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# Clinically Approved Treatment and Recent Updates of Acute Aluminium Phosphide Toxicity: A Review Article

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**Mini Review**

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## Abstract

Aluminium phosphide is a dangerous cheap toxin used for suicidal purposes in developing countries as Egypt and India. Many studies recently target to reach to the most effective protocol of treatment to decrease its mortality rate. This review aims to draw attention to the latest treatments in this issue..

**Keywords:** Aluminium Phosphide; Toxicity; Management

**Abbreviations:** AlP: Aluminium phosphide; ROS: Reactive Oxygen Species; ATP: Adenosine Tri Phosphate; ARDS: Acute Respiratory Distress Syndrome; ABG: Arterial Blood Gases; VBG: Venous Blood Gases; CBC: Complete Blood Count; CPK: Creatine Phosphokinase; CK-MB: Creatine Kinase Myocardial Band; IM: Intra Muscular; HR: Heart Rate; BP: Blood Pressure.

## Introduction

Aluminium phosphide (AlP) is an insecticide and rodenticide that protects grains from rodents and other household pests [1].

Aluminum phosphide comes in the form of pills or pellets. It has nearly excellent pesticide properties: it is harmful to all stages of insects; it is highly effective; it preserves seed viability; and it leaves only a trace of non-toxic Al $(OH)^3$ residue on food grains [2].

Furthermore, its low cost and widespread availability in the market enhance its use as a suicide weapon, particularly in developing agricultural countries [3].

In Egypt, AlP tablets have recently been a popular method of suicide, and as a result, admission to poison control centers has increased [4].

### Toxicological Mechanism

The exact mechanism by which phosphine causes death in people has yet to be determined. Despite this, phosphine suppresses the mitochondrial respiration in cells. This leads to cellular hypoxia. It also inhibits cytochrome oxidase by inducing oxidative stress through the formation of highly reactive hydroxyl radicals and other reactive oxygen species (ROS). Phosphine also inhibits oxygen intake, resulting in tissue damage. There may be direct toxicity to the adrenal and cardiac myocytes, causing a reduction in blood pressure. Furthermore, phosphine has been associated to decreased blood supply to vital organs. The total creation of ATP (adenosine triphosphate) has decreased. Severe poisoning causes multi-organ system failure. Phosphine transforms ferric iron to ferrous iron, releasing iron from its protein binding condition [5].

### Management of AlP-Toxicity

**Clinical Picture:** Patients typically report airway irritation and shortness of breath after inhaling tiny amounts of phosphine gas. Possible symptoms include dizziness, fatigue, chest tightness, headache, nausea, vomiting, diarrhea, ataxia, numbness, paresthesia, tremor, muscular weakness, visual impairment, and jaundice. After inhaling substantial amounts of the gas, the patient may develop acute respiratory distress syndrome (ARDS), heart failure, cardiac arrhythmias, convulsions, and coma, as well as late symptoms of hepatotoxicity and nephrotoxicity [6].

The primary causes of death in ALP poisoning are cardiac toxicity, cardiac dysfunction, and circulatory failure, all of which culminate in cardiomyocyte death [7].

**Investigation**: Common ECG anomalies include sinus tachycardia, ST segment abnormalities, an inverted T wave, myocardial infarction, AV block (particularly, right bundle branch block), and total heart block If the patient survives the first 24 hours, the ECG alterations will normalize within 10-25 days. Chest x-rays often show pulmonary edema, pleural effusion, and sub-pericardial hemorrhage. A blood sugar test detects hypoglycemia, which can be caused by gluconeogenesis, glycogenolysis, or adrenal insufficiency. The arterial blood gases (ABG) or venous blood gases (VBG) assay detects metabolic acidosis or a combination of metabolic acidosis and respiratory alkalosis. Some of the most common tests for Alp poisoning include liver and kidney function [7,8].

Serum electrolytes can demonstrate hypo- or hypermagnesemia, which is connected to cardiotoxicity and severe myocyte death. The levels of sodium and potassium in the blood might be excessive or low. A complete blood count (CBC) reveals low numbers of both white and red blood cells. Methemoglobinemia and intravascular hemolysis could be identified. Biochemical markers such as creatine phosphokinase (CPK), creatine kinase myocardial band (CK-MB), and Troponin-T have been related with ALP-induced myocardial damage in some cases [9].

However, some sources show that these biomarkers change after ALP poisoning, however these indicators are unreliable [10].

**Stabilization of the Patient: Airway and Breathing:** The airway is patent if the patient responds in a normal voice. If left untreated, a blocked airway might quickly result in cardiac arrest. Any health care practitioner, regardless of environment, can execute a head tilt and chin-lift procedure to open the airway if necessary. With the proper equipment, suctioning the airways to remove obstructions is possible. If breathing is insufficient, assisted ventilation is necessary. If there is a bag mask accessible, trained workers should utilize it [11].

**Decontamination by Gastric lavage:** It is theorized that gastric lavage with oil, such as Paraffin oil 50ml, creates a protective barrier around wounded stomach mucosa, preventing PH3 gas absorption. We can use 80 mL of olive oil, 60 mL of turpentine oil, or 100 mL of almond oil to dissolve one gram of phosphorus [12].

Water-based stomach decontamination procedures should be avoided during acute ALP poisoning. Using vegetable oils for gastric lavage or castor oil to reduce the elevated PH3 release [13].

**Elimination with Coconut Oil**: Researchers believe that its most likely mode of action is to block the systematic absorption of phosphine produced from the swallowed tablets by lining the stomach wall [14].

**Antioxidants as Vitamin E and N- Acetyle Cysteine (NAC):** In a randomized controlled trial of 36 ALP-intoxicated patients, giving 400 mg of vitamin E twice a day via intramuscular (IM) route with other supportive therapies reduced the need for mechanical breathing (30 vs. 62%) and the fatality rate (15 vs. 50%) compared to controls [15].

Furthermore, NAC may have a good function in lowering mortality and the prevalence of mechanical breathing in patients with phosphide poisoning [16].

**Lipid Emulsion:** Theory of the lipid sink According to the "lipid sink" theory, which argues that a lipid soluble toxin may be sequestered inside the lipid emulsion, limiting its impact site concentration and toxicity, is utilized to create intra-lipid emulsion [17].

**Symptomatic and Supportive:** Patients should be given 100% oxygen and should be treated for fluid and electrolyte imbalances. Adrenal insufficiency may occur because of shock; thus, a hydrocortisone infusion is given. f magnesium sulfate in ALP poisoned patients were rather contradictory. Nano particles were also targeted in research. Magnesium nanocarriers were reported to improve heart rate (HR), blood pressure (BP), and reduce lipid peroxidation. If severe acidosis occurs, dialysis is considered. Sodium bicarbonate at a dose of 50-100meq/8hrs is used if bicarbonate level is ≤ 15 ml eq/l [14].

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