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STUDY OF SOCIO CLINICAL, BIOCHEMICAL AND ECG ASPECTS OF YELLOW OLEANDER POISONING

Dr. T. Grashia,MD and Dr. J. Bharath*

Senior Assistant Professor in Medicine Department of Medicine Tirunelveli Medical College Tirunelveli.

***Corresponding Author: Dr. J. Bharath**

Senior Assistant Professor in Medicine Department of Medicine Tirunelveli Medical College Tirunelveli.

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ABSTRACT

Introduction: The yellow oleander, an evergreen shrub or a small tree of Apocyananceae family is widely distributed over the tropics. Yellow oleander (Thevetia peruviana), is one of the most poisonous plants which contains numerous toxins .The toxic effects resemble that of digitalis toxicity. The use of seeds or fruits of yellow oleander as a method of self-harm is common in South India. Severely poisoned patients may die in DC shock resistant ventricular fibrillation. It can lead to severe Hyperkalaemia which is a marker of a poor outcome in cardiac glycoside poisoning, which may worsen further toxicity and lead to serious Arrhythmias. **Aim & Objectives:** To find out the incidence of yellow oleander poisoning and to analyze the socio-clinical aspects. Also to correlate the clinical and biochemical data with electrocardiographic changes. **Materials and methods:** All patients admitted with consumption yellow oleander poison was studied. The study was carried out over a one year from June 2008 – May 2009. Clinical parameters, CBC, Serum Urea, Creatinine, Serum Na+, Serum K+ and ECG were done to analysis the changes on hourly basis. **Results:** 160 patients with yellow oleander poisoning were included in the study of which 54 were males and 106 were females. Most patients had consumed oleander in a suicidal intention. 1.73% of patients had underlying psychiatric illness. Case fatality rate was 7.5%. The most common electrocardiographic abnormality was sinus bradycardia. Life threatening arrhythmias were seen in 6.8%. There was significant correlation between outcome and serum potassium levels, bicarbonate levels, and delay in seeking medical attention, and number of symptoms at presentation. There was no correlation between number of seeds consumed and outcome or cardiotoxicity. Oleander seed is still used as a suicidal agent. Oleander plant is easily available as an ornamental plant in urban, semi-urban and rural areas. Highly symptomatic patients and those who has delay in seeking medical attention showed more cardio toxicity. Hence earlier treatment and symptomatic management will help in reducing complications and mortality.

KEY WORDS: Oleander seed, Hyperkalaemia, ECG abnormalities, Cardiac glycoside poisoning.

INTRODUCTION

The yellow oleander (Thevetia Peruviana) is an ornamental tree, which is common throughout the tropics and subtropical countries. It contains cardiac glycosides that are toxic to cardiac muscle and the autonomic nervous system[1].

Deliberate ingestion of oleander seeds has become a frequent method of self-harm in India. Oleander is an ornamental tree of the Apocyanaceae family that is common throughout the tropics and subtropics. It is wide spread in India, Nepal and Srilanka[2]. Its sap contains cardiac glycosides (thevetins A and B and neriifolin) including the roots and the smoke produced from burning, toxic to cardiac muscle and the ingestion of its seeds results in a clinical picture similar to that of digoxin poisoning[3]. Oleander leaf also contains other biologically active constituents that have antimitotic and insecticidal properties. Oleander is also reported to have emetogenic, cathartic, insecticide, parasiticidal, anthelmintic, menstrual stimulant, and abortifacient activities. The majority of deaths occurring after ingestion of plant are due to yellow Oleander or 'pita kaner' (cerebra thevetia), pink eyed cerebra or 'sea mango' (cerebra manghas), and white oleander or 'kaner' (nerium odorum) are reported in South India.Many patients with moderate poisoning show PR interval prolongation and progression to atrioventricular (AV) dissociation. Severely poisoned patients may die in DC shock resistant ventricular fibrillation. However, deliberate ingestion of yellow oleander seeds has recently become a popular method of self-harm[4]. So, our study aimed to find out the incidence of yellow oleander poisoning. Also to analyze the socio-clinical aspects and to correlate the clinical and biochemical data with electrocardiographic changes.

MATERIALS AND METHODOLOGY

The study was conducted on patients admitted to the Medicine Department in Government Medical College Hospital, Chengalpattu. The study was a prospective study conducted for a period of one year from June 2008 – May 2009. A total of 160 cases of yellow oleander poisoning who satisfied the inclusion and exclusion criteria were included in the study. Patients who were admitted in general medicine wards with history of yellow oleander ingestion were included in study while patients who had underlying severe cardia, renal or hepatic disease and pediatric cases less than 12 years of age were excluded. Also patients who were taking the following drugs-digoxin, diuretics, verapamil, diltiazem, beta blocker, ACE inhibitors, amiodarone, calcium and potassium supplements were excluded.

Sociodemographic, clinical, biochemical, electrocardiographic prepared proforma details were collected from the patients and recorded in a pre-prepared proforma. Data regarding poisoning comprised of part ingested, quantity of poison, method of ingestion, whether consumption on empty stomach or after food, first aid at home, consumption to admission interval, treatment given, duration of hospital stay and type of outcome everything were collected. Socioeconomic status was based on Modified Kuppuswamy Classification.[5]

Clinical data collected were as following - Symptom analysis, pulse rate, rhythm, Blood Pressure and systemic examination. Laboratory investigations like blood sugar, urea, creatinine and electrolytes were estimated. 12 -lead ECG was taken on admission before instituting treatment and repeated depending on clinical status. This research work was approved by institutional ethics committee and informed consent were obtained from patients.

Yellow oleander poisoning cases were divided into no cardio toxicity, some cardio toxicity and severe cardio toxicity groups based on electrocardiographic changes. Patients with normal sinus rhythm or sinus tachycardia only comes under no cardiotoxicity. While patients with Sinus bradycardia, First degree atrioventricular block, ST segment and T wave changes were considered having some cardiotoxicity. Whereas patients who comes under severe cardiotoxicity includes those who have Sino atrial block, Junctional rhythm, Mobitz type-II second degree atrioventricular block, Third degree atrioventricular block, Atrioventricular dissociation and ventricular fibrillation. Statistical analysis was done using Epidemiological Information Package (EPI 2008). Kruskal Wallis chi square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

The present study was a prospective study which included 160 patients with yellow oleander poisoning. There were 198 cases of oleander poisoning during the study period out of which 160 were included in the study according to the inclusion and exclusion criteria. Oleander poisoning contributed to 16% of total poisoning cases in Chengalpattu medical college hospital. Among 1234 total poisoning cases in our study period 198 cases were oleander poisoning which shows the prevalence of oleander poisoning around 16%. Female were common among them with 136 patients were female while only 62 are male with a male: female ratio of 1:2.1. Around 16 patients died which shows a case fatality rate of 7.5%.

Table 1: Social Profile.

Social profile	CASES	
	No.	%
Sex		
Male	54	33.8
Female	106	66.2
Marital Status		
Unmarried	62	38.8
Married	98	61.2
Divorced	-	-
Widower/Widow	-	-
Domicile		
Rural	144	90
Urban	16	10
Socio-Economic status		
Upper	0	0
Upper Middle	5	3.1
Lower Middle	39	9.4
Upper Lower	95	59.4
Lower	29	18.1

Coming to age distribution it was seen that most of the cases were in the age group <30 years (73%, n=119)). Only 3.8% of patients were above the age of 50 years. Almost 50% of cases are in the prime period of adulthood in the age group 20-30 yrs., in this study. Range was between 16-62 years and the mean age among our study group is 27.5 ± 9.7 years.The social profile of the cases was studied. It was seen that among the 160 cases studied, among which 106 were females (66.2%) and 54 were males (33.8%). Male: Female ratio was around 1:1.96. Also coming to marital status 62 patients were unmarried (38.8%) and 98 patients were married (61.2%). Among our study group 144 patients hailed form rural areas (90%) and the rest from urban areas. When we analysed the socio-economic status. It was also seen around 75% (n=134) of patients were from a lower socioeconomic background out of which 60% (n=95) belonged to upper lower classes and 18% (n=29) belonged to lower socioeconomic status.

Table 2: Poison profile.

Poison profile	CASES	
	Number of patients	%
Place of Ingestion		
Home	147	91.9
Others	13	8.1
Part of plant		
Fruit	86	53.8
Seed / Cover	74	46.2
Flower	0	0
Others	0	0
Method of ingestion		
Crushed	102	63.8
Swallowed	18	11.3
Chewed	40	25
Relation to food		
After	72	45
Before	34	33.8
With	34	21.2
Intention		
Suicidal	160	100
Homicidal	-	-
Accidental	-	-
Reason		
Psychiatric illness	3	1.9
Inter personal conflict	90	56.3
Situational reaction	60	37.5
Grief reaction	60	3.8
Others	1	0.6

The following observations were made in relation to the poison consumption. Most of the patients had consumed the poison at home (91.9%, n=147). A minority had consumed it outside their homes. The part of the plant consumed most commonly was the fruit in 53.8% (n=86) and the seed in 46% of patients (n=74). Sixty three percent of patients (n=102) had crushed the poison before consumption compared to 11.3% (n=18) who swallowed it as whole and 25% (n=40) who chewed it. The intention was suicidal in 100% of cases. Similarly the reason for consumption was psychiatric illness in 1.9% (n=3), interpersonal conflict in 56.3% (n=90), situational reaction in 37.5% (n=60), and grief reaction in 3.8% (n=6).

We analyzed the prognostic outcome of these patients where out of 160 patients, 148 improved (92.5%) and were discharged. The mortality was 7.5% (n=12).

Table 3: Clinical Profile of the Patients.

CLINICAL PROFILE	Cases	
	No.	%
Co morbid illness		
Present	-	-
Absent	160	100
Psychiatric illness		

Present	3	1.9
Absent	157	98.1
Cardiovascular system		
Palpitation	62	38.8
No Symptoms	98	61.2
Respiratory system		
Dyspnea	10	6.3
No symptoms	150	93.2
Gastrointestinal system		
Vomiting	52	32.5
Abdominal pain	19	11.9
No symptoms	89	55.6
Central Nervous System		
Giddiness	51	31.9
Numbness	8	5
Seizures	6	3.8
No symptoms	95	89.4
Pulse rhythm		
Regular	124	77.5
Irregular	36	22.5
Sensorium		
Normal	133	83.1
Abnormal	27	16.9

The following observations were made on analyzing the clinical presentation of the patients. Co morbid illness was not present in any of the patients. Psychiatric illness was present in 1.9% (n=3) of patients and was the root cause in these patients. On systemic examination the predominant cardiovascular symptom was palpitation which was present in 62 patients (38%). Dyspnea on admission was present in 10 patients (6.3%). Emesis and abdominal pain were the chief gastrointestinal symptoms and were present in 32.5% (n=52) and 11.9% (n=19) respectively. Neurological symptoms were present in around 40% (n=65) of patients of which giddiness was the commonest. Altered sensorium was present in 16% (n=27) of patients on admission. Pulse rhythm was irregular in 22.5% on admission (n=36) and regular in 77.5% (n=124) of patients. We also analyzed the biochemical profile where mean sodium, potassium and bicarbonate levels were 137.8, 4.19 and 19.17 meq/1 respectively. None of the patients had renal dysfunction.

Our next important analysis was ECG findings. Four serial ECG recording were done and the findings were tabulated as follows.

Table 4: ECG Changes.

ECG finding	**ECG change**							
	1		**2**		**3**		**4**	
	No	**%**	**No**	**%**	**No**	**%**	**No**	**%**
Sinus rhythm	69	43.1	114	71.3	141	88.1	133	83.1
Sinus Bradycardia	41	25.6	29	18.1	4	2.5	15	9.4
First degree AV Block	9	5.6	2	1.3	-	-	-	-
Second degree AVB MOB	5	3.1	1	0.6	1	0.6	-	-
Mobitz –II	2	1.3	1	0.6	1	0.6	-	-
Complete Heart Block	1	0.6	5	3.1	5	3.1	4	2.5
SA Exit Block	3	1.9	-	-	1	0.6	2	1.3
Sinus Tachycardia	6	3.8	1	0.6	-	-	-	-
MAT	2	1.3	-	-	-	-	-	-
Ventricular Bigemini	4	2.5	2	1.3	1	0.6	-	-
Ventricular Tachycardia	1	0.6	2	1.3	2	1.3	2	1.3
Ventricular Fibrillation	2	1.3	1	0.6	-	-	-	-
Non-Specific ST-T changes	15	9.4	1	0.6	-	-	-	-
Total	160	100	160	100	160	100	160	100

The following observations were made on analyzing the electrocardiogram of the patients: Sixty-nine patients had sinus rhythm on admission (43). Sinus bradycardia was the most common abnormality noted in 25% of patients

(n=40). Life threatening arrhythmias were seen in four patients which included complete heart block and ventricular fibrillation, Cardio toxicity was graded as mild and serve according to the ECG findings. 42.5% of patients (n=68) had no evidence of cardio toxicity; 35% (n=56) has mild cardio toxicity and 22.5% (n=36) had serve cardio toxicity.

Next parameter analyzed was time gap between poison consumption and delay in treatment. The mean delay in seeking medical attention was 4.37±2.87 hours.

Table 5: Delay Treatment.

Delay in treatment	CASES	
	No.	%
Up to 1 hour	10	6.3
1.1-3 hours	60	37.5
3.1-5 hours	43	26.9
5.1-10 hours	41	25.6
<10 hours	6	3.7
Total	160	100
Mean ± S.D	4.37 ±2.87 hours	

Correlation was analyzed between cardio toxicity and the biochemical variables. It was seen that the mean serum potassium value was 4.77±1.55 meq/1 in patients with serve cardio toxicity compared to 4.07±0.62 meq/1 in patients with no cardio toxicity. That difference was statistically significant (**p=0.002).** A similar correlation was seen between cardio toxicity and bicarbonate levels where it was seen that bicarbonate levels were less in patients with serve cardio toxicity compared to the patients with no cardio toxicity. The difference was statistically significant **(p=0.0001).** There was no correlation between cardio toxicity and renal parameters or serum sodium levels.

Table 6: Cardio Toxicity and Biochemical Variables.

Parameter	Cardio toxicity						'p Value'
	None		Mild		Severe		
	Mean	S. D	Mean	S. D	Mean	S. D	
Blood Urea							
1	28.7	8.9	29.1	9.6	29.3	6.8	0.779
2	30	14.9	28.8	8.5	28	6.2	0.9865
3	27.3	7.7	30.2	15.2	28.4	7.8	0.6375
4	29.4	11.3	27.7	7.7	28.8	7.1	0.5723
Sr. Creatinine							
1	1.01	0.49	1.01	0.53	1.15	0.95	0.5042
2	1.05	0.56	1012	0.6	1.01	0.42	0.2456
3	1	0.33	1.05	0.49	1.05	0.64	0.8408
4	0.98	0.22	1.0	0.52	1.0	0.41	0.4657
Sodium	138.5	5.1	137.4	4.4	136.9	3.9	0.1753
Potassium	4.07	0.62	3.96	0.43	4.77	1.55	0.002
Bicarbonate	19.4	2.5	19.8	2.2	12.3	2.9	0.0001

Cardiotoxicity was also categorized and analyzed with other parameters like delay in treatment, quantity of poison and Clinical symptoms. The mean delay in treatment was 3.49 ± 2.05 hours in patients with no toxicity; 3.89 ± 2.4 hours in patients with mild cardio toxicity and 6.75 ± 3.5 hours in patients with serve cardio toxicity. The difference was statistically significant (p=0.0001). Similarly there was no significant difference in cardio toxicity in relation the quantity of poison (p-0.1523). The relation between clinical features and cardio toxicity was analyzed. It was seen that patients with severe cardio toxicity had one or more symptoms pertaining to cardiovascular, neurological or gastrointestinal systems. The relationship was significant statistically.

Table 7: Symptoms and Cardio Toxicity.

Symptoms	Cardio toxicity					
	None		Mild		Severe	
	No.	%	No.	%	No.	%
CVS						
Yes (62)	19	30.6	26	41.9	17	27.4
No (48)	49	50	30	30.6	19.4	19.4
'p'	**0.0245 Significant**					
R S						
Yes (10)	3	30	5	52	2	20
No (150)	65	43.3	51	34	34	22.7
'p'	0.3155 Not significant					
G I T						
Vomiting (52)	18	34.6	17	32.7	17	32.7
Abdominal Pain (19)	1	5.3	-	-	17	94.7
No symptoms (89)	49	55.1	39	43.8	1	1.1
'p'	**0.0006 Significant**					
C N S						
Yes (65)	21	32.3	18	27.7	26	40
No (95)	47	49.5	38	40	10	210.5
'p'	**0.0461 Significant**					

Outcome of the intoxication was correlated with Cardiotoxicity, Delay in treatment, Quantity of poison and number of symptoms. A significant association was seen between cardiovascular toxicity and final outcome. Twelve out of 36 patients who had severe cardio toxicity died (33.3%). The relationship was statistically significant. **(p=0.0001).** Similarly there was also significant association between the time delay in seeking medical attention and outcome as it was seen that delay in treatment was 8.58±3.13 hours in patients who died compared to 4.02±2.57 hours in patients who improved. The relationship was statistically significant **(p=0.0001).** The mean quantity of poison consumed was 3.67±1.07 in patients who died as opposed to 2.13±1.25 in patients who survived. The relationship was statistically significant **(p=0.0002).** It was seen that patients who died had more number of symptoms (2.25±1.14) than patients who survived. (1.2± 1.0). The relation was statistically significant. (p=0.0027).

DISCUSSION

The Yellow Oleander plant is a common source of accidental poisoning worldwide. In parts of India and Sri Lanka it has become a popular Means of self-harm. Manifestations range from mild to potentially fatal. It has significant cardiovascular effects with carrying rhythm abnormalities. There are now tens of thousands of yellow oleander poisoning cases in South Asia each year and probably thousands of deaths. Management of patients with severe poisoning may be difficult and costly, placing great stress on the health system due to requirements for expensive treatment cardiac pacing. Although timely medical intervention reduces the mortality, a sub group of patients who sustain sudden cardiac death, the cause remains elusive. In animal models cardiac damage in Yellow Oleander Poisoning is caused by inflammation and degenerative changes. There are not many human studies on risk factors for cardio toxicity and sudden death in Yellow Oleander Poisoning.

We studied all patients with yellow oleander poisoning admitted to a secondary care hospital in north Tamilnadu from June 2008 to 2009, with the objective of determining the outcome of management using currently available treatment. During the study period 160 patients with Yellow Oleander poisoning were admitted to the hospital (male: female = 54:106).

The present study was undertaken to establish risk factors for cardio toxicity induced by cardiac glycosides in oleander. Many of the findings in our study correlated well with past literature. In our study there was more number of female victims which was in concurrence with world literature. Studies done by Rajapakshe, Eddleston and Fonseka et al showed a high proportion of female patients (1:2). Also seen was the high number of younger of younger patients (75%) compared to older patients,[6,7] True psychiatric illness was present only in 1.9% of patients whereas in other it was situational circumstances that led to poisoning. This was seen in a study done by Eddlestion et al where borderline personalities and non-psychiatric causes were seen in most of the patients. In the study in eastern India by Bose et al which included 300 patients with Yellow Oleander poisoning, Majority i.e., 246 (82%) were females and 226 (75.33%) were young in the age group 11-20 years.

Mostly patients reported for treatment 6 to 8 hours after ingestion of seeds. The number of seeds swallowed varied from half to fifteen. Two hundred and ninety-two (97.33%) ingested seeds in the crushed form. Fonseka et al also showed that patients who had consumed oleander in a suicidal intent always consumed it in a crushed form rather than the whole fruit[8]. This was seen in our study also.

The case fatality rate in our institution was 7.5%. Case fatality from yellow oleander poisoning varies between institutions and studies, with an overall mortality of 3-10%. This wide variability in mortality may reflect the availability of temporary cardiac pacing facilities, intermittent supply of ant digoxin Fab antitoxin, the higher proportion of severely poisoned patients at referral hospitals, inadequate cardiac monitoring or complication associated with high doses of atopic. The only other eastern India by Bose et al showed a mortality rate of 2.5%.43 Fonseka et al from Sri Lanka showed a mortality rate of 2.4%.[8]

Amongst the electrocardiographic abnormalities, Sinus Bradycardia was the most common finding accounting for 25% of cases. In addition, eleven patients (6.8%) had life threatening arrhythmias. In the study by Fonseka et al, twenty-five (14.8%) patients have arrhythmias that were considered life threating (second-degree heart block type II, third-degree heart block and nodal bradycardia).[8] The electrocardiogram (ECG) of patients in north eastern India, 138 (46%) revealed varying types of arrhythmias including sinus bradycardia in 68 cases (49.27%). Ischemic changes were present in 118 cases (39.33%). During discharge, 256 (85.33%) had normal ECG, 14 (4.66%) had sinus bradycardia and 16 (5.33%) demonstrated ischemic changes.[9] Our study also showed similar findings with 85% of patients showing sinus rhythm at discharge and only 5% having persistent sinus bradycardia.

Significant hyperkalemia was seen in patients with serve cardio toxicity in our study. Robert et al found that hyperkalemia was associated with increased toxicity.[10] Higher mean admission serum K+ was seen in patients with severe cardio toxicity (5.4mmol/L) than those with mild cardiotoxicity (4.3mmol/L). However, it does not appear to be a reliable marker. Other factors may alter serum potassium such as acid-base disturbances from the use of sodium bicarbonate in forced emesis solutions or even as a result of metabolic acidosis due to cardiovascular shutdown. Serum bicarbonate was also significantly low in patients with severe cardio toxicity, the significance of which is unknown. This probably reflects metabolic acidosis due to cardiovascular failure.

There was significant association between delay in treatment and degree of cardio toxicity at admission. This is well explained in various studies. There was no association between the number of seeds consumed and the degree of cardio toxicity. This was proven in various studies in the past as even one seed can cause death. Bose et al also showed that the quantity of poison did not matter.[9] It was also seen in our study that there was significant association between overall outcome and the number of seeds consumed. This can probably be explained by the fact that several patients had progression of cardiotoxicity while in the hospital.

There was good correlation between number of symptoms and the degree of cardio toxicity and overall outcome. In our study patients with serve cardio toxicity presented with at least two or more symptoms pertaining to cardiovascular, neurological or gastrointestinal systems. This has been described in serval forensic literature and larger studies on Yellow Oleander poisoning. In the study by Bose et al from North Eastern India, 156(52%) were asymptomatic, 92(30.66%) had vomiting and 36 (12%) had palpitation. In our study nearly 39% had palpitation and 32% had nausea/vomiting[9]. In our study, the mean duration of stay in the hospital was 3.2±1.3days. This was in concurrence with study done by Bose et al where median Hospital stay was 5 days.[9]

CONCLUSION

Most patients hailed form rural areas and came from lower socioeconomic strata of society. The part of the plant most commonly used was the fruit. Sinus bradycardia was the most significant electrocardiographic abnormality. Life threatening arrhythmias were seen in 7% of patients. Highly symptomatic patients and those who has delay in seeking medical attention showed more cardio toxicity. Hence earlier treatment and symptomatic management will help in reducing complications and mortality.

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