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# Prognostic significance of poison-related factors and consumption patterns in acute aluminum phosphide poisoning

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**Abstract:**

**OBJECTIVES:** The prognosis of acute aluminum phosphide poisoning is usually based on toxidrome features, with little focus on poison-related factors. We aimed to study the prognostic significance of poison-related factors, consumption patterns, and time delays to treatment.

**METHODS:** We performed a prospective cohort study in an academic hospital in North India in patients aged ≥ 13 with aluminum phosphide poisoning from July 2019 to December 2020. During data collection, a particular emphasis was made on the poison formulation, the ingested dose, the reconstitution of poison, vomiting, and time intervals to initiate various treatments. The primary outcome was inhospital mortality.

**RESULTS:** Fifty-eight patients were enrolled (median age, 32 years; 37 males). The mean dose of the ingested poison was 6.56 (±5.42) g. The predominant formulation of poison was pellet ($n$ = 41), followed by powder ($n$ = 16). Twenty patients performed reconstitution of poison before consumption, and 13 stirred the poison while reconstituting. All patients but three developed vomiting after consumption. Inhospital mortality ($n$ = 23, 39%) was significantly high with a higher ingested dose ($P < 0.001$), nonstirred reconstitution before consumption ($P = 0.042$), fewer vomiting episodes ($P = 0.010$), a delay in detection of the victim by someone ($P = 0.001$), and delayed initiation of intravenous fluids ($P = 0.043$). The secondary outcomes (shock and requirement of vasopressor or ventilation) remained unaffected by the stirring in the reconstitution group.

**CONCLUSIONS:** Poison-related factors and time intervals determine early risk stratification at admission in aluminum phosphide poisoning.

**Keywords:**

Aluminum phosphide, ingested dose, poison forms, poisoning, poison-related factors, prognosis, reconstitution

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## Introduction

Aluminum phosphide is widely used as a fumigant pesticide against insects and rodents to preserve stored grains in the agricultural economies of low- and middle-income countries (LMICs). Because of its easy availability, it remains a common agent for acute poisoning in these communities. Because of rapid systemic toxicity and the unavailability of a specific antidote, acute aluminum phosphide poisoning is associated



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**Box-ED Section**

**What is already known on the study topic?**

- Acute aluminum phosphide poisoning is associated with high mortality. Early prediction of toxidrome severity is needed to improve outcomes by leading to prompt therapeutic interventions and intensive care unit transfer
- The prognosis of aluminum phosphide toxicity is usually based on clinical and laboratory features such as shock, metabolic acidosis, or altered mental status.

**What is the conflict on the issue? What is its importance for the readers?**

- The influence of poison-related factors (e.g., poison formulation, the ingested dose, reconstitution of poison before ingestion, or stirring with a spoon for reconstitution) is less well studied for the prognosis of aluminum phosphide poisoning
- Poison-related factors are often the only available predicting factors in resource-constraint settings. Moreover, the baseline parameters determine rapid triaging and identifying the patients who need priority for intensive care unit admission.

**How is this study structured?**

- This was a single-center, prospective, observational study conducted with 58 patients admitted to the ED of an academic hospital in North India.

**What does this study tell us?**

- Mortality was significantly high with a higher ingested, nonstirred reconstitution before consumption, fewer vomiting episodes, a delay in victim detection by someone, and delayed initiation of intravenous fluids
- Poison-related factors and time intervals determine early risk stratification at admission in aluminum phosphide poisoning.

with a mortality rate of 40%–80%.[1-5] The predominant manifestation of severe toxidrome is cardiotoxicity or circulatory failure.[6-8] Early recognition of systemic toxicity and immediate high-quality supportive care remain the mainstay of management.

The toxic dose is approximately 150–500 mg for an average-built adult.[9-11] The toxicity is primarily mediated by phosphine gas released from aluminum phosphide. Consumption of poison from a freshly opened container causes high toxicity because "exposed" forms lose their potency from reaction with atmospheric moisture with subsequent phosphine release.[10] Conversion into powder formulation usually results in lesser toxicity than the tablet or pellet form.

The prognosis of acute aluminum phosphide poisoning is usually based on clinical and laboratory features such as severe metabolic acidosis, refractory shock, cardiorespiratory failure requiring mechanical ventilation, or the presence of stupor or coma (low Glasgow Coma Scale [GCS]).[12-20] These toxidrome features are considered accurate and reproducible in predicting mortality. Furthermore, scoring tools based on these variables, such as the PGI score (representing low pH, low GCS score, and impaired or low systolic blood pressure), Acute Physiologic Assessment and Chronic Health Evaluation II score, or the Sequential Organ Failure Assessment score have demonstrated excellent predictive abilities.[21-24] In contrast, the influence of poison-related factors on prognosis is less well studied. They are often the only available factors in primary health-care settings in LMICs. Moreover, the baseline parameters determine rapid triaging and identifying patients who need priority for intensive care unit admission. Therefore, this study aimed to evaluate the effect of poison-related factors on prognosis in aluminum phosphide poisoning.

## Methods

### Study design

This is a prospective cohort study conducted at the medical emergency department (ED) of a tertiary care hospital and an apex referral center in North India from July 2019 to December 2020. About 100–120 patients are admitted daily to the ED of this 1948-bedded hospital from a large population of North India.[25,26] The age criterion for admission to our adult medical emergency is ≥13 years. The institutional ethics committee approved the study (No.: INT/IEC/2019/002071, date October 10, 2019). We obtained written informed consent from all study patients or legally authorized representatives. There was no funding source for the study.

### Study participants

All patients aged ≥13 years diagnosed with acute aluminum phosphide poisoning based on a history of compound ingestion with the presence of clinical features consistent with the toxidrome were enrolled. Patients who had consumed multiple compounds and those with a history of doubtful consumption of aluminum phosphide were excluded from the study. No case was excluded after recruitment.

### Data collection

Sociodemographic details, route of exposure, the intention of poisoning, and baseline clinical parameters were recorded. A particular emphasis was made on the poison formulation and the ingested dose, reconstitution of poison before ingestion, vomiting episodes, and time delay in contact with the health-care facility and the initiation of primary care. The data regarding

poison-related factors and various time intervals were collected from the patients or the primary caregivers if the patients were unfit to answer. Basic laboratory investigations included blood gas analysis, lactate, complete blood count, serum electrolytes, renal and liver function tests, international normalized ratio, electrocardiography, and chest X-ray.

The management of the study patients was according to the standard intuitional protocol [Figure 1]. Gastric lavage with 0.9% saline was done in patients who had ingested the compound 1–2 h before admission. The shock was managed with intravenous fluids and vasopressors. Noradrenaline was the initial vasopressor of choice. After initial resuscitation, the patients were shifted to an ED observation unit or intensive care unit based on the severity of the toxidrome and followed up throughout their hospital stay. The outcome (died or survived) and length of hospital stay were documented.

## Outcomes

The primary outcome was to study the effect of the following factors (available at admission) on inhospital mortality – (a) poison formulation (e.g., tablet and sachet), (b) the ingested dose (in grams), (c) additives used during consumption (e.g., water, alcohol, and juice), (d) reconstitution of poison before consumption and stirring with a spoon for reconstitution, (e) presence of vomiting and the total number of episodes, and (f) time elapsed in contact with the health-care facility and the initiation of treatment, i.e., gastric lavage and intravenous fluid administration.

The secondary outcomes were to detect the effect of reconstitution of the poison before ingestion on (a) the presence of shock on admission, (b) the requirement of vasopressors during hospitalization, and (c) the requirement of ventilation (e.g., noninvasive or invasive mechanical ventilation) during hospitalization.

## Statistical analysis

Statistical analysis was performed using the SPSS software version 22.0 (SPSS Inc., Chicago, IL, USA). The categorical variables were presented as frequencies and analyzed using Chi-square or Fisher's exact test. Quantitative variables were expressed in the form of the mean (± standard deviation [SD]) and median (interquartile range [IQR]) as applicable. The normality of quantitative variables was assessed using Kolmogorov–Smirnov test. For normally distributed variables, between-group comparisons were made using the unpaired Student's *t*-test. Mann-Whitney test was

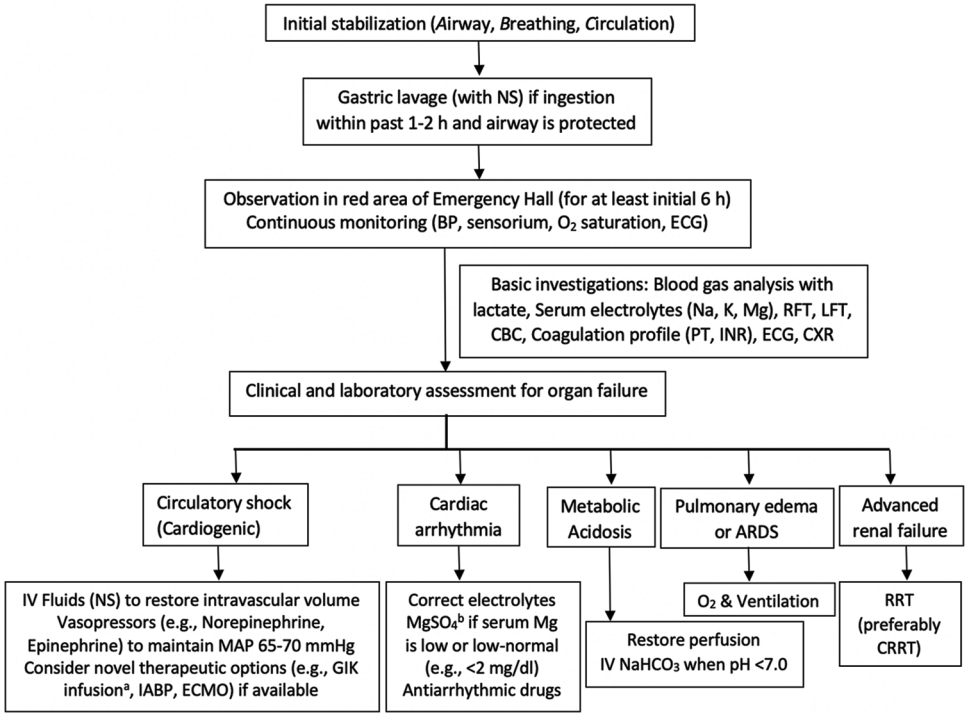


**Figure 1:** Management protocol for acute aluminum phosphide toxicity at our center (PGIMER, Chandigarh, India). [a]GIK protocol: Insulin regular loading dose of 0.5–1 IU/kg, followed by 0.5 IU/kg/h infusion (can be increased up to 10 IU/kg/h). Dextrose 0.5–1 g/kg loading dose, followed by 0.5–1 g/kg/h infusion; maintaining plasma glucose level between 140 and 180 mg/dL. IV K supplementation to maintain normal serum levels; monitoring levels every 8 h along with continuous ECG, [b]MgSO4 protocol: Loading dose 3 g over 3 h infusion, followed by 1.0-1.5 g every 6 h. ARDS: Acute respiratory distress syndrome, BP: Blood pressure, CBC: Complete blood count, CRRT: Continuous renal replacement therapy, CXR: Chest X-ray, ECG: Electrocardiography, ECMO: Extracorporeal membrane oxygenation, GIK: Glucose-insulin-potassium, IABP: Intra-aortic balloon counterpulsation, INR: International normalized ratio, IV: Intravenous, LFT: Liver function test, NS: Normal saline, PT: Prothrombin time, RFT: Renal function test, RRT: Renal replacement therapy

used for nonnormally distributed variables. Univariate analysis was performed to find factors associated with mortality. A two-sided $P < 0.05$ was considered statistically significant.

## Results

### Participants and baseline characteristics

A total of 58 patients were enrolled in the study. The median age was 32 years (range: 16–80 years), and most patients were 21–40 years ($n$ = 35, 60%). Table 1 shows the patients' baseline sociodemographic, clinical, and laboratory characteristics. The intention of the poisoning was mainly suicidal ($n$ = 55, 95%), followed by accidental ($n$ = 2) and homicidal ($n$ = 1).

The predominant formulation of poison consumed was pellet ($n$ = 41, 71%) [Table 2]. The mean ± SD (95% CI) dose of the ingested poison was 6.56 ± 5.42 (5.17–7.96) g. At least one-third ($n$ = 20) of patients gave a history of reconstitution of toxin (i.e., exposed form) before consumption and 13 of them stirred the poison while reconstituting. All patients but three developed vomiting after consumption. The median time elapsed for someone to find the victim and contact the first health-care facility was 30 min and 1 h, respectively. The median time interval between poison consumption and initiation of gastric lavage was 1.25 h [Table 2].

### Treatment and outcomes

During hospitalization, 40 (69.0%) patients needed vasopressor support for hypotension (vasopressors – single in 26, two in 13, and three in 1 patient). Ventilation was required in 33 (56.9%) patients in the form of invasive mechanical ventilation ($n$ = 24) and oxygen supplementation through a venturi mask ($n$ = 9). Four patients received renal replacement therapy for advanced azotemia or refractory metabolic acidosis. Inhospital mortality was 39% ($n$ = 23). The median duration of hospital stay was 1 day (IQR: 4 h–2.375 days, range: 2 h–21 days).

### Analysis of the outcomes

The primary outcome of this study was inhospital mortality, which was significantly high with a higher ingested dose ($P < 0.001$), nonstirred reconstitution before consumption ($P = 0.042$), less number of vomiting episodes after consumption ($P = 0.010$), a delay in detection of the victim by someone ($P = 0.001$), and delayed initiation of intravenous fluids ($P = 0.043$) [Table 2]. The secondary outcomes, i.e., shock at admission and requirement of vasopressor or ventilation during hospitalization, remained unaffected by the stirring with a spoon in the reconstitution group [Table 3]. The effect of baseline demographic, clinical, and laboratory characteristics on mortality is shown in Table 4.

**Table 1: Baseline sociodemographic, clinical, and laboratory characteristics of patients with acute aluminum phosphide poisoning ($n$=58)**

| Parameter | Value |
|---|---|
| Sociodemographic profile | |
| Age, median (IQR) | 32.0 (25.0–40.0) |
| Male gender, $n$ (%) | 37 (63.8) |
| Occupation | |
| Daily-wedge worker | 20 (34.5) |
| Housewife | 12 (20.7) |
| Student | 9 (15.5) |
| Unemployed | 5 (8.6) |
| Miscellaneous | 12 (20.7) |
| Clinical features, mean±SD (95% CI) | |
| Systolic blood pressure (mmHg) | 87.19±26.37 (80.40–93.97) |
| Diastolic blood pressure (mmHg) | 55.38±18.08 (50.73–60.03) |
| Mean arterial pressure (mmHg) | 66.47±20.46 (61.20–71.74) |
| Shock, $n$ (%) | 27 (46.6) |
| Pulse rate (/min) | 104.24±16.55 (99.98–108.50) |
| Respiratory rate (/min) | 20.90±5.01 (19.61–22.19) |
| GCS score, median (IQR; range) | 15 (15–15;3–15) |
| GCS score ≤8, $n$ (%) | 6 (10.3) |
| Laboratory parameters | |
| Blood pH, mean±SD (95% CI) | 7.24±0.19 (7.19–7.29) |
| Bicarbonate (mEq/L), mean±SD (95% CI) | 14.27±6.20 (12.67–15.87) |
| Lactate (mg/dL), mean±SD (95% CI) | 8.29±5.51 (6.87–9.71) |
| Hemoglobin (g/dL), mean±SD (95% CI) | 12.45±2.09 (11.91–12.99) |
| White blood cells (/mm$^3$), mean±SD (95% CI) | 13084.21±7072.80 (11263.98–14904.44) |
| Platelet count (×10$^3$/mm$^3$), mean±SD (95% CI) | 192.23±71.76 (173.76–210.70) |
| Plasma glucose (mg/dL), mean±SD (95% CI) | 138.30±72.70 (119.59–157.01) |
| Serum sodium (mEq/L), mean±SD (95% CI) | 140.65±4.08 (139.60–141.70) |
| Serum potassium (mEq/L), mean±SD (95% CI) | 3.82±0.70 (3.64–4.00) |
| Serum creatinine (mg/dL), mean±SD (95% CI) | 1.18±0.93 (0.94–1.42) |
| Serum bilirubin (mg/dL), mean±SD (95% CI) | 0.92±0.57 (0.77–1.07) |
| Alanine transaminase (IU/L), median (IQR) | 30.0 (16.5–56.5) |
| Aspartate transaminase (IU/L), median (IQR) | 37.0 (21.0–76.5) |
| Alkaline phosphatase (IU/L), median (IQR) | 89.0 (81.5–102.0) |
| INR, mean±SD (95% CI) | 1.17±0.19 (1.12–1.22) |
| Corrected QT interval (ms), mean±SD (95% CI) | 400.60±42.93 (389.55–411.65) |
| Prognostic scoring systems, median (IQR) | |
| PGI score, median (IQR) | 1 (0–2) |

*Contd...*

**Table 1: Contd...**

| Parameter | Value |
|---|---|
| PGI score>1, *n* (%) | 20 (34.5) |
| APACHE-II score, median (IQR) | 8.0 (2.0–14.25) |

PGI: pH, GCS, and Impaired systolic blood pressure. APACHE: Acute Physiologic Assessment and Chronic Health Evaluation, GCS: Glasgow Coma Scale, IQR: Interquartile range, SD: Standard deviation, CI: Confidence interval, INR: International normalized ratio

## Discussion

Determining prognosis at admission is crucial for rapid risk stratification or triaging patients with acute poisoning and identifying high-risk patients for intensive care unit transfer in LMICs. Our study evaluates poison-related factors and time intervals in detail. We found that the ingested dose, reconstitution of poison by stirring before consumption, number of vomiting episodes, and time delay to find the victim after ingestion or to administer intravenous fluids help provide prognosis at admission.

The dose of the poison consumed has historically been one of the most important prognostic factors in most toxidromes. Higher doses of aluminum phosphide lead to higher blood phosphine levels and subsequent inhibition of oxidative phosphorylation and anti-oxidant enzymes, resulting in free radical damage.[10,27,28] Like most studies, the dose was significantly associated with mortality in our data.[14-16,27] The inhospital mortality was 39% in our study, with a mean ingested dose of 6.5 g. In contrast, in previous studies, the ingested dose varied from 1.2 to 5.1 g, with a mortality rate ranging from 30% to 60%.[9,18,26,29] The reason for this discrepancy may be multifactorial such as consumption of expired formulations that exert lesser toxicity, differences in the method of consumption, including reconstitution, and early institution of treatment. The dose taken into consideration in our study is the actual dose of formulation consumed by the patients. The composition of aluminum phosphide in standard tablet or granulation form available in North India is 56%. In contrast, the previous studies have either considered the effective dose of toxin or did not mention the toxin or formulation.[9,18,26,29] Although government protocols have dictated standardization of tablet and granulation formulations into 3 and 10 g, their easy accessibility to the general public makes aluminum phosphide a typical "suicide poison."[6] More steps need to be taken to reduce the dose of formulations available in the market and restrict their availability to select groups.

Tablets or pellets are considered more toxic than powder or granules because of lower surface area, reduced exposure to the environment, and higher phosphine release in the body. We also found a severe toxidrome with tablet forms; however, it was not statistically significant. Similarly, reconstitution of the poison before ingestion (exposed form) resulted in better outcomes but did not reach a level of significance, as demonstrated in a few other studies.[9,15] The mortality was 1.9 times higher in patients who had ingested the nonreconstituted form. It was hypothesized that reconstitution with stirring leads to the escape of phosphine, resulting in a reduced systemic burden of the toxin. Although an association was demonstrated in our study, this interpretation is difficult to determine given the smaller sample size and the power of the reconstitution group. Moreover, stirring did not influence the incidence of shock and the requirement of vasopressor or ventilation in the reconstitution group. Future studies should elucidate and verify the prognostic significance of stirring.

Vomiting after consumption tends to remove the poison from the gastrointestinal tract before releasing phosphine, resulting in reduced systemic absorption of the toxin. Many studies demonstrated that the presence of vomiting, its early occurrence after consumption, and the number of episodes are associated with a less severe toxidrome.[16,27] In our study, patients with more vomiting episodes before admission had better outcomes than those with fewer.

Early recognition of a toxidrome and initiation of appropriate treatment are two essential factors in any poisoning because a delay worsens systemic toxicity.[30] In this study, the detection of the victim the first time after poison ingestion and initiation of intravenous fluid were two critical time intervals. Initial fluid resuscitation is often required to restore intravascular volume in acute aluminum phosphide poisoning as the toxin disrupts the vascular wall integrity, leading to congestion of vital organs, transudation of fluid into the serous cavities, and inadequate response to vasopressors.[10,27] The time intervals to contact the first health-care facility and gastric lavage were also higher in the nonsurvivor group but were not significant. Overall, the time elapsed to reach the first health-care facility after poison exposure was significantly less in our study compared to our center's previous data of 2009 (1 h vs. 3 h).

### Limitations

This study has three main limitations. First, a single-center experience and tertiary hospital referral bias lack the generalizability of the results. Second, we caution that some poison-related information and time intervals were collected from previous hospital records, and there could have been some differences in their subjective interpretation. Third, the sample size was relatively small, partly because of the coronavirus disease 2019 pandemic.[26] Therefore, we extended the patient enrollment period from 1 year to 1.5 years to increase the sample size. Our study incites larger multicentered studies, including primary health centers, to establish

**Table 2: Effect of poison-related factors and consumption patterns on mortality in aluminum phosphide poisoning (*n*=58)**

| Parameters | Total (*n*=58) | Died (*n*=23) | Survived (*n*=35) | *P* |
|---|---|---|---|---|
| Poison formulation, *n* (%) | | | | |
| Pellet | 41 (70.7) | 18 (78.3) | 23 (65.7) | 0.728 |
| Powder | 16 (27.6) | 5 (21.7) | 11 (31.4) | |
| Liquid | 1 (1.7) | 0 | 1 (2.9) | |
| Dose of poison (g), mean±SD (95% CI) | 6.56±5.42 (5.17–7.96) | 10.11±6.19 (7.58–12.64) | 4.16±3.10 (3.13–5.19) | <0.001 |
| Additives used during consumption | | | | |
| Water | 53 (91.4) | 22 (95.7) | 31 (88.6) | 0.783 |
| Alcohol | 4 (6.9) | 1 (4.3) | 3 (8.6) | |
| Juice | 1 (1.7) | 0 | 1 (2.8) | |
| Reconstitution of the poison before ingestion, *n* (%) | | | | |
| Yes (exposed form) | 20 (34.5) | 5 (21.7) | 15 (42.9) | 0.098 |
| No (unexposed form) | 38 (65.5) | 18 (78.3) | 20 (57.1) | |
| Stirring with spoon for reconstitution | *n*=20 | *n*=5 | *n*=15 | |
| Yes | 13 (65.0) | 2 (40.0) | 11 (73.3) | 0.042 |
| No | 7 (35.0) | 3 (60.0) | 4 (26.7) | |
| Vomiting after poison ingestion | | | | |
| Present, *n* (%) | 55 (94.8) | 22 (95.6) | 33 (94.3) | 1.000 |
| Numbers of episodes, mean±SD (95% CI) | 5.84±3.91 (4.83–6.85) | 4.26±2.28 (3.33–5.19) | 6.89±4.40 (5.43–8.35) | 0.010 |
| Time interval between poison consumption and treatment initiation (h), median (IQR) | | | | |
| Detection of the victim by someone | 0.50 (0.25–0.94) | 0.75 (0.50–1.0) | 0.25 (0.17–0.5) | 0.001 |
| Contact with first health-care facility | 1.0 (0.50–2.0) | 1.0 (1.0–3.12) | 1.0 (0.5–2.0) | 0.089 |
| Initiation of gastric lavage | 1.25 (0.67–2.58) | 1.25 (0.75–4.0) | 1.25 (0.67–2.19) | 0.312 |
| Initiation of intravenous fluids | 1.25 (5.0–15.0) | 2.0 (1.0–3.5) | 1.0 (0.67–2.0) | 0.043 |

IQR: Interquartile range, SD: Standard deviation, CI: Confidence interval

**Table 3: Effect of reconstitution of poison before consumption on secondary outcomes**

| Parameter | Reconstitution | | *P* | Stirring with spoon for reconstitution | | *P* |
|---|---|---|---|---|---|---|
| | Yes (*n*=20), *n* (%) | No (*n*=38), *n* (%) | | Yes (*n*=13), *n* (%) | No (*n*=7), *n* (%) | |
| Shock at admission | 9 (45.0) | 18 (52.6) | 0.864 | 7 (53.8) | 2 (28.6) | 0.374 |
| Need of vasopressor | | | | | | |
| Any | 13 (65) | 27 (71.1) | 0.636 | 9 (69.2) | 4 (57.1) | 0.651 |
| Number of vasopressors | *n*=13 | *n*=27 | | *n*=9 | *n*=4 | |
| 1 | 8 (61.5) | 18 (66.7) | 1.000 | 6 (66.7) | 2 (50.0) | 1.000 |
| >1 | 5 (38.5) | 9 (33.3) | | 3 (33.3) | 2 (50.0) | |
| Need of ventilation | | | | | | |
| Any | 9 (45.0) | 24 (63.2) | 0.184 | 5 (38.5) | 4 (57.1) | 0.642 |
| Mode of ventilation | *n*=9 | *n*=24 | | *n*=5 | *n*=4 | |
| Invasive | 5 (55.6) | 19 (79.2) | 0.212 | 2 (40.0) | 3 (75.0) | 0.524 |
| $O_2$ through venturi mask | 4 (44.4) | 5 (20.8) | | 3 (60.0) | 1 (25.0) | |

the prognostic role of poison-related factors and time delays in aluminum phosphide poisoning.

## Conclusions

Our study demonstrates the prognostic significance of poison-related factors such as ingested dose, stirring for reconstitution, number of vomiting episodes, and time delay to the first detection of the victim or intravenous fluid administration. These factors are immediately available at admission and, along with toxidrome features, might improve early risk stratification of aluminum phosphide poisoning. The significant mortality and higher dose of ingested toxin in our study reflect easy access of the poison to the general public in our area and require tailored government regulations.

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**Author contributions**

LAC: Data curation (lead), formal analysis (supporting), and writing – original draft (lead).

AKP: Conceptualization (supporting), methodology (supporting), formal analysis (lead), writing – original draft (supporting), writing – review, and editing (lead).

**Table 4: Effect of baseline demographic, clinical, and laboratory characteristics on mortality in aluminum phosphide poisoning (*n*=58)**

| Parameters | Died (*n*=23) | Survived (*n*=35) | *P* |
|---|---|---|---|
| Demographic data | | | |
| Age (years) | 34 (26.5–43.5) | 30 (23–38.5) | 0.141 |
| Male gender, *n* (%) | 16 (69.6) | 21 (60.0) | 0.458 |
| Clinical features, mean±SD (95% CI) | | | |
| Systolic blood pressure (mmHg) | 70.13±30.22 (57.78–82.48) | 98.40±15.76 (93.18–103.62) | <0.001 |
| Diastolic blood pressure (mmHg) | 44.96±20.75 (36.48–53.44) | 62.23±12.15 (58.21–66.26) | 0.001 |
| Mean arterial pressure (mmHg) | 53.70±23.15 (44.24–63.16) | 74.86±13.13 (70.51–79.21) | 0.001 |
| Shock, *n* (%) | 17 (73.9) | 10 (28.6) | <0.001 |
| Pulse rate (/min) | 108.96±15.50 (102.63–115.30) | 101.14±16.70 (95.61–106.67) | 0.075 |
| Respiratory rate (/min) | 21.13±5.99 (18.68–23.58) | 20.74±4.34 (19.30–22.18) | 0.340 |
| GCS score, median (IQR) | 15 (9–15) | 15 (15–15) | 0.021 |
| Laboratory parameters | | | |
| Blood pH, mean±SD (95% CI) | 7.11±0.18 (7.04–7.18) | 7.32±0.15 (7.27–7.37) | <0.001 |
| Bicarbonate (mEq/L), mean±SD (95% CI) | 9.86±4.11 (8.18–11.54) | 17.16±5.63 (15.30–19.03) | <0.001 |
| Lactate (mg/dL), mean±SD (95% CI) | 11.02±5.14 (8.92–13.12) | 6.37±4.99 (4.72–8.02) | 0.002 |
| Hemoglobin (g/dL), mean±SD (95% CI) | 12.31±2.49 (11.29–13.33) | 12.54±1.84 (11.93–13.15) | 0.718 |
| White blood cells (/mm$^3$), mean±SD (95% CI) | 13309.09±6524.31 (10642.73–15975.45) | 12942.86±7486.59 (10462.60–15423.13) | 0.743 |
| Platelet count (×10$^3$/mm$^3$), mean±SD (95% CI) | 172.05±62.05 (146.69–197.41) | 204.91±75.33 (179.95–229.87) | 0.079 |
| Plasma glucose (mg/dL), mean±SD (95% CI) | 163.65±91.32 (126.33–200.97) | 121.15±51.57 (104.07–138.24) | 0.055 |
| Serum sodium (mEq/L), mean±SD (95% CI) | 141.23±4.61 (139.35–143.11) | 140.29±3.73 (139.05–141.53) | 0.235 |
| Serum potassium (mEq/L), mean±SD (95% CI) | 3.67±0.72 (3.38–3.96) | 3.91±0.69 (3.68–4.14) | 0.107 |
| Serum creatinine (mg/dL), mean±SD (95% CI) | 1.43±0.77 (1.11–1.75) | 1.02±1.00 (0.69–1.35) | <0.001 |
| Serum bilirubin (mg/dL), mean±SD (95% CI) | 1.07±0.61 (0.82–1.32) | 0.83±0.54 (0.65–1.01) | 0.037 |
| Alanine transaminase (IU/L), median (IQR) | 35 (18.5–90.5) | 25 (17.0–43.5) | 0.324 |
| Aspartate transaminase (IU/L), median (IQR) | 58.5 (18.0–90.0) | 31 (21.5–50.0) | 0.377 |
| Alkaline phosphatase (IU/L), median (IQR) | 94.5 (86.0–101.5) | 86 (80.5–102.0) | 0.441 |
| INR, mean±SD (95% CI) | 1.23±0.21 (1.14–1.32) | 1.13±0.17 (1.07–1.19) | 0.004 |
| Corrected QT interval (ms), mean±SD (95% CI) | 423.56±58.40 (399.70–447.430) | 389.79±28.48 (380.36–399.23) | 0.053 |
| Prognostic scoring systems | | | |
| PGI score, median (IQR) | 2 (1–3) | 0 (0–1) | <0.001 |
| PGI score>1, *n* (%) | 17 (73.9) | 3 (8.6) | <0.001 |
| APACHE-II score, median (IQR) | 16.0 (10.0-22.0) | 3.0 (2.0-9.0) | <0.001 |

APACHE: Acute Physiologic Assessment and Chronic Health Evaluation, GCS: Glasgow Coma Scale, IQR: Interquartile range, SD: Standard deviation, CI: Confidence interval, INR: International normalized ratio, PGI: pH, GCS and Impaired systolic blood pressure

AB: Conceptualization (lead), methodology (lead), writing – review, and editing (supporting).

DPD: Conceptualization (supporting) and methodology (supporting).

NS: Conceptualization (supporting) and methodology (supporting).

### Conflicts of interest
None declared.

### Ethical approval
The Institutional Ethics Committee of Postgraduate Institute of Medical Education and Research, Chandigarh (India) approved the study (No.: INT/IEC/2019/002071, date October 1, 2019).

### Consent to participate
Written informed consent was taken from all study patients or a legally authorized representative (if the patient could not consent). The study patients were explained about confidentiality and their information will be used for educational purposes only.

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