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Research Article

COCONUT OIL, A COUNTER-AGENT IN ALUMINIUM PHOSPHIDE POISONING

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A B S T R A C T

Aluminium phosphide (ALP) poisoning is one of the major causes of suicidal deaths. Toxicity is caused by phosphine gas produced from the reaction of Aluminium phosphide with water and hydrochloric acid in the stomach which leads to multi-organ dysfunction. There is no antidote available for aluminium phosphide poisoning, only supportive therapy can be given. In this case, in spite of all supportive measures and care the patient did not survive. No coconut or almond oil was administered to the patient. Some of the case reports and articles suggested the role of coconut oil and increased survival chances in the absence of specific antidote for aluminium phosphide poisoning.

INTRODUCTION

A major cause of poisoning with suicidal intent in many countries is due to Aluminium Phosphide (AlP) [1, 2]. It is a solid fumigant available in form of tablets, pellets, granules or as a dust. Commercially, it is a dark grey coloured tablets of 3.0 g each, consisting of Aluminium phosphide (56%) and carbamate (44%), some of the brand names include Celphos, Phostoxin, Phosfume, Degesch, Synfume, Aluminium phosphidehos, Chemfume, Talunex, Phostek or Delicia, Quickphos [8,10].

Statistics

- ✓ Approximately 300,000 deaths occur annually worldwide due to pesticide poisoning [3].
- ✓ Death rate with aluminium phosphide poisoning is severe, ranging from 37% to 100% [4, 5].
- ✓ In a study of acute poisoning of about 559 cases in India, 68% were due to exposure of Aluminium phosphide, with 60% mortality [6, 7].

Effects

Aluminium phosphide poisoning may lead to multi-organ dysfunction. Presentation varies depending on variables such as doses, exposure route, and the time interval between exposure and treatment initiation [9].

Clinical Manifestations

The symptoms of mild phosphide inhalation are similar to upper-respiratory-tract infection including nausea, cough, vomiting, headache, diarrhoea, dizziness and fatigue.

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On severe exposure, it may cause lung irritation with persistent coughing, paraesthesia, diplopia, tremor, hypotension, ataxia, jaundice and weak pulse can also be seen. Severe metabolic acidosis, oliguria, proteinuria, anuria and finally cardiovascular collapse may occur, haemodialysis should be done as soon as possible. Sudden death occurs within 4 days and can be delayed for 1–2 weeks. Focal myocardial infiltration and necrosis may be revealed on Post-mortem examinations, widespread small vessel injury and pulmonary oedema.

Diagnosis

The diagnosis of phosphine poisoning can be done by a silver nitrate-impregnated paper test which is used to test the gastric fluid and breath of the patients exposed to phosphine/phosphide. Phosphine/phosphides react with silver nitrate to form silver phosphide confirming the diagnosis.

Monitoring Parameters

Laboratory investigations including haematocrit, haemoglobin, full blood count, arterial blood gas analyses, liver and renal function tests and cardiopulmonary monitoring are essential for the assessment of organ effects [11-14].

Mechanism of toxicity

Aluminium phosphide reacts with moisture, water and hydrochloric acid in the stomach to produce phosphine gas [15].

$$2\ AlP + 6H_2O \rightarrow Al_2O_3 \cdot 3H_2O + 2PH_{3\uparrow}$$
$$AlP + 3HCl \rightarrow AlCl_3 + PH_{3\uparrow}$$

The mechanism includes cellular respiration failure because of the effects on mitochondria, it inhibits cytochrome C oxidase

and leads to the formation of hydroxyl radicals which are highly reactive [17, 18]. Lipid peroxidation causing cellular injury is also suggested [19]. There is an increase in the activity of superoxide dismutase and a decrease in the levels of catalase [20]. Cellular injury can also be caused due to a decrease in glutathione concentration in different tissues. Glutathione acts as a protecting factor against oxidation, it works by catalysing the reduction of the oxygen peroxide to oxygen and water [21]. Indicators of oxidative stress reach peak levels within 48 hours of exposure which normalises by day 5 [19, 22].

Levels >50 ppm of phosphine are dangerous to all creatures, whereas the permissible exposure limit of phosphine is <0.3 ppm in working environment, while at 400–600 ppm it is lethal within half an hour [23, 24].

People involved in the manufacturing of Aluminium phosphide or methamphetamine where phosphine is a by-product, engaged in placing Aluminium phosphide tablets on the stacks of grains (farmers) and in the vicinity of application are at risk for unintentional exposure of phosphine gas, with few reported fatalities [25–28].

Case Report

The following case report describes the case of 38 years old male who ingested 2 tablets of aluminium phosphide (Celphos) with suicidal intent. He was admitted in ICU and gastric lavage with potassium permanganate and 4 tabs charcoal was performed. Intubations were done under all aseptic conditions. 1gm of MgSo4 in 100ml 5% Dextrose was given every 4th hourly and ECG was repeated every 6th hourly. Inj. Atropine 3ml/hour infusion was given. His heart rate, blood pressure and peripheral capillary oxygen saturation (SpO2) went on decreasing with a feeble pulse, pupils dilated and non-reacting to light. Inj. Adrenaline 3 doses were given and CPR was continued. BP, PR, and HR were not recordable and the patient was declared as dead.

Treatment

Personal precautions including a face mask, gloves during decontamination.
↓
Securing airway, IV access preferably Central Venous Pressure and routine investigations.
↓
Gut decontamination with KMnO4 (1:10,000), vegetable or coconut oil within 6 hours.
↓
Symptomatic and Supportive care.

Make sure that airway is patency, to prevent aspiration pneumonitis protect the airways with an endotracheal tube if necessary. Start O_2 inhalation, check for pulse rate regularly and instilled intravenous access, ideally central venous, to begin 0.9% normal saline and vasopressor therapy as suitable. Monitor vitals closely. The initial investigation must include ECG, arterial blood gas, chest X-ray, electrolytes including magnesium, blood glucose, routine hemogram, renal function test and liver function test. Cardiac dysfunction can be early identified by Regular ECG and echocardiography.

Current Treatment

Potassium permanganate ($KMnO_4$) (1:10000) is used for gastric lavage. It oxidizes phosphine to nontoxic phosphate. Some recently published articles assist the use of KMnO4 in Aluminium phosphide [29, 30], Marashi and Nasri Nasrabadi discovered that phosphine is a hard nucleophile and there is no interaction of free oxygen radicals from the resolution of KMnO4 [31]. Hence effectiveness on $KMnO_4$ is unsure against Aluminium phosphide poisoning.

Activated charcoal helps to adsorb phosphine from the gastrointestinal (GI) tract in most of the literature. But Marashi concluded that activated charcoal has large internal surface area comprises of pores (10 Å to 20 Å). It efficiently adsorbs toxins of moderate molecular weight (100 Da to 800 Da). But the molecular weight of Aluminium phosphide is about 58 Da, hence the role of activated charcoal in Aluminium phosphide poisoning is again uncertain [32].

The use of Magnesium sulphate (both high and low dose) did not improve survival in controlled clinical trials. Hence using it is not suggested [33].

Intervention

In the case of Aluminium phosphide poisoning, Vegetable oil administered orally or through a nasogastric tube inhibits the release of phosphine due to physiochemical properties of non-miscibility with fat and Aluminium phosphide [34, 35]. Coconut oil inhibits the breakdown of phosphide, protects the gastric mucosa, reduces the toxicity of phosphides, dilutes gastric acid to some extent and prevents the absorption of phosphine gas. In this case, no oils were used.

Positive Outcomes with Coconut Oil

The positive clinical effects of coconut oil against Aluminium phosphide poisoning in humans have been reported [36, 37]. Mechanism of action is not clear, but it prevents the absorption of phosphine gas by forming a protective layer around the gastric mucosa. Also, it reduces the breakdown of phosphide by dilute hydrochloric acid in the stomach. Saidi and Shoajaie [38] described that intra-gastric lavage with swe*et al*mond oil reduced the mortality of rats poisoned with Aluminium phosphide and also significantly lowered plasma cholinesterase levels. The authors recommended that swe*et al*mond oil should be given orally immediately after Aluminium phosphide ingestion, but it is yet to be confirmed in case of humans.

According to a case report, out of 7 Aluminium phosphide patients, coconut oil was used in 4 of them and only those 4 patients survived [39]. Another case report of a patient with Aluminium phosphide survived with 500ml of coconut oil instilled [42].

Even after 6 h of post-ingestion, the possible role of coconut oil in managing acute Aluminium phosphide poisoning was concluded in a case report [36]. The solution of sorbitol at a dose of 1–2 ml/kg can be used as cathartic. In vitro, experimental studies suggest that fat and oil, mainly vegetable oils and liquid paraffin inhibits phosphine release from the ingested Aluminium phosphide [40].

Some of the articles propose boric acid as a non-toxic and efficient trapping agent and an antidote for PH3 poisoning by investigating the chemical reaction between them [41].

CONCLUSION

Aluminium phosphide poisoning has a high mortality rate. There is no specific antidote available for aluminium

phosphide poisoning. Only supportive measures can be taken to save a life in poisoning. Young people and children should be prohibited from keeping and using aluminium phosphide at the home without proper verification and confirmation. Open sales of this pesticide should be restricted by Official health care system. The manufacturers should be advised to make small packs of 2 – 3 tablets with suitable packing. If possible all of the phosphide derivatives compounds should be banned forever for everyone. Coconut oil plays a major role in Survivability of aluminium phosphide poisoning patients even after 6hours of post ingestion as already observed in the above case reports.

Administration of coconut oil may decrease the mortality rate and increase chances of survivability in aluminium phosphide poisoning.

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