Original Research Article

Study of clinical profile and outcome of Ratol poisoning in tertiary care hospital

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Abstract

Background: Ratol paste or powder which is easily available rodenticide in the open market in India. Due to easy availability it has become a compound used in poisoning. Poisoning with yellow phosphorus classically manifest with acute hepatitis leading to acute liver failure. First stage: Gastrointestinal stage-occurs during first 24 hours. Symptoms like vomiting, diarrhoea, abdominal pain are present. Laboratory investigations will be normal. Sudden deaths may occur due to cardiovascular arrythmias and cardiovascular collapse, usually due to consumption of large quantities of yellow phosphorous. Second stage: Occurs between 24-72 hours after ingestion. It is essentially a symptom free period but liver enzymes become elevated and toxic hepatitis begins to spread. Third stage: Multiorgan failure stage occurs after 72 hours until the resolution of symptoms or death. Acute liver failure, acute renal failure and metabolic derangements. Hepatomegaly and jaundice appear acute fulminant hepatic failure mandating liver transplantation. Bleeding can occur due to coagulopathy and thrombocytopenia. Encephalopathy, arrythmias and cardiogenic shock occurs **Method:** Study Design: Analysis of data of ratol poisoning patient from April 2017 to March 2018.Study setting: KIMS Hospital Medicine wards & ICU. Period of study:1 Year. Sample size: 30 patients. **Results:** A total of 30 admitted patients were analysed and evaluated. Out of 30 patients, 23(76.7%) were males and 07(23.3%) were females. Out of 30 patients, vomiting was present in 27(90%) patients which was followed by abdominal pain which was present in 14(46.66%) patients, followed by lethargy in 4(13.33%) patients. No patient was having symptoms of altered sensorium, convulsion on the day of admission. Out of 30 patients, 3(10%) patients died because of fulminant hepatic failure and 27(90%) patients recovered and discharged on day 14. All patients were treated with N acetylcysteine. **Conclusion:** N Acetylcysteine has significant role in Acute Liver injury if given early. This is probably attributed to the Anti-oxidant property and Hepato- protective nature of N Acetylcysteine.
Key Word: Ratol poisoning.

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Received Date: 11/01/2019 Revised Date: 08/02/2019 Accepted Date: 17/03/2019
DOI: https://doi.org/10.26611/10219312

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 21 March 2019

INTRODUCTION

Rodent control is important as rodents cause diseases and disrupt the food supply. They multiply fast if left unchecked. One-dose rodenticides are often used because using a toxin that must accumulate after multiple feedings will kill only a fraction of target animals. For this purpose numerous rodenticides are available. Ratol paste and powder is easily available rodenticide in the open market in India. Yellow Phosphorous is a commonly used rodenticide containing 3-5% Yellow Phosphorous.[1] It is highly toxic and is responsible for many deaths after oral ingestion. Suicidal Ratol tablet and paste overdose is the most common cause of poisoning in India.[2] Most cases of fatal or severe exposure to Yellow Phosphorus resulted in adults or children accidentally or deliberately swallowing the rat poisons.[3,4] Yellow Phosphorous is a protoplasmic poison, spontaneously combusts in air and causes cardiac, hepatic, renal, and multi organ failure when ingested.[5] It

How to cite this article: Shilpa Patil, Mohit Khatri, Aniket Avhad, Gaurav Jaju. Study of clinical profile and outcome of Ratol poisoning in tertiary care hospital. *MedPulse International Journal of Medicine.* March 2019; 9(3): 200-204. https://www.medpulse.in/Medicine/

is rapidly absorbed from the GI tract[6] and is primarily metabolized by the liver. A dose of 60mg of phosphorus can cause severe poisonings in humans and as little as 15 mg may be fatal.[7] The vomitus after phosphorus ingestion is luminescent and has a characteristic garlic odour. If the patient survives the initial gastrointestinal irritation phase, secondary systemic poisoning usually in form of hepatic toxicity may ensue.

Three stages are known:

First stage: Gastrointestinal stage-occurs during first 24 hours. Symptoms like vomiting, diarrhoea, abdominal pain are present. Laboratory investigations will be normal. Sudden deaths may occur due to cardiovascular arrythmias and cardiovascular collapse, usually due to consumption of large quantities of yellow phosphorous.

Cardiac failure may occur because of fluid and electrolyte loss due to vomiting and diarrhoea in addition to cardiac toxicity. Electrolytes abnormalities like hypocalcaemia and hyperkalaemia causes cardiac dysaryhtmias.

Investigations which are done are LFT, RFT, serum Na, K, Ca and ECG.

Second stage: Occurs between 24-72 hours after ingestion. It is essentially a symptom free period but liver enzymes become elevated and toxic hepatitis begins to spread. Investigation which are done are LFT, ECG, INR.

Third stage: Multiorgan failure stage occurs after 72 hours until the resolution of symptoms or death. Acute liver failure acute renal failure and metabolic derangements. Hepatomegaly and jaundice appear acute fulminant hepatic failure mandating liver transplantation. Bleeding can occur due to coagulopathy and thrombocytopenia. Encephalopathy, arrythmias and cardiogenic shock occurs. Central nervous system effects include changes in mental status like confusion, psychosis, hallucinations and coma. Investigations which are done are LFT, serum Na, K, Ca, Mg, INR.

AIM

Study of clinical profile and outcome of ratol poisoning in tertiary care hospital.

OBJECTIVES

- Study of clinical profile of patients consuming ratol.
- Assessment of liver dysfunction, cardiac arrhythmias.
- Study of clinical outcome of the patients consuming ratol and treated with N acetylcysteine.

Detail Research Plan

Study Design: Analysis of data of ratol poisoning patient from April 2017 to March 2018.

Study setting: KIMS Hospital Medicine wards & ICU.

Period of study: 1 Year.

Sample size: 30 patients.

MATERIALS AND METHODS

This will be a retrospective study carried out in Krishna Hospital and Medical Research centre, Karad. A detailed history and physical examination, findings will be collected from patients record papers. Laboratory investigations will also be obtained from the records. Patients clinical progress on day to day basis will be collected and analysed.

Inclusion Criteria

1. All the patients above age of 18 years of either sex will be included.
2. All patients who have consumed yellow phosphorus either in solid or paste form will be included.

Exclusion Criteria

1. Patients with other rodenticide. consumption will be excluded from the study.
2. Patients having underlying liver disease and cardiac disease will be excluded from the study.

METHODOLOGY

Cases will be patients who have consumed ratol either in paste or powder form from medical wards and ICU in the hospital. The obtained data will be analyzed in terms of age, sex, duration of hospitalization from compound consumption, quantity of compound consumption, symptoms, signs, laboratory investigations, treatment given and clinical outcome.

Investigations

1. Complete blood count.
2. Prothrombin time with INR
3. Liver Function Test.
4. Ultrasonography
5. ECG

OBSERVATION AND RESULTS

1. Age group and gender wise distribution

A total of 30 admitted patients were analysed and evaluated. Out of 30patients, 23(76.7%)were males and 07(23.3%)were females. Out of 30 patients, 15(65.2%) males and 6(85.71%) females were in the age group of 18-40 years. 6(26.08%) males and 2(28.57%) females were in the age group of 40-60 years. 2(8.69%) males were in the age group of more than 60 years.

Table 1: Age group and gender wise distribution

Age group/ gender	Males	Females	Total
18-40	15	5	20
40-60	6	2	8
More than 60	2	0	2
Total	23	7	30

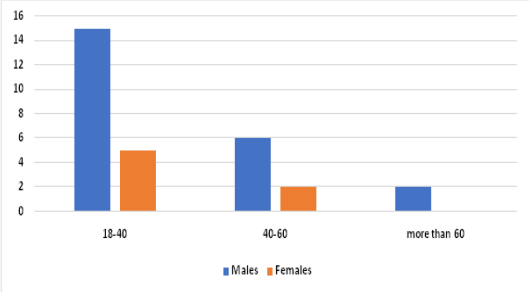

Figure1: Age group and gender wise distribution

2. Duration of compound consumption and hospital admission of patients

The patients are analysed for duration of hospitalisation from the compound consumption. Out of 23 males, 15(65.21%) were admitted within 6 hours and 8(34.78) were admitted within 24 hours of compound consumption and out of 7 females 6(85.71%) admitted within 6 hours and 1(14.28%) admitted within 24 hours of compound consumption. The duration of hospital admission and compound consumption are very important. The lesser the duration the prognosis was good.

Table 2: Duration of compound consumption and hospital admission of patients

Duration of compound consumption and hospital admission	Males	Females	Total
Within 6 hours	15	6	21
Within 24 hours	8	1	9
Total	23	7	30

3. Symptoms according to the three Stages

Symptoms evaluated are vomiting, lethargy, altered sensorium, convulsion, coma, abdominal pain. Out of 30 patients, vomiting was present in 20(66.66%) patients followed by abdominal pain in 10(71.42%), lethargy in 3(75%) in the first stage. In second stage vomiting was present in 7(25.92%), abdominal pain in 4(28.57%), lethargy in 1(25%) patients.In third stage 3(100%) patients landed into coma. No patient was having symptoms of altered sensorium, convulsion on the day of admission and also during the course of illness.

Table 3: Symptoms according to Stages

Stages	Vomiting	Abdominal pain	Lethargy	Altered sensorium	Convulsion	Coma
First stage (within 24 hrs)	20	10	3	0	0	0
Second stage(24-72 hrs)	7	4	1	0	0	0
Third stage(more than 72 hrs)	0	0	0	0	0	3
Total	27	14	4	0	0	3

4. Laboratory parameters and hospital course of patients

First 48 hrs the patients had no signs of hepatic encephalopathy and deranged INR, ECG abnormalities, Liver enzymes noted. Out of 30 patients on DAY 3, 13(43.33%) patients developed signs of hepatic encephalopathy, 20(66.66%) patients were having deranged PT and INR, 21(70%) patients were having deranged liver enzymes(total bilirubin, SGOT, SGPT), 2(6.66%) were having cardiac arrythmias. On day7, 3(10%) patients still have signs of hepatic encephalopathy, 10(33.33%) patients have deranged PT and INR ,15(50%) patients have deranged liver enzymes (total bilirubin, SGOT, SGPT), no patients were having cardiac arrythmias. On day 10, 3(10%) patients still have deranged PT INR and 6(20%) patients have deranged liver enzymes. All patients were admitted and supportive treatement was started immediately. Gastric lavage was given thoroughly to all patients.

Table 4: Laboratory parameters and hospital course of patients

Variables	Day 3	Day 07	Day 10
Signs of hepatic encephalopathy	13	3	3
Deranged PT, INR	27	10	3
Deranged liver enzymes	28	15	06
Cardiac arrythmias	2	0	0

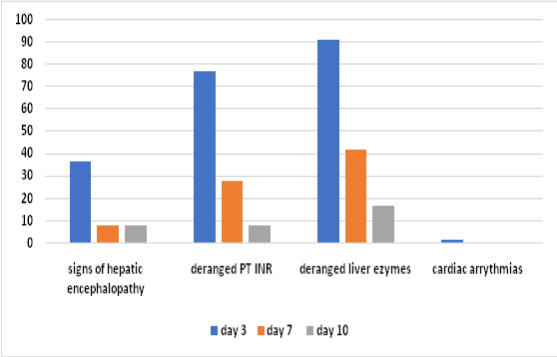

Figure 2: Laboratory parameters and hospital course of patients

5. Treatment protocol

All patients received N acetylcystine. It was given in a dose of 150mg/kg as loading dose then 50mg/kg in 500ml D5W over 4 hours and 100mg/kg in 1000ml D5W over 16 hours. Patient who reports early were having prognosis good while patient who reported late, some of them had complications. N acetylcystine was started on the day of admission to all patients.

6. Outcome of all patients

Out of 30patients, 3(10%) patients died because of fulminant hepatic failure which and 27(90%) patients recovered and discharged on day 14.

Table 5: Outcome of all patients

Outcome	No of patients (n=30)
Recovered	27
Death	03

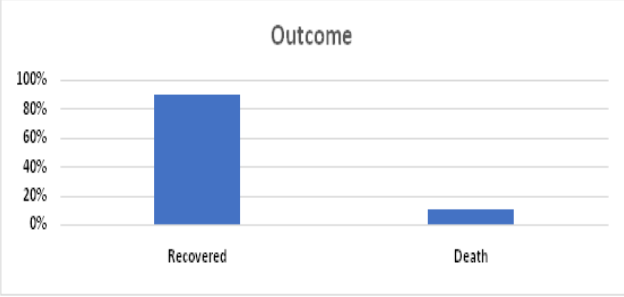

Figure 3: Outcome of all patients

DISCUSSION

Rodenticide compounds are commonly used to kill rats, mice and rodents. They are heterogeneous group of compounds that exhibit markedly different toxicities to Humans and Rodents. In our study all the patients consumed yellow phosphorous which is highly toxic in nature. In a Retrospective study conducted in South India in the year 2002, Organic Phosphorus compounds were reported as the most common cause of poisoning (36.0%).[2] In our study, the incidence is slightly higher in Males than Females. The patients who recovered sometimes suffered from psychiatric disorder like depression and adjustment disorders. Chikkaveeraiah KS et al.[4] (noted that the systems affected in their study due

to Yellow Phosphorous were gastrointestinal tract (100%), Liver (66.70%), Cardiovascular, Nervous and respiratory systems along with associated metabolic abnormalities (66.7%). In our study 93% patient had derangement in liver enzymes,10% patient had respiratory depression. No patient had cardiac or metabolic abnormalities. Fernandez and Canizares stated that in a series of 15 patients observed a mortality of 27% is recorded, confirming that Yellow Phosphorus is extremely lethal when ingested.[12] In our study the mortality rate was 10%. The mortality rate in Yellow Phosphorous poisoning from previous studies been reported between 10 and 50%. We have noticed good prognosis in patients who have been started early on N acetylcysteine therapy after consumption of poisoning. The duration of hospital admission and compound consumption are very important. The lesser the duration the prognosis is good. This is mainly because of gastric lavage given to those patients presenting early, which decreases the amount of yellow phosphorous entering circulation and early treatment with N acetylcyteine.

CONCLUSION

Among the patients presented, yellow phosphorus compound is common because of easy availability and cheaper. From the solid or paste form solid one is easily available in the form of chalks. Patients who presented early and started with N Acetylcysteine had good prognosis. Patients who presented late and started with N Acetylcysteine had bad prognosis. N Acetylcysteine has significant role in Acute Liver injury if given early. This is probably attributed to the Anti-oxidant property and Hepato- protective nature of N Acetylcysteine.

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Source of Support: None Declared
Conflict of Interest: None Declared