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Clinical Profile of Cases Admitted with Rat Killer Paste Poisoning in a Government Tertiary Care Hospital - A Cross Sectional Study

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Abstract: ***Background: Across the country, rodenticide toxicity is not as common. The death rates also vary significantly. This study was done to evaluate the clinical profile of patients who were hospitalised after swallowing rat poison paste. This was done because of the mortality and morbidity caused by rodenticides. Methods: A cross-sectional investigation was carried out among the cases with suspected histories of rat killer paste poisoning in the Department of General Medicine at Thanjavur Medical College and Hospital between October 2019 and August 2020. The study consisted of 80 patients in total. All cases' clinical profiles and outcomes were evaluated. Using SPSS, all data were entered and analysed. Results: 52.5% cases had toxic hepatitis, 33.8% cases had toxic myocarditis with overall mortality of 20%.Significant association was noted between toxic hepatitis and amount of rat poison consumed, the amount of time it took a patient to arrive at the hospital after ingesting rat poison paste, INR levels, and the presence of hepatic encephalopathy. Age and the amount of time it took a victim to arrive at the hospital after ingesting rat poison paste poison were related to mortality. Conclusion: Among the victims who ingested rat killer paste poison, mortality and morbidities were strikingly high, particularly due to toxic hepatitis and toxic myocarditis. It is possible to forbid the use of phosphorous in rat poison paste in order to reduce mortality and morbidities.***

Keywords: Toxic myocarditis, toxic hepatitis, rat killer paste poisoning, mortality

1. Introduction

In developing agrarian nations, poisoning is rarely an unintentional cause of death and is frequently caused by suicide. Poisoning accounts for a large portion of emergency medical care in rural India and also accounts for one third of admissions to intensive care units. Rat poison or rodenticide poisoning continues to be the second most frequent poisoning in our area, right after organophosphorus poisoning. However, there aren't as many pieces of literature on rodenticide toxicity as there are on organophosphorus poisoning.[1,2]

Rodenticide toxicity affects almost every system, and there are no established therapy recommendations. Few investigations have been done on its incidence, method of action, and management. Additionally, there are no conclusive clinical trials for phosphorous paste poisoning, the deadliest of all rodenticide toxins[3].To protect their stored grains against mice, who are present everywhere, rodenticides are frequently available in practically every household. because it is more readily available and less expensive than other insecticides available on the market. Due of its widespread availability, it is frequently consumed by people who are suicidal or mistakenly swallowed by kids[4]. Rodenticide comes in a variety of forms, including powder, paste, and cakes.

Elements of phosphorus are typically present in paste forms. Yellow, white, and red phosphorus are the three kinds of phosphorus that are now understood. Red phosphorus is the least harmful and least absorbed of all three types. White phosphorus is primarily utilised as an explosive agent in ammunition and fireworks. Yellow phosphorus, the active component, is present in the paste form in 3% strength. It is corrosive and damages any tissue it comes into contact[4].Phosphorus is the most prevalent and deadly poison among these rodenticides, especially after 3–4 days of consumption when liver damage begins. Since there is a lack of adequate information on rodenticides[5], Self-harm was projected to cause 2 million cases of poisoning annually, with 200000 deaths, in 1990. In contrast, it was predicted that occupational and unintentional exposure would result in 100,000 illnesses and 20,000 fatalities. According to a study intentional self-poisoning results in a much greater mortality rate than accidental poisoning[6].

The use of pesticides specifically formulated to kill rodents, such as rodenticides, increases the risk of unintentional poisoning for a number of reasons. They are toxic to people as well because they were made to kill mammals. Since rats frequently coexist with humans, using rodenticides carries a risk of exposure to humans, especially children and their pets, as well as other non-target species. They are some of the most dangerous substances frequently discovered in households. The prevalence of rodenticide poisoning varies across the nation. The rates of mortality also differ greatly. The type and dosage of poison taken, the absence of a known antidote for some rodenticides, and the length of time between exposure and treatment all have an impact on the result[7,8]. Considering the mortality and morbidity caused due to rodenticides, this research was conducted to assess the

clinical profile of cases who were hospitalised for consuming rat poison paste.

2. Materials

This study was conducted as a cross sectional study in the Department of general medicine in Thanjavur Medical College and hospital, Thanjavur, a tertiary care Government teaching hospital during the period of October 2019 to August 2020. Patients with alleged history of rat killer paste poisoning presented to our hospital during the study period were included in the study. Patients with history of previous hepatic disorder, cases who consumed other poisoning substance along with rate killer paste and cases aged less than 18 years were excluded from the study. A total of 80 cases who were admitted as inpatient in the department of general medicine during the study period were included in the study. All the cases were assessed for detailed history, clinical examination and assessed for the presence of toxic myocarditis, toxic hepatitis and mortality was also documented. All data were entered and analysed using Statistical Package for Social Sciences version 19 software. Chi square test and Yates chi square test were used, appropriately to calculate the statistical significance. P value of< 0.05 was considered as statistically significant.

3. Results

Among the 80 cases included in the present study, 27(33.8%) cases had toxic myocarditis. On assessing the association between the presence of toxic myocarditis and parameters like age, gender, quantity of rat poison consumed and Time taken topresent to hospital after consuming rat killer paste poison, there were no statistical significant association noted.

Table 1: Association between clinical profile and Toxic myocarditis

Variables	Toxicmyocarditis				P value
	Present (n=27)		Absent (n=53)		
	n	%	n	%	
Agecategory					
<20 years	5	18.5	8	15.1	0.454
20– 39years	19	70.4	33	62.3	
40– 59years	3	11.1	12	22.6	
Gender					
Female	13	48.1	21	39.6	0.484
Male	14	51.9	32	60.4	
Quantity of Rat poison consumed					
<10 grams	9	33.3	22	41.5	0.374
10–20 grams	14	51.9	19	35.8	
>20 grams	4	14.8	12	22.6	
Time of presentation					
<6 hours	11	40.7	18	34	0.942
6–12 hours	11	40.7	25	47.2	
>12 –48 hours	3	11.1	6	11.3	
>48 hours	2	7.4	4	7.5	

Among the 80 cases included in the present study, 42(52.5%) cases had toxic hepatitis. On assessing the association between the presence of toxic hepatitis and parameters like quantity of rat poison consumed, timetaken topresent to hospital after consuming rat killer paste poison, INR levels and presence of hepatic encephalopathy, there were statistical significant association noted however there were no significant association reported between toxic hepatitis and parameters like age and gender.

Table 2: Association between clinical profile and Toxic hepatitis

Variables	Toxic Hepatitis				P value
	Present (n=42)		Absent (n=38)		
	n	%	n	%	
Age category					
<20 years	7	16.7	6	15.8	0.444
20– 39years	25	59.5	27	71.1	
40– 59years	10	23.8	5	13.2	
Gender					
Female	17	40.5	17	44.7	0.821
Male	25	59.5	21	55.3	
Quantity of Rat poison consumed					
<10 grams	10	23.8	21	55.3	0.005*
10–20 grams	19	45.2	14	36.8	
>20 grams	13	31	3	7.9	
Time of presentation					
< 6 hours	20	52.6	9	21.4	0.015*
6–12 hours	15	39.5	21	50	
>12 –48 hours	2	5.3	7	16.7	
>48 hours	1	2.6	5	11.9	
INR category					
INR1–2	1	2.6	36	94.7	<0.001*
INR 2.1–3	19	45.2	2	5.3	
INR 3.1–5	15	35.7	0	0	
INR > 5	7	16.7	0	0	
Hepaticence phalopathy					
Present	23	54.8	0	0	<0.001*
Absent	19	45.2	38	100	

*Significant

Among the 80 cases included in the present study, 16(20%) cases were dead. On assessing the association between the mortality status and parameters like age and time taken to present to hospital after consuming rat killer paste poison, there were statistical significant association noted however there were no significant association reported between mortality status and parameters like gender and amount of rat paste consumed.

Table 3: Association between clinical profile and mortality status

Variables	Mortality status				P value
	Death (n=16)		Recovered (n=64)		
	n	%	n	%	
Age category					
<20 years	5	31.2	8	12.5	0.003*
20– 39years	11	68.8	41	64.1	
40– 59years	0	0	15	23.4	
Gender					
Female	9	56.2	25	39.1	0.263 (NS)
Male	7	43.8	39	60.9	
Quantity of Rat poison consumed					
<10 grams	5	31.2	26	40.6	0.752(NS)
10–20 grams	7	43.8	26	40.6	
>20 grams	4	25	12	18.8	
Time of presentation					
<6 hours	2	12.5	27	42.2	<0.001*
6–12 hours	6	37.5	30	46.9	
>12 –48 hours	2	12.5	7	10.9	
>48 hours	6	37.5	0	0	

*Significant

4. Discussion

Around the world, poisoning is directly or indirectly to blame for more than a million illnesses[9]. Young adults' intentional self-harm is now one of the most prevalent causes of emergency hospital admission and has become a common response to mental distress. The WHO estimates that about 800,000 people die prematurely each year as a result of suicide. The medications people typically use in overdoses in industrialized nations—analgesics, tranquillizers, and antidepressants—are comparatively non-toxic[10-12]. For instance, in England, the case fatality rate for overdose is believed to be around 0.5 %. In poor nations, things are very different. Agricultural pesticides, particularly rodenticides, are the compounds that are most frequently employed for self-poisoning. The overall case mortality rate ranges from 10% to 20%[13]. For this reason, suicide rates in developing countries, especially in rural regions, are significantly influenced by deaths from pesticide poisoning[14]. Every year, there are three million incidents of pesticide poisoning worldwide, resulting in 2,20,000 deaths, the majority of which are deliberate[15].

In Indian homes, ratol paste, a typical rodenticide, includes 3% yellow phosphorus. A general protoplasmic toxin that causes multiorgan failure is yellow phosphorus[16]. Doses more than 1 mg/kg are almost always lethal.

Ratol paste poisoning can be intentional or unintentional. In contrast to most other poisons, ratol paste poisoning has a peculiar clinical course. During the first 72 hours after consumption, patients are often asymptomatic, though they may exhibit signs and symptoms of gastrointestinal irritation. They experience coagulopathy, abrupt hepatic failure, and altered liver function after 72 hours. Changes in mental status including disorientation, psychosis, hallucinations, and coma are examples of central nervous system consequences. Cardiogenic shock, tachycardia, hypotension, and arrhythmias are all symptoms of cardiac poisoning. Acute renal failure and acute tubular necrosis can both occur in some people[12,17]. Few patients disclose that they have consumed ratol paste since they are first asymptomatic and arrive at the hospital later than necessary. Late-arriving patients experience fulminant liver failure with 100% death after ingesting the deadly amount[18]. Patients with severe ratol paste ingestions skip the initial asymptomatic period and pass away from shock and cardiac arrest in the early stages[4]. Yellow phosphorus toxicity has no specific treatment. The goal of treatment is to remove the toxin and provide supportive therapy[19,20].

The other available rodenticides include zinc and aluminium phosphides. In contrast to ratol paste, which is used in homes, they are primarily utilised in agricultural fields[21]. Additionally, ratol paste is frequently mistaken for toothpaste and eaten by kids. Additionally, the paste should be spread to bread, according to the product's instructions, to encourage rodents to eat it. This will also make it appetising to kids[22]. Therefore, poisoning by accident is more frequent when using ratol paste.

A higher prevalence of poisoning among farmers and labourers was caused by poverty, insufficient money to support the family, and monsoon failure. The increased rate of poisoning among housewives is caused by a number of factors, including dowry, in-law brutality, family disputes, poor marital adjustment, and dependence on the spouse. The prevalence of poisoning among children has increased as a result of exam failure or an inability to live up to the high expectations of parents and teachers. Higher mortality from rat poison is caused by its high toxicity and lack of any known antidote. After consuming yellow phosphorus for 2–3 days, LFT derangements are primarily observed[23].

In India, a number of studies on poisoning were conducted. The most often impacted age group, according to Song ZY et al[24], was 20 to 40 years old. In the age group, our study agreed with Banerjee et al. According to a study by Winek CL et al[25], a greater proportion of patients consumed rat poison while mixing it with alcohol, however our analysis revealed that many patients consumed the poison either raw or while mixing it with water.

As soon as someone suspects they have ingested rat poison, they must go to the hospital for treatment. In their study, Elizabeth J, et al[26] discovered that patients were typically asymptomatic for the first 72 hours after intake or that they could have gastrointestinal irritation symptoms.

When rat killer poison was consumed, the biological markers AST and ALT showed a larger rise in patients, indicating that the liver was the first organ to be affected, followed by the kidney and other organs. In numerous cases of rat poisoning, imbalances in total bilirubin, total protein, albumin, AST, ALT, urea, creatinine, PT, and INR were also documented[27].

The best outcomes were observed in individuals where NAC (N Acetyl Cysteine) was started early in the course of their illness, according to Watson WA et al[28]. There are no recommendations for the regular use of NAC in cases of hepatic failure brought on by the intake of rodenticides and non-acetaminophen-induced ALF. NAC is occasionally used to treat patients who were admitted with ALF following phosphorus consumption. In contrast to the 35.7% death rate reported by Lipton RA et al[29] and the 19% mortality rate in our study. Patients that survived were given a psychiatric evaluation and released with stable conditions.

Due to hepatotoxicity, Fernandez et al. discovered a very high mortality rate (27%) and came to the conclusion that yellow phosphorus is particularly deadly when taken (33%)[30]. According to these data, yellow phosphorous was ingested by 5 out of 7 patients who experienced bleeding signs[31].In another study, five patients with yellow phosphorus toxicity displayed encephalopathy. Two patients with yellow phosphorus poisoning were aggravated by hypotension; one of the patients also had myocarditis and cardiogenic shock. N-acetyl cysteine was administered to the 10 out of 26 (38.6%) patients who had yellow phosphorous intake who had fulminant hepatic failure. But just four patients made improvements, while four others received referrals for liver transplants and two passed away[32].

Hepatitis was discovered to be a frequent consequence in the study (32.1%), followed by cardiogenic shock (8.9%). 18

hepatitis patients experienced three cases of hepatic encephalopathy and three cases of bleeding complications. Hepatitis was brought on by yellow phosphorus in 11 individuals (19.67%), zinc phosphide in 4, and aluminium phosphide in 3, respectively. Aluminum phosphide poisoning was the cause of cardiogenic shock[20].

According to Prosser PR et al[33], the gastrointestinal tract (100%), liver (66.7%), cardiovascular, nervous, and respiratory systems were all adversely affected by yellow phosphorus in their investigation, along with any accompanying metabolic problems (66.7%). In our study, 10% of patients exhibited respiratory depression, and 93% of patients had abnormal liver enzymes. No patient had any problems of the heart or metabolism. In a group of 15 patients, Simon FA et al[34]reported that a death rate of 27% was observed, demonstrating that Yellow Phosphorus is particularly dangerous when consumed. The prognosis is influenced by the reporting window to the hospital and compound ingestion. This is primarily due to early treatment with N acetylcysteine and stomach lavage given to patients who appear early, which reduces the quantity of yellow phosphorous entering the blood.

5. Conclusion

Mortality and morbidities especially due to toxic hepatitis and toxic myocarditis were remarkably high among the cases who consumed rat killer paste poison. In order to prevent the mortality and morbidities, the use of phosphorous in rat killer paste can be outlawed. Also appropriate health education and mental health programmes are to be implemented in order to reduce the burden of suicides.

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