

## Research Article

# Risk assessment in poisoning with special reference to odollam and organophosphorous compounds

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## ABSTRACT

**Background:** Poisoning with organophosphorous compounds, odollam and other substances are common. Affected persons present with a wide variety of clinical features. Prognostic indicators of high mortality are important for management and to decide on intensive care unit admission.

**Methods:** Admissions due to poisonings from April 2015 to March 2016 were assessed. The clinical features, electrocardiographic changes and laboratory investigations of 915 patients were obtained and statistically analysed.

**Results:** The most commonly ingested poisons were odollam (26%) and organophosphorous compounds (22%). The overall mortality was 22%. Mortality was highest in organophosphorus compounds (38%) and odollam (33%) compared to other causes. Hyperkalemia, low systolic blood pressure, consumption of poison in powder or paste form, delayed presentation, bradycardia and extensive chest signs correlated with mortality in odollam and organophosphorous compound poisoning.

**Conclusions:** Poisoning with odollam or organophosphorous compounds is an increasingly common life threatening condition. Markers of mortality identified in this study were incorporated into a simple scale for assessment of risk, namely the PoPPER scale: potassium level >5.5 mE/L, systolic blood pressure <90 mmHg, paste/powder form of poison ingestion, extremely late presentation, rate of QRS <40/min or impending respiratory paralysis.

**Keywords:** Odollam, Organophosphorous, Mortality

## INTRODUCTION

Deliberate self-harm by poisoning takes an alarmingly increasing number of young lives in the state of Kerala each year. One reason for this is easy accessibility to poisons in the general population.

This is especially true in Alappuzha and its surrounding areas like Kuttanad where the major occupations are farming and fishing. Organophosphorous compounds are a cheap, commonly used and widely available pesticide that is potentially fatal when ingested.<sup>1</sup>

Cerbera odollam is a small tree that grows commonly in Kerala, called colloquially as othalanga.<sup>2</sup> Its fruits look like small mangoes and contain cerberin, a cardiac glycoside.<sup>2,3</sup> Ingestion of odollam kernel by adults can be fatal.<sup>2</sup> A commonly used method of suicide, it produces nausea, vomiting, abdominal pain, lethargy and cardiotoxic effects.<sup>2</sup>

There is great variability in the individual response following the ingestion of these above listed and other poisons. The objective of the study was to identify simple risk markers of high mortality that would be easy to apply to all patients at presentation without requiring any costly

investigation. These would serve to direct those patients to intensive care who need it the most, thus ensuring efficient, effective and life-saving use of limited critical care unit resources and personnel.

## METHODS

Prospective observational analytical study was done at Department of General Medicine, Government TD MC Hospital, Vandanam, Alappuzha, Kerala, India from April 2015 to March 2016.

### Inclusion criteria

All patients between the ages of 15 and 45 years with a history of ingestion of poison admitted in medical wards.

### Exclusion criteria

- Established coronary artery disease.
- Other known structural heart disease.
- Known pre-existing electrocardiographic abnormalities.
- History of cardiac chest pain in 48 hours prior to admission.
- Diabetes mellitus.
- Hypertension.

Patients who satisfied inclusion criteria were included in the study. Standard protocols and established clinical guidelines were followed for patient evaluation and management. Data obtained included age, gender, nature and amount of poison, time elapsed between ingestion and admission to hospital, clinical features,

electrocardiographic findings, cardiac enzyme levels, outcome and mortality. This data was entered into the proforma designed for the study.

### Statistical analysis

The prognostic significance and predictive value of various parameters and their relationship to in-hospital mortality and survival to discharge was assessed using Fisher's exact test and chi-square test.

## RESULTS

915 patients were included in the study. The most commonly ingested poisons were odollam (26%) and organophosphorous compounds (OPC) (22%) (Table 1). There was a female preponderance with a male:female ratio of 1:1.4 in the number of poisonings. Females predominated when the poison consumed was odollam, rat killer, sedatives, paracetamol, miscellaneous medications and ant killer. Males led in consumption of organophosphorous compounds, other pesticides and methanol, almost always mixed with ethanol. The overall mortality was 22%. Mortality was highest in organophosphorous compounds and odollam compared to other causes. There was no significant gender difference in mortality. Hyperkalemia was found in 143 (60%) odollam consumers at presentation (Table 2). 79 (55% of hyperkalemics) were corrected to normokalemia. Untreated or incompletely treated hyperkalemia carried high mortality (60%).

Hypotension (systolic BP <90 mmHg) at presentation correlated significantly with mortality for odollam (Table 3) and OPC (Table 8).

**Table 1: Profile of poisoning-April 2015 to March 2016.**

Nature of poison	No. of poisonings Total=915	Percentage	Male 384 (42%)	Female 531 (58%)	No. of deaths 197 (22%)
Odollam	238	26	93	145	79 (33%)
OPC*	201	22	183	18	77 (38%)
Other pesticides	119	13	78	41	23 (19%)
Rat killer	92	10	26	66	12 (13%)
Sedatives	82	9	25	57	3 (3.7%)
Paracetamol	82	9	33	49	2 (2.4%)
Multiple drugs**	78	8.5	26	52	0
Ant killer	9	1	0	9	0
Methanol	5	0.5	5	0	1 (20%)
Others***	5	0.5	3	2	0
Unknown substance	4	0.5	1	3	0

\*Organophosphorous compound, \*\*Multiple drug overdose - antibiotics, antacids, antihistaminics, antipsychotics, \*\*\*toilet cleaner, paint thinner, acid

Consumption of the inner kernel of the odollam fruit was significantly more fatal than consumption of the outer fruit alone (Table 4). Also, consuming the inner kernel of the odollam fruit after grinding it into paste increased the chance of mortality (Table 5) when compared to swallowing small pieces as such. 45% of patients with

paste ingestion died. Ingestion of the paste of 1½ or 2 kernels was uniformly fatal when the patient presented after 8 hours, which occurred in 11 patients. In contrast the mortality was less in those who consumed the kernel as such (30%) and still less in those who had consumed

only the outer portion of the fruit excluding the kernel (12%). Death was due to cardiac arrest in all cases.

**Table 2: Potassium levels in odollam consumers.**

K <sup>+</sup> level (mE/L)	Deaths	Survivors
>5.5 (Remained untreated)	39	25
3.5 to 5.5 (Initially hyperkalemic, treated to normokalemia)	27	52
3.5 to 5.5 (At presentation)	18	77

Hyperkaleemics vs normokaleemics : Fisher’s exact test P < 0.0001,  $\chi^2 = 23.694$ , P < 0.0001, statistically significant.

**Table 3: Systolic blood pressure in odollam consumers at presentation.**

Systolic BP at presentation (mmHg)	Deaths	Survivors
< 90	40	28
>90	39	131

Fisher’s exact test P < 0.001,  $\chi^2 = 26.6$ , P < 0.0001, statistically significant.

**Table 4: Odollam consumption-inner kernel/ outer fruit.**

Mode of ingestion of odollam	Deaths	Survivors
Inner kernel	74	123
Outer fruit alone	5	36

Fisher’s exact test P < 0.001,  $\chi^2 = 8.738$ , P = 0.0031, statistically significant.

**Table 5: Odollam inner kernel consumption-paste or powder/pieces.**

Inner kernel mode of ingestion	Deaths	Survivors
Paste or powder	45	55
Small pieces swallowed as such	29	68

Fisher’s exact test P < 0.0391,  $\chi^2 = 4.167$ , P = 0.04, statistically significant.

**Table 6: Time elapsed between ingestion of odollam and presentation to hospital.**

Time (hours)	Deaths	Survivors
>8H	46	66
<8H	33	93

Fisher’s exact test P = 0.018,  $\chi^2 = 5.269$ , P < 0.02, statistically significant.

Delayed presentation more than 8 hours following ingestion worsened the prognosis and increased mortality in odollam (Table 6) and OPC (Table 9). In odollam ingestion, deaths usually occurred within the first 36 hours following admission (75%), of which 40% occurred in the first 12 hours.

**Table 7: Electrocardiographic patterns in odollam poisoning.**

Normal sinus rhythm	15
Sinus bradycardia	201
Junctional rhythm	56
Atrial fibrillation with slow ventricular rate	78
1 <sup>st</sup> degree AV Block	150
2 <sup>nd</sup> degree type 1	127
2 <sup>nd</sup> degree type 2	139
Advanced AV block	102
Complete heart block	84
ST T changes	171
QRS rates < 40/min or RR pauses > 40 mm	82

**Table 8: Systolic BP in organophosphorous consumers at presentation.**

Systolic BP (mm Hg)	Deaths	Survivors
<90	50	47
>90	27	77

Fisher’s exact test P < 0.001,  $\chi^2 = 12.84$ , P = 0.0003, statistically significant.

**Table 9: Time elapsed between ingestion of organophosphorous compound and presentation to hospital.**

Time (hours)	Deaths	Survivors
>8H	48	57
<8H	29	67

Fisher’s exact test P = 0.03,  $\chi^2 = 4.467$ , P = 0.03, statistically significant.

ECG findings were maximum and most varied in odollam poisoning (Table 7). Patients had rapidly fluctuating rhythms from one arrhythmia to another and multiple arrhythmias occurred in the same patient over the course of a single admission in both survivors and fatalities. Hence no particular arrhythmia could be singled out as a marker of poor survival. Patients who were admitted with a so called benign arrhythmia like Mobitz type 1 advanced to complete heart block over a varying time span of 1 to 48 hours. However those patients who died invariably developed slow QRS rates <40/min or RR pauses >40 mm (recorded in 69% of deaths).

Cardiac enzymes including troponins and CK MB were not elevated in any case of poisoning, even in those with severe bradyarrhythmias. Thus evidence of myocarditis induced by odollam or other poisons was not obtained in this study. Hence troponins or cardiac enzyme levels are not useful in predicting prognosis.

Atropine improved heart rates in odollam poisoning but hindered temporary pacing in 6 cases due to atropine psychosis.

None of the patients who were put on temporary pacemaker (56) died. In OPC poisoning, the cardiac findings were normal sinus rhythm (59%), sinus bradycardia (41%) and hypotension (21%). Hypotension at presentation correlated significantly with mortality (Table 8) but sinus bradycardia did not.

In OPC poisoning, the most common cause of death was respiratory failure (64%), followed by bronchorrhea (30%). There was gasping pattern of shallow breathing, frothing from mouth and extensive chest signs in the form of bilateral crepitations.

## DISCUSSION

This study found a high incidence of cases admitted with OPC (22%). This correlated well with the studies conducted by Maharani et al which had 58%, the study by Ramesha et al with 36% and the study by Paranthakan et al with 44%.<sup>4-6</sup> The highest mortality occurred in OPC (38%) which was similar to the 35% mortality reported by Celine et al and the 58% mortality reported by Paranthakan et al.<sup>7</sup>

Lesser overall mortality in this study (22%) compared to the study conducted by Paranthakan et al, which showed a mortality of 38%, might be due to smaller amounts of poisons consumed by females and earlier seeking of health care.

The highest mortality occurred in OPC (38%) and odollam (33%) poisonings, which were analysed in this study. The third highest percentage occurred with methanol (20%), followed by other poisons, but the numbers were too small to allow meaningful inferences.

As this study aimed to look at ECG changes as prognostic risk factors, the particular inclusion and exclusion criteria were chosen to exclude those patients who might develop ECG changes as a result of acute coronary syndromes and who could have confounded the results. As per the above results, the markers that correlated significantly with mortality were incorporated into a simple scale of risk assessment for patients admitted with odollam or organophosphorous compound poisoning:PoPPER.

- Po = Potassium level > 5.5 mE/L.
- P = Pressure ( Systolic Blood Pressure < 90 mmHg).
- P = Paste/powder form of poison consumption.
- E = Extremely late presentation (>8 H after ingestion).
- R = Rate of QRS <40/min or RR ≥40 mm in the case of odollam  
Or
- Respiratory paralysis impending with copious secretions in the case of OPC

If any of the above is present it is given a score of 1. Any score above 0 is a red flag signal that indicates a need for

aggressive management in the form of intensified antihyperkalemic measures, temporary pacing, mechanical ventilation and/or intensive care unit admission for the same.

## CONCLUSION

Poisoning with odollam or organophosphorous compounds is a life threatening condition that is becoming increasingly common in today's stress filled materialistic world. Identifying early signs of high risk and prompt intervention can help save lives. Signs that correlated with high mortality in this study were hyperkalemia, low systolic blood pressure, consumption of poison in powder or paste form, delayed presentation, bradycardia and extensive bilateral chest crepitations with gasping pattern of respiration.

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