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Review article

Aluminium phosphide poisoning

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ABSTRACT

Aluminium phosphide (AlP) is a commonly used rodenticide, is easily available and very cheap. However, it is a highly toxic compound (mortality ranging from 30%–100%) and is one of the most commonly used suicidal poison. It is the liberation of phosphine from AlP which produces the toxic effects. Clinical symptoms of AlP poisoning can range from nausea, vomiting to cardiac arrhythmias, ARDS and disseminated intravascular coagulation. Diagnosis is based on history of ingestion and if in doubt, by testing gastric contents on silver nitrate impregnated paper test. Different studies have noted certain clinical and laboratory features that predict mortality in AlP poisoning. Treatment is mainly supportive with no specific antidote. Gastric lavage with coconut oil has been effective and so has intravenous treatment with 20% intra lipid emulsion. Boric acid has been proposed as an antidote but requires further validation. Being commonly used by agricultural workers for their crop protection, proper handling of AlP needs to be done to prevent accidental poisoning.

Contents

1. Introduction

Aluminium Phosphide (AlP) is a commonly available insecticide and rodenticide [1]. It is available in the form of pellets, tablets, compressed discs and powder under the brand names of Celphos, Alphos, Quickphos, Phosfume, Phostoxin, Talunex, Synfume, Chemfume, Phostek etc [1].

The first reported case from India was in 1980 and the case was reported from M.G.M. Medical College, Indore [2]. Since then, the incidence of poisoning has been increasing. AlP was found to be the most common suicidal poison in Northwest India between 1992–2002 [3]. In a study by Gupta et al, aluminium phosphide poisoning was the second most common form of poisoning [4]. This is because of its easy availability. It is used commonly by the agricultural workers for preserving their crops. No wonder, maximum cases are reported from young adult rural population [5]. In a study from Northern India done on children having AlP poisoning, 93.3% were from rural background [6].

The AlP tablet is available in the strength of 0.5–3.0 gm. 56% is aluminium phosphide with remaining 44% being carbamate [5]. Fatal dose is 0.5 g [7].

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2. Mechanism of toxicity [5]

AlP tablets after coming in contact with atmosphere or hydrochloric acid in the stomach liberate phosphine (PH_3). The phosphine gas gets absorbed throughout the gastrointestinal tract and produces toxic effects on heart, liver, lungs, kidneys. The phosphine gas is excreted from the lungs in unchanged form and is also metabolized to hypophosphite to get excreted from the kidneys. At molecular level, phosphine causes inhibition of cellular respiration at mitochondrial level, inhibition of cytochrome C, formation of hydroxyl radicals and lipid peroxidation. Decreased levels of glutathione, decrease activity of catalase and increased activity of superoxide dismutase are also observed. Phosphine can cause direct alveolar damage leading to lung injury.

3. Occupational hazard

AlP is used as a rodenticide by agricultural workers. Also, the workers where these AlP products are manufactured are likely to get exposure to this lethal poison. National Institute of Occupational Safety and Health (NIOSH) recommended exposure limit for phosphine is 0.3 ppm as a time weighted average for upto 10 h per day in a 40-h work week [8]. NIOSH has also established guidelines for workers and employers to prevent accidental exposure [8].

4. Clinical and laboratory features of AlP poisoning

In a case series from Northern Iran, nausea and vomiting were present in nearly all the patients [9]. Other symptoms observed were epigastric pain and loss of consciousness. Hemodynamic instability was noted in 87.5% of patients. Metabolic acidosis was noted in all the patients with cardiac arrhythmias and deranged liver functions being noted in 62.5% patients.

A prospective study from South India, showed almost similar clinical and laboratory findings as the study from North Iran [10].

The various cardiac arrhythmias observed in a study were supraventricular and ventricular ectopics (in all patients), supraventricular tachcardia (46.7%), ventricular tachycardia (40%), ventricular fibrillation (23.3%) and atrial flutter/fibrillation (20%) [11]. Variable degrees of heart block were observed in 33% patients. Changes simulating myocardial ischemia were observed in all patients (ST depression in 90% and ST elevation in 10%). In a recent study from Pakistan, almost similar findings were noted [12]. In this study mortality was significantly more in those who had cardiac arrhythmias.

In a recent study done on seven patients of AlP poisoning, low ejection fraction was demonstrated on 2-D Echocardiography in all patients [13].

Other symptoms noted after AlP poisoning are oliguria (in 50% patients), jaundice and breathlessness [14].

Adult Respiratory Distress Syndrome (ARDS), disseminated intravascular coagulation, intravascular hemolysis, gastrointestinal bleeding, fulminant hepatic failure, congestive cardiac failure, pericarditis, hypomagnesemia, hypermagnesemia and hypokalemia have all been documented in AlP poisoning [14].

Both hypoglycemia and hyperglycemia and methemoglobinemia have been reported in AlP poisoning [15,16]. Hypo and hypernatremia have also been reported [17].

After inhalation of AlP, cough, chest tightness and breathlessness have been observed [14]. If the exposure is severe, pulmonary edema and ARDS can occur [14]. Other systemic manifestations as mentioned above can also occur after inhalation.

Death occurs within first 24 h mainly due to shock and arrhythmias [14]. Deaths occurring later are due to ARDS, liver failure, renal failure or due to other complications [14].

Survivors can have long term sequelae in the form of peripheral neuropathy, esophageal strictures and tracheoesophageal fistulas [1].

Post mortem of patients dying due to AlP poisoning showed garlicky odour close to the body in 50% with histopathological examination showing necrosis in kidneys (100%), lungs (100%), liver (88%), spleen (82%), adrenals (71%), stomach (56%) and heart (36%) [18]. Necrosis and edema were also seen in many organ systems.

5. Diagnosis of AlP poisoning

This is a clinical diagnosis. Most of the times, history of ingestion is evident. However, at times the history is not very forthcoming. In doubt, the diagnosis can be readily made by silver nitrate- impregnated paper test on viscera, gastric contents or on breath [5]. This test is based on the principle that the silver nitrate-impregnated paper will turn black when exposed to phosphine. Diluted gastric contents are heated in a flask for 15–20 min with the silver nitrate-impregnated paper on the mouth of the flask. If phosphine is liberated, it will turn the paper black. Addition of ammonium molybdate to the black turned paper and change of colour to blue further confirms the presence of phosphine [5]. Hydrogen sulphide can also lead to blackening of paper [5]. To further distinguish, lead acetate paper is used. Phosphine will not turn lead acetate black [5]. Chugh et al in a study on 50 AlP poisoning found that silver nitrate impregnated paper test was positive in all the patients when used for gastric contents but it was positive in only 50 percent of the patients when tested on breath [19]. In a recent study from Iran on post-mortem in patients with AlP poisoning, the silver nitrate test was positive in 75 percent when done on intra-abdominal organs and in 50 percent when done on gastric contents [20]. It was also noted in the same study that positivity of silver nitrate test on intra abdominal organs was related to number of AlP tablets ingested. Those with ingestion of a single tablet had negative silver nitrate test [20].

Measuring levels of phosphine in blood and urine is not recommended [5].

For the viscera and gastric contents obtained from autopsy of patients with suspected AlP poisoning, gas chromatography with nitrogen-phosphorous detector is the most sensitive and specific method to confirm poisoning by AlP [5]. It detects phosphine. Head space Gas Chromatography with Mass Spectrometry (HS-GC–MS) has been used to detect metabolites of phosphine [21]. This test can detect phosphine as low as 0.2 mcg/mL in a sample. Advantage of this test is that it tests metabolites of phosphine even when phosphine has been converted.

Inductive Coupled Plasma- Mass Spectroscopy (ICP-MS) has been used to detect phosphorous in the dead body of a patient [22]. The dead body was found ten days after the poisoning and phosphine was not detectable in blood and urine. Phosphorous was found in high concentrations in blood and liver.

6. Prognostic factors

The mortality from AlP poisoning ranges from 30 to 100% [5]. In the study by Gupta et al, mortality from AlP poisoning was 70% [4].

In a study of 12 patients with AlP poisoning, it was found that non-survivors were slightly older with most being females [23]. Most of the non-survivors had consumed fresh tablets of AlP and also, more quantity [23]. Shock, cardiac arrhythmias, pneumonia, ARDS, confusion and uremia were more common among non-survivors [23].

Louriz et al in their study from Morocco studied various parameters in patients with AlP poisoning and found that presence

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