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General Medicine

# ROLE OF N ACETYL CYSTEINE IN YELLOW PHOSPHOROUS POISONING IN A TERTIARY CARE CENTER

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| **Dr.Sundaram Rajan*** | Assistant Professor, Department Of Medicine, Madurai Medical College<br>*Corresponding Author |
| **Dr.Sangumani Jayaram** | Professor, Department Of Medicine, Madurai Medical College |
| **Dr.Dinesh Ravichandran** | Post Graduate, Department Of Medicine, Madurai Medical College. |

**ABSTRACT** Rat killer paste(Yellow phosphorous) is one of the most common from of poisoning in south india. It causes hepatotoxicity by free radical production. No specific antidote has been found yet. N acetyl cysteine is used as a supportive therapy in many cases of acute liver failure due to yellow phosphorus.our study was prospective analytical study in patients of yellow phosphorous poisoning receiving N acetyl cyteine(NAC).

**AIM OF THE STUDY:** To analyse the treatment success and mortality rates in patients of yellow phosphorous poisoning treated with N acetyl cysteine.

**MATERIALS AND METHODS:** All patients admitted with ingestion of rat killer paste(yellow phosphorous) in our tertiary medical college hospital from January 2017 to june 2017 and treated with N acetyl cysteine. Patients were analysed with respect to age, sex, time to initiaiton of NAC.

**RESULTS:** Of the total 62 patients 12 patients died. 10 of these deaths were in patients who had presented more than 3 days after ingestion. Patients treated with NAC had a mortality rate 19.3% and there was no age,sex based variation in mortality rates. Delayed presentation (>72hrs) after ingestion had a significant mortality rate of 91%.

**CONCLUSION:** N acetyl cysteine reduces mortality when given earlier in patients with rat killer paste poisoning.The time to initiate therapy plays a major role in the prognosis of the patients. Since most of the primary care providers are unaware of this therapy, educating them will improve the survival rate among rat killer paste poisoning.

**KEYWORDS :** Rat killer paste, ratol, yellow phosphorous poisoning, N acetyl cysteine, acute liver failure

## INTRODUCTION:

Yellow phosphorous causes hepatotoxicity by production of phosphoric acid, which causes free radical damage and is a protoplasmic poison. No specific antidote is available for yellow phosphorous poisoning although stomach wash with various compounds, and N acetyl cysteine() and methionine(1) have been tried. N Acetyl cysteine through replenishment of glutathione stores of SOD (superoxide dismutase) is proposed to have a beneficial effect. It is also said to improve cerebral perfusion in fulminant hepatic failure in rats (2). No specific dosages have been formulated for use in non-paracetomol induced hepatic failure. The more commonly used dosage for oral tablets are 140mg/kg stat then 70mg/kg every 4th hourly for 72hrs.intravenous dosage are 150mg.kg stat and 50mg/kg for the next 4 hrs and 100mg/kg over the next 16hrs.

As there is no harm in using N acetyl cysteine for rat killer paste poisoning RCT are not available. Majority of trials have used retrospective cases as controls and have yielded positive result (3, 4, and 5).

## MATERIALS AND METHODS:

The study was conducted at government Rajaji hospital during January 2017 to June 2017 among the 62 patients admitted with alleged history of consumption of rat killer paste (yellow phosphorous 3%) and treated with NAC.Daily serial monitoring of LFT and PT/INR was done and charted.

## INCLUSION CRITERIA:

Consumption of toxic doses of rat killer paste (>0.5g)
Normal baseline LFT

## EXCLUSION CRITERIA:

Known chronic liver disease patients
Hepatitis B, C positive patients
Congestive cardiac failure
No biliary obstruction on USG abdomen
H/o hepatotoxic drug intake

## ETHICAL COMMITTEE:

## TREATMENT PROTOCOL:

Patients were treated with stomach wash and started in Inj.N Acetyl Cysteine IV 150mg/kg in 200ml 5%dextrose over 15mins then 50mg/kg IV in 5%dextrose over next 4 hrs and 100mg/kg IV in 500ml 5%dextrose over next 16 hrs on the day of admission irrespective of the LFT. Patients were kept on nil per oral for first three days and started on low fat diet over the next days. Patients with bleeding manifestations were given Fresh frozen plasma transfusions.

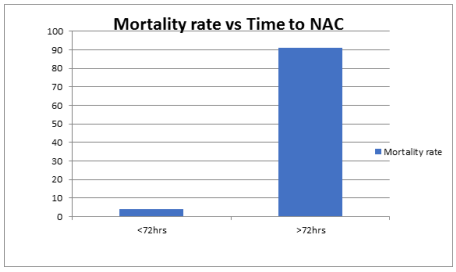
## STATISTICALANALYSIS:

Daily LFT was entered into master chart and analysed

## RESULTS:

The overall mortality rate in our hospital was 19.3%. Mortality rate among those who presented late (>72hrs) was 91%. The mortality rate among those who presented early (<72hrs) was 3.8%.

the mean AST/ALT was 328/180 on the 3rd day among those who survived. The mean AST/ALT among those who died was 450/190. Individuals with earlier rise in LFT had with higher mortality rate. Highest level of bilirubin recorded among patients who survived was 16.2mg/dl among patients who died the highest was 25.9mg/dl. The mean bilirubin levels on 5 th day in patients started on NAC was 6.2mg/dl and in those with late initiation was 14.6mg/dl.




## DISCUSSION:

Phosphorous is present in two forms white and red. White phosphorous

in its pure form is combustible when exposed to air. White phosphorous with its impurities is called yellow phosphrous. sources are that in firework industry,in bombs in military use and as rodenticide. In south india it is commonly available as rat killer paste(3%).it has a garlic odour. Among the rat killer chemical the most common form to be used as a suicidal poison is rat killer paste().it is also common to have accidental poisoning in children as it resembles toothpaste and toxic dose in children being very much less.the toxic dose of yellow phosphorous is 100mg/kg body weight and toxicity increases when taken with a fatty meal.

Yellow phosphorous is a protoplasmic poison and is both hepatotoxic and cardiotoxic. The effects of yellow phosphorous are described in three phases initially consisting of nausea,vomiting, abdominal pain and smoking stools. Then second phase the patient may feel symptomatically better and the third stage consists of systemic organ damage due to absorbed phosphorous.although distinction of these stages is usually not possible.hepatoxicity usually is recognized on 3rd day by LFT. enzymes may be elevated upto 1000u/ml. it causes necrosis of zone 1 unlike acetaminophen and carbon tetrachloride which cause zone 3 necrosis.the changes of fatty liver can be seen in liver after 6 hrs of ingestion.

Ingestion of large doses of yellow phosphorous can cause cardiotoxicity, apart from electrolyte disturbance, in form of fatty degeneration of myocardial cells. a large number of early deaths(<24hrs) are due to cardiotoxicity.ECG changes include QTc 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 and direct toxicity.hyponatremia, hyperkalemia and hyperphosphatemia are observed.metabolic parameters that need to be montored apart from serum electrolytes is blood glucose level.adequate blood glucose is essential for production of reducing equivalents(NADH and NADPH),which are overutilzed in the poisoning.so a hyperglycaemic state may be beneficial to the patient.

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 hypoglycaemia.

n acetyl cysteine is used as a mucolytic,nephroprotective agent to prevent contrast induced nephropathy,COPD and as antidote in paracetomol poisoning. since its mechanism of hepatoprotectivity 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.

All these pertain to acute poisoning with yellow phosphorous chronic poisonng due to occupations may cause phossy jaw or osteomyelitis of jaw.

## REFERENCES


1. Diaz-Rivera RS, Collazo PJ, Pons ER, Torregrosa MV. Acute phosphorus poisoning in man: a study of 56 cases. Medicine. 1950;29:269-298.
2. Ytrebo LM, Korvald C, Nedredal GI, Elvenes OP, Nielsen Grymyr OJ, Revhaug A. N-Acetylcysteine increases cerebral perfusion pressure in pigs with fulminant hepatic failure. Crit Care Med. 2001;29(10):1989–1995. doi: 10.1097/00003246-200110000-000232009;137(3):856-864.e1. doi:10.1053/j.gastro.2009.06.006.
3. Lee W, Hynan L, Rossaro L, et al. INTRAVENOUS N-ACETYLCYSTEINE IMPROVES TRANSPLANT-FREE SURVIVAL IN EARLY STAGE NON-ACETAMINOPHEN ACUTE LIVER FAILURE. Gastroenterology.
4. Safety and efficacy of N-acetylcysteine in children with non-acetaminophen-induced acute liver failure.Kortsalioudaki C, Taylor RM, Cheeseman P, Bansal S, Mieli-Vergani G, Dhawan A. Liver Transpl. 2008 Jan;14(1):25-30.
5. N-Acetyl Cysteine in the Management of Rodenticide Consumption — Life Saving?Smitha Bhat and Kumar P. Kenchetty. J Clin Diagn Res. 2015 Jan; 9(1): OC10–OC13. doi: 10.7860/JCDR/2015/ 11484.5455