Original Article

Investigating the Outcomes of Aluminum Phosphide Poisoning in Khorshid Referral Hospital, Isfahan, Iran: A Retrospective Study

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Abstract

Objective: Aluminum phosphide (ALP) (rice-tablet) is a common cause of adult poisoning in Iran, including Isfahan. So far, no effective treatment has been identified for this poisoning. We aimed to investigate the outcome of ALP poisoned patients admitted to the clinical toxicology ward of Khorshid Hospital in Isfahan from 2017 to 2019. **Methods:** This chart-review study was performed on the population of ALP poisoned patients admitted to the clinical toxicology ward of Khorshid University Hospital from 2017 to 2019 treated with the hospital's new treatment protocol, using the complete enumeration approach. The outcomes were determined by reviewing and abstracting medical charts of ALP poisoned patients from the hospital archive. **Findings:** The most common complaints at admission were depressed consciousness (41.9%) and vomiting (32.2%). There was no significant change in blood sugar, pH, base excess (BE), and venous blood bicarbonate throughout their hospitalization ($P > 0.05$). Treatment outcomes had a significant relationship with blood pH 2 h and 6 h after admission and the BE 6 h after admission ($P < 0.05$). There was also a significant relationship between the outcome and the length of stay, initial ejection fraction (EF), and EF in predischarge echocardiography ($P < 0.05$). Out of 31 patients, 24 (77.4%) died within 72 h, 5 (16.1%) recovered without any complication, and 2 (6.5%) recovered with some complications. **Conclusion:** The mortality rate of ALP poisoned patients was reasonably high and can be attributed to the poor efficacy of the new treatment protocol or the long time it takes for patients to reach the hospital and start receiving treatments.

Keywords: *Aluminum phosphide, poisoning, rice tablet, therapeutic outcomes, treatment protocol*

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Introduction

Aluminum phosphide (ALP), locally called rice-tablet, was first introduced in India in 1973. It is an inexpensive and effective rodenticide and insecticide to protect rice and cereals during storage and transportation. It is widely utilized in developing countries due to its low cost, ease of use, and high effectiveness.[1] This toxic agent is formulated in 3 g tablets in dark gray or greenish-gray in eco-tube packaging and is available under various brands such as Celphos, Phosfume, Quickphos, and Phostoxin. ALP tablets available in Iran, under the brand name Phostoxin, contain ALP, hydrogen phosphide, urea, and ammonium carbamate.[2]

The ALP, in the presence of water, water vapor, moisture, or gastric acid, releases colorless and dangerous phosphine gas (also called hydrogen phosphide or PH_3), which is known to be the main cause of toxicity of this agent; the fresher the tablets, the more gas they release and the more dangerous they are. Swallowing 500 mg (one-sixth) of this tablet in an adult can be fatal (LD_{50} = 10 mg/kg). Each tablet in the presence of moisture is usually able to produce about

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1 g of PH_3. This chemical has no dermal absorption and exerts its poisoning through oral and respiratory routes.[3] The phosphine is a colorless and flammable gas with decaying fish or garlic-like odor. This gas is extremely toxic and is a protoplasmic poison, which interferes with enzymes and proteins. It causes noncompetitive inhibition of mitochondrial cytochrome c oxidase, electron transport chain, and oxidative phosphorylation. The inhibition of energy production in mitochondria leads to toxicity, extensive cell death, and damage and necrosis of the gastrointestinal tract, liver, and kidneys. Due to the ability of phosphine to form free radicals in many tissues, studies show that the organs in need of more oxygen such as the heart, kidneys, lungs, and liver are more sensitive to phosphine-induced damage.[4]

The symptoms of metal phosphide poisoning appear very quickly within 10–15 min after ingestion. Vomiting has been reported as the first sign of gastrointestinal effects of ALP. The classic signs and symptoms include epigastric pain, nausea, and refractory shock. Other symptoms of ALP poisoning include delirium, coma, pulmonary edema, metabolic acidosis, methemoglobinemia, adrenal insufficiency, liver failure, and kidney failure. Phosphine poisoning is associated with a high risk of death. Most deaths occur within the first 12–24 h of poisoning and are often due to the direct effect of phosphine gas on the myocardium, arrhythmia, and cardiac arrest. Mortality happens after 24 h, mainly due to liver failure.[5] Acute respiratory distress syndrome may occur following acute lung injury due to the direct effect of phosphine gas on the alveoli and their destruction.[6]

ALP poisoning has been reported as one of the most important causes of severe poisoning leading to death in Iran, with a mortality rate of up to 60%. The mortality rate following the ingestion of metal phosphide is estimated to be between 31% and 77%, related to the dose of ingested toxin.[7-15] National statistics show the high prevalence and growing use of this deadly poison among young people to attempt suicide.[8,9] Unfortunately, it is impossible to detect toxins in the body early quickly, and the diagnosis is based solely on clinical signs and history taking. In suspected cases, silver nitrate-impregnated paper can be used, which darkens in the presence of phosphine gas. After 24 h, the ALP poisoning can be confirmed by measuring methemoglobin level.[16-20]

Immediate treatment is a critical determinant of survival in patients poisoned with ALP tablets. Unfortunately, there are no specific antidotes for the treatment of phosphine gas poisoning. The general treatment principles include careful and ongoing supportive measures and prompt transfer to the intensive care unit (ICU). Despite such care, many patients are unable to survive.

Methods

The present retrospective study was conducted after receiving the scientific code (No. 399395) and the code of ethics (No.: IR.MUI.MED.REC.1399.455) by reviewing the medical records of patients poisoned with rice tablets referred to the toxicology ward of Khorshid hospital in Isfahan, who were treated with the new therapeutic protocol in 2017–2019. The participants were selected by census sampling method, all of whom were diagnosed with rice tablet poisoning were treated with a new therapeutic protocol from 2017 to 2019.

The inclusion criteria were rice tablet poisoning leading to hospitalization and treatment with a new therapeutic protocol. Accordingly, from November 22, 2017, to March 19, 2019, 105 patients with rice tablet poisoning had been referred to the Khorshid hospital emergency department, of which 13 medical records were unavailable. According to the findings, 61 admitted patients had no symptoms of ALP poisoning or later refused to eat rice tablets and had been discharged a few hours after receiving maintenance treatment and being monitored or returning to regular status. Finally, 31 patients who were definite cases of rice tablet poisoning and had been prescribed the new therapeutic protocol were included in the study.

Based on up-to-date international studies and previous clinical experiences, a protocol for rice tablet poisoning was proposed by Dorooshi, Zoofaghari *et al*.;[21] according to this protocol, a new treatment of rice tablet poisoning was designed as follows.

After confirming the ingestion of rice tablets, the patient's clothes were first removed, and the areas contaminated during a vomiting episode were washed. After that, 100% oxygen was applied to the patient and kept warm. In case of acidosis, hypotension, or a lower level of consciousness, the patient was admitted to the ICU. Thus, the patient gave 250 ml of vegetable oil (preferably sweet almond oil) orally, and vomiting was stimulated. In case of persistent hypotension, lowered level of consciousness, metabolic acidosis with pH below 7.2, oxygen saturation below 90% or respiratory distress (whichever was higher), the patient underwent endotracheal intubation, oil administration through nasogastric tube (NGT) (with the largest size), and suctioning. The patient was then sedated with fentanyl (3–5 mic/kg) and midazolam (0.1 mg/kg); midazolam injection is continued at a dose of 3–5 mg/h to maintain sedation. Laboratory and clinical factors are analyzed simultaneously according to the protocol.

[21] After examination and confirmation of poisoning, intravenous calcium gluconate (1 g) was first given to the patient slowly over 5 min and then repeated every 6 h provided normal or low serum calcium levels. In addition, intravenous magnesium sulfate (1 g) was given to the patient first over 20 min and then every 6 h provided normal or low serum magnesium levels. Then, 1 L of dextrose/normal saline (D/S) was given to the patient over half an hour and then every 4 h in the absence of previous hypertension and diabetes. In the case of diabetic patients, normal saline (N/S) was prescribed to patients by the mentioned method. For nondiabetic patients with controlled blood pressure, 0.5 N hypertonic saline (H/S) with dextrose 5% in water (D/W) was given as much as 3 L of each over 24 h. In case of uncontrolled hypertension in nondiabetic or diabetic patients, one liter of 1.2–2.3 normal saline is given to the patient every 6 h. Vitamins E and C are administered according to the protocol proposed by Dorooshi *et al*.[21] Pantoprazole and hydrocortisone were then injected intravenously. Finally, in the case of vasopressor-resistant hypotension (norepinephrine or epinephrine), high-dose insulin. at a dose of 1 U/Kg is given as a direct intravenous injection (in a bolus) and then at a dose of 1–5 U/Kg/h.

It should be noted that none of the patients received pralidoxime, silymarin, and curcuma mentioned in the original protocol.

By referring to the hospital archive and extracting the file numbers of eligible patients, the information required for the study was extracted from the medical records to review the desired outcomes and examinations. Data were collected in a preprepared form consisting of information about age, gender, routes of poisoning (inhalation, dermal absorption, ingestion or injection), causes of poisoning (intentional/unintentional), treatments used for the patient, the need or not for dialysis, final outcome, and duration of hospitalization.

Statistical analyzes were performed in two steps: Descriptive and analytical. The descriptive part included frequency, frequency percentage, mean, standard deviation, and proportional statistical tables and graphs. Data were analyzed using SPSS version 24 software using dependent *t*-test, one-way analysis of variance (ANOVA), and repeated measures ANOVA at a significance level of $P < 0.05$.

Results

A total of 31 patients, including 22 (71%) males and 9 (29%) females, entered the study. The patients had a mean age of 27.51 ± 8.59 years, with the oldest being 53 and the youngest being 18 years of age. Out of these 31 patients, 21 (72.41%) were single, 8 (27.58%) were married, 27 (87%) were first-time suicide attempts, and 28 (90%) had no history of substance abuse. It should be noted that all (100%) of these patients had consumed the rice tablet orally with suicide intention. The mean number of rice tablets consumed was 1.91 ± 1.15, with the highest being four and the lowest being half a tablet.

The mean length of stay was 93.14 ± 36.27 h for recovered patients and 9.66 ± 8.38 for deceased patients ($P = 0.0$). Out of 31 patients, 24 required intubation, all of whom died. The mean time from admission to intubation was 2.68 ± 1.91 h.

The most common complaint at admission was lowered consciousness level (38.7%) followed by vomiting (32.25%), with 16% of patients having both conditions [Table 1]. For 8 (25.8%) of the patients, the amount of poison and the poisoning time was unknown. All of these eight patients died in <24 h. Out of 31 patients, 24 (77.4%) patients died within 72 h, 5 (16.1%) recovered without any complication, and 2 (6.5%) recovered with some complications. One of these two patients had sinus tachycardia, an ejection fraction (EF) of 40%, and global hypokinesia at the time of admission and showed elevated troponin levels of up to 11,000 during the hospitalization. For this patient, angiography was performed, showing normal results, and another echocardiography was performed before discharge showing EF = 60%. This patient was discharged after 120 h of hospitalization.

The second patient remained hospitalized for 80 h, during which INR increased from 1.1 to 1.45, but other liver tests were normal. Therefore, a follow-up INR check 1 week after discharge was recommended. Furthermore, one additional patient underwent hemodialysis at the 10th h- of hospitalization because of having a creatinine level of 2.4 (at admission), but died after 13 h of hospitalization.

Table 1: Main complaint of the patients with rice tablet poisoning at admission

Main complaint at admission	Outcome		Total (*n*=31), *n* (%)
	Survived (*n*=7)	Death (*n*=24)	
Decreased level of consciousness	0	12	12 (38.70)
Vomiting	4	6	10 (32.25)
Decreased level of consciousness with concurrent vomiting	0	5	5 (16.12)
Lethargy and weakness with concurrent vomiting	2	0	2 (6.45)
Decreased level of consciousness and seizures	0	1	1 (3.22)
Asymptomatic	1	0	1 (3.22)

In line with the research objectives, pH, HCO3, and BE at admission and 2, 4, and 6 h after admission were analyzed using the repeated measures test. The test results showed significant differences between deceased and recovered patients in terms of pH 2 h, 4 h, and 6 h after admission and BE 6 h after admission ($P < 0.05$) but not in terms of HCO3 ($P > 0.05$) [Table 2].

All of the patients had received similar amounts of calcium gluconate, magnesium sulfate, Vitamins E and C, and pantoprazole and hydrocortisone. The statistical analysis showed no significant relationship ($P > 0.05$) between the clinical outcome and aspartate transaminase (AST), alanine transaminase (ALT), ALP, blood urea nitrogen, Cr, Bili. Total and Bili. Direct, Na, K, Ca, Mg, the amount of oil received (in cc), the total amount of bicarbonate received (in vials), the total amount of insulin received (in units) BS at admission, 1 h after admission, 2 h after admission, and 3 h after admission, and the total amount of crystalloid IV solution received during the first 6 h (in liters). It should be noted that the serum phosphorus level at admission also had no significant relationship with the outcome (P>0.05) [Tables 3 and 4]; but had a significant relationship with pH at admission and 2 h after admission, HCO3 at admission and 2 h after admission, BE at admission and 2 h after admission, EF at admission and at discharge, and AST, ALT, INR, Mg, Cr levels ($P < 0.05$) [Table 5]. There was a significant difference between recovered and deceased patients in terms of BE and pH 2 h after admission and EF at admission ($P < 0.05$) [Table 5]. A significant relationship was found between EF at discharge and pH and BE 6 h after admission and the phosphorus level [Table 5]. (Alternative:) A significant relationship was found between pH and BE 6 h after admission and between EF and phosphorus level at discharge [Table 5].

Table 2: Changes in venous blood gas of the patients with rice tablet poisoning during hospitalization

VBG	Time	Survived	Death	*P*
pH	T0	7.26±0.14	7.18±0.20	0.349
	T2	7.30±0.13	7.13±0.12	0.015
	T4	7.28±0.16	7.05±0.12	0.051
	T6	7.31±0.10	7.06±0.11	0.001
$HCO3^-$	T0	19.64±7.76	15.46±6.16	0.160
	T2	17.78±5.97	14.18±5.34	0.187
	T4	17.95±5.16	17.80±8.79	0.982
	T6	17.10±7.28	13.72±2.69	0.221
Base excess	T0	−6.71±9.14	−13.34±9.13	0.111
	T2	−9.00±7.28	−15.00±5.66	0.054
	T4	−8.85±6.43	−13.04±8.66	0.533
	T6	−8.33±7.26	−16.25±3.45	0.013

T0=Immediately at admission, T2=2 h after admission, T4=4 h after admission, T6=6 h after admission, VBG=Venous blood gas

Table 3: Results of the electrolyte tests of the studied patients at the hospital admission

Variables	Survived	Death	*P*
Na^+	139.57±4.50	141.05±4.29	0.446
K^+	3.69±0.39	3.41±0.56	0.234
Ca^{++}	8.66±0.49	8.63±1.49	0.970
Mg^{++}	2.17±0.24	2.67±1.01	0.208
Phosphorus	3.65±1.92	4.85±2.51	0.311

Table 4: Effect of the treatment protocol on the outcome of the studied patients

Treatment protocol	Survived	Death	*P*
Total oil administered (cc)	233.33±93.09	227.08±109.32	0.774
Total IV fluid administered in first 6 h (L)	1.85±0.38	2.21±0.79	0.070
Total bicarbonate vials	20.43±14.47	14.91±10.16	0.315
Total NAC (g)	72.00±36.57	19.46±13.13	<0.001
Total insulin administered (units)	-	392.86±240.53	-

IV=Intravenous, NAC=N-acetylcysteine

Discussion

Our results showed that out of 31 patients admitted to the ward for rice tablet poisoning in the said period, 71% were male and 29% were female, and the majority (87.1%) were first-time suicide attempts. This is consistent with the findings of a study by Shayeste *et al*., which reported that most of the patients who had attempted suicide by eating rice tablets were men,[22] but not with the reports of the studies conducted by Hosseinian *et al*. in Mazandaran (2007–2008), Montazer *et al*. in Ghaemshahr and Sari (2013–2014), and Farzaneh *et al*. in Ardabil (2006–2016). However, like the present study, the studies of Taromsari *et al*. in Rasht (2010–2013), Sinha *et al*. in India (2005), and Murali *et al*.(2004–2009)[23-28] observed more men with this kind of poisoning than women. Since most rice tablet poisoning cases reported in studies are intentional, one can infer from the above results that men may be more committed to suicide in this way and that men tend to have more access to this poison than women.[25]

In this study, the mean age of rice tablet poisoning patients was 27.51 ± 8.59 years. The study of Shayeste *et al*. in Gorgan also reported that most cases were in the age group of 20–29 with an average of 27.4 years.[22] Similar results have been reported in many studies conducted in Iran and other countries, which indicates the high prevalence of rice tablet poisoning among young people and highlights the importance of having prevention programs in place, focusing on this age group.[24,29-31] Attention to the emotional, social and

Table 5: Relationship between serum phosphorus level and ejection fraction with other variables at admission and discharge

Variables	Outcome	pH				$HCO3^-$				Base excess			
		T0	T2	T4	T6	T0	T2	T4	T6	T0	T2	T4	T6
Serum phosphorus level													
Correlation	0.239	−0.838	−0.726	−0.245	−0.053	−0.644	−0.486	−0.394	−0.334	−0.723	−0.685	−0.312	−0.255
P	0.311	<0.001	0.001	0.524	0.864	0.002	0.041	0.295	0.264	<0.001	0.002	0.414	0.401
EF at admission													
Correlation	−0.534	0.437	0.792	0.235	−0.033	0.335	0.381	−0.704	0.201	0.407	0.589	−0.589	0.139
P	0.022	0.079	<0.001	0.575	0.928	0.189	0.161	0.051	0.578	0.105	0.021	0.125	0.703
EF at discharge													
Correlation	−0.768	−0.655	−0.300	−0.365	0.954	0.600	−0.890	−0.683	0.999	−0.067	−0.756	−0.256	1.00
P	<0.001	0.546	0.806	0.768	0.194	0.590	0.302	0.232	0.034	0.957	0.454	0.864	0.013

T0=Immediately at admission, T2=2 h after admission, T4=4 h after admission, T6=6 h after admission, EF=Ejection fraction

financial needs of young people at greater risk of suicide and banning the sale of this poison in stores can reduce the frequency of this kind of suicide attempt. However, since many of the reported cases have been in the middle-aged age range, this group also needs to receive due attention.[22] Considering that the majority of patients in the present study were young, this age skewness may be related to the inability of young people to overcome or deal with the problems in marital life (or love-life in general), which increases the tendency toward rash suicide attempts.[25]

Of this study's 31 rice tablet poisoning patients, 12 (38.7%) had a lowered consciousness level upon arrival, and 5 (16%) had reduced consciousness with vomiting. In the study of Taromsari *et al*., 41% and in the study of Khodabandeh *et al*., 44% of patients had lowered consciousness and lethargy upon admission to the emergency room.[23,32] The cause of this condition can be the hypoxia of brain cells due to the formation of oxygen free radicals and the dropping of blood pressure. It should be noted that there was no information available on complaints upon arrival for 11 of the patients who had been transferred from other facilities under sedation or intubation.

In the study of Shayeste *et al*., the most frequent complaints upon admission were vomiting followed by drowsiness and unconsciousness[22]. In the study of Khodabandeh *et al*., vomiting was described as the first and most prominent symptom of the effect of ALP on the gastrointestinal tract.[32] Some studies have suggested that vomiting contributes to the discharge of ALP and reduces the release of phosphine gas.[33-37] The presence of classic signs and symptoms of ALP poisoning, such as epigastric pain, nausea and shock, and severe hypotension in rice tablet poisoning patients, has been reported in many studies. In the present study, people with lowered consciousness at admission had a low survival rate despite using supportive methods and drug treatments such as vasopressors and bicarbonate, which is consistent with the findings of other studies.[38] The study of Farzaneh *et al*. also reported that lowered consciousness was the most frequent complaint and had a significant relationship with mortality.[39] In the case report published by Babu *et al*. also, the patient had a lowered level of consciousness.[40] Like the present study, Taromsari *et al*. also found that the most common symptom upon admission was lowered consciousness. Some studies have attributed this symptom to the hypoxia of brain cells, which can be due to the release of free radicals in these cells and the dropping of blood pressure in these patients. Furthermore, the brain autopsy of these patients has shown significant hyperemia along with exudate and small hemorrhages.[29] The cause of hypotension and the resulting shock in these patients could be a cardiogenic shock, shock due to peripheral circulatory failure, or both. The increased vascular permeability due to damage to vascular wall cells and the intoxication of cardiac cells also affect this condition.[22]

In this study, 33% of the patients had a normal electrocardiography (ECG), but 67% had cardiac arrhythmias, the most common of which was AF (18.5%) followed by bradycardia (14.8%), and tachycardia, V. Tach, and VF (11% each). In the study of Mathai *et al*., cardiac arrhythmias and treatment-resistant hypotension were the most frequent complications leading to death in rice tablet poisoning patients.[36] In a study by Hsu *et al*. on the ECG of patients with ALP poisoning, they observed a variety of cardiac arrhythmias, including tachycardia, V. Tach bradycardia, and AF.[33] In the present study, the mean EF at admission was 40 ± 17.32% for surviving patients and 22.91 ± 11.37% for deceased patients and was found to have a significant relationship with the outcome ($P = 0.02$, $R = –0.5$). The most common complications leading to death in rice tablet poisoning patients are cardiovascular collapse and

fatal arrhythmias due to the direct effect of phosphine gas on heart cells, fluid loss, and treatment-resistant hypotension.[31] In this study, the mean serum phosphorus level at admission was 3.65 ± 1.92 mg/dL for surviving patients and 4.85 ± 2.51 mg/dL for deceased patients, with the lowest level being 1.8 mg/dL and the highest being 12.2 mg/dL. No significant relationship was found between mortality and the serum phosphorus level at admission (P = 0.311). However, this could be due to the small size of the sample, because phosphorus level had a relationship with EF at admission (P = 0.03, R = –0.5), pH at admission (P = 0, R = –0/8), PH 2 h after admission (P = 0, R = –0.7) and other important parameters including liver enzymes and serum creatinine (INR) [Table 5] and also with EF at discharge [Table 5]. Considering the relationship between mortality and EF at admission (P < 0.05), one may argue that phosphorus level affects the outcome indirectly and could still be a significant predictor of whether or not the patient survives. Therefore, it is recommended to conduct further research with a larger sample size on the hourly changes in the phosphorus level during hospitalization and their relationship with venous blood gases (VBG) and EF, the outcome, and other parameters mentioned above. Future studies are also recommended to evaluate the effect of emergency dialysis (at most 3 h after admission) as an acute treatment of hyperphosphatemia with metabolic acidosis on the outcome.

This study also showed that the EF and mortality of the rice tablet poisoning patients had a significant relationship with their condition in the 2nd h and the 6th h of hospitalization, which makes them critical hours for these patients. In a study by Nasa *et al*. in Northern India, two patients with rice tablet poisoning were successfully treated with early initiation of continuous renal replacement therapy along with other supportive measures. Since this study found no significant relationship between the outcome and HCO3 at different hours after admission and also with the total number of bicarbonate vials received (P = 0.315), future studies are recommended to try other treatments including plasmapheresis and hemodialysis for the treatment of metabolic acidosis in the early hours of admission and publish the result. However, this must not be interpreted as a recommendation against the early administration of bicarbonate to rice tablet poisoning patients.

This study found no significant relationship between the outcome and the total amount of insulin received (in units) and BS at admission and 1, 2, and 3 h after admission. However, considering the effect of hyperphosphatemia on the development of insulin resistance, future studies are recommended to investigate the relationship between hyperphosphatemia treatment and changes in BS. While the P value obtained for the relationship between mortality and the total number of N-acetylcysteine (NAC) vials received was significant, no reliable interpretation could be made from this result because surviving patients remained hospitalized for more extended periods.

In this study, 77.4% of the patients died within 72 h. This rate was 34.3% in the study of Khodabandeh *et al*., 30.8% in the study of Montazer *et al*. in Ghaemshahr and Sari (2013–2014) 18.6% in the study of Hosseinian *et al*. in Mazandaran (2007–2008), 62.5% in the study of Nosrati *et al*. in Sari (2011–2012), and 24% in the study of Sultan Nejad in Tehran (2007–2010).[24,28,41,42] The study conducted by Christophers *et al*. in India (2002) also reported that two-thirds of rice tablet poisoning patients died after hospitalization.[23] In some studies, this rate has been even higher. For example, the studies of Farzaneh *et al*. in Ardabil (2006–2016) and Reyna-Medina in Mexico (2009–2010) reported this rate to be 70.4% and 78%, respectively, which reflect the high lethality of rice tablets.[26,43] Despite the restrictions put in place by the Iranian authorities to limit the circulation of rice tablets in the country, these tablets can still be easily acquired and continue to cause a significant number of deaths by poisoning. In European countries, where this substance is only available to certified individuals or organizations, the number of suicides by this substance and the resulting deaths are much lower than in Iran and other Asian countries.[24] By comparing the mortality statistics of this study and other similar studies, it is reasonable to conclude that the treatment protocol of the studied hospital is less successful than the protocols of other large medical facilities in Iran and may need to be reviewed. However, it should be remembered that the findings of such studies can be influenced by many factors including how strictly the treatment protocols are enforced, how long it has taken for the patient to start receiving treatment after taking the tablet, whether the patient has started vomiting immediately after taking the tablet, etc.

The results of this study regarding the mortality rate suggest that the treatment protocol of the studied hospital is weak compared to other similar medical facilities. Considering the relationship observed between the serum phosphorus level and VBG and EF and several other important parameters and the effectiveness of early hemodialysis in treating hyperphosphatemia with acidosis and also improving the efficacy of some other treatments (such as insulin), which may lead to improved EF and prognosis, it is recommended to conduct further research with a larger sample size to

investigate whether this relationship truly exists and whether early hemodialysis can help achieve better outcomes for these patients.

Respiratory support, fluid therapy, prevention of absorption, accelerated detoxification, and antioxidants and immunosuppressants are some of the suggested and sometimes used strategies in the treatment of this poisoning. No standard treatment management has been developed so far for ALP poisoning globally, and this poisoning is treated differently worldwide. The present study was designed and conducted to prepare a comprehensive, evidence-based protocol for ALP poisoning management at the Poison Control Center in Isfahan province for proper and timely treatment and reduction of unnecessary measures during treatment.

Authors' Contribution

G. Dorooshi and M. Mirzae contributed in conceptualization, data collection, and manuscript preparation and editing. N. Tavakoli Fard and S. Zoofaghari performed idea processing, study design, project administration and supervision, and manuscript preparation and editing. N. Eizadi Mood contributed in conceptualization, study design, and manuscript preparation and editing.

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Conflicts of interest

There are no conflicts of interest.

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