

## Aluminium Phosphide Poisoning : A Challenge for the Physician



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### Abstract

Acute Aluminium Phosphide (ALP) Poisoning by inhalational or ingestional exposure is seen world-wide. Except in Morocco, Denmark and India, nowhere in the world, it is ingested for deliberate self-poisoning (DSP). The rural belts of North states of India are worst affected with high and variable mortality rates in the young population with male dominance. The poison affects all systems; the shock, cardiac arrhythmias with varied electrocardiographic (ECG) changes and gastrointestinal (GIT) features being the most prominent. The role of magnesium sulphate ( $MgSO_4$ ) in reducing the cardiac arrhythmias and mortality is well documented and the problem needs multi-faceted approach in the form of preventive measures, strengthening of medical-aid services at gross root levels, stringent restrictions on supply of ALP in open market, updating the management techniques and overall a search for an antidote.

### Introduction

During the earlier times, the use of ALP as grain fumigant for bulk shipment of wheat led to isolated fatal inhalational exposure to phosphine in stray reports of accidental ALP toxicity on board a grain freighter and in labourers engaged in loading of wheat on ships (1,2). Washington State's five year experience (1992-1996) reports ALP exposure as inhalational or ocular linked to fumigate shipping containers when later were opened, improper disposal of ALP at a warehouse, to fumigate grain towers or hay trucks in handlers and applicators of ALP (3). No reports are available in English literature from any part of the world where ALP has been ingested as a

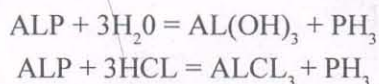
poison for committing suicide except in Morocco (1995) where recently death rate from suicide due to self administration of ALP pills (Phostoxin®) is high (4) and in Denmark (1996) a case of ALP ingestion has been reported and during last two decades poisoning due to ALP ingestion has been reported (5) widely from different Northern States of India where the ALP poisoning was unknown before 1980. First case was reported in 1981, but since then, the number of cases is progressively increasing throughout the North India so much so that it is now the single most suicidal method and the problem has acquired an epidemic proportion (6-8).

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## Aluminium Phosphide

ALP is a solid fumigant and ideal pesticide since 1940 as it is cheap, most efficacious and easy to use and freely available on the counter in India (as Alphos, Celphos, Quickphos, Phostek, Phosfume and Synfume) and in Morocco (as Phostoxin), in form of chalky white or brown 3 gm. tablets containing 56% of ALP and 44% of ammonium carbonate (4,9,10). The tablets are taken out of sealed container and placed on stored grains and storage container is closed for few days to combat moles and vermines in granaries (4). ALP has relatively high vapour pressure that allows it to penetrate porous material effectively. On coming into contact with water or moisture or OH radical of air or hydrochloric acid in stomach, 3 gm. tablet of ALP liberates 1 gm. of phosphine or phosphorus hydrogen (10,11), i.e.,



## Phosphine (PH<sub>3</sub>)

PH<sub>3</sub> is a colourless gas with fishy or garlic odour with odour threshold of 0.14 ppm and a vapour density of 1.17 (2,3). It is removed with half life of 5-24 hours which is dependant upon surface and permeability of solid matrix. The non-toxic residues i.e. phosphites and hypophosphites of aluminum left in the grains, are less than 0.1mg./kg of PH<sub>3</sub> for raw cereals, the WHO/FAO recommended permissible levels regarded safe for human consumption (10,11).

## PATHOGENESIS OF TOXIC EFFECTS

Irrespective of routes of exposure, viz., the inhalational, ingestional or ocular, the toxic effects of PH<sub>3</sub> are same except initially and are dose dependant. Some of ALP is directly absorbed from stomach to reach liver to liberate PH<sub>3</sub> slowly to prolong the toxic effects of poisoning (10).

PH<sub>3</sub> is rapidly absorbed from stomach or lungs by simple diffusion, oxidised slowly and is excreted in urine as hypophosphite and also excreted unchanged through lungs significantly. The systemic toxic effects appear 1 to 60 minutes after ingestion (8).

PH<sub>3</sub> inhibits the electron transport resulting from preferential inhibition of cytochrome oxidase leading to respiratory chain inhibition which leads to cellular hypoxia and small vessel injury which is further potentiated by cardiotoxicity due to anoxic myocardial damage and shock (12).

Direct toxic effect of ALP on myocardium or hypomagnesemia brought on by focal myocardial damage leads to arrhythmias. Hypotension and shock ensue within 3-6 hours of ingestion of ALP. In survivors, the cardiotoxicity and hypoxia disappear within 5-7 days due to excretion of PH<sub>3</sub> and restoration of normal cellular metabolism. The toxic chemical myocarditis leads to varied fatal ECG changes, 6 to 24 hours after ingestion in non-survivors, in the form of VE beats, conduction disturbance, LBBB, ventricular fibrillation, aberrant conduction and idioventricular rhythm terminally leading to asystole (13-16). The non-fatal ECG changes appear within 12 to 24 hours in survivors and disappear within 56 to 80 hours. Death in first 24 hours appears to be cardiogenic as evidenced by shock and ECG abnormality. The serum electrolytes are within normal limits and not correlated with ECG changes. Since the survivors show complete normal ECG recovery, it denotes that the effect of poisoning is due to some reversible factor leading to disturbance in the permeability of sodium (Na<sup>+</sup>), potassium (K<sup>+</sup>), calcium (Ca<sup>2+</sup>) and magnesium (Mg<sup>2+</sup>) ions leading to change in transmembrane action potential due to focal myocardial involvement and subsequent myocardial necrosis, resulting in release of reactive O<sub>2</sub> intermediates (17-19).



Hypomagnesemia seen in ALP poisoning may be due to an oxidative stress which buffers the  $Mg^{2+}$  resulting in increased susceptibility of oxygen free radicals injury and accelerated lipid peroxidation (20). High level of SOD (Superoxide Dismutase) and MDA (Malonyl dialdehyde) seen in non-survivors suggests their direct relation to mortality, whereas the catalase has inverse relationship. The return of SOD and MDA to normal or near normal levels in survivors suggests abolition of an oxidative stress due to elimination of phosphine. (21)

Intractable shock may be due to arrhythmia, conduction disturbance and myocardial damage. The peripheral circulatory failure (PCF) due to wide spread small vessel injury leads to peripheral vasodilatation leading to shock. Excessive vomiting due to fluid loss results into shock. Direct toxic effects of  $PH_3$  on adrenal cortex accompanied by decreased cortisol levels, leads to shock and high mortality (22,23).

Injury to alveolar capillary membrane by  $PH_3$  while being inhaled, leads to ARDS (Adult Respiratory Distress Syndrome) which may also occur rarely if patient has consumed more than 2 to 3 tablets of ALP. (24)

Wide spread capillary damage leads to bleeding diathesis, disseminated intravascular coagulation (DIC) and acute tubular necrosis (ATN). Shock and DIC lead to terminal renal failure. (25)

Direct toxic effects of ALP or its absorbed products or metabolic acidosis on muscles leads to muscular ischemia and degeneration of nonspecific nature leading to myopathy. (26)

In blood, the  $PH_3$  changes the circular dichroic spectra of haemoglobin and change in haeme iron leading to conformational changes of the prosthetic group (12). The blood  $PH_3$  levels are positively co-related to the clinical grades of ALP toxicity and to the dose of the pesticide consumed (27). The occurrence of intravascular

haemolysis with ALP poisoning in a patient with normal G6PD levels is of significance as jaundice in patients with this poisoning is often attributed to the hepatic damage alone (28).

In addition to the effect on the adrenal cortex, ALP also affects glucose metabolism leading to hypoglycemia, hyperglycemia or no change effects which can be attributed to the wide variety of changes in  $Mg^{2+}$ ,  $Ca^{2+}$ , phosphate, citrate or cortisol levels which act as active stimulatory or inhibitory modulators to enzymes and hormones that catalyse and regulate glucose metabolism (29).

### Epidemiology

In the largest series comprising of 418 cases reported from Rohtak, India, the hospital incidence of ALP poisoning was 0.06/1000 hospital admissions which progressively increased to 10/1000 in 1989-90 with male-female ratio of 2 : 1 and now is continuously increasing so as to surpass any other poisoning in Haryana (6,7). In Washington State, ALP poisoning formed 38.42% (15 out of 39 cases) of the fumigant-related illness (3). Stray incidence of ALP ingestion are reported from Morocco and Denmark (4,5).

ALP poisoning is more common in rural belt of North India where agricultural community irrespective of sex is more at risk which correlates positively with illiteracy, frustration, depression, failure in examination, disputes in the family, inability to find suitable avenues of income and easy availability of ALP in the house-hold. (8,30)

The occupational threshold value of  $PH_3$  is 0.3 PPM (3) and a concentration of more than 7 PPM in air can cause serious illness; concentration of 290-300 PPM is dangerous to life; 400 to 600 PPM is lethal in half an hour and that of 1000 PPM is rapidly fatal. The fatal dose after ingestion is 150 mg for a 70 Kg person and fatal period is 1 to 96 hours, the average being 28 hours. (6-8)

## Clinical Manifestations

The clinical features are more or less the same irrespective of the mode of toxicity, except the initial symptoms pertaining to the route of entry. The signs and symptoms depend upon the dose and severity of poisoning (22).

### (a) Inhalational Exposure (Primary Toxicity)

- (i) Mild : Skin, mucous membrane and eye irritation : dizziness, easy fatigability, tightness in chest, cough, nausea, vomiting, headache, diarrhoea and respiratory distress.
- (ii) More Severe : Vomiting, abdominal pain or cramps, ataxia, numbness, paraesthesias, tremors, diplopia, jaundice, muscular weakness, incoordination and paralysis.
- (iii) Very Severe : ARDS (Adults Respiratory Distress Syndrome), cardiac arrhythmias, pulmonary odema, convulsions, coma, hepatotoxicity and acute renal failure.

### (b) Ingestional Toxicity

- (i) Mild : Nausea, vomiting, headache, abdominal pain and discomfort. These patients usually recover.
- (ii) Moderate and Severe Systemic Manifestations :
  - **GIT System** : Nausea, vomiting, diarrhoea (20%), pain epigastrium, retrosternal pain and epigastric burning sensation (60%).
  - **Hepatobiliary System** : Acute hepatic failure (6%), jaundice, hepatitis and soft tender hepatomegaly. (31)
  - **Cardiovascular System (60-100%)** : Increased JVP, feeble heart sounds, S<sub>3</sub> gallop and muffled S<sub>1</sub>, hypotension, shock, arrhythmias, myocarditis and pericarditis (4,32).

- **Respiratory System (within 2 to 3 hours)** : Cough, dyspnoea, cyanosis, bilateral basal rales and rhonchi, respiratory failure and ARDS. (24)
- **Renal System** : Acute (oliguric or non-oliguric) renal failure.
- **Central Nervous System (50%)** : Headache, dizziness, diplopia, paraesthesias, ataxia, altered sensorium, restlessness, intention tremors, convulsion, hypoxic encephalopathy, coma and delayed haemorrhagic stroke (13,33).
- **Muscular System** : Muscle pain, severe muscle weakness, myopathy with muscle wasting and proximal muscle weakness (26).
- **Haemopoietic System** : Bleeding diathesis, DIC and jaundice (25,28).
- **Endocrinal System** : Hypoglycemia and hyperglycemia.
- **Shock** : (22,23).

### (c) Bad Prognostic Signs

Intractable shock, anemia, chest infection, metabolic acidosis, severe hypoxia, electrolyte disturbances, arrhythmias, oliguria, aspiration pneumonia, haemolysis, coma and DIC (8,34,35).

## LABORATORY INVESTIGATIONS

**Haemogram** : Haemoglobin is usually normal and there may be leucopenia.

**Urine** : Haematuria and albuminuria.

**ECG Changes (80%)** : ST-T changes (40%), ST depression, elevation or coving, hyperacute T-waves, multiple multifocal resistant ectopic beats, tachycardia, bradycardia, atrial or ventricular fibrillation, aberrant conduction, LBBB, RBBB, complete heart block and asystole (15).

**ABG-Analysis :** Hypoxia, hypercapnea or eucapnea, decreased bicarbonate ( $\text{HCO}_3$ ) levels and metabolic acidosis.

**X-ray Chest :** Normal except in ARDS, bilateral diffuse haze from hilum to periphery without cardiomegaly (24).

**Serum Biochemistry :** Transaminases (SGOT, SGPT) may increase 10 to 12 times, there may be low serum cholinestrase activity, increase plasma renin activity, serum electrolytes are usually normal initially and may increase terminally. Deranged renal function in ARF, increased serum amylase and increased CPK muscle fraction with myopathy has been reported in ALP (26,36).

**Skeletal Muscle Biopsy :** Swelling of muscle fibres with loss of striations.

**Echocardiography :** Marked L.V. systolic dysfunction with mean E.F.  $43.52 \pm 4.97\%$  on day 1 of poisoning (32,37).

**Silver Nitrate ( $\text{AgNO}_3$ ) Impregnated Paper Test :** It is based on property of  $\text{PH}_3$  to reduce  $\text{AgNO}_3$  to Silver (Ag) which gives black colour on the filter paper. It has high sensitivity as it is positive at low concentration of  $\text{PH}_3$  and even up to 2nd or 3rd day of poisoning in survivors. Test is 100% positive with gastric juice and 50% positive with breath (38).

**Atomic Absorption Spectrophotometry in Non-Survivors:**

**(a) Tissue Magnesium Content**

It is increased in different organs of patients who receive  $\text{MgSO}_4$  as compared with normal controls who did not receive it (18).

**(b) Histopathological Changes**

Seen in all organs viz ; in lungs, liver, kidneys, heart, brain and adrenals in form of congestion, odema leucocyte infiltration suggestive of cellular hypoxia.

**DIAGNOSIS**

Diagnosis is based on clinical suspicion, inhalational exposure or history of ingestion of ALP, corroborated by seeing the tablets of ALP or its empty container left in the house, clinical manifestations with garlic or decaying fish odour, unexplained shock (40), positive  $\text{AgNO}_3$  (38) test, chemical analysis of gastric juice and viscera (18) and histopathological changes (39).

**Management**

**(a) To Decrease  $\text{PH}_3$  Absorption**

- Gastric lavage with  $\text{KMnO}_4$  (1:10,000) immediately after admission and to be repeated twice or thrice.
- Activated charcoal 100 gm orally for absorbing  $\text{PH}_3$  from GIT.
- Medicated liquid paraffin and vegetable oils to accerlate the excretion of ALP and  $\text{PH}_3$  from the gut and inhibit release of  $\text{PH}_3$  from ALP (42).
- Reduction of organ toxicity can be achieved by using membrane stablizer like  $\text{MgSO}_4$  which corrects hypomagnesemia (43).
- Enhancing  $\text{PH}_3$  excretion with adequate hydration and renal perfusion with I/V fluids. In shock after raising SBP around 90 mm Hg, a 40 to 60 mg dose of furosemide may be tried. Dialysis has been tried and found beneficial in patients with BP maintained within normal limits.
- Detoxication of absorbed  $\text{PH}_3$  can't be done as there is no specific antidote (43,44).

**(b) Treatment of Hypoxia**

- $\text{O}_2$  administration by poly mask.
- Endotracheal intubation in unconscious patients.



- Assisted ventilation monitored by ABG-analysis.

### (c) Treatment of Shock

- Intravenous fluids 4 to 6 liters within 3-6 hours the 50% of which should be normal saline, with concomitant central venous pressure (CVP) monitoring to raise it to 10 cm. of H<sub>2</sub>O.
- I/V hydrocortisone 400 mg every 4 to 6 hours is highly effective to decrease capillary leakage in lungs in ARDS and to potentiate the responsiveness of the shock to the endo-or exogenous catecholamines.
- Monitoring vitals.

### (d) Treatment of Cardiac Arrhythmias

MgSO<sub>4</sub> is effective in first 24 hours in dose of 1 gm. I/V stat after dissolving in 100 ml of 5% dextrose and 1 gm. every hour for next 3 hours and then 1 gm. every 6 hours for 5-7 days in continuous I/V infusion in 5% dextrose (45,48). It corrects cardiac arrhythmias by modulating sympathetic, parasympathetic and slow channel kinetics (43,44,48-52). Mg<sup>2+</sup> level should be maintained at less than toxic levels of 10 meq/l. The magnesium level achieved with said dosage schedule is 2.00 to 3.1 meq/l.

The conventional antiarrhythmic drugs, viz; digoxin and xylocaine are not effective and cardioversion is not attempted due to diffuse myocarditis (48,53).

### (e) Metabolic Acidosis

The presence of moderate to severe acidosis with HCO<sub>3</sub> levels of less than 15 mmol/l is corrected by I/V Sodabcarb to raise HCO<sub>3</sub> level to 18 to 20 mmol/l for 3 to 4 days till PH<sub>3</sub> is excreted. The peritoneal or haemodialysis is useful in case, if metabolic acidosis persists in haemodynamically stable patients.

### (f) Complications

Other complications, viz; pericarditis, acute CCF, acute massive G.I. bleeding and ARDS require appropriate management (49).

### Mortality

It is high and variable and depends upon the dose and freshness of ALP consumed, rapid onset of clinical manifestations, delay in arrival at the hospital and delayed institution of the treatment, duration and severity of shock, vomiting (the earlier the vomiting the better the prognosis), the presence or absence of bad prognostic features, complications, lack of antidote and the modalities of treatment used, viz; only supportive : 70 to 100%, supportive + calcium gluconate : 60% and supportive + MgSO<sub>4</sub> : 25 to 45% (20,44,47). The mortality is 85 to 100% if ECG reveals infarct patterns, various blocks and arterial fibrillation. With the use of MgSO<sub>4</sub>, mortality has been further reduced to 20% in comparison to 44% in persons who did not receive MgSO<sub>4</sub> (20).

### A Clinical Study of ALP-Poisoning in Jammu

In a prospective study of 56 cases of ALP poisoning in Govt. Medical College Hospital Jammu during the period from 1995-1999, we found out the prevalence of DSP and accidental ingestion in young population in age group of 16-30 years with male-female ratio as 1.03 : 1.00; having marital discord and family quarrels as prominent predisposing factors. The majority of patients had GIT symptoms (73.2%), cardiac arrhythmias (62.5%) and shock (53.3%). The commonest ECG abnormalities were tachycardia (96%), atrial fibrillation (58%) and ventricular-ectopic (VE) beats (59%). The management was supportive in the form of stomach wash, intravenous (I/V) fluids, dopamine, hydrocortisone, sodabcarb and assisted ventilation in intensive care unit (ICU) setting with mortality rate of 75%. The fatal dose was 300 to 600 mg. and fatal period as 2 to 16 hours.



## Conclusion

The mortality due to ALP poisoning is very high and variable and depends upon a number of factors, the lack of antidote and the bad prognostic signs being the most prominent. The use of  $MgSO_4$  in reducing the cardiac arrhythmias and mortality is well documented and recommended.

One most important factor which shall help to improve survival is providing preliminary medical-aid within  $\frac{1}{2}$  to 1 hour of ALP intake at grass root levels. Other preventive measures are the caging of tablets in plastic packs with holes and spikes and more stringent restrictions on its supply in open market. The applicators of ALP must be licensed or working under the supervision of a licensed person. Improved education and enforcement of safety regulations would help to improve the frequency of the illness. Thus, the problem needs to be tackled by using multi-faceted approach in form of preventive measures, updating the management modalities at all levels and overall a study and research for an antidote which will prevent further loss of human lives as a result of poisoning.

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