Available online at www.medicinescince.org

CASE REPORT

Medicine Science 2016;5(Supp):143-5

Medicine Science International Medical Journal

A race against time in lethal aluminum phosphide intoxication: a case report

Ismail Altintop, Mahmut Firat Kaynak

Department of Emergency Medicine, Kayseri Training and Research hospital, Kayseri, Turkey

Received 23 February 2016; Accepted 04 April 2016
Available online 8 Apr 2016 with doi: 10.5455/medscience.2016.05.8457

Abstract

Aluminum phosphide (AP) is a condition that results in death in case of use for suicide purpose. The phosphine gas, which is released as a result of aluminum phosphide intake, has high level of toxicity. It leads to cellular level hypoxia and inhibition of oxidative phosphorylation. Aluminum phosphide intoxication may cause metabolic acidosis, arrhythmia, acute respiratory distress syndrome, and shock. High-dose intakes may lead to death within hours. 22 year-old female patient was taken to our emergency service because of the suicide attempt with 6000mg aluminum phosphide tablet. We aimed to share our experience with our aluminum phosphide intoxication, which has no known antidote and leads to rapid death in follow-up. We aim to present our experiences with our case of intoxication from aluminum phosphide, which causes high mortality after the intake. 22 year-old female patient was transferred to our hospital due to suicide attempt with 6000mg aluminum phosphide tablet. The patients have no known comorbidities, medicine use or allergic history. We believe that, in preventing the high mortality rate in aluminum phosphide intoxication, metabolic acidosis, arrhythmia, ARDS and measures against the shock and the hemodialysis in early period should be performed. Although its efficiency has not been clearly proved and its use has not entered into algorithms, the use of $MgSO_4$ and N-Acetyl-Cysteine in supportive care is an option for such cases. Further studies and proven treatment methods are needed in AP toxicity.

Keywords: Alüminyum fosfat, zehirlenme, acil servis, magnezyum sülfat

Introduction

Aluminum phosphide is used as disinfectant against the insects damaging the stocked cereals. Aluminum phosphide, which is produced in solid form, is stocked in form of large tablets or in plastic-coated small tablets like drugs. In case of contact with moisture, the phosphine gas is released. Once it is swallowed, these metal phosphide compounds are combined with hydrochloric acid (HCl) in stomach and creates toxic phosphine gas ($AlP + 3\ H+ \rightarrow Al^{3+} + PH_3$) [1,2]. The released phosphine mixes into the blood through stomach and bowels, and is then transported to the liver. It is retained by the lungs. Within short time after the formation of phosphine gas, the death occurs [3]. For humans, the toxic dose is 20 mg/kg, while lethal dose is 50 mg/kg [4]. Most of the deaths, which occur generally due to cardiovascular toxicity, occur within first 12-24 hours [5]. In case of intoxication, main organs affected are heart, lungs, kidneys, and gastrointestinal system. Phosphine inhibits the cytochrome C oxidase [6]. It causes cellular level hypoxia and inhibition of oxidative phosphorylation [7]. At the same time, it also leads to the formation of reactive oxygen species caused from lipid peroxidation [6]. These effects may cause changes in transmembrane action potentials in heart. The effects of phosphine gas on heart are multifactorial and the most common mortality reason is the cardiogenic shock [4].

We aimed to present our experiences with our case of intoxication from aluminum phosphide, which leads to high mortality after the intake.

Case Presentation

22 year-old female patient was transferred to our hospital due to suicide attempt with 6000mg aluminum phosphide tablet. The patients had no known comorbidities, medicine use or allergic history. In physical examination of the patient 5 hours after the intake of tablet, the Glasgow coma score was 14 and she was inclined to sleep. Vital parameters were as follows; TA: 88/59 mmHg, KTA: 102/minute (min), SpO_2: 94%, Temp.: 36.40 C. For the patient taken into resuscitation unit of emergency service, the treatment components

***Corresponding Author:** Ismail Altintop, Department of Emergency Medicine, Kayseri Training and Research hospital, Kayseri, Turkey
E-mail: draltintop1@hotmail.com

implemented were isotonic saline 200 mL hour-1, dopamine 10 mcg kg-1 min-1 and steradine 0.1 mcg kg-1 min-1. Stomach lavage was applied. Due to the development of respiratory distress and loss of consciousness, the patient was intubated and then connected to mechanic ventilation. In artery blood gas analyses, the metabolic acidosis of the patient was found to be (pH: 7.14, PO_2: 59 mmHg, PCO_2: 17 mmHg, HCO_3: 12.5 mEq/L, SpO_2: 99%) and sodium bicarbonate treatment was implemented. In biochemical examination of the patient, the blood glucose level was found to be 237 mg/dl, while no other anomaly was detected in hemogram and biochemical examinations. Except the sinusal tachycardia at the level of 110/min, no other characteristic was determined in ECG. The hemodialysis treatment was planned but, due to the cardiac arrest development, sinus rhythm of the patient was ensured by implementation of cardiopulmonary resuscitation for 5 minutes. In addition to the supportive care, $MgSO_4$ treatment was planned for the patient. The treatment was planned and started with 4g $MgSO_4$ initial dose, 2g 1 hour later, and 1g every 3 hours. Within approximately 1 hour, upon the development of cardiac arrest again, the cardiopulmonary resuscitation was implemented. No response was obtained after the implementation of cardiopulmonary resuscitation for approximately 1 hour. During this period, ventricular fibrillation was observed for once, and then defibrillated. Patients could not be recovered despite all the interventions. The patient was considered as exitus.

Discussion

Agricultural pesticide intoxications exhibit seasonal characteristics in our country, as in entire world, but they constitute the intoxication cases that are most frequently observed in emergency services [8]. In rural areas, where the sociocultural level is low, the intoxication cases due to suicide or accidentally intake may occur [1,8]. In a study, it has been observed that 68% of a total of 559 acute intoxication cases in India was with AF. In AF intoxication, the clinic findings are severe vomiting attacks, resistant hypertension, and metabolic acidosis. Through mitochondrial cytochrome C inhibition, it leads to cardiac and pulmonary toxicity [4,9]. Cardiac depression causes resistant hypertension. In a study on rats, it has been shown that N-acetyl cysteine decreased the myocardial oxidative damage. Although hypo/hypermagnesemia may be seen after AP, its pathogenesis has not been clearly detected. Although they are not very common, in AP intoxication, intravascular hemolysis, acute adrenocortical deficiency, hepatitis, acute tubular necrosis, pancreatitis, hypo-hyperglycemia, meth-hemoglobinemia, micro-angiopathic anemia, and disseminated intravascular hemolysis may develop. Again, in surviving cases, esophageal structure and trachea-esophageal fistula development are reported in following days. There are also studies reporting 50% decrease in mortality in AF intoxications upon the use of magnesium sulfate in treatment [8,10]. It is thought to have reducing effect on mortality due to its antioxidant effect on free oxygen radicals caused from AF and its reducing effect on fatal arrhythmia. In different studies, the use of magnesium sulfate is recommended at different doses. The detection of hyper-glycaemia was considered as bad prognostic factor in application of the patient [11]. It is thought that the treatment of hyperglycemia contributed to the prognosis of patient [6].

The most common clinic presentation of the intoxication is the vomiting-nausea and dopamine-resistant hypotension [11]. Moreover, due to the damage in myocardium and liver, hypermagnesemia is observed [8,12]. Majority of deaths occurs within first 24 hours. The blood pressure of the patient, who has been taken to our hospital 5 hours after the intake of aluminum phosphide, was low and her clinic was in accord with aluminum phosphide intoxication. Despite the vasopressor treatment, cardiac arrest developed in patient. We believe that cardiac arrest was caused from negative effects of phosphine gas on cardiac transmission system and myocardial metabolism.

Shadnia et al. [13], in their study, have reported the ST segment elevation in electrocardiograms of patients due to phosphine gas' effects in aluminum phosphide intoxications [13].

In aluminum phosphide intoxication, the treatment is symptomatic and there is no antidote. In a study of Vijay Kumar Agrawal et al. [7] on 7 cases, only 4 individuals could survive. Besides the symptomatic treatment after the intake of aluminum phosphide, also the dialysis is recommended for early period [7]. In addition to the supportive care, N-acetyl-cysteine and glutathione and magnesium, which have been reported to have antioxidant features, are recommended [15]. In rats exposed to aluminum phosphide, N-acetyl-cysteine decreased the oxidative damage on myocardium and increased the survival duration [14,15]. In addition to that, the change created by cardio-protective trimetazidin on myocyte metabolism may have a potential role by decreasing the oxygen consumption.

Conclusion

We believe that, in preventing the high mortality rate in aluminum phosphide intoxication, the metabolic acidosis, arrhythmia, ARDS and measures against the shock and the hemodialysis in early period should be performed. Because no antidote could be found despite the high mortality

risk, the supportive care constitutes the main component of treatment in AP intoxication. On the other hand, in literature, there are positive data on the use of $MgSO_4$ and N-Acetyl-Cysteine. Although its efficiency has not been clearly proved and its use has not entered into algorithms, the use of $MgSO_4$ and N-Acetyl-Cysteine in supportive care is an option for such cases.

References

1. Jaishankar M, Tseten T, Anbalagan N, Mathew BB, Beeregowda KN. Toxicity, mechanism and health effects of some heavy metals. Interdiscip Toxicol. 2014;7(2):60-72.
2. Mirakbari SM. Proposal for a new mechanism of action for aluminum phosphide (ALP) for causing local injuries in ALP poisoning: Should treatment strategies be modified? Hum Exp Toxicol. 2015 Nov 30. pii: 0960327115619221.
3. Behera C, Krishna K, Bhardwaj DN, Rautji R, Kumar A. A case of accidental fatal aluminum phosphide poisoning involving humans and dogs. J Forensic Sci. 2015;60(3):818-21.
4. Elabbassi W, Chowdhury MA, Fachtartz AAN. Severe reversible myocardial injury associated with aluminium phosphide toxicity: A case report and review of literature. Journal of the Saudi Heart Association. King Saud University. 2014;26(4):216-21.
5. Mohan B, Gupta V, Ralhan S, Gupta D, Puri S, Wander GS, Singh B. Role of Extracorporeal Membrane Oxygenation in Aluminum Phosphide Poisoning-Induced Reversible Myocardial Dysfunction: A Novel Therapeutic Modality. J Emerg Med. 2015;49(5):651-6.
6. Siddaiah L, Adhyapak S, Jaydev S, Shetty G, Varghese K, Patil C, Iyengar S. Intra-aortic balloon pump in toxic myocarditis due to aluminum phosphide poisoning. J Med Toxicol. 2009;5(2):80-3.
7. Agrawal VK, Bansal A, Singh RK, Kumawat BL, Mahajan P. Aluminum phosphide poisoning: Possible role of supportive measures in the absence of specific antidote. Indian J Crit Care Med. 2015;19(2):109-12.
8. Bogle RG. Aluminium phosphide poisoning. Emerg Med J. 2006;23(1):e03–e03.
9. Ntelios D, Mandros C, Potolidis E, Fanourgiakis P. Aluminium phosphide-induced leukopenia BMJ Case Rep. 2013;30:2013. pii: bcr2013201229.
10. Sulaj Z, Drishti A, Çeko I, Gashi A, Vyshka G. Fatal aluminum phosphide poisonings in Tirana (Albania), 2009 - 2013. Daru. 2015;23(1):8.
11. Mehrpour O, Alfred S, Shadnia S, Keyler DE, Soltaninejad K, Chalaki N, Sedaghat M. Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor. Hum Exp Toxicol. 2008;27(7):591-5.
12. Chugh SN, Kolley T, Kakkar R, Chugh K, Sharma A. A critical evaluation of anti-peroxidant effect of intravenous magnesium in acute aluminium phosphide poisoning. Magnes Res. 1997;10(3):225-30.
13. Soltaninejad K, Nelson LS, Khodakarim N, Dadvar Z, Shadnia S. Unusual complication of aluminum phosphide poisoning: Development of hemolysis and methemoglobinemia and its successful treatment. Indian J Crit Care Med. 2011;15(2):117-9.
14. Dülger H, Durmu A, Sezgi C. KOAH Akut Atak Tedavisinde Antioksidan Olarak N-Asetilsistein'nin Etkinliği. 2002;9(3):91-4.
15. Chaudhry D, Rai AS. N-acetyl cysteine in aluminum phosphide poisoning: Myth or hope. Indian J Critical Care Med. 2014;18(10):646-7.