ORIGINAL ARTICLE

A Study on Electrocardiography Echocardiography and Cardiac Biomarkers in Aluminium Phosphide Poisoning and Their Prognostic Correlation

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Abstract

Background: Aluminum phosphide (AlP) poisoning is a medical emergency with an alarmingly high mortality rate, primarily due to its rapid cardiotoxic effects.

Objective: To identify and evaluate key prognostic indicators—clinical, electrocardiographic, echocardiographic, and biochemical—in patients with AlP poisoning.

Materials and methods: A cross-sectional observational study was conducted on 100 patients with confirmed AlP ingestion. ECG changes, cardiac biomarkers (troponin-I, CPK-MB, LDH, and CPK-NAC), 2D echocardiography findings, and acid-base disturbances were analyzed in relation to survival outcomes.

Results: ECG abnormalities and decreased ejection fraction were significantly associated with mortality. Elevated cardiac biomarkers and profound acidosis were strong independent predictors of poor prognosis.

Conclusion: AlP poisoning causes critical cardiovascular compromise. Early identification of high-risk patients may guide aggressive intervention and resource allocation in intensive care settings.

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Introduction

Aluminum phosphide (AlP) poisoning represents one of the most fatal toxicological emergencies encountered in clinical practice, particularly in countries such as India, where the compound is widely used and poorly regulated.[1] Once ingested, AlP reacts rapidly with water and gastric acid to release phosphine (PH_3) gas, a potent mitochondrial poison.[2] Phosphine inhibits cytochrome c oxidase, disrupts the electron transport chain, reduces ATP production, and triggers a cascade of oxidative stress and cellular injury.[3] These biochemical disruptions predominantly affect vital organs with high metabolic demand, such as the heart, brain, and liver.[4]

Among the complications, cardiotoxicity remains the most prominent and prognostically significant. The majority of AlP poisoning-related deaths are attributed to refractory hypotension, cardiac arrhythmias, and severe myocardial depression.[5] Electrocardiographic changes such as ST-T wave abnormalities, conduction defects, tachyarrhythmias, and QT prolongation are frequently reported.[6] Additionally, echocardiographic findings such as global hypokinesia and reduced ejection fraction provide structural evidence of myocardial compromise.[7] Elevated cardiac biomarkers—troponin-I, CPK-MB, CPK-NAC, and LDH—further confirm the extent of myocardial damage.[8]

Despite these well-documented effects, timely identification of patients at greatest risk of death remains a clinical challenge. Early and accurate risk stratification can significantly influence management decisions, resource allocation, and potentially patient outcomes.[9] This study aims to evaluate the prognostic significance of these clinical and biochemical markers in patients with AlP poisoning. By doing so, the study seeks to contribute to both improved clinical outcomes and rational use of intensive care resources in this high-fatality poisoning scenario.[11]

Materials and Methods

This cross-sectional observational study was carried out in the Department of Medicine, Government Medical College, Kota, and affiliated hospitals over a 12-month period. Ethical clearance was obtained, and written informed consent was secured from all participants or their legally authorized representatives.

The study enrolled 100 patients above 18 years of age who presented with a clear history of AlP ingestion. Patients with preexisting structural or ischemic heart disease, congenital heart disease, or mixed poisoning were excluded. The objective was to assess parameters predictive of mortality and develop a practical nomogram model.

Each patient underwent detailed clinical evaluation on admission, including assessment of vitals, level of consciousness, oxygen saturation, and systemic examination. ECG was performed on admission and repeated every 24 hours or earlier if deterioration occurred. Electrocardiographic findings such as QT interval, ST-T changes, arrhythmias, or conduction defects were documented.

All patients underwent 2D echocardiography (using Wipro GE Vivid T8, 3S probe) within the first 24 hours of admission. Key echocardiographic parameters assessed included left ventricular ejection fraction (LVEF), global hypokinesia, regional wall motion abnormalities, and septal motion. Cardiac biomarkers—troponin-I, CPK-MB, CPK-NAC, and LDH—were analyzed at baseline and repeated every 24–48 hours depending on clinical status.

Laboratory tests included arterial blood gas (ABG), serum electrolytes, renal and liver function tests, and complete blood count. Patients were closely monitored for survival outcomes and duration of hospitalization.

Statistical analysis included multivariate logistic regression to identify independent predictors of mortality. This study emphasized the prognostic utility of early cardiac assessment and metabolic profiling in predicting clinical outcomes in AlP poisoning cases.

Results

This study analyzed 100 patients to assess prognostic indicators in AlP poisoning, with special emphasis on cardiac involvement. Correlation between specific ECG abnormalities and mortality in patients

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with AlP poisoning showed that, in those with atrial fibrillation and heart block, the mortality rate was 100%, indicating a strong association between these arrhythmias and fatal outcomes ($p = 0.008$). ST-T changes were the most prevalent abnormality, observed in 36 patients, with a high mortality rate of 72.2% ($p = 0.001$). Both ST elevation and ST depression were associated with significantly increased mortality ($p = 0.001$). These results emphasize that certain ECG abnormalities, especially atrial fibrillation, heart block, and ST-T changes, serve as strong prognostic indicators in AlP poisoning. The findings highlight the utility of ECG as a noninvasive, rapid bedside tool for early prognostication in AlP poisoning. The presence of malignant arrhythmias such as atrial fibrillation and heart block should immediately raise concern for poor prognosis and necessitate intensive monitoring. The statistically significant *p*-values reinforce the clinical relevance of ECG patterns in predicting patient outcomes and guiding the level of care required (Table 1).

A significant correlation between echocardiographic findings and patient outcomes in AlP poisoning was noted. Of the 60 patients evaluated, all 12 individuals with normal echocardiography findings survived (100%), strongly suggesting that normal cardiac function is a reliable predictor of survival. In contrast, global hypokinesia was present in 40 patients, with a mortality rate of 50%, indicating moderate prognostic risk. Most notably, decreased ejection fraction (EF) was observed in 45 patients, among whom 66.6% succumbed to the poisoning, establishing it as a powerful predictor of poor prognosis. The statistical significance of these findings ($p < 0.001$) confirms that echocardiographic abnormalities, particularly reduced EF, are closely linked to mortality. These results affirm the role of echocardiography as a noninvasive, rapid, and critical bedside tool for early prognostication in AlP poisoning. Identifying global hypokinesia and impaired EF early can aid clinicians in initiating aggressive supportive measures. This strengthens the case for routine echocardiographic evaluation in all moderate to severe cases of AlP poisoning. Integrating echo parameters into triage protocols may ultimately help reduce preventable deaths in resource-limited settings (Table 2).

A strong association was noted between elevated cardiac biomarkers and poor outcomes in patients with AlP poisoning. Among the 26 patients with positive troponin-I levels, 84.6% died, establishing troponin-I as a critical prognostic indicator ($p < 0.001$). Similarly, elevated CPK-MB and CPK-NAC levels were associated with 87.5 and 89.3% mortality rates, respectively, reinforcing their role in predicting myocardial injury severity. LDH, though a nonspecific marker, also showed a 90% mortality rate in positive cases, further confirming widespread cellular damage. The *p*-values for all markers were statistically significant (<0.001), underscoring their prognostic reliability. This reinforces the concept that myocardial damage, as evidenced by biochemical markers, plays a central role in AlP-related mortality (Table 3).

Multivariate analysis identified several independent predictors of mortality in AlP poisoning, emphasizing the critical role of both cardiac and systemic parameters. Cardiac biomarkers demonstrated the highest odds of predicting death, with positive CPK-NAC (OR 4.34), CPK-MB (OR 2.11), and troponin-I (OR 0.31) showing strong associations with fatal outcomes

Table 1: Correlation between specific ECG abnormalities and mortality ($n = 60$)

ECG abnormality	*Survived*		*Died*		*p-value*
	No.	*%*	*No.*	*%*	
Sinus tachycardia ($n = 24$)	16	66.7%	8	33.3%	0.045*
Sinus bradycardia ($n = 12$)	6	50%	6	50%	0.834
Atrial fibrillation ($n = 4$)	0	0%	4	100%	0.008*
VPCs ($n = 10$)	4	40%	6	60%	0.122
Heart block ($n = 4$)	0	0%	4	100%	0.008*
ST-T changes ($n = 36$)	10	27.8%	26	72.2%	0.001*
ST elevation ($n = 22$)	6	27.3%	16	72.7%	0.001
ST depression ($n = 14$)	4	28.6%	10	71.4%	0.001
BBB ($n = 8$)	2	25.0%	6	75.0%	0.038
QT prolongation ($n = 15$) (QTc)	12	80%	3	20%	0.001

*Indicates statistical significance $p < 0.05$

Table 2: Correlation between echocardiographic findings and outcome ($n = 60$)

Echo findings	*Survived*		*Died*		*p-value*
	No.	*%*	*No.*	*%*	
Normal ($n = 12$)	12	100%	0	0%	<0.001*
Global hypokinesia ($n = 40$)	20	50%	20	50%	
Decreased EF ($n = 45$)	15	33.3%	30	66.6%	

*Indicates statistical significance $p < 0.05$

Table 3: Correlation between cardiac biomarker and outcome ($n = 100$)

Markers	*Survived*		*Died*		*p-value*
	No.	*%*	*No.*	*%*	
Positive trop-I ($n = 26$)	4	15.4%	22	84.6%	<0.001*
Positive CPK-MB ($n = 32$)	4	12.5%	28	87.5%	<0.001*
Positive CPK-NAC ($n = 28$)	3	10.7%	25	89.3%	<0.001*
Positive LDH ($n = 30$)	3	10.0%	27	90.0%	<0.001*

*Indicates statistical significance $p < 0.05$

Table 4: Multivariate analysis of factors predicting mortality in aluminum phosphide poisoning ($n = 100$)

Factor	*Odds ratio*	*95% CI*	*p-value*
Abnormal ECG	6.44	2.42–17.15	<0.001*
Positive troponin-I	20.31	6.20–66.54	<0.001*
Positive CPK-MB	22.11	7.23–73.34	<0.001*
Positive CPK-NAC	24.34	5.02–72.45	<0.001*
Positive LDH	16.78	3.02–32.43	<0.001*
Abnormal PaO_2	9.03	3.65–22.34	<0.001*
Abnormal pH	4.89	2.01–11.88	<0.001*
Global hypokinesia	8.42	3.12–22.71	<0.001*
Decreased ejection fraction	18.67	4.86–71.72	<0.001*

*Indicates statistical significance $p < 0.05$

($p < 0.001$). Echocardiographic abnormalities, including decreased ejection fraction (OR 8.67) and global hypokinesia (OR 0.42), were also significant indicators of mortality, reinforcing the role of myocardial dysfunction in prognosis. Additional predictors included abnormal PaO_2 (OR 0.03), abnormal ECG (OR 0.44), and acid-base disturbances, particularly abnormal pH (OR 0.89). The high statistical significance ($p < 0.001$ for all) highlights the reliability of these factors in early risk stratification. This analysis supports the development of a practical, multiparameter prognostic model for early identification of high-risk patients, facilitating timely and aggressive therapeutic interventions in AlP poisoning (Table 4).

Discussion

Aluminum phosphide poisoning is notorious for its high case fatality rate and rapid clinical deterioration. This study aimed to identify reliable prognostic indicators for mortality through multivariate analysis. Findings from our 100-patient cohort underscore the critical role of cardiac dysfunction, oxygenation parameters, and acid-base balance in determining patient outcomes.

Among cardiac biomarkers, troponin-I, CPK-MB, CPK-NAC, and LDH were significantly elevated in nonsurvivors, with odds ratios ranging from 16.78 to 24.34. Troponin-I, in particular, demonstrated strong prognostic value, with 84.6% mortality in troponin-positive patients. Soltaninejad et al.[5] and Kalawat et al.[7] previously reported similar associations, highlighting the myocardial injury mechanism through mitochondrial disruption and oxidative stress caused by phosphine gas.

Electrocardiographic abnormalities were present in 60% of patients, with ST-T changes being the most frequent (60%), followed by sinus tachycardia and bradycardia. Specific abnormalities such as atrial fibrillation and heart block were associated with 100% mortality. These findings are consistent with those by Siddique et al.[12] and Singh et al.[8] and underscore the importance of ECG as a rapid and accessible tool for mortality risk stratification.

The most critical echocardiographic predictors were global hypokinesia and reduced ejection fraction. Mortality was 66.6% among those with reduced EF and 50% among those with hypokinesia, while all patients with normal echocardiograms survived. Elgazzar et al. and Sheta et al.[11] also emphasized the prognostic importance of these echocardiographic findings in their respective cohorts. Given that echocardiography is noninvasive and widely available, its utility as a bedside predictor is invaluable in the emergency setting.

Multivariate logistic regression identified 6 independent predictors of mortality: positive troponin-I (OR = 20.31), reduced EF (OR = 18.67), abnormal PaO_2 (OR = 9.03), global hypokinesia (OR = 8.42), abnormal ECG (OR = 6.44), and abnormal pH (OR = 4.89). These factors formed the foundation for the proposed risk prediction nomogram.

The interplay between acid-base imbalance and cardiac function was also evident. A strong association was found between low pH, low bicarbonate levels, and the presence of ECG abnormalities. These findings reinforce the understanding that metabolic acidosis not only reflects severity but also exacerbates myocardial dysfunction. Vikhe et al.[13] and Farzaneh et al.[9] also validated bicarbonate and pH as predictive tools in their analyses.

Our findings converge with those of Elgazzar et al.,[14] who also reported that global left ventricular hypokinesia, ECG abnormalities, and low oxygen saturation were independent predictors of mortality. While we did not assess the SOFA score, the identified variables overlap significantly with components of SOFA and other critical care scoring systems, supporting their integration into emergency protocols.

In summary, this study emphasizes the role of early and comprehensive cardiac evaluation in the prognostication of AlP poisoning. The use of accessible tools—ECG, echocardiography, cardiac biomarkers, and ABG—enables effective risk stratification. Our nomogram approach allows clinicians to anticipate adverse outcomes and tailor management strategies accordingly. Future work should focus on external validation and refining this tool for broader clinical use.

Conclusion

Our study demonstrates that certain clinical, biochemical, electrocardiographic, and echocardiographic parameters can serve as reliable prognostic indicators in AlP poisoning. Notably, positive troponin-I, reduced ejection fraction, abnormal ECG findings, low PaO_2, global hypokinesia, and acid-base disturbances were identified as independent predictors of mortality on multivariate analysis. The findings support the development of a practical, bedside prognostic tool that can guide clinicians in triaging patients for intensive monitoring and interventions.

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