Review Article

A Review on Complications and Management of Aluminium Phosphide (Rice Pill) and Zinc Phosphide Poisoning

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Received: Feb 18, 2024; Accepted: 10 August, 2024

DOI: 10.22037/nbm.v12i4.44690

Abstract

Background: Various fumigant products containing aluminium phosphide, known as rice pills, are widely used to kill insects in many parts of Iran, including the northern provinces. Poisoning with zinc phosphide as a rat poison also has the same side effects as aluminium phosphide. This poisoning has no specific antidote, and the treatment is auxiliary, and one of the effective auxiliary treatments is L-carnitine. This review study aimed to determine the prevalence of aluminium phosphide poisoning and treatment with intravenous L-carnitine.

Materials and Methods: This study was a narrative review conducted in 2023. Search keywords aluminium phosphide, zinc phosphide, intravenous L-carnitine, and poisoning in Persian and English languages in databases including Magiran, PubMed, Wiley, Science Direct, web of Sciences, SID, Scopus, and Google Scholar were done from 1970 to 2022. Relevant articles were identified, and the most important and valuable points were presented after review.

Results: According to the findings of most studies, aluminium phosphide pills had the most adverse effects as toxic substances.

Conclusion: Due to the prevalence of its consumption among young people with low education levels, there was a need for physicians to treat these patients as quickly as possible accurately. It is also necessary to raise awareness about its potentially dangerous side effects and to monitor closely the distribution of this poisonous pill in its distribution centers. Moreover, the quick start of supportive treatment with antioxidants and L-carnitine can help in this poisoning that does not have a specific antidote. Very effective and useful.

Keywords: Aluminium phosphide, Zinc phosphide, L-Carnitine, Poisoning

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Please cite this article as: K Saravani Kh, Alizadeh Ghamsari A, Dadpour B, Ghassemi Toussi A, Mousavi SR, Pouramiri F. A Review on Complications and Management of Aluminium Phosphide (Rice Pill) and Zinc Phosphide Poisoning. Novel Biomed. 2024;12(4):176-84.

Introduction

One of the hazardous pesticide agents is aluminium phosphide, the rice pill in Iran[1,2]. This compound was proposed for the first time in 1973 as an ideal pesticide. Currently, it is used in dark grey colour or in the form of yellow crystals with different brands as a pesticide,

insecticide, and rodenticide[3,4].

This substance has no skin absorption in humans, and the main ways of poisoning are oral consumption and respiratory exposure. Also, the main mechanism of poisoning is the production of phosphine gas, which is produced due to the contact of aluminium phosphide with water[2]. Aluminium phosphide has a high poisoning intensity in both digestive and respiratory routes, and the phosphine gas is easily absorbed from the digestive system and lung epithelium[3].

The mechanisms of the effects of phosphine gas on cellular function are as follows: cytochrome oxidase, electron transfer chain, and cellular respiration inhibition. Free radicals are the main products of these inhibitions. These radicals damage cells.

The phosphine gas affects the cardiovascular system and causes myocardium infarction and arrhythmia. Unfortunately, there is no specific and effective antidote for the treatment of aluminium phosphide poisoning, and this poisoning causes a high mortality rate in the first 24 to 48 hours[1,3-6] (1,6-3). From a general point of view, aluminium phosphide poisoning is one of the most common causes of death in many countries, especially developing countries[1].

Most of the poisonings happen with the intention of suicide and sometimes accidentally and rarely with the intention of crime[4,7,8]. This compound was presented to the market for the first time in India, and in this country, there are about 15,000 accidental or intentional poisonings, two-thirds of which lead to death[6]. In Nepal, more than 50% of suicides were caused by pesticides, of which 14% were due to the consumption of aluminium phosphide[9].

Several other reports indicate the consumption of aluminium phosphide for suicide in recent decades in Tehran and other parts of Iran[10]. According to the reports of the legal medical organization of Iran, during the first four months of 2011, there were 116 cases of suicide with aluminium phosphide in Tehran, Mazandaran, Gilan, and Lorestan provinces[1].

Zinc phosphide, known as rat poison, has a similar effect to aluminium phosphide (rice pill). Zinc phosphide is a solid grey chemical compound whose commercial sample is dark in colour or even black and white. Severe drop in blood pressure and cardiac toxicity are among the symptoms of zinc phosphide poisoning and are accompanied by a high mortality rate[11].

The effects of acetyl-L-carnitine (ALCAR) on mitochondrial respiratory chain function, ATP production, oxidative stress, and cell apoptosis were investigated in rodents. The study results showed that it significantly increases necrosis and oxidative stress without reactive oxygen species (ROS), improves plasma iron levels, and increases cytochrome oxidase activity, enhancing ATP production[12]. In another study conducted by Elgazzar et al., the results showed that patients who received L-carnitine supplements significantly reduced the need for intubation and mechanical ventilation and a significantly shorter hospital stay. Clinical evaluations have shown that L-carnitine administration is safe, and no side effects have been reported[13].

There is no comprehensive study on the effect of L-carnitine on aluminium phosphide, so this review aimed to determine the prevalence of intravenous L-carnitine administration in patients with rice pills (aluminium phosphide) and zinc phosphide poisoning.

Methods

The current narrative review was conducted in 2023 using the keywords aluminium phosphide, zinc phosphide, intravenous L-carnitine, and poisoning in Persian and English languages. Eligible articles published between 1970 and 2022 were searched in international databases SID, Magiran, Scopus, Web of Sciences, Pubmed, Wiley, Science Direct, and Google Scholar. Experimental or semi-experimental articles written in English or Persian that matched our study's purpose were included. Articles with unavailable full text, systematic reviews, and meta-analyses were excluded.

Results

Aluminium phosphide and zinc phosphide: Aluminium phosphide (Alp) is available as dark grey or yellow crystal pills. Several brands include Celphos, Phosphume, Phostoxin, Synfum, Phostek, Quickphos, Degesch, Cheffume, Alphosm, Talunex, and Delicia. One gram of phosphine gas is evaporated from 3 grams of a pill in contact with water.

In Iran, the Alp pill, Phostoxin, contains aluminium phosphide, ammonium carbamate, and urea. This gas is

flammable. Ammonium carbamate prevents the spontaneous combustion of the Alp pill. It should contain Alp and ammonium carbamate in a ratio of 56:44, respectively. In contact with water, the pill produces Phosphine and ammonia-carbon dioxide (2). Contact with acids may even increase the release of Phosphine (PH_3) (3). Phosphine gas has a molecular weight of 34 Da. It is colourless, flammable, and smells like rotten fish. The density is 1.52 g/L. The gas is detectable at ≥ 2 mg/L (2 ppm) concentrations. The smell of spoiled fish is a feature of Alp and Arsine; diphosphine (P_2H_4), hydrogen, and methane impurities are causes of this smell[1].

Zinc phosphide is a dark grey crystalline compound. It is a rodenticide against small mammals such as mice, rats, and squirrels. Humans may be exposed to zinc phosphide poisoning by accident or suicide. After entering the body, it turns into phosphine gas, and then it is mixed in the blood with the help of the stomach and intestine, and the liver and lungs catch it. There is currently no known antidote. Organophosphate poisoning, like zinc phosphide poisoning, is an important cause of morbidity and mortality in economically active age demographics, especially in developing countries. The mortality rate of zinc phosphide poisoning is around 37-100%[2].

Toxicokinetic: Absorption: Phosphine evaporates because of Alp and water and/or acid contact in the gastrointestinal (GI) tract. Phosphine can be rapidly absorbed via the GI tract and lungs. A part of the phosphide of the Alp is absorbed via the GI tract and then converted to Phosphine in the cells. Absorption through the skin and eyes is uncommon but may occur.

Distribution: Phosphine concentration increases in the liver and blood after consumption. Then, the small molecules of phosphonine can be easily distributed in all body parts[3]. Metabolism and elimination: hypophosphite is the most important metabolite of Phosphine in the urine, and phosphite and phosphate are other metabolites. The Phosphine is eliminated via exhalation. It should be noted that Alp may be excreted in urine without any changes[4].

Toxicodynamics: PH3 is responsible for most of the toxic consequences of metal phosphides. This substance is a protoplasmic poison and disrupts the function of cellular proteins. PH3 inhibits electron transfer and cytochrome C oxidase, and these events inhibit oxidative phosphorylation, cellular respiration, and peroxide radical activation[6]. Depleted glutathione and catalase are inhibited by Phosphine, which can lead to dysfunction of membrane channels[9].

Phosphine inhibits cellular respiration, the entry of amino acids into the protein production cycle, and cytochrome C oxidase of the heart myocardium[10]. These changes in mitochondria and myocardial proteins disrupt the permeability of the cell to ions and cause a change in the potential of the cardiac cell membrane. The pathophysiological changes of lung cells and peripheral small vessels of the myocardium are more obvious following the effect of Phosphine on these cells[4].

Alp and Phosphine both inhibit cholinesterase, and it is not known whether this inhibition correlates with clinical symptoms[15]. Another toxic effect of Phosphine is the change in heme capacity. Humans and rats can absorb the aluminium phosphide salt without hydrolysis, which reacts with free haemoglobin and haemoglobin in normal red blood cells (RBC) to produce hemichrome, a derivative of methemoglobin[10]. Phosphine can affect oxyhemoglobin, which interacts with CO and causes methemoglobinemia, increasing CO findings. Carbon monoxide can help diagnose and diagnose Alpine poisoning, which is measured by CO oximetry[16].

Because Phosphine produces free radicals, these radicals are absorbed in the organs that need more oxygen (heart, lung, kidney, and liver). These organs are more sensitive to the damage caused by phosphine gas, which is consistent with post-mortem histopathology. In addition, Heinz bodies, which represent haemoglobin degradation in vitro, are increased in poisoned patients by 1.25 μg/ml[17].

Poisoning dose: The lethal dose of Alp in a 70 kg adult is 500 mg. Phosphine levels of 50 mg/L (50 ppm) in the air in occupational environments may be hazardous to health. If this level reaches 400-600 mg/L (400-600 ppm), it can cause death in 30 minutes[18].

Clinical manifestations of aluminium phosphide poisoning

Acute poisoning after digestion: The first manifestation of poisoning occurs in 10-15 minutes and affects the cardiovascular and respiratory systems with a rapidly progressive course. If the pill is swallowed, it

also affects the digestive system[11].

The initial symptoms of nausea, vomiting, epigastric and retrosternal pain, shortness of breath, anxiety, irritability, and the smell of garlic or rotten fish from the breath of patients can also be recognized in the early stages of poisoning. Initial gastrointestinal manifestations include epigastric pain, vomiting, and hematoma. Dysphagia is common but late in onset. Destruction of the oesophagus, stomach, and duodenum can be seen in endoscopy. Esophageal obstruction or fistula may also occur[19]. Central nervous system poisoning manifests as anxiety, irritability, dizziness, ataxia, numbness, paresthesia, and tremors. These symptoms occur in case of hypoxia or low blood pressure. Delayed and severe neurological symptoms include delirium, seizures, and coma[20].

The most common findings of liver toxicity are increased aspartate transaminase and alanine transaminase. Jaundice can be a sign of liver dysfunction. The most common histopathological findings of hepatotoxicity are sinus congestion and cytoplasmic emptying of liver cells[21,22].

Tachypnea, shortness of breath, crepitation, and crackles are signs of respiratory involvement. Acute respiratory distress syndrome and pulmonary edema are common in these patients and are associated with accumulating blood fluids containing protein in the pleural space[11].

Severe hypotension, decreased cardiac output, increased size of the ventricles, increased systemic venous pressure, hypokinesis of the left ventricle and septum, akinesia, and disproportionate contraction of systemic vessels with normal capillary wedge pressure are among the most common findings of heart poisoning with Alp[19]. The length of time since taking Alp affects electrocardiographic (ECG) changes. Sinus tachycardia occurs during the first three to six hours, followed by conduction delays and arrhythmias six to twelve hours after Alp administration. The most common arrhythmias are supraventricular tachycardia (46%), ventricular tachycardia (40%), ventricular fibrillation (23%), and atrial flutter and fibrillation (20%)[23]. Autopsy findings show severe heart failure, subendocardial infarction, heart congestion, cardiac fibre separation due to edema, pericarditis, destruction of cardiac fibres, non-specific myocyte vacuolation, neutrophilic and eosinophilic infiltration, and local necrosis[24].

Electrolyte abnormalities, including glucose, potassium, sodium, calcium, and magnesium changes occur. Hypernatremia, hyperkalemia, and hyponatremia are associated with poorer prognosis[25,26]. Changes in magnesium, calcium, phosphate, citrate, and cortisol levels can affect blood glucose levels, associated with a poorer prognosis[27].

Other uncommon manifestations include pancreatitis, hepatitis, ascites, pericarditis, acute myocardial infarction, pleural effusion, adrenal gland congestion with haemorrhage or necrosis, acute tubular necrosis (ATN), ventricular tachycardia (VTlysiscardia), disseminated intravascular coagulation (DIC), and methemoglobinemia[10,28-30]. Fistula and oesophagal stricture are usually delayed. Most deaths occur in the first 12-24 hours, often due to cardiac arrest[31], but death after 24 hours is often due to liver failure[11].

Acute inhalation poisoning: After inhaling small amounts of phosphine gas, respiratory tract irritation and shortness of breath often occur[28]. Chest heaviness, dizziness, headache, paresthesia, numbness, muscle weakness, ataxia, nausea, vomiting, diarrhea, tremors, and diplopia may occur[32]. Excessive gas inhalation can lead to acute respiratory distress syndrome (ARDS), heart failure, dysrhythmias, seizures, coma, hepatotoxicity, and renal toxicity. Hepatotoxicity and renal toxicity are late manifestations[18].

Chronic inhalation poisoning: Chronic exposure to phosphine gas (silo workers) can cause loss of appetite, epigastric pain, cough, shortness of breath, drowsiness, and chest pain. Chronically low levels of Phosphine can cause toothache, swelling of the lower jaw, and gum necrosis. Chronic phosphine gas contact with the skin (0.4 mg/L (0.4 ppm)) can cause hypersensitivity and skin congestion[33,34].

Laboratory findings of Alp poisoning: Laboratory tests are essential for managing Alp poisoning and can predict the outcome. Generally, these tests include CBC evaluation, blood sugar, liver and kidney function tests, electrolytes, arterial or venous blood gases (VBG, ABG), ECG, heart monitoring, and chest X-ray. It has been said that hypomagnesemia or hypermagnesemia is associated with widespread destruction of myocytes[28]. Magnesium levels can be high/low and even normal in poisoned patients. CBC shows a decrease in white and

red blood cells[1,35]. Sodium and potassium levels can be high or low. Hypokalemia occurs mainly after swallowing Alp pills and due to vomiting or the release of catecholamines[10]. Hypoglycemia can be caused by adrenal insufficiency, gluconeogenesis disorder, or glycogenolysis and can be severe and persistent[25]. These findings indicate a poorer prognosis. Metabolic acidosis accompanied by respiratory alkalosis or metabolic acidosis can be diagnosed based on blood gas analysis[19,27].

Pleural effusion, lung edema, and pericardial bleeding can be found in the chest X-ray. Sinus tachycardia, inverted T wave, ST segment changes, AV block, features of myocardial infarction, and complete heart block (CHB) are also presentations of Alp poisoning in ECG[10]. If the patient survives, ECG abnormalities return to normal within 10-25 days. Life-threatening changes in ECG can be seen in 50% of patients[36].

Biosample tests in aluminium phosphide poisoning to detect PH_3: Phosphine is quickly oxidized to phosphate. We cannot evaluate hypophosphite for clinical investigations[37]. Also, it is unnecessary to prove its presence in tissues to diagnose Alp poisoning. Although quantitative evaluation of Phosphine is helpful in forensic investigations, these evaluations include exhaled air analysis (50% of patients poisoned with Alp have a positive exhaled breath test)[28], stomach contents (vomiting and gastric lavage), urine sample (only checking the metabolites resulting from the oxidation of PH_3). In an acidic environment, heating the liver tissue causes the release of PH_3 in the tissue matrix. It can be an excellent method to detect Phosphine after death[17].

Routine Alp detection and methods of quantification: Qualitative chemical colourimetric tests of biological samples such as mercury chloride in ethanol, silver nitrate (0.1 N or 16.987 g/L), diethyl carbamate 0.1 or acidic potassium permanganate (0.1 N or 79.017 g/L) detect Phosphine. The sample is heated in an acidic environment to release PH3 and produce a specific colour (dark grey or black) by chemical interaction. Silver nitrate paper is the most common and valuable method for detecting PH3 in clinical and medical tests, as it can detect exhaled air and stomach contents (35). This method detects PH3 at low concentrations (0.05 mg/L (0.05 ppm). Arsenic, ammonium, and ammonium molybdate are less commonly used as tracers. Ammonium and arsenic are used to evaluate exhaled air. Ammonium molybdate is also used to evaluate gastric contents[4].

A semi-quantitative test (potentiometer) is used to determine the concentration of PH3. PH3 reacts with mercuric chloride, which causes changes in oxidation-reduction potential and, as a result, changes in the electrical conductivity of electrochemical cells. The chromatographic test is currently the most sensitive and specific quantitative method for using phosphorus nitrogen as a tracer. This method can measure even the lowest level of PH3 in the air[39].

Differential diagnoses of phosphine poisoning: Evidence of mild poisoning is similar to symptoms of upper respiratory tract infections. Severe poisoning can be confused with cardiopulmonary apoplexy, bacterial pneumonia, viral pneumonia, or ARDS[11].

The signs and symptoms of zinc phosphide poisoning and Alp poisoning can be similar. However, the symptoms of zinc phosphide poisoning are usually demonstrated more slowly, and the mortality rate is lower[36-40].

Treatment of phosphine poisoning: The first step to diagnosis is whether the patient has been exposed to the Alps. (using the above diagnostic items). The entire related medical staff should examine the patient. A face mask and rubber gloves should be used for the examination. It should be noted that masks with small pores cannot prevent contamination with PH3[39].

The patient's clothes should be removed if they are contaminated. Infected skin and eyes should be washed thoroughly. If vomited contents are present, they should be cleaned and discarded as they may contain PH3 and be dangerous to others. The cleaner should be warned of the hazards. In inhaling phosphine gas, the patient should immediately leave the contaminated environment, clothes should be changed, and washing should be done with the recommended precautions[28].

Treatment of AlP poisoning is palliative, and there is no specific antidote for the treatment. Time significantly affects prognosis. The treatment of AlP poisoning should be started based on the history and physical examination of the suspected cases, and it should not be delayed until laboratory results are confirmed[3]. Symptomatic patients should be monitored in the intensive care unit (ICU) for at least 72 hours. Administration of 100% oxygen and intravenous fluids

should be initiated, and electrolyte disturbances should be monitored and treated. Based on what was said, a complete laboratory investigation is necessary[10]. Intravenous (IV) infusion of nine ampoules (9 g) of ALCAR in 500 mL of normal saline should be continued continuously until recovery or death[14].

Mechanism of L-carnitine: It is an amino acid that causes fatty acid to enter mitochondria and causes fatty acid beta-oxidation. Causes of deficiency in the body are valproate poisoning, metabolic disorders of newborns, hemodialysis, zidovudine use, which causes mitochondrial myopathy, and cardiomyopathy in children.

There are two isomers for L-carnitine, including L and D, whose active form is L. It does not bind to plasma protein; its distribution volume is 0.7L/Kg. L-carnitine is a part of category B drugs during pregnancy[40].

Side effects and contraindications of L-carnitine: The most common side effects are nausea and vomiting in very high doses, fishy odour of the body, diarrhea, and periods of convulsions (case reports). There is no contraindication[40].

Prognosis and long-term effects of phosphine poisoning: The mortality rate of phosphine poisoning in more than 500 mg is 30% to 100%. Cardiovascular collapse is the main cause in the first 12-24 hours[41]. Mortality mainly occurs because of severe acidosis, persistent shock, and ARDS after 24 hours of poisoning. Complete liver failure occurs in the first 72 hours, and it is one of the lethal causes[28]. Lower doses of Phosphine expired pills or pills exposed to air are more likely to survive. Vomiting and initial treatments increase the survival rate[4,19].

Higher phosphine levels, no vomiting after drug use, acidosis, hyperglycemia, hyperkalemia, hyponatremia, hypernatremia, hyperuricemia, leukocytosis, methemoglobinemia, low prothrombin time, abnormal ECG, shock, need for inotropes, need for mechanical ventilation, high grades of simple acute physiology score (SAPS), high Glasgow Coma score, and high levels of acute physiology, and chronic health evaluation II (APACHE II) are associated with higher mortality rate and poor prognosis. The higher blood levels of phosphonine increase the death risk[42,43].

The patient will be asymptomatic if there are no remarkable symptoms six hours after inhalation or ingestion[44]. Cardiorespiratory involvements are related to the poor prognosis[41]. The first 24 hours is the critical time associated with the highest mortality rate. The severity of the poisoning and the time between poisoning and starting treatment are important. GI complications, including dysphagia, esophageal stricture, and esophageal fistula, occur in the first month of poisoning. Patients should undergo GI examination with barium swallow or endoscopy, and if complications occur, they should be treated as soon as possible[28]. L-carnitine administration is safe, and no side effects have been reported[45-47].

Discussion

The study by Shekarzadeh et al. entitled "Comparison of the death rate caused by aluminium phosphide with other deaths caused by poisoning in Golestan province in the years 1389-1394", which was carried out in a retrospective descriptive manner on all corpses - referred to the General Department of Forensic Medicine of Golestan province[36].

Concluded that out of 420 cases of death caused by poisoning, 105 cases (25%) were due to aluminium phosphide, which is the second cause of death caused by poisoning after drugs. Out of the total 105 people, 63.8% i.e. 67 people, were men, and 42.9% i.e. 45 people, belonged to the age group of 20-29 years. 46.7% of cases, 46 people are married, and 40% of them, 42 people have education up to secondary level. 99% of deaths caused by aluminium phosphide are suicides, which often occur in winter. The conclusion of the results shows that poisoning with aluminium phosphide is the second cause of death after drugs in the province, which mostly occurs in men due to suicide[37].

In comparing the present study with this study, 31% of the investigated subjects, i.e., 15, were men, which does not fit this study's population ratio. The present study examined poisoning with phosphines, including zinc and aluminium phosphide, where 44% of the cases were poisoned with aluminium phosphide. In terms of comparison, the largest age group among the examined cases of poisoning was 35% of people belonging to the age group of 11-20 years and 33% of the age group of 21-30, which, according to the proximity of the frequency of these two age groups can be He concluded that it is the same as the above study that the highest

number of poisoned people were in the age group of 20 to 29 years.

In the study by Khodabandeh et al. entitled "Investigation of the frequency of complications leading to death in poisoning with rice tablets" in 2013 at Shahid Beheshti University of Medical Sciences and by examining 111 cases of death, they concluded that the most common symptom following the consumption of rice tablets was vomiting. It was seen in 96%. Restlessness, with 37%, and thirst, with 28%, were the most common complaints of patients when entering the emergency room. Although various complications such as hypotension, acute kidney failure, and multiorgan failure have been the cause of mortality in the cases under study, cardiac arrhythmia with 36.9% (41 people) was determined to be the most common complication leading to death due to rice pill poisoning in this study. Metabolic acidosis (pH less than 7.30) was reported in 62.2% of patients[39]. Comparing this study with the mentioned study, it can be pointed out that metabolic acidosis (pH less than 7.35) in this study was found in 81.25% and metabolic acidosis (pH less than 7.30) in 77% of the studied subjects. Before starting the treatment and upon arrival, it was observed that there was a significant similarity between the number of people with metabolic acidosis in the mentioned study and the number of people with metabolic acidosis.

In a study conducted in 2017 by Dr. Ismail Farzaneh and colleagues on prognostic factors in acute poisoning with aluminium phosphide. In this study, clinical and laboratory data, using Acute Physiology and Chronic Health Evaluation II (APACHE II), the order of evaluating organ failure (SOFA), and Simple Acute Physiology Scoring Systems (SAPS II), were compared for them. A total of 68 poisoned patients with confirmed acute AlP poisoning were included in the study for evaluation. Thirty-six of them did not survive. The parameters and values obtained from the patient's clinical and laboratory data showed that four factors were significant for predicting mortality: Glasgow Coma Score (GCS), systolic blood pressure (SBP); urine output (UOP), and 〖HCO〗_3. These four nomogram variables are designed to identify high-risk patients and predict mortality risk. The results of the study showed that (SBP <92.5 mmHg (p = 0.006); 〖HCO〗_3- <12.9 (p = 0.01), UOP <1725 ml/day (p = 0.04); and GCS <14.5(38)

Conclusion

Based on our findings and the lack of correlation between aluminium phosphide poisoning and venous blood gas parameters, as well as the correlation of these parameters in age and gender groups in zinc phosphide poisoning, this study can be used as a foundation for further studies to help to improve the management of patients with zinc and aluminium phosphide poisoning. The addition of L-carnitine to the treatment protocol may improve the prognosis of poisoned patients. However, it should be evaluated in clinical settings, and further studies should investigate this issue.

Acknowledgment

None.

Conflict of interest

The authors further declare that they have no conflict of interest.

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