

Aluminium Phosphide (Slow) Poisoning: A Narrative Case Review

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Abstract : Aluminum phosphide poisoning has been difficult to manage since there is a lack of specific antidote to treat it. The current case review provides an insight on various methods that has been handled by physician to manage the poison, and this review also provides the clinical manifestations that play a significant role in deciding the appropriate treatment. A retrospective study of the scientific literature reporting on cases of aluminum phosphide ingestion was carried out, using the inclusion criteria: 1) patient of all age groups 2) patient with an accidental exposure, 3) patient with an intention of self-harming, 4) successful and unsuccessful management of the aluminum phosphide poisoning. A total of 14 case reports were collected, from which 7 cases has been reviewed, of which four cases were due to the intention of self-harming and the other three cases were due to accidental ingestion of aluminum phosphide in various forms (tablets, granules, powders). The important manifestations that were seen in all these cases were 1) changes in ECG mostly resembling ST elevation or depression, 2) metabolic acidosis and 3) severe changes in hematocrit values affecting almost all the hemodynamic parameters. Management of aluminum phosphide poisoning has its major setback due to the unavailability of specific antidote, successfully managed cases shows that early diagnosis and detection of important clinical manifestations provides an advantage, other factors like appropriate history collection from patient relative is significant in providing an accurate treatment in a timely manner.

Key words: Aluminum phosphide poisoning, Phosphine, Cardiogenic shock, Hypotension

1. INTRODUCTION

Pesticide intoxication is a leading cause of mortality and morbidity worldwide¹, around 200,000 individuals has been suffering from pesticide intoxication globally, which was estimated by The World Health Organization (WHO), Geneva. Changes in lifestyles as well as modifications in cultural frameworks are the main factors for the enhanced incidence of poisoning cases². In addition, simple accessibility, low-cost and unrestricted sales of pesticides are the other main reasons^{3, 4}. The raised occurrence of pesticide poisoning is generally connected with population socio-demographic patterns and restricted pesticide regulation^{5, 6}. Aluminium phosphide (ALP) is a pesticide used extensively as a grain preservative for solid fumigant. Many trade names such as Quickphos, Celphos, Alphos, Fumigant and Phostoxin are accessible as greenish gray tablets on the market (packed in metal pipes, weighing 3 gms each capable of releasing 1 gm of phosphine gas. In 1973, aluminum phosphide was proclaimed as an optimal fumigant pesticide and is now one of the most frequently used suicidal poison in northern and central agricultural nations. The fatality in this poisoning is very big that in some areas of our nation this is epitomized as an agent of certain death⁷.

MATERIALS AND METHODS

Data Collection:

The key words aluminium phosphide poisoning/phosphine/case report was searched in PubMed google scholar and Scopus. Articles published between 1997 and 2019 were selected for this study. A total of 14 cases were collected from various demographics and 8 cases were reviewed among these with a motive of covering at least one case from 6 different countries. Out of 8 cases, four were due to the intention of self-harming and the other four due to accidental ingestion of aluminum phosphide in various forms (tablets, granules, powders). The published case reports originates from 6 different countries (Table 1). Most of the case reports were from India (n=4) and Iran (n=5) followed by Malaysia (n=1), Turkey (n=1), Srilanka (n=2) and USA (n=1).The case report includes patients of all age groups. The selected articles met the following requirements: (1) the articles contain a case report (2) Describes an intoxication with aluminum phosphide (3) accidental and self-harming ingestion (4) Management of aluminum phosphide poisoning Data Analysis: All case reports in this study was read and interpreted by at least two authors of the current paper.

The Chemistry

Aluminum phosphide is rapidly absorbed through inhalation, ingestion and through mucosal contacts; the active element of aluminum phosphide is phosphine gas (PH₃), which has a high potency for ignition when exposed to humidity. Thus, aluminum carbonate is added to aluminum phosphide in order to prevent the ignition⁸. Each 3g of aluminum phosphide tablet produces precisely 1g of phosphine gas on humidity exposure and leaves behind an aluminum hydroxide nontoxic grey residue⁸. In relation to these chemical properties, phosphine may ignite spontaneously at levels above a limit in the presence of oxygen at high concentrations^{9, 10}.

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Mechanism of Toxicity

Aluminum phosphide releases phosphine that can be rapidly absorbed through mucosal and inhalation routes, when exposed to atmospheric moisture. Aluminum phosphide releases phosphine gas that is absorbed into the circulation after ingestion. It is a protoplasmic poison that inhibits the synthesis of different enzymes and proteins. It is a powerful enzyme inhibitor in the respiratory chain with its greatest impact on cytochrome c oxidase. Resulting in the production of free radicals (superoxide, peroxidases) causing cell injury¹¹. Hypoxic damage is seen when there is an increased activity of superoxide dismutase and reduced levels of catalase. It impairs mitochondrial metabolism and inhibits the respiratory chain's electron transport system, stimulates superoxide radicals generation, and results in protein denaturation and lipid per-oxidation⁷, rapid absorption and metabolism of aluminum phosphide can be seen in liver which is subsequently proceeded by, slow release of phosphine can occur in the body, leading to delayed toxicity characteristics¹¹.

Epidemiology

A research was conducted to explore the epidemiology on health endpoints among Indian workers involved in stored grain fumigation. The average work duration of these employees was 11.1 years, and phosphine levels in the workforce during the inquiry area ranged from 0.17 to 2.11 components per million (ppm). The commonly reported signs included dyspnea, headache and chest tightness instantly after finishing fumigation operations. These employees physical examinations were unnoticeable and no important defects were noted in the motor and sensory nerve conductions¹². In connection with epidemiological accounts of random morbidity and mortality, phosphide fumigants are frequently involved outside the workplace. Out of a total of 13,100 visits, 301 presumed toxicity instances (2.3 percent) were reported in a one-year research span in Allahabad (India). In this area, the incidence of ALP toxicity was 11.7 times and the proportion between male and woman was 2:1. Study of age allocation showed maximum incidence was at the age between 11 to 30 years of which most of them belonged to the rural region, while others belonged to the urban region¹². While another study claimed that on exposure to Phosphine gas causes adverse neurobehavioral developmental and neurological effects among children¹³.

Diagnosis

Diagnosis can be made easily by simple silver nitrate-impregnated paper test on gastric content or on breath¹⁴. Laboratory evaluation is conducted primarily to achieve the prognosis. Serious toxicity to ALP leads to Leucopenia. Mild overdose of ALP causes increased SGOT (Serum glutamic oxaloacetic transaminase) and SGPT (Serum glutamic transaminase) levels along with metabolic acidosis. Decrease in magnesium level can be seen, renin plasma elevated concentration is important because its concentration is directly related to mortality and is proportional to ALP dose. In serious ALP toxicity, the serum concentration of cortisol is generally reduced¹⁵. Development of hypotension and the frequency of vomiting correlate with the outcome of intoxication after ingestion.

Development of metabolic acidosis, refractory shock, Acute respiratory distress syndrome (ARDS), anaemia, gastrointestinal bleeding and electrolyte imbalance, are observed following after ALP ingestion¹⁶.

Clinical Manifestations

Hemodynamic and cardiac effects:

ALP induces significant abnormalities like tachycardia, hypotension, electrocardiogram (ECG) abnormalities and bradycardia^{17,18}. ST elevation or depression along with PR and QRS interval prolongation can be observed in ECG, reversible myocardial injury along with fibrillation are also commonly seen in ALP poisoning¹⁹. Significant reduction in hematocrit, platelet count, red blood cell count, and hemoglobin along with severe metabolic acidosis were shown in animal studies including rodents and mice which were exposed to phostoxin^{20,21}.

Neurotoxic Effects:

Reduction in cytochrome in brain except for cytochrome b was seen along with neuronal lipoperoxidant damage and concurrent modifications in antioxidant production with severe impacts on nervous system structure and function was reported with exposure to ALP^{22,23}. Ataxia and tremors along with convulsions are commonly seen. Along with acute hypoxic encephalopathy, paralysis of respiratory center and shutting of central nervous system in brain may lead to death of the patient²⁴.

Hepatotoxic Effects:

There was a significant reduction in peroxidation of unsaturated fatty acids, along with reduction in the catalytic activity of effective oxidase molecules in cattle treated with ALP²⁵. Inhalation of phosphine induced a substantial reduction in GSH level as well as an improvement of 19–25 percent in lipid peroxidation and an increase of 39 percent in mitochondrial DNA of 8-hydroxydeoxy guanosine²⁶. Significant biologically appropriate rises were noted at 10-PPM, ALP exposure in the serum activity of alanine aminotransferase (ALT) and sorbitol dehydrogenase²⁷. ALP has been shown to induce a cellular energy deficit that results in impaired liver and brain energy status combined with important modifications in homeostasis of insulin²⁵.

Gastrointestinal Effects

Hematemesis, vomiting, and epigastric pain are the common gastrointestinal signs of ALP poisoning. Endoscopy shows esophagus and stomach with corrosive lesions, serious gastric erosions, duodenal erosions, and fistula or esophageal rigidities, while signs of excessive thirst, abdominal pain, epigastric tenderness are seen. Arrhythmias, hypotension, myocarditis, heart failure, dry pericarditis are common cardiovascular abnormalities²⁸.

CASE REPORTS AND MANAGEMENT

Accidental Ingestion:

A man of 45 years was admitted to the hospital due to the accidental ingestion of ALP, he was presenting with symptoms like nausea and severe diarrhea for 3 days, the patient condition deteriorated with signs of severe dyspnea even after the administration of intravenous saline. The

patient was transferred to ICU, as he showed compromised Glasgow Coma Scale (GCS) scale with a score of 6/15. He presented with symptoms of severe metabolic acidosis and liver dysfunction, which is a classical symptom of aluminum phosphide ingestion. He was offered support for respiration, along with venous hemofiltration; IV bicarbonate infusion was given as inotropic agent at a dose of (5meq/kg). Patient did not respond to the treatment and eventually passed away²⁹. A girl of 3 years developed cardiogenic shock, along with respiratory failure, after accidental ingestion of aluminum phosphide, she was also presenting with mild liver and kidney injury along with unfrequented episodes of ventricular arrhythmia. ECMO (extracorporeal membrane oxygenation) was given for 16 days veno-arterially, for support. Ventricular arrhythmia was treated with IV Lidocaine at a dose of 50mg and IV Magnesium sulfate (2g) along with hemodialysis for 3 weeks for the management of metabolic acidosis along with acute kidney injury. Despite presenting with all these clinical manifestation, she had complete normalization of her end-organ dysfunction. The case demonstrates the significance of early suspicion required for prevention of severe multi organ toxicity during the management of ALP poisoning³⁰. A woman of 22-year- was presenting with complaints of nausea, multiple episodes of vomiting and epigastric pain after her food intake, the patient was accidentally exposed to ALP. The patients past medical history showed celiac disease. The patient was hemodynamically stable on examination without any abdominal tenderness. Patient condition deteriorated with time as she was presenting severe hypotension and tachycardia. Severe metabolic acidosis was seen during venous blood gas analysis (VBG), while inferno lateral ST elevation myocardial infarction (STEMI) as seen in ECG. IV Bicarbonate at a dose of (5meq/kg) was initiated. Cardiopulmonary resuscitation (CPR) was initiated as Rhythm became ventricular fibrillation (VF) after 8 hours of admission. Peritoneal dialysis was performed. On the next day patient had repetitive episodes of ventricular fibrillation, patient failed to respond to CPR which was initiated. She was declared dead, the case reveals that determining the dose of ingestion is, as important as early diagnosis along with supportive treatment³¹. A girl of 12-year- was presenting with complaints of acute respiratory distress syndrome, persistent cough, cyanosis, multiple episodes of vomiting and irritability, the patient was accidentally exposed to ALP. The patient was hemodynamically stable on examination. Patient condition deteriorated with time as she was presenting with pleural effusion, pulmonary edema, severe hypotension and tachycardia. IV Dobutamine (20mcg/Kg/min) and IV Gentamicin (1.5mg/kg) was initiated as (BP 60/40 mm Hg) was critically low. Cardiopulmonary resuscitation (CPR) was initiated along with two boluses of Ringer lactate (20ml/kg), chest X ray depicted pulmonary edema, thus IV Hydrocortisone (200mg) and ceftriaxone (0.1 µg/kg/min) was added to manage acute respiratory distress syndrome. The patient condition substantially improved and was discharged. The case reveals the importance of considering pleural effusion as an important complication when presented with symptoms of persistent cough and cyanosis accurate diagnosis for successful management of Aluminum phosphide poisoning³⁷.

Self -Harming (Suicidal):

A woman of 26 years had allegedly ingested two tablets of 3g aluminum phosphide, of which each tablet, releases about 1g of phosphine gas following ingestion. Patient was hemodynamically unstable (Pulse rate 146 bpm; BP 60/40 mm Hg), resuscitation was initiated with intravenous fluids. She became unresponsive and pulseless. Cardiopulmonary resuscitation was initiated as the patient turned unresponsive and pulseless, while she was resending with Ventricular Tachycardia. Intravenous (IV) Amiodarone at a dose of 300mg was given along with defibrillation. Drugs like IV Magnesium sulphate (1g), Calcium gluconate (4g) and Sodium bicarbonate (3 mg) were also administered in response to severe metabolic acidosis. The patient had multiple episodes of seizures after resuscitation, which was managed with IV Midazolam at a dose of (0.15mg/kg bolus). Patient was presenting with persistent hypotension thus he was managed by Noradrenaline (1mg/ml), Adrenaline (1mg/ml), and Dobutamine (5mg/ml). Resuscitation was terminated as patient suffered a cardiac arrest for the second time. Post mortem showed edematous gut lining while ALP was still present as a gastric content. Pleural effusion was found during lung autopsy²⁸. A man of 19 years was admitted to hospital with alleged intake of 3g of ALP; Glasgow consciousness scale (GCS) of 8 was reported. (BP 80/60 mm Hg, pulse rate 116/min) Investigations revealed hemoglobin (Hb) 15.1 g/dl and hematocrit 58.3. Severe metabolic acidosis with a pH of 6.9 was shown in arterial blood gas analysis, while ECG was normal except for sinus tachycardia, patient had hypoglycemic event which as intervene with 50% IV dextrose infusion. Decontamination was initiated for gastric lavage with oral ingestion sodium bicarbonate at a dose of (3 mg), activated charcoal was administered orally with a dose of (1 g/kg,) along with permanganate potassium with a ratio of (1:10,000), and IV Magnesium sulfate 4 g and calcium gluconate of 4g was given as infusion along with plenty of hydrating fluids. The patient failed to respond to supportive treatment and died 2 hours later due to refractory hypotension leading to cardiac arrest³². A 35-year-old man was admitted with complaints of vomiting, excessive sweating, excruciating colicky pain epigastrium, dyspnea, and suffocation. 10gms of celphos powder was consumed by the patient in order to attempt self-harm. Patient presented with peripheral cyanosis, and cold sweaty skin (BP 126/70 mm Hg; R/R 20/min). Colonic tenderness was seen during abdominal examination without any abdominal distention, along with rigidity and hepato-splenomegaly. Cardio vascular system examination implied tachycardia. While neurological and respiratory systems were normal. Blood was withdrawn for investigations followed by resuscitation. He was managed with saline lavage, followed by IV Fluids, drugs like broad spectrum antibiotics along with Inj. Magnesium sulphate of 1 gm, and Inj. Hydrocortisone of 200mg were given, as the patient was presenting with typical symptoms of aluminum phosphide ingestion³³. A women of 55 year old had allegedly consumed ALP granules due to family issues, she was presenting with repeated episodes of vomiting and decreased level of consciousness (BP 140 / 70 mm Hg; PR 63; SPO₂ 95%). She was on Tab. Captopril 25mg BD for management of hypertension, ECG findings showed ventricular ectopic with atrial fibrillation. Patient's alertness

level was getting compromised with passage of time. Decontamination process was intimated with 2 L of Potassium permanganate of 0.1 percent solution for gastric lavage, accompanied by 50 gm in 400 ml of water-activated charcoal irrespective of exposure to boost its potential savings. Considering the ECG, IV Magnesium sulfate was initiated, patient responded with increased level of consciousness and orientation, whereas the atrial fibrillation did not subside, IV Magnesium sulfate at a dose of 6gm/day was maintained until atrial fibrillation was subsided after five days of admission³⁴.

CONCLUSION

This review provides an overview of the management strategy of aluminum poisoning followed in various countries. Aluminum phosphide has been in use in India for centuries as a strong fumigant pesticide. It is inexpensive, User-friendly, efficient and readily available in the market, it is being extensively used as the commonest means of self-harming in North India³⁵. Due to the lack of specific antidote, ingestion these pills, is more likely to result in fatality. The primary guiding principle for management includes aggressive lavage with potassium permanganate (1:10,000), and effective management of hypotension and shock. The favorable outcome of managing aluminum phosphide poisoning correlates best with the appropriate removal of poison from the body along with an effective supportive treatment. The rapid development to life-threatening diseases, inadequate therapy and constrained information on the effectiveness of therapeutic measures present difficulties for clinicians to combat their intoxication.

TABLE 1: CASE REPORTS FROM VARIOUS DEMOGRAPHIES AND ITS MANAGEMENT

DEMOGRAPHY	CASE REPORT TITLE	DETAILS	CLINICAL MANIFESTATION	MANAGEMENT
MALAYSIA (28)	A case report of aluminium phosphide poisoning	Age/sex=26/Female Self-harming vomiting and multiple episodes stools 10 times, drowsy, pale, cold and clammy	Ventricular Tachycardia, metabolic acidosis, Bilateral pleural effusion.	Mechanical ventilation, Gastric lavage was done with potassium permanganate(1:10,000) and sodium bicarbonate,(44meq)
TURKEY (29)	Fatal aluminium phosphide poisoning	Age/sex=45/Male Self-harming Severe diarrhoea, nausea and vomiting for 3 days	Dyspnoea, Increased respiratory rate, Bilateral alveolar-interstitial infiltrate, Acute respiratory distress syndrome	Respiratory support, veno-venous hemofiltration, inotropic support and bicarbonate infusion were immediately started, but The patient eventually died 10 h later.
USA (30)	Aluminium phosphide poisoning: Successful recovery of multi organ failure in a paediatric patient	Age/sex=3/Female Accidental ingestion Non-bilious emesis	Respiratory failure, Ventricular arrhythmia, Metabolic acidosis	ECMO (extracorporeal membrane oxygenation), IV Lidocaine at a dose of 50mg and IV Magnesium sulphate (2g)
IRAN (31)	Severe Hypoglycaemia Following Acute Aluminium Phosphide (Rice Tablet) Poisoning	Age/sex=19/Male Self-harming Cold and Clammy extremities	Sinus tachycardia, Hypoglycaemia, Refractory hypotension, mononuclear infiltration, Haemorrhagic necrosis	Two boluses of 50% dextrose, gastric Decontamination with sodium bicarbonate (44 mEq, orally), permanganate potassium (1:10,000), and activated charcoal (1 g/kg, orally), Magnesium sulphate 4 g by IV infusion, Calcium gluconate 4 g by IV infusion
IRAN (32)	An interesting case of aluminium phosphide poisoning	Age/sex=22/Female Self-harming Nausea, vomiting and epigastric pain	Hypotension, Tachycardia, Severe metabolic acidosis. Infero lateral ST elevation	Bicarbonate(44meq) and dopamine(50 mcg/kg/min IV) was initiated, cardiopulmonary resuscitation, Peritoneal dialysis.
SRI LANKA (33)	Acute Severe Suicidal Poisoning by Celphos Powder	Age/sex=35/Male Self-harming vomiting, excessive sweating, excruciating colicky pain epigastrium, dyspnoea, and suffocation,	No abdomen rigidity Hepato-splenomegaly, Tachycardia, SGOT & SGPT 72.2 and 70 IU/ Lt.	Saline lavage, IV Fluids, inj.Magnesium sulphate(4g), inj. Hydrocortisone (200mg)
SRILANKA (34)	Acute severe suicidal poisoning by herbicide pendimethalin	Age/sex=unrevealed Multiple episodes of vomiting(5 times) and decreased level of consciousness (BP 140 / 70 mm Hg, PR 63, SPO2 95%).	Hypertension, ECG findings showed ventricular ectopic with atrial fibrillation	Decontamination 2L of Potassium permanganate of 0.1 percent solution for gastric lavage 50 gm in 400 ml of water-activated charcoal IV Magnesium sulphate at a dose of 6gm/day
INDIA (36)	Pleural effusion in aluminium phosphide poisoning	Age/sex=30/male Self-harming pungent odour vomiting,	Hypotension Respiratory distress Delayed development of pleural effusion	Resuscitation with intravenous fluids, inotropes like Dopamine(50 mcg/kg/min IV) and norepinephrine(0.05 -0.1 µg/kg/min) Hydrocortisone(200mg)IV, Pantoprazole, Sodium

		abdomen pain Consciousness (BP 130 / 70 mm Hg, PR 70, SPO2 96%).		Bicarbonate(44meq), Calcium gluconate(4g), Magnesium sulphate(4g), and Prophylactic antibiotics. Oxygen supplementation
INDIA (37)	PLEURAL EFFUSION aluminium Phosphide Poisoning	Age/sex=12/Female Accidental ingestion Vomiting, irritable, restless, cyanosed with cool extremities	Acute respiratory distress syndrome Pleural effusion, pulmonary edema shock, tachycardia Gastrointestinal upset	Dobutamine(20 mcg/kg/min) Gentamicin(1.5 mg/kg) Hydrocortisone(200mg)I.V and ceftriaxone(0.1 µg/kg/min) Two boluses of Ringer lactate (20 ml/kg each)
INDIA (38)	The Successful Treatment Of aluminium Phosphide Poisoning With Limited Resources	Age/sex=unrevealed Self-harming Absent peripheral pulses Cold clammy Extremities.	Epigastric pain Restlessness, Tachypnea and Altered sensorium Atrial fibrillation	Oxygenation, Inj. Hydrocortisone 200mg I.V. stat, Inj. magnesium sulphate 1g I.V. stat and 0.5g I.M. IV fluids Ringers lactate. Dopamine infusion 10 mcg/kg/min. Potassium permanganate(1:10000) lavage and enema, Inj. Dexamethasone 8 mg
INDIA (39)	(1) Successful management of aluminium phosphide poisoning using intravenous lipid emulsion: Report of two cases	Age/sex=40/Female Self-harming conscious, irritable with low pulse rate	Abdominal tenderness	1 L of IV Ringer's lactate, Gastric lavage, intralipid emulsion 20% at 10 ml/h, IV magnesium sulphate (MgSO4)1 g over 20 min
	(2)	Age/sex=30/Female Self-harming Cold and clammy skin	Tachycardia, Dilated pupil	1 L of IV Ringer's lactate, Gastric lavage, , intralipid emulsion 20% at 10 ml/h, Infusion of Magnesium sulphate at 1 g/h
IRAN (40).	aluminium Phosphide Poisoning: A Case Report	Age/sex=16/male Self-harming vomiting and nausea	Severe hypotension Tachycardia Reduced oxygen saturation (65%) Leucocytosis	Mechanical ventilation, Gastric lavage was done with potassium permanganate and bicarbonate(1:10000), High dose inotrope drugs (norepinephrine0.05 -0.1 µg/kg/min and dopamine(50 mcg/kg/min IV)), bicarbonate, calcium gluconate(4g),magnesium sulphate(4g),hydrocortisone(200mg)
IRAN (41)	aluminium Phosphide Poisoning and Ignition in a Forensic Case	Age/sex=35/Female Self-harming Burnt face and neck due to phosphine gas	Erosions of the Stomach mucosa and a garlic odour.	Patient was dead after 6 hours of ingestion.

IRAN (42)	Hot charcoal vomitus in aluminium phosphide poisoning - A case report	Age/sex=34/Female Self-harming Drowsy, cold and hypotonic, and her skin was pale with mottling	Sensorium decreased, Apnoea, sinus tachycardia, pulse oximetry showed O ₂ saturation of 91% metabolic acidosis with PH of 7.1	Gastric washing with Sodium bicarbonate(44meq), gastric lavage was performed using Potassium permanganate (1:10,000) and then activated charcoal (100 g) was administered, IV infusion of Calcium gluconate and Magnesium sulphate
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