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Infusion of GIK (Glucose-Insulin-Potassium) for Treatment of Acute Aluminium Phosphide (Rice Tablet) Poisoning: A Case Report

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Dear Editor,

Aluminium Phosphide (AlP), commonly known as rice tablets, is an efficient rodenticide mostly used in rural populations to preserve grains (1, 2). While this substance is highly toxic, it is also easily available and used as a suicide poison, accounting for a great number of cases, including those in Iran. There is no particular antidote for this toxin; therefore, intoxication is associated with a high mortality rate, causing death in the early days of admission. Intoxication usually occurs through the ingestion of AlP, less commonly through inhalation and rarely through skin exposure. Phosphine gas is released when the rice tablet is exposed to moisture. This reaction is catalyzed in the presence of gastric hydrochloric acid. It is rapidly absorbed through the gastrointestinal tract, forming reactive free radicals through the inhibition of Cytochrome C Oxidase, which leads to cardiac, pulmonary, renal, and hepatic symptoms, including cardiac arrhythmia, shock, acidosis, and pulmonary edema (1).

The treatment for AlP toxicity is supportive; vasopressor drugs, mechanical ventilation, and therapeutic means to exclude Phosphine, including magnesium sulfate, potassium permanganate, sodium bicarbonate, coconut oil, N-acetyl cysteine, lipid emulsion, etc. These treatments are yet to be studied further to confirm their definite usefulness (1-3).

The usage of hyperinsulinemia-euglycemia or infusion of GIK (glucose-insulin-potassium) was proven beneficial, with its positive inotropic effects improving myocardial contractility in various patients suffering from calcium channel blocker and beta-blocker toxicity (1, 2). The metabolic acidosis and hyperglycemia in calcium channel blocker poisoning resemble the state of diabetic ketoacidosis. The free radicals produced due to the release of Phosphine also cause insulin resistance and a similar hyperglycemic state (2). GIK infusion was reported to have a favorable outcome when used as an additional treatment in various AlP toxicity cases (1, 2, 4, 5).

Here, we present a case of acute AlP intoxication that suffered various systemic symptoms, including metabolic acidosis, hypoxemia, and hypotension. However, with the introduction of GIK infusion as an additional treatment, he had a favorable outcome and was discharged from the hospital in good condition.

A 26-year-old Iranian male presenting symptoms of nausea and vomiting was admitted to the emergency ward of a medical center. The patient claimed to have ingested 4 rice tablets, each containing 3 grams of Aluminium Phosphide, an hour and a half prior to his reference. He also claimed to have had a past medical history of Mitral Valve Prolapse and psychiatric disorders which he did not take any medications for. There was a family history of Major Depression, which the patient's mother suffered from.

On admission, the patient had a Blood Pressure of 90/60 mmHg, a Pulse Rate of 88 beats/min, a Body Temperature of 36.5°C, a Respiratory Rate of 20 breaths/min, an Oxygen Saturation of 92%, and a Glasgow Coma Scale of 11/15. Treatment began with an intravenous infusion of 1 liter of Sodium Chloride Serum, which was repeated every 4 hours. A gavage of 60 grams of charcoal plus 60 grams of Sorbitol was given. The patient was then given 60 cc of castor oil and 2 vials of Sodium Bicarbonate (100 cc) through gavage, followed by an intravenous

infusion of 2 vials of Sodium Bicarbonate (100 cc). He was then referred to the higher facilitated Emam Khomeini Hospital in Urmia, Iran. During the one-hour dispatch to Urmia, the patient received 1 liter of Sodium Chloride Serum plus 2 vials of Sodium Bicarbonate (100 cc) and a 10 cc Potassium Chloride intravenous injection.

When the patient was received in Emam Khomeini Hospital's Emergency Department, he had on his examination a Blood Pressure of 70/30 mmHg, a Pulse Rate of 80 beats/min, a Body Temperature of 36.5°C, a Respiratory Rate of 21 breaths/min, an Oxygen Saturation of 90% and a Glasgow Coma Scale of 11/15. The patient's primary Venous Blood Gas included a pH of 7.08, a Bicarbonate (HCO_3) of 20.7 mEq/L, and a Carbon Dioxide (CO_2) of 12.30 mmHg. The patient was admitted to the Intensive Care Unit due to his unstable status and underwent mechanical ventilation.

A complete blood count showed no specific abnormalities. Sodium was reported to be 139 mEq/L; Potassium was 3.5 mEq/L; Blood Sugar was 130 mg/dL; Aspartate aminotransferase was 48 IU/L, Alanine aminotransferase was 23 IU/L, Alkaline Phosphatase was 173 IU/L, Blood Urea Nitrogen was 34 mg/dL and Creatinine was 1.16 mg/dL. Cardiac Troponin I and Creatine Kinase - Myocardial Band were within the normal limits.

Sinus Tachycardia was detected in the early Electrocardiogram. Echocardiography reported an Ejection Fraction of 40%, Mild Global Hyperkinesia, and Mild Mitral Regurgitation. Chest X-rays appeared to have no significant abnormalities.

Treatment began with a gavage of 100 cc castor oil plus 3 vials of sodium bicarbonate (150 ccs). The patient then received 2 vials of sodium bicarbonate (100 cc) intravenously. Because of low blood pressure and signs of a shocking state, the patient was given 1 liter of Sodium Chloride Serum intravenously (stat), which was repeated every 8 hours, plus 2 vials of Sodium Bicarbonate and 10 ccs of Potassium Chloride in each liter of the given serum. Infusions of norepinephrine were also started (10 - 20 micrograms per minute). The patient was also given a slow infusion of 2 grams of Magnesium Sulfate and a vial of Calcium Gluconate (both repeated every 8 hours). With regards to the prevention of gastrointestinal complications, 80 milligrams of Pantoprazole were intravenously infused (stat), which was repeated in doses of 8 milligrams per kilogram per hour. During his admission, the patient also received 3 more gavages of 50 cc castor oil every 4 hours and was given 4 gastric lavages with 60 grams of charcoal and 60 grams of Sorbitol at 4-hour intervals.

In addition to the mentioned medications, the patient then underwent the GIK protocol for the following 24 hours. An infusion of 1 liter of Dextrose Sodium Chloride Serum was given every 8 hours plus 2 vials of 50% Glucose, 3 vials of Sodium Bicarbonate, and 15 cc of Potassium Chloride. A subcutaneous injection of 10 units of Regular Insulin plus 1 vial of 50% Glucose (intravenous infusion) was given stat, followed by an intravenous infusion of 6 units of Regular Insulin every hour. The patient's blood sugar was checked every 2 hours, and potassium was checked every 6 hours. Serum glucose was maintained in the range of 150 - 250 mg/dL, and Serum Potassium levels were maintained in the range of 3.5 - 5.5 mEq/L.

The signs and symptoms of hypotension and acidosis gradually resolved. After 5 days, the patient was moved from the ICU to the General intoxication ward, from which he was discharged 3 days later in a good and satisfied condition.

Aluminum phosphide has a high mortality rate (approximately 90%), and there is no specific antidote for its intoxication (1, 4). The GIK protocol improves myocardial contractility due to its positive inotropic effects (1, 2). GIK reverses acidosis by increasing myocardial carbohydrate uptake and also possibly by increasing the metabolism of lactate (1).

In a study by Pannu et al. on 60 patients intoxicated by AlP, 30 patients received GIK protocol (insulin regular 0.1 - 0.5 IU/kg/h with glucose and potassium) and maintained their serum glucose between 150 - 200 mg/dL and serum potassium was maintained between 3.5 - 4.5 mEq/L. The survival rate was significantly higher in patients who received the GIK treatment (2).

In another study by Hassanian-Moghaddam and Zamani On 88 cases of AlP intoxication, 44 patients were given the GIK treatment (loading dose of regular insulin set at 1–3 IU/kg/h, followed by 0.2 - 0.5 IU/kg/h with glucose and potassium). Serum glucose was maintained at about 150 mg/dL, and serum potassium was in the range of 3.5 - 4 mEq/L (4).

In a similar case reported by Sedaghattalab, GIK administration (regular insulin of 1 IU/kg/h, 1 cc/kg hypertonic dextrose 50% and potassium to maintain serum glucose around 150 mg/dL and serum potassium between 3.5 and 4.5 mEq/L) on a 30-year-old Iranian woman intoxicated with AlP, proved to be highly beneficial regarding the reverse of shock and acidosis in the patient (1).

In conclusion, aluminum phosphide intoxication has a high mortality rate, and no antidote has yet been found. Here, we describe a patient who was discharged in good condition within a few days after the administration of GIK, although initially suffering from a severe hypotensive state and metabolic acidosis. As a result, the GIK protocol could be suggested as an important treatment in addition

to classical supportive therapy used for acute aluminum phosphide intoxication.

Footnotes

Authors' Contribution: B. B. conceived and designed the study. P. B. participated in designing the evaluation, collecting the clinical data, and helping revise the manuscript. K.F. re-evaluated and interpreted the clinical data and drafted and revised the manuscript. All authors read and approved the final manuscript.

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References

1. Sedaghattalab M. Treatment of critical aluminum phosphide (rice tablet) poisoning with high-dose insulin: a case report. *J Med Case Rep*. 2022;**16**(1):192. [PubMed ID: 35578361]. [PubMed Central ID: PMC9112492]. https://doi.org/10.1186/s13256-022-03425-4.
2. Pannu AK, Bhalla A, Gantala J, Sharma N, Kumar S, Dhibar DP. Glucose-insulin-potassium infusion for the treatment of acute aluminum phosphide poisoning: an open-label pilot study. *Clin Toxicol (Phila)*. 2020;**58**(10):1004–9. [PubMed ID: 32129101]. https://doi.org/10.1080/15563650.2020.1719131.
3. 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. [PubMed ID: 25722553]. [PubMed Central ID: PMC4339895]. https://doi.org/10.4103/0972-5229.151019.
4. Hassanian-Moghaddam H, Zamani N. Therapeutic role of hyperinsulinemia/euglycemia in aluminum phosphide poisoning. *Medicine (Baltimore)*. 2016;**95**(31). e4349. [PubMed ID: 27495040]. [PubMed Central ID: PMC4979794]. https://doi.org/10.1097/MD.0000000000004349.
5. Shams VS, Ojaghi HSHP, Pooya FR, Tajoddini S. One year evaluation of trauma patients' death. *J Emergency Practice Trauma*. 2015;**2**(2):31–2. https://doi.org/10.15171/jept.2016.07.