



## Review Article

## Phosphide poisoning: A review of literature

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## ABSTRACT

Metal phosphides in general and aluminium phosphide in particular are potent insecticides and rodenticides. These are commercially used for protection of crops during storage, as well as during transportation. However, these are highly toxic substances. Their detrimental effects may range from nausea and headache to renal failure and death. It is, therefore, pertinent to ensure their circumspect handling to avoid poisoning episodes. Its poisoning has a high mortality and recent years have seen an increase in the number of poisoning cases and deaths caused by suicidal ingestion. Yet due to their broad spectrum applications, these chemicals cannot be written off. The present communication reviews the various aspects of toxicity associated with metal phosphides.

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## 1. Introduction

Metal phosphides are highly effective insecticides and rodenticides. These are frequently used to protect grains in stores and during its transportation. Poisoning with these compounds may be direct due to ingestion of salts and indirect from accidental

inhalation of phosphine generated during their approved use. Both, metal phosphides and phosphine have corrosive actions [1]. Once ingested, the metal phosphides generate highly toxic phosphine gas by the action of dilute hydrochloric acid content of stomach. Phosphine is an insecticide and rodenticide in its own right [2].

Aluminium phosphide is used extensively as a cheap and effective grain fumigant and rodenticide in developing countries [3]. The reason being that it is highly potent against a broad spectrum of insect species, does not affect seed viability, is cost effective and leaves little residue on food grains [4]. Yet it elicits extreme toxic effects to humans for which no suitable antidote is available [5]. Its poisoning has a high mortality and the 1990s saw a

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dramatic increase in the number of poisoning cases and deaths caused by suicidal ingestion, particularly in India. In fact, it is the most common cause of poisoning in sub-urban and rural parts of Northern India [3,6,7]. Besides, poisoning cases have also been reported in France, Turkey, Germany and Iran [8–11].

## 2. Synthesis, exposure pathways and toxicokinetics

Metal phosphides are schedule 7 poisons and require a permit for usage [3]. As a rodenticide, aluminium phosphide is formulated in solid form as tablets or pellets placed in porous bags or blister packs. Aluminium phosphide may be synthesized as dark gray or dark yellow crystals. Zinc phosphide, on the other hand, is a steel gray crystalline powder which is synthesized by direct combination of zinc and phosphorus. It is a slow acting rodenticide as compared to aluminium phosphide [12]. Table 1 shows the general characteristics of some commonly available metal phosphides.

Exposure generally occurs by way of accidental or suicidal ingestion. It may also occur by consuming food products which may have been exposed to aluminium phosphide during processing. Dermal absorption of aluminium phosphide is quite rare as it is a solid material in its natural state. Nevertheless, it may be absorbed through the broken skin causing systemic toxicity. Yet another route to exposure is inhalation of phosphine which, in turn, may be generated by the action of moisture on metal phosphides [12]. Stephenson mentioned the possibility of zinc phosphide injection as a route of exposure [13].

Following the ingestion of metal phosphide, phosphine gas is generated which is rapidly absorbed throughout the gastrointestinal tract, reaches the blood stream, a part of it is carried to the liver by portal vein. It is also rapidly absorbed through lungs. After peak exposure, most of the phosphine is excreted unchanged in expired air, while the residual quantity is oxidized to phosphite and hypophosphite ions which are excreted in urine. Hydrolysis of metal phosphides on the skin could lead to the evolution of gaseous phosphine which could be absorbed by inhalation. Small amount of zinc phosphide reach liver and kidneys after ingestion and hydrolyze slowly in the tissues to phosphine and zinc salts [12]. It also gets distributed to the brain [14].

## 3. Mechanism of toxicity

Once ingested, aluminium phosphide is decomposed into highly toxic phosphine gas by the action of dilute hydrochloric acid content of the stomach. Phosphine acts as a respiratory poison. Even 20:100,000 part of phosphine in air is reported to fatal [10]. It blocks the enzyme cytochrome C oxidase as a result of which mitochondrial oxidative phosphorylation is inhibited [15–17]. It also disturbs the mitochondrial morphology, inhibits oxidative respiration by 70% and causes a severe drop in mitochondrial membrane potential [1], causing, in turn, the cells to die rapidly [16]. Mitochondrial cytochrome C oxidase inhibition may also lead to pulmonary and cardiac toxicity [18]. Phosphine is also known to inhibit protein synthesis and enzymatic activity, particularly in the mitochondria of lung and heart cells. This can lead to a blockage of

mitochondrial electron transport chain. It may also cause denaturing of various enzymes involved in cellular respiration and metabolism. Phosphine is responsible for the denaturation of oxyhaemoglobin molecule [12]. It progressively converts oxyhaemoglobin to methaemoglobin and hemi chrome species. The reaction of phosphine with oxyhaemoglobin leads to formation of phosphite and phosphate ions [19]. Phosphine thus, reduces the oxyhaemoglobin of blood [15]. Phosphine is known to induce oxidative damage in brain, lung and liver of rats [20].

Aluminium phosphide causes widespread organ damage due to cellular hypoxia by inhibition of enzyme cytochrome C oxidase of mitochondria [21,22]. Chugh, et al. [23] stated that ingestion of aluminium phosphide leads to a high superoxide dismutase activity and low catalase levels that result in formation of a high quantum of free radicals and accelerate lipid peroxidation. The latter, in turn, results in damage to cellular membrane, disruption of ionic barrier, nucleic acid damage and finally, cell death.

## 4. Clinical signs and symptoms

Cytotoxic phosphine gas produced due to acid hydrolysis of metal phosphide affects heart, lungs, kidneys and gastrointestinal tract [24]. Poisoning with metal phosphides causes nausea, restlessness, abdominal pain, palpitation, pulmonary edema, cyanosis, hypotension, shock and cardiac arrhythmias. Other rare effects include hepatitis, acute tubular necrosis, disseminated intravascular coagulation and respiratory alkalosis [1,10,25–31]. The inhalation of phosphine gas causes diarrhoea, pulmonary edema, cold and clammy sweats, tremors, convulsions, delirium, coma and death from respiratory and cardiac arrest [15]. Misra et al. [32] reported that occupational exposure to phosphine causes cough, tightness around the chest, headache, giddiness, numbness, lethargy, anorexia and epigastric pain. The abnormal physical signs included bilateral diffuse rhonchi and absence of ankle reflex.

Chugh et al. [33] reported abdominal pain, palpitation and sweating, tachypnea, cold clammy skin, metabolic acidosis, unrecordable blood pressure and shock as the most common symptoms following ingestion of zinc phosphide. Rodenberg et al. [34] advocated that after ingestion of zinc phosphide death results from pulmonary edema. Due to the absorption of trace amount of phosphide into body, delayed effects are observed on heart, liver and kidney; delayed death resulting from cardiotoxicity. Guale et al. [27] described the central nervous system intoxication, depression and vomiting as clinical signs in case of zinc phosphide poisoning.

Aluminium phosphide poisoning can result in either hypomagnesaemia or hypermagnesaemia [35]. Chugh et al. [28] reported that the most common signs and symptoms in case of aluminium phosphide poisoning are gastrointestinal haemorrhages and shock which results in congestive cardiac failure and acute respiratory arrest. Chugh et al. [36] reported that patients suffered shock after ingestion of aluminium phosphide tablets and developed adult respiratory distress syndrome within 6 h. Cardiogenic shock remains the most common cause of death in aluminium phosphide

**Table 1**  
Properties of commonly available metal phosphides.

S. no.	Metal phosphide	Source	Chemical formula	Physical form	Lethal dose (LD50)
1.	Aluminium	Fumigant insecticide, rodenticide	AlP	Yellow or dark gray crystals	20 mg/kg <sup>a</sup>
2.	Zinc	Insecticide, rodenticide	Zn <sub>3</sub> P <sub>2</sub>	Gray tetragonal crystals/gray black powder	4–5 g (toxic dose) <sup>b</sup>
3.	Magnesium	Fumigant insecticide, rodenticide	Mg <sub>3</sub> P <sub>2</sub>	Yellow-green crystals	10.4 mg/kg <sup>a</sup>
4.	Calcium	Rodenticide	Ca <sub>3</sub> P <sub>2</sub>	Red-brown crystalline powder or gray lumps	8.7 mg/kg <sup>a</sup>

<sup>a</sup> European Food Safety Authority Scientific Reports (2008). Available at <http://www.efsa.europa.eu/> (accessed on 17.06.11).

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poisoning [37]. Gupta et al. [38] have reported feeble heart sounds, tachy and bradycardia as cardiovascular manifestations in aluminium phosphide poisoning. Chugh [39] mentions that phosphine gas produces a multi organ failure in acute aluminium phosphide poisoning. The other complications in acute aluminium phosphide poisoning are hepatic failure, renal failure, cardiac failure, clotting abnormalities and pleural effusion. Pérez Navero et al. [40] reported that accidental inhalation of aluminium phosphide produces sudden vomiting, cardiac arrhythmias, shock, dyspnea, pulmonary edema, metabolic acidosis and hepatic dysfunction. Methaemoglobin formation in aluminium phosphide poisoning has been investigated by Lakshmi [25]. In cases of severe aluminium phosphide poisoning, the breath of the victim acquires a characteristic garlic-like odour [3]. Madan et al. [41] and Verma et al. [42] reported the development of oesophageal strictures after the ingestion of aluminium phosphide tablets.

Mortality with aluminium phosphide is very high. Thus, one of the vital issues in acute aluminium phosphide is predicting its outcome. APACHE II (Acute Physiology and Chronic Health Evaluation II) and the SAPS II (Simplified Acute Physiology Score II) have demonstrated an ability to predict the mortality rates in aluminium phosphide poisoning. APACHE II and SAPS II are one of the several ICU (Intensive Care Units) scoring systems designed to measure the severity of the disease in patients admitted to ICUs. Hajouji Idrissi et al. [43] evaluated the efficacy of APACHE II and SAPS II to determine the severity of AIP poisoning and found that they were positively correlated with poor outcome. Louriz et al. [44] evaluated the predictive power of APACHE II in aluminium phosphide poisoning. Shadnia et al. [45] in a recent study determined the impact of the SAPS II in the prediction of outcome in patients with acute aluminium phosphide poisoning and concluded that SAPS II calculated within the first 24 h was a good prognostic indicator. Studies have also been conducted on the role of a single clinical and/or paraclinical finding in predicting the outcome of acute aluminium phosphide poisoning [44,46–48].

## 5. Treatment

In episodes of metal phosphide poisoning, the treatment depends on route of exposure. If the victim has ingested metal phosphide, slurry of activated charcoal may be administered (1 g charcoal per kg body weight). Milk, fats or saline emetics should not be given orally. Metabolic acidosis must be treated by administering sodium bicarbonate and shock should be treated with appropriate vasopressors [12].

Goel and Aggarwal [18] and Gupta and Ahlawat [24] advocated that the victim be given a dose of saline solution along with supportive care. Activated charcoal, sorbitol suspension or sodium bicarbonate solution should be administered orally. Intravenous administration of magnesium sulfate, sodium bicarbonate and calcium gluconate is also an alternative. Specific therapy with intravenous magnesium sulfate is recommended. No known specific antidote is however, available for metal phosphide poisoning.

Shadnia et al. [49] concluded that coconut oil has a positive clinical significance and can be used as an antidote in acute aluminium phosphide poisoning in humans. In animal studies, Hsu et al. [20] held that melatonin protects against phosphine induced oxidative damage to brain, lung and liver in rats. Hsu et al. [50] indicated that glutathione plays a crucial role as a protective factor in phosphine induced oxidative damage in rats.

## 6. Autopsy findings and histopathological examination

Post-mortem examination carried out in wake of aluminium phosphide poisoning sometimes reveal a distinct garlic odour from mouth and close to the body. The face is usually livid with distinct

bluish discolouration and blood mixed froth from and around the nostrils. Internal examination reveals froth in trachea and edematous lungs with haemorrhages in the interlobular areas and at the margins of the lungs. Stomach usually contains grayish brown fluid or pasty material. Gastric mucosa becomes slugged and the stomach wall appears thinned out. Necrosis of mucosa in fundus region of stomach wall is observed. Lungs, trachea, myocardium, gastric mucosa, kidneys, liver, spleen and brain are congested. In fact, almost all the vital organs become clogged in cases of aluminium phosphide poisoning [51]. Besides congestion of organs, heart is full of dark blood. Tongue, mouth and esophagus are edematous and corroded. The mucous membrane of stomach is rendered corrugated, loosened or hardened and inflamed along with inflamed intestines [15].

The histopathological examination in studies has revealed varying degree of congestion, edema and leucocytic infiltration, suggesting cellular hypoxia in aluminium phosphide poisoning. The most dramatic effects are produced in lungs, kidneys and adrenals [52]. Animal studies on chickens revealed severe pulmonary edema and congestion of heart, liver and kidney in cases of zinc phosphide poisoning [53]. Focal myocardial infiltration with necrosis, pulmonary edema and widespread small vessel injury are reported at autopsy in a child who died of acute phosphine poisoning [54].

Abder-Rahman [55] reported the fatalities resulting from aluminium phosphide intoxication at mild exertion in asymptomatic children. The children suffered in relation to some physical activities such as running, walking and bathing, without any prior complain/symptom. Viscera showed intense congestion with moderate to severe pulmonary edema. In fatal cases, the cause of sudden death was cardiac arrest. Physical exertion may precipitate death due to increased cardiac stress, increased oxygen demand and by aggravating metabolic acidosis. The absence of clinical symptoms before death may be attributed to low level of aluminium phosphide or a possible occurrence of death in early stages after exposure to aluminium phosphide.

Aggarwal et al. [56] described the occurrence of intravascular haemolysis in patient with normal glucose-6-phosphate dehydrogenase (G-6-PD) levels in aluminium phosphide poisoning. Generally, intravascular haemolysis occurs in patients who are deficient in glucose-6-phosphate dehydrogenase (G-6-PD) enzyme. It has also been reported that inhalation of phosphine gas causes pulmonary injury and edema [57]. Saleki et al. [14] stated that sinusoidal congestion, central vein congestion, centrilobular necrosis, hepatocytes nuclear fermentation, sinusoidal clusters of polymorphonuclear leucocytes and mild macro vesicular steatosis, as well as fine cytoplasmic vacuolization occur in cases of phosphine poisoning.

Tripathi and Pandey [58] reported distinct changes in the cerebral and cerebellar cortex due to the effect of aluminium phosphide on human brain. Cerebral cortex showed disorganization of different layers, round shaped neurons with convex border and deeply stained degenerated eccentric nucleus. Cerebellar cortex revealed degenerated neurons, infiltration of round cells into the molecular layer. Degenerate nucleus was surrounded by scavenger cells in the granular layer. The subcortical zone of brain showed a paucity of glial cells, degeneration of nerve fibers and the appearance of necrotic patches. Mehrpour et al. [59] observed mild capillary dilation and congestion of cortex, cerebral edema, and cerebellar edema. They also observed degenerated Nissel granule in the cytoplasm and deeply stained degenerated eccentric nucleus in brain cortex and degenerated neuron and infiltration of round cell in to the molecular layer in cerebella. Sinha et al. [60] reported that almost all the vital organs were found to be congested in cases of aluminium phosphide poisoning. On histopathological examination, liver showed central venous congestion, degeneration,

sinusoidal dilation, haemorrhage and fatty changes. Lungs showed alveolar thickening, edema, dilated capillaries and haemorrhage. Kidney showed infiltration, degeneration and cloudy swelling. Stomach showed congestion and haemorrhage. Coagulative necrosis and congestion was observed in brain.

## 7. Analysis

A range of chemical tests and analytical methods may be used for the analysis of sample/s in case of phosphide poisoning. Diagnosis of phosphide poisoning can be confirmed by detecting the phosphine gas in sample/s. Chugh et al. [61] described the use of silver nitrate impregnated paper test on the gastric fluid of patients in cases of aluminium phosphide poisoning. It is a simple, reliable and sensitive method to detect phosphine. A combination of ammonium molybdate reagent and commercially available detector tubes can be used as qualitative and quantitative procedures for stomach contents and non-biological materials [27]. Ammonium molybdate test has been recommended for the detection of phosphorus and phosphides in the stomach contents and non-biological materials in the event of poisoning cases [62]. For this test to be conducted; a small amount of minced tissues (10 g) or other biological material suspected to contain phosphide is taken into a steam distillation flask, mixed with an equal amount of water and then acidified with dilute sulfuric acid, followed by steam distillation. The distillate is collected in an ice cold receiver containing 5 ml of 1% silver nitrate solution by dipping the adapter into the silver nitrate solution. In the presence of the phosphide, the silver nitrate solution turns black. 5 ml of concentrated nitric acid is added to this black precipitated material and boiled till the solution becomes practically colorless. Then 5 ml of ammonium molybdate solution is added and heated for 1 min. Appearance of canary yellow precipitates confirms the presence of phosphide [63]. Kashi and Muthu [64] developed a rapid, sensitive and reliable mixed indicator paper strip impregnated with dimethyl yellow (0.05%), cresol red (0.1%) and mercuric chloride (1%) in methanol for detection of phosphine. The appearance of red color on strip indicates the presence of phosphine. It is a highly sensitive method and the reagent has a better shelf life than indicator strip impregnated with dimethyl yellow plus mercuric chloride or cresol red plus mercuric chloride.

Chan et al. [65] reported an autopsy case of ingestion of aluminium phosphide tablets. Blood and viscera were preserved during autopsy and were mixed with 10% H<sub>2</sub>SO<sub>4</sub> for 30 s in head space vial. Porapak Q column with 100–200 mesh (1.4 m × 4 mm) was used in Head Space Gas Chromatograph. Nitrogen gas was used as carrier gas at flow rate of 30 ml/min. The column was operated at 80 °C with Nitrogen–Phosphorus Detector and phosphine was detected in specimens. In another case reported by Musshoff et al. [66], a 25 year old man was found dead in his house. The intense odour of rodenticides was observed in the room in which the man was found dead. A package of rodenticide

containing aluminium phosphide as main ingredient was found together with other medicine pills and pill residues. The autopsy was performed after 3 days. Various body fluids and tissues were collected at the autopsy and stored at –20 °C for further analysis. 1 g of sample material was mixed with 10 ml of 0.1 N sulfuric acid in an air tight headspace vial and used for gas chromatographic analysis. Phosphine gas was detected in stomach contents, blood and liver specimen of victim by employing Head Space Gas Chromatography (HS-GC) technique with Nitrogen–Phosphorus detector. HP-Plot Q GC column (30 m × 0.32 mm i.d.; film thickness 20 µm) was used. Hydrogen was used as a carrier gas. The temperature of injector and detector were 150 °C and 310 °C respectively. Gas Chromatography (GC) is one of the most widely used techniques for the analysis of sample/s. The technique is reliable, sensitive, highly selective and destructive in nature. In HS-GC, head space is used to isolate analyte of interest from sample matrix. The volatility of analyte should be higher than that of solvent used for extraction. Corley et al. [67] described the method for analysis of zinc phosphide in various agricultural food commodities, as well as for stomach contents of cattle. The method involved the production of phosphine gas which was then analyzed by Gas Chromatography (GC) technique using nitrogen – phosphorus or flame photometric detector. The detection limit was 5 ng of zinc phosphide per gram of sample. Gas chromatography (GC) is a dynamic method of separation and detection of volatile organic compounds and several inorganic permanent gases in a mixture. It is very sensitive and specific technique for the analysis of gases. The sensitivity of GC ranges from ng/l to g/l levels. Tiwary et al. [53] developed a sensitive and highly specific Gas Chromatography–Mass Spectrometry (GC–MS) method for analysis of gastrointestinal samples in animal studies on chickens. It was found that determination of zinc concentrations in the liver or kidney tissue or stomach contents was not a reliable indicator of zinc phosphide poisoning. GC–MS is most useful technique for routine analysis of postmortem material/s in forensic science laboratories. In GC–MS, GC separates the components of a sample and MS identifies it and it also acts as a detector. Although GC–MS is destructive method, yet it is most frequently used for analysis of sample/s due to its high specificity and sensitivity. General analytical procedure for determination of phosphine is described in Table 2. Tang et al. [68] described a reliable method for the determination of aluminium in bone by Electro Thermal Atomic Absorption Spectrometry (ETAAS). Accurate and precise results were obtained. The limit of detection in bone was found to be 0.023 µg/g. With some minor modifications, same method was used for determination of aluminium in serum and dialysis fluid. The Neutron Activation Analysis technique may be used to detect and quantify the concentration of zinc in viscera in cases of zinc phosphide poisoning [69]. Mantler and Wernisch [70] described the method for determining qualitative and quantitative composition of aluminium phosphide by employing X-ray electron spectrometry, X-ray fluorescence analysis and X-ray

**Table 2**  
General analytical procedures for determination of phosphine.

S. no.	Analytical technique	Pretreatment procedure	Column	Detector	Detection limit	Reference
1.	GC	1 g sample + 3 ml of 10% H <sub>2</sub> SO <sub>4</sub> + 5 ml toluene	30 m × 0.53 mm DB-17	FPD	0.10 µg/g	Tiwary [53]
2.	GC–MS	1 g sample + 5 ml of 10% H <sub>2</sub> SO <sub>4</sub>	30 m × 0.32 mm GS-Gaspro	MS	1 µg/g	Tiwary [53]
3.	GC	10 g sample + 20 ml of 5 N H <sub>2</sub> SO <sub>4</sub> + 10 ml toluene or 1 g of grass and spinach + 30 ml of 1 N H <sub>2</sub> SO <sub>4</sub> + 10 ml toluene	30 m × 0.53 mm DB-5	NPD/FPD	0.005 µg/g	Corley [67]
4.	GC	Aluminium Phosphide + water + 1 ml of nitrogen	2 m × 4 mm id Packed with Porapak Q (60–80 mesh)	N-P alkali Flame Ionisation	0.01 ng/ml	Chughtai [71]
5.	HS-GC	1 g sample + 10 ml of 0.1 N H <sub>2</sub> SO <sub>4</sub>	30 m × 0.32 mm HP-Plot Q GC Column	NPD	2 µg/Kg	Musshoff [66]



diffraction, as well as gravimetric and gas volumetric methods. In addition to aluminium phosphide, aluminium hydroxide was found which was produced by reaction of aluminium phosphide with water. Chughtai et al. [71] developed a method for the determination of phosphine in gas samples by packed column gas chromatography with alkali flame ionization detection. The limit of detection was 0.01 ng/ml. Response was linear over the ranges 0.1–1.0 and 2.5–25 ng/ml. Reproducibility at 1 ng mass of phosphine on column was 6.3%. No interference was found for hydrogen sulfide, ammonia or methane. Hydrogen sulfide produced a negative response from this detector and ammonia produced no response. Water eluted at the same retention time as hydrogen sulfide but produced a positive response and hence did not interfere with the elution of phosphine. Azoury and Levin [72] identified zinc phosphide in falsely labeled rodenticide bait using microscopic examination, scanning electron microscope-energy dispersive X-ray spectroscopy (SEM-EDX) and XRD.

Anger et al. [8] reported a case in which a 39 year old man ingested aluminium phosphide (available at victim's workplace) and committed suicide. The victim was discovered after 10 days of ingestion of aluminium phosphide. An autopsy was carried out as per the order of judicial authority. Blood, urine, liver, kidney, adrenal and heart samples were analyzed. Phosphine gas was not detectable in blood and urine. The concentration of phosphine in brain, liver and kidneys were 94 ml/g, 24 ml/g and 41 ml/g respectively. The phosphorus was measured by Inductive Coupled Plasma – Mass Spectroscopy (ICP-MS) that confirmed a diagnosis of poisoning by aluminium phosphide.

Garry et al. [73] and Anger et al. [8] described the method for the measurement of aluminium in blood by employing electro thermal atomic absorption spectrometry (ETAAS) or Inductive Coupled Plasma – Mass Spectroscopy (ICP-MS). Chugh et al. [74] correlates the blood phosphine levels to clinical grades of toxicity and to dose of active pesticide consumed. Higher the blood phosphine level, higher was mortality. The limit of phosphine toxicity was  $1.067 \pm 0.16$  mg%.

## 8. Conclusions

Metal phosphides are toxic to rodents and are used to protect the grain in stores and fields. Aluminium phosphide is a frequently used grain fumigant because of its highly potent characteristic, cost effectiveness and easy availability. Once administered in the body, a metal phosphide gets decomposed by dilute hydrochloric acid in stomach, and liberates highly toxic phosphine gas; the latter acts as respiratory and mitochondrial poison. Almost all the vital organs are affected in cases of aluminium phosphide poisoning. Silver nitrate impregnated paper test and ammonium molybdate test can be used for the detection of phosphide in biological and non-biological materials. Head Space-Gas Chromatography method with either nitrogen–phosphorous detector or mass selective detector confirms the presence of phosphine gas in biological and non-biological materials. Despite the extreme toxicity of metal phosphides no effective antidotes are available for treating victims of poisoning episodes.

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