ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 05 | May - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICAL PROFILE OF PATIENTS ADMITTED WITH RODENTICIDE POISON INGESTION AT TERTIARY CARE HOSPITAL

General Medicine

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ABSTRACT

Aim of the Study: The clinical profile of patients admitted with rodenticide poisoning and their course in the hospital. **Material & Methods:** 60 Patients will be selected with history of ingestion of rodenticide poison who satisfied the inclusion criteria. A detailed history regarding their complaints, onset, duration of symptoms and other relevant history was taken. **Result:** Total 60 patients, 22 patients showed SGPT values of more than Normal value (40iu/l), which almost identical with elevated bilirubin level. 10 patients had values more than five to ten times normal value, indicates acute Hepatocellular failure, it carries almost 100% mortality. 12 patients show elevated serum creatinine values, which indicates the renal toxicity of the phosphorus poison, which also carries the worse prognosis. **Conclusion:** Male to female ratio in the study is 1.7: 1, majority of the patients were in 20 to 30 years age group. Jaundice develop following ingestion of phosphorus, carries most mortality, which also correlates with elevated serum bilirubin and SGPT level.

KEYWORDS

Rodenticide poison ingestion; Serum Glutamic Pyruvic Transaminase (SGPT)

INTRODUCTION

Rodenticides are commonly available in almost every household, to prevent their stored grains from rodents, which is found everywhere. Since it is easily available and also cheaper than other pesticides in the market, it is often taken with suicidal indent or ingested accidentally by children[1].

Rodenticide is available in three different forms, as follows[2]-

1. Powder form - zinc phosphide
2. Paste form - yellow phosphorous
3. Bait form -super warfarins

Among these rodenticides, phosphorous being the most commonly and also most lethal poison, especially after 3 – 4 days of ingestion , as liver injury sets in.

Poisoning has been well documented as early as the 1950s. The number of cases of pesticide poisoning is on the increase globally, with around 300,000 cases being reported every year[3].

AIM OF THE STUDY

- To Study the clinical profile of patients admitted with Rodenticide poisoning and their course in the hospital.
- To correlate various parameters with the mortality.

MATERIAL & METHODS

60 patients who have ingested rodenticide poison, Patients coming to causality in Tertiary Care Hospital, Telangana from November 2019 to August 2021.

Inclusion Criteria:

Patient ingesting rodenticide (bait, paste or powder) immediately before admission, maximum period of within 24 hours.

Exclusion Criteria:

- Patients treated outside and referred to the hospital
- Patient presenting after 24 hours of ingestion.
- Patient consuming the poison along with alcohol or other compounds
- History of Chronic Alcoholism or Jaundice in the recent past
- Suffering from any other serious systemic diseases.

Method Of Collection Of Data

Patient admitted in emergency medical ward with history of ingesting rodenticide compound were taken as study subjects. A complete history, clinical examination of toxicity of individual compounds and certain relevant biochemical investigations were performed .patients fitting into the inclusion criteria as described by the patients or identification of rodenticide compound. After obtaining complete history, a complete clinical examination was performed, with particular importance to the vitals, jaundice, bleeding manifestations, as explained in the proforma. This evaluation was done in our emergency ward. Patient was further followed till their stay in hospital, they are noticed for development of jaundice or further prognosis of the patient and relevant investigation datas are collected.

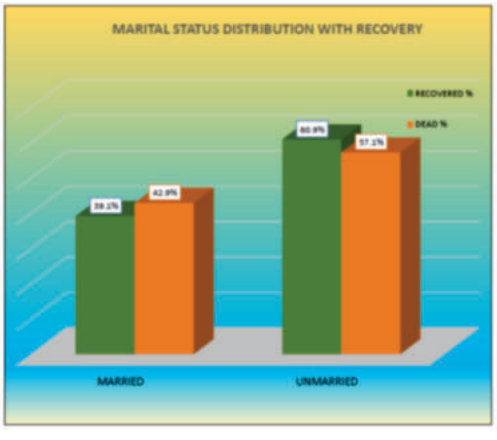
Importance to jaundice, bleeding tendencies, oliguria and seizures or headache, serum bilirubin value on admission, on 4th day and afterwards, INR value, serum creatinine and SGPT value on 4th day were collected.

All patients were managed with usual decontamination method including nasogastric lavage and treated according to nature of compound with intramuscular vitamin k and laxatives. Inj. N-acetyl cystiene was tried in intensive care unit. Counselling was given to the survivors. Autopsy was done for all expired patients.

RESULTS

Table 1: Marital Status Of The Patient

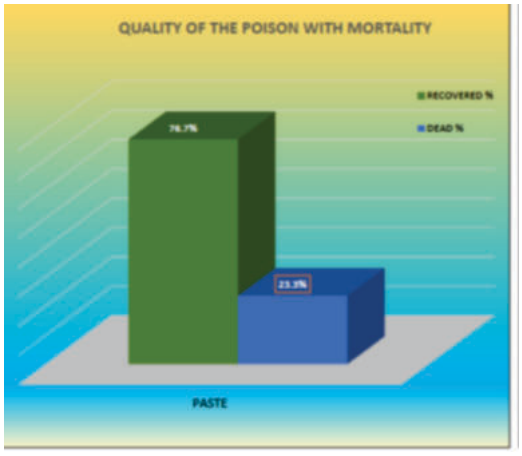
Martial Status	No. of Patients (60)	Percentage (%)
Married	24	40%
Un Married	36	60%



Graph 1: Marital Status Compared With Mortality

Table 2 : Quality of Ingested Poison

Quality of Ingested Poison	No. of Patients (60)	Percentage (%)
BAIT	8	13%
Paste	40	67%
Powder	12	20%
Total	60	100%



Graph 2 : Quality of the Poison with Mortality

Table 3: Quantity Of Poison Consumed

Quantity of Ingested Poison	No. of Patients (60)	Percentage (%)
5 gm	18	30%
10 gm	17	28%
15 gm	12	20%
20 gm	8	13%
30gm	1	2%
Total	60	100%

Table 4: Time Delay Of Patient From Ingestion To Hospitalisation

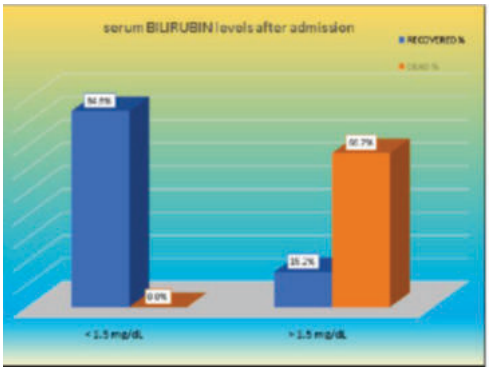
Time Delay	Ingestion to Hospitalization	Percentage (%)
< 4 hours	30	50%
4 – 8 hours	23	38%
> 8 hours	7	12%
Total	60	100%

Table 5: Clinical Manifestation

Symptoms	No. of patients	Percentage (%)
Vomiting	55	92%
Abdominal Pain	28	47%
Thirst	19	32%
Headache	5	8%
Diarrhea	25	42%
Jaundice	21	35%
Bleeding	10	17%
Oliguria	12	20%

Table 6: Serum Bilirubin Levels

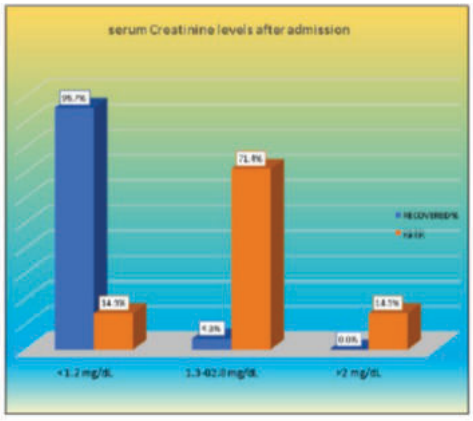
4th day of Admission	No. of Patients	Percentage (%)
< 1.5 mg/DL	39	65%
> 1.5 mg/dl	21	35%



Graph 3 : Serum Bilirubin compared with mortality

Table 7 : Serum Creatinine Value Of The Patients

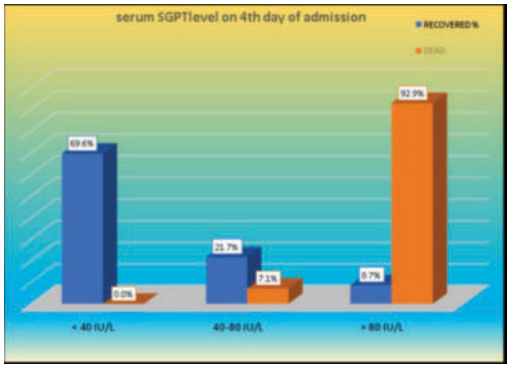
Sr. Creatinine	No. of Patients	Percentage (%)
< 1.2 mg/dl	48	80%
1.3 – 02.0 mg/dl	9	15%
> 2mg/dl	3	5%



Graph 4 : Serum Creatinine level compared with mortality

Table 8: SGPT LEVEL ON 4TH DAY OF ADMISSION

SGPT	No. of Patients	Percentage
< 40 IU/L	38	63%
40 – 80 IU/L	12	20%
> 80 IU/L	10	17%



Graph 5 : SGPT LEVEL COMPARED WITH MORTALITY

Table 9: Recovery Of The Patient

	No. of Patients	Percentage (%)
Recovered	46	77%
Dead	14	23%

DISCUSSION

In a Retrospective study conducted in south India in the year 2002, Organic Phosphorus compounds were the most common cause of poisoning (36.0%) followed by Snake bite (16.2%), Drugs (11.0%), Rat poison (7.3%) and Others[4].

A prospective study conducted from 2008-2010 in West Bengal, (total 4,432 patients) Snakebite (31.90%) was the most common cause of poisoning followed by Organophosphorus compounds (21.84%), Rodenticide (16.49%), Alcohol (13.80%), Chemicals (9.04%), and Drugs (2.3%)[5]

Rodenticide poisoning constitutes 3% of all poisonings in India. Rodenticide compounds are commonly used to kill rats, mice and rodents. They are heterogeneous group of compounds that exhibit markedly different toxicities to Humans and Rodents.

Rodenticides are composed of superwarfarins, thallium, barium carbonate, aluminium phosphide (AlP) and zinc phosphide.

They are inexpensive and highly toxic pesticides. They are easily available in the Indian subcontinent and are common agents in deliberate self-harm, especially among the agricultural community.

The high fatality due to ALF induced by rodenticides makes the need for an antidote urgent and imperative.

Elemental phosphorus is commonly available as red and white phosphorus. The latter is also known as yellow phosphorus and is used widely for military ammunition, fire crackers, fertilizers and in rodenticides, which contain yellow phosphorus in varying strengths of 2- 5%.

Rodenticides are toxic to the liver and heart. Rodenticides containing aluminium or zinc phosphide are hepatotoxic and can cause acute fulminant hepatic failure (ALF). Patients admitted with aluminium or zinc phosphide poisoning often develop hepatic necrosis, renal failure, metabolic acidosis and refractory hypotension[6].

In the absence of any known antidote for AlP poisoning, ALF in the setting of rodenticide poison consumption is often fatal, with mortality rates ranging from 40 – 80%[7].

Yellow phosphorus is readily absorbed by the skin and mucous membranes, especially the gastrointestinal tract.

Skin exposure following industrial accidents causes severe thermal burns. Once absorbed, the level of phosphorus in the blood, renal and hepatic system increases within a few hours.

In one study, the authors stated that zinc phosphide rodenticide poisoning is a cause of acute liver failure in many Indian patients.

Aluminium phosphide (AlP) and zinc phosphide are the hepatotoxic forms of rodenticide. AlP solid fumigant and is available in the form of dark grey tablets. Commercial names include celphos, alphos, quickphos and phostoxin[8].

When ALP comes in to contact with moisture it liberates the highly toxic phosphine gas. Phosphine inhibits cellular oxygen utilization by its effect on mitochondria, and also by decreasing the activity of cytochrome oxidase[9]. It also liberates highly reactive hydroxyl radicals[10].

Reactive oxygen species toxicity is believed to be key in the genesis of AlP induced toxicity. Rodenticides also contain 2 – 5% of yellow phosphorus. Phosphorus poisoning is also known to cause ALF[11,12]

Toxicity of ingested yellow phosphorus is through direct tissue damage by exothermic production of phosphoric acid causing tissue corrosion, and the formation of phosphorus pentoxide which reacts with organic molecules. In addition, hypocalcaemia results by preferential binding of calcium by phosphorus in serum proposed that toxicity may be the result of changes in ribosomal function and protein synthesis, failure of regulation of blood glucose, alteration of lipoprotein synthesis and secretion of triglycerides, leading to intracellular accumulation and fatty degeneration of multiple organs, especially the liver, the kidney, the heart and brain.

Acute liver failure (ALF) is a rare and severe, life threatening medical emergency. It is defined as the rapid development of acute liver injury with impaired synthetic function (INR > 1.5) and encephalopathy in a person who previously had a normal liver[13].

Causes of ALF include infections, metabolic derangements like Reyes syndrome and acute fatty liver of pregnancy. Toxins like paracetamol, tetracycline, and Amanita phalloides can also cause ALF.

The clinical features of ALF include progressively worsening jaundice, fetor hepaticus and coma. Biochemical derangements include increased INR and elevated transaminases.

Of the 60 patients included in the study, majority were younger patients under the age group of 40yrs and all were suicidal in intent. shows the high risk of the younger age group towards suicidal tendencies.

In this study , most number of the patient were in the age group of 21 to 30 yrs (58%), of less than 30 yrs being 73%. maximum number of patients was 21 to 30 years as compared with p.s Shankar et al[14] and Doshi et al study[15].

Various studies on poisoning done in India Indranil Banerjee et al[5] in west Bengal, K N Ramesha et al[4] in Karnataka and Gupta S et al in Gujarat also noticed most commonly effected age group was 20 – 40yrs.

In this study there was no significant difference in both incidence and mortality in age groups of the patients.

In this study, majority of the patients were unmarried being 36 patients (60%), while married patients being 24 (40%).

Murali N et al [16] in their study found that 69 % belonged to lower SES and 31 % belonged to Middle SES.

In this study, there is no significant difference in mortality in both married and unmarried groups, both groups carrying mortality of 42.9% and 57.1 % in each.

In this study, the most common symptom was vomiting observed in 92 % of the patients, followed by abdominal pain (47%), next by diarrhoea (42%) and thirst (32%). However, jaundice is observed only in 21 patients (35%), but mortality is higher.

In a study by Gopalakrishnan S et al, they observed that 72.73% of patients manifested after 24 hours of poison exposure. The predominant features included abdominal pain (52.53%), jaundice (22.21%), bleeding manifestations (15.15%), encephalopathy (10.10%), shock (10.10%), AKI (7.08%) and multi-organ dysfunction (17.17%).

Murali N et al [16] in their study found that, in Rodenticide poisoning common GI side effects like nausea, vomiting is seen in 83 members (85.7%) and Abdominal pain in 55 (56.7%). These findings are comparable to previously done studies. Bleeding manifestations are seen in 16 (16.4%), Icterus is seen in 46(46.3%) and Altered Sensorium seen in 29 (29.8%).

In this study, 21 patients (35%- almost all ingested phosphorus compound) showed elevated serum bilirubin on 4th day of more than 1.5 mg, out of which 14 patients (100%) had expired, which also correlate with elevated SGPT values measured on 4th day; indicating the acute hepatocellular toxicity of the phosphorus compound, measuring more than five times the normal value of SGPT.

Serum creatinine was also elevated in 12 patients ingesting phosphorus compound.

Murali N et al, noticed that, mortality in patients who presented with ingestion of Zinc Phosphide was 35.7% and patients who lost follow up 7.1%.

Study done by chugh et al16 on zinc phosphide showed mortality of 25%. Study done in Tehran by Abdollahi M et al [17] showed mortality of 28.3%.

In this study, 21 patients (35 %) presented with jaundice of which 14 patients (100 %) were expired. only one patient recovered, who was been treated with N-acetyl cysteine from the day of admission, who had only survived among all the icteric patients.

In this study, 21 patients showed elevated bilirubin of more than 1.5 mg/dl, out of which 14 patients (67%) had expired, others 39 who recovered 84.8% show only mild increase, which on subsequent days decreased and patients improved.

In this study, 17 patients (28%) showed elevated SGPT value of more than 80 IU/ L, out of which 13 patients (92.9%) expired and 4 patients recovered(8.7%). 11 patients (18%) showed values of 40 to 80 iu /l, and 10 (21.7%) recovered and one dead (7.1%). Thus SGPT value of more than five times normal along with elevated bilirubin and jaundice carries nearly full mortality.

Literature search revealed that AST/ALT elevations >10 times the normal value, derangements in PT/INR, metabolic acidosis, and hypoglycemia were important port enders of death.

Nalabothu M, et al[18] studied that three of the patients in their study who came with consumption of zinc phosphide had Acute Pancreatitis. Sarma et al[19] have reported that zinc phosphide ingestion leads to Acute Pancreatitis.

In this study, 14 patients (20%) showed elevated serum creatinine values, out of which all patients (100%) had expired, who also had oliguria. Thus it is not of much significance as other parameters in determining the mortality. Thus out of 60 patients got admitted, 14 patients had expired, all of them had ingested paste – phosphorus compound.

Chi square test shows, there was no significant association between age, sex, socio-economic status, marital status, abdominal pain, bleeding manifestation, duration of stay and outcome of the patient, which shows calculate value greater than table value ($p > 0.05$).

But there was significant association between quality, quantity, time delay, thirst, diarrhoea, jaundice, oliguria, head ache, serum bilirubin 4th day, SGPT, creatinine value of the respondents and their outcomes. They show calculate value less than table value ($p < 0.05$).

In one study of zinc phosphide poisoning, only 4 of 102 patients died. Conversely, in a smaller study, 5 of 20 patients died (25% mortality rate). The mortality rates in a study by Hassanain et al[20], which included 455 patients, was low (<10%). However, the other studies were performed in different countries that might have used different zinc phosphide products or different management protocols; this may have contributed to the differences in the mortality rates.

Inference

There is a significant difference between bilirubin on admission, 4th day, >1 wk, sgpt, INR, creatinine of the respondents and their quality. Hence the calculate value less than table value ($p<0.05$).

Phosphorus-based rodenticides claim many human lives. Given the fact that there is no specific antidote, constant vigil and timely resuscitation could be the only factors to tilt the balance favorably.

Stringent legislative measures to restrict the indiscriminate and over-the- counter sales of yellow phosphorus are much needed in our country. In this study, we identified delayed resuscitation, jaundice, hepatic encephalopathy, elevation of AST and ALT to >1000 IU/L, metabolic acidosis, and refractory shock as the significant predictors of bad outcome in this patient population. The common mode of death was fulminant hepatic failure.

Despite all patients receiving supportive therapy, a poor outcome or death resulted in the majority. Early referral to a tertiary care center, meticulous monitoring and supportive measures are key elements of patient management as there are no specific antidotes available at present. Increase in public and physician awareness to the toxin and implementation of preventive policies is of utmost importance.

CONCLUSION

1) Majority of the patients were in 20 to 30 years age group.
2)Phosphorus compound (paste) forms the major share of poison.
3)Bleeding manifestation is common in warfarin group (bait).
4)Mortality in this study was 23 %.
5)Increased time delay in hospitalisation carries more mortality.
6)Mortality is seen only in phosphorus compound.
7)Jaundice develop following ingestion of phosphorus, carries most mortality, which also correlates with elevated serum bilirubin and SGPT level.

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