythmias, sinus bradycardia with all types of AV nodal block, junctional rhythms, and sinus arrest. Dysrhythmias characterized by increased automaticity and conduction blockade are highly suggestive of cardiac glycoside toxicity. These include tachydysrhythmias such as atrial tachycardia with block, paroxysmal atrial tachycardia with block, bidirectional ventricular tachycardia, and ventricular fibrillation.[4,5]

The objectives of this study were to identify the various cardiac arrhythmias and electrolyte abnormalities in yellow oleander poisoning. This study was also designed to identify clinical and biochemical parameters at presentation which can predict serious arrhythmias.

MATERIALS AND METHODS

Setting

The study was conducted at the Department of Medicine, Tirunelveli Medical College Hospitals.

Design of Study

This was a prospective study.

Period of Study

The study duration was from August 2015 to January 2016.

Sample Size

A total of 111 cases of yellow oleander poisoning that satisfied the inclusion and exclusion criteria were selected.

Inclusion Criteria

1. Those admitted in general medicine wards with a history of yellow oleander ingestion during the period of August 2015–January 2016 were included in the study.

Exclusion Criteria

1. Pediatric cases were excluded (<13 years of age).
2. Those with underlying severe cardiac, renal, or hepatic disease were excluded.
3. Patients who were taking the following drugs – digoxin, diuretics, verapamil, diltiazem, β-blockers, angiotensin-converting enzyme inhibitors, amiodarone, and calcium and potassium supplements were excluded from the study.

Definitions

The definitions adopted during the study period with reference to selected entities are furnished below.

1. Cardiotoxicity: Yellow oleander poisoning cases were divided into no cardiotoxicity, some cardiotoxicity, and severe cardiotoxicity groups based on electrocardiographic changes.
   a. No cardiotoxicity: This group included patients whose electrocardiogram (ECG) showed sinus rhythm or sinus tachycardia only.
   b. Some cardiotoxicity: This group included patients whose ECG showed any one of the following changes.
      1. Sinus bradycardia.
      2. First-degree AV block.
      3. Mobitz Type I second-degree AV block.
      4. Atrial ectopic.
      5. Segment and wave (ST-T) changes that are characteristic of digitalis effect/toxicity.
   c. Severe cardiotoxicity.

This group included patients whose ECG showed any one of the following changes:

1. Sinoatrial (SA) block.
2. Junctional rhythm.
3. Mobitz Type II second-degree AV block.
4. Third-degree AV block.
5. AV dissociation.

Methods

Selected sociodemographic, clinical, biochemical, electrocardiographic, and treatment details were collected from the patients and recorded in a pro forma.

Data regarding poisoning comprised of part ingested, quantity of poison, method of ingestion, whether consumption in empty stomach or after food, the intention behind poisoning, time of ingestion, first aid at home, consumption to admission interval, treatment given, duration of hospital stay, and the type of outcome. Clinical data comprised of symptom analysis, pulse rate, rhythm, blood pressure, and systemic examination.

Blood urea, sugar, serum creatinine, and serum electrolytes values were estimated using ERBA XL300 Automated Analyzer. The blood urea, sugar, creatinine, and serum electrolytes were measured at the time of admission before instituting treatment. 12-lead ECG, including rhythm strip in Leads II and V1, was taken in all patients. 12-lead ECG including rhythm strip was taken at admission before

instituting treatment and repeated depending on the clinical status.

Ethical committee approval obtained from the institutional ethical committee.

Consent

An informed consent was obtained from all patients who were included in the study.

Statistical Analysis

Data were entered in Microsoft Excel spreadsheet and analyzed utilizing the software – Epidemiological Information Package 2002 – developed by the Center for Disease Control and Prevention, Atlanta, for the World Health Organization. Frequencies, percentages, range, mean, standard deviation, and "*P*" values were calculated using this package. Chi-square test was performed to find out the significance of the relationship between the groups. Since the variances were not homogeneous, Kruskul–Wallis (X^2 test) was performed to find out the significance of the difference. The difference was considered to be statistically significant if "*P*" < 0.05.

RESULTS

The incidence of yellow oleander poisoning among a total number of admissions in general medicine wards of Tirunelveli Medical College Hospital, during the study period, was 13.2 per 1000 admissions. It accounted for 91.7% of the cases of plant poisoning. In general, 10% of the cases among the total number of admission in general medicine wards were poisoning cases. Among the poisoning cases, 85.7% were chemical poisoning and 14.3% plant poisoning. Among the total number of poisoning cases, yellow oleander accounted for 13.1% of the cases.

The maximum number of cases occurred in the age group of 20–29 years (50.5%) followed by the age group of 13–19 years (21.6%) and 30–39 years (16.2%), respectively. The age of the patients ranged from 13 to 62 years. The mean age and standard deviation was 27.05 ± 9.76. The mean age of poisoning among males was 27.96 ± 9.6 and that of females 26.31 ± 9.91. The difference in the mean age of males and females was not statistically significant ($P = 0.2458$). Among the total of 111 cases, 50 (45.05%) were males and 61 (54.95%) were females. The ratio of females:males was 1.22:1, but the difference was not statistically significant ($P = 0.56$) [Table 1]. The intention behind poisoning was suicidal in majority of cases (82.9%) and accidental in 1.8%.

The most common electrocardiographic abnormalities were sinus bradycardia (33.6%) and ST-T changes (41.8%) (which were similar to that described for digoxin effect/toxicity). SA block, third-degree AV block, and first-degree AV block were noted in 7 cases each. Mobitz Type II second-degree AV block which is not described in digitalis toxicity occurred in two cases. Depending on the ECG changes, all cases of yellow oleander poisoning were divided into no, some, and severe cardiotoxicity groups (refer definitions in materials and methods). In majority of the cases (61.8%), some form of cardiotoxicity was present. Severe cardiotoxicity was present in about one-fifth of the cases [Table 2].

Majority of the patients had taken either the fruit or seed, 52.8% or 44.6%, respectively. Three patients had taken the outer fleshy covering of the nut alone and these patients had no cardiotoxicity. There was no statistically significant difference in the cardiotoxicity caused by fruit and seed ($P = 0.9582$) [Table 3].

Table 1: Age and sex-wise distribution of study population

Age group	Male	Female	Total	*P* value
13–19	10	14	24 (21.6)	0.246
20–29	25	31	56 (50.5)	
30–39	8	10	18 (16.2)	
40–62	7	6	13 (11.7)	
Total	50 (45.05)	61 (54.95)	111 (100)	
Mean±SD	27.96±9.6	26.31±9.91	27.05±9.76	

SD: Standard deviation

Table 2: ECG changes in yellow oleander poisoning

Cardiotoxicity	ECG changes	cases *n* (%)
No	Sinus rhythm	36 (32.7)
	Sinus tachycardia	6 (5.5)
Some	Sinus bradycardia	46 (41.8)
	Atrial ectopics	1 (0.9)
	ST-T changes (digoxin effect/toxicity)	37 (33.8)
	First-degree AV block	7 (6.4)
	Mobitz Type I second-degree AV block	2 (1.8)
Severe	SA block	7 (6.4)
	Junctional rhythm	3 (2.7)
	Mobitz Type II second-degree AV block	2 (1.8)
	Third-degree AV block	7 (6.4)
	AV dissociation	2 (1.8)

ECGs: Electrocardiograms, ST-T: ST segment and T-wave, AV: Atrioventricular

Table 3: Part ingested and cardiotoxicity

Part ingested	Cardiotoxicity *n* (%)		
	No cardiotoxicity	Some cardiotoxicity	Severe cardiotoxicity
Fruit	20 (48.8)	27 (57.4)	10 (50)
Seed	18 (43.9)	20 (42.6)	10 (50)
Other parts	3 (7.3)	–	–

The mean number of seeds/fruits ingested in no cardiotoxicity group was 2.33 and that in severe cardiotoxicity group was 3.85. The difference was statistically significant ($P = 0.0001$). The range and mean and standard deviation of quantity of poison in each of the groups are depicted in Table 4.

The method of the ingestion of poison was crushed in 50.5% of the cases, chewed in 40.5%, and swallowed in 9%. 76.8% of those who had taken the poison in the crushed form had some form of cardiotoxicity compared to 50% in those who had taken the poison chewed. The difference between the groups was statistically significant ($P = 0.0001$).

The most common symptoms of yellow oleander poisoning were vomiting (73%), numbness of tongue and lips (57.7%), giddiness (56.8%), and diarrhea (27.9%). The relationship between three symptoms, i.e., vomiting, diarrhea, and altered mental status with cardiotoxicity was analyzed. In those patients who did not have any one of the above three symptoms, only 27.3% had features of cardiotoxicity. In those patients who had at least one of the three symptoms, 66.7% had features of cardiotoxicity. Approximately 75% of the patients who had at least two symptoms or all the three symptoms had features of cardiotoxicity. The difference between the groups was statistically significant ($P = 0.0005$). The details are shown in Table 5.

Serum potassium levels were measured at time of admission in 97 of 111 cases. Of these, only two cases had hyperkalemia and 7 cases had hypokalemia, and in the majority (90.7%), serum potassium values were within normal limits. The mean serum potassium value in no, some, and severe cardiotoxicity groups was 3.85, 4.09, and 4.16 meq/l, respectively. The difference between the groups was statistically significant ($P = 0.0201$).

Gastric lavage was given in 90.9% of the cases. In those who were given gastric lavage, 39.0% did not develop any cardiotoxicity compared to 33.3% in those who were not given gastric lavage. The difference between the groups was statistically insignificant ($P = 0.5675$).

The mean duration of hospital stay was 4.55 days, range being 1–9 days. The mean duration in no, some, and severe cardiotoxicity groups was 3.64, 4.96, and 5.65 days, respectively. The difference between the groups was statistically significant ($P = 0.0001$).

Of 111 cases, 86.4% of the cases were discharged well. Death occurred in two cases (1.9%, one male and female patient).

Table 4: Number of seeds ingested and cardiotoxicity

Cardiotoxicity	Range	Mean±SD
No cardiotoxicity	1–5	2.33±1.65
Some cardiotoxicity	1–10	3.47±1.54
Severe cardiotoxicity	1–18	3.85±1.93
Total	1–18	3.26±2.27

SD: Standard deviation

DISCUSSION

Yellow oleander poisoning is a common method of deliberate self-harm among young adults in Sri Lanka and Southern India. This study was done to find out the burden of this poisoning among admissions in general medicine wards of Tirunelveli Medical College Hospital, Tirunelveli, to correlate clinical and biochemical parameters with cardiotoxicity and to identify the possible risk factors for cardiotoxicity and outcome. The incidence of yellow oleander poisoning among a total number of admissions in general medicine wards of our hospital during the study period was 13.2 per 1000 admissions.

Among the 111 cases studied, the mean age of patients was 27.05 ± 9.76 years, range being 13–62 years (pediatric cases not included). 71.7% of the cases occurred in the age group between 13 and 29 years. This observation confirms the observation of previous Indian and Sri Lankan studies that yellow oleander poisoning was found commonly among adolescents and young adults. Eddleston *et al.*, in a study of 415 cases in Sri Lanka, observed that the patients were

Table 5: Symptoms and Cardiotoxicity

Presence of symptoms	Cardiotoxicity *n* (%)			Total *n* (%)
	No cardiotoxicity	Some cardiotoxicity	Severe cardiotoxicity	
Vomiting (80)	24 (30.00)	39 (48.75)	17 (21.25)	80 (100)
Diarrhea (31)	7 (22.58)	15 (48.38)	9 (30.03)	31 (100)
Altered mental status (18)	5 (27.78)	9 (50.00)	4 (22.22)	18 (100)
None of the above three symptoms	16 (72.70)	4 (18.20)	2 (9.10)	22 (100)
At least one symptom	17 (33.30)	26 (51.00)	8 (15.70)	51 (100)
At least two symptoms	8 (24.25)	17 (51.50)	8 (24.25)	33 (100)
All the three symptoms	1 (25.00)	1 (25.00)	2 (50.00)	4 (100)

young (mean age 25.8 years and range 11–-71). In that study, more than 50% of women and 35% of men were under 21 years.[3].

Regarding the sex distribution, 54.95% of the cases were females and 45.05% of cases were males, the ratio being 1.22:1. In the present study, there was only a slight female preponderance when compared to findings of previous Indian and Sri Lankan studies. Eddleston *et al.* observed a female:male ratio of 1.6:1 in his study involving 415 patients in Sri Lanka 4. In general, this poisoning was found to be more common among females when compared to males.[3]

The intention behind the poisoning was suicidal in 82.9% of the cases. The reasons included interpersonal conflict, unemployment, failure to achieve goal, situational reaction, grief reaction, and physical illness. There were two cases of accidental poisoning among adolescents. There were no cases of homicidal poisoning probably due to the bitter taste of the poison. The observations regarding the intention behind poisoning were similar to that observed by Mandal *et al.*[4]

The electrocardiographic changes that were noted in this study were mainly due to depressed conduction. Most common abnormalities were sinus bradycardia, ST-T changes suggestive of digoxin effect/toxicity, first-degree AV block, third-degree AV block, and SA block. Others included second-degree AV block, junctional rhythm, AV dissociation, and atrial ectopics. The observations were similar to that of Eddleston *et al.* except that tachyarrhythmias (0.5–1%) such as atrial flutter, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation observed by Eddleston *et al.* which were not observed in the present study. In the same study, 3–6% had supraventricular tachycardia and 2% had ventricular ectopics which were not observed in the present study. Mobitz Type II second-degree AV block which is not described in digoxin toxicity occurred in two cases.[6] In yellow oleander poisoning, arrhythmias due to depressed conduction were more common than tachyarrhythmias and thus differ from digoxin poisoning, in which tachyarrhythmias were found to be more common.

Majority (61.8%) of cases showed some form of cardiotoxicity. Severe cardiotoxicity was present in approximately 20% of the cases. The common method of poisoning in the present study was ingestion of fruits or seeds in the crushed or chewed form. Toxicological studies in albino rats have shown that all parts of the plant were poisonous, especially the seeds/kernels of fruit.[5] Other parts such as roots, leaves, or flowers were not taken. The mean number of seeds/fruits ingested in severe (3.85) and some cardiotoxicity groups was 3.47 higher than that in no cardiotoxicity group (2.33). The range in no, some, and severe cardiotoxicity groups was 1–5, 1–10, and 1–18 (seeds/fruits), respectively. Although there was a positive correlation between the quantity of poison and cardiotoxicity in the present study, it should be noted that even one seed/fruit was found to cause some or severe cardiotoxicity. The relationship between quantity of poison and outcome was not present as only two deaths had occurred and patients who had taken six and ten seeds had all survived. Two patients who died had consumed 5 and 18 seeds, respectively. Eddleston *et al.* also observed no relationship with seeds ingested and outcome. Hence, the quantity of poison alone cannot determine outcome, and it may be influenced by other factors such as method of ingestion, consumption in empty stomach or not, delay in gastric lavage, and treatment given. There was a higher incidence of cardiotoxicity in those who had taken the seeds/fruits crushed compared to those who had chewed or swallowed the poison. This is probably because more amount of cardiac glycoside is available to be absorbed once the seeds/fruits are crushed. The method of ingestion observed in the present study was similar to that reported in a study from Eastern India, in which majority (97.33%) of the patients had ingested the poison in the crushed form.[7]

In 42.3% of cases, first aid was given at home after the ingestion of poison. The commonly used method was to induce vomiting using soap water, tamarind water, salt water, etc. There was no statistically significant difference in cardiotoxicity between patients who were given first aid and those who were not given first aid. This is because, in many patients, there was a delay in giving first aid.

The mean delay in getting admitted to Tirunelveli Medical College Hospital, Tirunelveli, after consumption of poison was 12.72 ± 4.6 h, the range being 1–62 h. A correlation between delay in getting admitted to our hospital and cardiotoxicity could not be obtained probably because many patients were given first aid and treated outside in other hospitals/institutions before being referred to our hospital. Bose *et al.* observed a delay of 6–8 h.[7] In patients with high suicidal intent, the relatives come to know of the poisoning only after several hours and this might have contributed to the delay. The most common symptoms of yellow oleander poisoning in the present study were vomiting (73%), numbness of tongue and lips (57.7%), giddiness (56.8%), and diarrhea (27.9%). Patients who had vomiting, diarrhea, or altered mental status had a higher incidence of cardiotoxicity compared to those who did not have any one of the above symptoms. Among patients who had at least two or all the three of above symptoms, 75% developed cardiotoxicity. Thus, patients with either vomiting, diarrhea, or altered mental status should be closely monitored for cardiotoxicity. Ellenhorn

and Barceloux have also noted that, in severe poisoning, diarrhea and vomiting are early features.[8] Many patients had a normal pulse rate and rhythm at admission only to develop the features of cardiotoxicity later usually within a day.

Hyperkalemia occurs in severe yellow oleander poisoning. Severe hyperkalemia can contribute to AV block and depressed myocardial excitability. In the present study, hyperkalemia was noted in only two of 97 cases (2.1%). Both of these patients had some cardiotoxicity. However, there was a correlation between serum potassium levels and cardiotoxicity in the present study. The mean serum potassium values were higher in some (4.09 meq/l) and severe cardiotoxicity groups (4.16 meq/l) compared to patients with no cardiotoxicity (3.85 meq/l) [Table 6]. Eddleston *et al.* noted hyperkalemia in 38 patients of 118 cases (32.2%). Very high values of potassium such as 7.2 meq/l, 8.4 meq/l, and 10.8 meq/l were observed in that study.[3]

Hypokalemia was noted in 7 of our cases. Eddleston *et al.*, noted hypokalemia in 9 of 118 cases. Hypokalemia may be probably due to persistent vomiting due to poisoning *per se* or due to induced emesis. Hypokalemia can exacerbate cardiac glycoside toxicity as if facilitates enhanced binding of cardiac glycosides to Na^+-K^+ ATPase pump. Both hyperkalemia and hypokalemia are dangerous in yellow oleander poisoning, and serial monitoring of potassium levels and adequate treatment is necessary.[9]

The mean duration of hospital stay in the present study was 4.55 days, range being 1–9 days. Those patients with some and severe cardiotoxicity had increased duration of hospital stay when compared to patients with no cardiotoxicity. This was similar to findings observed by Bose *et al.* who observed a median hospital stay of 5 days.[7]

Death occurred in two cases (one male and female patient) within 1 h after admission. The male patient had AV dissociation while the female patient died before ECG could be taken. The case fatality rate was 1.9%. Bose *et al.* observed a case fatality rate of 4.6% among 300 patients in Eastern India.[7] In Sri Lankan studies, Eddleston *et al.* observed a case fatality rate of approximately 10%. The lower case fatality rate in the present study may be due to less severe poisoning in Tamil Nadu when compared to that in Sri Lanka and probably due to lesser number of patients studied. As the deaths were very few, there was no statistically significant difference in outcome among no, some, and severe cardiotoxicity groups and also there was no statistically significant difference in outcome among male and female patients.

Table 6: Serum potassium levels and cardiotoxicity

Cardiotoxicity	Serum potassium values (meq/l)	
	Range	Mean±SD
No cardiotoxicity	3–4.60	3.85±0.38
Some cardiotoxicity	3.4–5.6	4.09±0.43
Severe cardiotoxicity	3.50–4.90	4.16±0.4
Total	3–5.60	4.01±0.42

SD: Standard deviation

CONCLUSIONS

1. The incidence of yellow oleander poisoning in general medicine wards of Tirunelveli Medical College Hospital, Madurai, during the study period was 13.2 per 1000 admissions.
2. Yellow oleander poisoning was most commonly observed among young adults and adolescents.
3. Although there was a slight female preponderance, the difference was not statistically significant.
4. The most common symptoms of yellow oleander poisoning in the present study were vomiting, numbness of tongue and lips, giddiness, and diarrhea.
5. Electrocardiographic changes that were noted were mainly due to depressed conduction. Most common abnormalities were sinus bradycardia, ST-T changes similar to digoxin effect/toxicity, first-degree AV block, third-degree AV block, and SA block.
6. Hyperkalemia as a manifestation of yellow oleander poisoning was uncommon in the present study compared to Sri Lankan studies.
7. There was a higher incidence of cardiotoxicity as the quantity of poison increased but even ingestion of one seed/fruit was found to cause severe cardiotoxicity.
8. There was a higher incidence of cardiotoxicity in those who had taken the seeds/fruits crushed when compared to those who had chewed or swallowed the poison.
9. The mean serum potassium values at presentation were higher in patients who had cardiotoxicity when compared to patients who had no cardiotoxicity.

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