Section: Emergency Medicine

Original Research Article

CLINICAL PROFILE AND OUTCOMES OF PATIENT ADMITTED WITH YELLOW PHOSPHOROUS POISONING IN THE EMERGENCY WARD OF GOVERNMENT MEDICAL COLLEGE PUDUKKOTTAI

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Abstract

Background: Yellow phosphorus is extremely toxic and can be fatal in small amounts, leading to suicide due to Ratol ingestion. It is corrosive and damages any tissue in contact, leading to liver damage and acute liver failure. This study is aimed to study the clinical profile of the patients with Ratol poisoning and their association with the outcome of the patients. **Materials and Methods:** The study included a cohort of 60 patients divided into two groups: This hospital-based prospective study was conducted in the Emergency Ward of Government Medical College Pudukkottai, Tamilnadu, for twelve months. Sixty-five patients admitted to TAEI with yellow Phosphorus poisoning were selected within 24 hrs of ingestion. Blood investigations like Complete haemogram, serum urea, serum creatinine, random blood sugar, Liver Function Test, prothrombin time, APTT, serum electrolytes, INR, and ECG are reordered. **Results:** Most patients (86.15%) reached the hospital within 6 hours, and 72.31% exhibited symptoms, with nausea and abdominal pain being the most common. Treatment outcomes showed that 90.77% of patients were successfully discharged, while six patients died. Gender did not significantly influence the outcomes, but age and the quantity of poison consumed were significantly associated with the outcomes. Among the hospitalised patients, 18 were discharged after over two weeks, while three could not be saved despite extended hospitalisation. All blood parameters like SGOT, SGPT, INR, and Prothrombin time have a statistically significant difference at admission and after two weeks for the deceased. **Conclusion:** In conclusion, age, poison amount, and treatment delay affect outcomes, and liver damage is the main complication.

INTRODUCTION

Phosphorus, a non-metallic irritant, is used in various sectors like firecrackers, match industries, rodenticides, and fertilisers.[1] In India, around 70% of rural households depend primarily on agriculture for their livelihood.[2] The unchecked growth of rodents like rats around the household in farms and fields can be problematic because of the potential spreading of diseases, food supply disturbance and consequential economic loss. Hence rodenticides are widely marketed in India, and various preparations are available, such as yellow phosphorus, Arsenic trioxide, Barium carbonate, α-naphthylthiourea etc., in the form of powders, pastes, pellets, cereal baits, or blocks. Yellow phosphorus is extremely toxic and can be fatal in very small amounts.[3,4] It is no longer registered as a rodenticide in the United States, whereas in India, it is available as Ratol paste and powder. This preparation is cheap and easily available in the open market and online e-commerce sites, contributing to the frequently reported cases of suicide due to Ratol ingestion. Accidental Yellow Phosphorus poisoning is common in children, while suicidal ingestion is common among adults.[5,6] Accidental consumption in children occurs since it looks like toothpaste. Since the initial phase is asymptomatic, some patients do not reveal ingesting

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Ratol paste and present late to a health care facility. It is one of the common causes of emergency health care admission; one-third of the patients are admitted to the intensive care unit.

Yellow phosphorus is a severe local and systemic toxin affecting the gastrointestinal, hepatic, cardiovascular and renal systems. It is corrosive and damages any tissue it comes into contact.[7,8] It causes mortality, especially after 3-4 days of consumption when liver damage begins, and the most dreaded complication of acute liver failure with coagulopathy happens. Clinical manifestation occurs in three stages. The first stage occurs with gastrointestinal symptoms like nausea and vomiting without laboratory abnormalities. The second stage occurs 24-48 hrs after ingestion, characterised by rising transaminases, although the patient may not be symptomatic.

In a few cases, it progresses to the third stage, characterised by acute liver failure with coagulopathy and encephalopathy, which may be fatal. It damages the liver by depleting glutathione stores. N-Acetyl cysteine (NAC) stimulates glutathione synthesis and enhances glutathione transferase activity. NAC has other beneficial effects like anti-inflammatory, ionotropic and vasodilatory effects. Early and excellent supportive care is key to reducing morbidity and mortality. Studies are available regarding the magnitude of the effects of different rodenticides in humans. This study is aimed to study the clinical profile of the patients with Ratol poisoning and their association with the outcome of the patients.

MATERIALS AND METHODS

This hospital-based prospective study was conducted in the Emergency Ward of Government Medical College Pudukkottai, Tamilnadu, for twelve months. Sixty-five patients admitted to TAEI with yellow Phosphorus poisoning within 24 hrs of ingestion were selected for the study.

Inclusion Criteria

Patients admitted with a history of yellow phosphorous poison (Ratol Paste) consumption were included.

Exclusion Criteria

Patients who have ingested other substances besides yellow phosphorous and those known to have preexisting liver disease. Patients with chronic kidney disease, mixed poisons, and heart disease absconded within 24 hrs of admission, and patients on chemotherapy or steroids were excluded.

Data collection methods consist of approval from the institutional ethics committee, which was taken before undertaking the study. Participants willing for the study were selected after getting informed written consent from them. Detailed history taking, including history were taken clinical and laboratory examinations, was made as per the study tool (pre-designed study Performa) for data collection.

Detailed histories were taken from the patients with particular importance to bleeding tendencies and gastrointestinal symptoms. Blood investigations like Complete haemogram, serum urea, serum creatinine, random blood sugar, Liver Function Test, prothrombin time, APTT, serum electrolytes, INR, and ECG are reordered. Treatment was initiated with gastric lavage and maintenance of hydration with intravenous fluids.

All patients were treated with Inj. Vitamin K 10 mg IM OD, Inj. Ondanserton 4 mg IV BD, Inj. Ranitidine 50 mg iv BD, Inj N acetylcysteine 150mg/kg in 200 ml of 5% D over 1 hour, Then 50 mg/kg in 500 ml of 5% D over 4 hours, Then 100 mg/kg in 2 pints of 5% D over 18 hours. In case of acute liver failure, patients were treated with Inj. Hepamerz and FFP.

Statistical Analysis

All data were entered in the Excel sheet, and statistical analysis was performed using statistical software. The demographic data were expressed in frequency and percentage. A chi-square test was used for categorical variables, and a p-value < 0.05 is considered statistically significant. The Wilcoxon sign rank test analysed the clinical parameters, a non-parametric test, as the sample size was small. This test was done after checking for normality with the help of the Shapiro-Wilk test.

RESULTS

Among 65 patients, 37 (57%) were males and 28 (43%) were female, and the mean age was 30.27 ± 10.84.

27 (42%) of the study participants were in the age group of 21-30 years, constituting the maximum number of study participants consuming rat poison. The minimum age was 14, while the maximum age was 65 years.

Most patients were 29 (44.62%) skilled workers, followed by 15 unemployed (23.08%). 36 (55.38%) were married; the maximum consumed was 45 grams, while the minimum was 7 grams. The mean quantity was 22.6 grams, with a standard deviation of 9.49 grams.

Table 1: Demographic data of the study

		Frequency	Percentage
Gender	Male	37	57
	Female	28	43
Age group	≤ 20 years	12	18
	21-30 years	27	42
	31-40 years	17	26
	41-50 years	5	8

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	>50 years	4	6
Occupation	Student	6	9.23
	Skilled Worker	29	44.62
	Semi-skilled	12	18.46
	Semi Professional	3	4.62
	Unemployed	15	23.08
Marital status	Married	36	55.38
	Unmarried	29	44.62
Ratol poison consumed (Grams)	7	2	3.08
	10	4	6.15
	15	18	27.69
	20	11	16.92
	25	12	18.46
	30	12	18.46
	40	1	1.54
	45	5	7.69
Time delay	Up to 6 hrs	56	86.15
	> 6 hrs	9	13.85
Symptoms	Asymptomatic	18	27.69
	Symptomatic	47	72.31
Nausea	Yes	24	36.92
	No	41	63.08
Bleeding manifestation	Yes	13	20
	No	52	80
Abdominal pain	Yes	17	26.15
	No	48	73.85
Oliguria	Yes	1	1.54
	No	64	98.46
Headache	Yes	11	16.92
	No	54	83.08
Outcome	Discharge	59	90.77
	Death	6	9.23

56 (86.15%) reached the hospital within 6 hrs, while only 9 (13.85%) took > 6 hrs. 47 (72.31%) had symptoms like nausea, bleeding manifestations, abdominal pain, oliguria and headache. Most symptoms were nausea 24 (36.92%) and abdominal pain 17 (26.15%). 59 (90.77%) had a successful treatment outcome and were discharged, while six died.

Table 2: Comparison of parameters between outcomes

		Outcome		P-value
		Discharge	**Death**	
Gender	Male	33	4	0.613
	Female	26	2	
Age	> 30 yrs	15	20	0.013
	< 30 yrs	22	8	
Quantity of Ratol poison consumed	Up to 15g	24	0	<0.0001
	16-30g	33	2	
	>30g	2	4	
Time delay	Up to 6 hrs	54	2	<0.0001
	6 hrs	5	4	
Presence of symptoms	Asymptomatic	17	1	0.526
	symptomatic	42	5	

No significant difference in gender between outcomes (p=0.613), but a significant difference in age between outcomes (p=0.013). A statistically significant association exists between the quantity of Ratol poison consumed and the outcome (p<0.0001).

The time between the consumption of poison and initiation of treatment plays an important role in determining the treatment outcome, and it was statistically significant (p<0.0001). No significant difference in the presence of symptoms between outcomes (p=0.526).

Out of 65 patients, 18 were hospitalised for more than two weeks and got discharged. Three were hospitalised for > 2 weeks but couldn't be saved. Trend analysis of the blood parameters of 18 discharged study participants and three deceased is presented. There is a notable peak in serum bilirubin values on day 2, which gradually declines over two weeks. There is no significant correlation between the pattern of serum bilirubin rise and mortality.

The Prothrombin levels peak between day two and day four and gradually declines over two weeks. There is no significant correlation between prothrombin time and mortality among yellow Phosphorus poisoning. INR rises to a peak on day two and gradually declines over two weeks in both categories of patients, and INR shows no correlation with mortality pattern.

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In discharged patients, SGOT gradually increases and attains a peak on Day 4. In contrast, in the case of dead patients, there is a twofold increase in SGOT levels, with a peak on Day 4; after that, it also increased, indicating a significant correlation between rising SGOT levels and mortality [Figure 1].

In discharged patients, SGPT gradually increases to peak on Day 4 and then decreases over two weeks. In contrast, there is a gradual increase in SGPT in dead patients, which attains a peak in seven days. A twofold increase in SGPT in patients who died indicates a significant association between rising SGPT and mortality [Figure 2].

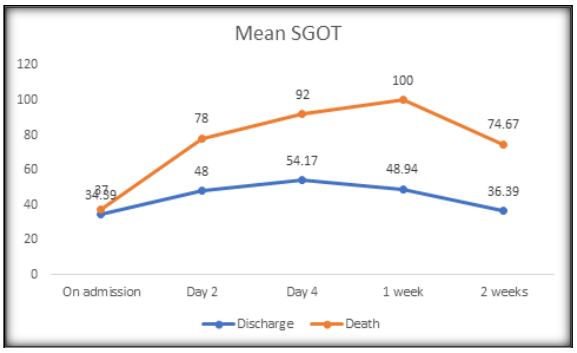

Figure 1: Mean SGOT

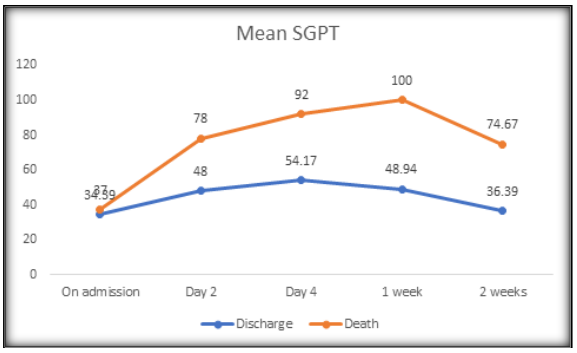

Figure 2: Mean SGPT

Table 3: The clinical parameters analysed by the Wilcoxon sign rank test

		Outcome (Median [IQR])	
		Discharge	**Death**
SGOT	On admission	39.5 (34,46)	40.6 (38,42)
	At two weeks	33.72 (0,64)	10.66
P-value		0.1503	0.046
SGPT	On admission	37.08 (32,42)	36.33 (36,38)
	At two weeks	18.18 (0,38)	6.66
P-value		0.0001	0.0459
INR	On admission	1.23 (1.1,1.3)	1.15 (1.1,1.2)
	At two weeks	0.4 (0,1.1)	0.18
P-value		<0.0001	0.033
PT	On admission	16.32 (15,17)	15.5 (15,16)
	At two weeks	5.54 (0,15)	2.5
P-value		<0.0001	0.033

All blood parameters like SGOT, SGPT, INR, and Prothrombin time have a statistically significant difference at admission and after two weeks for the deceased [Table 1].

DISCUSSION

Poisoning is one of the common causes of admission to the emergency department. A systematic analysis shows that rodenticides and pesticide poisoning account for nearly 60% of all poisonings. When accidentally exposed to them, an ideal rodenticide should kill the rodents and be non-toxic to humans and pets. Unfortunately, such a rodenticide is not available. Rodenticides like warfarin and super warfarin usually cause major toxicity on chronic exposure, whereas yellow and white phosphorus and phosphides cause acute toxicity. Yellow phosphorus has worse outcomes than others, commonly available as a paste, and the frequent product brought to the hospital is Ratol.

Toxicity resulting from poison ingestion can be divided into three stages. The first stage occurs within 24 hours and is generally symptom-free, although gastrointestinal tract irritation may lead to symptoms such as vomiting and abdominal pain. The second stage, lasting from 24 to 72 hours, is also mostly asymptomatic but may show slight increases in bilirubin and liver enzymes. The third stage, lasting beyond 72 hours, is characterised by hepatomegaly, jaundice, and the possibility of acute fulminant liver failure. Complications include bleeding tendencies, acute tubular necrosis (rare), and hepatic encephalopathy. These stages provide an understanding of the progression and potential complications associated with poisoning.[9]

The total number of patients in our study was 65, who fulfilled the inclusion criteria. Out of 65, the maximum number of study participants were in the age group of 21 – 30 years, similar to Karanth S and Nayyar V.'s study, in which 61.3% of the population was less than 30 years.[10] More than half of the study participants were male, married and skilled workers.

Banerjee I et al. study was done over two and a half years and showed a male-to-female ratio of 1: 1.3 with female predominance.[11] About 86.15% of people got Medical care within 6 hrs of consumption of Ratol Poison, and the time delay in seeking medical attention plays a role in the outcome, i.e. discharge and death. The most

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common symptom in our study was nausea (37%), and nearly 18% were asymptomatic. The presence or absence of symptoms doesn't affect our study's outcome.

The amount of poison consumed plays an important role in the outcome of poisoning. The more the amount, the more the chance of death rather than discharge. There is a statistically significant association between the patient's age, amount of poison consumed, time taken between poison consumption and seeking medical help and the outcome of discharge or death.

The mortality rate in our study was 9.23%. In the Nalabothu M et al. study, out of 43 patients, 21 (48.8%) survived, 12 (27.9%) died, and 10 (23.3 %) were discharged AMA.[12]

In Fernandez and Canzares et al. case series of 15 patients with yellow phosphorus, the mortality rate is 27%.[13] A study by Tahir Gokdemir M et al. showed a mortality rate of 28.3%. The study done by McCarron et al. has shown varying mortality rates depending on the presenting symptoms. It was found to be 23% for patients with GI symptoms and 73% for those with CNS manifestations.[15]

Mean serum bilirubin, prothrombin time and INR play a role in morbidity. Duration of hospital stay and has no role in determining mortality. Mean SGPT and SGOT correlate with mortality. This is similar to the study by Gopalakrishnan et al.[16], which showed that delayed resuscitation, jaundice, hepatic encephalopathy, the elevation of SGOT and SGPT to >1000 IU/L, metabolic acidosis, and refractory shock are reliable predictors of a bad outcome.

CONCLUSION

The age of the patient, amount of the poison and duration of delay in treatment influence the patient's outcome. Liver damage is found to be the most common complication of yellow phosphorus poisoning. Banning the sale and use of yellow phosphorus as rat killer poison will help in reducing the suicide rate considerably. Awareness creation, mental health promotion, and devising a proper protocol are necessary for emergency management.

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