

Original Research Article

Study of impact of clinical and biochemical parameters in aluminium phosphide poisoning

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ABSTRACT

Background: The aim of the study was to assess the relationship between clinical and biochemical parameters with outcome of aluminium phosphide toxicity.

Methods: Total 46 patients with aluminium phosphide poisoning admitted to the Hospitals attached to BMCRI between November 2018 and April 2019 were prospectively studied. After adequate history was obtained and immediate first aid measures given, clinical and biochemical parameters such as liver enzymes, serum creatinine, serum potassium and magnesium levels were studied. The patients were then followed up until outcome. Data was analysed using Descriptive statistics like mean, Standard deviation and percentages were calculated. Inferential statistics like Mann-Whitney test was used to compare the parameters between the two outcome groups (discharged and death) using SPSS software.

Results: Among the study population, between the two outcome groups males were 80.6% and females were 19.4%. the outcome was death in 10 patients and 36 patients were discharged. Tachycardia and hypoxia at presentation, Low Serum Magnesium and higher levels of serum creatinine at admission were found to be associated with higher mortality in patients with aluminum phosphide toxicity and were statistically significant.

Conclusions: These clinical and laboratory parameters can be considered as manifestation of hemodynamic compromise and are hence associated with poorer prognosis. Serum magnesium levels, can be used in prognostication of aluminium phosphide toxicity, owing probably to their effects of cardiotoxicity. Increased serum creatinine levels as a result of acute kidney injury, could also indirectly point to circulatory compromise. Hence these clinical and laboratory parameters at presentation have important implications.

Keywords: Aluminium phosphide, Biochemical, Poisoning

INTRODUCTION

Aluminum phosphide (ALP) is a commonly found compound in powder, pellet, or tablet form used for the safeguarding grains from pests especially rodents during transportation and storage.¹ Most commonly encountered form of exposure is inhalation of phosphine gas. Deliberate suicidal ingestions with aluminum phosphide include other modes of exposure.

Upon ingestion, phosphides are converted by gastric acid to phosphine gas, the primary toxic agent. Subsequently phosphine gas is absorbed from the gastrointestinal tract into the blood stream. The mechanisms of toxicity include;

- Inhibition of oxidative phosphorylation,
- Free radical production with promotion of lipid peroxidation

- Cholinesterase inhibition.

Lethal dose of ALP is 0.15 to 0.5 g.² Phosphide toxicity occurs rapidly, typically within 30 minutes of exposure. Exposure results in airway irritation and breathlessness. Other features include nausea, diarrhea, vomiting, jaundice, dizziness, headache, numbness and paresthesia.

Severe toxicity manifests as disseminated intravascular coagulation (DIC) and even Myocardial damage resulting in refractory hypotension, Congestive cardiac failure. Respiratory Complaints such as cough, cyanosis, dyspnea and pulmonary edema, and even ARDS Neurological complications such as headache, convulsion and altered sensorium can also be present.

Few studies have shown the importance of biochemical parameters in assessing the prognosis, but with contradictory results. This study aims at finding a correlation between the lab parameters and the outcome in these patients with aluminium phosphide toxicity.

METHODS

A total of 46 patients who had alleged history of ALP poisoning and presented to the emergency department at hospitals attached to BMCRI between November 2018 and April 2019 were studied. All patients more than 18 years old, who gave informed consent were included. Exclusion criteria was those patients with underlying Chronic Kidney disease or liver disease, Sepsis, known leucopenia.

Relevant Data was collected for socio-demographic parameters such as age, sex, duration of stay in the hospital. Clinical profile was analyzed for symptoms at onset, pulse, blood pressure (BP), respiratory rate, oxygen saturation total leukocyte count, Serum Creatinine, Liver enzymes, Serum Potassium and Magnesium levels, all measured at the time of presentation. All the patients were managed in Intensive Care Unit (ICU) with mainly supportive treatment. Gastric lavage was done on every patient. Blood gas analysis and an Electrocardiogram was done for all patients. All patients were prospectively studied and outcome of the patients was taken as death or discharged.

Statistical analysis

Data was entered in the excel spread sheet. Descriptive statistics like mean, standard deviation and percentages were calculated. Inferential statistics like Mann-whitney test was used to compare the parameters between two groups (discharged and death). Chi square test was also used for association of qualitative variables using SPSS (statistical Package for Social Sciences) version 2.0. A p value of less than 0.05 or lesser was taken as statistically significant.

RESULTS

The mean Age of the population was 29.64±7.74 years, 74 % were males and 26% were females. Out of the total of 46 patients, 10 died and 36 were discharged.

Table 1: Cross tabulation of pulse rate and outcome in the study population.

Outcome		Pulse rate			Total
		No tachycardia	Not Recordable	Tachycardia	
Death	Count(n)	3	1	6	10
	% of Total	6.5%	2.2%	13.0%	21.7%
Discharged	Count	33	1	2	36
	% of Total	71.7%	2.2%	4.3%	78.3%
Total	Count	36	2	8	46
	% of Total	78.3%	4.3%	17.4%	100.0%

Chi-square value- 18.08 p- 0.00

Table 2: Cross tabulation of oxygen saturation and the outcome groups.

Outcome		SPO ₂			Total
		Hypoxia	No hypoxia	Not Recordable	
Death	Count(n)	3	6	1	10
	% of Total	6.5%	13.0%	2.2%	21.7%
Discharged	Count	1	35	0	36
	% of Total	2.2%	76.1%	0.0%	78.3%
Total	Count	4	41	1	46
	% of Total	8.7%	89.1%	2.2%	100.0%

Chi-square value- 18.08 p- 0.00

Tachycardia at presentation was found to have a worse outcome and was statistically significant as shown in Table 1.

Hypotension at the time of presentation although associated with poorer outcome, was not found to be statistically significant in this study. Patients with Tachypnea and hypoxia at presentation were also found to have a graver outcome as shown in Table 2 and was statistically significant.

The most common finding on Electrocardiogram (ECG) was Sinus tachycardia, 1 patient had Sinus bradycardia and all other patients had normal ECG.

Among the Biochemical Parameters, WBC count did not have any effect on the outcome. Acidosis on ABG, Liver Function Tests and serum potassium levels were also assessed in these patients and did not have any significant correlation with the outcome. However, Increased Serum Creatinine Levels at presentation as shown in Figure 1, had a worse outcome. It was statistically significant (p=0.032).

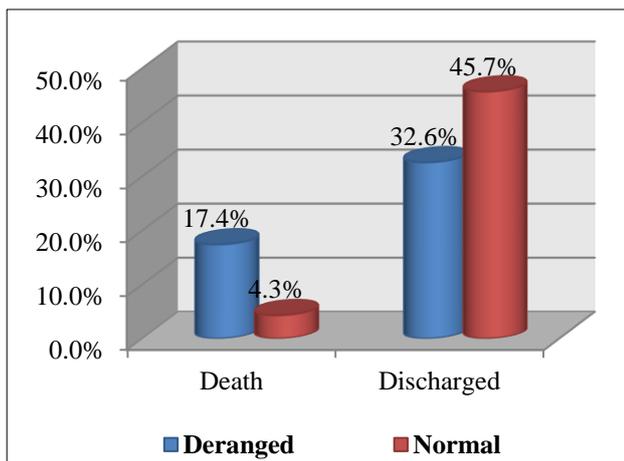


Figure 1: Cross tabulation of serum creatinine levels and final outcome.

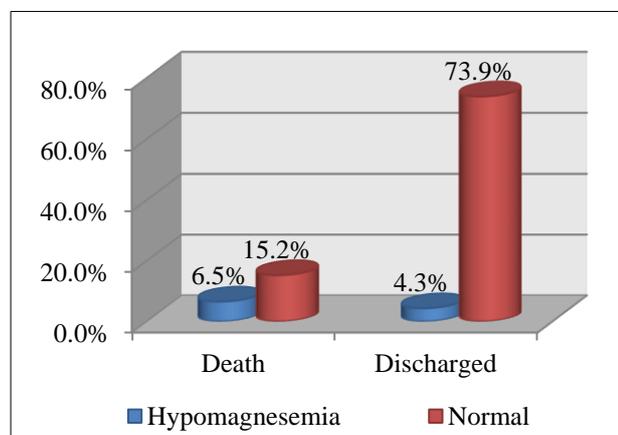


Figure 2: Cross tabulation of serum magnesium levels and final outcome.

It was also found that lower serum magnesium levels at presentation had a worse prognosis, and the values were statistically significant (p 0.028) as depicted in Figure 2.

DISCUSSION

ALP is a powerful insecticide and widely used in the agricultural community in India. In this study, 46 patients with ALP poisoning were evaluated to determine the correlation between clinical and biochemical parameters and ALP poisoning.

The pulse rate at presentation had a statistically significant outcome in our study (Tachycardia had a worse outcome) This was unlike the study conducted by Chugh et al, where there was no significant difference in pulse rate among survivors and nonsurvivors.³ This can be attributed to the severity of hemodynamic compromise, caused by the toxin.

Hypotension was also predominantly found in the group that did not survive, although this was not statistically significant. This justifies that the patients with more cardiac stability improved as compared to the expired. Primary care physicians must examine for hypotension and tachycardia and triage the patients because in the present study these two parameters were associated with poor prognosis. Chopra et al, also found that hypotension was the most important cause of death in these patients.⁴

In another study done by Chugh et al, the incidence of cardiac abnormalities as assessed by ECG changes was 50% in the study.⁵ The arrhythmias, conduction disturbances and ischaemic pattern were common when hypomagnesaemia was severe. In our study, Tachycardia at presentation was associated with a higher mortality and was statistically significant. This could be as a result of the underlying electrolyte disturbances itself, or because of cardiotoxicity of the compound.

Mathai et al, reported that AST, ALT, bilirubin had an effect on mortality rate.⁶ However Taramasari et al, found no statistical significance.⁷ Even in this study, no statistical correlation was found between liver enzymes and the outcome of the patients significantly, probably because liver enzymes take longer time to rise, after consumption of the compound.

Serum creatinine was found to elevated in the cases who had died in a study conducted by Singh et al, who found increased blood urea, low magnesium and potassium levels.⁸ Study showed similar results with a statistically significant correlation with mortality in patients presenting with high creatinine levels.

In a study conducted by Jaiswal et al, there was no significant difference in pH among survivors and nonsurvivors.⁹ This study did not show a statistically significant correlation as well. In a study conducted by Mathai et al, no statistical significance was found

between potassium levels and mortality.⁶ However in this study, mortality was found to be higher in those with hypokalemia and was statistically significant

The outcome of the study was death in 10(21.7%) and discharge in 36 patients. Mortality rate was low in this study than reported by various workers. Singh S et al, 73% mortality, Chugh SN et al, who reported 66% mortality in their studies.^{3,8} This was probably due to Immediate gastric decontamination with KMNO₄ and coconut oil, rapid identification of electrolyte disturbances and cardiac membrane stabilization. Also among the survivors, 22 consumed previously opened tablets (stale), which probably had already released the phosphine on exposure to moisture.

Limitations of the study includes short sample size and short time period of study.

CONCLUSION

Clinical parameters such as tachycardia and hypoxia, biochemical parameters such as raised creatinine levels, at presentation, low magnesium levels have been associated with worse outcomes in patients with aluminium phosphide toxicity.

Since the compound is extremely toxic with death resulting within a very short duration after consumption, identifying variables that predict the outcome at the time of presentation, play a very important role. Complications should be predicted, with aid of these biochemical parameters, and treatment should be aimed at correcting them.

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