

Aluminium Phosphide Poisoning: A Case Series in South India

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Abstract: Aluminium phosphide (AIP) poisoning is a life threatening emergency. AIP is a cheap, highly toxic fumigant pesticide used for grain preservation from rodents and bugs. In this study, demographic characteristics, clinical profiles and outcomes of a series of patients presented with AIP poisoning in a tertiary care centre is described. 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 <40 years. The most common signs and symptoms at admission were nausea & vomiting (100%), metabolic acidosis (82%), hemodynamic instability (82%) and pain abdomen (64%). All cases were poisoned as a result of suicidal attempt leading to 9 (82%) deaths. Compared with the patients who survived, those who died had taken higher dose of AIP, developed cardiovascular dysfunction, had severer metabolic acidosis, refractory shock and presented late to emergency department (ED). All patients were admitted to intensive care unit, symptomatic treatment is initiated and received gastric lavage with activated charcoal, coconut oil. There is no antidote for treatment of patients with AIP poisoning, so symptomatic management should be initiated early and preventive measures should be undertaken to decrease the number of cases due to AIP poisoning.

Keywords: Aluminium phosphide, phosphine gas, poisoning, refractory shock, supportive therapy

1. Introduction

Aluminium phosphide (AIP), a solid fumigant pesticide widely used in India for grain preservation. It protects the stored grains from pests, insects and rodents by liberating toxic phosphine (PH₃) gas after contact with moisture. It is available in the form of tablets, each weighing 3g and packets, each weighing 10g packed in container. Each tablet/packet contains 56% of AIP and 44% of ammonium carbonate (1). AIP which is commercially available under various brand names such as Celphos, Phostoxin and Quickphos, and zinc phosphide are solid products used as grain fumigants and rodenticides (2-4). The intention of poisoning with these compounds is usually suicidal, occasionally accidental and rarely homicidal (4). Irrespective of the sex, the incidence is higher in the rural population (5).

When phosphides are ingested or exposed to moisture, they release phosphine gas (PH₃). Phosphine is a colourless, flammable and toxic gas with garlic or decaying fish like-odour (2). Phosphine impairs myocardial contractility and fluid loss which results to pulmonary oedema. Hence, metabolic acidosis or mixed metabolic acidosis and Respiratory alkalosis and acute renal failure may also occur (4, 6). Other reported features include disseminated intravascular coagulation, hepatic necrosis and hypo or hypermagnesaemia (4, 6). Mortality is highly variable (37-100%) depending on the various factors like dose of fresh compound consumed, severity of shock and its response to resuscitative measures. Death is due to acute cardiovascular collapse. In this study, demographic characteristics, clinical profiles and outcomes of a series of patients presented with AIP poisoning in south India are described.

2. Methodology

This is a prospective observational study of AIP poisoning patients in S S Institute of Medical Sciences & Research Centre, Davangere, Karnataka from July 2013 to Dec 2014. Diagnosis of AIP 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.

3. Results

During the study period, 8 out of 11 patients were men and 3 were women were admitted with AIP poisoning with a mean age of 29.2 years. 2 patients had been discharged without any complications and 9 (82%) patients had been died. Most of the cases were 25-35 yrs old. Female/male ratio is 0.37. Among 11 cases, 2 were single and 9 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 (82%), hemodynamic instability (82%) 7 pain abdomen (64%). Majority of deaths occurred during first 36hrs. Compared with patients who survived, those who died had

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 4.5 hours. Also mean time gap between tablet ingestion and appearance of symptoms was 60 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 2 (1-4) days.

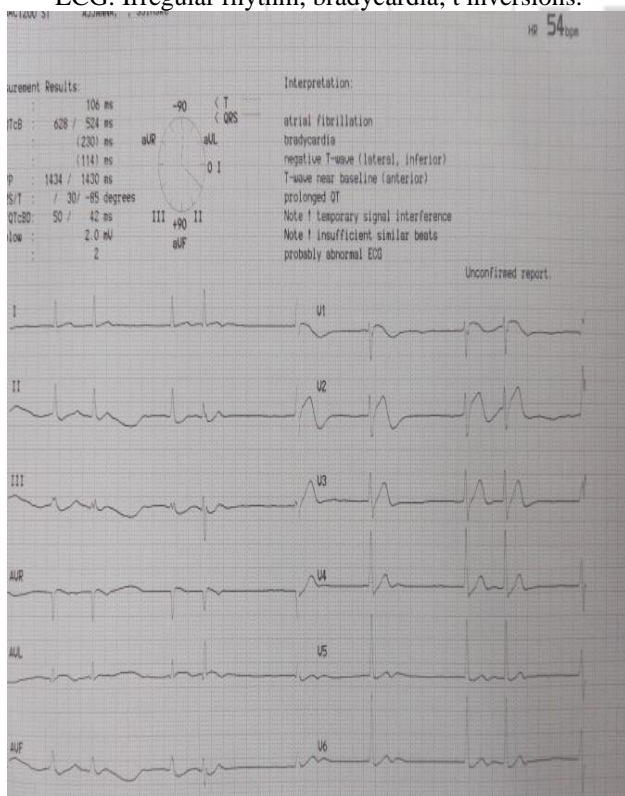
Table 1: Demographic characteristics of patients (n = 11)

Male sex (%)	80
Female sex (%)	30
Mean age (years)	29.2
Dose of AIP, Mean	3.3
Length of hospital stay (days), Mean	02
Duration of onset of symptoms (min), Mean	60
Intention of poisoning (%) -Accidental	-
Homicidal	100

Table 2: Clinical manifestations of patients (n=11)

Variable	No (%)
Nausea & Vomiting	11 (100)
Metabolic acidosis	9 (82)
Hemodynamic instability	9 (82)
Cardiac arrhythmias	8 (72)
Epigastric pain	7 (64)
Liverdamage	6 (54)
Seizures	0 (0)

ECG: Irregular rhythm, bradycardia, t inversions.



ABG: Metabolic acidosis with respiratory alkalosis

Blood Gas Values		
↓ pH	6.954	[7.350 - 7.450]
↓ pCO ₂	20.5 mmHg	[32.0 - 48.0]
↑ pO ₂	229 mmHg	[83.0 - 108]
Oximetry Values		
↓ ctHb	11.0 g/dL	[12.0 - 17.5]
sO ₂	98.6 %	[95.0 - 99.0]
Electrolyte Values		
↓ cNa ⁺	132 meq/L	[136 - 146]
↓ cK ⁺	3.2 meq/L	[3.4 - 4.5]
↓ cCa ²⁺	1.95 mg/dL	[4.10 - 4.90]
cCl ⁻	103 meq/L	[98 - 106]
Metabolite Values		
↑ cLac	15 meq/L	[0.5 - 1.6]
Calculated Values		
cBase(B) _c	-26.7 mmol/L	
cBase(Ecf) _c	-25.2 mmol/L	
cHCO ₃ ⁻ (P, st) _c	6.3 mmol/L	
cHCO ₃ ⁻ (P) _c	4.3 mmol/L	
ctCO ₂ (P) _c	11.1 Vol%	
ctO ₂ e	15.6 Vol%	
ABE _c	-26.7 mmol/L	
SBE _c	-25.2 mmol/L	
Notes		
↑	Value(s) above reference range	
↓	Value(s) below reference range	
c	Calculated value(s)	
e	Estimated value(s)	

4. Discussion

AIP is a easily available, low-cost highly-toxic rodenticide. Aluminium phosphide is used as a grain preservative, particularly for wheat. AIP is a lethal compound. Its lethal characteristic is attributed to the fact that in the presence of moisture, it releases phosphine gas absorbed easily by inhalation, ingestion or through dermal contact (7). Although the exact mechanism of phosphine toxicity is not clearly known, low oxygenation and consequently the failure of cellular respiration (non competitive inhibition of cytochrome oxidase of mitochondria) and formation of highly reactive hydroxyl radicles has been identified as the culprit (8). Phosphine gas has systemic toxic effects on heart, liver, kidney, lung with manifestations of severe intractable shock, cardiac arrhythmias, acidosis and pulmonary oedema (8). Cardiovascular manifestations include profound hypotension, arrhythmias, congestive heart failure, blocks, myocardial injury (9). Most cases of AIP poisoning were suicidal in our study; a finding which is similar to many relevant studies (10-11). Various factors such as family conflicts, drug and alcohol abuse, emotional distress, depression, medical diseases, social isolation, and financial and work-related problems may contribute to suicidal acts (12).

In the present study, we prospectively assessed 11 patients with AIP poisoning. Most of the patients had metabolic acidosis, hemodynamic instability, nausea and vomiting. Nine patients died and these victims had lower pH levels, hemodynamic instability and ingested higher amounts of AIP tablets in comparison to subjects who survived.

Fatal dose of AIP has been reported to be in the range of 150-500 mg/70 Kg (13). The deceased patients in our study had ingested over 1 g AIP.

Louriz et al. in a study on 49 patients demonstrated hepatic dysfunction as a risk factor of poor prognosis (10). Likewise, we found that 6 of the deceased cases had impaired liver function tests (LFT), and survived cases did not develop such impairments. ECG changes, blood bicarbonate level and PH have related with outcome in ALP poisoning in study of Shadnia (14).

There is no specific antidote, treatment is mainly supportive. Few reports showed beneficial effects of coconut oil to prevent absorption of phosphine gas from gut (15).

5. Conclusion

ALP is easily available and used as a toxic compound for suicide in Asia region. There has been no effective antidote available for treatment of AIP poisoned patients. AIP poisoning is an untoward condition needs steps such as limited sells, public awareness rising about its dangers, and doctors training about patient pre hospital and emergency management to combat its occurrence. Intensive hemodynamic monitoring, supportive measures, symptomatic treatment has been proposed for management of patients.

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