SHORT COMMUNICATION

Aluminum Phosphide Poisoning: A Case Series in North Iran

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Abstract

Background: Aluminum phosphide (AlP) poisoning is one of the most life threatening emergencies. In this study, demographic characteristics, clinical profiles and outcomes of a series of patients presented with AlP poisoning in north Iran are described.

Methods: The study was a retrospective descriptive medical chart review of AlP poisoned patients who were admitted to internal ward of Imam Khomeini hospital, Sari, Iran, from July 1st 2011 to July 1st 2012. Collected data included gender, age, intention of poisoning, amount of AlP ingested, clinical manifestations at admission, therapeutic interventions, laboratory tests and outcome.

Results: During the one-year period, 8 patients which were all men with mean (SD) age of 40.5 (22.5) years were admitted with AlP (rice tablet) poisoning. The most common signs and symptoms at admission were nausea and vomiting (100%), metabolic acidosis (100%) and hemodynamic instability (87.5%). All cases were poisoned as a result of suicidal attempt leading to 5 (62.5.6%) deaths. Compared with the patients who survived, those who died had taken higher doses of AlP, developed hepatic dysfunction in higher rates and had severer metabolic acidosis. All patients were admitted to intensive care unit and received gastric washing with sodium bicarbonate, followed by activated charcoal therapy and intravenous calcium gluconate for decontamination purposes. The median (IQR) of length of hospital stay was 2 (1-4) days.

Conclusion: AlP is a low-cost highly-toxic rodenticide. It is easily available and used as a toxic compound for suicide in the Asia region. There has been no effective antidote available for treatment of AlP poisoned patients; thus, symptomatic management should be taken into consideration as soon as possible.

Keywords: Aluminum Phosphide; Iran; Phosphine; Poisoning

INTRODUCTION

Aluminum phosphide (AlP) 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, respectively (1-3). The rate of poisoning with these chemicals is relatively low in Iran as it has been reported to allocate 0.2% of poisoning cases (4). However, based on mortality, these compounds are the most lethal among other poisons. The mortality rate among AlP poisoned patients has been reported to be 18.6%-24% in Iran (5,6). The intention of poisoning with these compounds is usually suicidal, occasionally accidental and rarely homicidal (3). Irrespective of the sex, the incidence is higher in the rural population (7).

When phosphides are ingested or exposed to moisture, they release phosphine gas (PH3). Ingestion of metallic phosphides can induce phosphine intoxication when the solid phosphide contacts with gastric acid (1). Phosphine is a colorless, flammable and toxic gas with garlic or decaying fish like-odor (1). Inhalation is the major route of phosphine toxicity (8). Phosphine causes myocardial contractility and fluid loss which results to pulmonary edema. Hence, metabolic acidosis or mixed metabolic acidosis and Respiratory alkalosis and acute renal failure may also occur (3,8). Other reported features include disseminated intravascular coagulation, hepatic necrosis and hypo or hypermagnesemia (3,8).

In this study, demographic characteristics, clinical profiles and outcomes of a series of patients presented with AlP poisoning in north Iran are described.

METHODS

This was a retrospective descriptive study of AlP poisoned patients who were admitted to internal department of Imam Khomeini hospital, Sari, Iran during July 1st 2011 to July 1st 2012. Data were collected by reviewing medical charts. Data collected included clinical and laboratory presentations and also treatments administered to each patient. Intention of poisoning was classified as accidental and suicidal. Data were analyzed using Microsoft® Excel software (Microsoft Corp., Redmond, WA, USA). Categorical data were reported as percentage. Continuous data were reported as mean and standard deviation (SD) if they were normally distributed and as median and interquartile range (IQR) if they were non-normally distributed.

RESULTS

During the one-year period, 8 patients which were all men with mean (SD) age of 40.5 (22.5) years were admitted with AlP (rice tablet) poisoning (Table 1). The most

Table 1. Demographic characteristics of patients (n = 8)

Male sex (%)	100
Age (year); mean (SD)	40.5 (22.5)
Dose of AlP (g); mean (SD)	2.5 (1)
Length of hospital stay (day); median (IQR)	2 (1-4)
Intention of poisoning (%)	
Accidental	-
Suicidal	100

common signs and symptoms at presentation were nausea and vomiting (100%), metabolic acidosis (100%) and hemodynamic instability (87.5%) (Table 2).

All cases were poisoned as a result of suicidal attempt leading to 5 (62.5.6%) deaths. The majority of deaths (4 out of 5 cases) occurred during the first 24 hours. Compared with the patients who survived, those who died had taken higher doses of AlP (3 (1.2) g vs. 1.4 (0.5) g), developed hepatic dysfunction in higher rates (5 out of 5 cases vs. 0 out of 3 cases) and had severer metabolic acidosis (pH (median (IQR)) = 7.14 (7.10-7.33) vs. 7.25 (7.24-7.32)). All patients were admitted to intensive care unit and received gastric washing with sodium bicarbonate, followed by activated charcoal therapy and intravenous calcium gluconate for decontamination purposes. The median (IQR) of length of hospital stay was 2 (1-4) days (Table 1).

Table 2. Clinical manifestations of patients (n = 8)

Variable	No. (%)
Nausea and vomiting	8 (100)
Metabolic acidosis	8 (100)
Hemodynamic disorders	7 (87.5)
Liver damage (abnormal liver function tests)	5 (62.5)
Cardiac arrhythmia	5 (62.5)
Epigastric pain	3 (37.5)
Loss of consciousness	2 (25)

DISCUSSION

AlP is a low-cost highly-toxic rodenticide. After exposure to moisture, it releases phosphine gas absorbed easily by inhalation, ingestion or through dermal contact (9). Although the exact mechanism of phosphine toxicity is not clearly known, low oxygenation and consequently the failure of cellular respiration has been identified as the culprit (8-11).

In the present study, we prospectively assessed 8 patients with AlP poisoning. All patients had metabolic acidosis and nausea and vomiting. Five patients died and these victims had lower pH levels and ingested higher amounts of AlP tablets in comparison to subjects who survived.

In a study by Hosseinian et al. metabolic acidosis was observed in 42% of patients and similar to our study nausea and vomiting were their most common findings (5). In consistent with our series that gastrointestinal symptoms, metabolic acidosis and hemodynamic disturbances were found as the commonest manifestations, in other studies gastrointestinal symptoms and shock were introduced as the most frequent (3,10).

Fatal dose of AlP has been reported to be in the range of 150-500 mg/70 Kg (11). The deceased patients in our study had ingested over 1 g AlP. Moreover, similar to the study by Hosseinian et al. those with lower pH were more likely to death (5).

Louriz et al. in a study on 49 patients demonstrated hepatic dysfunction as a risk factor of poor prognosis (12). Likewise, we found that all deceased cases had impaired liver function tests (LFT), and survived cases did not develop such impairments.

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 AlP poisoned patients; thus, symptomatic managements should be taken into consideration as soon as possible.

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REFERENCES

1. Hayes, WJ and Laws ER. Solvent, Fumigants and Related Compounds. In: Gehring PJ, Nolan RJ, Watanabe PG, Schumann AM, editors. Handbook of Pesticide Toxicology. New York: Academic Press; 1991. p. 285-300.
2. Goel A, Aggarwal P. Pesticide poisoning. Natl Med J India 2007 Jul-Aug;20(4):182-91.
3. Bumbrah GS, Krishan K, Kanchan T, Sharma M, Sodhi GS. Phosphide poisoning: a review of literature. Forensic Sci Int 2012 Jan 10;214(1-3):1-6.
4. Afshari R, Majdzadeh R, Balali-Mood M. Pattern of acute poisonings in Mashhad, Iran 1993-2000. J Toxicol Clin Toxicol 2004;42(7):965-75.
5. Hosseinian A, Pakravan N, Rafiei A, Feyzbakhsh SM. Aluminum phosphide poisoning known as rice tablet: A common toxicity in North Iran. Indian J Med Sci 2011 Apr;65(4):143-50.
6. Soltaninejad K, Nelson LS, Bahreini SA, Shadnia S. Fatal

aluminum phosphide poisoning in Tehran-Iran from 2007 to 2010. Indian J Med Sci 2012 Mar-Apr;66(3-4):66-70.

7. Chugh SN, Dushyant, Ram S, Arora B, Malhotra KC. Incidence & outcome of aluminium phosphide poisoning in a hospital study. Indian J Med Res 1991 Jun;94:232-5.

8. Proudfoot AT. Aluminium and zinc phosphide poisoning. Clin Toxicol (Phila) 2009 Feb;47(2):89-100.

9. Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. Emerg Med J 2006;23(1):e3.

10. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arh Hig Rada Toksikol 2012 Mar;63(1):61-73.

11. Moghadamnia AA. An update on toxicology of aluminum phosphide. Daru 2012 Sep 3;20(1):25.

12. Louriz M, Dendane T, Abidi K, Madani N, Abouqal R, Zeggwagh AA. Prognostic factors of acute aluminum phosphide poisoning. Indian J Med Sci 2009 Jun;63(6):227-34.