ORIGINAL ARTICLE

Polyserositis: An Unusual Complication of Aluminum Phosphide Poisoning

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Abstract

Background: Aluminum phosphide is the common cause of poisoning in adults in India, with a very high case fatality ratio. We studied five patients of aluminum phosphide poisoning with polyserositis.
Methods: We enrolled all patients with aluminum phosphide poisoning presenting to emergency medical department, at a tertiary care hospital in northwestern India from January to July 2006. These patients were managed according to a standard treatment protocol and their complications were recorded.
Results: During the study period, total of 35 patients were admitted with 57.5% mortality in the first 12 hours. Among the rest, 5 patients were found to develop polyserositis. All these patients had severe hypotension at presentation and developed respiratory distress requiring mechanical ventilation after an average stay of 3.8 days post-ingestion. They were managed conservatively and four of them were discharged from the hospital after the average stay of 10 days.
Conclusion: In this case series, features of polyserositis (pleural effusion, ascites and pericardial effusion) were found in 15% patients of severe aluminum phosphide poisoning. We postulate systemic capillary leak syndrome, secondary to mitochondrial damage in the endothelium, as a possible mechanism.

Keywords: Systemic capillary leak syndrome; Endothelial dysfunction; Phosphine gas; Aluminum phosphide; Polyserositis

INTRODUCTION

Aluminum Phosphide (AlP) poisoning is commonly used as an agent of self-harm in northwestern India (1). Its use as a pesticide has increased tremendously in the last decade due to increasing need of storing the grain. Various physical and chemical properties, such as low persistence and rapid action has made AlP as the agent of choice (1,2). The highest incidence of AlP poisoning is seen among males and in the age group of 14 to 40 years (2). Over the past 15 years, the frequency of AlP poisoning cases has increased with high case fatality ratio due to its easy availability (1).

AlP poisoning manifests with nausea, vomiting, hypotension and metabolic acidosis. Common complications are cardiac arrhythmia, acute renal failure, and hemorrhage with disseminated intravascular coagulopathy (3). Moreover, pulmonary complications including pulmonary edema in the form of acute respiratory distress syndrome (ARDS) (4,5), and pleural effusion has also been reported (6,7).

We recently managed five adult patients of acute AlP poisoning who developed concurrently bilateral pleural effusion, ascites and pericardial effusion. This is an unusual complication and there are few available reports regarding this context (7).

METHODS

We studied all the patients of aluminum phosphide poisoning presenting to emergency medical department, at a tertiary care hospital in northwestern India from January to July 2006. All patients were managed according to a standard treatment protocol. Patients who developed polyserositis were included and their complications were recorded. The diagnosis of polyserositis was suspected when patients started developing respiratory distress. The diagnosis was confirmed on radiological evidence of pleural, pericardial and peritoneal fluid.

RESULTS

During the study period a total of 35 patients were admitted with 57.5% mortality in the first 12 hours. Fifteen patients out of 35 survived for more than 12 hours. Five patients out of 35 patients (14.7%) developed polyserositis. Out of these five patients three were male. Their mean age was 27.6 years, (range 23-32 years). All of them had ingested fresh tablets (unexposed to the air) and the average dose was 3.6 grams. The time interval between onset of initial symptoms after ingestion ranged from 30 minutes to 3 hours (median: 86 minutes) (Table 1). All these patients had

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Table 1. Demographic profile of five patients with polyserositis following aluminum phosphide ingestion.

Cases	Age	Sex	Dose	Onset of manifestations	Nausea/ vomiting	Palpitation	Dyspnea	Altered mental status
1	23	F	1.5 g (0.5 tab)	30 min.	+	+	-	-
2	32	M	9.0 g (3 tabs)	<1 hr.	+	+	-	+
3	27	F	1.5 g (0.5 tab)	<1 hr.	+	+	+	-
4	26	M	3.0 g (1 tab)	<3 hr.	+	+	-	-
5	30	M	3.0 g (1 tab)	<1 hr.	+	+	-	-

M; Male, F; Female

multiple episodes of vomiting and were in shock at the time of presentation. All except one had no respiratory distress at presentation and developed respiratory distress after 12 hours of admission. One patient was irritable and confused at the time of admission but was not hypoxic (Table 2). All 35 patients were managed initially with a standard treatment protocol including gastric decontamination, intravenous fluids, and inotropic support with dopamine and noradrenalin. 20 patients died within first 12 hours after admission and out of the rest, 14 patients could be discharged while only a patient died on the 3rd day post-ingestion. The baseline clinical and paraclinical parameters of all cases with polyserosits are described in the table 3.

During admission (median: 3.8 days post-ingestion), five patients developed respiratory distress requiring endotracheal intubation and mechanical ventilation. The mean duration of ventilatory support for these patients was 3.4 days. All these patients had bilateral pleural effusion (median: 3.6 days post-ingestion) and ascites (median: 4.6 day post-ingestion). The pleural effusion was tapped and was transudative in nature. Three patients developed pericardial effusion on day 5. Ultrasonography and echocardiography was done in all 20 patients. It showed systolic dysfunction with low ejection fraction and mild pericardial effusion in three patients with polyserositis (Table 3). Aggressive management with mechanical ventilation was continued in all patients developing polyserositis and four patients could be successfully weaned from ventilator and discharged. The median period of hospitalization was 10 days.

DISCUSSION

Aluminum phosphide is a solid fumigant pesticide, commonly used as a food grain preservative, in India. It is available in 3 gram tablets and pellets. The ingredients of the tablets are aluminum phosphide and ammonium carbonate (commonly marketed in India as Celphos and Quickphos tablets). These compounds are cheap, easily available, and effective grain fumigants with little residue. These properties made them ideal for poisoning. Use of AlP products has tremendously increased over last decade due to the increasing need of grain storage (1).

Following exposure to the air and moisture, this compound generates a highly toxic phosphine gas, which dissipate very rapidly into the air and leaves very little residue on the food grains. The chemical reaction of this compound will be enhanced in the presence of acidic medium, so the liberation of this gas increases due to the presence of hydrochloric acid in the stomach (ALP +3H2O → Al(OH)3 + PH3) (2,8). The phosphine gas, after absorption through the mucosal layer, enters into blood circulation and causes immediate both local and systemic manifestations. Consequently, extensive complications with cardiopulmonary instability may occur which lead to high mortality ranging from 37 to 100% (1,9).

Phosphine gas can cause toxicity by ingestion (more common mode of toxicity) and inhalation route. The exact pathophysiology of the toxicity of this compound is not clear. After absorption through the gastrointestinal tract by simple diffusion, it is mainly excreted through kidneys and lungs. It has been postulated that in case of high concentration of phosphine gas in the lungs, direct alveolar damage might occur. This may produce acute respiratory distress syndrome (ARDS) like picture (5,9,10). Phosphine causes non-competitive inhibition of the cytochrome oxidase system of the mitochondria, inhibition of cellular respiration (by inhibiting oxidative phosphorylation) and inhibition of catalase. This results in damages due to free oxygen radicals and furthermore, produces a state of cellular or global hypoxia (11,12). Multi-organ failure and shock following AlP poisoning, which are seen in majority of such patients, are likely to be due to direct extensive cytotoxic oxidative damage by phosphine (12,13).

We propose that hypoxic injury causes endothelial dysfunction and cellular apoptosis which lead to increased capillary permeability and leakage of fluid into third space, a mechanism similar to systemic capillary leak syndrome (14). The increased capillary permeability will thus lead to bilateral pleural effusion, ascites with mild pericardial effusion, as were seen in our patients. Since majority of patients with severe AlP poisoning dies, the evidence of capillary leak could be under-reported. However, diffuse

Table 2. Onset of complications in the study group.

Study No.	Respiratory Distress	Pleural Effusion	Ascites	Pericardial Effusion
1	D-3	D-5	D-5	-
2	D-3	D-3	D-5	D-5
3	D-3	D-3	D-3	-
4	D-5	D-5	D-5	D-5
5	D-5	D-5	D-5	D-5

D; day of onset

Table3. Routine investigations in the study group

	Cases				
Variables	1	2	3	4	5
SBP at presentation (mmHg)	80	80	70	90	70
DBP at presentation (mmHg)	60	40	30	70	30
Hemoglobin (g/dL)	11.5	14.6	11.7	10.6	12.4
WBC count (count/L)	10600	6000	10100	7700	9000
BUN (mg/dL)	35	36	45	30	46
Serum Creatinine (mg/dL)	0.6	0.6	1.3	0.7	0.9
Total Bilirubin (mg/dL)	0.7	0.7	0.7	1.4	1.0
AST (IU/L)	21	36	42	88	74
ALT (IU/L)	27	42	60	116	80
Serum Albumin (mg/dL)	3.7	3.5	4.1	3.9	3.8
ECG	Tachycardia	Tachycardia	Nonspecific ST-T changes	Tachycardia	Atrial fibrillation
CXR	Diffuse infiltrates	Perihilar infiltrates	Pleural effusion (right)	Pleural effusion (bilateral)	Infiltrates
Ultrasonography	Ascites + Pleural effusion	Ascites + Pleural effusion	Ascites + Pleural effusion	Ascites + Pleural effusion	Ascites + Pleural effusion
Echocardiography	EF 60%	EF 45%	EF 40%	EF 50%	EF 45%

SBP; Systolic blood pressure
DBP; Diastolic blood pressure
WBC; White blood cell
BUN; Blood urea nitrogen
AST; Aspartate aminotransferase
ALT; Alanine aminotransferase
EF; Left ventricle ejection fraction
ECG; Electrocardiography
CXR; Chest X-ray

hemorrhages and edema of all the major organs have been commonly reported in autopsy studies (15,16). Correspondingly, diffuse congestion and hemorrhage of lungs, liver, spleen, kidney and heart along with evidence of free fluid in pericardial, peritoneal and pleural cavity was found in one patient who died in our study.

Pleural effusion (6,17), and coexisting ascites (7), have been reported in a few patients with severe AlP poisoning to date. Our study is the first study reporting the development of polyserositis in patients with aluminum phosphide poisoning and its correlation with mortality. Although unusual, polyserositis should be actively considered for and managed in patients with severe poisoning.

LIMITATIONS

The inability to demonstrate pathological evidence of capillary leak and endothelial damage is the major limitation of our study.

CONCLUSION

Following aluminum phosphide poisoning, apart from the usual complications such as ARDS, arrhythmia and aspiration syndrome, the possibility of polyserositis should be kept in mind especially if patients develop respiratory distress during the course of the illness.

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