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 breath.
(d) The ECG changes and metabolic acidosis.

Observation And Results
Table 1 (Age incidence)

<table>
<thead>
<tr>
<th>Age (Yrs.)</th>
<th>No. of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20 yrs</td>
<td>09</td>
<td>18</td>
</tr>
<tr>
<td>21-40 yrs</td>
<td>38</td>
<td>76</td>
</tr>
<tr>
<td>&gt;40 yrs</td>
<td>03</td>
<td>06</td>
</tr>
</tbody>
</table>

Table – 2 (Cause of Poisoning)

<table>
<thead>
<tr>
<th>Cause</th>
<th>No of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suicidal</td>
<td>48</td>
<td>96</td>
</tr>
<tr>
<td>Accidental</td>
<td>02</td>
<td>04</td>
</tr>
</tbody>
</table>

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The common ECG abnormalities in this study are ST-T changes following sequence – gastrointestinal tract involved in 100% cases. Chugh et al. (1991) in his study on 191 patients recorded a similar cases were of accidental in nature. In our study, out of 50 patient 96% cases were of suicidal and 4% cases were of accidental in nature.

In the study, 62%, 38% died within 6 hours of admission, 16% died within 6 -12 hour, 4% in 12-24 hour, 2% in 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.

The ECG changes which appear in aluminum phosphate 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% in 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 phosphate causes cellular and sub cellular hypoxia due to free radical injury leading to widespread endothelial damage resulting in multorgan failure, ARDS and cardiac toxicity. Out of all cardiac 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 6-6 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 phosphate 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.

**References**