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IN-HOSPITAL SURVIVAL OUTCOME AMONG PATIENTS WITH ACUTE ALUMINIUM PHOSPHIDE (WHEAT-PILL) POISONING RECEIVING INTRAVENOUS DEXTROSE-CONTAINING SUPPORTIVE THERAPY: A PROSPECTIVE CASE SERIES.

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Abstract

Objective: *To determine the frequency of in-hospital survival among patients with acute aluminium phosphide (wheat-pill) poisoning receiving intravenous dextrose-containing supportive therapy in the Accident and Emergency Department of a tertiary care hospital.*

Study Design: *Prospective, observational case series.*

Setting: *Accident and Emergency Department, Jinnah Postgraduate Medical Centre (JPMC), Karachi.*

Period: *January to April 2025.*

Material & Methods: *Ninety-five adult patients (≥18 years) with a compatible history and presentation of acute aluminium phosphide wheat-pill ingestion, who received physician-directed intravenous dextrose-containing supportive therapy, were enrolled by non-probability purposive sampling. Demographic, clinical, biochemical and treatment variables were recorded on a structured proforma. Data were coded and analysed in IBM SPSS Statistics v26. Continuous variables were tested for normality (Shapiro-Wilk) and reported as mean±SD or median (IQR); categorical variables as frequency/percentage. Survivors and non-survivors were compared using the independent-samples t-test/Mann-Whitney U test and chi-square/Fisher's exact test, and a parsimonious multivariable binary logistic regression was fitted for in-hospital survival. $p \leq 0.05$ was considered significant.*

Results: *In-hospital survival occurred in 42/95 patients (44.2%; 95% CI 34.6%-54.2%). Non-survivors presented with significantly higher pulse, respiratory rate, QTc, lactate and baseline glucose, and lower systolic/diastolic blood pressure, SpO2, GCS, pH and bicarbonate than survivors (all $p<0.05$), and had higher rates of shock, vasopressor requirement, mechanical ventilation, dysrhythmia and other major complications (all $p<0.001$). On multivariable logistic regression, systolic blood pressure (adjusted OR 1.11, 95% CI 1.05-1.17, $p<0.001$) and lactate (adjusted OR 0.57, 95% CI 0.34-0.96, $p=0.033$) were independent predictors of survival (Nagelkerke $R^2=0.70$; model correctly classified 84.2% of cases).*

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Conclusion: *Just under half of patients with acute aluminium phosphide poisoning receiving dextrose-containing supportive therapy survived to discharge, with presenting haemodynamic and metabolic severity not dextrose exposure itself driving in-hospital outcome. Early recognition of shock, acidosis and hyperlactataemia should guide triage and resource allocation.*

INTRODUCTION

Aluminium Phosphide (AlP), known as the 'wheat' or 'rice' pill in South Asia, is a pesticide that, once ingested, releases phosphine gas. Symptoms of AlP poisoning include cardiac shock, acidosis, and rapid failure of multiple organs. Symptoms of AlP poisoning typically appear within 12 hours.[1] There is currently no known antidote for AlP poisoning. Because no specific treatment exists, the best approach is to manage symptoms and provide supportive care.[2] The outcomes for patients who experience AlP poisoning depend on how rapidly symptoms are identified, how quickly supportive care is initiated, and the overall availability of that care. AlP poisoning is one of the leading causes of suicide in South Asia. A series of 110 patients at a Pakistani hospital reported that 85.5% of the patients were between the ages of 12 and 30, and 56.4% of all patients died.[3] Outcomes for AlP poisoning patients also vary by location. A series of patients in Northwest Ethiopia reported a mortality rate of 31.2%,[4] and another more recent series in Iran reported 39.2%[5] mortality.

Disturbed glucose homeostasis is now recognised as an important aspect of phosphine toxicosis. Elevated blood glucose at presentation is associated with higher case fatality.[6] Proposed simple bedside methods for assessing severity in aluminium phosphide poisoning are based on admission glucose and potassium levels.[7] In light of this, standard practice has shifted from administering intravenous glucose alone to a combination of insulin and potassium. A recent pilot study reduced case fatality from 73.3% to 46.7% with additional use of a glucose-insulin-potassium solution.[8] A randomised controlled study showed lower case fatality (39.2% vs 96.3%) with insulin to control blood glucose compared with standard care.[9] A 2024 systematic review noted that small, unrelated studies have evaluated the use of insulin to control blood glucose levels, but their outcomes have not been reported. Lastly, several recent research studies have shown that it is the existing metabolic and haemodynamic derangement at the time of presentation that determines the prognosis of patients with aluminium phosphide poisoning. [10-13]

In-hospital survival data for patients in Pakistan administered IV dextrose-containing supportive therapy is unavailable. This study will determine the in-hospital survival rate of patients who presented with acute aluminum phosphide (wheat-pill) poisoning to the Accident and Emergency Department (A&E) of a tertiary care hospital and who were given IV dextrose-containing supportive therapy. This study will also determine the clinical and biochemical variables that are associated with in-hospital survival.

MATERIAL & METHODS

Study Design: Prospective, observational case series.

Settings: Accident and Emergency Department, Jinnah Postgraduate Medical Centre (JPMC), Karachi.

Duration: January to April 2025, a four-month enrolment period following approval from the Institutional Review Board, JPMC.

Sample Size: Using the WHO formula for estimating sample size for a single population proportion,[14] with an expected population proportion of survival of 43.64%, based on a tertiary care series from Pakistan,[3] and taking a 95% confidence interval (Z=1.96) and a relative precision of 0.10, the required minimum sample size was found to be 94.5. Hence, the sample size is taken as 95.

Sampling Technique: Non-probability, purposive sampling.

Inclusion Criteria: Adult (18 years and above) patients presenting to the emergency department

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of Jinnah Postgraduate Medical Centre within 24 hours of ingesting aluminium phosphide wheat pills. An emergency physician documented and evaluated each case. The emergency physician then provided the patient with an IV infusion of dextrose and normal saline solution. A consultant physician (of the speciality to which the patient belongs) confirmed the action taken.

Exclusion Criteria: The patient was excluded from the study in case of reported co-ingestion of any other pesticide/psychotropic drug/substance, exposure to zinc phosphide and not ingesting the same, presence of any of the following conditions: insulin-dependent diabetes mellitus (with or without ketoacidosis and ketosis) and hyperosmolar hyperglycemic state, end-stage renal disease and chronic liver disease and malignant (terminal) cancer, and/or cardiopulmonary arrest.

Data Collection Procedure: Once the JPMC IRB approved the study, potentially wheat-pill poisoned patients were identified and recruited to the study. Recruitment did not delay the patient's resuscitation. The study team had no authority to direct or change the treating physician's management of the patient; therefore, the study was not intended to modify, alter, or affect the treatment provided to the patient as part of routine clinical practice[15]. At the time, recommendations for managing this poisoning were to provide supportive care. At baseline and at 1, 3, 6, 12 and 24 hours, capillary blood glucose levels were measured. This study also recorded pertinent clinical and electrocardiogram (ECG) data, as well as all laboratory and other medical procedures. We recorded any of the following as study endpoints: patient discharge, patient transfer, or in-hospital patient death.

Data / Statistical Analysis: This study coded and analysed the data using IBM SPSS Statistics version 26. We analysed continuous variables using normality tests. We reported continuous variables as mean ± standard deviation or median and interquartile range, depending on normality. Categorical variables were described using frequencies and percentages. The primary outcome is reported as a proportion with a confidence interval. Continuous variables, if applicable, were compared across survivor and non-survivor groups using independent sample tests (i.e. independent sample t-test or Mann-Whitney U test), and $\chi 2$ test or Fisher's exact test for categorical and continuous variables, respectively. For multivariable analysis, this study used logistic regression to analyse in-hospital mortality. To assess collinearity, we used Spearman's rank correlation among variables. This kept lactate along with other factors, including age, systolic blood pressure, and time from exposure to arrival, to improve the events-to-variables ratio for the smaller outcome group (i.e., the in-hospital mortality group). This study evaluated model fit using the Hosmer-Lemeshow test and Nagelkerke R2. This study considered p-values < 0.05 statistically significant.

Ethical Approval: Jinnah Postgraduate Medical Centre's Institutional Review Board authorised this research study. After authorisation, the researchers obtained informed consent from participating patients. In cases where patients were unable to give consent, their legally authorised representatives provided informed consent.

RESULTS

A total of 95 patients were assessed for the study. The mean age of the patients was 29.9 years (range 18-47). 52 patients (54.7%) were males. The tablet container was fresh or recently opened (62.1%). Thirty-nine (41.1%) patients were in shock. The median Glasgow Coma Scale score was 14, and 48.4% of patients had metabolic acidosis. Table I presents the other clinical and demographic findings. According to the protocol, all 95 patients received an IV solution containing dextrose. Of these, 46.3% received 5% dextrose, while 33.7% received 10% dextrose. The remaining patients received other dextrose combinations (Figure 1).

Table-I: Baseline demographic and clinical characteristics of patients (n=95)

Variable	Category	Value (n=95)
Age (years), mean ± SD		29.9 ± 7.2

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Variable	Category	Value (n=95)
Gender	Male, n(%)	52 (54.7%)
Intent	Self-harm, n(%)	84 (88.4%)
Time ingestion-to-arrival (hours)	Median (IQR)	3.1 (2.5–4.5)
Tablet container	Fresh/opened, n(%)	59 (62.1%)
Estimated tablets ingested	Median (IQR)	1.0 (0.5–1.0)
Shock at presentation	n(%)	66 (69.5%)
Glasgow Coma Scale (/15)	Median (IQR)	14 (13–15)
Systolic BP (mmHg), mean ± SD		99.0 ± 22.5
Baseline glucose (mg/dL), mean ± SD		139.4 ± 42.8
pH	Median (IQR)	7.32 (7.24–7.40)
Lactate (mmol/L)	Median (IQR)	3.9 (2.4–5.7)
Dextrose concentration	5% / 10% / mixed or rescue, n(%)	44 (46.3%) / 32 (33.7%) / 19 (20.0%)
Concomitant insulin	n(%)	21 (22.1%)
Concomitant potassium	n(%)	32 (33.7%)
Vasopressor required	n(%)	65 (68.4%)
Mechanical ventilation	n(%)	41 (43.2%)
Final outcome	Alive discharge / transfer / death, n(%)	38 (40.0%) / 4 (4.2%) / 53 (55.8%)

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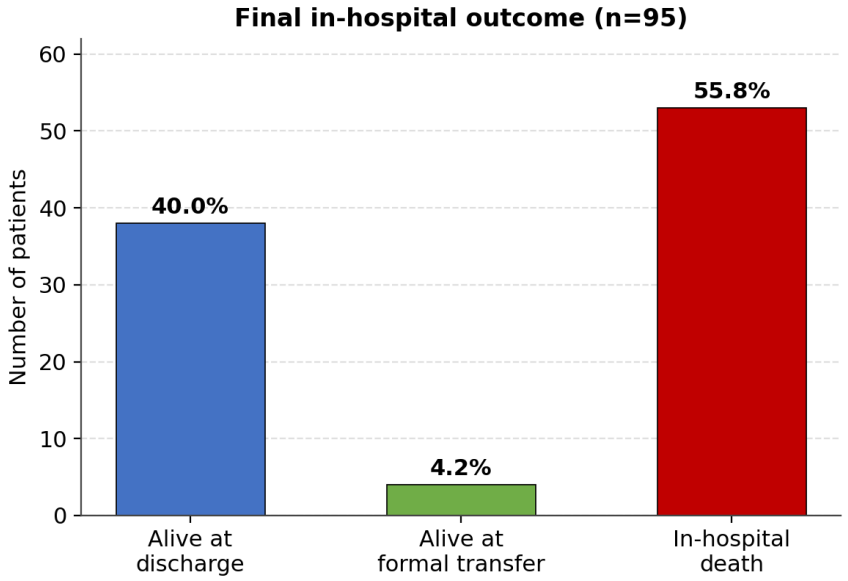


Figure-1: Final in-hospital outcome distribution (n=95).

This portion of the analysis explores meaningful differences among patient attributes (refer to Table II). With regard to vital signs, survivors had a slower pulse of 88.7±14.9 versus 109.8±14.7, a respi-ratory rate of 18.4±3.9 versus 24.3±4.6, and a systolic blood pressure of 116.8±15.0 versus 84.9±16.8 (mmHg) (p<0.001 for all). The survivors also had a longer QTc (442.3±33.8 versus 461.1±30.9 ms) (p=0.006). The survivors also had a higher level of consciousness as indicated by a higher Glasgow Coma Scale score of 15 vs 13 (p<0.001). The survivors also had a lower baseline blood glucose level of 128.5±36.6 versus 148.0±45.6 mg/dL (p=0.023).

Table-II: Comparison of continuous clinical and biochemical variables between survivors and non-survivors

Variable	Survivors (n=42)	Non-survivors (n=53)	Test	p-value
Age (years)	30.5 (27.0–34.0)	31.0 (23.0–36.0)	MW-U	0.610
Time to arrival (h)	3.2 (2.6–4.2)	3.0 (2.4–4.8)	MW-U	0.922
Estimated tablets	1.0 (0.5–1.0)	1.0 (1.0–2.0)	MW-U	0.017*
Pulse (/min)	88.7 ± 14.9	109.8 ± 14.7	t-test	<0.001*
Respiratory rate (/min)	18.4 ± 3.9	24.3 ± 4.6	t-test	<0.001*
Systolic BP (mmHg)	116.8 ± 15.0	84.9 ± 16.8	t-test	<0.001*
Diastolic BP (mmHg)	71.6 ± 10.3	55.5 ± 10.1	t-test	<0.001*
SpO2 (%)	96.5 (95.0–98.0)	94.0 (92.0–95.0)	MW-U	<0.001*
Temperature (°C)	36.8 ± 0.4	36.8 ± 0.4	t-test	0.677
GCS (/15)	15.0 (14.0–15.0)	13.0 (12.0–14.0)	MW-U	<0.001*

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Variable	Survivors (n=42)	Non-survivors (n=53)	Test	p-value
QTc (ms)	442.3 ± 33.8	461.1 ± 30.9	t-test	0.006*
Baseline glucose (mg/dL)	128.5 ± 36.6	148.0 ± 45.6	t-test	0.023*
pH	7.40 ± 0.06	7.26 ± 0.10	t-test	<0.001*
Bicarbonate (mmol/L)	23.3 ± 2.6	17.0 ± 4.4	t-test	<0.001*
Lactate (mmol/L)	2.6 ± 1.3	5.5 ± 2.1	t-test	<0.001*
Total dextrose, 24h (mL)	2036.9 ± 588.9	1480.2 ± 797.1	t-test	<0.001*
Length of stay (hours)	91.3 (71.4–103.1)	25.9 (11.1–51.3)	MW-U	<0.001*

MW-U = Mann-Whitney U test. *statistically significant at p<0.05.

As shown in Table III, all victims in the non-survivor group suffered from circulatory shock, while only 33.3% of survivors had shock. The non-survivor group was also more vasopressed on presentation (98.1% vs. 31.0% of survivors, $p < 0.001$). Additionally, non-survivors were more likely to require mechanical ventilation (62.3% vs. 19.0% of survivors, $p < 0.001$), and had a higher incidence of life-threatening dysrhythmias (56.6% vs. 19.0% of survivors). Non-survivors had more cardiac arrests, and the arrests occurred in the ED (62.3% vs. 4.8% of survivors). Metabolic acidosis was more prevalent in the non-survivors (81.1% vs 7.1%). Study drug container freshness differed, with 77.4% of non-survivors and 42.9% of survivors using a recently supplied study drug ($p < 0.001$). Regarding the other variables of the study, the type of insulin and potassium, baseline hyperglycemia, and ED patient and study personnel factors, did not impact survival ($p > 0.05$).

Table-III: Comparison of categorical variables between survivors and non-survivors

Variable	Survivors n(%)	Non-survivors n(%)	p-value
Male gender	20 (47.6%)	32 (60.4%)	0.301
Fresh/opened tablet container	18 (42.9%)	41 (77.4%)	<0.001*
Shock at presentation	14 (33.3%)	52 (98.1%)	<0.001*
Vasopressor required	13 (31.0%)	52 (98.1%)	<0.001*
Mechanical ventilation	8 (19.0%)	33 (62.3%)	<0.001*
Major dysrhythmia	8 (19.0%)	30 (56.6%)	<0.001*
Cardiac arrest after arrival	2 (4.8%)	33 (62.3%)	<0.001*
Any other major complication (AKI/ARDS/hepatic/myocardial)	10 (23.8%)	47 (88.7%)	<0.001*
Metabolic acidosis	3 (7.1%)	43 (81.1%)	<0.001*
Baseline hyperglycaemia (>180 mg/dL)	4 (9.5%)	13 (24.5%)	0.066
Concomitant insulin	5 (11.9%)	16 (30.2%)	0.060

*statistically significant at p<0.05 (chi-square or Fisher's exact test as appropriate).

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As shown in Table IV, the presence of hypotension and each millimole per liter of lactate, in a positive analysis, decreased the likelihood of survival (OR = 0.57 and 0.36 respectively, and $p < 0.05$ for both). Increasing the systolic blood pressure, in 1 mm of mercury, was positively associated with the survival of the patient (OR = 1.11 and $p < 0.001$).

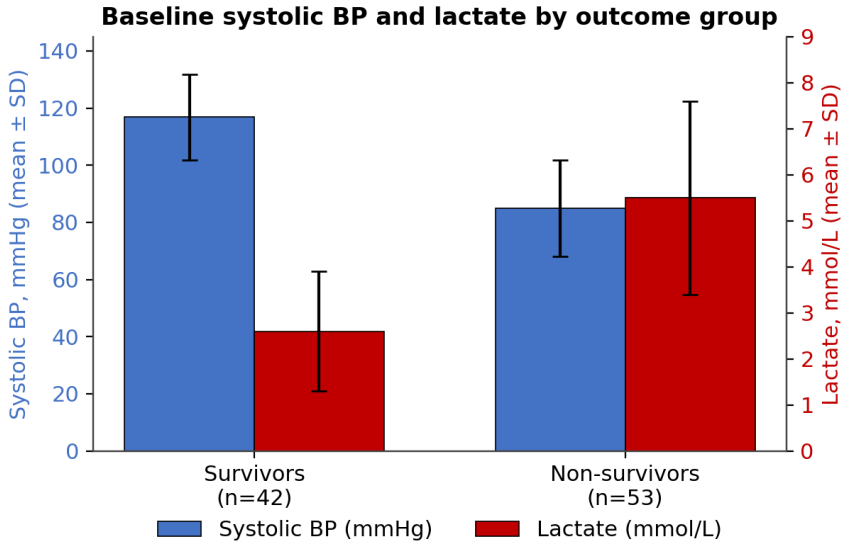


Figure-2: Baseline systolic blood pressure and lactate by outcome group (mean ± SD).

Table-IV: Multivariable binary logistic regression for in-hospital survival (dependent variable: 1=survivor, 0=non-survivor)

Predictor	β	Adjusted OR	95% CI for OR	p-value
Age (years)	0.044	1.045	0.954 – 1.144	0.344
Time to arrival (h)	0.023	1.023	0.737 – 1.420	0.891
Systolic BP (mmHg)	0.101	1.106	1.045 – 1.171	<0.001*
Lactate (mmol/L)	-0.560	0.571	0.341 – 0.956	0.033*
Constant	-9.858	0.000	–	0.005

*statistically significant at $p<0.05$. Model: $\chi^2=70.36$, df=4, $p<0.001$; Hosmer-Lemeshow p=0.158; Nagelkerke $R^2=0.70$; overall correct classification 84.2%.

DISCUSSION

The survival rate in our study is comparable to the rate reported in a previous study from Pakistan (43.64%)[3], and to the rates reported in other regional studies like 68.8% from Northwest Ethiopia[4] and 60.8% from central Iran[5]. The rates of mortality in Ethiopia and Iran were 31.2% and 39.2%, respectively. A more recent study from Pakistan's Khyber Pakhtunkhwa showed that mortality exceeding half of admitted cases,[16] the toxicity from aluminium phosphide led to death, demonstrating that across different geographical areas, including those in which the healthcare personnel use dextrose-containing fluids for supportive therapy, aluminium phosphide poisoning remains a public health burden.

The features that distinguished survivors from non-survivors among our cohort (including tachycardia, hypotension and tachypnea,

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hypoxemia, altered level of consciousness, prolonged QTc, metabolic acidosis) are similar to those described with phosphine poisoning.[17] Our non-survivors had more critical illness, shock on presentation, mechanical ventilation, vasopressor requirements, cardiac arrest and dysrhythmias, which is consistent with other case series describing similar phosphine poisoning incidents. As with these prior reports, early aggressive resuscitative efforts determined survival. A case series from India published in 2026 described phosphine poisoning and cardiogenic shock. [18] They showed that early bicarbonate and hemodialysis arrested progressive multi-organ failure and supported that survival was not determined by the quantity of phosphine to which patients were exposed.

This research identified another point of differentiation related to the management of hyperglycemia. As reported previously, hyperglycemia increases the risk of mortality in patients with aluminium phosphide poisoning.[6] This also supports the hypothesis that the glucose-to-potassium ratio can be used to estimate the severity of aluminium phosphide poisoning.[7] Survivors and non-survivors in our study did not receive an insulin infusion according to a standard protocol. Thus, we cannot attribute the outcome to insulin therapy. The lower total 24-hour dextrose infusion volume in non-survivors may have been due to aggressive therapy to lower their blood glucose level. This supports concerns about the potential harm of the open-label study and a randomised controlled trial, in which glucose-insulin-potassium infusion therapy was associated with reduced mortality.[9] The 2024 Systematic Review also concluded that evidence for using insulin to treat aluminium phosphide toxicity is inadequate and limited.[10] In the setting of hyperglycemia due to various causes in other forms of shock, a stress response may explain the hyperglycemia observed in patients with aluminium phosphide toxicity.[19] Thus, in aluminium phosphide toxicity, hyperglycemia may reflect an overall increase in the body's stress response.

In multivariable analysis, both systolic blood pressure and lactate remained independent predictors of survival, and the model was overall accurate in 84% of cases. The discriminative performance of the current model is comparable to recently developed prognostic models of aluminium phosphide poisoning that incorporate a combination of cortisol and/or arterial blood gas levels, as well as machine learning. The model is also comparable to prognostic models that utilise other advanced artificial intelligence and machine learning techniques, as well as prognostic models for aluminium phosphide poisoning that use the relevance-vector machine learning technique.[11,12,13,20] Based on the above models and the current model, it is appropriate to assess the vitals of patients who have been poisoned with aluminium phosphide and, based on the evidence, determine which patients need urgent medical intervention. [21]

This study has several limitations. It was a single-centre study. The sampling method increases the risk of bias and limits the generalizability of the results to other centres. Patients at the study site were offered dextrose-containing therapy, and the treating team also administered insulin and potassium. Because there was no comparison group, the study aimed to evaluate the relationship between different factors and survival; this study was not designed to evaluate the effectiveness of dextrose therapy or other glucose management strategies. One limitation was that the primary outcome had a 95% confidence interval of 34.6% to 54.2%. The model used to analyse factors associated with the primary outcome was also limiting because it could evaluate only four variables. The study was designed to evaluate the effect of a more rigorously controlled glucose management system in the critically ill.

CONCLUSION

Most patients surviving to discharge or transfer in this series of aluminum phosphide (wheat-pill) poisoning cases received intravenous dextrose-based therapy. We reported that in the presence of severe metabolic and haemodynamic derangement, as evidenced by elevated lactate levels and hypotension, prior exposure to intravenous dextrose solutions was associated with improved survival. The impact of dextrose

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exposure was independent of co-interventions, including dialysis. Dextrose-based solutions may facilitate early triage and resource allocation in under-resourced emergency departments.

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