

Case Report

Aluminium phosphide induced pancreatitis

Mir Nadeem^{1*}, Vishal Phogat¹, Mir Waseem², Tabinda Ayub Shah³

¹Department of Medicine, SGT Medical College and Research Centre, Gurgaon, Haryana, India

²Department of Medicine, GMC Baramulla, Jammu and Kashmir, India

³Department of medicine GMC Srinagar, Jammu and Kashmir india

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*Correspondence:

Dr. Mir Nadeem,

E-mail: mirnadeem44@gmail.com

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ABSTRACT

Aluminium Phosphide (AIP) is a commonly used agricultural pesticide. It is cheap, effective, and easily available. Aluminium Phosphide is used as a rodenticide, insecticide, and fumigant to preserve stored cereal grains, also known as "Wheat pills". In Iran, it is known as the rice tablet. There have been frequent incidents of accidental or intentional deaths. There have been only a few case reports on aluminum phosphide-induced pancreatitis in the literature available. In this report, we present the case of a young man who developed acute pancreatitis following ingestion of aluminum phosphide pellet in the absence of the usual risk factors and after exclusion of other possible causes of pancreatitis. 35-year-old male came to the ER of SGT Hospital, Gurugram, one hour after ingestion of a single 3 g tablet of Aluminium Phosphide (Celphos) at home, with a suicidal intent. He had three episodes of Vomiting on the way to the hospital. On Day 1 of admission, USG abdomen showed heterogeneity of head and the body of pancreas with minimal peri-pancreatic fluid, suggestive of Pancreatitis. Serum Amylase and lipase levels were raised throughout the hospital course. CT images were suggestive of pancreatitis. The signs and symptoms of Acute AIP Poisoning are non-specific, dose dependent and evolve with time. After ingestion, toxic features usually develop within a few minutes. The major lethal consequence of AIP ingestion is profound circulatory collapse, secondary to direct effects of toxins on cardiomyocytes, fluid loss, and adrenal gland damage. Our patient was diagnosed with acute pancreatitis in first 24 hours of admission with high suspicion of pancreatitis and managed well with iv fluids and supportive treatment and was discharged after 3 weeks of in hospital stay.

Keywords: Aluminium phosphide, Pancreatitis, Sepsis, Shock

INTRODUCTION

Aluminium Phosphide (AIP) is a commonly used agricultural pesticide. It is cheap, effective, and easily available.

Aluminium Phosphide is used as a rodenticide, insecticide, and fumigant to pre-serve stored cereal grains, also known as Wheat pills. In Iran it is known as the rice tablet.¹ There have been frequent incidents of

accidental or intentional deaths. Since the first available report of AIP poisoning in 1980s from India, it is now one of the most common and lethal poisonings with no available antidote.

Highly poisonous, aluminium phosphide has been used for suicide.² It has been reported to be the most common cause of suicidal deaths in North India.^{3,4}

The fatal dose has been reported as 0.5 g for a 70 kg adult with a mean time interval between poisoning and death

being 3 hours, with a range of 1-48 hours.⁵ The mortality rates from acute AIP poisoning (AAIPP) vary from 40 to 80 percent. The actual numbers of cases may be much larger, as less than five percent of those with AAIPP eventually reach a tertiary care center. Death results from profound shock, myocarditis, and multi-organ failure.⁶

The toxic effects of the AIP are due to deadly phosphine gas liberated when it reacts with water or hydrochloric acid in the stomach.⁷ Phosphine is a cytotoxic compound that causes free radical mediated injury, inhibits vital cellular enzymes and is directly corrosive to tissues.

There have been only a few case reports on aluminum phosphide-induced pancreatitis in the literature available. In this report, authors present the case of a young man who developed acute pancreatitis following ingestion of aluminum phosphide pellet in the absence of the usual risk factors and after exclusion of other possible causes of pancreatitis.

CASE REPORT

Patients with 35 year old male came to the ER of SGT Hospital, Gurugram, one hour after ingestion of a single 3 g tablet of Aluminium Phosphide (Celphos) at home, with a suicidal intent. He had three episodes of Vomiting on the way to the hospital. Pt was conscious, cooperative, and well-oriented to time, place, and person.

HR was regular at 118 bpm, BP 112/80 mmHg, and systemic examination was unremarkable. RT was inserted and Gastric lavage was performed with 2L of NS, pt was stabilized and managed in ICU with I.V. fluids, Magnesium Sulfate, Thiamine, Trimetazidine, and PPIs.

At the time of admission, his serum electrolytes were normal except for potassium which was 3.2mg/dl. ECG, CBC, Coagulation profile, Urine routine exam, Kidney functions, and LFTs were normal except for a slightly raised ALT at 44 IU/L.

On Day 1 of admission, USG abdomen showed heterogeneity of head and the body of pancreas with minimal peri-pancreatic fluid, suggestive of Pancreatitis. Serum Amylase and lipase levels were raised throughout the hospital course. CT images were as shown below:

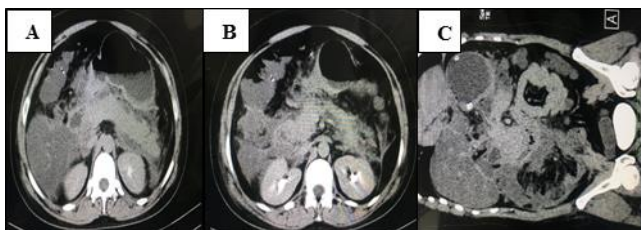


Figure 1 : CT images.

Table 1: Lab parameters of patient admitted with aluminium phosphide poisoning.

Labs	At admission	At discharge
Hb (g/dL)	16.4	16.0
TLC (/cmm)	7600	5500
PC (lac/cmm)	2.45	2.00
MCV (fL)	89.4	89.0
Hct/PCV (%)	47.2	47.0
MCHC (%)	34.7	33.8
MCH (pg)	31.1	31.2
Amylase (U/L)	1300	50
Lipase (U/L)	890	28
Urea (mg/dL)	12.5	13
S. creatinine (mg/dL)	0.9	0.89
Uric acid (mg/dL)	6.8	6.2
Total protein (g/dL)	7.2	6.9
S. albumin (g/dL)	4	4.1
S. globulin (g/dL)	3.2	2.8
Total calcium (mg/dL)	9.3	9.5
S. Sodium (mmol/L)	137	140
S. Potassium (mmol/L)	3.2	3.5
Total bilirubin (mg/dL)	0.8	0.8
Direct bilirubin (mg/dL)	0.2	0.2
AST(IU/L)	15	25
ALT(IU/L)	44	31
ALP(IU/L)	99.3	77

DISCUSSION

The signs and symptoms of Acute AIP Poisoning are non-specific, dose dependent and evolve with time. After ingestion, toxic features usually develop within a few minutes. The major lethal consequence of AIP ingestion is profound circulatory collapse, secondary to direct effects of tox-ins on cardiomyocytes, fluid loss, and adrenal gland damage. The dominant clinical feature is severe hypotension refractory to dopamine therapy. Other features may include dizziness, fatigue, tightness in the chest, headache, nausea, vomiting, diarrhea, ataxia, numbness, paraesthesia, tremor, muscle weakness, diplopia, and jaundice. If severe inhalation occurs, the patient may develop ARDS, heart failure, arrhythmias, convulsions, and coma.³ AIP can rarely induce complications including hepatitis, acute tubular necrosis, gastroduodenitis, bleeding diathesis, corrosive like esophageal stricture and intravascular. The diagnosis of AAIPP usually depends on the clinical suspicion or history. Garlic like odor is present at mouth and nostrils. Gastric lavage turns black in presence of silver nitrate in acute AIP poisoning. The management of AAIPP remains purely supportive because no specific antidote exists. After ingestion, removal of unabsorbed poison from the gut can be effective, especially if administered within 1-2 hours. Potassium per-manganate (1:10,000) gastric lavage can decompose the toxin. Administration of H2

receptor antagonists has been recommended after AIP ingestion to reduce the gastric acidity and prevent further liberation of phosphine gas. Administration of sorbitol solution as a cathartic and vegetable oils and liquid paraffin as inhibitor of phosphine release has been suggested. It was reported that coconut oil has a role in managing acute AIP poisoning even 6 h post ingestion.⁶ Correction of metabolic acidosis is a cornerstone of treatment. Hypomagnesemia is a common outcome of acute poisoning of phosphine gas exposure, but magnesium sulfate use did not improve survival in controlled clinical trials. Oral administration of the anti-ischemic drug trimetazidine decreases the production of oxygen-derived free radicals.

All patients of severe AIP poisoning require continuous invasive hemodynamic monitoring and early resuscitation with fluids and vasoactive agents. The average time interval between ingestion of AIP and death is three hours (1-48 hours), 95% of the patients die within 24 hours and the commonest cause of death in this group is cardiac dysrhythmias. In this case patient was managed well with iv fluids and supportive treatment and was discharged after 3 weeks of in hospital stay.

CONCLUSION

Pancreatitis is a very rare complication of aluminium phosphide poisoning and needs early recognition and proper treatment to have a favorable outcome.

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