

A STUDY ON THE CLINICAL AND BIOCHEMICAL PROFILE OF PATIENTS ADMITTED WITH PLANT POISONING AND THEIR OUTCOME IN A TERTIARY CARE CENTRE IN CHENNAI

T.S. Karthigeyan¹, Balamanikandan Paulchamy², A.T. Jayaraj³, T.S. Santhi⁴, S. Vigneswaran⁵

Received : 05/08/2023

Received in revised form : 08/09/2023

Accepted : 20/09/2023

Keywords:

Plant poisoning, Oduvanthalai, Yellow oleander, Datura, Sinus bradycardia.

Corresponding Author:

Dr. A.T.Jayaraj,

Email: vetriventhan51@gmail.com.

DOI: 10.47009/jamp.2023.5.5.155

Source of Support: Nil,

Conflict of Interest: None declared

Int J Acad Med Pharm

2023; 5 (5); 790-796



¹Assistant Professor, Institute of Internal Medicine, Madras Medical College, Tamilnadu, India.

²Assistant Professor, Institute of Internal Medicine, Madras Medical College, Tamilnadu, India.

³Assistant Professor, Institute of Internal Medicine, Madras Medical College, Tamilnadu, India.

⁴Professor, Institute of Internal Medicine, Madras Medical College, Tamilnadu, India.

⁵Junior resident, Institute of Internal Medicine, Madras Medical College, Tamilnadu, India.

Abstract

Background: Poisoning is a prevalent method for deliberate self-harm, contributing to mortality and morbidity worldwide. India has a higher prevalence of self-poisoning due to poisons, socioeconomic status, religious influences, and drug availability. Hence, this study was undertaken in our hospital to assess the incidence, clinical, biochemical profile, and Electrocardiographic changes of patients admitted with various types of plant poisoning and to see their outcome with the management protocol followed in our hospital. **Materials and Methods:** This single-centre prospective descriptive study was conducted at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, for six months (April 2021 to September 2021). The patient underwent a detailed examination including recording of vital signs, systemic examination, ECG, and continuous cardiac monitoring. Lab investigations included blood sugar, urea, creatinine, potassium, sodium, and liver function tests. **Results:** Out of the 60 cases, 26 were males and 34 were females. The incidence of poisoning is more in the age group between 26-35 years. Yellow oleander was the common type of plant poison consumed. Plant poisoning often presents with gastrointestinal symptoms like vomiting and cardiac symptoms. Oduvanthalai poisoning often presents with dyspnea. ECG changes -sinus bradycardia, ST T changes, and second-degree AV block are common. Oduvanthalai poisoning had more ICU stay associated with increased seed intake and serum potassium levels, but there was no mortality. **Conclusion:** Yellow oleander, Oduvanthalai, and datura poisonings often cause electrocardiographic abnormalities, dyselectrolytemia, renal tubular acidosis, ARDS and CNS manifestations. Hence, Early intervention is necessary to reduce morbidity.

INTRODUCTION

Poisoning is the most common method for deliberate self-harm, including suicidal attempts. Poisoning, both accidental and intentional, significantly contributes to mortality and morbidity worldwide. About 800,000 people commit suicide worldwide every year. Among them, 18% are Indian, which amounts to 1.39 lakh Indians committing suicide yearly, and the national suicide rate is around 10.4. Acute poisoning forms one of the most common causes of emergency hospital admissions. The pattern of poisoning in a region depends on various factors, such as the availability of the poisons, socioeconomic status of the population, religious and cultural

influences and availability of drugs. In low-middle-income countries like India, where socioeconomic factors such as poverty, unemployment, and domestic violence are prevalent, it is unsurprising to find a higher prevalence of self-poisoning compared to psychiatric conditions in the West.^[1]

Suicidal and homicidal cases of poisoning are common in India, as poisons can be easily obtained, and many poisonous plants grow wild. Various methods of self-poisoning, as seen in another study done in South India, are ingesting pesticides, plant poisons, rodenticides, and drugs.^[2] The local availability of highly toxic plants, predominantly in the rural areas of India, provides easy access to self-poisoning. A deadly combination of low intention, high lethality, and lack of proper medical

infrastructure poses a risk of severe mortality and morbidity. In India, more than 4,000 medicinal plants are growing as herbs, shrubs, and trees, many of which local druggists use in traditional medicine to make potions for various diseases. These medicinal plants are usually seen along with many toxic plants, flowering shrubs, and herbs.

A lack of knowledge or, perhaps, the constituents' knowledge can prove fatal when used in large doses.^[3] The ingestion of medicinal compounds in large doses can be fatal, as most believe them to be particularly safe because of their natural origin. Despite the high prevalence of Deliberate self-poisoning,^[4] using plant poisons, a few studies have been undertaken to understand the patterns involved in such cases. Hence, this study was undertaken in our hospital to assess the incidence, clinical, biochemical profile, and Electrocardiographic changes of patients admitted with various types of plant poisoning and to see their outcome with the management protocol followed in our hospital.

MATERIALS AND METHODS

This single-centre prospective descriptive study was conducted at the Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, for six months (April 2021 to September 2021).

Inclusion Criteria

All patients more than 18 years of age with clinical history and features suggestive of plant poisoning were included.

Exclusion Criteria

Pediatric patients, patients with coexisting cardiac illness, patients not willing to study, patients with known cases of dyselectrolytemia, and those with known cases of chronic kidney disease were excluded.

Ethical committee approval and informed consent were obtained before the study started. All cases were admitted and examined in detail in the wards, clinical data was recorded in the proforma annexed herein, and all cases were followed till discharge or death. Personal particulars like age and sex were obtained. Clinical details regarding the type of plant poisoning, and individual details about them regarding what part of the plant consumed, associated intake, number of seeds consumed, whether taken on an empty stomach or with food, the Time interval between poisoning and hospitalisation and details of first aid were obtained are enquired and recorded. Time of poisoning, time of hospital admission, episodes of vomiting, symptoms like chest pain, palpitation, breathlessness, giddiness, syncope, and altered sensorium were asked.

The examination was done in detail; vitals were recorded (BP, PR, RR, SpO₂), and a system examination was carried out. ECG was taken in all cases after admission, and conventional limb leads, chest leads and long strips were recorded. Continuous

cardiac monitoring was done in the first 24-hour period and after that for the required patients. ECG was recorded twice for the second day and once daily until discharge. Lab investigations such as random blood sugar, urea, serum creatinine, serum potassium, serum sodium, urine routine, liver function tests, ABG, LDH/CPK/CKMB, and complete blood count were obtained.

All patients were admitted and initially treated with gastric lavage with multidose-activated charcoal. Oleander seed poisoning was treated with IV fluids, correction of hyperkalemia, symptomatic bradycardia was treated with atropine and orciprenaline, and some patients required temporary cardiac pacing. Oduvanthalai poisoning was treated with fluid resuscitation, correction of dyselectrolytemia, and ventilation in the case of an ARDS setting. Datura poisoning was treated with supportive measures along with gastric lavage.

Statistical analysis

The patient's data were collected prospectively and entered in the proforma, and the data was digitalised in Microsoft Excel software. Statistical analysis was done using SPSS version 26 software. The categorical variables were described as proportions and percentages. The continuous variables were expressed as Mean, Standard deviation, and range. The effect of various factors on the presence of ECG and no ECG changes was analysed by unpaired 't' test (difference between means) for continuous data. The Chi-square and Fischer's exact test (difference between proportions) were used to compare the categorical data. A p-value <0.05 was considered as significant.

RESULTS

Out of the 60 cases, 26 were males and 34 were females. The male-female ratio was 0.7:1, and the percentage of males was 43.3%. Incidence appears to be more in the females 29 (59.2%). The number of patients below the age of 25 was 23 (38.3%), and between 26 and 35 were 31 patients (51.7%), and the age above 35 was 6 (10%). The incidence of poisoning is more in the age group between 26-35 years.

Fifty-one patients had yellow oleander poisoning, five had Oduvanthalai poisoning, and 4 had datura poisoning. Fifty-five patients consumed seeds, five consumed leaves (All Oduvanthalai patients), and other parts of the plant were not consumed. Out of seeds, chewed was the most common form of consumption (47.27%), followed by paste form (38.18%), and grounded form was the least consumed form.

Forty-eight patients had a suicidal intention, and 12 patients had accidentally ingested. Thirty-eight patients took the plant substance in raw form, nine patients took it with alcohol, and 13 patients mixed it with food. Forty patients received first aid in gastric

lavage before visiting our hospital, and 20 patients didn't receive any first aid [Table 1].

Out of 60 patients, 48 patients had GIT symptoms, and 12 patients had no GIT symptoms. In those patients, vomiting is the most common clinical presentation. Twenty patients had cardiac symptoms, and all the patients had palpitations. CNS symptoms were seen in 8 patients, and 52 patients didn't have any CNS complaints. Seven patients had respiratory symptoms, and 53 patients had no respiratory symptoms.

Thirty-eight patients had ECG changes, and 22 patients didn't have ECG changes. Various type of ECG changes was encountered, mainly in oleander poisoning. Sinus bradycardia is the most common ECG finding (25%), followed by ST T changes (18.3%) followed by second-degree AV Block [Table 2].

Of 26 males, 21 male patients took yellow oleander, two male patients took Oduvanthalai, and three male patients took datura. Of 34 females, 30 female patients took yellow oleander, 3 took Oduvanthalai, and one took datura. Yellow oleander poisoning patients are seen most among the age group between 26-35 (25 patients), the 15-25 age group had 23 patients, and more than 35 age has three patients. In Oduvanthalai poisoning ingested patients, three patients were from the 26-35 age group, and two patients were older than 35. Three patients from the 26-35 age group and one patient from more than 35 age group had taken Datura poisoning.

All patients were taken seed form of Datura and Yellow Oleander poisoning. The chewed form is the most common form of consumption of yellow oleander (49%), followed by the paste form (41.2%). Vomiting is present in 75% of Datura patients, and around 58.8% of patients who consumed oleander seed had vomiting. Palpitations were present in 19 patients of yellow oleander poisoning out of 20 patients who presented with cardiac symptoms. Cardiac symptoms were not present in 40 patients. Altered sensorium is the predominant feature in Datura poisoning seen in all patients. (4 patients) Two cases of oleander and Oduvanthalai poisoning had CNS features. Dyspnoea is present in 4 patients of Oduvanthalai poisoning (80%) and not present in 1 patient. Respiratory symptoms are not present in Datura poisoning, and it is present in three patients with yellow oleander poisoning [Table 3].

ECG changes were present in 34 cases of yellow oleander poisoning (66.7%), and ECG changes were not present in 17 cases of yellow oleander poisoning. ECG changes were present in 3 cases of Oduvanthalai poisoning and one case of Datura poisoning. Patients who took Datura and Oduvanthalai poisoning had no other changes, but 15 (29.4%) patients had sinus bradycardia who took Yellow

Oleander. 15 of 60 patients (29.4%) who took Yellow Oleander had sinus bradycardia.

Patients who took Yellow Oleander had ST T changes 8 (15.7%), Exit block/sinus arrest 3 (5.9%), Premature atrial contractions 2 (3.9%), AV dissociation 3 (5.9%), First-degree AV block 4 (7.8%), Second-degree AV block 8 (15.7%), Third-degree AV block 3 (5.9%), and Junctional rhythm 3 (5.9%) patients [Table 4].

The mean age across the type of plant poison was 32.25±4.11 for Datura poisoning, 34.20±6.30 for Oduvanthalai poisoning, and 26.55±5.69 for yellow oleander poisoning.

The mean number of seeds/leaves taken across the plant poisons were Datura (7.75±1.26) seeds, Oduvanthalai (8.80± 2.59) leaves, and yellow oleander (5.22±2.18).

The mean time interval from poisoning to admission in hrs is datura (5.75±2.87), Oduvanthalai (4.80±2.39), and yellow oleander (4.63±2.79).

ICU stay was found to be increased in Oduvanthalai poisoning (7.00± 1.41) as comparable to yellow oleander (2.88±1.45) and datura poisoning (4.25±0.50). Total stay also increased in Oduvanthalai poisoning (8.20± 1.64), then in datura (5.25±0.50) and yellow oleander (3.69±1.49) [Table 5].

The systolic BP and Diastolic BP were found to be on the lower side in cases of oleander seed poisoning compared to datura and Oduvanthalai poisoning. The mean pulse rate of oleander seed poisoning was around 68±12, whereas, in Datura poisoning, it was 113.25±9.25. The respiratory rate was normal in oleander and datura-ingested patients, whereas it was elevated at 18±2.92 in the case of Oduvanthalai poisoning. QTc prolongation as evident in Oduvanthalai patients (471.20±16.53) ms. RBS were near normal in all groups. There was a slight elevation of urea and creatinine in Oduvanthalai poisoning compared to the other two. Hypokalemia was present in Oduvanthalai poisoning patients (3.08± 0.51), and hyperkalaemia was found in yellow oleander poisoning. Liver enzymes (AST, ALT) were elevated in a case of Oduvanthalai poisoning patients. There were reduced serum bicarbonate levels in Oduvanthalai poisoning (16.20±1.64) [Table 6].

ECG changes in the time interval from poisoning to admission (hours) were 4.92 ± 2.99 hours, and no changes were 3.36 ± 2.28 hours. Patients who experienced ECG changes after consuming oleander seed poisoning had an average of 6.55 ± 2.18 seeds; no changes were 4.18 ± 2.08. ECG changes were present in high potassium values (4.82±0.87), and ECG changes were not present in low potassium values (4.31 ± 0.71). Patients who experienced ECG changes had an average hospital stay of 4.76 ± 1.60 days, and no changes were 3.14 ± 2.05 days [Table 7].

Table 1: Demographic data of the study

		Frequency (N)	%
Gender	Male	26	43%
	Female	34	57%
Age group	15-25	23	38.30%
	26-35	31	51.70%
	>35	6	10%
Type of plant poison taken	Datura	4	7%
	Oduvanthalai	5	8%
	Yellow Oleander	51	85%
Form of the plant taken	Leaves	5	8.3%
	Seeds	55	91.7%
Type of intake	Chewed	26 (47.27%)	43.30%
	Grounded	8 (14.5%)	13.30%
	Paste	21(38.18%)	43.30%
Associated intake	Alcohol	9	40.90%
	Mixed with food	13	59.10%

First aid	No	20	33.30%
	Yes	40	66.70%

Table 2: Various symptoms and ECG changes

		Frequency (N)	%
GIT symptoms	Abdominal pain	2	3.30%
	Abdominal pain, diarrhoea	1	1.70%
	No symptoms	12	20%
	Vomiting	34	56.70%
	Vomiting, abdominal pain	7	11.70%
	Vomiting, diarrhoea	4	6.70%
Cardiac symptoms (palpitation)	No	40	66.70%
	Yes	20	33.30%
CNS symptoms (altered sensorium)	Altered sensorium	8	13.30%
	Nil	52	86.70%
Respiratory symptoms	Dyspnea	7	11.70%
	Nil	53	88.30%
ECG changes	No	22	36.70%
	Yes	38	63.30%
ECG changes present	Sinus bradycardia	15	25%
	Exit block/sinus arrest	3	5%
	Premature atrial contractions	2	3.30%
	AV dissociation	3	5%
	First degree	4	6.70%
	Second degree	8	13.30%
	Third degree	3	5%
	Tall t wave	4	6.70%
	Junctional rhythm	3	5%
	ST T changes	11	18.30%

Table 3: Comparison of patient characteristics across types of plant poison taken

		Type of plant poison taken			Total
		Datura	Oduvanthalai	Yellow Oleander	
Gender	Male	3 (75%)	2 (40%)	21 (41.2%)	26 (43.3%)
	Female	1 (25%)	3 (60%)	30 (58.8%)	34 (56.7%)
Age group	15-25	0 (0%)	0 (0%)	23 (45.1%)	23 (38.3%)
	26-35	3 (75%)	3 (60%)	25 (49%)	31 (51.7%)
	>35	1 (25%)	2 (40%)	3 (5.9%)	6 (10%)
Form of the plant taken	Seeds	4 (100%)	-	51 (100%)	55 (91.7%)
Form of consumption of seeds	Chewed	1 (25%)	-	25 (49%)	26 (47.3%)
	Grounded	3 (75%)	-	5 (9.8%)	8 (14.5%)
	Paste	0 (0%)	-	21 (41.2%)	21 (38.2%)
GIT symptoms	Abdominal pain	0 (0%)	1 (20%)	1 (2%)	2 (3.3%)
	Abdominal pain, diarrhoea	0 (0%)	1 (20%)	0 (0%)	1 (1.7%)
	Nil	0 (0%)	0 (0%)	12 (23.5%)	12 (20%)
	Vomiting	3 (75%)	1 (20%)	30 (58.8%)	34 (56.7%)
	Vomiting, abdominal pain	1 (25%)	1 (20%)	5 (9.8%)	7 (11.7%)
Cardiac symptoms (palpitation)	Vomiting, diarrhoea	0 (0%)	1 (20%)	3 (5.9%)	4 (6.7%)
	No	3 (75%)	5 (100%)	32 (62.7%)	40 (66.7%)
CNS symptoms (altered sensorium)	Yes	1 (25%)	0 (0%)	19 (37.3%)	20 (33.3%)
	Altered sensorium	4 (100%)	2 (40%)	2 (3.9%)	8 (13.3%)
Respiratory symptoms	Nil	0 (0%)	3 (60%)	49 (96.1%)	52 (86.7%)
	Dyspneic	0 (0%)	4 (80%)	3 (5.9%)	7 (11.7%)
	Nil	4 (100%)	1 (20%)	48 (94.1%)	53 (88.3%)

Table 4: Comparison of electrocardiographic changes across types of plant poison taken

		Type of plant poison taken			Total
		Datura	Oduvanthalai	Yellow Oleander	
ECG changes	No	3 (75%)	2 (40%)	17 (33.3%)	22 (36.7%)
	Yes	1 (25%)	3 (60%)	34 (66.7%)	38 (63.3%)
Sinus bradycardia	No	4 (100%)	5 (100%)	36 (70.6%)	45 (75%)
	Yes	0 (0%)	0 (0%)	15 (29.4%)	15 (25%)
ST T changes	No	4 (100%)	2 (40%)	43 (84.3%)	49 (81.7%)
	Yes	0 (0%)	3 (60%)	8 (15.7%)	11 (18.3%)
Exit block/sinus arrest	No	4 (100%)	5 (100%)	48 (94.1%)	57 (95%)
	Yes	0 (0%)	0 (0%)	3 (5.9%)	3 (5%)
Premature atrial contractions	No	4 (100%)	5 (100%)	49 (96.1%)	58 (96.7%)
	Yes	0 (0%)	0 (0%)	2 (3.9%)	2 (3.3%)
AV dissociation	No	4 (100%)	5 (100%)	48 (94.1%)	57 (95%)
	Yes	0 (0%)	0 (0%)	3 (5.9%)	3 (5%)

First-degree AV block	No	4 (100%)	5 (100%)	47 (92.2%)	56 (93.3%)
	Yes	0 (0%)	0 (0%)	4 (7.8%)	4 (6.7%)
Second-degree AV block	No	4 (100%)	5 (100%)	43 (84.3%)	52 (86.7%)
	Yes	0 (0%)	0 (0%)	8 (15.7%)	8 (13.3%)
Third-degree AV block	No	4 (100%)	5 (100%)	48 (94.1%)	57 (95%)
	Yes	0 (0%)	0 (0%)	3 (5.9%)	3 (5%)
Junctional rhythm	No	4 (100%)	5 (100%)	48 (94.1%)	57 (95%)
	Yes	0 (0%)	0 (0%)	3 (5.9%)	3 (5%)

Table 5: Comparison of mean parameters across types of plant poison taken

	Type of plant poison taken		
	Datura	Oduvanthalai	Yellow Oleander
Age	32.25 ± 4.11	34.20 ± 6.30	26.55 ± 5.69
No. of seeds/leaves taken	7.75 ± 1.26	8.80 ± 2.59	5.22 ± 2.18
The time interval from poisoning to admission (hours)	5.75 ± 2.87	4.80 ± 2.39	4.63 ± 2.79
ICU stay	4.25 ± 0.50	7.00 ± 1.41	2.88 ± 1.45
Total hospital stay	5.25 ± 0.50	8.20 ± 1.64	3.69 ± 1.49

Table 6: Comparison of mean lab parameters across types of plant poison taken

	Type of plant poison taken		
	Datura	Oduvanthalai	Yellow Oleander
SBP	136 ± 11.43	135.20 ± 14.94	116.98 ± 17.16
DBP	88.50 ± 5.972	89.60 ± 6.23	76.43 ± 9.95
Pulse rate	113.25 ± 9.25	96.20 ± 19.54	68.55 ± 12.24
Respiratory rate	16.50 ± 0.58	18.0 ± 2.92	13.82 ± 1.41
SpO2	96.25 ± 2.06	95.40 ± 2.07	97.71 ± 1.39
QTc	384.75 ± 23.20	471.20 ± 16.53	388.59 ± 39.81
RBS	84.00 ± 25.07	105.00 ± 18.37	98.53 ± 8.98
Blood urea	31.00 ± 12.36	41.40 ± 6.11	30.04 ± 8.89
Serum creatinine	0.95 ± 0.13	1.34 ± 0.53	0.93 ± 0.17
Na+	140.75 ± 3.59	136.80 ± 4.66	133.82 ± 17.62
K+	4.45 ± 0.47	3.08 ± 0.51	5.11 ± 0.74
AST	55.25 ± 6.89	140.40 ± 88.66	74.06 ± 44.26
ALT	35.50 ± 6.66	93.20 ± 60.92	48.59 ± 22.99
LDH	220.25 ± 128.98	234.80 ± 69.31	172.90 ± 94.76
CPK	82.75 ± 35.04	147.00 ± 63.41	137.08 ± 102.14
CKMB	41.50 ± 36.39	74.00 ± 22.64	61.88 ± 46.56
ABG (blood HCO3-)	26.50 ± 3.11	16.20 ± 1.64	25.71 ± 1.90

Table 7: Comparison of mean parameters with ECG changes in oleander seed poisoning

	ECG changes		Unpaired t-test P value
	Yes	No	
The time interval from poisoning to admission (hours)	4.92 ± 2.99	3.36 ± 2.28	0.05
No. of seeds taken	6.55 ± 2.18	4.18 ± 2.08	<0.001
K+	4.82 ± 0.87	4.31 ± 0.71	0.021
Total hospital stay	4.76 ± 1.60	3.14 ± 2.05	0.001

DISCUSSION

Three types of plant poisoning were commonly reported in our region. They are yellow oleander, Oduvanthalai and Datura. They are most commonly found in the age group below 35 (54 patients). This causes increased morbidity and mortality in the working community. Out of 60 cases, 26 were males and 34 were females. Compared to other studies, the female preponderance cannot be accounted for in our study.

Yellow oleander seed poisoning is present in a younger age group (26.55±5.69), which is similar to De Silva HA et al. series,^[5] as compared to older age of presentation in Oduvanthalai (34.20±6.30) and datura poisoning (32.25±4.11). Yellow oleander poisoning is most commonly seen in the 26-35 age group, followed by the 15- 25 age group.

Regarding the symptoms of plant poisoning, GIT symptoms are the most common manifestation, followed by cardiac symptoms. It may be due to toxin-induced gastritis. In GIT Symptom analysis, vomiting is the main clinical symptom. It is present in 58.8% of patients who consumed oleander and was found in 75% of datura-consumed patients. Altered sensorium is the most frequent manifestation of datura poisoning, and it is due to atropine effects on the CNS. Dyspnoea is present in 4 patients of Oduvanthalai poisoning (80%), possibly due to ARDS.

Unlike previous studies, ECG changes were predominantly found in yellow oleander poisoning. Sinus bradycardia is the most common ECG finding (25%), followed by ST T changes and second-degree AV block. ECG changes were present in 34 cases of yellow oleander poisoning (66.7%), and ECG changes were not present in 17 cases of yellow

oleander poisoning. ECG changes were present in 3 cases of Oduvanthalai poisoning and one case of Datura poisoning.

In our study, sinus bradycardia was the most common ECG change, followed by ST T changes and 2nd degree AV block. The cardiac glycosides inhibit the Na⁺/K⁺ ATPase pump, increasing intracellular Na⁺ and Ca²⁺. Intracellular hyperkalemia leads to spontaneous depolarisations and increased arrhythmogenicity. Zamani et al.^[6] published the incidence of various arrhythmias in yellow oleander poisoning. The incidence of arrhythmias is similar to our study. Hence, ECG monitoring is important in yellow oleander poisoning to detect arrhythmias at the earliest and prevent death. Among 60 patients, 48 patients had a suicidal intention, and 12 patients had accidentally ingested.

Comparing various modes of consumption of seeds of plant poisons showed that the chewed form was the most common mode of consumption, accounting for 47.3% in our study. A statistically significant association between the mode of consumption and the outcome of poison ($p=0.002$).

The systolic BP and Diastolic BP were found to be on the lower side in cases of oleander seed poisoning compared to datura and Oduvanthalai poisoning. This may be due to the Cardiotoxic effect of oleander seed poisoning. The mean pulse rate of oleander seed poisoning was around 68 ± 12 , whereas, in Datura poisoning, it was 113.25 ± 9.25 . The myocardial depressant effect reduced the mean pulse rate for oleander seed poisoning. The respiratory rate was normal in oleander and datura-ingested patients, whereas it was elevated at 18 ± 2.92 in the case of Oduvanthalai poisoning. It may be due to renal tubular acidosis-related hyperapnea or may be due to ARDS. QTc prolongation was evident in Oduvanthalai patients (471.20 ± 16.53) ms, also seen in the Mohan et al.^[7] study and was statistically significant. RBS were near normal in all groups. There was a slight elevation of urea and creatinine in Oduvanthalai poisoning compared to the other two. Hypokalemia was present in Oduvanthalai poisoning patients (3.08 ± 0.51) due to distal RTA and hyperkalemia in yellow oleander poisoning. Liver enzymes (AST, ALT) were elevated in a case of Oduvanthalai poisoning patients, which may be a spectrum of MODS. There was a reduction in serum bicarbonate levels in Oduvanthalai poisoning (16.20 ± 1.64) due to acidosis.^[8,9]

ICU stay was found to be increased in Oduvanthalai poisoning (7.00 ± 1.41) as comparable to yellow oleander (2.88 ± 1.45) and datura poisoning (4.25 ± 0.50). Total stay also increased in Oduvanthalai poisoning (8.20 ± 1.64), then in Datura (5.25 ± 0.50) and Yellow oleander (3.69 ± 1.49) due to Multiorgan dysfunction, which is present in Oduvanthalai poisoning. Long ICU stay may have an impact on morbidity. Total hospital stay also increased in Oduvanthalai poisoning, comparable to the Mohan et al.^[7] study.

In our study, the mean time interval from oleander poisoning to the development of ECG changes was found to be 4.92 ± 2.99 hrs, which was statistically significant compared to those who didn't develop ECG changes. The patients who reached the hospital early had less morbidity, and this is because oleander toxicity is time-bound, and toxicity increases with time elapsing after ingestion.

The mean number of seeds consumed by the patients with oleander seed poisoning who developed ECG changes was 6.55 ± 2.18 , which was statistically significant, proving that consuming more seeds increases Cardiotoxicity. The lethal dose may be between 4-8 seeds, similar to studies conducted by Sreeharan et al. and Saravanapavanandhan et al. Paste form has more toxicity.^[10,11]

The relationship between ECG changes and serum potassium was analysed in our study. The mean serum potassium with ECG changes was 4.82 ± 0.87 , which was statistically significant. This is comparable with the Zamani et al. and Lokesh et al. study.^[6,12]

Morbidity and mortality in oleander glycoside toxicity are due to cardiac effects. Hence, those with ECG changes needed pharmacological intervention. Drugs used by us are Inj. Atropine and orciprenaline, and we also used temporary cardiac pacing. There was no mortality in our oleander seed poisoning group.

In Oduvanthalai poisoning, as there was no antidote, we treated them symptomatically by treating dyselectrolytemia, oxygen support, and renal failure, if necessary, by renal replacement therapy. Cleistanthus collinus is a common cause of plant poisoning in rural south India. It is associated with high mortality, and no definitive antidote exists. Consumption of the aqueous extract of the plant is associated with greater mortality. Further research is required to identify putative toxic molecules in C. collinus and definitive antidote. Without a definitive antidote, the management of C. collinus poisoning remains symptomatic and supportive. In datura poisoning patients, delirium was managed by benzodiazepine and symptomatic management. Future research should be focussed on the detailed pathophysiology and toxicity of these plants. Despite many cases, an antidote has not been found for oleander seed poisoning. Digoxin-specific antibody fragments are very expensive. Hence, research should be made to develop an alternate antidote for oleander seed poisoning

CONCLUSION

Electrocardiographic abnormalities and dyselectrolytemia were most common in yellow oleander poisoning. Oduvanthalai poisoning presents with renal tubular acidosis, hypokalemia and ARDS. Datura poisoning generally presents with CNS manifestations. Early intervention is required to prevent the morbidity of the patients. Studies

regarding plant poisons were minimal; hence, more research is needed to reduce morbidity and mortality. The local authorities must restrict easy access to these lethal plant plants to decrease the incidence of plant poisoning. All physicians should be aware of the initial management of the most common poisons in their region and their acute management.

Limitations

The study faced several limitations, including an uneven distribution of cases of plant poison, making it challenging to discern individual characteristics. Additionally, there was a lack of follow-up for the patients, and the research was conducted at a single centre. Future studies should consider multicentric approaches to obtain a more accurate understanding of the prevalence of specific plant poisons.

REFERENCES

1. Nongpiur A, Tesia SS, Vijaya R. Pattern of deliberate self-harm seen at a tertiary teaching hospital in Meghalaya, India. *Open J Psychiatry Allied Sci* 2018;9:34.
2. Asawari R, Atmaram P, Bhagwan K, Priti D, Kavya S, Jabeen GA. Toxicological pattern of poisoning in urban hospitals of Western India. *J Young Pharm* 2017;9:315–320.
3. Knipe D, Williams AJ, Hannam-Swain S, Upton S, Brown K, Bandara P, et al. Psychiatric morbidity and suicidal behaviour in low- and middle-income countries: a systematic review and meta-analysis. *PLoS Med* 2019;16: e1002905.
4. Jegaraj MK, Mitra S, Kumar S, Selva B, Pushparaj M, Yadav B, et al. Profile of deliberate self-harm patients presenting to Emergency Department: a retrospective study. *J Family Med Prim Care* 2016;5:73–76.
5. De Silva HA, Fonseka MMD, Pathmeswaran A, Alahakone DGS, Ratnatilake GA, Gunatilake SB, et al. Multiple-dose activated charcoal for treatment of yellow oleander poisoning: A single-masked, randomised, placebo-controlled trial. *Lancet Lond Engl*. 2003;361:1935–8.
6. Zamani J, Aslani A. Cardiac findings in acute yellow oleander poisoning. *J Cardiovasc Dis Res* 2010;1:27–9.
7. Mohan A, Harikrishna J. *Cleistanthus collinus* poisoning. *Indian J Crit Care Med* 2019;23: S256-259.
8. Devaprabhu S, Manikumar S, David SS. Toxicology-epidemiology and prognostic profile of patients with *Cleistanthus collinus* poisoning. *Indian J Trauma Anaesth Crit Care*. 2007;8:642.
9. Shankar V, Jose VM, Bangdiwala SI, Thomas K. Epidemiology of *Cleistanthus collinus* (oduvan) poisoning: Clinical features and risk factors for mortality. *Int J Inj Contr Saf Promot*. 2009;16:223–30.
10. Sreeharan N, Putharasingam S, Ranjadayalan K, Satkurnathan K, Ganeshamoorthy J. Yellow oleander poisoning—clinical manifestations and prognostic criteria. *Jaffna Med J*. 1985;20:100-1.
11. Saravanapavanathan N, Ganeshamoorthy J. Yellow oleander poisoning- A study of 170 cases. *Forensic Sci Int*. 1988;36:247–50.
12. Lokesh S, Arunkumar R. A clinical study of 30 cases of acute yellow oleander poisoning (*Thevetia neriiifolia*). *J Curr Trends Clin Med Lab Biochem*. 2013;1:28–31.