ORAL ALUMINIUM PHOSPHIDE POISONING

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Aluminium phosphide is a solid fumigant pesticide, commonly used for grain preservation in almost every household of North India. Due to its efficacy, it was declared an ideal pesticide(l). However, in recent years, due to its low cost, free availability and highly toxic nature, it has emerged as the most commonly used, self-poisoning agent(2-4). It is reported that the total number of deaths due to aluminium phosphide poisoning has exceeded the number of fatalities in the Bhopal gas tragedy(5). Almost all the reported series have shown a higher incidence of poisoning in the adolescent age. Children can ingest the drug accidentally, but usually they are the innocent victims when it is given to them by parents who take it for suicidal reasons(2-4,6). Despite the alarming increase in the incidence of this poisoning, there is hardly any mention about it in the literature and text books.

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Aluminium phosphide is available as 3.0 g tablet (trade names Celphos, Alphos or Quickphos). On exposure to moisture, aluminium phosphide liberates highly toxic phosphine gas (PH_3)(7,8). Each tablet (containing 56% aluminium phosphide and 44% aluminium carbonate) liberates 1.0 g of phosphine. The lethal dose for a human weighing 70 kg is 150-500 mg(9). When used for grain storage, the gas is lethal to insects and rodents. Phosphine gas dissipates rapidly into the air leaving very little residue. Such a grain is safe for human consumption(9).

In human beings, phosphine causes toxicity by inhalation and ingestion. Whereas only isolated cases of toxicity by inhalation (primary toxicity) from fumigated bulk of grain have been reported (10), it is the latter route which is commoner(2-5).

After ingestion of aluminium phosphide tablets, phosphine gas. is liberated in the stomach in the presence of hydrochloric, jfcid. The liberated phosphine gas is absorbed throughout the gastrointestinal tract by simple diffusion and is mainly excreted through kidneys and lungs(9).

Epidemiology

The exact magnitude of aluminium phosphide poisoning is unknown due to social and legal reasons. The mode of poisoning is usually suicidal, occasionally accidential and rarely homicidal. Irrespective of the sex, the incidence is higher in the rural population. This may be due to illiteracy and easy availability

of the toxic pesticide in agriculture based households(2,4-6).

Pathogenesis and Pathophysiology

The exact mechanism of action of phosphine is not clearly understood but appears to be hypoxic as supported by post-mortem findings(2,3,10). Like cyanide, phosphine produces hypoxia by non-competitive inhibition of cytochrome oxidase at mitochondrial level(ll). Within 24 hours there are features of severe metabolic acidosis, peripheral capillary leakage, electrocardiographic changes and enzymatic evidence of global myocardial injury, depressed left ventricular ejection fraction and a late development of adult respiratory distress syndrome(2,3,12).

Clinical Features

The clinical features depend upon the number and freshness of tablets ingested. Clinical toxicity and mortality is less with pre-exposed tablets because of already released phosphine gas from such tablets(2,3). The onset of the symptoms is almost instantaneous(2-4) and include:

(a) *Mild ingestional toxicity:* Patient usually has nausea, vomiting, pain abdomen, thirst, headache; these patients usually recover.

(b) Moderate to severe poisoning: This is usually associated with marked symptoms and systemic manifestations which are progressive and invariably fatal. Usually gastrointestinal symptoms appear first and are followed by irreversible shock. Epigastric burning, persistent vomiting, thirst, hypotension, shock and respiratory distress are most common features described in all reports(2-4,6,12-14).

Patients can present with epigastric burning, pain abdomen, persistent vomiting and nausea. Cardiovascular symptoms include hypotension, shock, bradycardia or tachycardia, acute congestive heart failure, toxic myocarditis presenting with electrocardiographic abnormalities like ST-T changes, conduction and rhythm disturbances. Cough, dyspnea, adult respiratory distress syndrome after 24 hours, oliguric and non-oliguric renal failure, headache and dizziness and restlessness without alteration in consciousness are other important features. Less commonly observed feature are diarrhea, jaundice and acute hepatotoxicity, convulsions, ataxia, paraesthesias, coma and pericarditis.

Death usually occurs within 24 hours due to acute cardiotoxkity. However, it may be delayed upto 4 days when adult respiratory distress syndrome supervenes(2,9). Other causes of death include gastrointestinal bleeding, metabolic derangements and hepatic failure(2,9).

Management

Since there is no specific antidote, treatment is basically symptomatic and aimed to maintain vitals till phosphine is excreted from body through lungs and the kidneys(2,9-12).

Measures may be taken to decrease phosphine absorption from the gastrointestinal tract. They include:

(a) Repeated gastric lavage with mild oxidizing agents *(e.g.,* 1:10,000 potassium permanaganate) followed by sodium bicarbonate lavage.

(b) Activated charcoal administered orally followed by a cathartic to increase phosphine excretion.

(c) H_2 receptor antagonist like intra venous ranitidine and oral antacids can be used.

(d) Liquid paraffin may be administered to increase the excretion of aluminium phosphide and phosphine from the gut.

In addition, measures may be taken to increase the excretion of phosphine from the body. Adequate hydration to maintain a satisfactory urinary output is recommended. Intravenous diuretics may be used if blood pressure is normal. Hemodialysis is effective in cases with uremia(15).

Other supportive measures include oxygen, treatment of shock with fluids, low dose dopamine, intravenous hydrocortisone, correction of acidosis with sodium bicarbonate and assisted respiration may be required. Magnesium sulphate has been tried with limited success(16) for its general membrane stabilizing effect in cardiac cells(17).

Outcome

The reported mortality of all aluminium phosphide poisoning varies from 37% to 100%(2-5). The outcome depends on the number and freshness of tablets, presence of shock and presence or absence of poor prognostic factors.

Prognostic Features

Poor prognostic features include shock not responsive to standard therapy, severe metabolic acidosis, hypoxia, electrolyte imbalance, arrhythmias, aspiration pneumonia and anemia due to gastrointestinal bleeding.

Complications

Reported complications of aluminium phosphide poisoning are gastrointestinal hemorrhage, adult respiratory distress syndrome, acute congestive cardiac failure, pericarditis and aspiration syndrome.

Prevention

Since death is rapid and survival after significant poisoning is rare, prevention is the logical option. The most effective way for prevention is to either ban or impose strict regulations on the sale of aluminium phosphide tablets. Strong and immediate measures should be taken by appropriate agencies to combat this problem.

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