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A Prospective Observational Study on Clinical Profile, Laboratory Parameter and Outcomes of Rodenticide Poisoning

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Abstract

Introduction: In India, rodenticide poisoning poses a serious threat to public health due to its high rate of morbidity and mortality. The purpose of this study was to assess rodenticide poisoning's clinical characteristics, laboratory results, and outcomes at an Indian tertiary care facility.

Material and Methods: 50 patients with rodenticide poisoning who were hospitalized to a tertiary care facility in India for a period of one and a half years participated in a prospective observational research. Results were assessed in connection to the type of rodenticide consumed, and clinical symptoms, indicators, and laboratory parameter evaluations were conducted.

Results: In our investigation, the prevalence of rodenticide poisoning throughout the study period was 6.7%. The bulk of the people (58.00%) are under the age of twenty decades. Vomiting (38%) and abdominal discomfort (22%) were the most common symptoms. Clinically, icterus was the most prevalent sign (38%) of all. Symptoms (p=0.043), signs (p=0.040), rodenticide kind (p=0.003), amount of rodenticide consumed (0.038), and metabolic acidosis presence (p=0.001) were all significantly correlated with survival status. Patients who passed away had considerably worse liver function tests, coagulation profiles, and renal functions than those who survived (p<0.05). There was a 12% overall mortality rate.

Conclusion: Young adults are the age group most affected by rodenticide poisoning, which is linked to considerable morbidity and mortality. Improving patient outcomes requires early detection, timely treatment, and careful observation.In environments with limited resources, the findings of this study can direct the creation of evidence-based management plans and preventative actions for rodenticide poisoning.

Keywords: Rodenticide poisoning; Clinical profile; Laboratory parameters; Outcomes; tertiary care center; India.

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INTRODUCTION

In underdeveloped countries, where the use of these poisonous compounds is common and sometimes poorly controlled, rodenticide poisoning remains a serious worldwide public health concern.[1]. Strong chemicals included in rodenticides, which are intended to eradicate rodents, can be harmful to people if they are swallowed, breathed, or absorbed through the skin. [2] By using a prospective observational method, this study seeks to give a thorough review of the clinical profile, laboratory data, and outcomes related to rodenticide poisoning.

Rodenticides are commonly used in agricultural and domestic settings to control rodent populations. These substances are typically classified into several categories based on their active ingredients, including anticoagulants (e.g., warfarin, brodifacoum), metal phosphides (e.g., zinc phosphide), and hypercalcaemic agents (e.g., cholecalciferol).[3] The toxicity of these agents can vary significantly, influencing the clinical manifestations and treatment strategies required for affected individuals.[4]

The incidence of rodenticide poisoning varies globally, influenced by factors such as regional pest control practices, socioeconomic status, and availability of medical care.[5] In many low- and middle-income countries, the burden of rodenticide poisoning is exacerbated by the widespread use of these agents in households and agricultural settings[6] Data from poison control centres and hospital records suggest a higher prevalence of rodenticide poisoning in rural locations with limited access to healthcare facilities, and the population is more likely to be involved in agricultural activities.[7]

Different rodenticides have different clinical presentations, based on the kind and concentration of rodenticide ingested, the duration since exposure, and the patient's overall health status. Anticoagulant rodenticides, which inhibit vitamin K-dependent clotting factors, can lead to severe coagulopathy, manifesting as spontaneous bleeding, hematuria, gastrointestinal haemorrhage, and intracranial haemorrhage.[8] Metal phosphides, upon ingestion, release phosphine gas, causing severe gastrointestinal distress, metabolic acidosis, cardiac toxicity, and, in severe cases, multi-organ failure.[9] Hypercalcaemic rodenticides such as cholecalciferol induce hypercalcemia, leading to nephrocalcinosis, renal failure, and cardiovascular complications.[10]

Management includes supportive treatment including gastric lavage with diluted potassium permanganate, coconut oil, sodium-bicarbonate, intravenous magnesium sulfate and vasopressors. [11]

MATERIAL AND METHOD

This study was a prospective observational study conducted in the Department of General Medicine at Ballari Medical College and Research Centre, Ballari over a period of one and a half years, from August 2022 to January 2024.

Inclusion Criteria

1. Any patient admitted with rodenticide intake.
2. Patients aged above 15 years.

Exclusion Criteria

3. Cases involving mixed poison ingestion.
4. Patients with chronic liver disease.
5. Patients who consumed alcohol within 24 hours before admission.
6. Patients on medications such as anticoagulants or antiplatelets.
7. Patients with bleeding disorders.
8. Patients with acute diarrheal disease.
9. Patients with known coronary artery disease.

Method of Data Collection

During the study period, patients who met the inclusion criteria and provided informed consent were selected. The assessment of these patients included preoperative procedures consisting of eliciting appropriate history, clinical symptoms, signs, and laboratory tests.

RESULTS

All 6 patients (100%) who died had a survival status of "Died," all those patients who have survived has a survival status of survive among the stable patients has the survival status as "survived".

Table 1: Amount of Rodenticide Ingested

Amount	Died (N=6)	Stable (N=44)	Total (N=50)	p-value
<5g	1(16.7%)	27 (61.3%)	28 (44%)	0.038
>5g	5(83.3%)	17(38.7%)	22 (56%)	

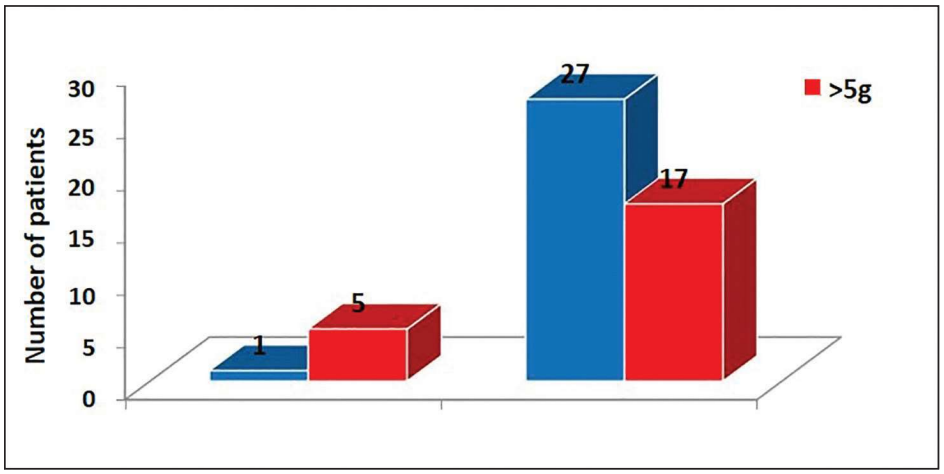

Fig. 1: Bar Diagram Presents Amount of Rodenticide Ingested

Among the patients who died, 16.7% ingested less than 5g of rodenticide, while 83.3% ingested more than 5g. Among the stable patients, 61.3% ingested less than 5g, and 56% ingested more than 5g. Overall, 44% of the total cases ingested less than 5g, and 56% ingested more than 5g. The p-value of 0.038 suggests significant association between the amount of rodenticide ingested and survival status.

Table 2: Type of Rodenticide

Type	Died (N=6)	Stable (N=44)	Total (N=50)	p-value
2% Zinc Phosphide Granules	1 (16.7%)	23 (52.3%)	24 (48%)	
Aluminium phosphide Pellets	1 (16.7%)	11 (25%)	12 (24%)	
Bromadiolone Cake	0 (0%)	7 (15.9%)	7 (14%)	**0.003***
Yellow Phosphorus	3 (50%)	2 (4.5%)	5 (10%)	
Yellow Phosphorus + Zinc Phosphide	1 (16.7%)	1 (2.3%)	2 (4%)	

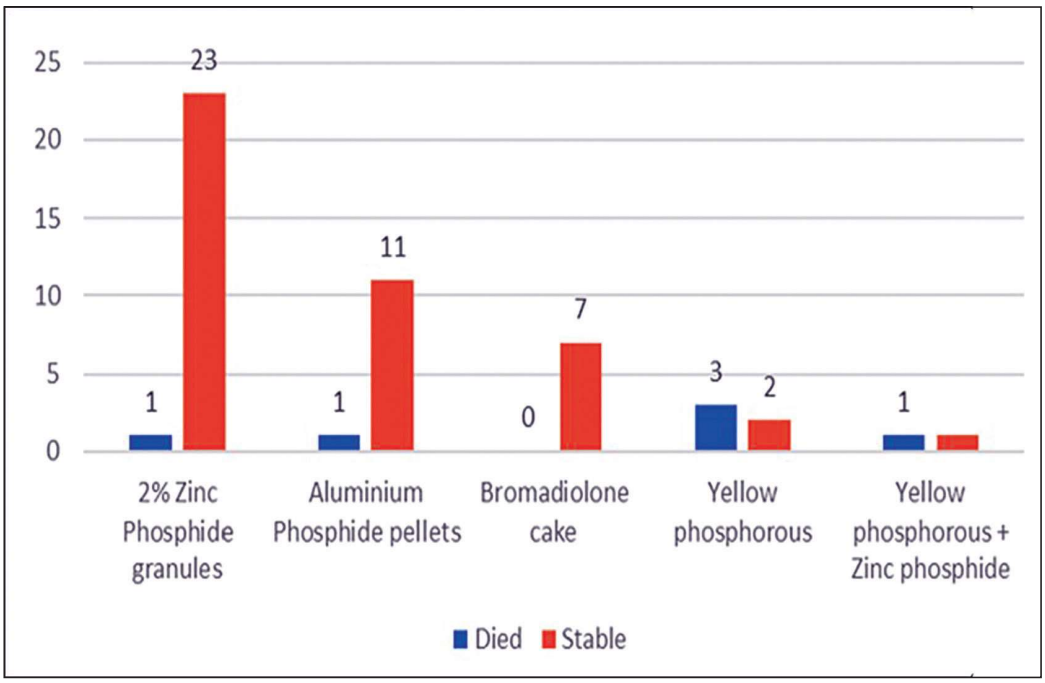

Fig. 2: Bar Diagram Depicting Type of Rodenticide Consumed with Respect to Survival Status

Among the patients who died, 16.7% ingested 2% zinc phosphide granules, 16.7% ingested aluminium phosphide pellets, 50% ingested yellow phosphorus, and 16.7% ingested a combination of yellow phosphorus and zinc phosphide. Among the stable patients, 52.3% ingested 2% zinc phosphide granules, 25% ingested aluminium phosphide pellets, 15.9% ingested bromadiolone cake, 4.5% ingested yellow phosphorus, and 2.3% ingested a combination of yellow phosphorus and zinc phosphide. Overall, 48% ingested 2% zinc phosphide granules, 24% ingested aluminium phosphide pellets, 14% ingested bromadiolone cake, 10% ingested yellow phosphorus, and 4% ingested a combination of yellow phosphorus and zinc phosphide. The p-value of 0.003 indicates a significant association between the type of rodenticide and survival status.

Table 3: Symptoms (Initial 24 hours)

Symptom	Died (N=6)	Stable (N=44)	Total (N=50)	p-value
Abdominal Pain	4 (66.7%)	7 (15.9%)	11 (22%)	0.043*
Nausea	0 (0%)	6 (13.6%)	6 (12%)	
None	1 (16.7%)	13 (29.5%)	14 (28%)	
Vomiting	1 (16.7%)	18 (40.9%)	19 (38%)	

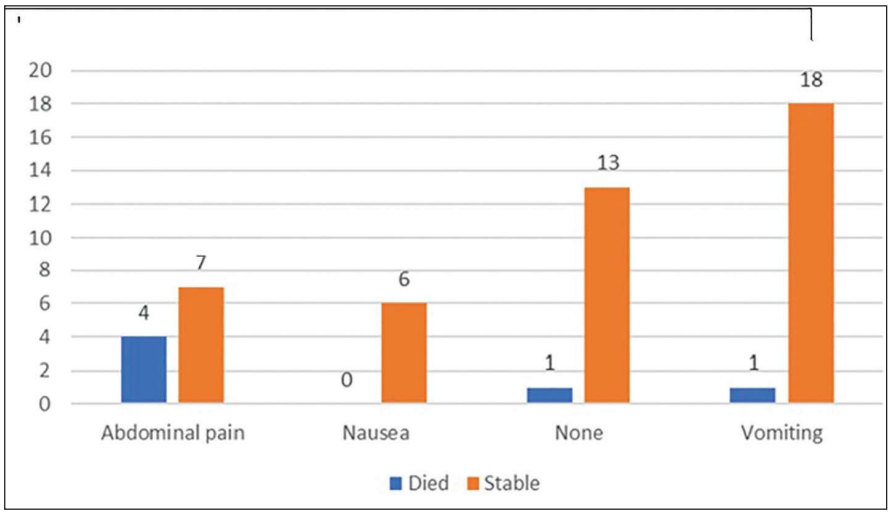

Fig. 3: Bar Diagram Depicting Symptoms of Patient with Respect to Survival Status

Among the patients who died, 66.7% experienced abdominal pain, 16.7% experienced vomiting, and 16.7% had no symptoms. Among the stable patients, 15.9% experienced abdominal pain, 13.6% experienced nausea, 29.5% had no symptoms, and 40.9% experienced vomiting. Overall, 22% experienced abdominal pain, 12% experienced nausea, 28% had no symptoms, and 38% experienced vomiting. The p-value of 0.043 suggests a significant association between symptoms and survival status.

Table 4: Clinical Signs

Sign	Died (N=6)	Stable (N=44)	Total (N=50)	p-value
Icterus	0 (0%)	19 (43.2%)	19 (38%)	0.040
Bleeding	0 (0%)	9 (20.4%)	9(18%)	
Altered Sensorium	0 (0%)	1 (2.0%)	1 (2%)	
Hepatic Encephalopathy	0 (0%)	1 (2.0%)	1 (2%)	
Hypotension	1 (16.7%)	5 (10.0%)	5(10%)	
None	5(83.3%)	10 (30%)	15 (30%)	

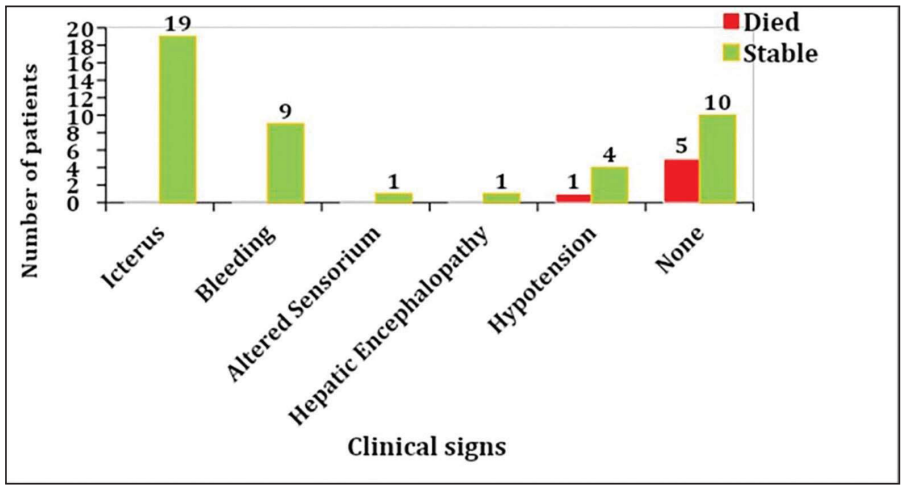

Fig. 4: Bar Diagram Depicting Clinical Signs with Respect to Survival Status

Among the patients who died, 16.7% had hypotension, and 83.3 had no signs. Among the stable patients, 43.2% had icterus, 20.4% had bleeding, 2% had altered sensorium, 2% had hepatic encephalopathy, 10 % had hypotension, and 30% had no signs. Overall, 38% had icterus, 18% had bleeding, 2% had altered sensorium, 2% had hepatic encephalopathy, 10% had hypotension, and 30% had no signs. The p-value of 0.040 indicates significant association between clinical signs and survival status.

Table 5: ABG (Metabolic Acidosis)

Status	Died (N=6)	Stable (N=44)	Total (N=50)	p-value
Absent	0 (0%)	31 (70.5%)	31 (62%)	0.001*
Present	6 (100%)	13 (29.5%)	19 (38%)	

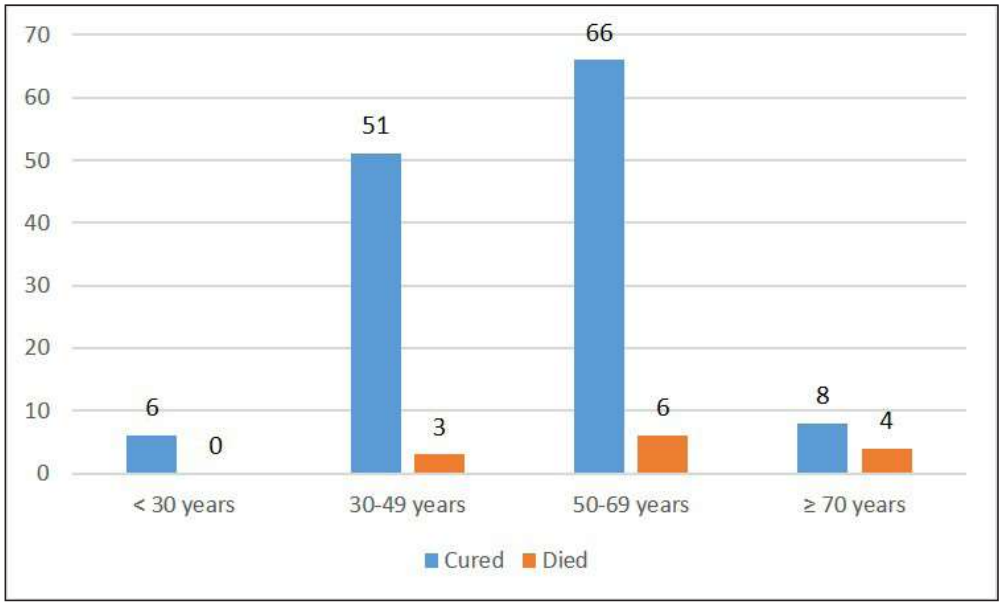

Fig. 5: Bar Diagram Depicting Abg (Metabolic Acidosis) with Respect to Survival Status

Among the patients who died, 100% had metabolic acidosis. Among the stable patients, 29.5% had metabolic acidosis, while 70.5% did not. Overall, 38% had metabolic acidosis, and 62% did not. The p-value of 0.001 suggests a significant association between metabolic acidosis and survival status.

Table 6: Liver Function Tests

Parameter	Died (Mean ± SD)	Survived (Mean ±SD)	Total (Mean ±SD)	p-value
AST (U/L)	1499 ± 684	388 ± 473	566 ± 650	0.000
ALT (U/L)	1690 ± 737	356 ± 392	569 ± 671	0.000
ALP (U/L)	970 ± 641	127 ± 167	262 ± 424	0.000
Bilirubin (mg/dL)	12.4 ± 5.6	5.5 ± 4.1	6.6 ± 5.0	0.000

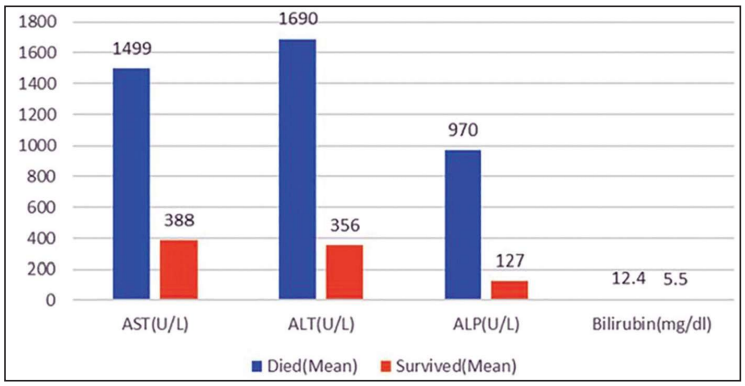

Fig. 6: Bar Diagram Depicting Liver Function Tests with Respect to Survival Status

The mean AST level was 1499 ± 684 U/L for those who died and 388 ± 473 U/L for survivors, with an overall mean of 566 ± 650 U/L (p=0.000). The mean ALT level was 1690 ± 737 U/L for those who died and 356 ± 392 U/L for survivors, with an overall mean of 569 ± 671 U/L (p=0.000). The mean ALP level was 970 ± 641 U/L for those who died and 127 ± 167 U/L for survivors, with an overall mean of 262 ± 424 U/L (p=0.000). The mean bilirubin level was 12.4 ± 5.6 mg/dL for those who died and 5.5 ± 4.1 mg/dL for survivors, with an overall mean of 6.6 ± 5.0 mg/dL (p=0.000). All p-values were 0.000, indicating significant differences between the groups for all liver function tests.

Table 7: Coagulation Profiles

Parameter	Died (Mean ±SD)	Survived (Mean ± SD)	Total (Mean ± SD)	p-value
PT (seconds)	59.2 ± 23.6	19.5 ± 12.1	25.9 ± 20.5	0.000
APTT (seconds)	91.6 ± 25.4	31.1 ± 12.4	40.8 ± 26.9	0.000
INR	8.7 ± 3.7	1.9 ± 1.2	2.9 ± 3.1	0.000

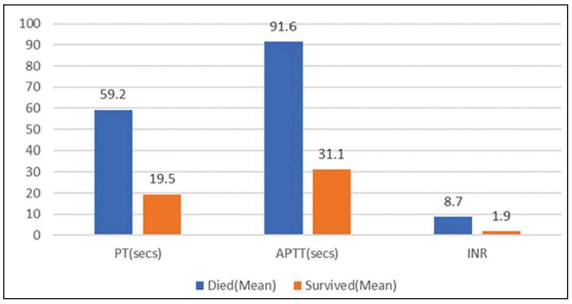

Fig. 7: Bar Diagram Depicting Coagulation Profile with Respect to Survival Status

The mean PT was 59.2 ± 23.6 seconds for those who died and 19.5 ± 12.1 seconds for survivors, with an overall mean of 25.9 ± 20.5 seconds (p=0.000). The mean APTT was 91.6 ± 25.4 seconds for those who died and 31.1 ± 12.4 seconds for survivors, with an overall mean of 40.8 ± 26.9 seconds (p=0.000). The mean INR was 8.7 ± 3.7 for those who died and 1.9 ± 1.2 for survivors, with an overall mean of 2.9 ± 3.1 (p=0.000). All p-values were 0.000, indicating significant differences between the groups for all coagulation profile parameters. However, bleeding and clotting time was increased in all the patients who died.

Table 8: Renal and Electrolytes

Parameter	Died (Mean ± SD)	Survived (Mean ±SD)	Total (Mean ±SD)	p-value
Creatinine (mg/dL)	3.54 ± 1.83	1.58 ± 2.05	1.90 ± 2.13	0.016
Sodium (mmol/L)	139.7 ± 3.1	140.5 ± 3.1	140.4 ± 3.1	>0.05
Potassium (mmol/L)	4.1 ± 0.4	4.3 ± 0.4	4.2 ± 0.4	>0.05
Calcium (mg/dL)	9.6 ± 0.6	9.4 ± 0.6	9.4 ± 0.6	>0.05

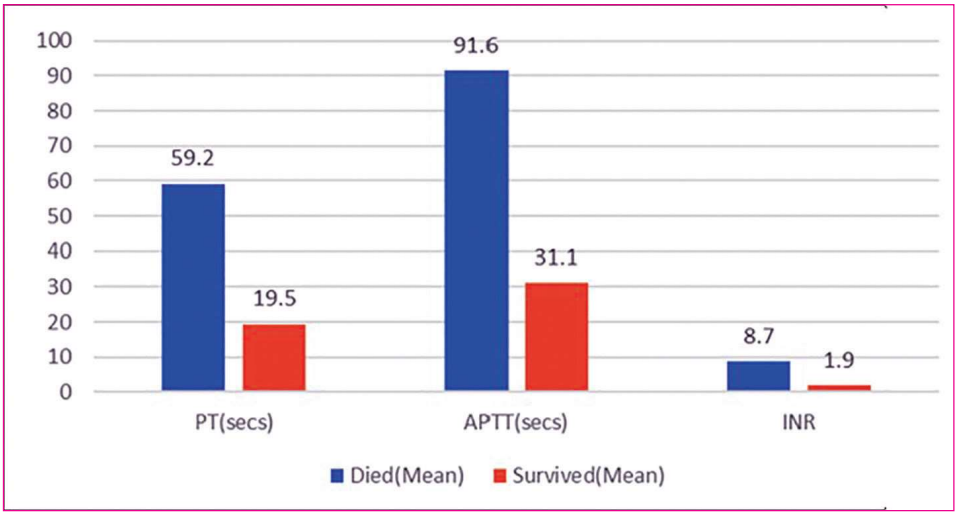

Fig. 8: Bar Diagram Depicting Renal Profile And Serum Electrolytes With Respect To Survival Status

The mean creatinine level was 3.54 ± 1.83 mg/dL for those who died and 1.58 ± 2.05 mg/dL for survivors, with an overall mean of 1.90 ± 2.13 mg/dL (p=0.016). The mean sodium level was 139.7 ± 3.1 mmol/L for those who died and 140.5 ± 3.1 mmol/L for survivors, with an overall mean of 140.4 ± 3.1 mmol/L (p>0.05). The mean potassium level was 4.1 ± 0.4 mmol/L for those who died and 4.3 ± 0.4 mmol/L for survivors, with an overall mean of 4.2 ± 0.4 mmol/L (p>0.05). The mean calcium level was 9.6± 0.6 mg/dL for those who died and 9.4 ± 0.6 mg/dL for survivors, with an overall mean of 9.4 ± 0.6 mg/dL (p>0.05). The p-value for creatinine was 0.016, indicating a significant difference between the groups, while the p-values for sodium, potassium, and calcium were greater than 0.05, suggesting no significant differences.

DISCUSSION

The present prospective observational study investigated the clinical profile, laboratory parameters, and outcomes of rodenticide poisoning in 50 patients admitted to the Ballari Medical College & Research Centre Ballar (Formerly known as VIMS Ballari Karnataka - 2024) over a period of one and a half years. The study aimed to evaluate the clinical outcomes in relation to the chemical composition of the ingested poison, examine the prevalence of rodenticide poisoning at Ballari Medical College & Research Centre Ballari (Formerly known as VIMS Ballari Karnataka - 2024), and assess the prognosis among different age groups. Prevalence A study was conducted to evaluate the prevalence of rodenticide poisoning over a period of one and half year (August 2020 to January 2024).[12]

Total number of rat poison cases were found to be 177 among 2605 of all poisoning cases, which accounts for prevalence of 6.7% of all cases. After meeting exclusion criteria, a total 50 patients were selected for the study; which was in comparison with study done by Ashok kumar pannu *et al.*,[13] which showed prevalence of 5 %. The prevalence in our study is more, which can be attributed to factors such as regional availability of rodenticides and cultural differences.

Age and Sex Distribution:

The majority of the individuals (58.00%) are in the youngest age category (< 20 years). This finding is consistent with a study by Chugh SN *et al.*,[14] which reported that the most common age group

affected by rodenticide poisoning was 21-30 years (42.5%) Similarly, a study by Bumbrah GS *et al*.[15] found that the majority of patients (64%) were in the age group of 21-40 years. The higher incidence of rodenticide poisoning in young adults may be attributed to various factors such as stress, relationship issues, and financial problems.

Regarding sex distribution, our study found a slight male predominance (52%) compared to females (48%). This finding is in line with a study by Sciuto AM *et al*.,[16] which reported a male predominance of 57.5%. However, a study by Anand R *et al*. observed a female predominance of 56%.[17] The variation in sex distribution across studies may be influenced by regional and cultural differences.

In the present study, 2% zinc phosphide granules were the most common type of rodenticide ingested (48%), followed by aluminium phosphide pellets (24%) and yellow phosphorus (10%). A significant association was found between the type of rodenticide and survival status (p=0.003). This finding is consistent with a study by Mehrpour O *et al*.,[18] which reported that zinc phosphide was the most common rodenticide (47.5%), followed by yellow phosphorus (27.5%) However, a study by Wahab A *et al*.[19] found that aluminium phosphide was the most common rodenticide (60%), followed by zinc phosphide (24%).

The present study highlights the clinical profile, laboratory parameters, and outcomes of rodenticide poisoning in a tertiary care centre in India. The majority of patients were young adults, with a slight male predominance. Zinc phosphide was the most common type of rodenticide ingested, and vomiting and abdominal pain were the most frequent symptoms. Significant associations were found between survival status and the type of rodenticide, symptoms, and the presence of metabolic acidosis.

The elevation of liver enzymes and bilirubin in rodenticide poisoning can be attributed to the hepatotoxic effects of the ingested substances. Zinc phosphide and aluminium phosphide release phosphine gas, which causes oxidative stress and mitochondrial dysfunction in hepatocytes. Yellow phosphorus directly damages the liver by causing steatosis, necrosis, and fibrosi. In our study, significant differences were observed between those who died and those who survived for all coagulation profile parameters, including PT, APTT, and INR (p=0.000 for all). The mean values of these parameters were markedly prolonged in patients who died compared to survivors.

In our study, a significant association was found between the presence of metabolic acidosis and survival status (p=0.001). All patients who died had metabolic acidosis, while only 29.5% of stable patients had metabolic acidosis. This finding is consistent with a study by Raghapriya *et al*., which reported a significantly higher incidence of metabolic acidosis in non-survivors compared to survivors (p<0.001). Similarly, a study by Mundhe *et al*. found a significantly higher incidence of metabolic acidosis in patients with severe poisoning compared to those with mild to moderate poisoning (p<0.001)

Liver function tests, coagulation profile, and renal function were significantly deranged in patients who died compared to survivors. The overall mortality rate was 12%. The findings of this study emphasize the need for early recognition, prompt treatment, and close monitoring of patients with rodenticide poisoning. Education and awareness programs should be conducted to prevent accidental or intentional ingestion of rodenticides. Stricter regulations on the sale and distribution of rodenticides may help in reducing the incidence of poisoning. Further research with larger sample sizes and multicentric designs is warranted to validate the findings of this study and explore effective management strategies for rodenticide poisoning.

In the present study, a significant difference was observed between those who died and those who survived for creatinine levels (p=0.016). The mean creatinine level was higher in patients who died compared to survivors. This finding is consistent with a study by Raghapriya *et al*., which reported significantly higher creatinine levels in non-survivors compared to survivors (p<0.001). Similarly, a study by Mundhe *et al*. found significantly higher creatinine levels in patients with severe poisoning compared to those with mild to moderate poisoning (p<0.001).

CONCLUSION

In a tertiary care facility in India, the current study offers important new information about the clinical characteristics, test results, and consequences of rodenticide poisoning. The results emphasize how critical it is to identify rodenticide poisoning patients early, treat them quickly, and closely follow them. Significant correlations between survival status and rodenticide kind, symptoms, and metabolic acidosis were found in the research.

Compared to survivors, individuals who passed away had markedly abnormal liver function tests, coagulation profiles, and renal functions. The 12% total mortality rate highlights the necessity of efficient management plans and preventative initiatives.

The study emphasizes how crucial education and awareness campaigns are in preventing rodenticide consumption, whether on purpose or by accident. The frequency of poisoning may be decreased with stricter laws governing the distribution and sale of rodenticides. To enhance patient outcomes, healthcare professionals should get training on how to identify and treat rodenticide poisoning early on. In environments with limited resources, the results of this study can direct the creation of management recommendations and treatment procedures for rodenticide poisoning.

To confirm the results of this study and investigate efficient management techniques for rodenticide poisoning, more research with bigger sample numbers and multicentric designs is necessary. Studies with long-term follow-up can offer important information about the quality of life and long-term effects of rodenticide poisoning survivors. Cooperation between public health and healthcare providers To address the problem of rodenticide poisoning and put into place efficient preventative and control measures, authorities and legislators are crucial.

In conclusion, young adults are the age group most impacted by rodenticide poisoning, which is a serious public health issue in India. Improving patient outcomes requires early detection, timely treatment, and careful observation. In environments with limited resources, the results of this study can direct the creation of evidence-based management plans and preventative actions for rodenticide poisoning.

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