OPEN ACCESS

IJABR

International Journal of Advanced Biotechnology and Research

ISSN 0976-2612, Online ISSN 2278–599X,
17 (4), 2026, 104-112

Research Article

Clinical Profile and Outcomes of Rodenticide Poisoning in a Tertiary Care Hospital: A Descriptive Hospital-Based Study

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DOI: 10.5281/zenodo.24214758

Received: 22/10/2026
Accepted: 09/11/2026
Published: 14/11/2026

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Abstract

Background: Rodenticide poisoning is a common cause of emergency admission in rural India. The compounds differ in toxicity, and outcomes vary widely. We describe the clinical and laboratory profile of patients with rodenticide poisoning and examine which factors are associated with outcome.

Methods: This hospital-based descriptive study was done over 18 months in the general medicine department of a government tertiary care hospital in southern India. Patients older than 12 years with rodenticide poisoning were enrolled after written informed consent. Data on demographics, compound, clinical features and laboratory values were collected on a proforma. Outcome was classified as discharged, died or discharged against medical advice (DAMA). Chi-square test and one-way ANOVA were used.

Results: Of 150 patients, 84 (56.0%) were aged 28 to 42 years and 89 (59.3%) were male. The mean age was 30.5 years. Most lived in rural areas (74.7%), and 97.3% of poisonings were deliberate. Yellow phosphorus was the commonest compound (47.3%), followed by bromadiolone (39.3%) and zinc phosphide (13.3%). Vomiting (45.3%) was the commonest symptom. In all, 103 patients (68.7%) were discharged, 16 (10.7%) died and 31 (20.7%) left against medical advice. Age, sex, residence, intent, psychiatric history, formulation and compound were not significantly associated with outcome. Direct bilirubin, SGOT, SGPT, INR, creatinine, bicarbonate, ionised calcium, potassium and lactate differed significantly across outcome groups ($p < 0.001$). Bicarbonate ($\eta^2 = 0.934$), lactate ($\eta^2 = 0.847$) and INR ($\eta^2 = 0.821$) showed the strongest association.

Conclusion: Rodenticide poisoning mainly affected young, rural adults and was almost always deliberate. Low bicarbonate and high lactate and INR were most strongly associated with death. These tests can help identify patients who need early intensive monitoring.

Keywords: Bromadiolone, Hepatotoxicity, Prognosis, Rodenticide poisoning, Yellow phosphorus

Introduction

Pesticide self-poisoning is one of the leading ways people end their lives in rural Asia.[1] Most deaths from pesticide self-poisoning occur in low- and middle-income countries, largely in Asia, where farming communities have easy access to these chemicals.[2] The problem is preventable, yet

poisoning remains a frequent reason for emergency admission and intensive care use.

India follows this pattern. A pooled analysis of Indian hospital studies from 2010 to 2020 found that pesticides accounted for 63% of poisoning in adults. The share was highest in North India (79.1%) and was 65.9% in the South.[3] Within the pesticide group, rodenticides deserve separate attention. They are cheap and sold without a prescription in small shops. Many households keep them to control rats, so the poison is within reach at the moment of crisis.

Several factors explain why rodenticides are used so often for self-harm. Debt, family conflict, failed examinations and relationship problems are common triggers, and many acts are impulsive. The decision to ingest a poison is often made within minutes. When a lethal agent is stored in the kitchen or the shed, there is little time for the urge to pass. Reducing access to the most toxic products is therefore one of the few prevention measures with a clear logic, but it needs local data on which products are actually causing harm. Young adults in their working years are the group most often affected, so each death also carries a large social and economic cost for the family. Describing who is affected, which compounds they take and how they fare in hospital is the first step towards targeted prevention.

Three types of rodenticide are common in India. Pastes and cakes containing yellow phosphorus are widely sold, especially in the southern states. Anticoagulant products such as bromadiolone are also common. Tablets and powders containing zinc phosphide make up the third group. Reviews of pesticide poisoning in India note that clinicians face a wide range of toxic agents and that specific antidotes exist for only a few of them.[4] Packaging is often similar across brands. Patients and relatives may not know which compound was taken, so the doctor has to work out the agent from the history and the clinical picture.

Anticoagulant rodenticides such as bromadiolone are long-acting vitamin K antagonists. They block the recycling of vitamin K and cause a delayed fall in clotting factors II, VII, IX and X. Patients usually present days after ingestion with bleeding from the gums, urine or gut and a prolonged prothrombin time. Vitamin K1 is the specific treatment, and the response is usually good when it is started early.[5]

Yellow phosphorus behaves very differently. It is a cellular toxin that is absorbed from the gut and concentrates in the liver. Vomiting and abdominal pain appear in the first hours. Many patients then seem to improve for a day or two. This quiet period can be misleading. It is followed by jaundice, coagulopathy and encephalopathy, and some patients go on to acute liver failure. There is no antidote, so care is supportive, and a few patients need referral for liver transplantation.

Zinc phosphide reacts with gastric acid and releases phosphine gas. Phosphine inhibits mitochondrial respiration. Patients develop early vomiting, low blood pressure, arrhythmias and metabolic acidosis. Death, when it occurs, is usually from circulatory failure within the first day.

Hospital care adds further difficulty. Many patients from rural areas first reach a primary health centre or a private clinic, and some travel for hours before arriving at a tertiary hospital. Symptoms in the first day are often mild and non-specific, so severity can be underestimated at the bedside. Intensive care beds and liver transplant services are limited, and families may decide to take the patient home when the cost of care rises. Early identification of patients likely to deteriorate would help staff decide who needs close monitoring and who can be managed on the ward.

Because outcomes differ so much between compounds and between patients, clinicians need simple ways to identify who is at highest risk. Liver enzymes, INR, bicarbonate and lactate are available in most district hospitals. Reports from tertiary centres often come from liver units or poison centres and tend to focus on a single compound. Fewer studies describe a general medical ward, where all three types of rodenticide are admitted together. Fewer still compare laboratory values across clinical outcomes. Data from government hospitals in this part of the country are limited.

We therefore carried out a hospital-based descriptive study at a government tertiary care teaching hospital in southern India. We aimed to describe the demographic profile, type of poison, clinical features and laboratory findings of patients with rodenticide

poisoning. We also aimed to examine how these factors relate to the outcome at discharge.

Materials and Methods

This was a hospital-based descriptive study. It was carried out in the Department of General Medicine of a government tertiary care teaching hospital in southern India. The study period was 18 months. All patients admitted with rodenticide poisoning during the study period were assessed for eligibility. We included patients older than 12 years who had a history or clinical features of rodenticide poisoning and who were willing to take part. We excluded patients who did not consent. We also excluded patients in whom the rodenticide was mixed with another poison or toxic substance, including alcohol, and patients with pre-existing liver disease, including acute or chronic hepatitis B or C infection. Patients with acute liver failure from any cause other than rodenticide poisoning were excluded. Written informed consent was obtained from all participants or their attenders. The sample size was calculated with PASS software using the chi-square procedure at 90% confidence and a 5% significance level, based on a previously published study. This gave a required sample of 150 patients. Participants were selected by convenience sampling. Data were collected on a predesigned proforma by interviewing patients and their attenders. We recorded age, sex, place of residence, whether the poisoning was deliberate or accidental, and any previous psychiatric illness. We also recorded the compound ingested, its form (cake, paste or powder), the amount taken in grams, and the presenting symptoms. Laboratory tests included direct and indirect bilirubin, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), international normalized ratio (INR), creatinine, bicarbonate, sodium, ionized calcium, potassium and serum lactate. Patients were followed until discharge. The outcome was classified as discharged, died, or discharged against medical advice (DAMA). Recovery at follow-up after discharge was also recorded. The study examined the association of clinical and laboratory abnormalities with outcome, and the relation of poisoning severity to the type of compound ingested. Data were entered in Microsoft Excel and analysed with SPSS version 26. Categorical variables are given as frequencies and percentages. Continuous variables are given as mean and standard deviation. The association of categorical variables with outcome was tested with the chi-square test. Mean laboratory values across the three outcome groups were compared with one-way ANOVA, and the strength of association was expressed as eta squared (η^2). A p value below 0.05 was considered statistically significant. Patient identity was kept confidential throughout the study.

Results

The gender distribution of the studied cases showed that out of 150 patients there were 89 (59.3%) males and 61 (40.7 %) females. There was a male preponderance with M:F ratio of 1:0.68. (Figure 1)

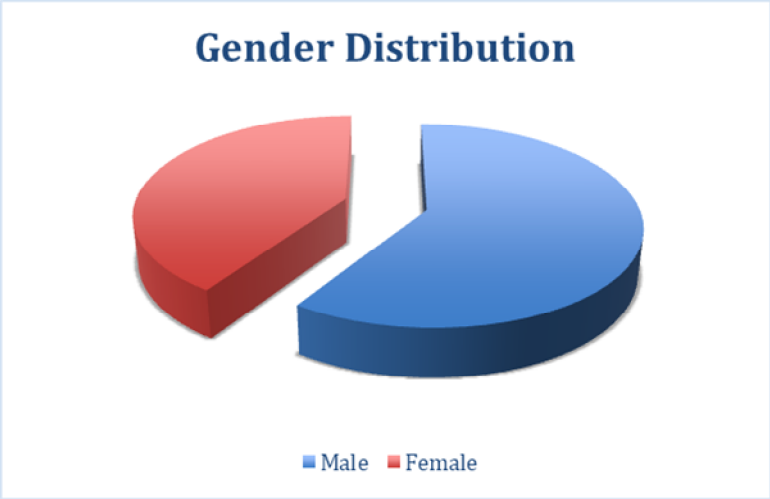


Figure 1: Gender Distribution of studied cases.

The analysis of age distribution of the studied cases showed that majority were in the 28–42 year age group (56.0%). The mean age was 30.5 years (SD 7.95; range 14–54 years). (Table 1).

Variable	Category	n	%
Age group (years)	13–27	48	32.0
	28–42	84	56.0
	43–56	18	12.0

Table 1: Age and gender distribution of the study population (n = 150).

Most patients lived in rural areas (74.7%). Nearly all poisonings were deliberate (97.3%), and 15.3% of patients had a previous psychiatric illness (Table 2).

Variable	Category	n	%
Residence	Rural	112	74.7
	Urban	38	25.3
Intent of poisoning	Deliberate	146	97.3
	Accidental	4	2.7
Previous psychiatric illness	Present	23	15.3
	Absent	127	84.7

Table 2: Residence, intent of poisoning and psychiatric history of the study population (n = 150).

Yellow phosphorus was the commonest compound (47.3%), followed by bromadiolone (39.3%) and zinc phosphide (13.3%). Paste (46.7%) and cake (42.0%) were the commonest forms. The amount ingested was most often 25 g (31.3%) or 30 g (24.7%) (Table 3).

Variable	Category	n	%
Compound	Yellow phosphorus	71	47.3
	Bromadiolone 0.005%	59	39.3
	Zinc phosphide	20	13.3
Form	Paste	70	46.7
	Cake	63	42.0
	Powder	17	11.3
Amount ingested	5 g	10	6.7
	10 g	23	15.3
	15 g	24	16.0
	20 g	3	2.0
	25 g	47	31.3
	30 g	37	24.7
	50 g	6	4.0

Table 3: Type, form and amount of rodenticide ingested (n = 150).

Vomiting was the commonest symptom (45.3%), followed by palpitations (42.7%), lethargy (40.7%) and bleeding tendency (40.0%). Jaundice and altered mental status each occurred in 32.0% of patients (Table 4).

Clinical feature	n	%
Vomiting	68	45.3
Palpitations	64	42.7
Lethargy	61	40.7
Bleeding tendency	60	40.0
Restlessness	57	38.0
Abdominal pain	56	37.3
Breathing difficulty	54	36.0
Seizures	53	35.3

Altered mental status	48	32.0
Jaundice	48	32.0

Table 4: Presenting clinical features of the study population (n = 150). Patients could have more than one feature.

Among the 150 patients, 103 (68.7%) were discharged, 16 (10.7%) died and 31 (20.7%) left against medical advice. At follow-up, 89 patients (59.3%) had recovered (Table 5).

Variable	Category	n	%
Outcome	Discharged	103	68.7
	Deceased	16	10.7
	DAMA	31	20.7
Status at follow-up	Recovered	89	59.3
	No follow-up	16	10.7
	Not applicable	45	30.0

Table 5: Outcome and follow-up status of the study population (n = 150). DAMA: discharged against medical advice.

Mean INR was 3.33 and mean SGOT was 1011.1 U/L in the whole group. Ranges were wide, for example SGOT 34 to 5917 U/L, INR 0.90 to 11.70, bicarbonate 2.00 to 24.82 mEq/L and lactate 0.54 to 21.50 mmol/L. All laboratory parameters except sodium differed significantly across outcome groups ($p < 0.001$). Patients who died had the highest bilirubin, transaminases, INR, creatinine and lactate and the lowest bicarbonate, ionised calcium and potassium. Values in the DAMA group lay between those of discharged and deceased patients. Bicarbonate ($\eta^2 = 0.934$), lactate ($\eta^2 = 0.847$) and INR ($\eta^2 = 0.821$) showed the strongest association with outcome. Sodium did not differ across groups ($p = 0.063$) (Table 6).

Parameter	Discharged (n = 103)	Deceased (n = 16)	DAMA (n = 31)	Overall (n = 150)	F	p	η^2
Direct bilirubin (mg/dL)	1.12 (0.50)	3.98 (1.97)	2.89 (1.57)	1.79 (1.46)	75.312	<0.001	0.506
Indirect bilirubin (mg/dL)	1.14 (0.46)	1.88 (0.79)	1.68 (0.65)	1.33 (0.61)	20.977	<0.001	0.222
SGOT (U/L)	260.8 (128.0)	3733.6 (1668.6)	2099.0 (1108.5)	1011.1 (1403.6)	195.216	<0.001	0.726
SGPT (U/L)	173.7 (84.5)	1193.1 (549.9)	680.2 (324.4)	387.1 (419.1)	154.887	<0.001	0.678
INR	1.64 (0.37)	7.43 (1.91)	6.86 (2.15)	3.33 (2.79)	337.333	<0.001	0.821
Creatinine (mg/dL)	0.77 (0.14)	1.56 (0.66)	0.90 (0.27)	0.88 (0.36)	59.908	<0.001	0.449
Bicarbonate (mEq/L)	22.40 (1.14)	5.35 (1.86)	19.45 (1.84)	19.97 (5.39)	1035.171	<0.001	0.934
Sodium (mEq/L)	138.15 (2.27)	136.44 (3.05)	137.42 (4.26)	137.81 (2.91)	2.818	0.063	0.037
Ionised calcium (mmol/L)	1.109 (0.051)	0.917 (0.048)	0.961 (0.078)	1.058 (0.095)	134.515	<0.001	0.647
Potassium (mEq/L)	3.58 (0.26)	2.99 (0.17)	3.25 (0.30)	3.45 (0.33)	46.412	<0.001	0.387
Serum lactate (mmol/L)	2.24 (0.95)	15.68 (4.32)	6.52 (1.89)	4.56 (4.58)	406.550	<0.001	0.847

Table 6: Laboratory parameters by outcome, shown as mean (SD) (n = 150). One-way ANOVA; η^2 = eta squared; DAMA: discharged against medical advice.

Outcome did not differ significantly by age group, gender, residence, intent of poisoning or previous psychiatric illness. Mortality was 16.7% in the 43–56 year group, compared with 10.4% and 9.5% in the younger groups. Eight of 23 patients (34.8%) with a psychiatric history left against medical advice, compared with 23 of 127 (18.1%) of those without (Table 7).

Variable	Category	Discharged n (%)	Deceased n (%)	DAMA n (%)	Total	χ^2	p
Age group (years)	13–27	34 (70.8)	5 (10.4)	9 (18.8)	48	1.144	0.887
	28–42	57 (67.9)	8 (9.5)	19 (22.6)	84		
	43–56	12 (66.7)	3 (16.7)	3 (16.7)	18		
Gender	Male	60 (67.4)	10 (11.2)	19 (21.3)	89	0.166	0.921
	Female	43 (70.5)	6 (9.8)	12 (19.7)	61		
Residence	Rural	77 (68.8)	11 (9.8)	24 (21.4)	112	0.421	0.810
	Urban	26 (68.4)	5 (13.2)	7 (18.4)	38		
Intent	Deliberate	100 (68.5)	16 (11.0)	30 (20.5)	146	0.500	0.779
	Accidental	3 (75.0)	0 (0.0)	1 (25.0)	4		
Previous psychiatric illness	Present	14 (60.9)	1 (4.3)	8 (34.8)	23	3.877	0.144
	Absent	89 (70.1)	15 (11.8)	23 (18.1)	127		

Table 7: Association of outcome with sociodemographic factors (n = 150). Chi-square test; row percentages shown. DAMA: discharged against medical advice.

Mortality was highest among patients who took powder (23.5%) and among those who took yellow phosphorus (15.5%), compared with 7.9% for cake, 10.0% for paste, 6.8% for bromadiolone and 5.0% for zinc phosphide. None of these differences was statistically significant (Table 8).

Variable	Category	Discharged n (%)	Deceased n (%)	DAMA n (%)	Total	χ^2	p
Form	Cake	47 (74.6)	5 (7.9)	11 (17.5)	63	5.548	0.236
	Paste	48 (68.6)	7 (10.0)	15 (21.4)	70		
	Powder	8 (47.1)	4 (23.5)	5 (29.4)	17		
Compound	Bromadiolone	41 (69.5)	4 (6.8)	14 (23.7)	59	3.637	0.457
	Yellow phosphorus	47 (66.2)	11 (15.5)	13 (18.3)	71		
	Zinc phosphide	15 (75.0)	1 (5.0)	4 (20.0)	20		

Table 8: Association of outcome with form and type of rodenticide (n = 150). Chi-square test; row percentages shown. DAMA: discharged against medical advice.

Discussion

This study describes 150 patients with rodenticide poisoning admitted to a general medicine ward over 18 months. Most were young adults from rural areas, nearly all had taken the poison deliberately, and yellow phosphorus was the commonest compound. In-hospital mortality was 10.7%, and one in five patients left against medical advice. Bicarbonate, lactate and INR showed the strongest association with outcome. Age, sex, residence, intent, psychiatric history, form and compound were not significantly associated with outcome.

Our profile is close to that of other Indian hospital series. Gopalakrishnan et al.[6] studied 99 adults at a tertiary centre. Yellow phosphorus accounted for most cases, 90.9% had taken a paste, mortality was 9.1%, and 77.8% of deaths were from fulminant hepatic failure. Paste was also the commonest form

in our patients (46.7%), and our mortality of 10.7% is similar. Abhilash et al.[7] reviewed 145 rodenticide poisonings and reported a mean age of 27.9 years, yellow phosphorus in 57% and in-hospital mortality of 9%. Our mean age was 30.5 years, and yellow phosphorus accounted for 47.3% of cases. In their series, age above 30 years independently predicted death (adjusted OR 2.2). Our oldest group also had the highest mortality (16.7%), but the difference was not significant ($p = 0.887$), and that group had only 18 patients.

Nearly all poisonings in our study were deliberate (97.3%), and only 15.3% of patients had a known psychiatric illness. Most self-harm episodes therefore occurred in people with no previous contact with mental health care. Easy access to cheap rodenticides may turn a moment of distress into a life-threatening event. Outcome did not differ by psychiatric history ($p = 0.144$). However, 34.8% of patients with a psychiatric history left against medical advice, compared with 18.1% of the others. This was not tested separately, and the numbers are small.

Liver injury was prominent. Mean SGOT was 3733.6 U/L in patients who died and 260.8 U/L in those who were discharged. Mean INR was 7.43 and 1.64 in the same groups. Vijayalekshmi et al.[8] studied 67 patients with yellow phosphorus liver injury, of whom 29 had acute liver failure, and linked disease severity to activation of the reticuloendothelial system. Saraf et al.[9] studied acute liver failure from zinc phosphide rodenticide and found that the MELD score, which includes INR and bilirubin, helped identify patients who needed liver transplantation. Our data point the same way, since INR and bilirubin rose step by step from discharged patients to those who died. We did not record encephalopathy grade or MELD scores, so we could not test these scores directly.

Metabolic acidosis was the strongest signal in our data. Mean bicarbonate was 5.35 mEq/L in patients who died and 22.40 mEq/L in those who were discharged ($\eta^2 = 0.934$). Mean lactate was 15.68 and 2.24 mmol/L ($\eta^2 = 0.847$). Trakulsrichai et al.[10] described 455 cases of zinc phosphide poisoning in Thailand, with a mortality of 7%. Eizadi-Mood et al.[11] compared phosphide poisoning in central Iran and reported mortality of 39.2% with aluminium phosphide and 22.2% with zinc phosphide. They found that systolic blood pressure predicted death. Only 1 of our 20 zinc phosphide patients died (5.0%), which is lower than in these series. The group was small, and we did not analyse acidosis by compound. We cannot say whether the acidosis in fatal cases came mainly from zinc phosphide or from liver failure after yellow phosphorus.

We did not collect treatment data, but published work gives context. Mark et al.[12] studied 229 patients and found higher survival with N-acetylcysteine ($p \leq 0.03$). Yellow phosphorus (relative risk 2.89) and delayed presentation were risk factors for death. Rashid et al.[13] pooled ten studies with 492 participants and found lower mortality with N-acetylcysteine than without it. Reddy et al.[14] developed 31 consensus recommendations on admission and discharge criteria, intensive care, extracorporeal therapy and early predictors of the need for liver transplantation in yellow phosphorus poisoning. Thomas et al.[15] reported one-month survival of 87.5% in 32 children with rodenticide hepatotoxicity treated with low-volume plasma exchange. Whether such treatments were used in our patients is not recorded. The 15.5% mortality we saw with yellow phosphorus should be read with that in mind.

One in five patients left against medical advice. Their INR (6.86), bilirubin and transaminases were far above those of discharged patients. Their INR was close to that of patients who died, although their bicarbonate was closer to that of discharged patients. We do not know what happened to them after they left. Counting them as survivors could understate the true mortality.

This study has limits. It was done at one center with convenience sampling, and the outcome was the status at discharge. We did not record the time from ingestion to arrival, the treatment given, or the timing of the blood tests. Eta squared describes association within the sample and does not give a cut-off for risk. Several cross-tabulations had small cells, so non-significant chi-square results may

reflect low power. We did not adjust for confounders.

Conclusion

In this hospital series, rodenticide poisoning mostly affected young rural adults and was almost always deliberate. Yellow phosphorus was the commonest compound, and 10.7% of patients died. Low bicarbonate and high lactate and INR were most strongly associated with death, followed by raised transaminases. Patients with these findings need early intensive monitoring. Prevention should focus on restricting the sale of yellow phosphorus products and on community mental health support. Larger multicentric studies with treatment and timing data should test these markers in multivariable models.

Declarations

Declarations

Ethical Approval

The study protocol was approved by the Institutional Ethics Committee of Government Medical College and Hospital, Chidambaram, Cuddalore District, India, before commencement of the study. The ethics committee approval/reference number and date should be provided by the authors.

Funding

The authors received no specific financial support from any funding agency in the public, commercial, or not-for-profit sectors for conducting this study.

Conflict of Interest

The authors declare that they have no conflict of interest.

Informed Consent

Written informed consent was obtained from the all participants and parents from the participants below 18, before enrolment in the study.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable institutional and ethical requirements.

Use of Artificial Intelligence

The authors used AI-assisted tools solely for grammatical correction, language refinement, and improving the readability of the manuscript. No AI tools were used to generate research data, perform statistical analyses, interpret findings, or draw scientific conclusions. The authors take full responsibility for the accuracy and integrity of the manuscript.

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