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Examining the effectiveness of prognostic scoring systems and mortality predictors in aluminum phosphide poisoning: insights from emergency department settings

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Abstract

Aluminum Phosphide (AIP), a potent rodenticide and fumigant pesticide, poses a significant health threat, especially in agricultural communities. This study aimed to investigate demographic trends and predictive factors influencing outcomes in AIP poisoning patients presenting to the Emergency Department (ED). With an alarming mortality rate of 80.4%, identifying predictors of mortality became imperative. Non-survivors tended to be older and presented with distinct vital signs, such as tachycardia and shock upon ED arrival. Prognostic scoring systems like the Modified Early Warning Score (MEWS), Shock Index (SI), Mean Arterial Pressure (MAP), Glasgow Coma Scale (GCS), and serum lactate emerged as robust mortality predictors, with good accuracy (Area Under the Curve, AUC, MEWS=0.904, SI=0.914, MAP=0.869, GCS=0.829, lactate=0.962). This study favors integrating these predictors into routine ED practices, particularly in Low- and Middle-Income Countries (LMICs), to prognosticate and enhance management outcomes in patients with AIP poisoning, offering essential guidance for emergency physicians.

Introduction

Aluminum phosphide (AIP) poisoning presents a significant health hazard, especially prevalent in agricultural communities within Low- and Middle-Income Countries (LMICs). Annually, an estimated 1-5 million cases afflict agricultural workers, predominantly in developing regions, with mortality rates ranging from 40% to 80%.¹⁻³ AIP is primarily used as a fumigant pesticide to safeguard stored grains against pests and is readily accessible in the form of 3-gram tablets, each containing 56% aluminum phosphide.⁴ Despite its widespread usage, its lethal potential is alarming, as ingestion of around 150-500 mg can have fatal consequences.⁵

Since the early 1990s, the unregulated sale of AIP in Indian markets has increased a notable surge in poisoning cases.³ Insufficient regulatory measures, deficient training, inadequate surveillance systems, and the absence of Personal Protective Equipment (PPE) can exacerbate the susceptibility to poisoning, especially in areas heavily reliant on these pesticides for agricultural activities. This contributes to the escalating challenge posed by a preventable yet highly toxic substance within the domain of public health emergencies.⁶

AIP toxicity is due to the production of phosphine gas (PH₃) upon contact with moisture, notably in the stomach's acidic environment.⁷ Phosphine disrupts cellular function by inhibiting cytochrome C oxidase, leading to cellular hypoxia and Adenosine Triphosphate (ATP) depletion. Additionally, it triggers oxidative stress, damaging cellular components and inhibiting enzymatic processes, increasing cellular damage.⁸ These manifest as circulatory failure, arrhythmias, and severe metabolic acidosis, which ultimately result in multi-organ dysfunction. The lack of a

specific antidote complicates treatment, making AIP poisoning particularly challenging to manage and frequently resulting in unfavorable outcomes.

The prognosis of acute AIP poisoning generally depends on clinical presentation, point-of-care investigations, and laboratory features. In LMICs, where resources may be limited, poison-related factors can also serve as the primary prognostic indicators in primary healthcare settings. These factors not only facilitate rapid triaging but also aid in identifying patients requiring priority admission to the Intensive Care Units (ICUs).⁹ Tools like the Acute Physiologic Assessment and Chronic Health Evaluation II (APACHE II) and Sequential Organ Failure Assessment (SOFA) scores demonstrate excellent predictive abilities in ICU, but their use in resource-limited Emergency Department (ED) is under-explored. Additionally, scoring tools such as the Modified Early Warning Score (MEWS), Shock Index (SI), Glasgow Coma Scale (GCS), Mean Arterial Pressure (MAP), serum lactate levels, and sodium levels, among others, play crucial roles in ED, which are based only on vital parameters and/or point-of-care investigations.¹⁰

Given the high incidence and mortality rates associated with acute AIP poisoning globally, there is a critical need for better prognostic tools. This study seeks to evaluate the predictive factors of mortality and outcomes in patients with AIP poisoning, with a special emphasis on evaluating the effectiveness of scoring systems like MEWS, SI, MAP, GCS, and others in the ED setting.

Materials and Methods

Study design and setting

This was a retrospective cross-sectional study. Data was collected from patients who arrived at the ED between 1st March 2013 and 1st March 2023. Our ED has an average annual visit of around

25,000 cases. Patients who presented to our ED with acute AIP poisoning were selected for the study.

Inclusion criteria

The patients who were diagnosed with AIP poisoning through history, evidence like poison bottles with clinical pictures consistent with the toxidromes within 48 hours of exposure.

Exclusion criteria

Patients with doubtful diagnosis, exposure to multiple toxins, presentation after 48 hours of exposure, partially treated at an outside hospital, pregnant patients, patients with cardiac diseases and hypertension.

Study protocol

Upon arrival at the ED, patients were initially assessed and stabilized by primary survey where airways, breathing, circulation, disability, and exposure were evaluated; adjuncts like Arterial Blood Gas Analysis (ABG), Electrocardiography (ECG), capillary glucose level were used and patients were stabilized. Patients with threatened airways were intubated by Rapid Sequence Intubation (RSI). Oxygen support to maintain partial pressure of oxygen above 80 mmHg. Mean arterial pressure was maintained above 60 by initial fluid bolus with Intravenous (IV) Normal Saline (NS) at 20 mL/kg and switching over to vasopressor like nor-epinephrine 0.05-0.1 mcg/kg/min as first line, then epinephrine at 0.05-2 mcg/kg/min as second line and vasopressin 0.01-0.03 units/min as third line. If shock still persisted, then novel therapy like High-dose Insulin Therapy (HIT) or Intra-Arterial Balloon Pump (IABP) was considered.¹¹

Arrhythmia was controlled by inj lidocaine 1-1.5 mg/kg IV over 5 min. Acidosis was treated with inj sodium bicarbonate 1-2 mEq/kg IV when pH was less than 7.1. Seizure was treated with inj lorazepam 0.1 mg/kg slow IV. Inj magnesium sulphate at 3g in 250 ml NS IV bolus over 30 min followed by 6 g in 500 mL NS over 24 hours for 5-7 days was administered to all patients. In all the cases, gastric lavage with normal saline was intentionally excluded. Instead, the stomach was instilled only with 100 mL of coconut oil and 100 g of activated charcoal via Ryle's tube.¹¹

A complete history from patient bystanders was collected, including circumstantial evidence, such as finding empty bottles of AIP at home. Demographic profile and clinical data, along with initial vital signs, were compiled, with calculations and observations of key scores such as MAP, SI, MEWS, and GCS. Basic blood investigations like complete blood count, renal function tests, liver function tests, and coagulation profiles were sent from ED. Patients were shifted to the ICU after initial stabilization. The patients were followed until death or discharge and grouped as non-survivors and survivors, respectively. Survivors were sent for psychiatric counseling before discharge when indicated.

Outcomes

The primary outcome was to assess the predicting factors of mortality and outcome of AIP poisoning. The secondary outcome was to determine the demographic profile of the patients with AIP poisoning in South India.

Ethical considerations

The research obtained approval from the SNMC Ethical Committee Board, ensuring compliance with the ethical guidelines outlined in the Declaration of Helsinki regarding medical research ethics.

Statistical analysis

Data analysis was conducted using the software SPSS version 23.0. Descriptive statistics were computed, such as frequencies, percentages, means, standard deviations, and medians. Inferential statistics, including the Mann-Whitney U test or student t-test for comparisons between survivors and non-survivors and the chi-square test for association between categorical variables, were performed. The significance level for all statistical analyses was set at 5%. Logistic regression analyses were conducted to explore the relationship between variables. The Receiver Operating Characteristic curve (ROC) was employed to determine the optimal cutoff point, providing sensitivity, specificity, and the Area Under the Curve (AUC), and a value between 0.9-1 is considered excellent, 0.8-0.9 as excellent, 0.7 -0.8 as fair, but 0.6-0.7 and 0.5-0.6 was considered as poor and fail as per academic point system. Statistical significance was defined as a p-value ≤ 0.05 .

Results

Throughout the study period, our institute admitted a total of 3214 patients who were exposed to various toxins. Among these, pesticides were the primary culprit for 26.1% of cases, totaling 839 patients. Within this subgroup, 8.1% (68 patients) were identified with AIP poisoning. However, nine patients were excluded due to incomplete documentation, two due to ambiguous medical history, four due to involvement with multiple poisoning agents, and seven due to seeking critical

care management only after initial treatment at another hospital. Of 46 patients, 44 were due to deliberate self-poisoning, and two were because of accidental exposure.

Among the study group, 9 (19.6%) were survivors, and 37 (80.4%) were non-survivors. Notably, while females comprised a slightly higher proportion among survivors (55.6%), males were more prevalent among non-survivors (56.8%). Age-wise, a significant majority of survivors fell within the 0-20 years bracket (44.4%), contrasting with non-survivors, where a larger proportion were aged 21-40 years (37.8%). Occupationally, survivors included a notable percentage of farmers (55.6%) followed by students (22.2%), whereas non-survivors had more farmers (45.9%) and office workers (16.2%). Additionally, a majority of both survivors (77.8%) and non-survivors (75.6%) resided in rural areas (Table 1).

Notable differences with other parameters include age, with non-survivors significantly older (41.00 ± 7.67 years) compared to survivors (24.11 ± 3.48 years). Non-survivors also displayed tachycardia (154.81 ± 19.56) and lower systolic blood pressure (57.02 ± 18.68) than survivors, with p-value <0.05 and <0.001 , respectively. Additionally, non-survivors had delayed presentation times after ingestion (215.67 ± 82.95 min) and shorter hospital stays (1.89 ± 0.73 days) compared to survivors (presentation time: 47.77 ± 23.73 min, hospitalization: 4.88 ± 1.45 days), due to early mortality, both with p-values <0.001 . Also, the mortality group exhibited lower MAP, higher SI, higher MEWS, and lower GCS scores, all with p-values <0.001 except for GCS ($p < 0.05$). They also had lower bicarbonate levels ($p < 0.05$) and raised serum lactate levels ($p < 0.001$) compared to survivors. Other parameters, including capillary glucose and pH, did not show significant differences between the two groups (Table 2).

Survivors demonstrated higher sodium levels (133.55 ± 2.29) compared to non-survivors (131.32 ± 2.12), while potassium levels showed no significant disparity. Notably, survivors

exhibited lower total white blood cell counts (8783.00 ± 2072.15) but higher platelet counts (3.51 ± 1.26). Additionally, creatinine levels were lower in survivors (1.74 ± 0.69) compared to non-survivors (2.47 ± 0.47), indicating better renal function. Markers of liver function, such as Serum Glutamic Oxaloacetic Transaminase (SGOT) and Serum Glutamic Pyruvic Transaminase (SGPT), were significantly lower in survivors (55.44 ± 16.17 and 82.66 ± 24.00 , respectively), suggesting less hepatic injury. However, the two groups had no significant difference in International Normalized Ratio (INR) levels. These findings underscore the importance of laboratory parameters in predicting patient outcomes and guiding clinical management decisions (Table 3).

Survival outcomes were associated with distinct clinical interventions and cardiac manifestations. Specifically, while 70% of survivors required vasopressor support, this intervention was universally necessary among non-survivors. Moreover, a notable contrast was observed in ventilator dependence, with 20% of survivors vs 100% of non-survivors requiring ventilation. On initial presentation at ED, abnormal ECG findings were prevalent, affecting 70% of survivors and all non-survivors. Further delineation of cardiac rhythms revealed a higher incidence of sinus tachycardia in both groups, with 80% of non-survivors and 70% of survivors affected. However, only non-survivors experienced atrial fibrillation (10%) and ventricular tachycardia (10%). None of the survivors exhibited atrial fibrillation or ventricular tachycardia (Figure 1).

In assessing the accuracy of mortality prediction, the area under the ROC curve was utilized. Exceptional accuracy was found for MEWS, lactate, and SI, boasting AUC values of 0.904, 0.962, and 0.914, respectively. Both MAP and GCS demonstrated commendable accuracy, recording AUC values of 0.869 and 0.829. Conversely, sodium displayed a moderate accuracy level with an AUC of 0.763, while creatinine exhibited fair accuracy with an AUC of 0.773 (Table 4, Figure 2).

Discussion

Poisoning constitutes a major public health challenge in developing nations, wherein the epidemiology of poisoning cases, the causative agents, and resultant morbidity and mortality exhibit regional variations and temporal trends.¹² In India, self-poisoning incidents comprise 18.0% of total poisoning cases over the previous decade. Geographical differences exist in the choice of pesticides employed for such self-harm, but Organophosphates (OP), followed by AIP, prevail in most regions.¹³ Although poisonings are the second-leading cause of death in India, after traffic accidents, they frequently go unnoticed and underfunded in the healthcare systems. Moreover, accurately diagnosing such cases presents a significant challenge, further complicating effective intervention strategies.

Our study found that 26.1% of poisoning cases stemmed from pesticide exposure, with 8.1% involving AIP specifically. These findings contrast with a global perspective presented in a 2020 systematic review, where 44% of farmers worldwide encountered pesticide poisoning, and 14% experienced AIP poisoning.¹⁴ This difference could be attributed to unique factors within South Indian farming communities. For instance, they benefit from interest subsidies, easier loan approvals, and higher literacy rates, which contribute to improved food security and a societal shift towards embracing information and technology. This indicates a reduced reliance solely on agriculture compared to other regions in Southeast Asia.^{15,16}

Regarding age and gender distribution, our study observed predominance in male patients within the 20-40 age group. This contrasts with findings from an observational study conducted in Egypt by Sheta *et al.*, which noted a higher incidence of AIP poisoning among females aged 10-20.¹⁷ Cultural differences may contribute to this variation, as in India, men often serve as the primary breadwinners of their families, assuming financial responsibilities immediately after marriage and

better access to fumigants. Moreover, given the legal marriage age of 21 years in India, men usually begin their careers and assume financial responsibilities during their second to fourth decade of life.¹⁸ Additionally, it's worth noting that our study included a significant proportion of farmers from rural areas, reflecting India's status as a predominantly agricultural country.¹⁵ These socio-cultural and occupational factors likely contribute to the varying demographic profiles of poisoning cases across different regions.

Clinical parameters are pivotal in triaging patients, guiding management decisions, and prognosticating outcomes in cases of AIP poisoning. However, limited studies have explored the correlation between vital parameters and point-of-care investigations upon arrival with survival outcomes, particularly in ED. A study conducted in Chandigarh, India, by Anbalagan *et al.* in 2022 sheds light on this aspect.¹⁹ They reported baseline vital parameters, including pulse rate (104.24 ± 16.55 bpm), systolic blood pressure (87.19 ± 26.37 mmHg), and GCS score (15/15). Interestingly, while this study did not separately analyze these vitals parameters for survivors and non-survivors, they did observe a difference in pH and bicarbonate levels. Among survivors, the pH levels were 7.32 ± 0.15 , contrasting with non-survivors at 7.11 ± 0.18 , potentially due to lactic acid accumulation from inhibited oxidative phosphorylation and compromised tissue perfusion. Some differences between our study and the Chandigarh findings may be attributed to the lower mortality rate observed in the latter. Chandigarh, as a premier government-run medical academy in India, offers treatment at subsidized rates, making it readily accessible to patients. This accessibility likely facilitates early patient presentation and enables a higher level of medical intervention, contributing to a lower mortality rate. This could elucidate why the majority of vital parameters appeared within normal ranges in their study.

Early identification of toxidromes and prompt implementation of life-supportive measures along with decontamination are pivotal in managing AIP poisoning, as delays can significantly exacerbate systemic toxicity. Our study revealed a strong association between timely presentation to the ED and improved mortality outcomes. Survivors arrived at our ED within an average of 47.77 ± 23.73 minutes after exposure, while non-survivors, unfortunately, presented significantly later, with an average delay of 215.67 ± 82.95 minutes and a difference found to be statistically significant ($p < 0.001$). In the Anbalagan *et al.* study, there were prolonged time intervals for both the initial contact with a healthcare facility and the initiation of decontamination procedures among non-survivors (ranging from 1.0 to 3.12 hours and 0.75 to 4.0 hours, respectively). However, these differences failed to attain statistical significance ($p = 0.089$ and $p = 0.312$, respectively). Nonetheless, it is critically important to minimize the duration from toxin exposure to the detection of symptoms, first contact with a healthcare provider, initiation of life-saving interventions, and commencement of decontamination strategies, as these measures collectively play a pivotal role in enhancing patient outcomes.¹⁹

On comparing the lab parameters of our study with two other studies done by Farzaneh *et al.* in 2015, a prospective study in Iran, and Mathai *et al.* in 2015 India, which was a retrospective study, significant differences in creatinine levels between survivors and non-survivors were observed.^{20,21} In the Iranian study, non-survivors exhibited higher creatinine levels (1.10 ± 0.49 mg/dL) than survivors (0.90 ± 0.19 mg/dL, $p = 0.04$), while in our study, non-survivors had elevated creatinine levels (2.47 ± 0.47 mg/dL) compared to survivors (1.74 ± 0.69 mg/dL, $p < 0.05$). Similarly, the Mathai *et al.* study also identified higher creatinine levels in non-survivors. However, while sodium levels did not significantly differ in the Iranian study, survivors in our study showed higher sodium levels compared to non-survivors ($p < 0.05$), reflecting a discrepancy between the two studies. Potassium

levels did not significantly differ between survivors and non-survivors in any of the studies. Late presentation of patients in India and the absence of well-trained Emergency Response Services (EMS) contribute to this situation. Ambulances in India are often manned solely by drivers without proper emergency response training, leading to delays in reaching medical facilities. In contrast, Iran established EMS in 1974 under the management of the Ministry of Health. This early implementation of EMS in Iran has facilitated timely field resuscitation efforts, a concept that is still largely theoretical in India.²²

A retrospective study conducted in Morocco in 2005 by Hajouji *et al.* unveiled concerning rates of ECG abnormalities (72%), alongside a majority of patients necessitating vasopressor support (61%) and mechanical ventilation (64%).²³ Similarly the study by Sheta *et al.* from Egypt, found ECG abnormalities in 41.2% of survivors and 76.9% of non-survivors.¹⁷ Our study echoes these observations where among survivors, 70% exhibited ECG changes and required vasopressor, with 20% needing ventilator support. In contrast, all non-survivors showcased ECG abnormalities and required both vasopressor and ventilator support. These findings underscore the cardiac toxicity of AIP secondary to cytochrome c oxidase inhibition and free radical injury to cardiac myocytes. In our study, we found that a MEWS score >7.5, MAP <60 mmHg, SI >0.99, GCS <11, and lactate >4.5 mmol/L were strong predictors of mortality, demonstrating good sensitivity, specificity, and excellent accuracy. Specifically, serum lactate, SI, and MEWS score exhibited superior AUC values in our analysis. These results align with previous research by other studies. Farzaneh *et al.* in Iran identified SBP <92.5 mmHg, HCO₃ <12.9 mEq/L, and GCS <14.5 as effective predictors of mortality in acute AIP poisoning, with good sensitivity and specificity.²⁰ Similarly, Sheta *et al.* demonstrated that SBP <80 mmHg and DBP <40 mmHg were associated with mortality, showing high sensitivity and specificity, with excellent accuracy.¹⁷

Our study has a few limitations that need to be acknowledged. Firstly, the relatively small sample size and retrospective design introduce inherent biases. Moreover, as our study was carried out in a tertiary care institute located in proximity to rural areas, it is important to acknowledge that the generalizability of our findings to healthcare settings worldwide may be constrained. Additionally, we did not account for variables such as the quantity and mode of AIP ingestion, whether the tablet was placed in the mouth before consumption (potentially increasing phosphine absorption) or mixed with water outside the mouth (potentially decreasing phosphine gas absorption). Factors like the freshness of the AIP tablet also remain unexplored, potentially impacting toxicokinetics. To enhance predictive accuracy, future studies should incorporate larger datasets, employ more sophisticated modeling techniques, explore novel biomarkers, and employ advanced data collection methods. Despite these limitations, our study offers valuable insights into prognostication in the ED for AIP poisoning, laying the groundwork for further research and refinement of patient care protocols.

Conclusions

Our study provides insights into the intricate clinical aspects of AIP poisoning, revealing demographic patterns, clinical manifestations, and predictive markers impacting patient outcomes in the ED. It emphasizes the critical significance of early diagnosis and intervention in AIP poisoning, pinpointing key mortality predictors such as MEWS, GCS, Shock Index, and MAP with robust sensitivity, specificity and accuracy thus implementing them in routine ED practices could potentially standardize ED protocols in LMICs, thereby enhancing the management of AIP poisoning cases.

References

1. Mew EJ, Padmanathan P, Konradsen F, et al. The global burden of fatal self-poisoning with pesticides 2006-15: systematic review. *J Affect Disord* 2017;219:93-104.
2. Chugh SN, Dushyant K, Ram S, Arora B. Incidence and outcome of aluminium phosphide poisoning in a hospital study. *Indian J Med Res* 1991;94:232-5.
3. Singh D, Jit I, Tyagi S. Changing trends in acute poisoning in Chandigarh zone: a 25-year autopsy experience from a tertiary care hospital in Northern India. *Am J Forensic Med Pathol* 1999;20:203-10.
4. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. *Arch Ind Hyg Toxicol* 2012;63:61-73.
5. Wahab A, Zaheer M, Wahab S, Khan R. Acute aluminium phosphide poisoning: an update. *Hong Kong J Emerg Med*. 2008;15:152-5.
6. Thundiyil JG, Stober J, Besbelli N, Pronczuk J. Acute pesticide poisoning: a proposed classification tool. *Bull World Health Organ* 86:205-9.
7. Anand R, Binukumar BK, Gill KD. Aluminum phosphide poisoning: an unsolved riddle. *J Appl Toxicol* 2011;31:499-505.
8. Chefurka W, Kashi KP, Bond EJ. The effect of phosphine (gas toxic to insects) on electron transport in mitochondria. *Pestic Biochem Physiol* 1976;6:65-84.
9. Villa-Manzano AI, Zamora-Lopez XX, Huerta-Viera M, et al. Mortality and factors associated with phosphide poisoning: prospective cohort study. *Rev Med Inst Mex Seguro Soc* 2019;57:156-61.

10. Ghonem MM, Abdelnoor AA, Hodeib AA. Shock and modified shock indices in predicting poisoning severity and outcomes in acute aluminum phosphide poisoned patients. *Toxicol Res* 2023;13:124-8.
11. Gurjar M, Baronia AK, Azim A, Sharma K. Managing aluminum phosphide poisonings. *J Emerg Trauma Shock* 2011;4:378-84.
12. Aggarwal N, Sawlani KK, Chaudhary SC, et al. Study of pattern and outcome of acute poisoning cases at tertiary care hospital in North India. *J Family Med Prim Care* 2023;12:2047-52.
13. Senapati RE, Jena S, Parida J, et al. The patterns, trends and major risk factors of suicide among Indian adolescents - a scoping review. *BMC Psychiatry* 2024;24:35.
14. Boedeker W, Watts M, Clausing P, Marquez E. The global distribution of acute unintentional pesticide poisoning: estimations based on a systematic review. *BMC Public Health* 2020;20:1875.
15. Sandhu N. Dynamics of banks' lending practices to farmers in India. *J Small Business Enterprise Develop* 2020;28:102-20.
16. Moeis FR, Dartanto T, Moeis JP, Ikhsan M. A longitudinal study of agriculture households in Indonesia: the effect of land and labor mobility on welfare and poverty dynamics. *World Dev Perspect* 2020;20:100261.
17. Sheta AA, El-Banna AS, Elmeguid RA, et al. A study of the predictive factors of mortality in acute poisoning with aluminum phosphide with special reference to echocardiography and SOFA score. *Environ Sci Pollut Res Int* 2019;26:33135-45.
18. Singh M, Shekhar C, Shri N. Patterns in age at first marriage and its determinants in India: a historical perspective of last 30 years (1992-2021). *SSM Popul Health* 2023;22:101363.

19. Anbalagan LC, Pannu AK, Bhalla A, et al. Prognostic significance of poison-related factors and consumption patterns in acute aluminum phosphide poisoning. *Turk J Emerg Med* 2023;23:88-95.
20. Farzaneh E, Ghobadi H, Akbarifard M, et al. Prognostic factors in acute aluminium phosphide poisoning: a risk-prediction nomogram approach. *Basic Clin Pharmacol Toxicol* 2018;123:347-55.
21. Mathai A, Bhanu MS. Acute aluminium phosphide poisoning: can we predict mortality? *Indian J Anaesth* 2010;54:302-7.
22. Shakeri K, Jafari M, Khankeh H, Seyedin H. history and structure of the fourth leading emergency medical service in the world; a review Article. *Arch Acad Emerg Med* 2019;7:e17
23. Hajouji Idrissi M, Oualili L, Abidi K, et al. Severity factors of aluminium phosphide poisoning (Phostoxin). *Ann Fr Anesth Reanim* 2006;25:382-5.

Table 1. Demographic profile in Aluminum Phosphide (AIP) poisoning.

Variables	Survivor (n=9)	Non-survivor (n=37)	Total (n=46)
Sex			
Male	4 (44.4)	21 (56.8)	25 (54.3)
Female	5 (55.6)	16 (43.2)	21 (45.7)
Age (years)			
0-20	4 (44.4)	7 (18.9)	11 (23.9)
21-40	2 (22.2)	14 (37.8)	16 (34.8)
41-60	2 (22.2)	9 (24.4)	11 (23.9)
>60	1 (11.1)	7 (18.9)	8 (17.4)

Occupation			
Student	2 (22.2)	9 (24.4)	11 (23.9)
Office worker	1 (11.1)	6 (16.2)	7 (15.2)
Farmer	5 (55.6)	17 (45.9)	22 (47.8)
Home maker	1 (11.1)	5 (13.5)	6 (13.1)
Residence			
Rural	7 (77.8)	28 (75.6)	35 (76.1)
Urban	2 (22.2)	9 (24.4)	11 (23.9)

Table 2. Clinical parameters in Aluminum Phosphide (AlP) poisoning on presentation to Emergency Department (ED).

Variables	Survivors (Mean ± SD)	Non- survivors (Mean ± SD)	p-value
Vitals on presentation to ED			
Age (years)	24.11±3.48	41.00±7.67	
Pulse rate (bpm)	121.77±24.31	154.81±19.56	p<0.05
Systolic BP (mmHg)	90.00±14.14	57.02±18.68	p<0.001
Diastolic BP (mmHg)	52.22±10.92	27.02±23.55	p<0.05
Temperature (°C)	38.11±0.33	37.35±0.67	p<0.05
Respiratory rate (cpm)	20.55±4.15	18.91±5.26	p>0.05
Time of presentation after ingestion (min)	47.77±23.73	215.67±82.95	p<0.001

Days of hospitalization	4.88±1.45	1.89±0.73	p<0.001
Scoring on ED presentation			
MAP	64.81±11.19	37.02±21.39	p<0.001
SI	1.43±0.57	3.40±3.43	p<0.001
MEWS	5.55±2.92	9.54±1.19	p<0.001
GCS	11.66±3.35	7.48±1.69	p<0.05
Point of care investigation on arrival to ED			
Capillary glucose (mg/dL)	188.88±65.13	195.16±89.99	p>0.05
pH	7.31±0.13	7.26±0.16	p>0.05
Bicarbonate	19.77±4.35	16.72±3.42	p<0.05
S. Lactate	3.23±0.72	8.68±3.77	p<0.001

BP, Blood Pressure; GCS, Glasgow Coma Scale; MAP, Mean Arterial Pressure; MEWS, Modified Early Warning Score; SI, Shock Index; SD, Standard Deviation

Table 3. Lab parameters in Aluminum Phosphide (AlP) poisoning.

Variables	Survivors (Mean ± SD)	Non-survivors (Mean ± SD)	P-value
Sodium	133.55±2.29	131.32±2.12	p<0.05
Potassium	4.06±0.31	4.57±0.92	p>0.05
Total count	8783.00±2072.15	25008.78±7042.57	p<0.001
Platelet	3.51±1.26	1.34±0.47	p<0.001
Creatinine	1.74±0.69	2.47±0.47	p<0.05

SGOT	55.44±16.17	230.00±123.62	p<0.001
SGPT	82.66±24.00	328.70±161.55	p<0.001
INR	1.36±0.10	1.46±0.42	p>0.05

INR, International Normalized Ratio; SD, Standard Deviation,SGOT, Serum Glutamic Oxaloacetic Transaminase; SGPT, Serum Glutamic Pyruvic Transaminase

Table 4. Factors evaluated for mortality predictors in Aluminum Phosphide (AlP) poisoning.

Parameters	Cutoff	Sn	1-Sp	Sp	Area	PPV	NPV	Accuracy	p-value
MEWS	7.5	0.946	0.333	0.667	0.904	0.92	0.75	0.89	p<0.001**
MAP	60	0.667	0.162	0.838	0.869	0.91	0.5	0.8	0.001
SI	0.99	100	0.778	0.222	0.914	0.84	1	0.85	p<0.001
GCS	11	0.667	0.027	0.973	0.829	0.92	0.86	0.91	0.002
S. Lactate	4.2	0.892	0.111	0.889	0.962	0.97	0.67	0.89	p<0.001
Sodium	133.5	0.556	0.162	0.838	0.763	0.89	0.45	0.78	0.015
Creatinine	2	0.73	0.444	0.556	0.773	0.87	0.33	0.7	0.012

GCS, Glasgow Coma Scale; MAP, Mean Arterial Pressure; MEWS, Modified Early Warning Score; NPV, Negative Predictive Value; PPV, Positive Predictive Value; SI, Shock Index; Sn, Sensitivity, Sp, Specificity

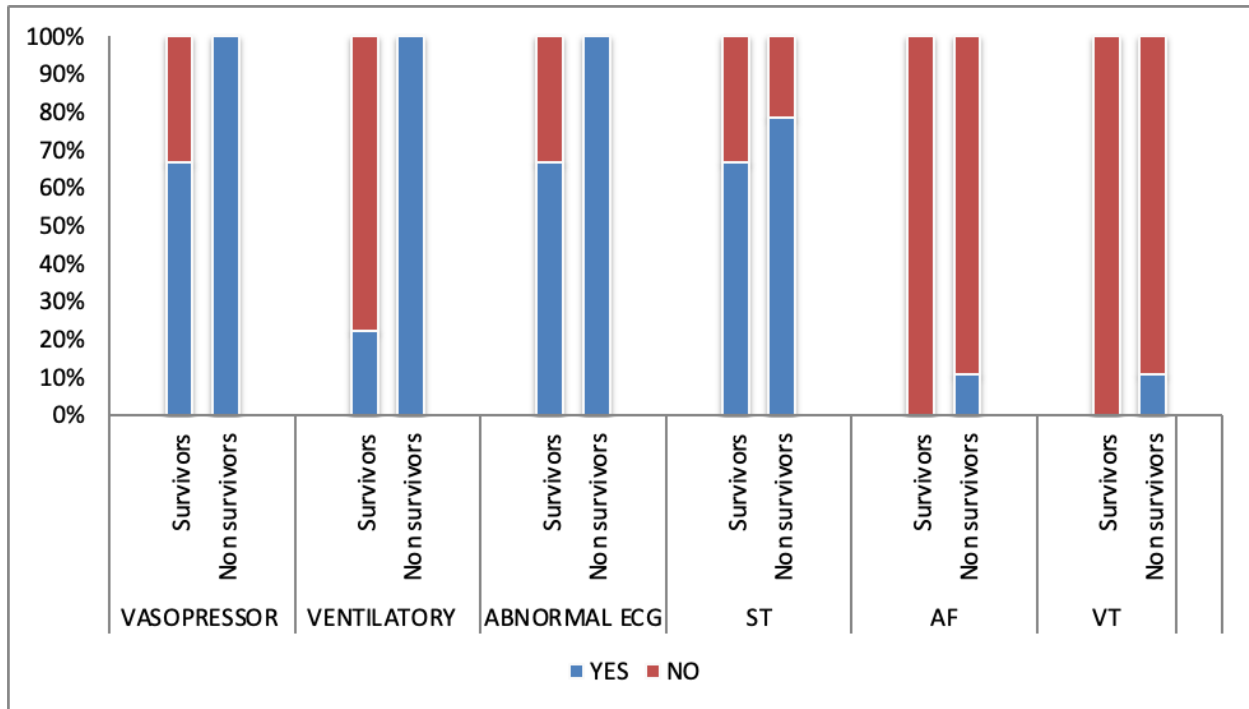


Figure 1. Cardiac manifestations and need for mechanical ventilator support in Aluminum Phosphide (AlP) poisoning.

AF, Atrial Fibrillation; ST, Sinus Tachycardia; VT, Ventricular Tachycardia

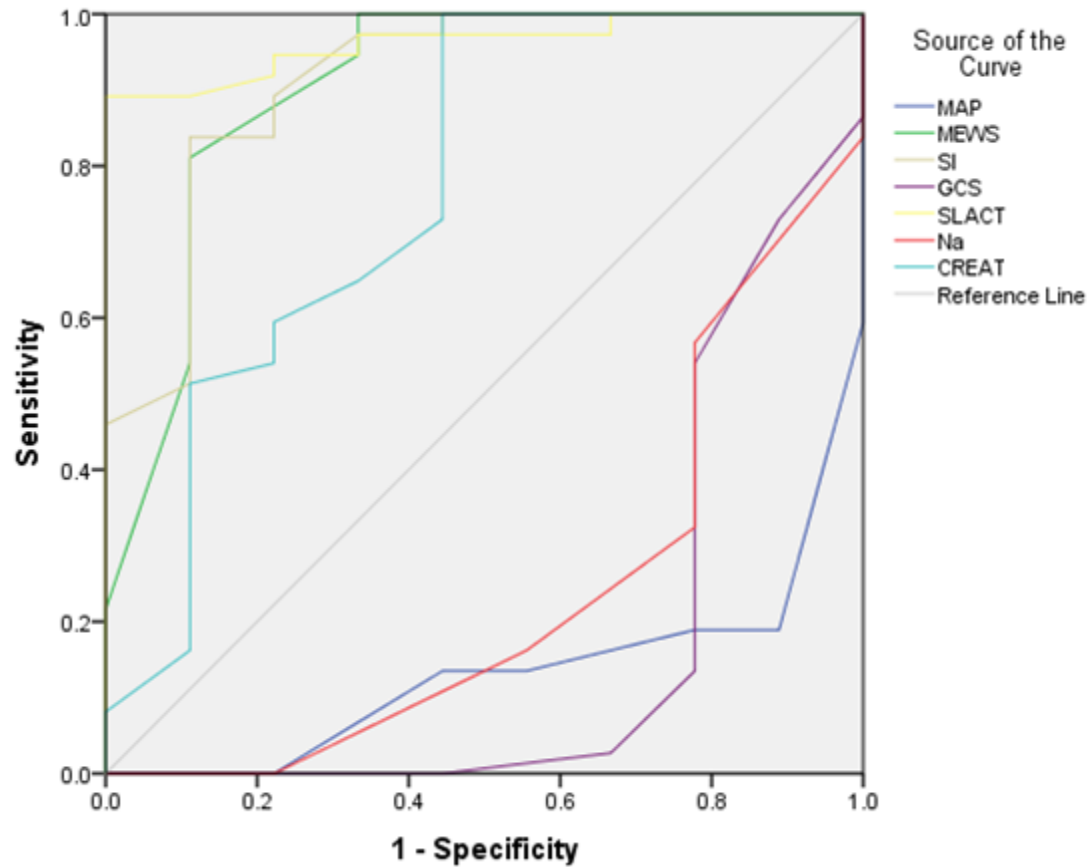


Figure 2. Receiver Operating Characteristic (ROC) curve for various mortality predictors in Aluminum Phosphide (AIP) poisoning.

Creat, Creatinine; GCS, Glasgow Coma Scale; MAP, Mean Arterial Pressure; MEWS, Modified Early Warning Score; SI, Shock Index; SLact, Serum Lactate)

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