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CASE REPORT

Normal Acid Base Balance in aluminum phosphide poisoning: Case reports of a rare presentation

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Abstract

Introduction: Aluminum phosphide (AlP) is an effective fumigant and rodenticide which is a commonly used agent for suicide in parts of developing countries. AlP poisoning results in serious manifestations involving many vital organs and it has high mortality. Electrolyte and metabolic abnormalities including metabolic acidosis is a common problem in AlP poisoning.
Case report: Here, we report two cases of AlP poisoning who take AlP tablets intentionally. The therapeutic intervention initiated for both of them including glucose, insulin, and potassium (GIK) protocol, antioxidants agents, intravenous magnesium, intravenous calcium, and norepinephrine. The arterial blood samples obtained in a serial manner and interestingly it showed no abnormality.
Conclusion: This is first cases of AlP poisoning with normal acid base balance which emphasis the importance the early initiation of therapeutic intervention.

Keywords: Aluminum phosphide; acidosis; Poisoning; Toxicity; blood gas

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INTRODUCTION

Aluminium phosphide (AlP) is a well-known insecticide and rodenticide. It is available in Asian markets such as India and Iran. Although its use has been forbidden in Iran, it is still used to keep rice and stored grains from rodents and other household pests. Hence it is known as rice tablet [1].

AlP was found to be the most common suicidal agent which accounting the 68.4 % of total deaths due to poisoning. The rate of AlP poisonings in Iran is also significant. Of 471 AlP poisonings, 146 (31 %) were fatal between 2000 and 2007 [2].

The exact mechanism of action of AlP is still unknown. However, some initial studies on different animals showed that phosphine mainly binds cytochrome oxidase and changes the valences of the haem component of haemoglobin. It also induces oxidative stress and boosts extra-mitochondrial release of free oxygen radicals that results in lipid peroxidation and protein denaturation of the cell membrane in various organs [3-5].

AlP poisoning affects most of the organs. Early symptoms include nausea, vomiting, retrosternal and epigastric pain, dyspnoea, anxiety, agitation, and garlic breath. The early signs of fatal toxicity (90 % to 100 %) are shock and peripheral circulatory failure [6, 7].

Electrolyte and metabolic abnormalities also is common in AlP poisoning. Hypokalaemia (primary or secondary to vomiting), metabolic acidosis or mixed metabolic acidosis, respiratory alkalosis, and acute renal failure have often been reported. Metabolic acidosis may be present probably due to the accumulation of lactic acid caused by blockage of oxidative phosphorylation and poor tissue perfusion [8, 9]. Here we report two cases of AlP poisoning with normal acid base balance as a rare presentation.

CASE PRESENTATION

A 55 years old man came to emergency department of hospital with history of taking one tablet of AlP. He ingested this tablet about two hours ago intentionally. He admitted to the medical toxicology ICU ward of hospital, then supportive care and full cardiorespiratory monitoring initiated. Primary vital signs were as follows: respiratory rate (RR) 12 breaths/minute, heart rate (HR) 80 pulses/minute, blood pressure (BP) 108/74 mmHg. He had not any past medical history and the drug history also was negative. Two hours later the patient developed hypotension (89/60 mmHg) in second day and he intubated for mechanically ventilation. The laboratory results showed fasting blood glucose 112 mg/dL, creatinine 1 mg/dL, potassium 4.1 mEq/L, and magnesium 2.1 mg/dL. There was not any abnormality in liver and kidney function tests. The arterial blood gas study also showed pH 7.426, PCO2 29.3 mmHg, and bicarbonate 19.3 mmol/L. Echocardiography was performed on the

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second, fourth and eighth days of hospitalization, The results showed ejection fraction (EF%) 20-15%, 20-25% and 40%, respectively. He presented mitral regurgitation (MR+) and tricuspide regurgitation (TR+) in echocardiography.

Treatment of AlP poisoning initiated promptly including glucose, insulin, and potassium (GIK) protocol, antioxidants agents, intravenous magnesium and calcium. Norepinephrine also considered for management of hypotension shock. Arterial blood gas checked repetitively to monitor any metabolic acidosis. The results of arterial blood gases showed a normal balances interestingly. This is summarized in Figure 1. These treatments continued for next days and the blood pressure increased to the normal range after two days. Hence, the norepinephrine tapered to discontinue. After seven days, the patient extubated and no complication occurred during hospitalization in medical toxicology ICU. Finally the patients transferred to the ward and discharged from the hospital.

Case 2

A 53 years old man came to emergency department of hospital with history of taking one tablet of AlP. He ingested this tablet about one hours ago intentionally. After admission to medical toxicology ICU ward, the supportive care and full cardiorespiratory monitoring initiated. The patient had history of HTN (Hypertension).

Primary vital signs were RR 22 breaths/minute, HR 109 pulses/minute, BP 118/77 mmHg. The patient intubated for airway management and while after he developed hypotension (101/71 mmHg) such as the pervious case. The laboratory results showed fasting blood glucose 105 mg/dL, creatinine 1.2 mg/dL, potassium 3.9 mEq/L, and magnesium 2.0 mg/dL. The liver and kidney function tests were in normal ranges. The arterial blood gas study also showed pH 7.440, PCO2 35.3 mmHg, and bicarbonate 24.7 mmol/L. Echocardiography was performed on the first day and show an EF 45%, mitral regurgitation (MR+) and tricuspide regurgitation (TR+).

Management of AlP poisoning initiated with considering of GIK protocol, antioxidants agents, intravenous magnesium and calcium, norepinephrine, and hydration. Serial arterial blood samples were taken for gas study. The results showed no abnormality in blood gas parameters (Figure 1).

The clinical condition of patient improved in next days and norepinephrine discontinued gradually. Finally, the patient extubated with no more complications and he transferred to the ward. He discharged successfully after 10 days of hospitalization.

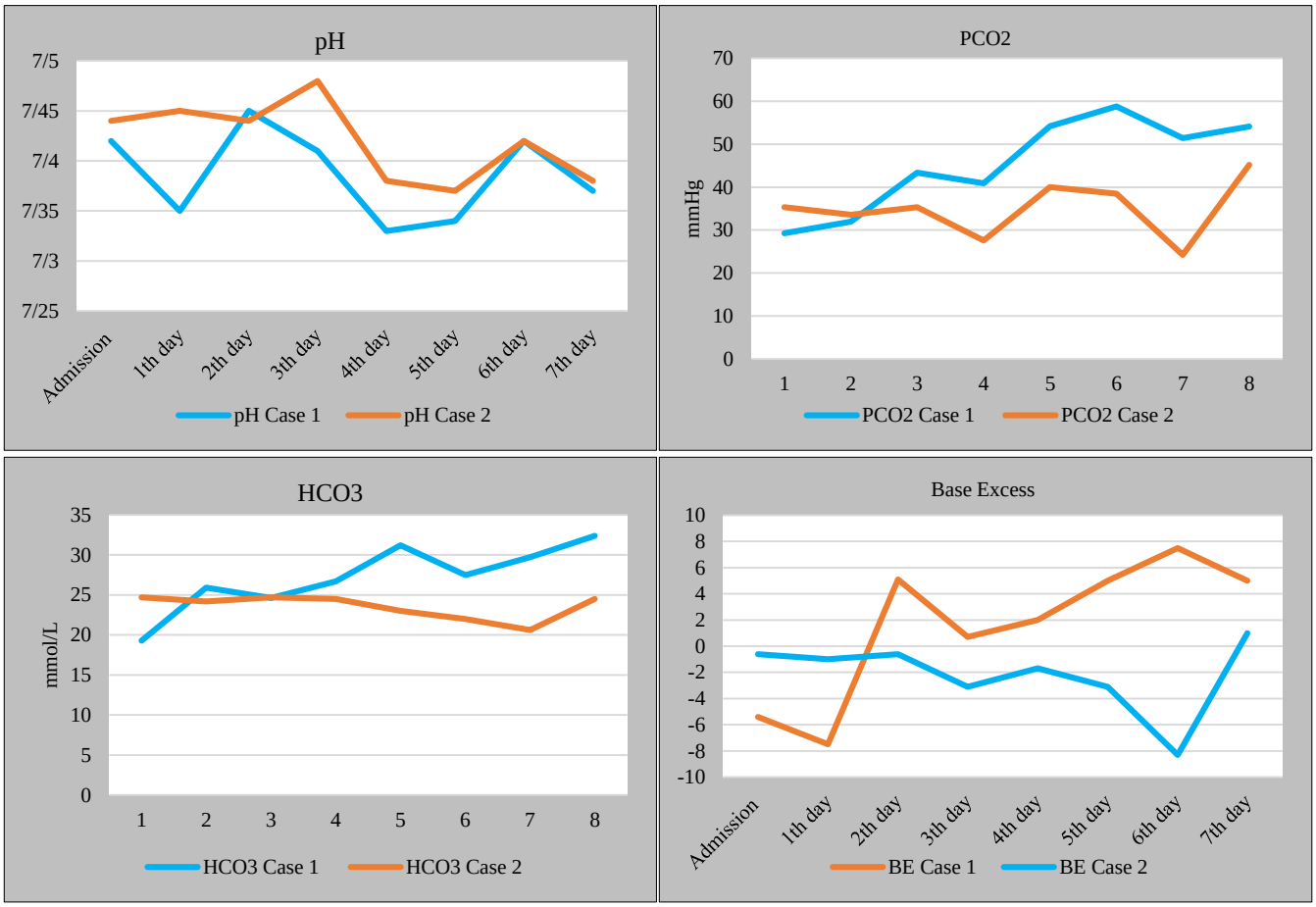

Figure 1. Results of the arterial blood gas parameters in two patients with aluminum phosphide poisoning.

DISCUSSION

Metabolic acidosis is a life threatening problem in AlP poisoning. It is probably due to the accumulation of lactic acid caused by blockage of oxidative phosphorylation and poor tissue perfusion. Severity of metabolic acidosis is also a prognostic indicator in AlP poisoning. The effect of full correction of acidosis on survival rate in aluminum phosphide poisoning is significant[10]. However, we found no abnormality in our cases in point of blood gases. These cases did not develop any metabolic acidosis which is extremely rare in the AlP poisoning. It is notable that the origin of AlP tablet should be investigated by clinicians carefully. Fake AlP tablets, with natural sources may be used by such patients and they are not dangerous itself. However, the AlP tablets which used by our cases, were not fake according to the obtained history. In addition, the hypotension episodes were associated with using of original AlP tablet. Our cases also experienced some episodes of respiratory alkalosis. This may be due to hyperventilation or respiratory infection. As our both cases experienced ventilator associated pneumonia. Some mechanism is expected for this phenomena. First, our cases came to the hospital early. It is known that early arrival, resuscitation, diagnosis, intensive monitoring and supportive therapy in AlP poisoning may result in a good outcome[11]. The management interventions also initiated promptly in both cases. Hence, blockage of oxidative phosphorylation did not occurred significantly and lactic acid did not accumulated.

In the conclusion, these cases emphasis the importance of early initiation of GIK protocol and other supportive cares to avoid development of electrolyte and blood gas abnormalities. Further reports and trials can be useful to understand the exact mechanism of this phenomena.

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REFERENCES

1. Shadnia S, Soltaninejad K. Spontaneous ignition due to intentional acute aluminum phosphide poisoning. The Journal of emergency medicine. 2011;40(2):179-81.
2. Shadnia S, Sasanian G, Allami P, Hosseini A, Ranjbar A, Amini-Shirazi N, et al. A retrospective 7-years study of aluminum phosphide poisoning in Tehran: opportunities for prevention. Human & experimental toxicology. 2009;28(4):209-13.
3. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. J Emerg Trauma Shock. 2011;4(3):378-84.
4. Chugh SN, Arora V, Sharma A, Chugh K. Free radical scavengers & lipid peroxidation in acute aluminium phosphide poisoning. The Indian journal of medical research. 1996;104:190-3.
5. Hsu CH, Chi BC, Liu MY, Li JH, Chen CJ, Chen RY. Phosphine-induced oxidative damage in rats: role of glutathione. Toxicology. 2002;179(1-2):1-8.
6. Proudfoot AT. Aluminium and zinc phosphide poisoning. Clinical toxicology (Philadelphia, Pa). 2009;47(2):89-100.
7. Singh RB, Rastogi SS, Singh DS. Cardiovascular manifestations of aluminium phosphide intoxication. The Journal of the Association of Physicians of India. 1989;37(9):590-2.
8. Easterwood L, Chaffin MK, Marsh PS, Porter B, Barr C. Phosphine intoxication following oral exposure of horses to aluminum phosphide-treated feed. Journal of the American Veterinary Medical Association. 2010;236(4):446-50.
9. Mehrpour O, Alfred S, Shadnia S, Keyler DE, Soltaninejad K, Chalaki N, et al. Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor. Human & experimental toxicology. 2008;27(7):591-5.
10. Ramadan N. Aluminum phosphide poisoning; a case of survival. Asia Pacific Journal of Medical Toxicology. 2019;8(1):28-9.
11. Ghasemi Tousi A. Successful Treatment of Aluminium Phosphide, is it possible? Asia Pacific Journal of Medical Toxicology. 2017;6(3):90-6.