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KEY WORDS:

A STUDY OF CLINICAL PROFILE IN ALUMINIUM PHOSPHIDE POISONING

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INTRODUCTION

Aluminum phosphide poisoning has now become popular and presently used due to its low cost, easy availability. Aluminum phosphide was declared an ideal fumigant pesticide in 1973 and now it has become one of the most commonly used suicidal poison in north and central agricultural states. The fatality in this poisoning is very high that this is epitomized as an agent of sure death in some parts of our country. Aluminum phosphide is a solid fumigant pesticide widely used as a grain preservative. Many trade names like Quickphos, Celphos, Alphos, Fumigant and Phostoxin are available in the market as greenish gray tablets (packed in metal tubes, weighing 3 gms each capable of liberating 1 gm of phosphine gas). In recent past poisoning due to aluminum phosphide has increased in northern and central parts of India reaching epidemic proportions. Poisoning is more common in the younger age group, in rural areas and is mostly suicidal. When Aluminum Phosphide comes in contact with water (Moisture of grain) or hydrochloric acid of stomach it is liberate phosphine gas (active ingredient), ammonia and carbon dioxide. Phosphine protects the stored grains from all types of insects. It is rapidly absorbed throughout the gastrointestinal tract after ingestion and through lungs after inhalation. Phosphine acts by inhibiting cytochromic oxidase enzyme. Free radicals are in excess due to their increased production and less destruction leading to lipid per-oxidation of cell membrane and ultimately the death of the cells. It impairs mitochondrial metabolism and inhibits electron transport system of the respiratory chain, stimulating generation of superoxide radicals and brings about protein denaturation and lipid per-oxidation. Epigastric burning, pain, nausea and vomiting are the common initial features. Later hypotension or shock, Brady or Tachycardia, conduction disturbances, arrhythmias, myocarditis, pericarditis, acute congestive heart failure, cough, dyspnea, cyanosis, ARDS, oliguric or non-oliguric renal failure, tender hepatomegaly, acute hepatic encephalopathy, altered mental state. restlessness without alteration in consciousness, bleeding tendency, multiorgan failure, may occur. Patients remain mentally clear till cerebral anoxia due to shock supervenes resulting in drowsiness, delirium and coma. Biochemical, metabolic and acid base disturbances in the form of hypoglycemia, hypokalemia, hypos or hypermagnesemia, hypoxia, hypo and hypercarbia and acidosis have been reported. The cardiovascular toxicity is the cardinal feature of the aluminum phosphide poisoning and is the major cause of death. Mortality in this poisoning depends mainly on cardiac involvement, persistence of shock, ARDS, electrolyte imbalance, amount and freshness of tablets and delay in institution of therapy.

MATERIAL AND METHODS

A total of fifty patients of aluminum phosphide poisoning with age groups ranging from 10 years to 50 years were selected for the present study (42 males and 8 females) from central causality, DMCH and from the medical ward and ICU of the Darbhanga Medical College and Hospital. Only those cases were included in this study who had definite history of aluminum phosphide poisoning or had poisoning with unknown substances foul or decaying fish like smell and physical verification of the poison consumed.

Detailed history was taken from the patients, their attendants and close relatives with a view to know whether it was suicidal, accidental or homicidal. The diagnosis of ALP poisoning is based on –

(a) History of ingestion of ALP compound.
(b) Clinical manifestations including shock.
(c) Foul or decaying fish like smell in breadth.
(d) The ECG changes and metabolic acidosis.

Observation And Results

Table 1 (Age incidence)

Age (Yrs.)	No. of Patients	Percentage. %
<20 yrs	09	18
21-40	38	76
>40	03	06

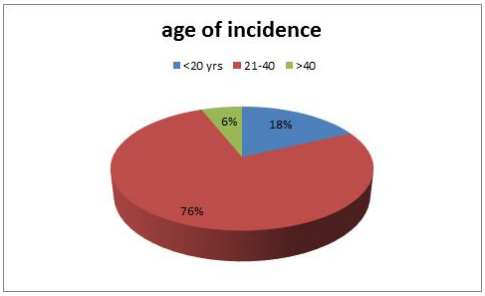

Table – 2 (Cause of Poisoning)

Cause	No of Patients	Percentage %
Suicidal	48	96
Accidental	02	04

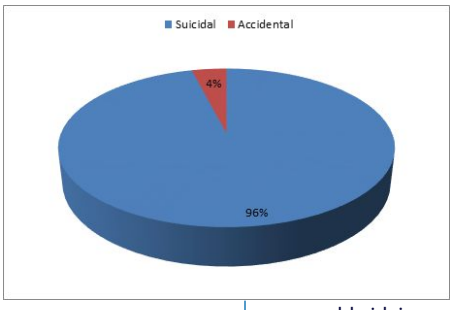

Table 3(Presentation)

Presentations	No of patients	Percentage %
Shock	48/50	96.00 %
Unconscious	20/50	40.00 %
ECG Abnormality	30/50	60.00 %
ST- T Changes	12/30	40.00 %
Tachy-arrhythmia	10/30	33.34 %
Brady-arrhythmia	08/30	26.63%
Metabolic Acidosis	34/50	68.00 %

Table 4 (Time of death after admission)

Time Since Admission	No of Patients	Percentage %
0-6 hours	19/50	38.00 %
06-12 hours	08/50	16.00%
12-24 hours	02/50	04.00 %
>24 hours	02/50	04.00 %
Total Death	31/50	62.00 %
Survived	19/50	38.00 %

Total Death in First 12 hr of admission 27/50 (54.00%)

Table 5 (System Involvement)

System	No of patients	Percentage
GIT	50	100 %
CVS	48	96 %
Respiratory	26	52 %
CNS	20	40 %
Hepatobiliary	12	24 %
Renal	03	06 %

DISCUSSION

Isolated cases of fatal exposure to phosphine gas have been reported in the world literature when Aluminum Phosphide (AlP) was used as a fumigant pesticide for bulk shipment of wheat. In India the first case of aluminum phosphide poisoning was reported in 1981 from Indore. Since then series of cases have been published from different Northern states of India. However, due to the green revolution in northern and central India and the majority of population being engaged in cultivation, potent pesticides have been available easily and their low cost and lethal effect have changed the pattern of poisoning in this region. In the current decade there has been a gradual increase in self poisoning by aluminum phosphide. Both accidental and suicidal cases have been reported. Easy availability of highly toxic substances like aluminum phosphide in the household at peak moments of frustration and depression has become the major cause of death by suicide in young men and women in this part of country. Aluminum phosphide poisoning is more frequent in young but cases in all age groups have been reported. Fatal dose of aluminum phosphide poisoning is 0.5-1 gm and fatal period is 30 minute to 1 hour. Immediate cause of death is intractable shock, acidosis, serious cardiac arrhythmias and ARDS. A total of 50 patient of aluminum phosphide were induced in this study most of the cases were found in the age group of 11-50 year. Among this age group 21-30 year the most vulnerable for this poisoning which is in agreement with the earlier studies by Chugh et al (1988) on 160 patients who reported most of their patients between the age group of 20-40 year. Maximum number of cases between the age group of 20-45 year in a study on 102 patients (Bajaj et al 1988). In our study, out of 50 patient 96% cases were of suicidal and 4% cases were of accidental in nature.

In our study, out of 50 patients 100% show GI toxicity while cardiovascular, respiratory, CNS, Hepatobiliary, renal were involved in 96%, 52%, 40%, 24%, 6% of cases respectively.

Chugh et al (1991) in his study on 191 patients recorded a similar toxic profile of patients with aluminum phosphide poisoning in the following sequence – gastrointestinal tract involved in 100% cases while cardiovascular system, cerebral nervous system, respiratory system and renal in 95%, 95%, 51% and 50% of cases respectively.

The common ECG abnormalities in this study are ST-T changes (20%), tachyarrhythmia (18%) and Bradyarrhythmia (16%). This observation agreed the earlier report of Chugh SN et al (1991) on 190 patients that the arrhythmias, conduction disturbances and S-T changes occurred more or less in equal frequency. There was no correlation between individual ECG abnormalities and mortalities.

In tachyarrhythmia, Supraventricular tachycardia was reported in 6% atrial fibrillation in 4%, ventricular premature beats in 4%, non sustained ventricular tachycardia in 2%, atrial premature beats in 2%.

In Bradyarrhythmia most common observation in this study was sinus bradycardia in 8% followed by RBBB in 4%, 1 Atrioventricular block in 4%.

The ECG changes which appear in aluminum phosphide poisoning could be due to toxic myocarditis developed due to cellular and sub cellular toxicity of phosphine.

Mortality observed in this study was 62%, 38% died within 6 hour of admission, 16% died within 6-12 hour, 4% in 12-24 hour, 2% within 24-72 hour and 2% in > 72 hour. Our study shows that first 24 hour of period after admission was critical as maximum number of mortalities was reported in this period. Metabolic acidosis was seen in 68% cases (34 patients out of 50); 28 patients died and 6 patients survived.

Phosphine gas released from aluminum phosphide causes cellular and sub cellular hypoxia due to free radical injury leading to widespread endothelial damage resulting in multiorgan failure, ARDS and cardio toxicity. Out of all cardio toxicity is the major cause of death emphasizing the need of intensive cardiac monitoring. . The usage of coconut/mineral oil in gastric lavage was found to improve outcome substantiating the claims of two case reports. A combination of coconut oil lavage, magnesium sulfate therapy and proper supportive care in an intensive care unit will definitely improve the survival rates from this deadly poison.

Summary And Conclusion

The Gastrointestinal tract involvement was present in all the cases (100%) followed by involvement of CVS (96%), Respiratory system (52%),Central nervous system (40%), Hepatobiliary system (24%) and Kidney (10%). Shock, was found in 92% of cases. Mortality was found in 62% of cases (31 out of 50 patients) more during 0-6 hour period (38%) since admission followed by 16% in 6 to 12 hour period. This shows most critical period from management point of view is first 12 hours. Metabolic acidosis was seen in 68% cases (34 out of 50) patients); 28 patients died and 6 patients survived.

It is concluded that the aluminum phosphide is highly toxic protoplasmic poison producing cardio-toxicity manifested as myocarditis (Evidenced by ST-T changes) tachy and Bradyarrhythmia, shock and other system involvement. The antidote against it is not known making the management of this poisoning very difficult. As mortality in this poisoning is very high, it is emphasized that there should be every effort to prevent this poisoning.

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