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Successful Management of Aluminum Phosphide (ALP) Poisoning in a Teaching Hospital in Shahroud: A Case Report

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Abstract

In Iran, herbal or chemical rice tablets containing ALP protect grains from pests. Deaths have been reported following both accidental and intentional exposure to aluminum phosphide. A 20-year-old woman who intentionally ingested a 3-gram ALP tablet and became poisoned is the primary focus of this case report. It outlines the successful management of a patient who had taken a rice tablet and suffered from acute cardiogenic shock, severe metabolic acidosis, and severe hypotension.

This patient survived successfully despite the higher mortality rate due to cardiac and multi-organ problems from exposure to phosphine gas, particularly the rice tablet and cellular hypoxia. In addition to daily supportive therapies, an antioxidant regimen, glucose, insulin, and potassium (GIK), and prompt intubation to improve cardiac function assisted with the recovery.

Keywords: Poisoning, Suicide attempt, Aluminum phosphide, Case report, Teaching hospital.
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Introduction

There are two types of rice tablets available in Iran: chemical and herbal. Three grams of ALP, which shields grains from rodents and insects, is present in the chemical tablets.[1] Despite the fact that can also be available in green or brown, these rice tablets are usually gray. Typically, they are round and large. ALP is a highly toxic substance that releases phosphine when exposed to moisture. Phosphine is a colorless, odorless gas, but it produces a garlic-like smell when it comes into contact with air. This gas is quickly absorbed by the lungs and intestines, leading to systemic toxic effects such as cardiac arrhythmias, shock, metabolic acidosis, and pulmonary edema. Early signs of ALP poisoning include shock and circulatory collapse.[2]

The wide availability of ALP leads to significant incidents of both intentional and unintentional poisoning within various countries, including Egypt, India, and Iran, regardless of their income levels.[3-5] Edde al.'s study indicates that prior research has shown an increase in suicide rates linked to ALP, particularly in developing nations like India, Sri Lanka, and Iran.[6] Research indicates that poisoning from rice tablets is often intentional and used for suicidal purposes.[7, 8]

A systematic review by Bagherian et al. indicates that the mortality rate of ALP poisoning in the Iranian population is approximately 27%, with a higher fatality rate observed in men compared to women.[9] The mortality associated with ALP poisoning is significantly high. It varies based on several factors, including the lack of a specific antidote, lack of standardized treatment guidelines, and the presence of poor prognostic indicators.[10]

When ALP expose to moisture, phosphine gas is released and easily absorbed through inhalation, ingestion, or skin contact. While the exact mechanism of phosphine toxicity remains unknown, reduced oxygen delivery and consequent cellular respiratory failure play a significant role.[11]

In severe cases of poisoning, patients ingest more than one-sixth of rice tablets, leading to delayed vomiting and symptoms such as shock (hypotension, tachycardia, tachypnea), severe metabolic acidosis, and further organ involvement, including the heart, brain, lungs, kidneys, and liver. Rice tablet poisoning typically affects both the heart and liver. Within the first 12 to 24 hours, patients often succumb to cardiac complications, followed by liver damage that can be fatal.[12] Cardiac-related complications from rice tablet poisoning can include decreased contractility, resistant hypotension, arrhythmias, ST-T wave changes, and conduction abnormalities.[13] Rice tablet poisoning also alters blood pressure, hemoglobin levels, platelets, and leukocytes. Monitoring these changes is essential to mitigate associated side effects and mortality.[14]

A systematic review indicated a lack of an effective antidote or standardized protocol for the treatment of acute ALP poisoning. Research interest in ALP poisoning is high, which could be caused by the high mortality rate.[15] The GIK protocol improves survival rates, increases hospital stays, and maintains the cardiovascular system in individuals with ALP poisoning.[16]

This report presents a successful management of rice tablet poisoning after 23 days. This case report aims to examine a patient with intentional ALP poisoning who received treatment in the intensive care unit (ICU) of a teaching hospital,

incorporating an antioxidant regimen, glucose-insulin-potassium (GIK) therapy, and prompt intubation to enhance cardiac function, with standard supportive care protocols.

Case Report

A 20-year-old female patient was admitted to the medical center one hour after ingesting a gray 3-gram ALP obtained from a herbal store with the purpose of suicide. Upon arrival, the patient displayed loss of consciousness and a weakened sinus pulse. She was unable to speak, had cold skin, tachycardia, and epigastric pain. Further examination confirmed that the ingested tablet was authentic and had a valid expiration date in the following year.

Blood gas analysis indicated metabolic acidosis, which worsened in subsequent tests.

The patient experienced severe hypotension that did not respond to fluid resuscitation with two liters of normal saline.

An antioxidant package, a combination of four drugs (Magnesium Sulphate, calcium gluconate, vitamin E, and NAC), was administered as adjuvant therapy, along with other supportive treatments such as fluid therapy and norepinephrine.

On the other hand, due to the prominent role of GIK in several studies for patients poisoned with ALP, insulin was also initiated for the patient considering the refractory hypotension unrespond to the supportive therapy.

To address the cardiogenic shock in the patient, intubation was executed to decrease the myocardial oxygen demand. A 6.5 mm endotracheal tube was placed utilizing a rapid sequence intubation approach. Following the initial laboratory evaluations and potassium level assessment, a GIK protocol was implemented for treatment.

The patient's potassium and blood glucose levels were continuously monitored. Initially, insulin administered at a dosage of 0.5 units per Kg per hour, which amounted to a total of 25 units per hour. Intravenous dextrose (50%) was administered at a rate of one vial per hour, adjusted according to the blood glucose levels. In response to the progression of acidosis and subsequent decline in blood pressure, the insulin dosage was titrated upwards, ultimately reaching 480 units per hour.

The patient received intravenous N-acetylcysteine at a dosage of 100 mg/kg every 8 hours for 7 days. Intravenous magnesium sulfate was given at 1 gram every 8 hours for 7 days, and calcium gluconate (10%) was provided at an initial dose of 1 gram every 8 hours for 7 days. Vitamin E was administered intramuscularly at 400 units every 12 hours for 7 days.

The patient's ECG initially demonstrated sinus tachycardia without ST-T changes; but on day five, it showed significant changes, including T-wave inversion in the precordial and lateral leads. By the Seventh day, the patient's blood pressure improved, and norepinephrine tapered off.

Table 1 depicts the key details regarding the patient's vital signs.

Table 1. Serial monitoring of patients' vital signs

Day	Glasgow Consciousness Scale (GCS)	Systolic Blood pressure (mmHg)	Diastolic Blood pressure (mmHg)	Heart rate (bpm)	Oral Temperature (˚C)
0	13	80	60	175	36.7
1	3i	75+NE	43	170	-
2	5i	80+NE	55	155	-
3	5i	80+NE	50	158	-
4	5i	85+NE	60	130	38.3
5	5i	95+NE	60	115	37.9
6	5i	98+NE	65	110	37.2
7	5i	104	75	108	-
8	5i	105	70	102	-
9	5i	108	72	98	-
10	14	95	65	104	-
11	15	105	75	92	-
19	15	100	68	80	36.6
20	15	105	72	82	-
21	15	108	70	88	-
22	15	110	74	78	-
23	15	115	76	85	-

On the tenth day, the patient developed pneumonia, which was confirmed with a positive culture for Pseudomonas. Consequently, intravenous cefepime was added to the patient's medication regimen.

Table 2 presents the serial measurement of arterial blood gas (ABG) recorded during hospital admission and over the subsequent 12 days.

Table 2. Serial measurements of arterial blood gases

Arterial Blood Gas (ABG)	Day												
	0	1	2	3	4	5	6	7	8	9	10	11	12
Arterial HCO3; Normal: 22-26 mEq/L	18.4	13.3	15.7	18.6	17.8	20.2	20	18.1	18.6	17.2	19.4	17.5	17.2
Serum CO2 Pressure; Normal: 35-45 mmHg	56.6	33.3	42.8	44.7	35.2	47.8	42.5	35.6	46.9	36.6	35.3	34.9	39.2
Arterial pH; Normal: 7.35-7.45	7.11	7.20	7.16	7.22	7.26	7.22	7.27	7.17	7.20	7.27	7.34	7.30	7.24

The International Normalized Ratio (INR) increased to 2 on the 12th day. The patient was anemic from the first day, with an Hb level of 11.5 (g/dL). The anemia worsened on the 11th day (Hb=7.8 g/dL). Leukocytosis was evident from the first day (WBC=17300) and gradually declined by the fifteenth day (WBC=9300).

The patient experienced moderate liver dysfunction, as indicated by increased liver transaminases from the day after admission (AST=167 IU/L) and (ALT=76 IU/L), which returned to normal levels by the 15th day. The patient developed rhabdomyolysis due to rice tablet poisoning, with CPK levels decreasing from 7532 (on the first day) to 180 on the fifteenth day following treatment.

Table 3 summarizes the details on echocardiograms.

Table 3. Summary of echocardiography results post-hospitalization

Day	Cardiac Echo showed
0	- Mild left ventricular enlargement - Systolic wall dysfunction with global hypokinesis - Mild right ventricle stenosis - Moderate right ventricular dysfunction - Tricuspid Annular Plane Systolic Excursion (TAPSE): 13 mm - Mild to moderate mitral stenosis - Mitral regurgitation - Pulmonary arterial pressure (PAP): 25 mmHg - No aortic stenosis or aortic insufficiency - Ejection fraction: 15-20% - No PE
2	- SE W Enlarge, - Systolic Wall Dysfunction, Global Hypokinesis, - No Left Ventricular Contractility Loss - Mild to Mild Mitral Stenosis, Tricuspid Stenosis, - Pulmonary Arterial Pressure (PAP)=30, - EF=15%, - No PE
12	- Normal Size of The Left Ventricle, and Mild Degree of Dysfunction. - Normal Size of The Right Ventricle, and Normal Function. - No Mitral Stenosis, Mild Mitral Regurgitation, No Aortic Stenosis, No Aortic Insufficiency, Mild - Tricuspid Regurgitation (Tr), TRG=18, - Pulmonary Arterial Pressure (PAP)=21 - EF=45%, - No PE
17	- Normal Size of The Left Ventricle, and Mild Degree of Dysfunction. - Normal Size of The Left Ventricle, and Normal Function, - Normal Size of The Right Ventricle, and Normal Function, - No Mitral Stenosis, No Mitral Regurgitation, No Aortic Stenosis, No Aortic Insufficiency, - Mild Mitral Regurgitation, - Mild Tricuspid Regurgitation (Tr), TRG=20 - No As, No Ps, EF By Sips=60 %, - EF=50 55%, No RWMA, Aps=25 - No Dd, - No PEF
21	- Normal Size of the Left Ventricle, and Mild Degree of Dysfunction. - Normal Size of the Right Ventricle, and Normal Function, - No Mitral Stenosis,

	- No Mitral Regurgitation, - No Aortic Stenosis, No Aortic Insufficiency, - Mild Tricuspid Regurgitation (Tr), TRG=17, - Pulmonary Arterial Pressure (Pap)=20, - EF=45% - No PE

On the initial day of assessment, an echocardiogram indicated that the patient's cardiac pumping function was severely compromised, measuring at only 15%. By the 12th day, this function demonstrated improvement, rising to 35% due to supportive therapies.

Discussion

This study highlights successful ALP poisoning management, early diagnosis, and timely therapeutic intervention to lower poisoning-related death. ALP poisoning presents a considerable clinical problem owing to the lack of a particular antidote, leading to a high mortality rate. Nonetheless, lower death rates result from recent progress in specialist medical care.[17] Current research suggests that pharmacological agents, including magnesium sulfate and trimetazidine, alongside interventions such as intra-aortic balloon pump insertion and extracorporeal membrane oxygenation (ECMO), may be beneficial in managing ALP poisoning.[12]

Key supportive measures to manage ALP poisoning encompass the rigorous monitoring of vital signs, blood gas levels, cardiac activity, and maintaining pH balance. It is imperative to ensure a continuous presence of medical staff at the patient's bedside and consider transfer to Critical Care Units (CCU) or Intensive Care Units (ICU) when necessary.[18]

Crucial factors contributing to the survival of individuals suffering from ALP poisoning include prompt referral and intervention, meticulous and ongoing care, rapid gastric lavage using sodium bicarbonate, and the administration of olive oil to facilitate the absorption of liberated phosphine gas within the gastrointestinal tract. Furthermore, ECMO may be necessary to provide crucial extracorporeal cardiac and respiratory support.[12]

According to the Iranian protocol for diagnosing and managing acute rice tablet poisoning, administering N-acetylcysteine, vitamin E, magnesium sulfate, and calcium gluconate is recommended.[19] In this study, we implemented a treatment regimen similar to that employed in prior research,[20, 21] utilizing magnesium sulfate, N-acetylcysteine, and vitamin E. Notably, Tehrani et al. demonstrated that N-acetylcysteine may be effective in the treatment of rice tablet poisoning.[19]

Research evidence indicates that administering N-acetylcysteine in managing acute rice tablet poisoning yields several therapeutic benefits, including reducing mortality rates, extending hospital stays for survivors,[22,23] and enhancing survival time among non-survivors.[23] By established treatment protocols for ALP poisoning,[24] and corroborated by similar studies,[12,21] magnesium sulfate is utilized for cellular membrane stabilization and the mitigation of lipid peroxidation. Magnesium sulfate therapy for ALP poisoning is reported to lower mortality rates.[25-27]

Most therapeutic agents utilized for ALP poisoning primarily aim to improve survival rates, allowing time for additional therapies. Antioxidant therapy has demonstrated potential benefits when combined with supportive care for acute ALP poisoning, as clinical outcomes indicate.[28] In this study, the GIK protocol was utilized. Previous research demonstrated that the GIK treatment protocol significantly improved hemodynamic status,[29] and improved cardiovascular function.[16] Furthermore, it was associated with prolonged hospitalization and increased patient survival rates.[16, 29]

Ullah et al. found that combining GIK therapy with supportive treatment improves survival rates for ALP poisoning patients. Their findings suggest using GIK treatment when combined with supportive measures to treat ALP poisoning. They concluded that GIK therapy could increase hospitalization, but it also improves survival outcomes.[30]

This study describes the effective treatment of a patient experiencing acute cardiogenic shock, metabolic acidosis, and hypotension following rice pill ingestion. This patient survived by following a protocol that included an antioxidant package, GIK, early intubation to support cardiac function, and all the usual supportive measures. These patients have a high mortality rate, especially when they experience cardiac and multi-organ complications due to phosphine gas released from rice tablets and cellular-level hypoxia.

We addressed a case by employing prompt supportive interventions. This case provides evidence for the efficacy of timely supportive interventions in treating ALP poisoning.

Ethical Considerations

The Research Ethics Committee of Shahroud University of Medical Sciences approved this study (IR.SHMU.REC.1402.158).

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Conflict of Interest

The authors declare no conflicts of interest.

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