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Epidemic Self-Poisoning with Seeds of *Cerbera manghas* in Eastern Sri Lanka: An Analysis of Admissions and Outcome

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Abstract

The use of plant fruits and seeds as a method of self-poisoning is common in South Asia, with most deaths being due to ingestion of (yellow oleander) seeds. Self-poisoning with the locally common *Cerbera manghas* (CM) fruit is prevalent in the Eastern Province of Sri Lanka. We carried out a retrospective study to determine the clinical manifestations, treatment and outcome of patients with *cerbera manghas* self-poisoning in Batticaloa teaching hospital. Data were collected retrospectively on all cases with *Cerbera manghas* self-poisoning from 1st January 2011 to 31st October 2013.There were 48 patients [mean age: 21(± 0.43) yrs], (male: female=35:13). Twenty four had ingested half of a seed (25 g). Most of the patients were symptomatic with vomiting (48), dizziness (24) and abdominal pain (20). Forty (83.3%) had cardiac arrhythmias that required transfer to the poisoning unit for specialized management. Severe cardiac toxicity was observed among patients (18) with fewer amount of seeds (25 g) compared to patients (8) with large amount of seeds (50 g). The 1st degree heart block (10, 20.8%), 2nd degree heart block (Type I:5, 10.4%, type II:12, 25.0%), complete heart block (10, 20.8%) and sinus bradycardia (5, 10.4%) were ECG findings. Fourteen (29.16%) had high serum potassium concentrations between 6.0 and 6.9 mmol/L; ten had life threatening hyperkalemia (>7.0 mmol/L). All patients were treated with multiple doses of activated charcoal. Temporary cardiac pacing was required in nine (18.7%) cases. There were eight deaths (16.7%), due to third-degree heart block and life threatening hyperkalemia. *Cerbera manghas* self-poisoning was common among young males in Batticaloa district of Sri Lanka's Eastern Province. Cardiac toxicity was observed in patients with fewer amounts of seeds. Patients presenting with complete heart block and severe hyperkalemia had great risk of mortality.

Key Words:

Plant poisoning; Suicide; Cardiac toxicity; *Cerbera manghas*

Introduction

While self-poisoning with Pesticides is the most common method of self-harm in Asia, self-poisoning with plant seeds or fruits is also common, especially in the South Asian region [1]. While most deaths follow ingestion of *Thevitia peruviana (*yellow oleander) seeds, other locally common plants are also implicated [2]. We noted that cases of self-poisoning with *Cerbera manghas* (sea mango, pink eyed cerbera, odollam tree) fruits, while being most commonly reported in Tamil Nadu and Kerala [3,4] are seen in the Eastern Province of Sri Lanka.

Cerbera manghas is a poisonous plant belonging to the Apocynaceae family notorious for its cardiac glycoside cardiotoxicity. *C. manghasis* called *diyakaduruin* Sinhala, *kattuarali* in Indian Tamil; *natchuchukkaiin* Sri Lankan Tamil. *Cerbera manghas* Linn (previously also *Cerberaodollam Gaertn*) is common along the coasts of south Asia and south East Asia, Northern Australia, and Polynesia. In Sri Lanka, especially along coastal regions, sea mango (*Cerbera manghas*) is a very common plant and readily accessible. It grows to a height of 5 to 10 metres and bears whorled branchlets with terminal lanceolate leaves. The fruit of the sea mango turns bright red at maturity and appear very much like the edible mango (*Mangifera* spp.) fruit. The latex present throughout the plant contains cardenolides (such as cerberin, neriifolin, and cerberoside) (Figure 1) that cause vomiting, cardiac dysrhythmias, and hyperkalemia via inhibition of the Na^+/K^+ATPase.

Figure 1: Cerbera odollam tree leaves and seeds.

Although widespread in Sri Lanka [5], and well known to be poisonous [6], only a one report has been published from the island.

However, there have been no publications reporting clinical data from poisoned patients.

The aim of this study was to evaluate the clinical manifestations, biochemical findings and outcome of management using currently available treatment in the poisoning unit of a tertiary care hospital in Eastern Sri Lanka.

Materials and Methods

Study population

Patients with *Cerbera manghas* poisoning admitted to tertiary care hospital in Eastern Sri Lanka from 1st January 2011 to 31st October, 2013 by were included using predesigned questionnaires, retrospectively.

Treatment of patients

Patients with *Cerbera manghas* poisoning were treated in medical wards and poisoning unit, tertiary care hospital in Eastern Sri Lanka with different treatment modalities such as activated charcoal, intravenous boluses of atropine/isoprenaline, temporary pacing and supportive measures depending on the clinical manifestations.

Electrocardiographic monitoring

12-lead standard electrocardiography (INNOMED Medical ECG machine) and 2-lead ECG monitoring were taken during the standard work up of each patient in this unit.

Blood samples

Five milliliters (5 ml) venous blood was collected from each patient. Serum was separated by centrifuging in a laboratory centrifuge at for three minutes after blood clotting and retraction at room temperature. Serum potassium (K^+) and Sodium (Na^+) were analyzed at the Department of Chemical Laboratory. Renal function including blood urea nitrogen [BUN], creatinine and liver function indices including SGOT, SGPT, PT and serum protein were analyzed in the clinical laboratory of Batticaloa teaching hospital using standard automated techniques.

Statistical analysis

Differences between the two groups (patients with significant arrhythmia vs. patients with insignificant arrhythmia) were analyzed with pair-wise comparisons. Baseline results are presented as counts and percentages and as mean ± SD for continuous variables. A P value <0.05 was considered significant.

Results

We noted 583 cases of poisoning from 1st January, 2011 to 31st October, 2013. Among these, 192 (32.93%) cases were due to Yellow Oleander poisoning, while 48 (8.23%) cases were due to *Cerbera manghas* poisoning. There were 48 patients [Mean age: 21 (± 0.43) yrs], (Male: Female=35:13) with *Cerbera manghas* self-poisoning. The great majority were in the 16-30 year age group (40, 83.3%). The ethnicity of the majority of patients was Tamil (42, 87.5) (Table 1). Twenty four (50%) of them had ingested half a seed (one seed weighted about 20 g) (Table 2).

Personal Characteristics		Number of cases (%)
Sex	Male	35 (72.9%)
	Female	13 (27.1%)
Ethnicity	Tamil	42, 87.5%),
	Muslims	6 (12.5%)
Age	<16yrs	1 (2.0%)
	16-30 years	40 (83.3%)
	31-45years	3 (6.25%)
	4-60years	3 (6.25%)
	>60years	1 (2.0%)

Table 1: Personal characteristics of patients.

Amount of seeds	No of cases (%)
10 g	24 (50%)
20 g	10 (20.8%)
40 g	8 (16.7%)
>40 g	6 (12.5%)

Table 2: Number of seeds ingested by patients.

Most of the patients were symptomatic with symptoms of cardiac glycoside toxicity including vomiting (48, 100%), dizziness (24, 50%) and abdominal pain (20, 41.6%). Abdominal pain and diarrhea alone was rare presentations. Neurological manifestations such as drowsiness and restless (8, 16.6%) were also associated. Cardiac dysrhythmias such as bradycardia or an irregular pulse were the examination findings. ECG findings showed 1st, 2nd, 3rd degree heart block and sinus bradycardia (Table 3). A normal ECG was found only in 6 patients. Atrial fibrillation was the tachyarrythmias. Mean serum potassium concentration was significantly higher in patients with significant cardiac arrhythmias that required specific management transfer to CCU and temporary pacemaker insertion.

ECG findings		No of cases (%)
1st degree heart block		10 (20.8%)
2nd degree heart block	Type I	5 (10.4%)
	Type II	12 (2%)
3rd degree heart block		10 (20.8%)
Sinus bradycardia		5 (10.4%)
Normal ECG		6 (12.5%)

Table 3: ECG findings of Patients.

Fourteen of them had higher serum potassium concentrations (6.4 (± 0.97) mmol/L). Ten patients had life threatening hyperkalemia [7.4 (± 0.17)]. All patients developed cardiac toxicity and hyperkalemia within 24 hrs of ingestion of seeds. These severe cardiac toxicity and

hyperkalemia were observed among patients (16, 33.3%) having half seed of ingestion. All other blood investigations including renal and liver markers are within normal range (Table 4).

Blood investigations		Range	No of cases (%)
Serum Electrolytes	Sodium	13-145 mEg/L	48 (100%)
	Potasium	<5 mEq/L	6 (12.5%)
		5-5.5 mEq/L	15 (31.25%)
		5.6-6.0 mEq/L	5 (10.4%)
		6.1-6.5 mEq/L	8 (29.16%)
		6.6-7.0 mEq/L	4 (8.33%)
		>7.0 mEq/L	10 (20.8%)
Liver enzymes	SGPT	<40 U/L	48 (100%)
	SGOT	12-20 U/L	48 (100%)
Blood urea		<30 mg	48 (100%)
Serum Creatinine		0.6-1.0 mg/dL	48 (100%)
PT/INR		1-1.3	48 (100%)

Table 4: Blood Investigations of patients.

All patients were treated with multiple doses of activated charcoal. Patients with bradyarrhythmias were treated with intravenous atropine (18, 37.5%) and intravenous infusions of isoprenaline (4, 12.5%). Temporary cardiac pacing was required in nine of them who had not responded to drug therapy. There were eight deaths who had both 3rd heart block and high serum potassium concentrations (>7.0), (Figure 2; ECG tracings). Three died before definitive treatment could be instituted. The majority of patients with cardiac toxicity had an uneventful recovery. Multiple doses of activated charcoal alone were safe and adequate in most cases in our study. However it was not statistically proven. All patients with hyperkalemia were managed only with insulin-dextrose (10 units Insulin with 50 ml 50% Dextrose regime).

Features of severe toxicity such as persistent vomiting, severe abdominal pain, neurological signs and persistent hyperkalemia were significantly associated with high risk of mortality and morbidity (P<0.05). The risk of cardiac toxicity was observed with patients with ingestion of less amount of seeds (half seed).

Discussion

A previous study in 2000/2001 showed that *Cerebera manghas* is the cause for 20% deaths from plant poisoning and 4.4% of all self-harm deaths in this hospital on the eastern coast of Sri Lanka [7]. It was the second commonest plant poisoning in Eastern Sri Lanka next to Yellow Oleander Poisoning in our study period. Review of our study confirmed that *Cerbera manghas* plant poisoning produces clinical features typical of cardenolide poisoning [8]: in particularly; vomiting, cardiac dysrrhythmias and hyperkalemia. Electrocardiographic changes are the most obvious marker of *Cerbera manghas* toxicity [9]. The most common ECG abnormality is bradycardia, which may be sinus bradycardia, sinus arrest or exit block, or AV nodal dissociation (third degree heart block).

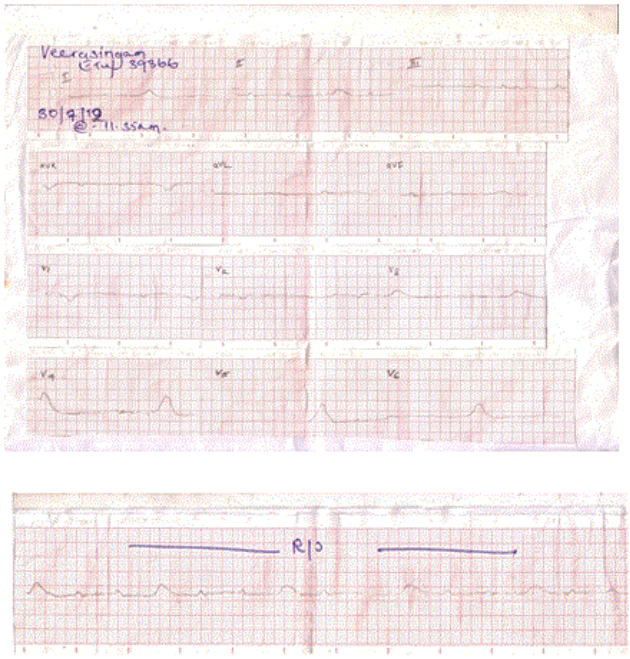

Figure 2: ECG tracing of a patient; 54 year old man presented with severe vomiting, abdominal pain and restless following ingestion of half seed of *cerbers manghas* after 8hrs of ingestion. His ECG showed complete heart block. His serum Potassium was 7.9 mmol/dL. He died within 2 hrs of hospital admission even before pacing was instituted.

Management involved gastric lavage, multiple doses of activated charcoal, administration of atropine to counter bradycardia and Insulin-Dextrose to treat hyperkalemia. The effect of atropine on outcomes in cases of bradyarrhythmia has not been formally assessed. However a study from Trivandrum reported a poor outcome if bradycardia did not respond to atropine and atropine was not effective [10].

Cardic arrhythmias and persistent hyperkalemia are important predictable mortality risk for *Cerbera manghas* plant poisoning. There were eight deaths (16.67%) due to third-degree heart block and life threatening hyperkalemia in our study. These deaths were observed among patients having half seed of ingestion. These severe cardiac toxicity and hyperkalemia were observed among patients having half seed of ingestion and not vomiting seeds immediately. This may be due to rapid absorption of seeds in gastrointestinal tract following ingestion. The patients taking more seeds vomited immediately within 2 hrs. As a result, they have less cardiac toxicity compared those having lees amount of ingestion of seeds.

Despite its relative uncommon prevalence compared to Yellow Oleander poisoning, it is much feared by the medical staff due to high case fatality and the lack of pacemakers or anti-digoxin Fab for therapy [11].

A limitation of our study is also same as in previous study [7] that neither blood nor gastric contents were tested for *Cerbera manghas*

cardenolides to prove ingestion. The responsible fruit ingestion was identified only from history from patient and relatives. However, *Cerbera manghas* is very well known in the region and easily recognized, increasingly the like hood that the ingested fruit would be accurately identified.

Conclusion

Cerbera manghas self-poisoning was common among young males in Batticaloa district of the Eastern Province. The cardiac toxicity observed within 24 hrs of ingestion of seeds. The severe toxicity was associated with less amount of seeds. Most patients had specific symptoms of *Cerbera manghas* poisoning. AV conduction defects are common. Patients presenting with features of severe toxicity such as persistent vomiting, severe abdominal pain, neurological signs and persistent hyperkalemia has higher risk of mortality.

Recommendations

Cerbera manghas poisoning is the second commonest plant poisoning in Eastern Sri Lanka but lack of resources can significantly impair the successful management in the hospital setting. We would like to emphasize the importance of equipping these hospitals with adequate resources, e.g: pacemakers for therapy *Cerbera manghas* poisoning to prevent adverse outcomes.

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