



## An Observational Study On Rat Killer Poisoning And Earlier Plasma Exchange Treatment In A Tertiary Care Hospital

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### Abstract

**Background:** Rat killer poisoning is one of the most commonly encountered poisoning in the country. There are many rat killer compounds available such as aluminium phosphide, zinc phosphide, thallium, strychnine, yellow phosphorous and sodium monofluoroacetate.

**Methods:** The study was conducted in a tertiary care hospital. 38 patients with a history of ratkiller ingestion were followed up for complications, PLEX was initiated and hence outcomes were compared.

**Results:** Total patients : 38, Male : 34 ( 89%) ,Female : 4 (11%). Rat killer cake was ingested by 21% , powder form by 68% and paste by 11 %. Liver function test were deranged in 5 patients . Highest value of serum bilirubin was 6.6 mg/dl. The AST, ALT, ALP levels did not raise or showed a decline after initiation of PLEX. Yellow phosphorous compounds in rat killer paste showed more toxicity in the form of exponential raise in liver enzymes whereas other compounds did not show much elevation. When compared among the patients who had undergone PLEX, those who had their first cycle of PLEX done earlier and with lower bilirubin values showed better decline in liver function tests and required lesser cycles.

**Conclusion :** Ratkiller paste contains yellow phosphorous compounds that are extremely harmful to the liver when compared to other compounds. If left untreated, mortality might reach as high as 100%. But in this study, a vigilant approach with earlier Plasma exchange (PLEX) has demonstrated a remarkable response and reduced mortality. As a result, PLEX can successfully be used to treat ratkiller paste toxicity and reduce mortality. Hepatotoxicity from other compounds, such as zinc phosphide and superwarfarins, was not as severe as it was from yellow phosphorous, but it still requires prompt treatment with drugs like N-acetyl cysteine to avoid complications.

**Keywords:** Rat killer, yellow phosphorous, plasma exchange

### Introduction

Rat killer poisoning is one of the most common toxicological encounters across the country. Morbidity and mortality rate is very high in yellow phosphorous compounds with fulminant liver cell failure occurring in many patients. Pro-inflammatory cytokines, which cause multi-organ failure, are eliminated by plasmapheresis in addition to supporting the primary functions of the failing liver. Coagulation factors, albumin, and immunoglobulins

are replaced by the replacement fluid. The whole process enhances the liver's microenvironment, which quickens regeneration and aids in functional recovery.

### Materials And Methods :

1. The study was conducted in a tertiary care hospital. A total of 38 patients who fulfilled the criteria were further followed up to note any

complications and to identify the efficacy of early PLEX therap.

2. The dialysis machine used for plasma exchange was 4008 S Fresenius and the filter used was Plasma Flux P2 filter.
3. Of the 38 cases with a definite history of rat killer paste poisoning, patients who had deranged INR, hepatic encephalopathy and elevation in the liver enzymes were taken up for plasmapheresis and the outcomes were compared.

**Inclusion Criteria**

Patients admitted to a tertiary care hospital with rat killer poison intake were studied.

**Exclusion Criteria:**

1. Patients who consumed rodenticide mixed with other poisons.
2. Patients who were <18 years of age.
3. Patients with preexisting chronic liver disease and chronic kidney disease .

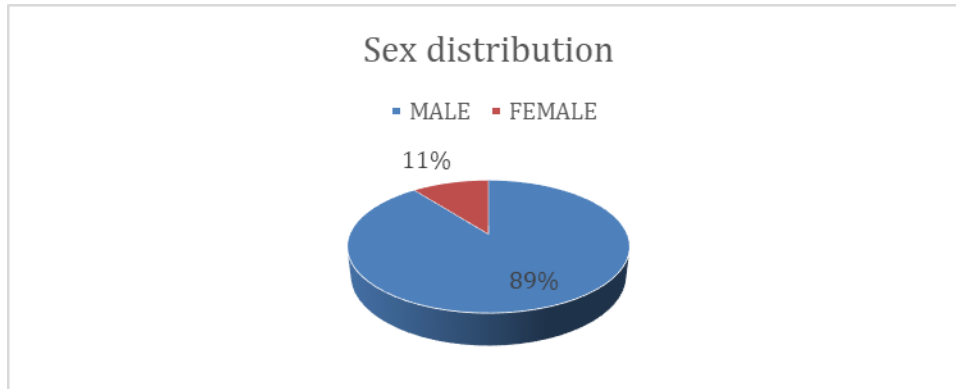
**Results :**

The following results were obtained from the study

Total patients : 38

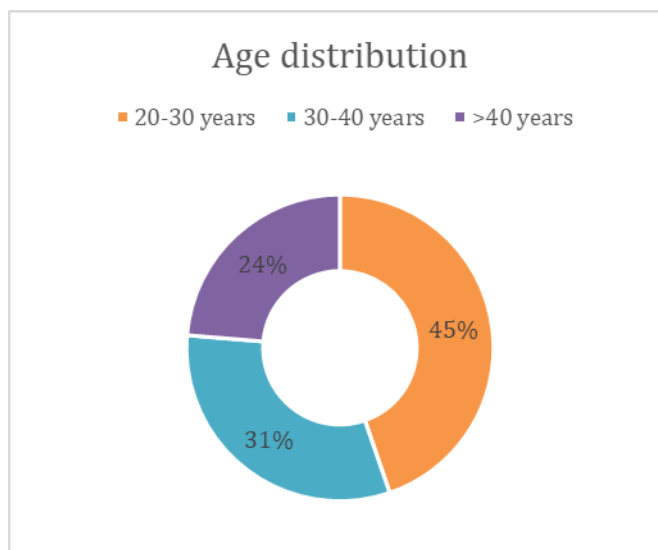
**Sec distribution**

Gender	No.
Male	34
Female	4



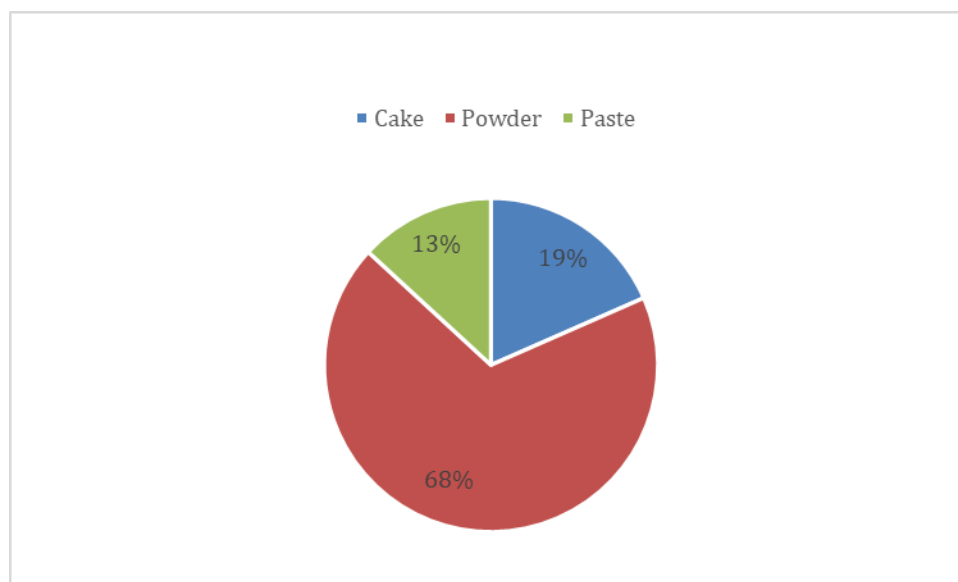
**Age distribution**

AGE	No.
20 -30	17 (45%)
30-40	12 (31%)
>40	9 (24%)

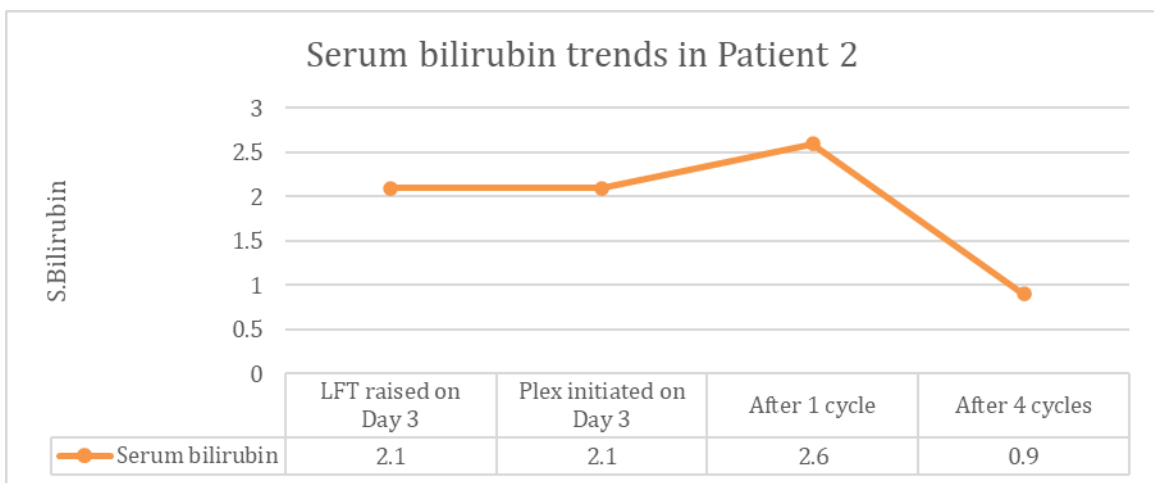
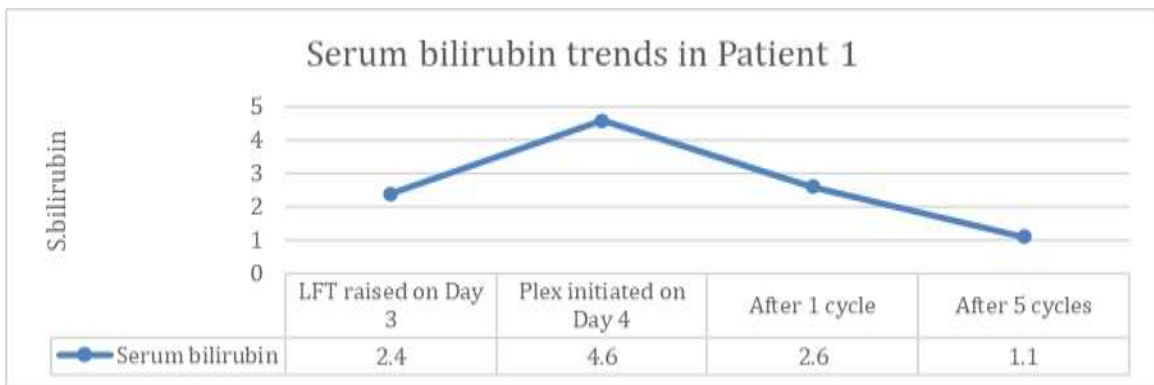
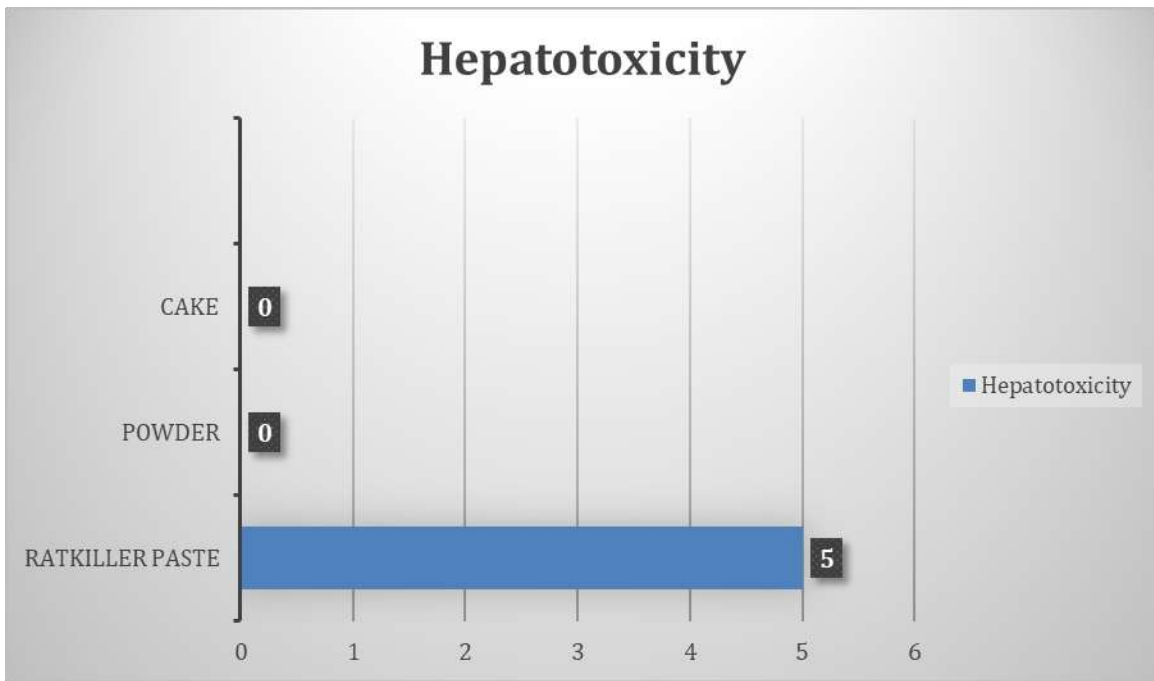


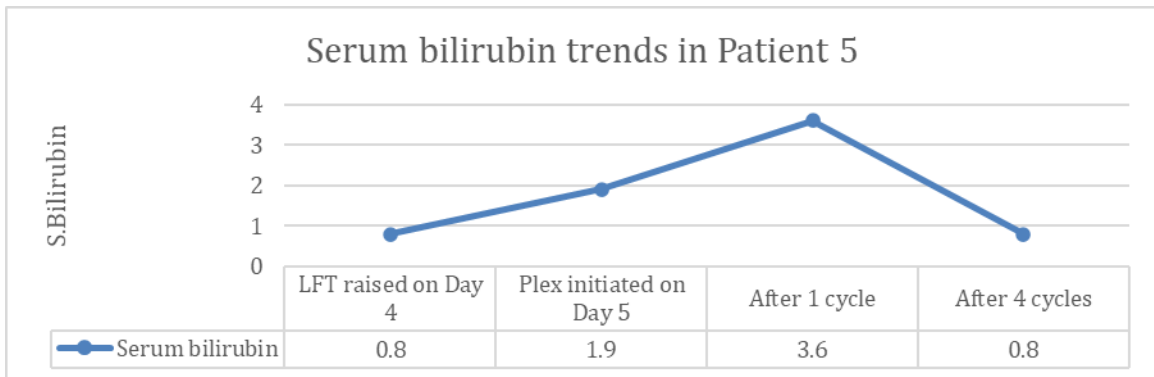
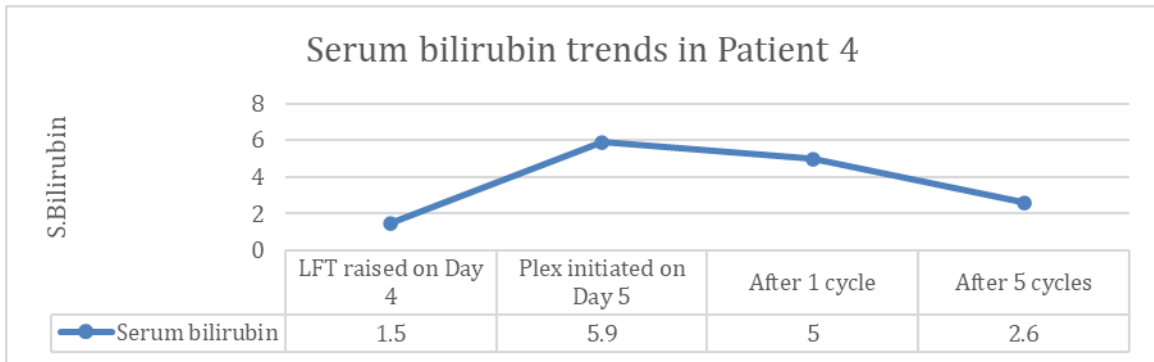
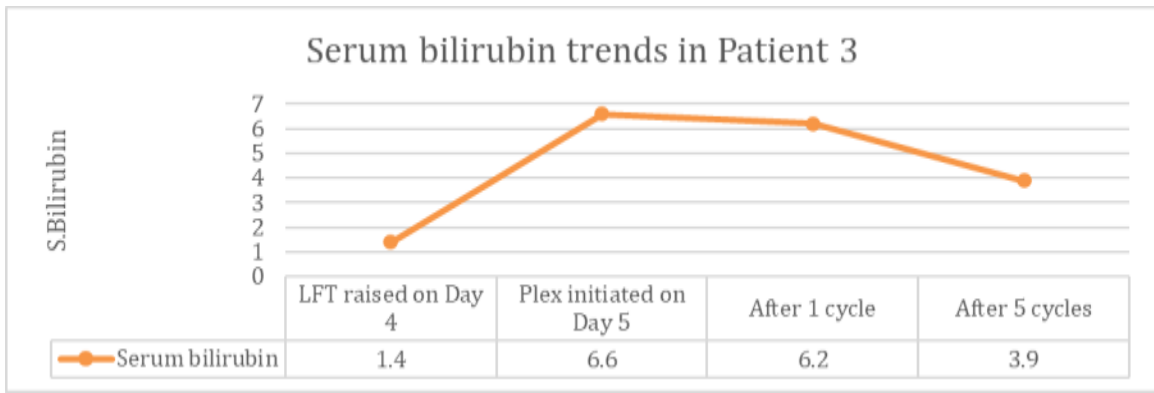
**FORM of RAT KILLER POISON**

FORM	No.
Cake ( bromadiolone- superwarfarin)	7 (19%)
Powder (Zinc phosphide)	26 (68%)
Paste ( Yellow phosphorous)	5(13%)



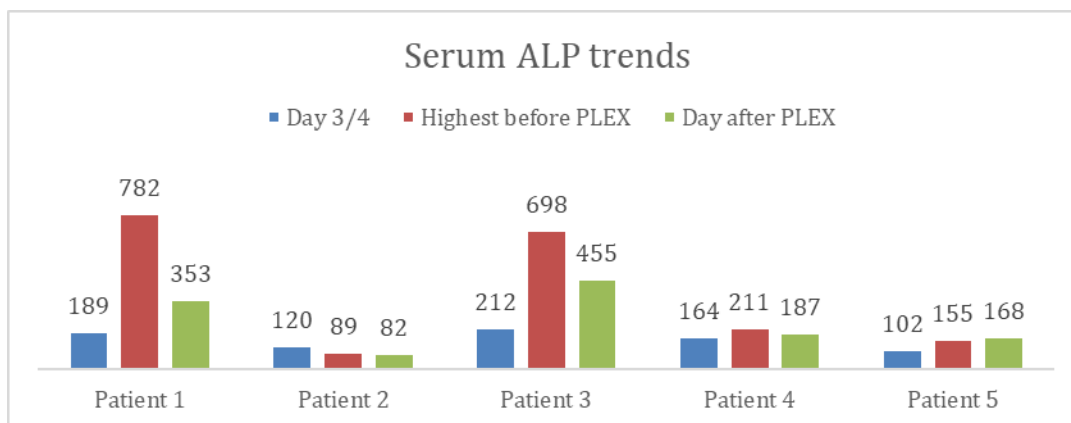
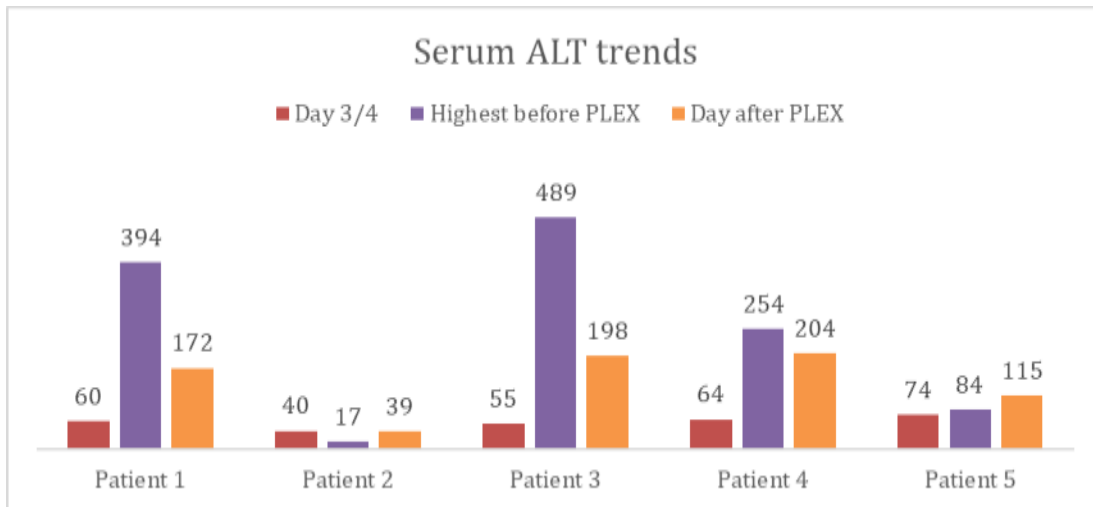
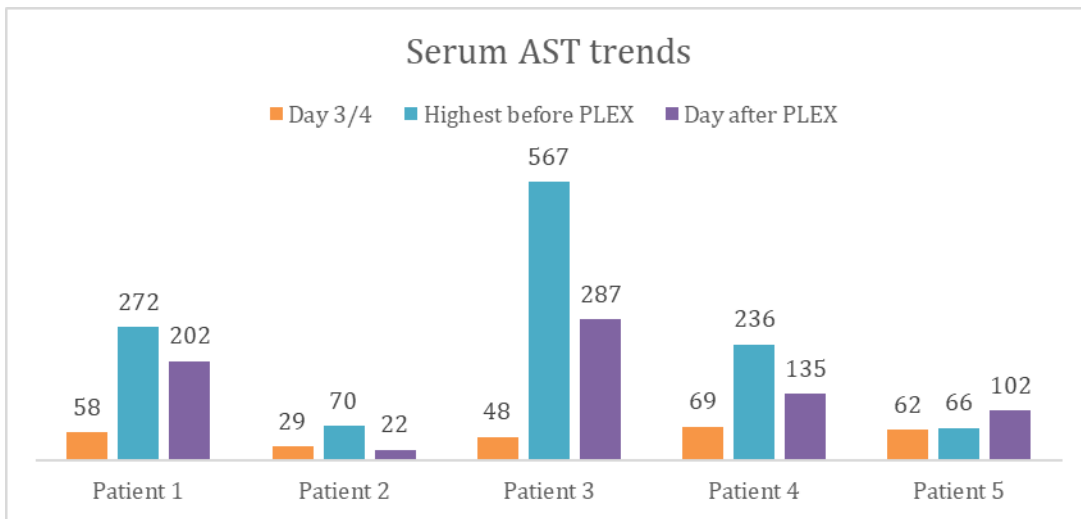
1. LIVER enzymes and bilirubin elevated in 5 PATIENTS- 13.15%
2. Patients who consumed yellow phosphorous compounds showed dramatic increase in liver function tests when compared to other compounds.
3. BILIRUBIN HIGHEST 6.6 mg/dl





Patients 1,3 and 4 had higher bilirubin and enzymes values when PLEX was initiated and hence a lesser decline and needed about 5 cycles to show a considerable fall. Whereas patients 2 and 5 were started earlier with lesser derangement of liver function tests and showed drastic fall or no exponential raise by 4 cycles.

When compared among the patients who had undergone PLEX, those who had their first cycle of PLEX done earlier and with lower bilirubin values showed better decline in liver function tests and required lesser cycles.

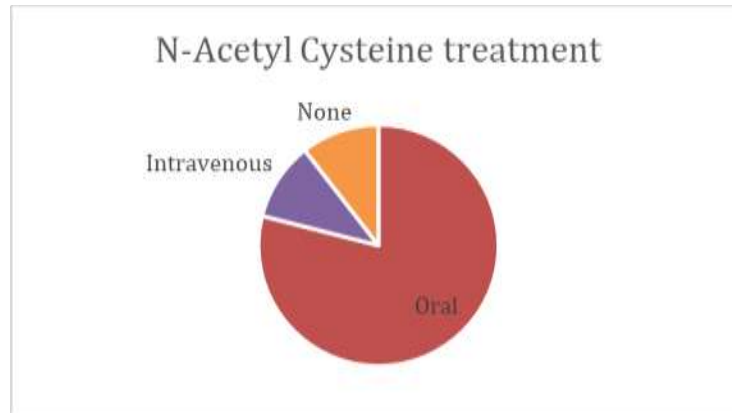


Serum aspartate aminotransferase and alanine aminotransferase levels declined after initiation of plasma exchange or did not show an exponential raise.

Serum alkaline phosphatase levels responded well to early initiation of Plex

## NAC

1. oral 30 ( 79% ) , intravenous 4 (10.5% ) , not given 4 (10.5%)
2. 19 patients had taken alcohol mixed with rat killer poison ( 50%)
3. 36 patients had no comorbidities ( 94.74%)



1. PLEX : Plex done in 5 patients
2. Paste produced 100 % acute hepatitis
3. Nil mortality in PLEX done patients

## Discussion

All the patients in the study were followed up for the development of complications such as increase in INR, hepatic encephalopathy, bleeding manifestations and fulminant liver cell failure. The patients who showed such features were immediately started on plasma exchange and the response was monitored. Patients who were initially started with early Plex were prevented of dreadful complications and did not show any mortality. The maximum number of cycles that a patient needed was around 5 cycles of plasma exchange. PLEX was done earlier in patients who showed bilirubin elevation and enzyme elevation. This has led to dramatic response in reducing the toxic effect of rat killer poison and hence has been life saving in these patients. Early initial of PLEX is instrumental in avoiding the catastrophic effects.

Rat killer compounds available are aluminium phosphide, zinc phosphide, thallium, strychnine , yellow phosphorous and sodium monofluoroacetate. Yellow phosphorous compounds in rat killer paste showed more toxicity in the form of exponential raise in liver enzymes whereas other compounds did not show much elevation.

Aluminium phosphide liberates a highly toxic phosphine gas when it comes in contact with water or moisture or with hydrochloric acid in the stomach[1-3]. Toxicity to humans is most commonly after ingestion, although toxicity from inhalation and absorption from the skin are possible[2,3]. When it is ingested, phosphine gas is released from the reaction with HCl and water in the stomach. This highly toxic phosphine gas then diffuses through the gastrointestinal tract and distributed throughout the body resulting in systemic toxicity. Phosphine inhibits cytochrome c oxidase which leads to the inhibition of oxidative phosphorylation and cellular respiration by up to 70%. Overproduction of reactive oxygen species with subsequent cellular damage leads to eventual cell death[4]. Organs with higher oxygen demands such as the heart, lung, kidney, liver and brain are more sensitive to damage involving oxygen free-radicals production[5].

Aluminium and zinc phosphides are highly potent insecticides and rodenticides that are widely accessible on the market in powder, pellet, or tablet form. Acute poisoning with these compounds can occur through direct ingestion of the salts or indirectly through accidental inhalation of phosphine produced during their approved use. Death usually

occurs within the first day of severe metallic aluminium phosphide poisoning. Death is usually caused by cardiac arrhythmias, refractory shock, or cardiac failure[6].

Elemental phosphorous is found in two forms: red and yellow. The red form is employed in the creation of matchsticks, is not absorbed, and has low toxicity. Yellow phosphorus compounds are widely employed as rodenticides, fertilisers, and in firecrackers and are widely available in Tamil Nadu. Phosphorous is easily absorbed by the GI system, resulting in high phosphorus levels in the kidney and liver within hours. Yellow phosphorous induces cardiac, hepatic, renal, and multiorgan failure in the same way that zinc and aluminium phosphides do.

Therapeutic plasma exchange is defined as the removal of patient's plasma and replacing it with plasma from a donor along with colloid by using an extracorporeal device. It is an effective method for the removal of accumulated toxins from plasma in liver failure patients [7,8]. Though TPE reduces blood ammonia levels, it has an added advantage of providing deficient clotting factors and albumin[9].

### Conclusion

Yellow phosphorous compounds in ratkiller paste are highly detrimental when compared to other compounds in terms of liver toxicity. If untreated mortality is as high as 100%. However in this study. A watchful strategy with an earlier initiation of Plasma exchange (PLEX) has shown a dramatic response and reduced morbidity and mortality . Hence ratkiller paste poisoning can be effectively treated with PLEX and mortality can be prevented. Hepatotoxicity from other compounds, such as zinc phosphide and superwarfarins, was not as severe as it was from yellow phosphorous, but it still requires prompt treatment with drugs like N-acetyl cysteine to avoid complications.

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