IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 16, Issue 9 Ver. X (September. 2017), PP 64-71
www.iosrjournals.org

Yellow Oleander Seed Poisoning – A Profile

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Abstract:
Aim: *The aim of this study was to estimate the incidence of yellow oleander seed poisoning,toanalyse theclinical and biochemicalaspects, tocorrelate the clinical andbiochemicaldata with electrocardiographicchanges and cardiotoxicity and toidentifythe possible risk factors for cardio toxicityand outcome following treatment.*
Methodology: *Cases of yellow oleander seed ingestion presenting to the Poison control Center, were included in the study. Selected sociodemo graphic, clinical, biochemical, electrocardio graphic and treatment detailswere collected from the patientsand recorded in a proforma.*
Results: *In a study of 50 patients, the clinical, biochemical and electrocardiographic profle of the patients with a history of ingestion of yellow oleander seed were studied and analysed.*
Conclusion: *Yellow oleander poisoning was most commonly observed among young adults and adolescents. Although there was a slight female preponderance the difference was not statistically significant.In majority of cases, the intention was suicidal.Electrocardiographic changes noted in the present study were mainly due to depressed conduction.Hyperkalemia as a manifestation of yellow oleander poisoning was uncommon in the present study compared to Srilankanstudies.There was a positive correlation between incidence of cardiotoxicity as the quantity of seeds ingested.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.*
Keywords: *Yellow oleander, Hyperkalemia, cardiotoxicity.*

Date of Submission: 03-09-2017 Date of acceptance: 14-09-2017

I. Introduction

Deaths occur due to various reasons, often preventable and curable. Suicidal deaths remain the common mode of preventable and unnaturaldeath which is widely prevalent both in developed and developing countries. The exact number of deaths and mode of suicide is grossly under reported in developing countries. The yellow oleander, an evergreen shrub or a small tree of dogbane family, Apocyananceae family is widely distributed over the tropics.Yellow oleander (Thevetiaperuviana), is one of the most poisonous plants which contains numerous toxins .The toxic effects resemble that of digitalis toxicity. The pathophysiology of cardiotoxicity is direct inhibition of Na+-K+-ATPase pump of the heart and increased vagal tone.The use of seeds or fruits of yellow oleander as a method of self harm is common in Sri lanka and South India. There has been a sudden increase of the number of cases of yellow oleander poisoning in Srilanka with thousands of suicidal attempts occurring every year[9.] Most poisonings have clear guidelines for the effective treatment and its complications. Though the chemical nature of yellow oleander is clearly described, the guidelines for its management has not been defined adequately and requires a larger study.The only Indian study is from Eastern India. (300 cases,Bose T K et al,1999). Hyperkalemia which was noted in about 30% of the cases of yellow oleander poisoning in the Srilankan study was not been described in Indian studies involving 300 patients[24.] This study was done to find out the incidence of yellow oleander poisoning among admissions in Toxicology unit of a tertiary care hospital,to study the clinical and biochemical aspects, and to find correlation between clinical, biochemical parameters and electrocardiographic changes of yellow oleander poisoning and to assess the risk factors for cardiotoxicity and the outcome and to compare the results of our observation with previous published reports.

II. Materials And Methods

We conducted an observational study for a period of 6 months in Institute of Internal Medicine, Department of Toxicology,Rajiv Gandhi Government General Hospital,Chennai. 50 cases of yellow ole and erpoisoning cases were included in the study. Paediatriccases, thosewith underlying severe cardiac, renal or hepatic disease and patientswho were taking the followingdrugs– Digoxin, Diuretics,Verapamil,

diltiazem, Beta blockers, ACE inhibitors, Amiodarone, calciumand potassium supplementswere excluded. Selectedsociodemographic, clinical, biochemical, electrocardio graphic and treatment detailswere collectedfrom the patients and recorded in a proforma. Dataregarding poisoning comprised of partingested, quantityofpoison, method of ingestion, whether consumptionin empty stomachor after food, time of ingestion, first aid at home, consumption to admission interval, treatment given, duration of hospital stayand the type of outcome. Symptomanalysis, pulserate, rhythm, blood pressure and systemicexamination were done. Bloodurea, sugar, serumcreatinine andserumNa^+,K^+,valueswereestimated using ERBAXL 300automatedanalyzer.The bloodurea,sugar,creatinineandserum Na^+,K^+,were measured atthe time of admission before instituting treatment. 12leadECG includingrhythm strip was takenatadmission before institutingtreatmentandrepeated depending on the clinical status.

III. Statistical Analysis

Datawereenteredin Microsoft Excel spreadsheet and analyzed utilizingthe software –Epidemiological Information Package 2002 (EpiInfo2002)– developedby the Centre for Disease Control and Prevention, Atlantafor World Health Organization, Frequencies, percentages, range, mean, standard deviation and 'p' values were calculated usingthispackage.ChiSquare testwas done to findoutthe significance of relationship between the groups. SPSS software was used for analysis. The difference was considered to be significantif the 'p'value was less than 0.05.

IV. Results

In our study, out of 50 patients, 6 patients (12%) were <20 years,27 patients (54%) were in the age group of 21-30 years, 12 patients (24%) were in the age group of 31-40 years, and 5 patients (10%) were above 40 years of age (TABLE: 1 & FIGURE: 1). 22 (44%) were males, and 28 (56%) were females. Male to female ratio was 1:1.17 (FIGURE: 2). Incidence appears more in females than males and also the common age group in females was 21 to 30 years.Most of the patients presented with vomiting (80%), giddiness (68.6 %), Palpitation (48.6%), abdominal pain (42.9%), breathlessness (34.3%). Only 2 patients who were very sick on admission presented with altered sensorium (5.7%) (TABLE: 4).Most of the patients had consumed 3-5 seeds (64%). Mortality was seen in patients who have taken more than 8 seeds (TABLE: 3).There appears to be a direct relationship between the no. of seeds, ECG abnormality and mortality.In any patient consuming 5 seeds or more, there was a definitive ECG abnormality (FIGURE: 3).In our study, most of the patients had consumed 3-5 seeds (64%). Mortality was seen in patients who have taken more than 8 seeds (FIGURE 4).Most of the patients in our study presented with sinus bradycardia 25(50%). 11 of patients (22%) presented with 1°AV block. 2 patients(4%) presented with 2°AV block. ST changes were seen in 2 patients (4%). 2 patients (4%) who presented late presented with CHB (TABLE: 5).Seeds were taken in many forms. Most common form of consumption was grounded form (64%) (TABLE: 7).No. of patients admitted within 3 hours of poisoning were 22 (44%), between 3-6 hours were 19 (38%) and after 6 hours were 9 (18%) (TABLE: 8).Mortality in male is 4.54 % and in female is 3.57 %.Overall mortality in the study was 2 (4%) - 23 years old female and a 45 year old male who presented late with CHB. High mortality could be due to delay in admission and consumption of more no. of seeds (TABLE: 9).Most patients recovered on symptomatic treatment. 26 patients (74%) recovered with atropine alone. 7 patients (20%) required both atropine and orciprenaline (TABLE 10).

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V. Discussion

Yellow oleander poisoning is a common method of deliberate self-harm among young adults in Srilanka and southern India. This study was done to find out the burden of this poisoning among admissions in toxicology unit of Rajiv Gandhi Govt General hospital Chennai to analyse the clinical aspects, to correlate clinical and biochemical parameters with cardiotoxicity and to identify the possible risk factors for cardiotoxicity and outcome. Among the total number of poisoning cases, yellow oleander accounted for 12.7% of the cases.Among the 50 cases studied, the mean age of patients was around 25 years, range being 18-56 years (pediatric cases not included). 62% of the cases occurred in the age group between 18-29 years. This observation confirms the observation of previous Indian and Srilankan studies that yellow oleander poisoning was found commonly among adolescents and young adults.Regarding the sex distribution 56% of the cases were females and 44% cases were males, the ratio being 1.17:1. In the present study there was only a slight female preponderance when compared to findings of previous Indian and Srilankan studies. The intention behind the poisoning was suicidal in more than 80% of the cases. The reasons included interpersonal conflict, unemployment, failure to achieve goal, situational reaction, grief reaction, physical illness etc. Twopatients had underlying psychiatric illness and were on antipsychotic drugs. In 24% of the patients, poisoning attempt was done just to frighten others for some personal gain or to resolve conflicts. 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 byEddlestonM et al.[4]

Regarding underlying illness, except for four patients which includes two cases of psychiatric illness, one case of diabetes mellitus,one case of hypertension, none had any previous illness. Both of these patients were on irregular treatment. This was similar to the findings observed by Eddleston M et al, who noted that only one patient had underlying illness (rheumatic heart disease)[8]. This observation differed markedly from patients with digoxin poisoning. Many of the patients with digoxin poisoning had underlying heart disease and were on multiple drugs.[27]This observation is important because of the young age and previously healthy state.

The electrocardiographic changes that were noted in this study was 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 sino-atrial block. Others included second-degree AV block, junctional rhythm, AV dissociation and atrial ectopics. The observations were similar to that of Eddleston M et al except that tachyarrhythmias (0.5-1%) like atrial flutter, atrial fibrillation, ventricular tachycardia and ventricular fibrillation observed by Eddleston M et al were not observed in the present study. In the same study 3-6% had supraventricular tachycardia and 2% had ventricular ectopics which was not observed in the present study.[8]Mobitz type – II second-degree AV block which is not described in digoxin toxicity occurred in two cases.[28] In yellow oleander poisoning arrhythmias due to depressed conduction were more common than tachy-arrhythmias and thus differs from digoxin poisoning in which tachy-arrhythmias were found to be more common.[27]

The common method of poisoning in the present study was ingestion of fruits or seeds in the crushed or chewed form. Two patients had taken the outer fleshycovering of the nut and these patients had no cardiotoxicity. Toxicological studies in albino rats have shown that all parts of the plant were poisonous especially the seeds / kernels of fruit[16.] Other parts like roots, leaves or flowers were not taken. Most of the patients in our study who had presented with cardiotoxicity had ingested about 3-5 seeds. Those who had taken more than 5 seeds had presented with severe cardiotoxicity.Although there was a positive correlation between number of seeds and cardiotoxicity in the present study it should be noted that even one seed / fruit was found to cause some or severe cardiotoxicity. Two patients who died had consumed more than 8 seeds. Majority had taken the seeds / fruits in the crushed form. 64% of those who had taken the poison in the crushed form had severe cardiotoxicity compared to 6% in those who had taken the poison chewed, which shows a lower 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 due to the fact that more amount of cardiac glycoside is available to be absorbed once the seeds/fruits are crushed and one patient developed severe cardiotoxicity. This finding is supported by the fact that in Srilankapeople usually eat the seeds whole and they develop cardiotoxicity. So even the seeds taken as a whole can cause cardiotoxicity but to a lesser extent when compared to crushed or chewed form. 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]

Most of the patients had consumed the poison in empty stomach. There was a higher incidence of cardiotoxicity in patients who had consumed the poison in empty stomach than after food. This is probably due to the fact that absorption of cardiac glycoside is better in empty stomach. In previous studies this relationship was not assessed.In 78% 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.In patients who were given first aid, delay in giving first aid was associated with increased incidence of cardiotoxicity. So bringing out the poison early before the poison has passed into intestine reduces the incidence of cardiotoxicity in yellow oleander poisoning. A correlation between delay in getting admitted to our hospital and cardiotoxicity could not be obtained probably due to the fact that many patients were given first aid and treated outside in other hospitals/institutions before being referred to our hospital. Bose TK et al (1999) observed a delay of 6-8 hrs.[7]. The reasons for the delay in getting to our hospital in the present study would be treatment of patients outside by others doctors and lack of adequate transportation in interior rural areas. In patients with high suicidal intent the relatives come to know of the poisoning only after several hours and this night have contributed to the delay.

The most common symptoms of yellow oleander poisoning in the present study were vomiting (80%), giddiness (68.6%) and diarrhea (37.1%). This was similar to findings noted by Saravanapavanathan N and Ganeshmoorthy J (1986) who observed vomiting, giddiness and diarrhea as the most common symptoms in their study of 170 cases in Srilanka.[2]. Patients who had vomiting, diarrhea or altered mental status had 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 vomiting, diarrhea or altered mental status should be closely monitored for cardiotoxicity.EllenhornandBarceloux (1988) have also noted that in severe poisoning diarrhea and vomiting are

early features[20.] The average pulse rate at admission was 71±18 per minute, range being 38-116 per minute. Many patients had a normal pulse rate and rhythm at admission only to develop features of cardiotoxicity later usually within a day.Eddleston M et al (1999) have described a patient who remained in sinus rhythm for three days before developing second degree AV block.[4]So patients may have to be observed for 3-4 days after ingestion of poison before being discharged home.

Hyperkalemia occurs in severe yellow oleander poisoning[8]. Severe hyperkalemia can contribute to atrioventricular (AV) block and depressed myocardial excitability19. In the present study hyperkalemia was noted in only two of 50 cases (4%) in whom the serum potassium levels were measured. Both of these patients had some cardiotoxicity. But there was no correlation between serum potassium levels and cardiotoxicity in the present study. The mean serum potassium values was 3.9meq/l.Eddleston M et al (2000) noted hyperkalemia in 38 patients out of 118 cases (32.2%). Very high values of potassium like 7.2meq/l, 8.4 meq/l and 10.8 meq/l were observed in that study.[8] In the present study the two patients who had hyperkalemia had values of 5.7 and 5.2 meq/l. The reason why hyperkalemia was not as common when compared to Srilankan study would be that poisoning might have been less severe or due to the persistent vomiting due to poisoning per se or due to induced emesis. Hypokalemia was noted in 3 of our cases. Eddleston M et al, (2000) noted hypokalemia in 9 out of 118 cases[2]. Hypokalemia can exacerbate cardiac glycoside toxicity as if facilitates enhanced binding of cardiac glycosides to Na+ -K+ ATPase pump[22]. Both hyperkalemia and hypokalemia are dangerous in yellow oleander poisoning and serial monitoring of potassium levels and adequate treatment is necessary. Severe hyperkalemia may require treatment with antidigoxin Fab fragments[18]

There was no statistically significant difference in cardiotoxicity between patients who were given gastric lavage and those who were not given gastric lavage. Gastric lavage is most useful when started within 60 minutes after ingestion[24]. Early gastric lavage is more important than whether gastric lavage is given or not in reducing the incidence of cardiotoxicity. Most of the patients were treated with supportive measures like gastric lavage, injection atropine, tabletorciprenaline. Antidigoxin Fab fragments were not used due to economical and technical constraints.

The mean duration of hospital stay in the present study was 5 days, range being 2-9 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 Boss TK et al (1999) who observed a median hospital stay of 5 days.[7] Death occurred in two cases (one male and female patient) within one hour after admission. Both patients had atrioventricular dissociation and presented to the toxicology unit very late after ingestion of more than 8 seeds with severe hypotension and died even before temporary pacing could be arranged. The case fatality rate was 4%. Bose TK et al (1999) observed a case fatality rate of 4.6% among 300 patients in eastern India [7]. In Srilankan studies, Eddleston M et al observed a case fatality rate of approximately 10%[4]. The lower case fatality rate in the present study may be due to less severe poisoning in Tamilnadu when compared to that in Srilanka and probably due to lesser number of patients studied.

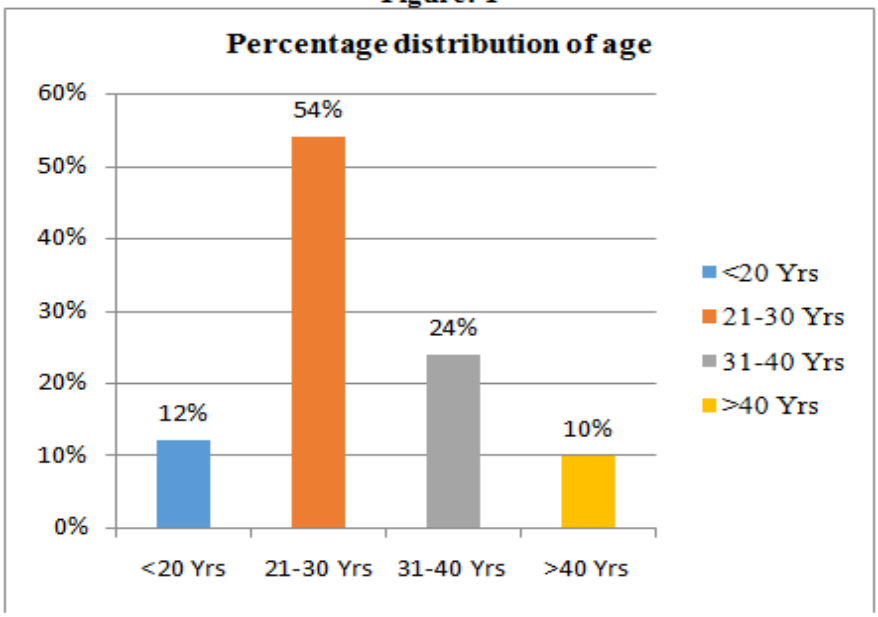
VI. Conclusion

1. Yellow oleander poisoning was most commonly observed among young adults and adolescents.
2. Although there was a slight female preponderance the difference was not statistically significant.
3. In majority of cases, the intention was suicidal secondary to interpersonal conflict, grief reaction, situational reaction, unemployment etc.
4. The most common symptoms of yellow oleander poisoning in the present study were vomiting, giddiness and diarrhea.
5. Electrocardiographic changes noted in the present study were mainly dueto 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 sino-atrial block.
6. Hyperkalemia as a manifestation of yellow oleander poisoning was uncommon in the present study compared to Srilankan studies.
7. There was a positive correlation between incidences of cardiotoxicity as the quantity of seeds ingested.
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 occurrence of cardiotoxicity was higher in patients who had vomiting, diarrhea or altered mental status compared to patients who did not have any of these symptoms.
10. The mean serum potassium values at presentation were normal in patients whohad cardiotoxicity when compared to patients who had no cardiotoxicity.

Tables and figures:

AGE GROUP			
TABLE: 1			
AGE GROUP		Frequency	Percent
	<20	6	12.0
	21-30	27	54.0
	31-40	12	24.0
	>40	5	10.0
	Total	50	100.0

Figure: 1



Sex Distribution

Table: 2

		Frequency	Percent
	Male	22	44.0
	Female	28	56.0
	Total	50	100.0

Figure: 2

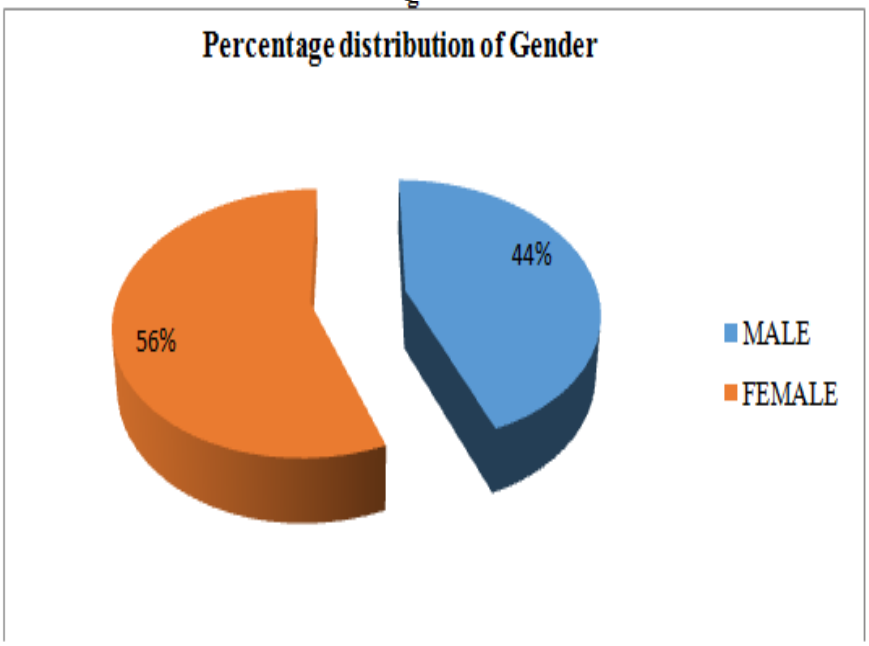


Table: 3 Number Of Seeds Ingested

No.of seeds		Frequency	Percentage
	1	2	4 %
	2	6	12 %
	3	11	22 %
	4	11	22 %
	5	10	20 %
	6	7	14 %
	7	1	2 %
	8	1	2 %
	12	1	2 %
	Total	50	100 %

Table: 4 Symptoms

Symptoms	No. of patients	%
Vomiting	28	80.0%
Giddiness	24	68.6%
Diarrhoea	13	37.1%
Abdominal Pain	15	42.9%
Altered sensorium	2	5.7%
Blurred_vision	5	14.3%
Palpitation	17	48.6%
Breathlessness	12	34.3%

Table: 5 Distribution Of Ecg Changes

Ecg changes	SB	1° AVB	2 ° AVB	ST	SAB	TI	CHB	STI	JER	VE
No of patients	25	11	2	2	2	5	2	2	2	1
%	50.00%	22.00%	4.00%	4.00%	4.00%	10.00%	4.00%	4.00%	4.00%	2.00%

Figure: 3 Number Of Seeds Ingested And Cardiotoxicity

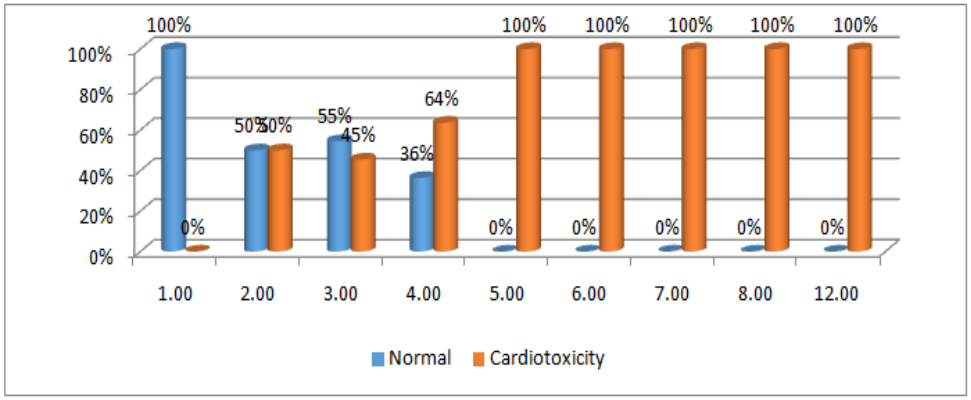
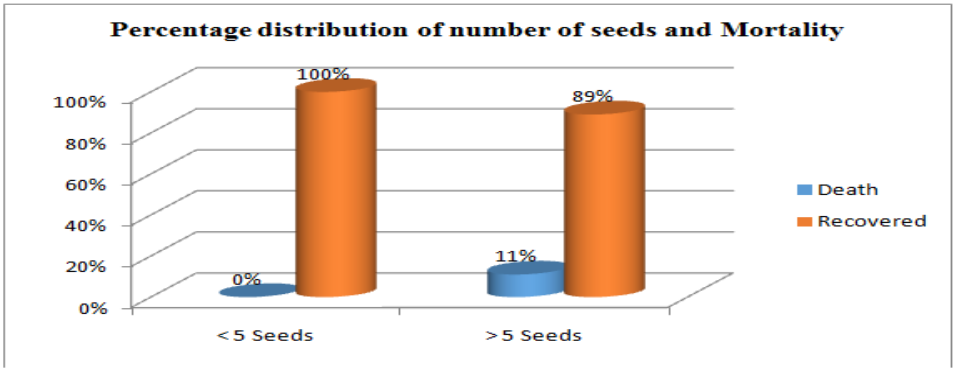


Figure: 4



Form Of Ingestion Table: 6

		Frequency	Percent
	Grounded	32	64.0
	Paste	10	20.0
	Bite & chewed	3	6.0
	Swallowed	4	8.0
	Leaves +seeds	1	2.0
	Total	50	100.0

Table: 7 Time Of Admission And Cardiotoxicity

			cardiotoxicity		Total
			Normal	Yes	
Time of admission	<3 Hrs	Count	9	13	22
		%	60.0%	37.1%	44.0%
	3 -6 Hrs	Count	4	15	19
		%	26.7%	42.9%	38.0%
	>6 hrs	Count	2	7	9
		%	13.3%	20.0%	18.0%
Total		Count	15	35	50
		%	100.0%	100.0%	100.0%

P value – 0.05 (statistically significant)

Time of admission & outcome table: 8

			outcome		Total
			death	recovered	
Time of Admission	<3 Hrs	Count	0	22	22
		%	0.0%	100%	
	3 -6 Hrs	Count	0	19	19
		%	0.0%	100%	
	>6 hrs	Count	2	7	9
		%	22%	78%	
Total		Count	2	48	50
		%	**8%**	**92%**	**100.0%**

P value - 0.009 (statistically significant)

Requirement Of Drugs Table: 9

Drugs	No of patients	Percent
Atropine	26	74%
Orciprenaline	7	20%
Both	7	20%

Reference

[1]. Driggers DA, Solbrig R, Steiner JF, et al : Acute oleander poisoning – A Suicide attempt in a geriatric patient. West J Med 1989 Dec; 151 : 660 -662

[2]. Saravanapavananthan N and Ganeshamoorthy J (1988) Yellow oleander poisoning - a study of 170 cases. Forensic Science International 36, 247 – 250.

[3]. Sreeharan N, Putharasingam S, Ranjadayalan K, et al. Yellow oleander poisoning – Clinical manifestations and prognostic criteria. Jaffna Medical Journal 1985 ; 100, 101.

[4]. Eddleston M, Ariaratnam CA, Meyer PW, et al. Epidemic of self poisoning with seeds of the yellow oleander tree (Thevetiaperuviana) in northern Srilanka. Trop Med Int Health 1999 ; 4-266-73.

[5]. Modi NS Modi's Textbook of Medical Jurisprudence and Toxicology. 22nd Edition (Editor Subrahmanyan BV). Butterworths India, New Delhi 1999.

[6]. Pearn J Oleander poisoning In : Toxic Plants and Animals : A guide for Australia 2nd Edition (EdsCovacevich J, Davine P and Pearn J). William Brooks, Brisbane, 1989:37-50.

[7]. Bose TK, Basu RK, Biswas B, et al. Cardiovascular effects of yellow oleander ingestion. J Indian Med Assoc. 1999 Oct;97(10): 407-410.

[8]. Eddleston M, Ariaratnam CA, Sjostrom L, et al. Acute yellow oleander (Thevetiaperuviana) poisoning : Cardiac arrhythmias, electrolyte disturbances and serum cardiac glycoside concentrations on presentation to hospital Heart 2000 : 83:301-306

[9]. De Silva HA, Fonseka MM, Pathmeswaran A, et al. Multiple – dose activated charcoal for treatment of yellow oleander poisoning : A single - blind, randomized, placebo – controlled trial. Lancet 2000, Jun 7; 363 (9373) : 1935 – 38.
[10]. Eddleston M, Rajapakse S, Rajakanthan et al. Anti – digoxin Fab fragments in cardiotoxicity induced by ingestion of yellow oleander : a randomized control trail.
[11]. Aggarwal A. The poison sleuths death by yellow kaner. Science Reporter, 1999 Sep;9 : 31-34.
[12]. Middleton WS, Chen KK, Clinical results from oral administration of thevetin, a cardiac glycoside. Am Heart J 1936 ; 11 : 75-88

*Sureshkumar Aparna. "Yellow Oleander Seed Poisoning – A Profile." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) 16.9 (2017): 64-71