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ORIGINAL ARTICLE

Clinical Characteristics and Associated Factors of Poor Outcome in Zinc Phosphide Poisoning; An Exploratory Analysis of 68 Cases

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Abstract: **Introduction:** Zinc phosphide (ZnP) poisoning may be considered a lethal toxicological emergency, although there is very little zinc-specific data concerning its prognosis. This study aimed to describe the clinical pattern of ZnP poisoning and determine admission criteria associated with poor clinical outcome. **Methods:** This retrospective cross-sectional study included patients with clinically diagnosed oral ZnP poisoning referred to hospital during 2020-2025. We extracted all demographic, clinical, biochemical, imaging, electrocardiographic, therapeutic, and outcome variables from the medical records. Poor outcome was defined as need for ICU admission, in-hospital mortality, or hospital stay exceeding 72 hours. Univariable exploratory logistic regression analysis was performed to calculate the crude OR and 95% CI. Multivariable logistic regression was not done because only 12 poor-outcome events occurred, and some candidate variables showed complete or quasi-complete separation. Sensitivity analyses included (1) an alternate outcome of severe illness, which is hospital death, need for vasopressors, or intubation; and (2) the complete case analysis without DAMA. **Results:** 68 consecutive cases of ZnP poisoning with a mean age of 32.79 ± 13.46 years were studied (61.8% male and 86.8% intentional). The most frequent symptoms were nausea (61.8%), vomiting (47.1%), generalized weakness (38.2%), and abdominal pain (33.8%). Poor clinical outcome occurred in 12 patients (17.6%; 95% CI: 10.4%-28.4%); all 12 were admitted to the ICU. Five had a hospital stay exceeding 72 hours, and two died in hospital (2.9%; 95% CI: 0.8%-10.1%). Poor outcome was associated with the reported higher-ingestion ($p < 0.001$), BUN ($p = 0.007$), creatinine ($p = 0.008$), transaminases (AST: $p = 0.027$; ALT: $p = 0.001$), CPK ($p = 0.001$), and base deficit ($p = 0.005$), hypotension (systolic: $p = 0.006$; diastolic: $p < 0.001$), lower bicarbonate ($p = 0.002$), prolonged PT/INR (PT: $p = 0.032$; INR: $p = 0.034$), abnormal ECG ($p < 0.001$), abnormal ECG ($p < 0.001$) and radiographic findings ($p = 0.029$), and bicytopenia/pancytopenia ($p = 0.004$). Five patients met the alternative severe endpoint. Excluding 10 DAMA patients left 58 patients and 9 poor-outcome events; the overall direction of the main associations remained similar. **Conclusion:** Easily identifiable admission features included hemodynamic instability, metabolic acidosis, abnormal laboratory findings suggesting organ dysfunction, ECG or X-ray abnormal findings, and cytopenia; all indicated poor prognosis. Given the small sample size, these features may reflect severity rather than poor prognosis.

Keywords: Phosphides; Poisoning; Pesticides; Emergency Medicine; Prognosis; Risk Assessment; Cohort Studies.

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1. Introduction

Zinc phosphide (ZnP), a metal phosphide rodenticide used for agricultural, urban, and industrial pest control, is available in certain low- and middle-income regions, presenting a significant opportunity for deliberate poisoning; however, the epidemiology is highly variable between regions, product formulations, and referral routes (1-6). Data on ZnP poisoning from recent studies of Iranian and Thai populations, as well as a mixed metal-phosphide case series from Egypt, reveal that the majority of patients are young to middle-aged adults who have ingested the drug deliberately; however, mortality rates vary widely between studies (5, 7, 8).

ZnP can form phosphine once ingested into the gastrointestinal tract. Phosphine inhibits mitochondrial energy generation, causes oxidative stress and lipid peroxidation, and leads to cellular hypoxia and multi-organ damage (1, 2, 6). While gastrointestinal symptoms usually appear first, severe intoxication can lead to such complications as hypotension, cardiac dysfunction, arrhythmias, acidosis, pulmonary edema, acute kidney injury, liver damage or acute liver failure, coagulopathy, and death (1, 2, 6, 8-11). Even minor or vague symptoms cannot exclude future deterioration.

No definite antidotes are described for this condition. Management involves rapid stabilization, gastrointestinal decontamination if indicated, hemodynamic and electrocardiographic monitoring, correction of metabolic and electrolyte abnormalities, treatment of shock and organ dysfunction, and critical care (1, 6, 12). Evidence for oil-based lavage, N-acetylcysteine, high-dose insulin/euglycemia therapy, and extracorporeal support is derived predominantly from aluminum phosphide poisoning. It is limited by heterogeneity, small studies, and risk of bias (13-17). A recent small randomized double-blind trial

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in ZnP poisoning found numerically lower mortality with intravenous N-acetylcysteine. Still, the difference was not statistically significant, reinforcing the need for larger zinc-specific trials (18).

Although several reports describe ZnP poisoning manifestations, relatively few studies have systematically evaluated zinc-specific early factors associated with clinical deterioration (5,8,10,20). This study therefore aimed to describe the demographic, clinical, laboratory, radiologic, and electrocardiographic findings, as well as the management and outcome profile of patients admitted with ZnP poisoning and to explore admission factors associated with poor clinical outcome.

2. Methods

2.1. *Study design and setting*

We recruited all eligible patients with ZnP poisoning admitted to Kosar Hospital, Karaj, Iran, from 2020 to 2025 for this retrospective cohort study. We used a cohort design because clinical and admission factors occurred before the in-hospital outcomes. The predictive factors of outcomes were explored from demographic, clinical, laboratory, radiologic, electrocardiographic, and management variables. Consecutive sampling was used to reduce selection bias. We did not perform a priori sample-size calculation because this exploratory retrospective study included all eligible patients available during the study period. This research was done in accordance with the Declaration of Helsinki and received approval from the Research Ethics Committee of Alborz University of Medical Sciences (IR.ABZUMS.REC.1404.126).

2.2. *Participants*

The study subjects were poisoned individuals with a history of ZnP exposure and documented symptoms, laboratory or/and radiographic signs, and sufficient medical-record data for analysis. The exclusion criteria were the absence of any contact with the poison, non-acute or combined poisoning, absence of sufficient medical history, and heart, liver, or kidney disease.

2.3. *Data collection and outcome definition*

We extracted data from medical records using the data extraction form created before the analysis. Variables include demographic and exposure variables, including age, sex, marital status, education status, place of residence, motive for poisoning, method of exposure, quantity of poison, and time to hospital admission. We extracted symptom, vital sign, and Glasgow Coma Scale values from the first documented clinical assessment after hospital admission. The laboratory parameters, including blood gases, bicarbonate, base deficit, renal function, liver enzymes, coagulation studies, creatine phosphokinase (CPK), and blood cell abnormalities, were collected based on the first results obtained after admission. ECG findings and chest and abdominopelvic X-ray interpretations were coded based on the first medical record interpretation obtained.

We pre-specified ingestion in two source-record categories: <10 g and ≥10 g; the 10-g threshold reflected how the information was documented and was not the outcome-selected cut-point. The time taken for patients to reach the hospital was noted as <1 hour, 1-3 hours, or >3 hours. This categorization was based on the source categories.

Predefined candidate admission covariates included amount ingested category, systolic/diastolic blood pressure, bicarbonate, base excess, BUN, creatinine, PT, INR, AST, ALT, CPK, electrocardiogram findings, chest X-ray findings, abdominopelvic X-ray findings, and bicytopenia/pancytopenia. Age, dose category, and delay to presentation were identified as potential confounders with clinical significance. Dose category and delay to presentation were also identified as potential effect modifiers; however, we did not conduct interaction tests or adjust for multiple covariates because of the small number of outcome events (n=12) and complete/quasi-complete separation.

Steps to minimize possible biases were considered wherever possible through inclusion of cases consecutively, use of a predetermined abstraction form, application of the same first evaluation criterion to all patients, and sticking to predetermined source-record categories as opposed to determining cut points from the results. As this research was retrospective, it was not possible to measure prospectively, blind reassess electrocardiograms and X-rays, and control all confounding factors.

2.4. *Outcomes*

We defined in-hospital mortality, ICU admission, or stay>72 hours as the primary outcomes. However, we included the component endpoints because of potential overlap and possible selection bias for the ICU endpoint.

2.5. *Statistical analysis*

Categorical variables were represented using frequencies and percentages. Continuous variables were reported using mean ± SD and range in the entire study population, while median (IQRs) was used to compare groups. The main comparison was done between subjects with a poor-outcome event and those without a poor-outcome event. Because several laboratory variables were skewed and the poor-outcome group was small, continuous variables were compared using the Mann-Whitney U test and Fisher's exact test. Wilson 95% CIs were calculated for the proportions of poor clinical outcome and in-hospital mortality. No data were missing for the variables used in the analyses. We performed simple exploratory logistic regression for each specified admission factor: quantity of substance ingested, systolic and diastolic blood pressure, bicarbonate, base excess, BUN, creatinine, PT, INR, AST, ALT, CPK, ECG results, chest X-ray, abdominopelvic X-ray, and bicytopenia/pancytopenia. CRUDE ORs and 95% CIs were calculated using units with clinical importance. For AST, ALT, and CPK, log2 transformation was used because the data were highly skewed, so ORs were calculated for a doubling of the respective biomarker. Binary variables with zero cells were corrected by the Haldane-Anscombe correction method. Multivariable logistic regression was not done because only 12 poor-outcome events occurred, and some candidate variables showed complete or quasi-complete separation. Multiple testing was not controlled for; thus, all association analyses were considered exploratory. Two sensitivity analyses were performed. First, we defined the alternative severe endpoint as in-hospital mortality, vasopressors, or intubation to minimize reliance on ICU admission, since only five patients met this endpoint. Second, we excluded all 10 patients discharged against medical advice, leaving 58 patients and 9 primary composite events; this complete-case sensitivity analysis was also limited to between-group comparisons and was not used to fit a new prediction model. All

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tests were two-tailed, and P<0.05 was taken as statistically significant. All analyses were done in SPSS version 23.

3. Results

3.1. *Patient characteristics*

In total, there were 68 patients clinically diagnosed with ZnP poisoning. The average age was 32.79 ± 13.46 (range: 15 - 68) years. Out of this group, 42 (61.8%) were male, 44 (64.7%) were unmarried, and 65 (95.6%) lived in urban localities. Intentional ZnP poisoning took place in 59 (86.8%) cases. Time to reach the hospital of≥3 hours was noted in 41 patients (60.3%). The high dose intake category (≥10 g) was found in 13 (19.1%) patients (Table 1).

3.2. *Clinical, laboratory, radiologic, and ECG findings*

The most common presenting symptoms were nausea in 42 (61.8%) patients, vomiting in 32 (47.1%), generalized weakness in 26 (38.2%), and abdominal pain in 23 (33.8%). Seven patients (10.3%) were asymptomatic at referral. Electrocardiographic abnormalities occurred in 15 (22.1%) patients, including sinus tachycardia in 5 cases, T inversion in 6 cases, QRS widening in 2 cases, prolonged QT interval in 1 case, and atrial fibrillation (AF) rhythm with rapid ventricular response in 1 case. Chest X-ray revealed bilateral pulmonary infiltration in 2 (2.9%) patients, and abdominopelvic X-ray showed radiopaque/opacity findings in 11 (16.2%) patients (Table 2).

3.3. *Management and clinical outcomes*

The most common decontamination method was polyethylene glycol, administered to 41 (60.3%) patients, while gastric lavage and activated charcoal were applied to 11 (16.2%) and 9 (13.2%) cases, respectively. A composite poor clinical outcome occurred in 12 (17.6%; 95% CI: 10.4%-28.4%) patients. All 12 patients with a poor outcome were admitted to the ICU; five had a hospital stay longer than 72 hours, and two died in hospital (2.9%; 95% CI: 0.8%-10.1%). These components overlapped. Ten patients were discharged against medical advice, and their post-discharge clinical status was unavailable (Table3).

3.4. *Factors associated with poor clinical outcome*

Compared with patients without the composite outcome, patients with poor clinical outcome had lower systolic and diastolic blood pressures (systolic: $p = 0.006$; diastolic: $p < 0.001$), lower bicarbonate ($p = 0.002$), a greater base deficit ($p = 0.005$), higher BUN ($p = 0.007$) and creatinine ($p = 0.008$), prolonged PT/INR (PT: $p = 0.032$; INR: $p = 0.034$), and higher AST ($p = 0.027$), ALT ($p = 0.001$), and CPK ($p = 0.001$) levels. The higher-ingestion category ($p < 0.001$), abnormal ECG ($p < 0.001$), abnormal abdominopelvic X-ray ($p < 0.001$), abnormal chest radiography ($p = 0.029$), and bicytopenia/pancytopenia ($p = 0.004$) were also associated with poor clinical outcome (Table 4).

In exploratory univariable logistic regression, each 10-mmHg decrease in systolic blood pressure was associated with higher odds of poor clinical outcome (OR: 1.88; 95% CI: 1.22-2.89), as was each 5-mEq/L decrease in bicarbonate (OR: 4.10; 95% CI: 1.75-9.60), each 10-mg/dL increase in BUN (OR: 2.77; 95% CI: 1.47-5.22), and an abnormal ECG (OR: 51.00; 95% CI: 8.65-300.74). All estimates were unadjusted and exploratory and should not be interpreted as independent predictors (Table 4).

3.5. *Sensitivity analysis*

Because the composite endpoints in the primary analysis related to ICU admission and utilization and may have been influenced by local practices and limited resources, we conducted a sensitivity analysis using objective outcomes such as in-hospital death, vasopressor use, or intubation. Five people (7.4%) had these outcomes. The trends in the primary analysis severity-at-admission variables were consistent with the sensitivity analysis. They included increased dose reported, abnormal ECG and radiologic findings, decreased blood pressure and bicarbonate, increased base deficit, and evidence of injury to the kidney, liver, coagulation, and muscle systems (Table 5). Given the limited sample size, these results are preliminary and not an independent prediction model.

After excluding all 10 patients discharged against medical advice, 58 patients remained; 9 (15.5%) had reached the composite endpoint, and 49 had not. The direction of the primary group difference was similar to that seen in the primary analysis for dose category, ECG, and X-ray findings, blood pressure, acid-base status, renal function, coagulation studies, transaminase levels, and CPK (Table 6). Because only nine poor-outcome events remained, this was an exploratory analysis, and we did not run a regression model.

4. Discussion

In this single-center retrospective cross-sectional study, gastrointestinal manifestations were the most frequent initial features of ZnP poisoning. In contrast, the poor-outcome group was associated with hemodynamic instability, metabolic acidosis, kidney and liver biochemistry disturbances, coagulopathy, electrocardiogram abnormalities, radiographic abnormalities, and cytopenia. The key finding is not that these factors are independent predictors, but that they can serve as reasonable early predictors of severity and should be confirmed in future studies.

The cohort was relatively young (mean age, 32.79 years), predominantly male (61.8%), and characterized by a high proportion of intentional ingestion (86.8%). These findings broadly resemble the 127-patient northern Iranian ZnP cohort reported by Ghasempouri et al., in which the mean age was approximately 25.5 years and 55.9% were male (5), and the Thai poison-center cohort of 455 patients, in which the mean age was 39.91 years, 60.5% were male, and 84.4% of exposures were intentional (8). In contrast, a large Egyptian mixed aluminum/zinc phosphide series contained a greater proportion of females and adolescents or young adults, with suicidal exposure in 78.6% (7). Differences in product access, referral patterns, inclusion criteria, and the proportion of aluminum versus zinc phosphide probably contribute to this geographic heterogeneity.

Nausea, vomiting, weakness, and abdominal pain predominated in the present study. Ghasempouri et al. reported nausea/vomiting in approximately 48% and abdominal pain in 25.9% of patients (5), while the Thai cohort reported gastrointestinal manifestations in 68.8% (8). The similar pattern supports direct gastrointestinal irritation and local phosphine generation as early mechanisms (1, 2, 6). However, seven patients in the present cohort were asymptomatic at referral, and the Thai study also found that many patients initially had normal vital signs and laboratory findings (8). Therefore, absence of prominent symptoms at first assessment should not be used alone to justify early discharge.

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In-hospital mortality in this cohort was 2.9% (95% CI, 0.8%-10.1%), compared with approximately 0.8% in the northern Iranian cohort (5) and 7% in Thailand (8). Hassanian-Moghaddam et al. reported four deaths among 102 patients (3.9%) in a radiography-focused Iranian ZnP series (10). These crude comparisons should be interpreted cautiously because case severity, formulation and dose, transfer delay, treatment protocols, and outcome ascertainment differed. The present composite poor-outcome rate of 17.6% is not directly comparable with mortality-based endpoints. Because all 12 composite events involved ICU admission, the endpoint largely reflected the need for ICU-level care and may also have been influenced by local ICU admission practices.

Among the clinical parameters that correlated with a bad outcome were lower systolic and diastolic blood pressure and metabolic acidosis. The findings align with the results from the Thai ZnP group, where low blood pressure and metabolic acidosis predicted mortality (8), as well as general phosphide poisoning research linking shock indices and metabolic abnormalities to disease severity (19, 20). On the mechanism side, myocardial depression, vasoplegia, impaired oxidative phosphorylation, and tissue hypoperfusion can potentially work together and result in shock and metabolic acidosis (1, 2, 6). Since shock indices have mostly been assessed for aluminum phosphide poisoning, direct extrapolation to ZnP is not justified.

Elevated BUN and creatinine, increased AST and ALT, prolonged PT/INR, and increased CPK in the poor-outcome group indicate multisystem involvement. Renal dysfunction could be due to impaired perfusion, direct toxicity, or both. Liver dysfunction is also biologically plausible and clinically relevant because delayed acute liver failure has been reported after ZnP intoxication, including cases requiring liver transplantation and recent reports of fulminant liver failure (11, 21).

The association between radiopaque material on abdominopelvic radiography and poor clinical outcome is particularly notable: 10 of 12 patients with poor outcome had an abnormal abdominal film, compared with 1 of 56 patients in whom no composite poor-outcome event was observed. In a 102-patient Iranian study, plain abdominal radiography had 81% sensitivity, 89% specificity, 47% positive predictive value, and 97% negative predictive value for ZnP ingestion, and positive films were associated with complications or death (10). The current results are consistent with the hypothesis that visible retained product may indicate a larger or more sustained gastrointestinal toxic burden.

Abnormal ECG findings were also strongly associated with poor clinical outcome, consistent with the known myocardial and electrophysiologic toxicity of phosphine (1, 8, 9). The very large univariable OR should not be interpreted as a precise effect size: only 15 patients had an abnormal ECG, the confidence interval was wide, and the estimate was not adjusted for blood pressure, acidosis, or dose. A more informative future analysis would classify specific ECG abnormalities, report their timing, and evaluate whether ECG findings add prognostic information beyond simple hemodynamic and acid-base markers.

For this study, treatment information is descriptive. Findings mention the frequency of treatments such as polyethylene glycol, gastric lavage, and activated charcoal. However, it does not present tables comparing indications, timing, dosage, contraindications, and outcomes across treatments. Therefore, no causal conclusions about the efficiency of treatments can be made. Recent systematic reviews of oil-based lavage, intravenous N-acetylcysteine, insulin-based therapy, and extracorporeal support suggest potential benefit for patients with severe aluminum phosphide poisoning, but their applicability to zinc-specific cases and efficacy remain uncertain (13-17). The ZnP study carried out in 2026 reported mortality rates of 6.7% compared to 20.0% with intravenous N-acetylcysteine in 60 patients (18). These interventions should therefore be interpreted within the context of supportive or rescue care rather than as established zinc-specific therapies.

Clinically, the combined evidence supports periodic reassessment of blood pressure, mental status, arteries/veins blood gas, kidney and liver function, clotting parameters, glucose, ECG, and abdominal radiography when clinically indicated. Those with low blood pressure, worsening acidosis, abnormal ECG, or signs of organ damage biochemically or radiologically are candidates for careful monitoring and possibly early toxicology/critical care consultation. Using the severe-endpoints sensitivity analysis as an alternative decreased the role of ICU admission, while the use of DAMA-exclusion sensitivity analysis as an alternative reduced the impact of the unclear post-discharge status; however, both demonstrated the same direction of findings, albeit with small numbers of events.

4.1. *Limitations*

This research has several advantages, including consecutive case selection for five years, specific attention to zinc, and analysis of multisystem clinical and laboratory abnormalities. However, it also has several limitations. The single-center, retrospective design makes the study susceptible to selection and information bias and therefore limits the external validity of the results, as they may not apply to settings where ZnP is produced using other methods. Exposure was clinically rather than analytically confirmed, the reported ingested amount was subject to recall and measurement error, and only admission laboratory values were analyzed. The composite poor clinical outcome included ICU admission, death during hospital stay, or hospitalization longer than 72 hours; the events overlapped, and all 12 composite events required ICU admission, making the hospital's ICU admission practice likely to affect the outcome. Ten patients (14.7%) were discharged against medical advice, without any post-discharge follow-up. Three of them had already experienced the composite primary endpoint event before discharge. At the same time, the other seven patients (10.3% of the total sample) did not have a composite in-hospital event, but their subsequent status was unknown. If any of these seven patients experienced an adverse development after discharge, this could have undercounted poor outcomes and underestimated the true percentage of poor outcomes. Bias direction and magnitude are hard to predict because they depend on the relationship between DAMA and further deterioration and on admission severity measures. Sensitivity analysis using the complete-case strategy, excluding all 10 DAMA patients, did not change the direction of our results. However, the number of analyzed patients decreased to 58, with only 9 poor outcome events. In addition, few or no cells, few primary and sensitivity analysis events, no multivariate adjustment, and no adjustment for multiplicity resulted in wide confidence intervals and unstable results.

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5. Conclusions

The features of admission that were associated with poor prognosis in cases of ZnP poisoning included hemodynamic instability, metabolic acidosis, kidney failure and liver dysfunction, coagulopathy, increased CPK levels, cytopenia, and changes on ECG or X-ray. Because the analysis was not adjusted and was exploratory, and because it was based on only 12 cases of primary outcome events, these results should not be viewed as independent predictors. Further studies with larger sample sizes are required for validation of these results.

6. Declarations

6.1. *Acknowledgments*

The authors would like to thank the patients and clinical staff involved in this study.

6.2. *Conflict of interests*

The authors declare no conflict of interest.

6.3. *Funding and supports*

Not applicable.

6.4. *Authors' contributions*

Farangis Sadeghi: Conceptualization, Methodology, Resources. Seyede Maryam Naghibi: Conceptualization, Validation, Formal Analysis, Writing – Original Draft, Writing – Review & Editing. Nazanin Bashiri: Conceptualization, Visualization, Project Administration. Ali Taherinia: Software, Data Curation, Investigation, Visualization. Lida Shojaei: Supervision, Project Administration, Writing – Original Draft, Writing – Review & Editing. Funding acquisition: None. All authors read and approved the final version of manuscript.

6.5. *Ethical statement*

This study was approved by the Research Ethics Committee of Alborz University of Medical Sciences (Approval Code: IR.ABZUMS.REC.1404.126). All procedures were performed in accordance with the Declaration of Helsinki.

6.6. *Informed consent*

Given the retrospective design of the study, data were collected from previously documented medical records. Patient information was handled confidentially and used solely for research purposes.

6.7. *Availability of supporting data*

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request and with the required approvals.

6.8. *Using artificial intelligence chatbots statement*

The authors used Grammarly (Grammarly, Inc.) solely for English language editing, including grammar, spelling, punctuation, and improvements in clarity and readability. The tool was not used for scientific content generation, data analysis, or interpretation of results. All suggested revisions were reviewed and approved by the authors, who take full responsibility for the final content of the manuscript.

6.9. *Other declarations*

Transparency Statement "The lead/corresponding author, Dr. Lida Shojaei, affirms that this manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained."

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Table 1: Baseline characteristics of studied cases (n=68)

Characteristic	Value
Age (years)	
Mean ± SD	32.79 ± 13.46
Sex	
Male	42 (61.8)
Female	26 (38.2)
Marital status	
Single	44 (64.7)
Married	24 (35.3)
Educational level	
Below university degree	59 (86.8)
University degree	9 (13.2)
Residence	
Urban	65 (95.6)
Rural	3 (4.4)
Time to hospital	
≤ 3 h	27 (39.7)
> 3 h	41 (60.3)
Reported ingested amount	
<10 g	55 (80.9)
≥10 g	13 (19.1)
Type of poisoning	
Intentional	59 (86.8)
Accidental	9 (13.2)

Values are mean ± standard deviation (SD) or n (%).

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Table 2. Clinical, laboratory, radiologic, and electrocardiographic findings at admission (n=68)

Variable	Value
Clinical symptoms and signs	
Nausea	42 (61.8)
Vomiting	32 (47.1)
Generalized weakness	26 (38.2)
Abdominal pain	23 (33.8)
Dizziness/vertigo	19 (27.9)
Drowsiness	14 (20.6)
Respiratory symptoms	13 (19.1)
Headache	9 (13.2)
Asymptomatic at referral	7 (10.3)
Palpitations	2 (2.9)
Sweating	2 (2.9)
Diarrhea	2 (2.9)
Cardiopulmonary arrest	1 (1.5)
Admission vital signs	
Glasgow Coma Scale score	14.79 ± 0.61 (11.00-15.00)
Temperature, °C	36.59 ± 0.47 (35.00-37.50)
Systolic blood pressure, mmHg	116.96 ± 19.58 (55.00-157.00)
Diastolic blood pressure, mmHg	72.37 ± 13.81 (35.00-95.00)
Pulse rate, beats/min	88.34 ± 17.75 (54.00-162.00)
Respiratory rate, breaths/min	17.84 ± 1.32 (14.00-21.00)
Oxygen saturation, %	96.13 ± 2.53 (83.00-99.00)
Laboratory findings	
pH	7.36 ± 0.13 (6.58-7.51)
PCO2, mmHg	41.75 ± 9.42 (18.00-73.30)
HCO3, mEq/L	23.58 ± 5.22 (6.70-32.90)
Base excess	-1.91 ± 6.22 (-33.00-5.60)
Sodium, mEq/L	140.66 ± 3.14 (134.00-155.00)
Potassium, mEq/L	3.95 ± 0.36 (3.00-5.20)
Blood glucose, mg/dL	98.25 ± 29.79 (35.00-242.00)
Prothrombin time, s	14.14 ± 2.62 (12.00-30.00)
Partial thromboplastin time, s	34.68 ± 4.89 (26.00-48.00)
International normalized ratio	1.11 ± 0.29 (1.00-2.60)
Aspartate aminotransferase, U/L	61.43 ± 215.14 (13.00-1748.00)
Alanine aminotransferase, U/L	45.44 ± 121.30 (11.00-893.00)
Alkaline phosphatase, U/L	190.01 ± 69.67 (26.00-416.00)
Blood urea nitrogen, mg/dL	28.47 ± 10.90 (9.26-65.00)
Creatinine, mg/dL	1.14 ± 0.56 (0.71-4.50)
Creatine phosphokinase, U/L	332.90 ± 1022.48 (47.00-8385.00)
Radiologic findings	
Normal chest radiography	66 (97.1)
Bilateral infiltration in chest radiography	2 (2.9)
Normal abdominopelvic X-ray	57 (83.8)
Radiopaque/opacity in abdominopelvic X-ray:	11 (16.2)
Electrocardiography findings	
Normal	53 (77.9)
Abnormal	15 (22.1)

Values are n (%) or mean ± SD (range). SpO2 is presented as percentage. PCO2, pressure of carbon dioxide; HCO3, bicarbonate.

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Table 3: Therapeutic management, complications, and clinical outcomes (n=68)

Characteristic	Value
Therapeutic management	
Polyethylene glycol	41 (60.3)
Gastric lavage	11 (16.2)
Activated charcoal	9 (13.2)
Bicarbonate therapy	8 (11.8)
Antioxidant therapy	7 (10.3)
Vasopressor support	5 (7.4)
Endotracheal intubation	5 (7.4)
Insulin-glucose therapy	2 (2.9)
Hemodialysis	1 (1.5)
Complications and outcomes	
Acute kidney injury	1 (1.5)
Bicytopenia/pancytopenia	3 (4.4)
Length of stay <24 h	31 (45.6)
Length of stay 24-48 h	22 (32.4)
Length of stay 48-72 h	10 (14.7)
Length of stay >72 h	5 (7.4)
ICU admission	12 (17.6)
Routine discharge	56 (82.4)
Discharge against medical advice	10 (14.7)
In-hospital mortality	2 (2.9); 95% CI, 0.8%-10.1%
Poor clinical outcome (composite)	12 (17.6); 95% CI, 10.4%-28.4%

Values are n (%). Poor clinical outcome was defined as ICU admission, in-hospital death, or hospitalization longer than 72 hours; components could overlap. The poor-outcome proportion was 17.6% (Wilson 95% CI, 10.4%-28.4%), and the in-hospital mortality proportion was 2.9% (Wilson 95% CI, 0.8%-10.1%). All 12 patients with poor clinical outcome were admitted to the ICU; five had hospital stays longer than 72 hours and two died in hospital. Discharge status was reported separately from the composite severity outcome. Of the 10 patients discharged against medical advice, 3 had already met the composite endpoint before leaving and 7 had no observed composite event during the index hospitalization; post-discharge status was unavailable.

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Table 4: Factors associated with poor clinical outcome based on univariate analysis

Variable	Observed poor outcome		P	OR (95% CI)
	No (n=56)	Yes (n=12)		
Reported ingested amount category ≥10 g	3 (5.4)	10 (83.3)	<0.001	88.33 (13.05-598.07)
Abnormal ECG	5 (8.9)	10 (83.3)	<0.001	51.00 (8.65-300.74)
Abnormal abdominopelvic X-ray	1 (1.8)	10 (83.3)	<0.001	275.00 (22.73-3327.72)
Abnormal chest radiography	0 (0.0)	2 (16.7)	0.029	26.90 (1.20-601.45)
Bicytopenia/pancytopenia	0 (0.0)	3 (25.0)	0.004	41.63 (1.99-871.98)
Systolic blood pressure, mmHg (per 10-mmHg decrease)	118.50 (110.00-135.00)	106.00 (84.25-112.00)	0.006	1.88 (1.22-2.89)
Diastolic blood pressure, mmHg (per 10-mmHg decrease)	78.00 (70.00-85.00)	60.50 (45.00-70.00)	<0.001	2.53 (1.47-4.37)
HCO3, mEq/L (per 5-mEq/L decrease)	24.75 (22.08-27.62)	16.70 (13.88-22.52)	0.002	4.10 (1.75-9.60)
Base excess (per 5-unit decrease)	-0.70 (-3.35-2.00)	-6.30 (-13.30--0.97)	0.005	3.69 (1.49-9.18)
BUN, mg/dL (per 10-mg/dL increase)	26.00 (22.00-29.00)	37.00 (27.50-54.25)	0.007	2.77 (1.47-5.22)
Creatinine, mg/dL (per 0.5-mg/dL increase)	0.99 (0.90-1.10)	1.15 (1.00-1.85)	0.008	4.49 (1.07-18.84)
PT, s (per 1-s increase)	13.50 (13.00-14.00)	14.10 (13.38-17.45)	0.032	1.44 (1.05-1.98)
INR (per 0.1-unit increase)	1.00 (1.00-1.06)	1.05 (1.00-1.52)	0.034	1.30 (1.04-1.62)
AST, U/L (per doubling)	25.00 (19.75-29.25)	30.50 (22.75-121.50)	0.027	3.95 (1.21-12.92)
ALT, U/L (per doubling)	20.00 (17.75-23.25)	43.00 (23.25-110.50)	0.001	8.67 (1.91-39.26)
CPK, U/L (per doubling)	165.00 (112.00-221.75)	289.00 (213.25-359.75)	0.001	4.41 (1.27-15.32)

Continuous variables are median (IQR); categorical variables are n (%). Poor clinical outcome was defined as ICU admission, in-hospital death, or hospitalization longer than 72 hours; components could overlap. The comparison group comprised patients in whom no composite poor-outcome event was observed during the index hospitalization and should not be interpreted as a confirmed favorable-outcome group. P values are from Mann-Whitney U tests for continuous variables and Fisher's exact tests for categorical variables. Crude ORs and 95% CIs are from exploratory univariable logistic regression; for binary variables with zero cells, the Haldane-Anscombe continuity correction was applied to the corresponding 2 × 2 table to obtain a finite estimate. ORs are not mutually adjusted, no multiplicity correction was applied, and all estimates are exploratory and should not be interpreted as independent predictors.

Table 5: Sensitivity analysis of factors associated with an alternative severe outcome definition

Variable	Severe outcome (n=5)	No severe outcome (n=63)	P value
Reported ingested amount category ≥10 g	5 (100.0)	8 (12.7)	<0.001
Abnormal ECG	5 (100.0)	10 (15.9)	<0.001
Abnormal abdominopelvic X-ray	5 (100.0)	6 (9.5)	<0.001
Abnormal chest radiography	2 (40.0)	0 (0.0)	0.004
Bicytopenia/pancytopenia	2 (40.0)	1 (1.6)	0.013
Systolic blood pressure, mmHg	85.00 (79.00-102.00)	116.00 (110.00-134.50)	0.002
Diastolic blood pressure, mmHg	56.00 (52.00-70.00)	75.00 (67.50-84.50)	0.016
HCO3, mEq/L	16.40 (7.70-17.00)	24.40 (21.90-27.40)	0.002
Base excess	-12.60 (-23.30 to -11.10)	-0.80 (-3.50-2.10)	<0.001
BUN, mg/dL	49.00 (38.00-55.00)	26.00 (22.00-29.00)	0.007
Creatinine, mg/dL	1.60 (1.31-3.20)	1.00 (0.90-1.10)	<0.001
PT, s	16.60 (16.00-20.00)	13.50 (13.00-14.00)	0.013
INR	1.40 (1.30-1.90)	1.00 (1.00-1.08)	0.001
AST, U/L	300.00 (62.00-400.00)	25.00 (20.00-29.00)	<0.001
ALT, U/L	271.00 (57.00-466.00)	20.00 (18.00-24.50)	0.002
CPK, U/L	365.00 (358.00-2156.00)	169.00 (115.00-230.00)	0.002

The alternative severe outcome was defined as in-hospital death, vasopressor requirement, or endotracheal intubation. Five patients (7.4%) met this definition. Continuous variables are median (IQR) and categorical variables are n (%). P values are from Fisher's exact tests for categorical variables and Mann-Whitney U tests for continuous variables. Because of the small number of severe events, this sensitivity analysis was exploratory; no multivariable model was fitted, and the results should not be interpreted as an independent prediction model.

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Table 6: Sensitivity analysis excluding patients discharged against medical advice

Variable	Observed poor outcome		P value
	Yes (n=9)	No (n=49)	
Reported ingested amount category ≥10 g	7 (77.8)	2 (4.1)	<0.001
Abnormal ECG	7 (77.8)	3 (6.1)	<0.001
Abnormal abdominopelvic X-ray	7 (77.8)	0 (0.0)	<0.001
Abnormal chest radiography	2 (22.2)	0 (0.0)	0.022
Bicytopenia/pancytopenia	2 (22.2)	0 (0.0)	0.022
Systolic blood pressure, mmHg	102.00 (85.00-115.00)	116.00 (110.00-131.00)	0.024
Diastolic blood pressure, mmHg	68.00 (52.00-70.00)	75.00 (70.00-84.00)	0.010
HCO3, mEq/L	16.10 (9.60-22.20)	24.40 (21.80-27.10)	0.002
Base excess	-6.90 (-15.40 to -4.80)	-0.80 (-3.50-1.70)	0.002
BUN, mg/dL	38.00 (31.00-54.00)	25.00 (22.00-28.00)	0.001
Creatinine, mg/dL	1.10 (1.00-1.60)	0.96 (0.90-1.10)	0.021
PT, s	14.70 (13.50-20.00)	13.50 (13.00-14.00)	0.026
INR	1.10 (1.00-1.90)	1.00 (1.00-1.10)	0.034
AST, U/L	32.00 (22.00-300.00)	25.00 (20.00-31.00)	0.046
ALT, U/L	53.00 (21.00-271.00)	20.00 (18.00-24.00)	0.014
CPK, U/L	282.00 (215.00-365.00)	167.00 (113.00-224.00)	0.001

This complete-case sensitivity analysis excluded all 10 patients discharged against medical advice, leaving 58 patients: 9 with the primary composite poor clinical outcome and 49 with no observed composite event. Continuous variables are median (IQR) and categorical variables are n (%). P values are from Fisher's exact tests for categorical variables and Mann-Whitney U tests for continuous variables. Because only nine poor-outcome events remained, the analysis was exploratory and no additional regression model was fitted.

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