

# Research Paper: Prognostic Factors of Aluminum Phosphide Poisoning in Urmia: A-five-years Cross-sectional Study



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## ABSTRACT

**Background:** Aluminum phosphide is a highly toxic fungicide. It causes such severe adverse effects as cardiac arrhythmias, metabolic acidosis, acute renal failure, shock, and even death. Therefore, this study investigated the prognostic factors of aluminum phosphide poisoning.

**Methods:** In this cross-sectional study, all hospitalized patients due to aluminum phosphide poisoning admitted to the poisoning center at Taleghani Hospital, Urmia City, Iran, from 2015 to 2019 were evaluated. The demographics characteristics, clinical findings, and laboratory profiles were retrospectively studied by an investigator-made checklist and evaluated concerning the explored patients' treatment outcomes. Then, the obtained data were analyzed by descriptive and analytical statistics using SPSS V. 16.

**Results:** The present study investigated 134 patients (96 males & 38 females). The mortality rate was equal to 29.8% (22.4% males & 7.4% females) in the study patients. The research patients' Mean±SD age was 28.6±11.5 years. The Mean±SD ingestion amount of aluminum phosphide was 1.48±1.06 g (min=0.2 g, max=15 g). Nausea and vomiting with 119 (88.8%), hypotension: 89 (66.4%), vertigo: 80 (59.7%), and sinus tachycardia: 74 (55.2%) were the most signs and symptoms in the study patients, respectively. Statistically significant relationships ( $P<0.05$ ) were found between the patients' treatment outcomes and white blood cells, direct bilirubin,  $HCO_3^-$  base excess, magnesium, Aspartate Aminotransferase, Alanine Aminotransferase, Blood Urea Nitrogen, creatinine, blood glucose, pH, prothrombin time, and the international normalized ratio. Furthermore, significant relationships were detected between the patients' treatment outcomes and leukocytosis, hypokalemia, hyperglycemia, and hypoglycemia ( $P<0.05$ ).

**Conclusion:** Such prognostic factors as demographics characteristics, clinical findings, laboratory profiles, and electrolytes could be used as good indices of the severity of toxicity in patients; accordingly, such data are beneficial for the proper management of patients by healthcare providers. Therefore, prognostic factors should be considered in the diagnosis, treatment, and follow-up stages for these patients.

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## 1. Introduction

The rate of mortalities due to acute poisoning has increased worldwide [1]. Approximately, 300000 deaths due to pesticide poisoning occur in Asian pacific countries annually [2]. Aluminum Phosphide (ALP) poisoning usually occurs intentionally following suicidal ideation; in some cases, it occurs accidentally and rarely occurs criminally [3]. Considering its availability and low price, ALP is used to protect grains, like rice. As a result, it is also called the rice pill in Iran [4]. According to recent studies, suicide with rice pills has become highly prevalent in Iran [5]. ALP is a common cause of acute poisoning and mortality in numerous developing countries, like Iran [1, 6]. ALP falls in the category of fungicides and rodenticides, i.e., available in the form of 3-gram tablets in dark gray with a smell similar to garlic or rotten fish [6, 7]. The average lethal dose of ALP for an adult is 0.5 g or one-sixth of a pill [3, 8]. The main routes of the involvement of ALP poisoning are oral administration and inhalation [6]. Rice pill, when combined with water or hydrochloric acid in the stomach, releases a highly toxic gas, called Phosphine (PH<sub>3</sub>). Phosphine gas acts by inhibiting the mitochondrial cytochrome oxidase system [5, 6]. The mortality rates of ALP intoxication were reported by Beyranvand et al. to range from 31% to 77% [9], Taghad-dosinejad et al. as 70%-100% [10], and Navabi et al. as 60%-90% [3]. Due to high mortality, the production and distribution of rice tablets have been stopped since 2007 in Iran [1]. Multi organs failure, such as gastrointestinal, cardiovascular, hepatic, renal, and neurological involvement is observed in ALP poisoning due to its hypoxic effects [5, 6].

The important clinical manifestations of ALP poisoning are gastrointestinal conditions (nausea, vomiting, & epigastric pain), cardiovascular disorders (hypotension & even shock, tachycardia, bradycardia, & congestive heart failure), central nervous system conditions (headache, dizziness, & finally the loss of consciousness), and severe metabolic acidosis [5, 11]. Life-threatening cardiac arrhythmias induced by severe metabolic acidosis are the main causes of death in ALP poisoning [3]. The diagnosis of ALP poisoning is usually based on clinical suspicions; however, silver nitrate testing on stomach contents or breathing vapors is also used to confirm the diagnosis [11]. ALP poisoning presents no specific antidote; however, previous studies have suggested some treatment approaches, such as gastric lavage with sodium bicarbonate and the ingestion of charcoal [12]. Additionally, some specific treatment methods of ALP poisoning have been suggested, including the intravenous administration of magnesium sulfate, sodium bicarbonate, N-acetyl cysteine, digoxin, vasopressor, vitamin C, and vitamin E [10,

13, 14]. Some studies demonstrated that hyperglycemia, hypotension, acidosis, leukocytosis, hyperuricemia, electrocardiographic abnormalities, the loss of consciousness or coma, low prothrombin rate, and acute renal failure were associated with a poor prognosis in ALP poisoning [3, 4, 7]. Accordingly, laboratory profiles and their effects on the prognosis of patients with ALP poisoning were reported in limited studies [15-17]. Therefore, the present study evaluated demographics characteristics, clinical features, laboratory profiles, and their correlations with prognosis in ALP intoxicated patients.

## 2. Materials and Methods

The current study investigated 134 patients hospitalized due to ALP poisoning. They were admitted to the poisoning center at Taleghani Hospital in Urmia City, Iran from 2015 to 2019. To conduct this cross-sectional study, we included all patients with ALP poisoning in the age range of 14 years or above. In each case, the diagnosis had been made based on history gathered from the patients or their relatives. The hospital's clinical toxicologists also had visited the patients and provided further information to establish the diagnosis on admission to the Emergency Department (ED). Besides, the diagnosis of ALP exposure was made based on clinical signs and symptoms, such as inhaling garlic or rotten fish odor during their clinical examinations. ALP poisoning has no effective antidote; thus, the patient's management was only supportive. For all cases, gastric lavage with sodium bicarbonate, potassium permanganate, and activated charcoal were administered. Other components of administered supportive medical care included sodium bicarbonate, magnesium sulfate, calcium gluconate, N-acetyl cysteine, dopamine, and norepinephrine. Patients with incomplete records and laboratory tests during admission and those who were discharged with personal consent were excluded from this study.

Table 1 presents the study patients' demographics characteristics and clinical features. All laboratory profiles were checked upon hospital admission. Laboratory profiles and prognosis were retrospectively collected by an investigator-made checklist, i.e., presented in Table 2. Moreover, the comparison of mortality rate in studied patients from 2015 to 2019 are presented in Table 3. No personal identification data were recorded and all information remained strictly confidential. Approval for performing this research was issued by the Ethics Committee of Urmia University of Medical Sciences, Iran. The obtained data were analyzed in SPSS version 16 using descriptive statistics, including Mean±SD. The data consisted of the demographic characteristics and clinical out-

comes for every patient. The research variables were also grouped into survivors and nonsurvivors. In our study, the differences in quantitative variables with normal distributions and abnormal distributions were evaluated by the t-test and Mann–Whitney U test, respectively. The relationships between categorical variables and the relevant outcomes were evaluated using Chi-squared test. Furthermore,  $P < 0.05$  and confidence interval of 95% were found to be statistically significant in this research.

### 3. Results

The present study explored 134 patients (96 males & 38 females). The mortality rate was calculated as 29.8%

(22.4% males & 7.4% females). The patients' Mean±SD age was measured as 28.6±11.5 (min=14, max=81) years. The Mean±SD ingestion amount of ALP was equal to 1.48±1.06 g (min=0.2 g max=15 g). The male to female ratio was calculated as 2:5. Table 1 presents the study patients' demographic characteristics and clinical features. The highest prevalence of aluminum phosphide poisoning (67.9%) was reported in the age group of 14-30 years. Approximately 12 (9%) of patients presented no history of substance abuse. Furthermore, 17 (12.5%) of the explored patients were rural and 117 (87.5%) of them were urban residents. About 63 (47%) of the investigated patients were married. Moreover, 95 (71%) of cases reported a history of suicidal attempts. Nausea and vomiting 119 (88.8%), hypo-

**Table 1.** Effects of some demographic characteristics and clinical features on the outcomes of the patients with ALP poisoning at admission

Characteristics	No. (%)		P (Chi-squared Test)
	Survivor Group (n=94)	Non-survivor Group (n=40)	
<b>Demographic</b>			
Admission service	Ward	37 (39.4)	1 (2.5)
	ICU	26 (27.6)	36 (90)
	Ward and ICU	31 (33)	3 (7.5)
The duration of hospital stay (days)	3 (1-18)	2 (1-5)	0.02 (Mann–Whitney U test)
Age (y),	Median (min-max)	25.5 (14-81)	29.5 (17-66)
	14-30	69 (73.4)	22 (55)
	31-45	21 (22.3)	9 (22.5)
	46-60	3 (3.2)	7 (17.5)
	Up to 60	1 (1.1)	2 (5)

Characteristics	No. (%)		P (Chi-squared Test)
	Survivor Group (n=94)	Non-survivor Group (n=40)	
<b>Clinical Features (Using Chi-squared Test)</b>			
The level of consciousness	Alert (GCS=15/15)	61 (64.9)	14 (35)
	8/15≤GCS<15/15	28 (29.8)	19 (47.5)
	Coma (GCS<8/15)	5 (5.3)	(17.5)
Hypoventilation	21 (22.3)	17 (42.5)	0.0001
Sinus bradycardia	2 (2.1)	6 (15)	0.004
Mydriasis	11 (11.7)	20 (50)	0.0001
Shivering	3 (3.2)	6 (15)	0.01
Ataxia	30 (31.9)	24 (60)	0.002
Pale	36 (38.3)	22 (55)	0.04
Extremity Coldness	23 (24.5)	27 (67.5)	0.0001
Hypotension	56 (59.5)	33 (82.5)	0.04

SD: Standard Deviation; GCS: Glasgow Coma Score; ARF: Acute Renal Failure.

**Table 2.** Effects of some laboratory profiles on outcome and P-value at admission

Quantitative Variables	Mean±SD/ No. (%)		P	
	Survivor Group (n=94)	Non-survivor Group (n=40)		
Normal distributions; using t-test	WBC (×103/L)	9.5±4.1	12.8±7.0	0.000
	Direct Bilirubin (mg/dL)	0.35±0.26	0.6±0.1	0.001
	Serum Hco <sub>3</sub>	21.7±5.9	14.0±5.15	0.000
	Base excess (BE)(meq/L)	(-3)±(+6.3)	(-14)±(-7.7)	0.000
	Hb (mg/dL)	14.1±2.1	14.2±1.7	0.93
	HCT (%)	41.7±5.2	42.0±7.3	0.49
	PLT (×103/L)	229.0±68.5	212.0±63.0	0.16
	Calcium (mg/dl)	9.3±0.7	9.0±0.6	0.22
	Total Bilirubin (mg/dL)	1.0±0.55	1.2±0.2	0.25
	Pco <sub>2</sub> (%)	36.6±9.9	34.9±13.5	0.42
Abnormal distributions [median (min-max)] using Mann-Whitney U test	Magnesium (mg/dL)	1.9 (1.3-9.4)	2.3 (1.4-13)	0.0001
	SGOT (IU/L)	19 (11-2465)	54.5 (16-218)	0.007
	SGPT (IU/L)	14 (7-2328)	50.5 (13-174)	0.006
	BUN (meq/L)	22 (11-53)	28 (16-71)	0.0001
	Creatinine (meq/L)	0.9 (0.6-2.7)	1.2 (0.7-2.1)	0.0001
	Blood Glucose (mg/dL)	111 (56-460)	148 (25-384)	0.003
	Blood pH	7.38 (7.06-7.55)	7.21 (6.8-7.5)	0.0001
	PT (s)	13.2 (11-20)	14.7 (12-60)	0.001
	INR	1.1 (0.88-2.6)	1.4 (1-10.48)	0.001
	Phosphor (mg/dL)	3.2 (1.2-8.2)	5.8 (1.9-3.9)	0.053
	ALKP (IU/L)	171 (84-411)	218 (141-262)	0.45
	Amylase (U/L)	71 (30-381)	135 (52-490)	0.10
	Lipase (U/L)	30 (16-214)	30 (26-54)	0.78
	PTT (s)	30.3 (25-180)	31.5 (23-125)	0.118
Abnormal blood chemistry and using Chi square test	Leukocytosis (WBC>11000 /mm <sub>3</sub> )	2 (24.5)	18 (45)	0.009
	Hypokalemia (K<3.5 meq/L)	9 (9.5)	11 (27.5)	0.03
	Hyperglycemia (BG>200 mg/dL)	7 (7.4)	12 (30)	0.02
	Hypoglycemia (BG<55 mg/dL)	0	3 (7.5)	0.008
	Leukopenia (WBC<4500 /mm <sub>3</sub> )	6 (6.4)	1 (2.5)	0.62
	Thrombocytopenia (PLT<150000/L)	10 (10.6)	2 (5)	0.45
	Hypernatremia (Na>145 meq/L)	0	4 (10)	0.41
	Hyponatremia (Na<135 meq/L)	2 (2.1)	0	0.19
Hyperkalemia (K>5 meq/L)	1 (1.1)	2 (5)	0.13	

**Table 3.** Comparing mortality rate in the survived and non-survived cases from 2015 to 2019

Year of Admission	No.				Total (n=134)	Mortality Rate (%)
	Survivor Group (n=94)		Non-survivor Group (n=40)			
	Male	Female	Male	Female		
2015	1	0	2	0	3	66.6
2016	13	3	7	2	25	36
2017	11	4	4	0	19	21
2018	22	10	8	6	46	30.4
2019	19	11	9	2	41	26.8

**Table 4.** Mortality and prognostic factors in our study, compared to some previous studies

Articles	Non-survived/Total Patients, (Mortality %)	pH	Hco <sub>3</sub>	GCS	Hypotension	WBC Hospital Stays	HCT	Prothrombin Rate	Blood Urea Nitrogen	Blood Glucose	PCO <sub>2</sub>	Number of AIP Tablets	Duration of Hospital Stay	Pulse Rate	Hypoventilation	time Elapsed from Consumption to Treatment	APACHE II	Mechanical Ventilation	Creatinine	Blood Levels of Methemoglobin
Our study (2019)	40/134, (29.8)	+	+	+	+	+	NS	+	+	+	NS	NS	+	+	+	#	#	+	+	#
Farzaneh et al. [7] (2018)	36/68, (52.9)	#	+	+	+	#	#	#	#	#	#	#	#	#	#	#	#	#	#	#
Navabi et al. [3] (2018)	41/77, (53.2)	+	+	#	+	#	#	#	#	#	#	+	#	#	#	+	#	#	#	#
Sharma et al. [15] (2018)	59/116, (51)	+	+	+	+	#	#	#	#	+	#	#	#	+	#	#	#	#	+	#
Erfantalab et al. [4] (2017)	15/39, (38.5)	+	+	NS	+	NS	NS	#	NS	NS	NS	NS	#	+	#	#	#	#	#	#
Mostafazadeh et al. [16] (2010)	9/48, (18.8)	+	#	#	NS	#	#	#	#	#	#	#	#	#	#	#	#	+	#	+
Louriz et al. [17] (2009)	24/49, (49)	#	#	+	+	+	+	#	#	NS	#	#	#	#	#	#	+	+	#	#

NS= Not Significant= notperformed; P<0.05

tension 89 (66.4%), vertigo 80 (59.7%), sinus tachycardia 74 (55.2%), respiratory failure 73 (54.5%), epigastric pain 70 (52.2%), the loss of consciousness 65 (48.5%), paler 58 (43.3%), agitation 58 (43.3%), ataxia 54 (40.3%), extremity coldness 50 (37.3%), mydriasis 31 (23.1%), the dryness of mucosa 31 (23.1%), tachypnea 27 (20.1%), shivering 9 (6.7%), miosis 9 (6.7%), diarrhea 9 (6.7%), and sinus bradycardia 8 (6%) were the most prevalent signs and symptoms in the study patients, respectively. The results of the blood chemistry analyses range of the study patients at admission are presented in [Table 2](#). Furthermore, 41 (30.6%), 4 (2.9%), 3 (2.2%), and 19 (14.2%) of the explored patients had leukocytosis, hypernatremia, hyperkalemia, and hyperglycemia, respectively. Additionally, 7 (5.2%), 2 (1.45%), 20 (14.9%), and 3 (2.2%) of the explored patients presented leukopenia, hyponatremia, hypokalemia, and hypoglycemia, respectively. The deceased patients, compared to the recovered ones (95% vs. 51.5%) swallowed  $\geq 3$  grams of rice pills. Statistically significant relationships ( $P < 0.05$ ) were found between the study patients' treatment outcomes and demographic (admission service & the duration of hospital stay), clinical features (the level of consciousness, hypoventilation, sinus bradycardia, mydriasis, shivering, ataxia, pale, & cold extremities) and laboratory profile, such as White Blood Cells (WBC), direct bilirubin,  $Hco_3$ , base excess, magnesium, aspartate transaminase (SGOT), alanine aminotransferase (SGPT), Blood Urea Nitrogen (BUN), creatinine, blood glucose, PH, Prothrombin Time (PT), and International Normalized Ratio (INR). Moreover, statistically significant relationships were observed between the study patients' treatment outcomes and leukocytosis, hypokalemia, hyperglycemia, and hypoglycemia ([Table 2](#)). However, there was no significant relationship between the study patients' treatment outcomes and demographic characteristics (history of substance abuse, living area, marital status, a history of suicidal attempts, and the amount of ingested ALP), clinical features (vomiting, nausea, agitation, vertigo, diarrhea, sinus tachycardia, epigastric pain, & the dryness of mucosa), and laboratory profile, such as Hemoglobin (Hb), Hematocrit (HCT), Platelets (PLT), Partial Thromboplastin time (PTT), calcium, phosphorus, total bilirubin, Alkaline Phosphatase (ALKP), amylase, lipase, and partial pressure of carbon dioxide ( $Pco_2$ ).

#### 4. Discussion

ALP is a highly-toxic pesticide with a high mortality rate. The mean age of the study cases was 28.6 years. Overall, the consequences of this investigation are compatible with recent studies, such as Erfantalab et al., Kordrostami et al., and Mostafazadeh et al., i.e., 31, 32.6, and 25.5 years respectively. These studies indi-

cated that ALP poisoning has increased among young individuals and the majority of the patients were male in Iran [4, 16, 18]. The highest prevalence of ALP poisoning (67.9%) belonged to the age group of 14-30 years. All explored patients were poisoned following suicidal attempts, i.e., similar to the study of Navabi et al. [3] as well as Hosseinian and associates [19]. In this study, the number of ingested ALP on admission were different in the survivor group, compared to the non-survivor group. Additionally, these data provided useful information; however, consistent with the study of Erfantalab et al., they had no prognostic value in ALP intoxicated patients [4]. In line with the recent studies, the early clinical findings at admission were gastrointestinal discomforts, such as nausea, vomiting, and abdominal pain [19, 20]. Consistent with the study of Jamshidi et al., approximately 15%, 2%, and 2% of the investigated patients reported hypokalemia, hyperkalemia, and hyponatremia, respectively [1]. Concerning Arterial Blood Gases (ABG), serum  $Hco_3$  and pH levels were very low in the non-surviving group, compared to the surviving group. Such finding is consistent with those of other studies [1, 15]. Interestingly, similar to the results of Sharma et al., the mean blood glucose level in this study was very low in the surviving group, compared to the non-surviving group [15]. Most studies have reported mortality rates of above 50%; however, in this study, the mortality rate due to ALP poisoning was measured as 29.8%, i.e., less than those reported by most previous studies. Approximately, the mortality rate of this study is consistent with those of Erfantalab et al. [4] and Mostafazadeh et al. [16] ([Table 4](#)). ALP poisoning has no specific antidote; thus, its management is supportive. Therefore, early diagnosis using appropriate predictors of mortality may provide desirable care and prognosis for these patients. Numerous prognostic factors for ALP poisoning have been used to evaluate the severity of patient's clinical conditions, such as blood pH (severe acidosis), serum  $Hco_3$ , hyperglycemia, GCS, and blood pressure, age, the lack of vomiting, dose ingested APACHE II, white blood cell count, mechanical ventilation, creatinine, blood urea nitrogen, blood levels of methemoglobin, hospital stays, hematocrit, prothrombin rate,  $PCO_2$ , the number of ALP tablets, the duration of hospital stay, blood urea nitrogen, blood glucose, pulse rate, hypoventilation, and time elapsed from consumption to treatment ([Table 4](#)) [3, 7, 15-17]. Furthermore, our study demonstrated that magnesium, aspartate transaminase, alanine aminotransferase, International Normalized Ratio (INR), direct bilirubin, mydriasis, shivering, ataxia, pale, and cold extremities were significantly different between survivors and non-survivors groups. Finally, to obtain

better results, follow-up studies are recommended to investigate the other prognostic factors.

The main limitation of this study was that the diagnosis of ALP poisoning was not confirmed by the silver nitrate test. Another limitation of this study was the incomplete recording of some laboratory data.

## 5. Conclusion

Laboratory abnormalities, such as severe metabolic acidosis, clinical suspicion (severe hypotension), and a positive history of ALP poisoning can be applied for early diagnosis and appropriate treatment of this intoxication. The amount of ingested ALP on admission provides useful information; however, these data presented no prognostic value in patients with ALP poisoning. Establishing the relevant prognostic factors in the clinical setting is crucial for managing patients with acute ALP poisoning. Previous research studies addressed some prognostic factors and their effects on the prognosis of ALP poisoning outcomes. Furthermore, prognostic factors should be considered in the diagnosis, treatment, and follow-up stage of these patients. However, some prognostic factors, such as laboratory profiles, electrolytes, and clinical findings could be used as appropriate indices of the severity of the toxicity of the patients; these data can be used for proper management by healthcare providers. Further studies are required to elucidate definitive prognostic factors in patients with ALP poisoning.

## Ethical Considerations

### Compliance with ethical guidelines

The Ethics Committee of Urmia University of Medical Sciences approved this study (Code: IR.UMSU.REC.1399.215).

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### Author's contributions

All authors contributed to preparing this article.

### Conflict of interest

The authors declared no conflicts of interest.

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