Case Report

A successful management of aluminum phosphide intoxication

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Abstract

Background: Aluminum Phosphide or rice tablet is one of the most common pesticides which leads to accidental or intentional acute intoxication and finally death. In this paper, we describe a successful management of intoxication with rice tablet in a young girl.

Case Presentation: A 14-year-old girl was admitted due to consumption of rice tablet. Gastric washing with two vials of sodium bicarbonate and discharge suction was done. In the first 24 hours, the patient underwent recurrent hydration, dopamine infusion with sodium bicarbonate, calcium, magnesium and amiodarone. On the second day of admission, the patient was transferred to intensive care unit (ICU) and five days later, she was discharged without liver or renal complications.

Conclusion: Short interval between consumption of this tablet and start of the treatment and on time rescue to the patient can be some of the important factors to prevent early death in intoxication with this tablet.

Keywords: Rice tablet, Intoxication, Aluminum Phosphide, Phosphine gas.

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Aluminum Phosphide (AP) or rice tablet is one of the most common pesticides which is used to protect stored grain from rodents and other insects in agriculture especially in the northern part of the country (1, 2). This substance next to the water, water stream or gastric acid produces a non-color, fire caching, with a fish-stench like smell called phosphine (PH3) which the causes intoxication. Phosphine is a protoplasmic poison which leads to a non-competitive inhibition of cytochrome oxidase enzyme in mitochondria, electron transfer system and oxidative phosphorylation (3, 4). This gas is immediately absorbed through the lungs and its skin absorption is trace (5).

Hence, to the production of free radicals in several tissues by phosphine, those organs which need more oxygen like heart, brain, lungs, kidney and liver have more sensitivity to phosphine-related damages and this is in association with histopathological changes in those organs (3, 6). The current lethal dose of AP in an adult with the average weight of 70 kg is estimated about 500 mg (3). Because of its low price, easy access and high potential for intoxication rice tablet is used in intentional acute intoxication targeting suicide and sometimes accidentally which finally results in death (2).

Most of the intoxications occur in the third decade of life (7). An investigation between 1997 and 2000 revealed that rice tablet was the third cause of suicide in Babol, Iran (8). Diagnosis of the intoxication with the rice tablet is mainly through history taking and physical examination, but in suspicious cases, it is possible to use silver nitrate-covered sheet which becomes black adjacent to the patient's breath or gastric discharge (9, 10).

In recent years, little prowess has been performed in order to reduce production, inhibition and restriction in selling these tablets, but high prevalence of intoxications due to these agents in the north province of the country implies the importance of more serious applications for this problem.

Case presentation

A 14-year-old girl was admitted with severe nausea, vomiting and irritation in the emergency ward of Yahyanejiad Hospital, Babol, Iran. The initial vital signs included blood pressure (BP): 85/60, pulse rate (PR): 120 mmHg, respiratory rate (RR): 22 min. Her weight was 45 kilograms. The patient was conscious and irritated, but did not answer to the questions. From history taking of the patient's companion, it showed that her problem was related to her father and no other information was given about taking the special drug.

Primary management with resuscitation and hydration was started. Blood samples for blood urea nitrogen (BUN), creatinine (Cr), Na, K, blood sugar (BS), complete blood count (CBC) were taken. Arterial blood gases (ABG) was taken and the patient's companion found a metal box of AP in the patient's bag with 2 tablets in it.

The patient confessed that she swallowed half of a tablet and put the other one in water and drank it. Nasogastric tube (NGT) was performed and perfume of the discharges and the history proved the probability of rice tablet consumption. The hypothesis also consisted of the physical signs of the patient. The patient's entrance to the emergency room took about 3 to 5 minutes to know her intoxication with AP.

Primary ABG showed PH: 7.33, PCO2: 33mmHg, HCO3: 17MEq/L and PaO2: 99%. Subsequent gastric washing with two vials of sodium bicarbonate, charcoal and oral sorbitol was administrated through NGT. Foley catheter was inserted to control the intake/outtake (I/O) chart and 1 lit. normal saline (NS) with two vials of sodium bicarbonate were infused in 10-15 minutes.

The initial ECG was normal. One hour after treatment, BP was 100/60 mm Hg and ABG showed PH: 7.31, PCO2: 37mmHg, HCO3: 15mEq/L, PaO2:103. Hydration with normal saline (NS) and two vials of sodium bicarbonate per lit with the velocity of 100-150 ^{cc}/h was continued, but during the second hour, systolic BP decreased to 70 mm Hg. Infusion of 500 ^{cc} NS in 10 minutes could not make any change in BP. Hydration velocity was raised up to 200 ^{cc}/h and Dopamine(dopamine hydrochloride) was started at a dose of 5 mcg/kg/minute and was increased gradually using 5 mcg/kg/minute increments at 15 minutes interval up to 10 mcg/kg/minute. At the end of third hour, systolic BP reached to 90/60 and ABG showed PH: 7.37, PCO2: 32mmHg, HCO3: 20mEq/L.

During the fourth hour, the patient had a seizure. Diazepam 2 mg IV was injected. The patient was overwhelmed with carpopedal spasm. Blood sample was sent for Mg, Ca serum levels and treatment with magnesium (Mg) (infusion of 50gr in 24 hours for 72 hours), Ca (10 cc calcium gluconate 10%, every 12 hours for 24 hours) supplementation was started which eradicated the patient's symptoms.

In order to prevent hypokalemia, 10 meq K in each vial of sodium bicarbonate was calculated and O2 saturation was controlled with pulse oxymetry. After the sixth hour, the patient received about 12 vials of sodium bicarbonate and 300 mg of Dopamine. ABG showed PH: 7.37, PCO2; 38, HCO3: 20 and systolic BP reached to 100-110 mm Hg with dopamine.

During the seventh hour, several arrhythmias with sinus tachycardia were seen, among them were recurrent premature ventricular contraction (PVC), atrial fibrillation (AF) and sinus bradycardia (only about 3-5 minutes with HR: 50-55 and normal PR Interval) were the commonest.

In the eighth hour, the patient was severely irritated and antipsychotic drug, haloperidol 2.5 mg IV was injected. In order to manage the arrhythmia, intravenous amiodarone (150 mg over 20 minutes followed by infusion of 350 mg over 6 hours and then 450 mg for 18 hours) was given which controlled arrhythmia after 3 hours. ABG at the end of the 8th hour showed PH: 7.41, HCO3: 30. Sodium bicarbonate level was decreased but ABG at the end of the 9th hour showed a decrease in the level of sodium bicarbonate to 18.

At the end of the 16th hour, BP was 110/70 mm Hg and ABG showed PH: 7.41, PCO2: 44, HCO3: 30. Dopamine was decreased as compared beforehand. The patient received about 11 lits. NS with 22 vials of sodium bicarbonate and about 9 lits. was extracted. The patient with the GCS of 15 was transferred to the ICU. The next day, liver and coagulation tests were performed and showed a mild rise in the liver enzymes with normal coagulation tests. Inquired calcium and magnesium levels tests were ready after 48 hours and were 7.1 and 1.4, respectively. The patient was discharged after five days without any complications.

Discussion

Hypotension and metabolic acidosis are inevitable complications of intoxication with rice tablet in which their degrees depend upon the amount of consumed material and existence or inexistence of previous vomiting. For its compensation, constant infusion of isotonic serums and sodium bicarbonate is required (11). Probable acidosis directly or indirectly affects heart and leads to several kinds of arrhythmias and hypotension. Calcium and magnesium are some of the elements that their levels decreased during intoxication with AP, especially Mg which can lead to lethal arrhythmia and their appropriate replacement is so helpful. Gastric acid content increases AP to phosphine change, so primary dilution of gastric acid with sodium bicarbonate can be useful (3). Using proton pomp inhibitors (PPI) also can be helpful. Potassium permanganate solution (1:10000) causes oxidation of phosphine in stomach and its change to phosphate decreases the poisonous phosphine gas especially when administrated in the first hour of intoxication (12). Recurrent gavage of charcoal, sorbitol and mineral oil are useful in prevention from releasing of phosphine and acceleration in its extraction (11). Again, it is emphasized that the history taking and physical examination are the main elements of the diagnosis and in this case, on time presence to the patient was the main factor of our success.

Short interval between consumption of this tablet and the immediate start of treatment and on time rescue to the patient can be some of the important factors to prevent the early death in intoxication with this tablet.

References

1. Cienki JJ. Non-anticoagulant the rodenticides. In: Ford MD, Delaney KA, Ling LJ, Erikson T (eds). Clinical toxicology. 1st ed. Philadelphia: W.B. Saunders Co 2001; pp: 858.
2. Shadnia SH, Soltaninejad K, Ghaemi M, Abdollahi M. Review on Rice tablet toxicology J Babol Univ Med Sci 2005; 8: 53-63.
3. Phosphine, Poisindex, Micromedex Healthcare Series, Vol 120, Thomson Healthcare Inc. 2004.
4. Chugh SN, Aggarwal HK, Mahajan SK. Zinc phosphide intoxication symptoms: analysis of 20 cases. Int J Clin Pharmacol Ther 1998; 36: 406-7.
5. Lall SB, Peshin SS, Mitra S. Methemoglobinemia in aluminum phosphide poisoning in rats. Indian J Exp Biol 2000; 38: 95-7.
6. Arora B, Punia RS, Kalra R, Chungh SN, Arora DR. Histopathological changes in aluminum phosphide poisoning. J Indian Med Assoc 1995; 93: 380-1.
7. Rahbar Taramsari M, Orangpour R, Zarkami T, Palizkar M, Mousavian SA. Survay patients poisoned with aluminum phosphid. J Guilan Univ Med Sci 2006; 14: 42-7.
8. Moghaddamnia AA, Abdollahi M. An epidemiologic study of poisoning in northern Islamic Republic of Iran. East Mediterr Health 2002; 8: 88-94.
9. Christophers AJ, Singh S, Goddard DG. Dangerous bodies: a case of fatal aluminum phosphide poisoning. Med J Aust 2002; 176: 403.
10. Mital HS, Mehrotra TN, Dwivedi KK, Gera M. A study of aluminum phosphide poisoning with special reference to its spot diagnosis by silver nitrate test. Assoc Physicians India 1992; 40: 473-4.
11. Dart RC. Medical Toxicology. 3rd ed, Philadelphia. PA: Lippincott. Williams and Wilkins 2004; pp: 1151-4.
12. Nelson LS, Lewin NA, Howland MA, et al. Goldfrank's toxicologic emergencies, 7th ed, USA, Mc Graw Hill Co 2002; pp: 1384-5.