Original article:

Acute yellow phosphorus poisoning – retrospective analysis in a tertiary care centre

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Abstract

Introduction: Rodenticides exposure accounts for 0.5% cases presenting with poisoning to health care facilities. Despite the limited use of yellow phosphorus these days, rodenticides provide the common available source of yellow phosphorus. After consumption the manifestations can be both local and systemic. The management is usually conservative with monitoring of renal and liver parameters.

Material and methods: Retrospective analysis of cases presenting with acute yellow phosphorus poisoning to our hospital between the study period of January 2013 to December 2017. All patients aged above 18 years were included in the study. The demographic data, clinical manifestations, laboratory parameters and complications were recorded.

Results: A total of 35 cases presenting with acute consumption of yellow phosphorus aged above 18 years were included in the study. The majority were within the age group of 18-40 year age group and of male gender. Patients presented with gastrointestinal manifestations most commonly followed by neurological and bleeding diasthesis. The outcome included death seen in about 25.7% of the cases and 54.3% of cases recovered

Conclusion: Yellow Phosphorous is very lethal on consumption with high mortality rates. Since there is no specific antidote available for treatment, rigorous first aid and supportive care should be given to have a good outcome. Measures should be taken towards the prevention of the suicidal attempts.

INTRODUCTION

Elemental phosphorus is a non-metallic substance that exists in two forms mainly red and yellow phosphorus. "Yellow phosphorus" is formed by a small amount of red phosphorus resulting discoloration of white phosphorus.[1] YP is a general protoplasmic toxin[2] and is used in the manufacture of fireworks, rodenticide, and fertilizers. Although poisoning may result from industrial accidents in developed countries, it also occurs in adults who are attempting suicide and accidentally by oral intake in children due to easy accessibility to rodenticides, containing 2 to 5 percent yellow phosphorus. .

On ingestion it has both local and systemic toxicity. LD50 of the compound after acute exposure is approx. 1mg/kg body weight.[3] The clinical manifestations are classically described in three phases, phase 1 occurs immediately after exposure. Initial symptoms include nausea, vomiting, diarrhoea with perioral and mucosal burns. The feces and vomiting may exhibit phosphorescence termed as smoking stool syndrome. In the second phase, a latent period of up to 2–4 weeks occurs, during which symptoms seem to resolve, as seen in our study. Finally, in a third phase, multi-organ involvement is seen.[4] Here we report a case series of patients presenting with yellow phosphorous consumption.

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MATERIALS AND METHODS

This is a Retrospective study conducted in KS Hegde Medical college and included all patients above the age of 18 years admitted with history of consumption of yellow phosphorus from January 2013 to December 2017. The patient demographics, amount of consumption along with time of presentation to the hospital were recorded. Initial assessment included symptoms, signs of poisoning along with blood investigations done to assess systemic toxicity. The laboratory parameters mainly assessing liver function/ kidney function and its severity were assessed. The need for ventilatory and vasopressor support was also included in the study. The total duration of hospital stay and outcome was also assessed.

STATISTICAL ANALYSIS

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Continuous data was represented as mean and standard deviation.

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, Pie diagram.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

RESULTS

A total of 35 patients were included in the study of which 60% were males and 40% were females. Majority of subjects were in the age group of 21 to 30 years (22.9%), 20% were in the age group <20 years and 31 to 40 years, 17.1% were in the age group 41 to 50 years, 14.3% were in the age group 51 to 60 years and 5.7% were in the age group >60 years.

Table 1: Age distribution

		Count	%
Age	<20 Years	7	20.0%
	21 to 30 Years	8	22.9%
	31 to 40 Years	7	20.0%
	41 to 50 Years	6	17.1%
	51 to 60 Years	5	14.3%
	>60 years	2	5.7%

Table 2: Gender distribution

		Count	%
Gender	Female	14	40.0%
	Male	21	60.0%

In the study 22.9% consumed 5ml, 57.1% consumed 10ml, 8.6% consumed 15 ml and 11.4% consumed 20 ml & all of them were suicidal. Of all the patients, 20% presented with in 3hrs, 22.9% presented with in 4 to 6 hrs, 37.1% presented between 7 to 24 hrs and 20% presented after 24hrs. There was no association between the time of presentation and complications like renal & liver failure. And also there was no correlation between quantity consumed and complications.

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Table 3: Quantity Consumed

		Count	%
Quantity Consumed (ML)	5	8	22.9%
	10	20	57.1%
	15	3	8.6%
	20	4	11.4%

Table 4: Time of Presentation

		Count	%
Time Of Presentation (Hours)	<3 hrs	7	20.0%
	4 to 6 yrs	8	22.9%
	7 to 24 hrs	13	37.1%
	>24 hrs	7	20.0%

In the study, 91.4% presented with vomiting, 85.7% presented with abdominal pain, 57.1% with jaundice, 28.6% with altered sensorium, 14.3% Bleeding Diasthesis, 5.7% with seizures, 2.8% with oliguria, fever & loose stools each. 14.3% with tachypnea & 5.7 % presented with tachycardia.

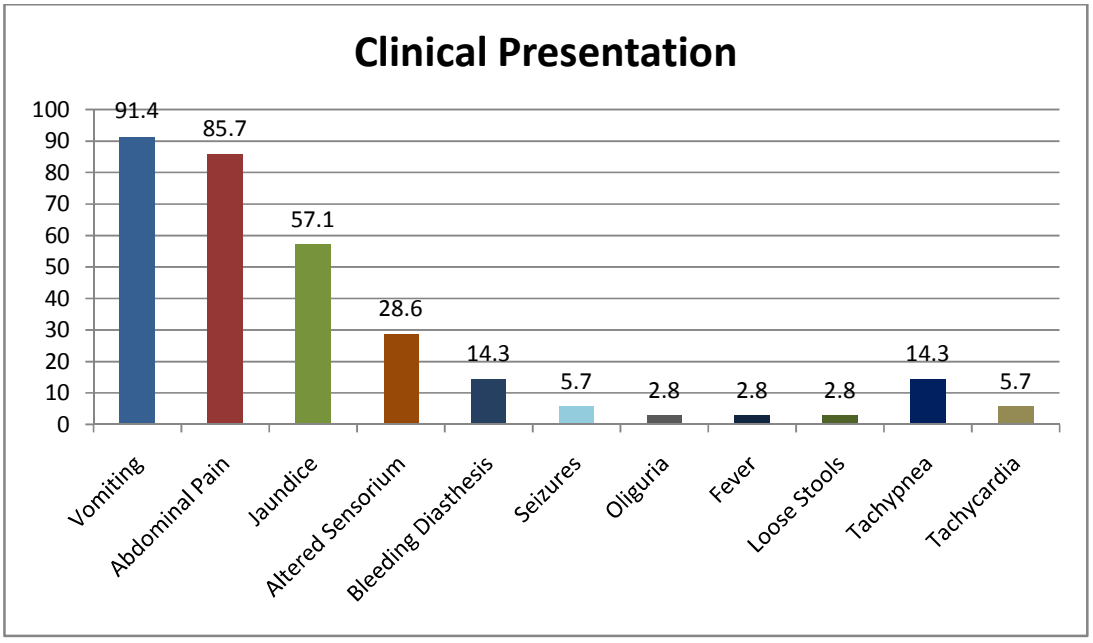

Chart 1: Clinical Presentation

Of the 35 patients, 68.6% had elevated Serum Creatinine, 60% had elevated serum bilirubin, 71.4% had elevated liver enzymes and 60% had elevated INR & 51.4% had reduced PAO2/FIO2.

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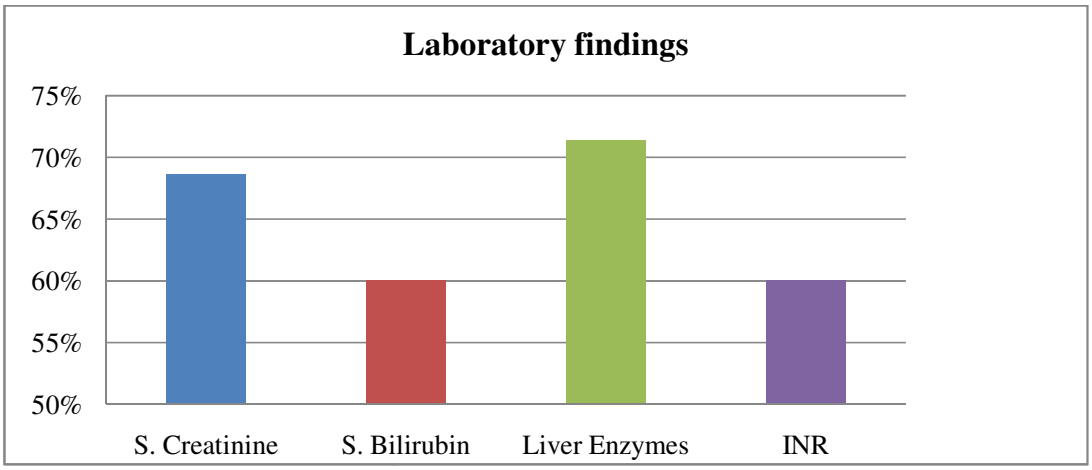

Chart 2: Lab Findings

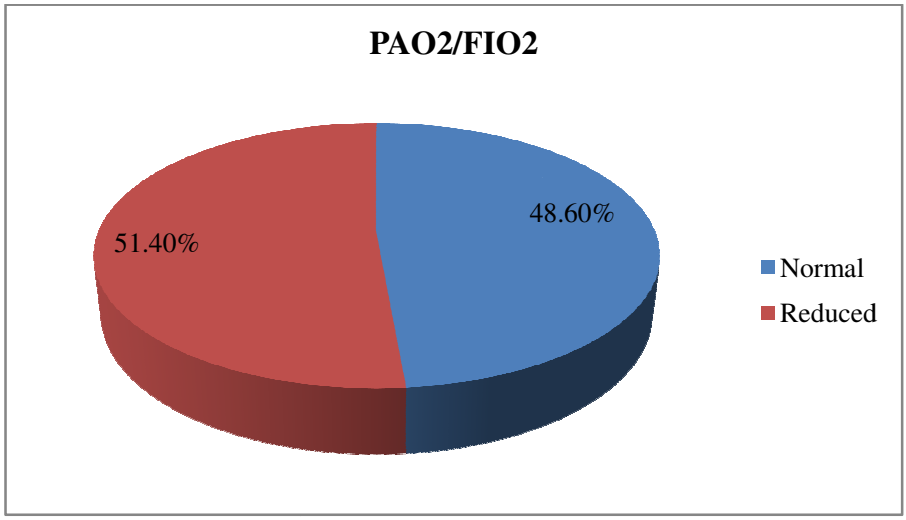

Chart 3: PAO2/FIO2 among patients

In the study, 22.9% received Ventilatory support, 37.1% received Inotropic Support and 40% received N Acetylcysteine.

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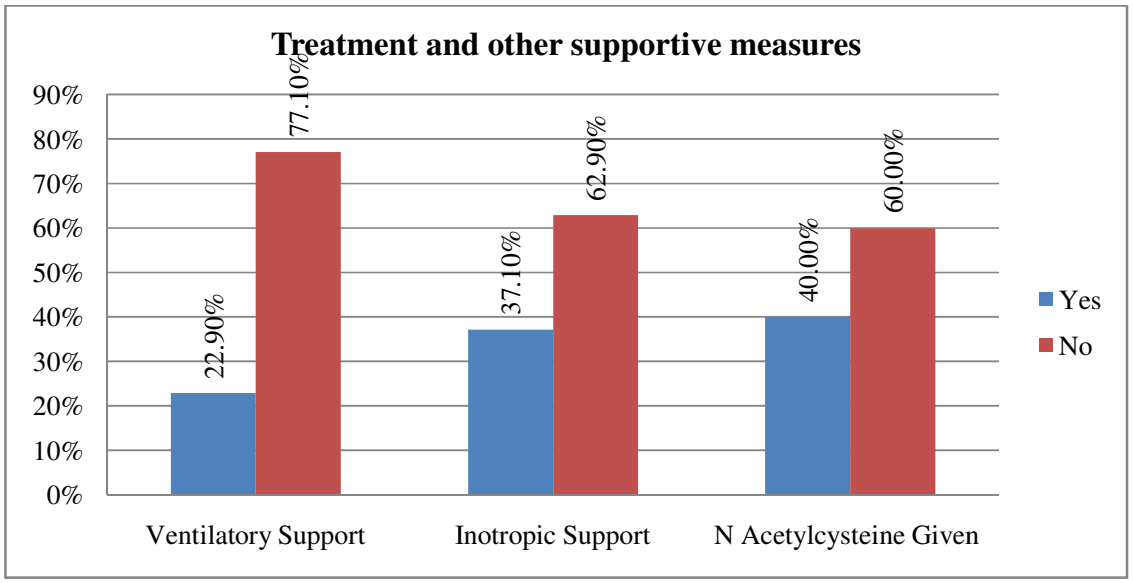

Chart 4: Treatment & Supportive measures

In the study, 54.3% recovered from Yellow phosphorus poisoning, 25.7% died and 20% were DAMA. The time of presentation varied among patients but there was no significant association between time of presentation and outcome. There was no significant association between Quantity of consumption and Outcome. However Mortality was high with higher doses of consumption.

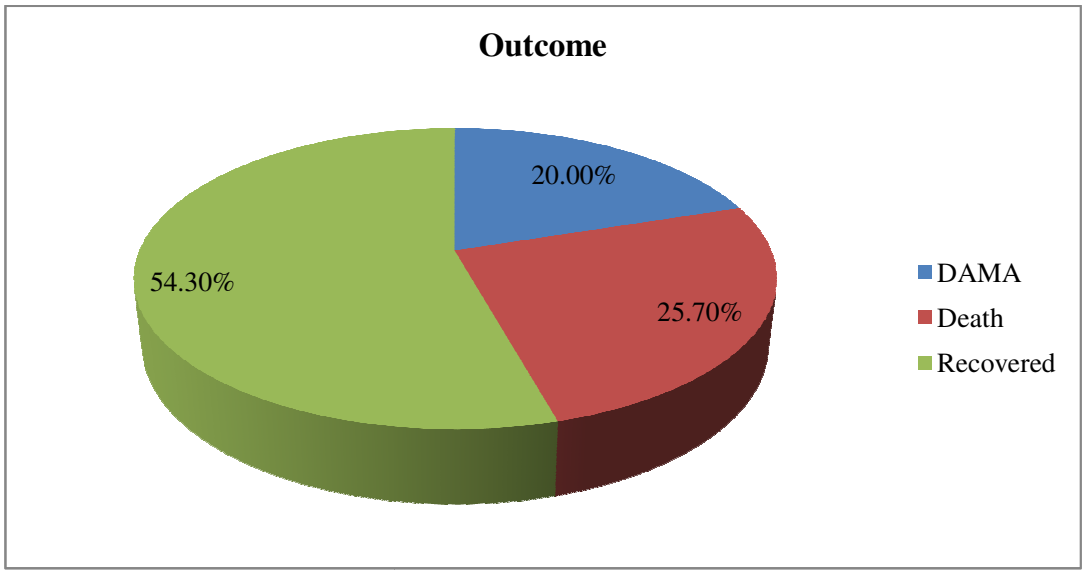

Chart 5: Outcome Distribution

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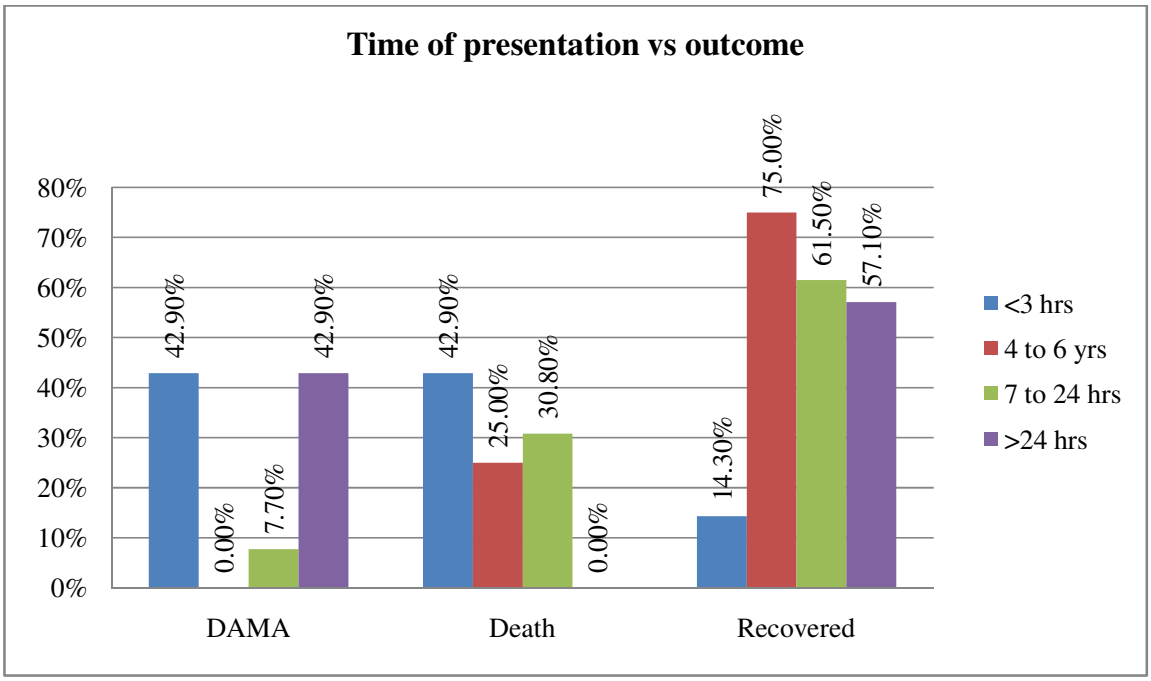

Chart 6: Time of Presentation vs Outcome

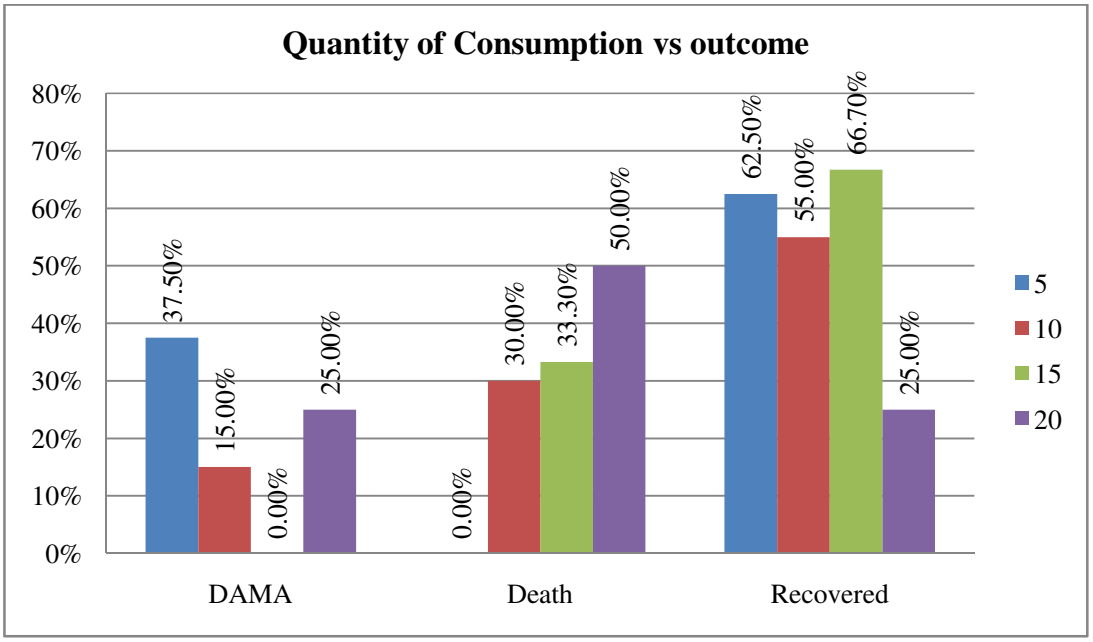

Chart 7: Quantity of Consumption vs Outcome

Among those who received N acetyl cysteine, 28.6% recovered, 57.1% died and 14.3% were DAMA and among those who were not given N Acetylcysteine, 71.4% recovered, 4.8% died and 23.8% were DAMA.

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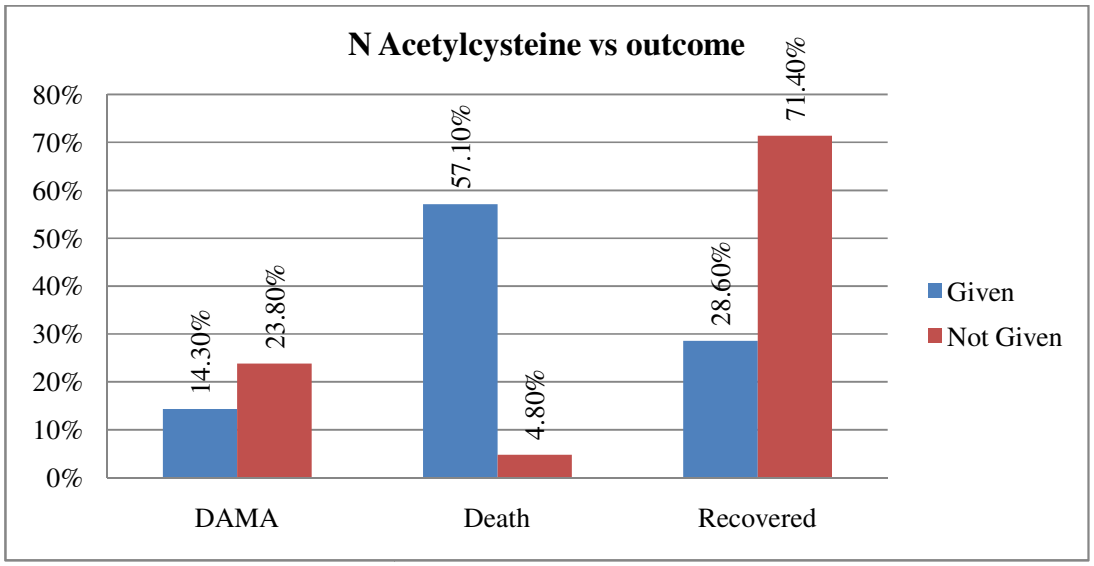

Chart 8: N Acetylcysteine vs Outcome

Among 19 subjects who recovered during the follow up, 10.5% stayed in hospital for <3 days, 73.7% for 4 to 7 days and 15.8% for >7 days.

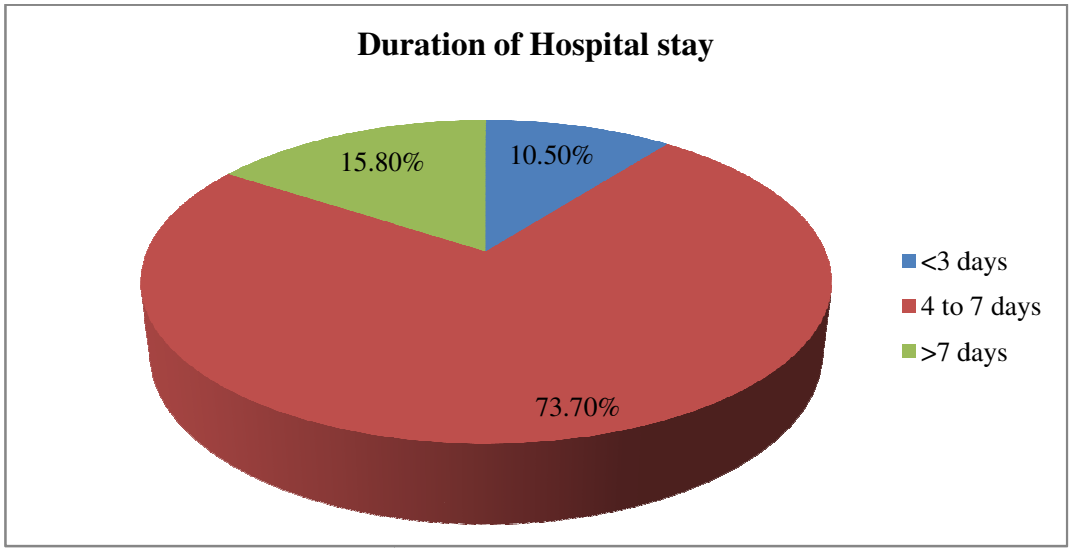

Chart 10: Duration of Hospital Stay Among Recovered

DISCUSSION

Elemental phosphorus is commonly available as red and white phosphorus. The latter is also known as yellow phosphorus and is used widely for military ammunition, fire crackers, fertilisers and in rodenticides, which contain yellow phosphorus in varying strengths in the range of 2–5%. Poisoning has been well documented as early as the 1950s.[5] Rodenticide poisoning constitutes 3% of all poisonings in India.[6] Yellow phosphorus is readily absorbed by the skin and mucous membranes, especially the gastrointestinal tract. Skin exposure following industrial accidents causes severe thermal burns.[7,8] Once absorbed, the level of phosphorus in the

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blood, renal and hepatic system increases within a few hours.[9] Toxicity of ingested yellow phosphorus is through direct tissue damage by exothermic production of phosphoric acid causing tissue corrosion, and the formation of phosphorus pentoxide which reacts with organic molecules.[10,11] In addition, hypocalcaemia results by preferential binding of calcium by phosphorus in serum.[12]

However, proposed that toxicity may be the result of changes in ribosomal function and protein synthesis, failure of regulation of blood glucose, alteration of lipoprotein synthesis and secretion of triglycerides, leading to intracellular accumulation and fatty degeneration of multiple organs, especially the liver, the kidney, the heart and brain. Of the 35 patients included in the study, majority were younger patients under the age group of 40 yrs and all were suicidal in intent. Males were more compared to females. Mishra et al[5] & Nalabothu et al[13] also showed similar results. This shows the high risk of the younger age group towards suicidal tendencies.

Patients presented with gastrointestinal manifestations most commonly followed by neurological and bleeding diasthesis. Acute renal failure, hepatic involvement with acute hepatic failure and coagulopathy with hypotention and circulatory collapse was seen in most of the patients. Mishra et al also showed similar results in his study. This is explained by the pathophysiology of the Yellow Phosphorous as mentioned above. . Decontamination and supportive therapy is advised as there is no specific antidote for yellow phosphorus. Reports on clinical improvement following timely initiation of NAC are contradictory. Even in our study early initiation of treatment with NAC did not show good outcome. NAC acts as a anti oxidant thus is used as a good hepatoprotective agent in treatment of Yellow phosphorous poisoning. Our patients also presented with renal failure, coagulopathy & circulatory collapse which explains the failure of good outcome even after starting NAC. Outcome among the Yellow phosphorous poisoning patients was poor as seen in other studies with high mortality rate even with rigorous treatment. This is attributed to the complications of poisoning, most common being renal failure, hepatic failure, coagulopathy and circulatory collapse.

CONCLUSION

Yellow Phosphorous is very lethal on consumption with high mortality rates. Since there is no specific antidote available for treatment, rigorous first aid and supportive care should be given to have a good outcome. Strict medical policies should be introduced to limit the use of these chemicals and create awareness among the people about the potential lethality of the substance. Measures should also be taken to provide good psychiatric counselling to all the high risk individuals to prevent the suicidal attempts.

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