

# Role Of Plasmapheresis In 3% Yellow Phosphorus Poisoning And Its Outcome

# Dr. Haneendhar R<sup>1</sup>, Dr. Sreelakshmi V K<sup>2</sup>, Dr. Melvin Dominic\*<sup>3</sup>, Dr. Sai Vineeth Moturi<sup>4</sup>, Dr. Arul Nivi P<sup>5</sup>, Dr. Pragnesh Rathnasami<sup>6</sup>, Dr. Alen Hussain Kalakkat<sup>7</sup>

<sup>1</sup>Post-graduate 3rd year, M.D Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem. Email ID: haneendhar@gmail.com

<sup>2</sup>Post-graduate 3rd year, M.D Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem

\*3 Associate Professor, Department of Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem

<sup>4</sup>Post-graduate 2nd year, M.D Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem

<sup>5</sup>Post-graduate 1st year, M.D Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem

<sup>6</sup>Post-graduate 1st year, M.D Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem

<sup>7</sup>Post-graduate 1st year, M.D Emergency medicine, Vinayaka Missions Kirupananda Variyar Medical College and Hospital, Chinnaseeragapadi, Salem

# \*Corresponding Author:

Dr.Melvin Dominic

Email ID: dr.melvindominic@gmail.com

Cite this paper as: Dr. Haneendhar R, Dr. Sreelakshmi V K, Dr. Melvin Dominic, Dr. Sai Vineeth Moturi, Dr. Arul Nivi P, Dr. Pragnesh Rathnasami, Dr. Alen Hussain Kalakkat, (2025) Role Of Plasmapheresis In 3% Yellow Phosphorus Poisoning And Its Outcome. *Journal of Neonatal Surgery*, 14 (19s), 38-43.

## **ABSTRACT**

**Background and Objectives:** Yellow phosphorus, commonly found in rodenticides, is a highly toxic substance frequently used in developing countries like India. Poisoning due to ingestion is often accidental or intentional, with a lethal dose of approximately 1 mg/kg. There is no specific antidote, and treatment primarily involves supportive care. This study evaluates the role of plasmapheresis in managing acute liver injury caused by yellow phosphorus poisoning.

**Methods:** A retrospective case analysis was conducted at VMKVMCH, Salem, Tamil Nadu, on patients admitted with yellow phosphorus poisoning between January 2021 and March 2022. Medical records of these patients were reviewed. Baseline vital parameters and laboratory investigations (liver biomarkers, renal parameters, serum electrolytes, coagulation profile, arterial blood gas analysis, and imaging studies). Daily monitoring of blood investigations and clinical status. Statistical analysis using SPSS version 23, with numerical data presented as mean, standard deviation, and categorical data as percentages.

**Results:** Early plasmapheresis (within 24 hours of ingestion) was associated with better clinical outcomes. Plasmapheresis reduced bilirubin, SGOT, SGPT, and INR levels significantly (p<0.05), though liver function parameters showed temporary elevation post-procedure. It was effective in preventing rapid deterioration, improving initial recovery, and stabilizing coagulation parameters. The overall survival rate was 67% in patients receiving plasmapheresis compared to a previously reported 76.2% mortality rate with standard care.

**Conclusion:** Yellow phosphorus poisoning predominantly affects young adults, with a female preponderance. Acute liver injury develops within 3–4 days post-ingestion, and early plasmapheresis significantly improves prognosis. Patients presenting within 24 hours of poisoning respond well to treatment, reducing the need for liver transplantation. Plasmapheresis plays a crucial role in managing toxin-mediated acute liver injury and should be considered as an early therapeutic intervention.

**Keywords:** Yellow phosphorus poisoning, plasmapheresis, acute liver injury, liver failure, rodenticide toxicity, coagulopathy, therapeutic plasma exchange, toxic hepatitis, poisoning management, prognostic factors.

#### 1. INTRODUCTION

The 3% yellow phosphorus is a rodenticide and are widely used in developing countries especially in India and also easily available. The 3% yellow phosphorus consumption is mostly accidental or intentional. The lethal dose is around 1mg/kg. Yellow phosphorus is a protoplasmin toxin affecting the hepatic, gastrointestinal, cardiovascular and renal systems.[1,2]

The poisoning patients present in ER with mild symptoms such as nausea, vomiting, abdominal pain, and diarrhoea, later on develops, jaundice, bleeding, hypoglycemiia, hypotension, confusion, hallucinations. If untreated death occurs due to Coagulopathy, internal bleeding, fluminent liver cell failure, hepatic encephalopathy and cardiovascular collapse.[3,4]

There is no specific antidote for 3% yellow phosphorus poisoning. Initial treatment includes decontamination and supportive therapy. Use of N-acetyl cystenine and Glutathione is well documented for paracetamol poisoning and has also been studied in acute liver failure due to 3% yellow phosphorus poisoning[5], hence we are starting these medication as early as possible.

Early plasmapheresis is effective tool in management of acute live injury in 3% yellow phosphorus poisoning and reveals the best outcome [6,7,8], hence I am taking the study on role of plasmapheresis in 3% yellow phosphorus poisoning patients and it's outcome.

#### 2. MATERIALS AND METHODS

It was a Retrospective case analysis conducted on the yellow phosphorus poisoning patients who were admitted between January 2021 and March 2022 in VMKVMCH, Salem, Tamil Nadu. Medical case records of these patients were collected. The study includes the duration period of 18 months

#### **Inclusion criteria**

The patients of both sex, age more than 18 years old are included.

All patients who have consumed 3% yellow phosphorus in paste form.

Patients presented with unknown amount of Yellow phosphorus consumption with Hepatitis /Acute Liver Injury or Liver Cell Failure.

#### **Exclusion criteria**

Patients with other rodenticide consumption or mixed poison consumption were excluded from the study.

Patients having underlying liver disease and cardiac disease.

## Methodology

Patients who were admitted in VMKVMCH hospital with yellow phosphorus poisoning and who had willingly given informed consent.

 $\downarrow$ 

Patient's initial vital parameters and blood investigations at the time of admission were assessed and recorded.

 $\downarrow$ 

Treated accordingly with Hospital poisoning protocol.

١

Ongoing assessment and monitoring of the patient, blood

investigations & vital parameters and stability on every day.

 $\downarrow$ 

Drawing conclusions, with recorded details and inferencing the effectiveness with regards to the aoutcome of the patient.

# Lab investigations

The main prognostic factors include liver biomarkers (Bilirubin,SGOT,SGPT,PT/INR), Renal parameters (urea, creatinine), Serum electrolytes, Blood glucose, Blood grouping and RH typing, Arterial blood gas analysis, All routine blood investigation (complete blood count, lipid profile, special serology, covid-19 screening test,urine routine).

## **Imaging studies**

Ultrasound abdomen Sonographic imaging of the abdomen revealed fatty liver with GB wall edema with mild ascites.

CT scan and MRI scan abdomen CT revealed multiple parenchymal hemorrhages of the liver, breach in the continuity of wall of second part of the duodenum with associated pneumo peritoneum.

X-ray Chest.

CT Brain - ruled out encephalopathy.

## Statistical Analysis-

Numerical parameters, such as Age, are typically represented using statistical measures such as mean, standard deviation (SD), median, and mode. Categorical factors is depicted using frequencies and percentages. Pie charts and bar graphs are employed as suitable visual representations. The data was inputted into an MS Excel spreadsheet and then analyzed using SPSS software, namely version 23.

# 3. RESULTS

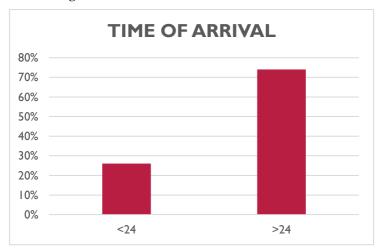


Figure 1- TIME OF ARRIVAL IN HOSPITAL

Plasmapheresis Found To Be More Useful And Effective When Taken Within 24 Hours Of Consumption Of Poison- With Above Analysed Reports.

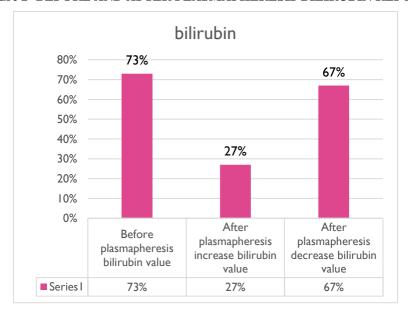


Figure 2- BEFORE AND AFTER PLASMAPHERESIS BILIRUBIN REPORT

Plasmapheresis Found To Be Ineffective With Above Analysed Reports – It Doesnot Cease The Elevation Of Lft Values Permanently.

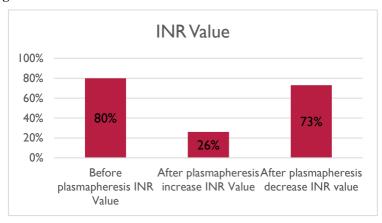


Figure 3- BEFORE AND AFTER PLASMAPHERESIS INR REPORT

Plasmapheresis is found to be effective in protecting the coagulation cascade during the initial stages of recovery but not permanently.

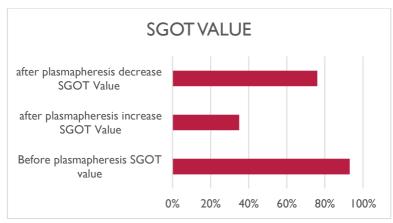


Figure 4- BEFORE AND AFTER PLASMAPHERESIS SGOT REPORT (A)

Plasmapheresis is found to be effective in preventing the more common liver cell injury during the initial stages of recovery but not permanently.

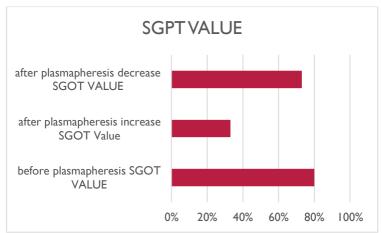


Figure 4 (B)- BEFORE AND AFTER PLASMAPHERESIS SGPT REPORT

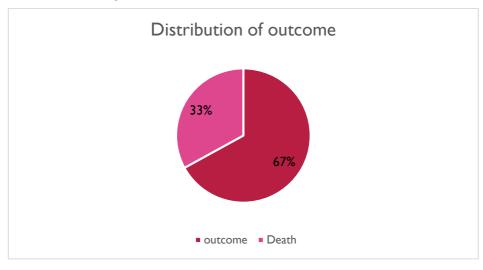


Figure 5- AFTER PLASMAPHERESIS REPORT

On Analysing The Proforma Reports Plasmaphersis Can Be Used As A Tool In Preventing The Patient From Rapid Worsening Of Signs And Symptoms Due To Ratol Paste Poison, Which Plays A Important Role In Initial Stages Of Recovery, Provide That Taken Within 24 Hours.

#### 4. DISCUSSION

In developed countries, spillage in industrial accidents is the most common form of yellow phosphorus poisoning, while in developing countries like India, this substance is often used in rodenticides and people often take it by suicidal intention and rarely by accidental ingestion. The 3% yellow phosphorus poisoning was commonly seen in farmers, field workers and housewives. In all the cases, the most common route of ingestion was oral. The majority of cases were in the age group of 21-40 years which can be explained by the fact that the persons of this age group are suffering from the stress of the modern lifestyles, family problems, nuclear family concept, etc.

The clinical effects of Yellow phosphorus poisoning are classically divided in three stages. The initial gastrointestinal stage (within the first 24 h after ingestion) is characterized by mild GI symptoms like vomiting, nausea, diarrhea, and abdominal pain. The laboratory tests at this stage are normal. Patients are usually asymptomatic in the second stage, which often lasts for 1 to 4 days. Liver biochemistry shows gross derangement during this stage. The third stage (4–7 days) manifests as Acute Liver Failure with multiple organ dysfunction syndrome that is characterized by acute renal failure with metabolic derangements, encephalopathy, coagulopathy, dysrhythmia, and cardiogenic shock. Liver Transplant is the only available option during this stage. The biological markers, AST and ALT showed a higher elevation in patients who ingested rat killer poison, which indicates that the first affected organ was liver followed by kidney and other organs.

Our study found that acute liver injury was the commonest complication. Other systems affected due to Yellow Phosphorous were gastrointestinal tract, Renal, Cardiovascular, Nervous systems along with associated metabolic abnormalities.

There is no specific antidote for Yellow Phosphorus Poisoning, the definitive treatment for acute liver failure is liver transplantation only. In our Hospital, we started therapeutic plasmapheresis as per the local experiences and observed that it performs especially well in toxin-mediated Acute liver failure such as Yellow phosphorus poison. [6,7]

This study was done with the intention to measure the safety and efficacy of Therapeutic plasmapheresis in terms of overall survival and transplant-free survival in yellow phosphorus poisoning. Our study was similar to other studies which reported that Yellow phosphorus poisoning with Acute Liver Injury presentation was seen in younger population (between 20 and 40 years) with a female gender predominance.

Our study reveals that the overall survival rate was 67% with early plasmapheresis therapy. We noticed that there was significant reduction (p<0.05) in the values of Prognostic factors includes Bilirubin, ALT, AST and INR following plasmapheresis. In contrast to a previous study on 334 Yellow phosphorus in a tertiary care center in the same geographical location (Thanjavur, Tamilnadu, India) showed very high mortality rate (76.2%) with standard medical care. [8,9,10] We found, out of 100% of patients received treatments like Plasmapheresis, N – acetyl cysteine and supportive measures, 67% of our patients survived and were discharged. Our study reveals that the best results were seen among patients in whom Plasmapheresis was started early in the course of illness.

From our observations, despite the small sample size, we state that patients who had biochemical improvement after plasmapheresis may not eventually require Liver Transplant. Early Plasmapheresis is an effective tool in management of

Acute Liver Injury and also Liver Failure following Ratol paste poison and may even serve as bridge to liver transplant in those listed for Liver Transplant. For definitive answers Multi-center, prospective study with a large sample size needed. We observed that, in Toxin mediated hepatitis, Acute Liver Injury or Acute Liver Failure due to Ratol paste poison, Early Plasmapheresis plays an important role and it is a safe, effective procedure.

# 5. CONCLUSION

Yellow phosphorus poison consumed with suicidal intention is commonly seen in the age group of 21-30 years with female preponderance. Most of them consumed around half of the tube (8gm) Ratol paste and presented to the hospital more than 24 hours after consumption. Acute liver injury sets in 3rd to 4th day of consumption. SGOT, SGPT, Serum Bilirubin, & PT/INR are the best prognostic factors to assess liver cell injury which significantly reduced after plasmapheresis. Patients who consumed less than a tube and those with early presentation (less than 24 hours) to the hospital responded well with our new protocol treatment. Treatment with Early Plasmapheresishas played an important role in yellow phosphorus toxic elimination and improving patient's prognosis as well as outcome.

### REFERENCES

- [1] Gargi J, Tejpal HR, Chanana A, Rai G, Chaudhary R. A retrospective autopsy study of poisoning in the northern region of Punjab. J Punjab Acad Forens Med Toxicol. 2008;8(2):17-9.
- [2] Ahuja H, Mathai AS, Pannu A, Arora R. Acute poisonings admitted to a tertiary level intensive care unit in northern India: patient profile and outcomes. J Clinic Diag Res. 2015;9(10):UC01.
- [3] Suneetha DK, Inbanathan J, Kannoth S, Reshma PK, Shashank MS. Profile of rat killer poisoning cases in a tertiary care hospital at Mysore. Int J Sci Study 2016;3(12):264–267.
- [4] Fernandez OU, Canizares LL. Acute hepatotoxicity from ingestion of yellow phosphoruscontaining fireworks. J Clin Gastroenterol 1995;21(2):139–142. DOI: 10.1097/00004836-199509000-00015
- [5] Alaga Venkatesan, et al. A study on the role of n-acetyl cysteine in yellow phosphorous poisoning (ratol) and postmortem toxicological findings. International Journal of Current Research Vol. 11, Issue, 01, pp.672-675, January, 2019 DOI: https://doi.org/10.24941/ijcr.33948.01.2019.
- [6] Varghese J, Joshi V, Bollipalli MK, Malleeswaran S, Patcha R, Nair H, Vij V, Sachan D, Subramanian P, Jain M, Venkataraman J. Role of therapeutic plasma exchange in acute liver failure due to yellow phosphorus poisoning. Indian J Gastroenterol. 2020 Dec;39(6):544-549. doi: 10.1007/s12664-020-01095-y. Epub 2021 Jan 6. PMID: 33409946; PMCID: PMC7787244.
- [7] Mathew J, Gnanaraj J, Basavarajegowda A, Venkateswaran R. Plasmapheresis in lethal yellow phosphorus poisoning: a scope for recovery. BMJ Case Rep. 2021 Apr 21;14(4):e239676. doi: 10.1136/bcr-2020-239676. PMID: 33883110; PMCID: PMC8061805
- [8] Appavu, Vinoth & Anthirias, Deborah & Dhanaraj, Haripriya & Lakshmanan, Mageshwaran & Ananthakumar, Sujitha & K, Swathi & Vinayagam, Swathy & Sekar, Chouthri. (2019). Yellow phosphorous poisoning: an observational study from a tertiary care hospital. Journal of Evidence Based Medicine and Healthcare. 6. 996-1002. 10.18410/jebmh/2019/208.
- [9] Ates M, Dirican A, Ozgor D, Aydin C, Isik B, Ara C, et al. Living donor liver transplantation for acute liver failure in pediatric patients caused by the ingestion of fireworks containing yellow phosphorus. Liver Transpl. 2011;17:1286–91. [PubMed] [Google Scholar]
- [10] Krishnakumar N, Krishnakumar S, Dalus MR, Balamugesh T. Yellow phosphorous poisoning--smoking stool syndrome. J Assoc Physicians India. 1997 Nov;45(11):885-6.PMID: 11229195