Clinical Audit

Clinical profile and therapeutic outcomes of yellow phosphorus (Ratol) poisoning: A retrospective analysis at Kauvery Hospital, Trichy

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Abstract

Background: Ingestion of yellow phosphorus (YP), primarily in the form of "Ratol" paste, is a leading cause of toxic hepatitis and fulminant hepatic failure (FHF) in Southern India. Despite the 2022 ban on 3% YP paste in Tamil Nadu, cases continue to present at tertiary centers.

Methods: A retrospective study of 67 patients admitted over one year in multispecialty tertiary care hospitals of Kauvery (Pan-Kauvery audit) was conducted. Data on demographics, formulation types, treatment (NAC and Plasmapheresis), and survival outcomes were analyzed.

Results: Of 67 cases, 67% were male (n=45). The "Paste" formulation was the primary toxicant (n=37). Management included N-acetylcysteine (NAC) and therapeutic plasma exchange (PLEX). The overall survival rate was 70.1%, with a 9% mortality rate and 21% discharged AMA.

Conclusion: Early initiation of NAC and PLEX as a "bridge to recovery" significantly improves survival in resource-limited settings where liver transplantation is not immediately available.

Key words: Yellow phosphorus (YP); Plasma exchange (PLEX); N-acetylcysteine (NAC)

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1. Introduction

Yellow phosphorus is a potent, non-metallic protoplasmic poison. In India, it is commonly available as "Ratol" (3%–5% concentration) and is frequently used for self-harm due to its low cost and high lethality [1]. While Western nations primarily deal with industrial exposure, the Indian subcontinent faces an epidemic of intentional oral ingestion.

In late 2022, the Government of Tamil Nadu banned the sale of 3% YP paste due to its high association with suicidal fatalities. However, existing stocks and alternative formulations (powders/cakes) ensure that it remains a critical public health challenge [4]. This study evaluates the clinical efficacy of current treatment protocols at across the Kauvery Hospitals, focusing on non-transplant options for liver salvage.[2]

2. Pathophysiology: How It Acts

Yellow phosphorus is rapidly absorbed (within 2–3 hours) from the gastrointestinal tract.[5].

Tissue Accumulation: Approximately 70% accumulates in the liver, with the remainder affecting the heart (12%), kidneys (4%), and brain.

Cellular Toxicity: It inhibits protein synthesis by causing ribosomal dissociation and interferes with carbohydrate and fat metabolism.

Hepatic Impact: The toxin leads to centrilobular necrosis and microvesicular steatosis. This results in the depletion of ATP and profound oxidative stress, triggering Fulminant Hepatic Failure (FHF).

3. Clinical Stages of Toxicity

The clinical course is classically divided into three distinct phases:

Stage I (0–24 hours): Local irritation; symptoms include vomiting (often luminescent/garlic odor) and abdominal pain.

Stage II (24–72 hours): The "Latent Period." The patient appears clinically stable, but biochemical markers (AST/ALT) begin to rise exponentially.

Stage III (>72 hours): Systemic toxicity manifests as jaundice, encephalopathy, coagulopathy, and multi-organ dysfunction (MODS).

4. Materials and Methods

This was a retrospective study conducted at all the multispecialty units of Kauvery Hospitals, India.

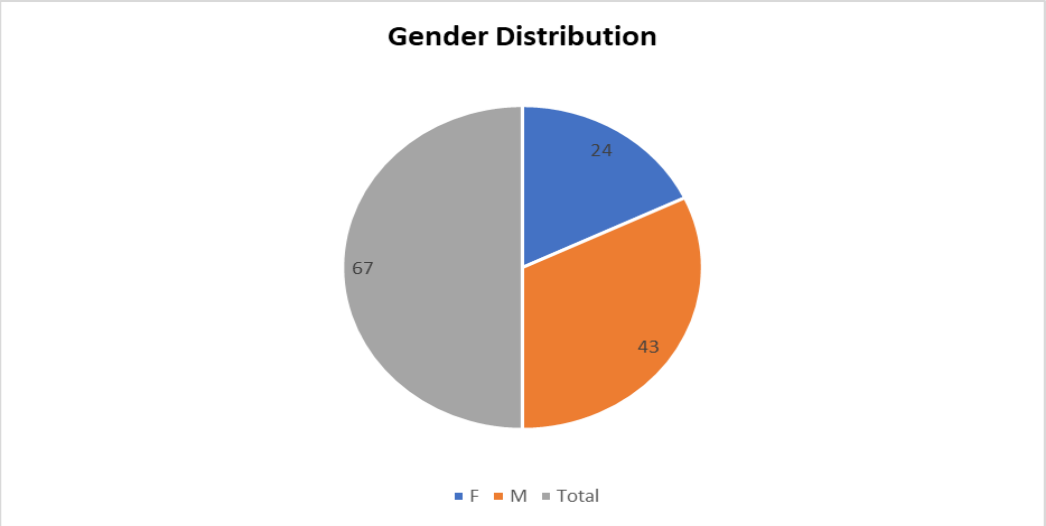
Sample Size: 67 patients with confirmed history of Ratol ingestion.

Inclusion Criteria: Patients >18 years with documented ingestion of Paste, Powder, or Cake formulations.

Data Collection: Derived from hospital records including treatment protocols (NAC infusions, PLEX sessions) and final disposition (Alive, Death, or AMA).

5. Results

Demographic Analysis

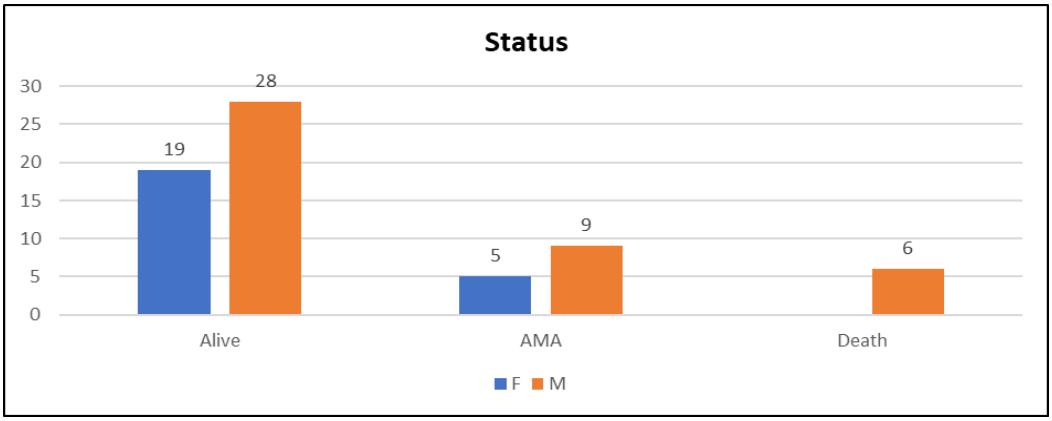


Gender Distribution: A clear male preponderance (43 vs. 24) suggests higher occupational or social stressors in the male cohort in the Trichy region.

Chronological Trends: Incidence peaked in September (n=12) and January (n=11). This "month-wise" seasonality often correlates with agricultural cycles and economic distress.

Age Profile: Literature suggests the 20–40 age group is most vulnerable, which aligns with the active workforce demographics of this study.

Patient Status



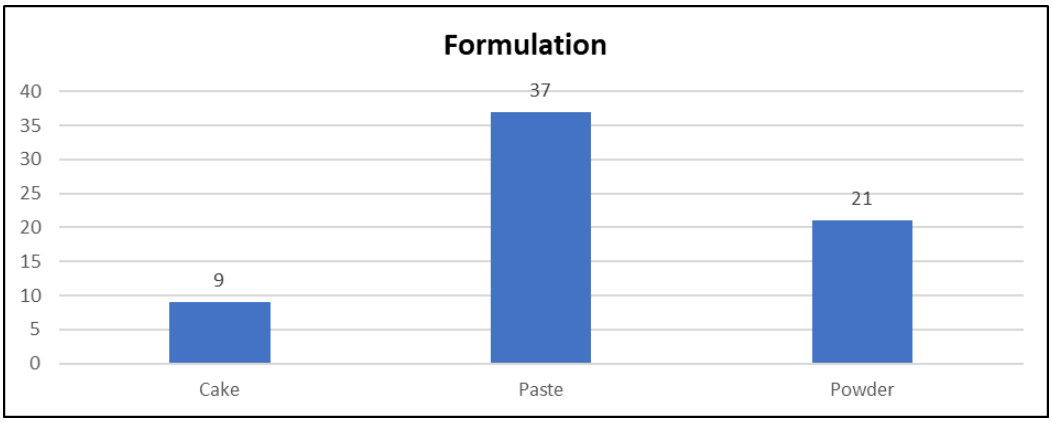
Alive (Discharged): 47 patients (70.1% Survival Rate).

AMA (Against Medical Advice): 14 patients (20.9%).

Death: 6 patients (9%) (4 from ratol paste, 2 from ratol powder).

6. Formulation Protocol and Clinical Distribution

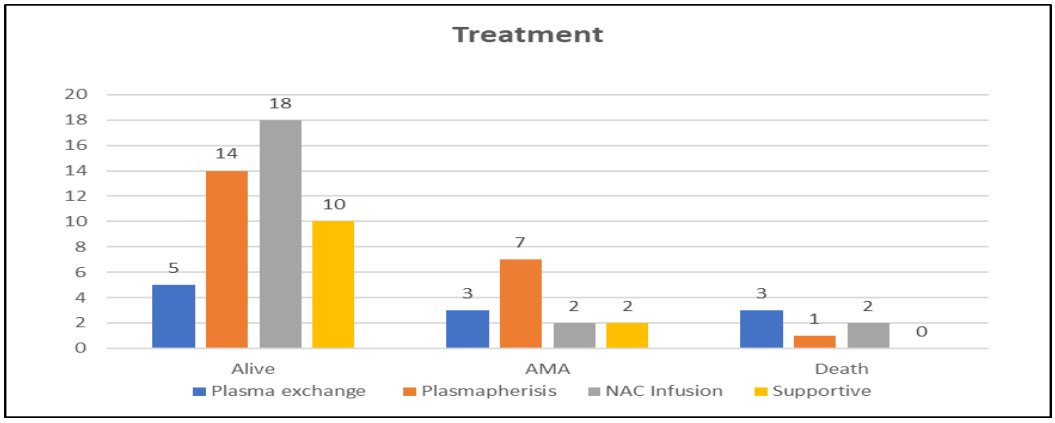
The data categorizes cases based on the physical form of the toxin ingested. The physical state appears to influence the volume of ingestion and subsequent absorption rates.



Formulation	Case Count	Percentage
Paste (Ratol)	37	55.2%
Powder	21	31.3%
Cake	9	13.5%
Total	67	100%

Paste formulation is the most common medium for ingestion, likely due to its high accessibility and concentration of yellow phosphorus.

7. Treatment Analysis



Treatment Modality	Frequency
NAC Infusion	High (Baseline) 22
Plasmapheresis (PLEX)	22 cases
Plasma Exchange	11 cases
Supportive	12 cases

In the study group, all patients received an NAC infusion. Depending on the severity of their condition following the infusion, some patients also underwent either plasmapheresis or plasma exchange.

Supportive therapy (Pantoprazole, Ondansetron, Liver Protectant: Ursocol, Thiotres, Vitamin K) were used only in mild cases.

Survival Rate: 70.1% (47/67).

Mortality: 9% (6/67). This is significantly lower than regional estimates (which range from 25% to 50%), highlighting the efficacy of the tertiary care interventions at Kauvery Hospital.

The "AMA" Factor: 20.9% (14 cases) were discharged Against Medical Advice. These likely represent patients in the latent phase or those facing financial constraints, who may have progressed to post-discharge mortality.

8. Discussion

The "Paste" formulation presents the highest mortality risk due to superior bioavailability followed by cake formulation, both caused 9% mortality rate at Kauvery Hospitals, compared to 28% in studies like Nalabothu et al., we have recorded less amount of mortality. Our study confirms that early plasmapheresis is a life-saving intervention when administered within 24 hours of ingestion. By rapidly clearing toxins from the blood, the procedure significantly reduced critical markers of liver damage.[6]. In our study population 6 deaths were recorded among them 4 deaths due to ratol paste and 2 deaths from ratol powder. This clearly showed that paste formulation is more dangerous than powder formulation due to the ingredient (yellow phosphorus) and its nature of fast absorption.

This study results the importance of a protocol featuring early plasmapheresis (within 24–48 hours) to mechanically remove the toxin and the off-label use of NAC for glutathione replenishment and enhanced oxygen delivery.

9. Conclusion

In conclusion, phosphorus-related hepatotoxicity from "Paste" ingestion can be managed through early intervention, specifically by prioritizing early Plasmapheresis and off-label NAC to prevent irreversible hepatic necrosis. Future efforts should focus on rapid identification of the toxin and immediate, aggressive, combined treatment protocols to reduce mortality.

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