

# Efficacy of Early N-Acetylcysteine in Rat Killer Paste Poisoning

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## Abstract

**Introduction:** Rat killer paste (yellow phosphorous) is one of the most common forms of poisoning in South India. It causes hepatotoxicity. No specific antidote has been found. Recently, N-acetylcysteine is used as supportive therapy in many cases of acute liver failure.

**Aim:** This study aims to evaluate the effectiveness of early N-acetylcysteine in preventing the rat killer paste poisoning.

**Methods:** Patients who ingested rat killer paste poison and age >12 years were included in the study. Patients having jaundice, liver disease, and age <12 years were excluded from the study.

**Results:** Among 30 patients studied, five patients died, seven patients developed hepatitis, one patient developed acute kidney injury with hepatitis, and one patient developed hyponatremia.

**Conclusion:** Early initiation of N-acetylcysteine had a significant impact in reducing mortality.

**Key words:** Acute hepatic failure, N-acetylcysteine, Rat killer paste

## INTRODUCTION

Rat killer paste poisoning is one of the causes of acute liver failure.<sup>[1]</sup> Rat killer (Ratol) paste contains yellow phosphorus. White phosphorous with its impurities is called yellow phosphorus. It is used in firework industry, in bombs, as Rodenticide. In South India, it is commonly available as rat killer paste (3%). The toxic dose of yellow phosphorous is 100 mg/kg body weight and toxicity increases when taken with a fatty meal. Yellow phosphorus causes hepatotoxicity by the production of phosphoric acid, which causes free radical damage.<sup>[2]</sup> This poisoning is associated with high mortality. The good prognostic factors are survival after 3 days and minimal elevation of LFT. Bad prognostic signs are altered sensorium, cyanosis, hypotension, metabolic acidosis, elevated prothrombin time, and hypoglycemia.

The first phase consists of nausea, vomiting, abdominal pain, and smoking stools. Then, in the second phase, the patient may feel symptomatically better and the third stage consists of systemic organ damage due to absorbed phosphorous. Hepatotoxicity usually is recognized on the 3<sup>rd</sup> day by liver function test (LFT).<sup>[3]</sup> A large number of early deaths (<24 h) are due to cardiotoxicity. Renal toxicity is due to acute tubular necrosis which may be due to hypotension. Hyponatremia, hyperkalemia, and hyperphosphatemia are observed. Furthermore, there is no antidote for this poisoning. N-acetylcysteine is used as an antidote in paracetamol poisoning. Since its mechanism of hepatoprotective is similar, it can also be used in yellow phosphorous poisoning. N-acetylcysteine has been used in many of non-paracetamol-induced acute liver failure. N-acetylcysteine through replenishment of glutathione stores of superoxide dismutase (SOD) is proposed to have a beneficial effect. It replenishes the free radical scavenging system of hepatocytes and also directly neutralizes the free radicals. It is also said to improve cerebral perfusion in fulminant hepatic failure in rats. Many randomized control studies were not available for this poisoning.<sup>[4,5]</sup> Hence, this study was conducted to evaluate the efficacy of early N-acetyl cysteine from preventing the mortality.

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## Aim

This study aims to evaluate the effectiveness of early N-acetylcysteine in reducing the mortality in rat killer paste poisoning.

## METHODS

This study was conducted among 30 patients admitted with ingestion of rat killer paste poison in the Department of Medicine, Pudukkottai Government Medical College Hospital, from July 2018 to December 2018. This study is a prospective analytical study. Patients who ingested rat killer paste poison and age >12 years were included in the study. Patients are having jaundice, liver disease, congestive cardiac failure, patients on hepatotoxic drugs, and age <12 years were excluded from the study. Demographic details, medical history, and clinical examination were recorded. Complete blood count, blood sugar, renal function test, LFT, prothrombin time/international normalized ratio, and electrocardiography (ECG), and ultrasound abdomen were done. Patients were treated with N-acetylcysteine at the dosage of 150 mg/kg in 200 ml 5% D over 15 min and 50 mg/kg in 500 ml 5% D over 4 h and 100 mg/kg in 2000 ml 5% D over 16 h. There is no specific dosage of N-acetylcysteine for rat killer paste poisoning patients. Since N-acetylcysteine is used for many non-paracetamol causes of liver failure, we used N-acetylcysteine and there is no harm to patients. LFT was done daily until discharge to monitor the development of liver failure. Patients were categorized into three groups based on the time interval between consumption of poison and starting of N-acetylcysteine. There was no delay in starting of the N-acetylcysteine after admission.

## RESULTS

In our study, among 30 patients studied, 17 were male and 13 were female. In that 19 patients were <30 years of age and nine patients were >30 years but <60 years. Two patients were >60 years [Figure 1].

Among 30 patients studied, five patients died, seven patients developed hepatitis, one patient developed acute kidney injury with hepatitis, and one patient developed hyponatremia. Of the patients developed complication, NAC was started within 6 h for 23 patients, 9 patients had complications, of which two died and NAC was started in >6–10 h for four patients, three had complications, of which two died and NAC was started in >10 h for three patients, one had complications and died.

In our study, of the 30 patients studied, 13 patients (43.34%) developed complication and in that 5 patients

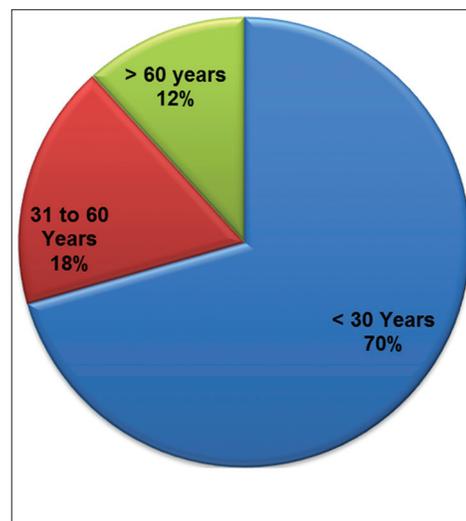


Figure 1: Age distribution

(16.67%) died. When comparing the time interval between starting of antidote and consumption of poison, 39.13% of patients developed complications and 22.23% of that patients died in <6 h group, but in 6–10 h group, 75% developed complications and mortality was 66.67%, whereas in >10 h group, 1 patient (33.33%) developed complication and died (100%) [Tables 1 and 2]

## DISCUSSION

Various studies on poisoning did in India Banerjee *et al.* in West Bengal also noticed most commonly affected age group was 20–40 years.<sup>[6]</sup> Saoji *et al.* found in her study that the patients were usually asymptomatic during the initial 72 h of ingestion, or they may have signs and symptoms of gastrointestinal irritation.<sup>[7]</sup> Nalabothu *et al.* showed 35.7% mortality rate, whereas our study showed 17% mortality rate.<sup>[8]</sup>

Yellow phosphorus is a protoplasmic poison and is both hepatotoxic and cardiotoxic. The first phase consists of nausea, vomiting, abdominal pain, and smoky stools. Then, in the second phase, the patient may feel symptomatically better and the third stage consists of systemic organ damage due to absorbed phosphorous. Hepatotoxicity usually is recognized on the 3<sup>rd</sup> day by LFT. Ingestion of the large doses of yellow phosphorous can cause cardiotoxicity. A large number of early deaths (<24 h) are due to cardiotoxicity. ECG changes include corrected QT INTERVAL prolongation and ST segment changes which are associated with worse prognosis. Renal toxicity is due to acute tubular necrosis which may be due to hypotension.<sup>[8,9]</sup> Hyponatremia, hyperkalemia, and hyperphosphatemia are observed. The good prognostic factors are survival after 3 days and minimal elevation

**Table 1: Correlation of time interval between the consumption of poison and starting of N-acetylcysteine with the development of complications**

Time of starting N-acetylcysteine	Number of patients	Number of patients developed complications	Number of patients died after complications
<1 h	2	0	0
1–2 h	4	2	0
2–3 h	3	1	0
3–4 h	8	4	1
4–5 h	2	0	0
5–6 h	4	2	1
6–10 h	4	3	2
<10 h	3	1	1

**Table 2: Comparison of three time interval groups and the development of complications and mortality**

Time of starting N-acetylcysteine	Number of patients	% of patients developed complications	Number of patients died after complications (%)
<6 h	23	39.13%	22.23
>6–10 h	4	75	66.67
>10 h	3	33.33	100

of LFT.<sup>[10]</sup> Bad prognostic signs are altered sensorium, cyanosis, hypotension, metabolic acidosis, elevated prothrombin time, and hypoglycemia. N-acetylcysteine is used as a mucolytic, nephroprotective agent to prevent contrast-induced nephropathy, chronic obstructive pulmonary disease, and as an antidote in paracetamol poisoning. Since its mechanism of hepatoprotective is similar, it can also be used in yellow phosphorous poisoning. It acts as a glutathione substitute and replenishes the free radical scavenging system of hepatocytes and also directly neutralizes the free radicals.

## CONCLUSION

As per our study after early N-acetylcysteine therapy, overall mortality has been reduced. However, there were no significant results in preventing the development of complications in patients started NAC within 6 h. To conclude from our study that there is strong evidence that early NAC therapy in rat killer paste poisoning has a significant impact in reducing the mortality.

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