

## Aluminium Phosphide Poisoning: A Case Series in North India

Dr Ambuj Giri, Dr Indresh yadav, Dr Shruti bhushan aparajita, Dr Ranjeet Kumar

*1-Senior resident, Critical care medicine, IMS, Banaras Hindu University*

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**ABSTRACT**-Poisoning with aluminium phosphide (AIP) is a serious medical emergency. AIP is a low-cost, extremely toxic fumigant insecticide used to protect grain from vermin and rats. This study describes the demographic details, clinical features, and treatment outcomes of a group of patients who presented with AIP poisoning in a tertiary care facility. During the study period, 8 out of 11 patients were male and 3 were female were admitted with AIP poisoning. Most of the patients were young with a age of <40years. The most prevalent signs and symptoms upon admission were abdominal pain (64%), nausea and vomiting (100%), metabolic acidosis (82%), and hemodynamic instability (82%). A suicidal attempt resulted in poisoning in every case, which led to 9 (82%) deaths. The patients who died had taken a greater dose of AIP, experienced cardiovascular failure, had more severe metabolic acidosis, had refractory shock, and arrived at the emergency department (ED) later than the individuals who lived. All patients were admitted to the intensive care unit, where they underwent gastrointestinal lavage using activated charcoal and coconut oil as well as symptomatic treatment. Since there is no treatment for people with AIP poisoning, it is important to start managing symptoms as soon as possible and adopt preventative steps to reduce the number of instances.

### I. INTRODUCTION

In India, a pesticide known as aluminium phosphide (AIP) is frequently used to preserve grain. It shields the grains from rodents, insects, and pests by releasing poisonous phosphine (PH<sub>3</sub>) gas when it comes into touch with moisture. It is offered in the form of 3g tablets and 10g packets that are packaged together as tablets. Each tablet or package has 44% ammonium carbonate and 56% AIP (1). Zinc phosphide and AIP, which are both solid chemicals used as grain fumigants and rodenticides and are marketed under a variety of brand names including Celphos, Phostoxin, and Quickphos, respectively (2-4). The purpose of

poisoning with these substances is typically suicide, sporadically unintentional, and infrequently homicidal (4). Regardless of gender, the frequency is higher in rural areas (5).

When phosphides are consumed or are in contact with water, they release the gas phosphine (PH<sub>3</sub>). Phosphine is a colourless, combustible gas that smells like fish that has decomposed (2). Myocardial contractility is compromised by phosphorus, and Loss of fluid causes pulmonary edema. Therefore, respiratory alkalosis, mixed metabolic acidosis, and abrupt renal failure may also ensue (4, 6). Disseminated intravascular coagulation, liver necrosis, and hypo- or hypermagnesemia have also been noted (4, 6). Mortality varies greatly (from 37 to 100%) based on a number of variables, including the amount of fresh compound taken, the severity of the shock, and the patient's responsiveness to resuscitative procedures. Acute cardiovascular collapse causes death. This study describes the demographics, clinical features, and prognoses of a group of individuals who presented with AIP poisoning in south India.

### II. METHODOLOGY

This is prospective observational study of AIP poisoning patients in Sir Sunderlal hospital, institute of medical sciences, IMS, BHU. Diagnosis of ALP poisoning was confirmed based on history, clinical presentation, garlic odour in breath, vomits or gastric washing. An enquiry was made as to whether the tablets ingested by the patients were taken from a freshly opened bottle or were lying exposed to the atmosphere. A careful note was made of the approximate amount of aluminium phosphide ingested, period of onset of symptoms after ingestion and pre-hospitalization period. Data collected included clinical manifestations, investigations and treatment administered to each patient. Intention of poisoning was classified as accidental and suicidal. All of cases had been admitted in ICU after initial assessment and supportive measures. Data were

analysed using Microsoft® Excel software. Categorical data were reported as percentage. Continuous data were reported as mean if they were normally distributed and as median and interquartile range (IQR) if they were non-normally distributed.

### III. RESULTS

During the study period, 7 out of 10 patients were men and 3 were women were admitted with AIP poisoning with a mean age of 30 years. 2 patients had been discharged without any complications and 8 (80%) patients had been died. Most of the cases were 25-35yrs old. Female/male ratio is 0.30. Among 10 cases, 2 were single and 8 were married. All cases were due to suicidal attempt none due to accidental or homicidal. Common signs and symptoms at presentation were nausea and vomiting (100%), metabolic acidosis

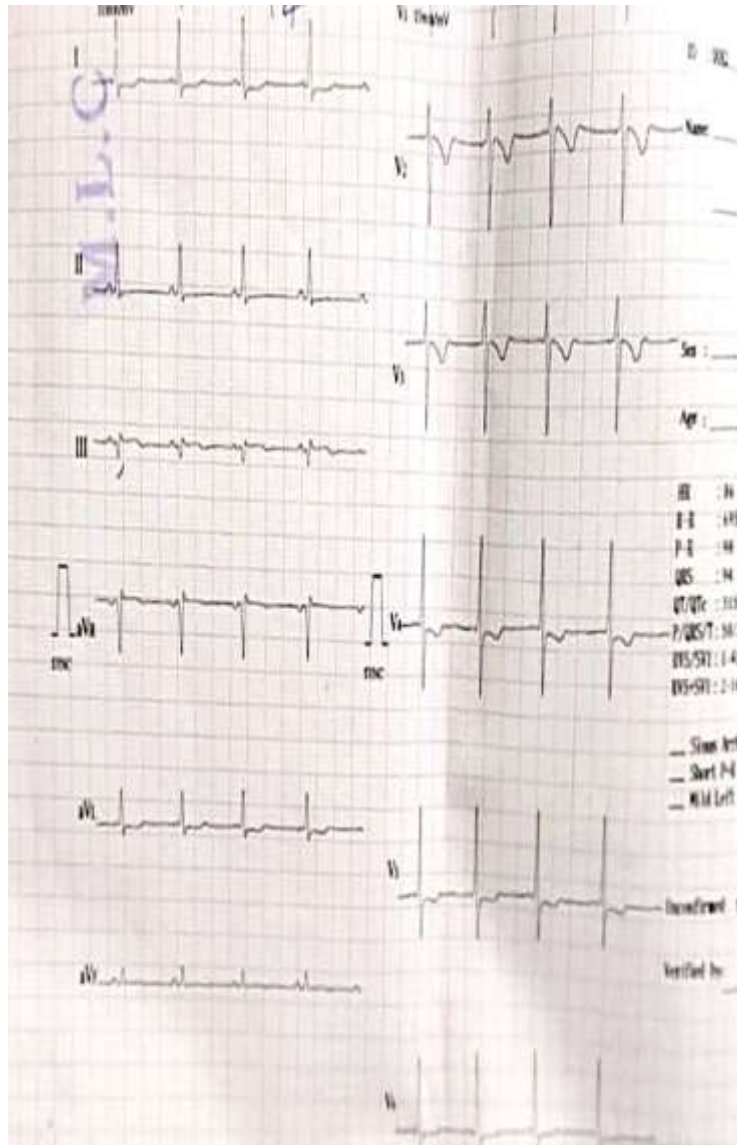
(80%), hemodynamic instability (80%), 6 were having pain abdomen (60%). Majority of deaths occurred during first 36 hrs. Compared with patients who survived, those who died taken higher dose of AIP (4. 2g vs 1. 5g), developed severe metabolic acidosis and hemodynamic instability. Mean time between tablet ingestion and start of medical intervention was 6 hours. Also mean time gap between tablet ingestion and appearance of symptoms was 75 min. 9 patients were intubated and connected to mechanical ventilator. All patients were admitted to intensive care unit received gastric lavage with activated charcoal, coconut oil, vasopressors, magnesium sulphate as membrane stabilising agent and free radicle scavenger, antiemetic, antacids, ascorbic acid as a free radicle scavenger. The median length of hospital stay was 3 (1-5) days.

**Table 1-Demographic characteristics of patient (n=10)**

<b>Male sex (%)</b>	<b>70</b>
<b>Female sex(%)</b>	<b>30</b>
<b>Mean age (years)</b>	<b>30</b>
<b>Dose of AIP (gram)</b>	<b>3.3</b>
<b>Length of hospital stays(days), Mean</b>	<b>03</b>
<b>Duration of onset of symptoms(min), Mean</b>	<b>75</b>
<b>Intention of poisoning(accidental)</b>	<b>-</b>
<b>Suicidal</b>	<b>100</b>

**Table 2-Clinical manifestations of the patient (n=10)**

<b>Variable</b>	<b>NO (%)</b>
<b>Nausea &amp; Vomiting</b>	<b>10</b>
<b>Metabolic acidosis</b>	<b>8</b>
<b>Haemodynamic stability</b>	<b>8</b>
<b>Cardiac arrhythmias</b>	<b>8</b>
<b>Epigastric pain</b>	<b>6</b>
<b>Liver damage</b>	<b>6</b>
<b>Seizure</b>	<b>1</b>



ECG: showing t wave inversions

Blood Gas Values			
↓ pH	7.015		[ 7.350 - 7.450 ]
↓ pCO <sub>2</sub>	23.6	mmHg	[ 38.0 - 45.0 ]
↓ pO <sub>2</sub>	22.3	mmHg	[ 83.0 - 108 ]
Electrolyte Values			
↓ cK <sup>+</sup>	1.6	mmol/L	[ 3.5 - 4.5 ]
↑ cNa <sup>+</sup>	152	mmol/L	[ 136 - 146 ]
↑ cCl <sup>-</sup>	132	mmol/L	[ 98 - 116 ]
↓ cCa <sup>2+</sup>	0.73	mmol/L	[ 1.15 - 1.27 ]
Metabolite Values			
↓ cGlu	49	mg/dL	[ 80 - 110 ]
↑ cLac	7.1	mmol/L	[ 0.5 - 1.6 ]
cCrea	0.51	mg/dL	[ 0.50 - 1.60 ]
ctBil	2	μmol/L	
Oximetry Values			
↓ ctHb	6.3	g/dL	[ 12.0 - 16.0 ]
↓ sO <sub>2</sub>	18.8	%	[ 94.0 - 99.0 ]
FMethHb	0.9	%	
FO <sub>2</sub> Hb	18.5	%	
FCOHb	0.5	%	
FHHb	80.1	%	
Acid Base Status			
cBase(B) <sub>c</sub>	-23.3	mmol/L	
cBase(Ecf) <sub>c</sub>	-23.1	mmol/L	
cBase(B ox) <sub>c</sub>	-24.3	mmol/L	
cBase(Ecf,ox) <sub>c</sub>	-23.9	mmol/L	
Calculated Values			
Anion Gap <sub>c</sub>	14.0	mmol/L	
AnionGap.K <sup>+</sup> <sub>c</sub>	15.6	mmol/L	
cHCO <sub>3</sub> <sup>-</sup> (P.st) <sub>c</sub>	6.6	mmol/L	
cHCO <sub>3</sub> <sup>-</sup> (P) <sub>c</sub>	5.7	mmol/L	
Hct <sub>c</sub>	19.8	%	
p50(st) <sub>c</sub>	27.98	mmHg	
mOsm <sub>c</sub>	306.6	mmol/kg	
cH <sup>+</sup> <sub>c</sub>	96.6	nmol/L	
Notes			
↑	Value(s) above reference range		
↓	Value(s) below reference range		
c	Calculated value(s)		
	1035. One or more derived parameters cannot be calculated		

ABG: Metabolic acidosis with respiratory alkalosis



**X-ray shows pleural effusion**

#### **IV. DISCUSSION**

AIP is a very lethal, easily accessible rodenticide. In especially for wheat, aluminium phosphide is utilised as a grain preservative. AIP is a poisonous substance. Its deadly property is a result of the fact that when moisture is present, it releases phosphine gas, which is easily absorbed by skin contact, ingestion, and inhalation (7). The cause of phosphine toxicity has been determined to be inadequate oxygenation, which leads to the failure of cellular respiration (non competitive inhibition of mitochondrial cytochrome oxidase) and the generation of extremely reactive hydroxyl

radicals (8). The effects of phosphorus gas on the heart, liver, kidney, and lung are systemic and toxic, and they might appear as severe persistent shock, cardiac arrhythmias, acidosis, and pulmonary edoema (8).

Deep hypotension, arrhythmias, congestive heart failure, blockages, and myocardial damage are examples of cardiovascular symptoms (9). In our analysis, the majority of AIP poisoning cases were suicidal; this finding is consistent with numerous other investigations (10–11). Suicidal behaviours may be influenced by a variety of reasons, including family conflicts, drug and



alcohol addiction, emotional discomfort, despair, physical conditions, social isolation, and issues with money and the workplace (12).

Ten individuals with AIP poisoning were prospectively evaluated in the current investigation. The majority of the patients experienced metabolic acidosis, unstable hemodynamics, and nausea and vomiting. In comparison to participants who survived, the eight patients who died exhibited lower pH levels, hemodynamic instability, and consumed more AIP tablets. AIP's lethal dosage has been estimated to be between 150 and 500 mg/70 kg (13). Over 1 g of AIP had been consumed by the study participants who passed away.

Hepatic dysfunction was shown to be a risk factor for a poor outcome in a research by Louriz et al. on 49 individuals (10). Similarly, we discovered that whereas lived subjects did not experience such impairments, 6 of the deceased cases had abnormal liver function tests (LFT). In Shadnia's study (14), ECG alterations, blood bicarbonate level, and PH were all connected to how ALP poisoning turned out.

Since there is no specific cure, supportive care is the mainstay of therapy. Only a few studies have shown that coconut oil can help your body avoid absorbing phosphine gas from your intestines (15).

## V. CONCLUSION

In Asia, AIP is a poisonous substance that is widely used for suicide. For the treatment of patients who have consumed too much AIP, there is currently no efficient antidote. To prevent the occurrence of AIP poisoning, measures such as limiting sales, increasing public awareness of its risks, and training doctors in patient pre-hospital and emergency management are needed. For patient management, extensive hemodynamic monitoring, supportive measures, and symptomatic treatment have been suggested.

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