Section: Emergency

Original Research Article

METABOLIC PROFILE AND OUTCOME OF PATIENTS PRESENTING WITH ACUTE TOXIN EXPOSURE AT THE EMERGENCY DEPARTMENT OF A TERTIARY CARE HOSPITAL IN NAVI MUMBAI

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ABSTRACT

Background: Acute toxin exposure represents a significant cause of morbidity and mortality worldwide. Metabolic derangements, particularly acid-base disturbances, are common manifestations of poisoning with a prognostic significance. We aimed to evaluate the metabolic profile and outcomes in patients with acute toxin exposure to the emergency room. **Materials and Methods:** A retrospective observational study was conducted on 150 patients with acute toxin exposure. Demographic data, toxin type, clinical parameters, laboratory values including arterial blood gas analysis, and mortality at 0-day, 2-day, and 8-day were analyzed. Acid-base disturbances were classified as primary and secondary disorders, and their association with mortality at 8-day was assessed. **Results:** The mean age of study population was 40.5 ± 9.8 years with 56% males. Aluminum phosphide (25.3%), hydrocarbons (24.7%), and organophosphates (20.7%) were the common toxins encountered. Acid-base analysis showed that 85.3% of patients had abnormal pH of which 74.2% were acidic, with primary metabolic acidosis being the most common primary disorder (43.3%) and respiratory alkalosis the most frequent secondary disorder (36%). The mortality rates were 4.7% at 0-hour, 12.7% at 48-hour, and 27.3% at 8-day. A significant association was found between primary acid-base disorders and 0-day mortality ($p<0.001$), with metabolic acidosis having the highest proportion of deaths (30.8%). **Conclusion:** This study found acute toxin exposure to be associated with significant acid-base disturbances, which correlate with mortality. Early recognition of these patterns can guide prognostication and therapeutic interventions.

INTRODUCTION

Acute poisoning is a major concern for global health, accounting for a large portion of emergency department (ED) visits and prolonged hospital stay. According to the World Health Organization, poisoning causes about 370,000 fatalities every year, with several cases leading to serious morbidity and mortality.[1] A wide variety of toxins are capable of inducing poisoning and controlling acute toxin exposure is complicated by the need to recognize and treat the severe metabolic disruptions that result from these exposures. India is known to have the highest burden of poisoning in the world. More than 50,000 people die every year due to toxic exposure. A study based on National Crime Records Bureau (NCRB) during the year 1995-2015, reported Maharashtra to have the highest number of suicide deaths due to pesticide i.e. 19.2% of total death followed by Andhra Pradesh with 17.6%. Considering this grave problem the State of Maharashtra imposed bans on selected pesticide in 2017.[2]

For the majority of poisoning cases, the emergency room is the initial point of contact, hence it is essential to set up quick and efficient evaluation procedures. Complex metabolic abnormalities that might have a major influence on patient outcomes are frequently discovered during the initial screening of these patients. Metabolic disturbances can occur due to acid–base imbalances, electrolyte abnormalities, alterations in glucose metabolism, and organ dysfunction. Recognizing these patterns is essential for early intervention and improving patient outcomes.

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The significance of metabolic profiling in poisoning cases has been brought to light by recent developments in clinical toxicology. This method entails a thorough evaluation of numerous biochemical indicators that can direct treatment efforts and offer vital insights into the degree of poisoning. According to studies, in cases of acute poisoning, specific metabolic rhythms can act as early indicators of mortality and morbidity.[3] For example, severe acidosis has been persistently linked to poor outcomes in a variety of poisonings (toxic alcohols, salicylates, and cyanide), especially when it is accompanied by a significant anion gap.

Early metabolic profiling has purposes beyond simple diagnosis. It is a useful tool for prognostication and risk stratification. Certain metabolic characteristics have been shown to predict the likelihood of complications and direct the allocation of resources in emergency situations when evaluated early in the course of poisoning.[4] Several scoring systems such as Poison Severity Score (PSS), New Poisoning Mortality Score (new-PMS, Simplified Acute Physiology Score (SAPS) II, Performance Grading Index (PGI) Score and other are available to grade the severity of poisoning, plan treatment and predict the outcome. This is especially important in environments with limited resources when making the best use of acute care units is essential.

The availability of point-of-care testing in emergency toxicology, allows quick evaluation of important metabolic indicators including blood gases, electrolytes, lactate, and renal function markers, has completely changed the way acute poisoning cases are managed. This technical development makes it possible to make therapeutic decisions instantly, which could lead to better results. Evidence supports that early identification and treatment of metabolic abnormalities can considerably decrease death from 30% to more than 55% even in severe cases of poisoning.[5]

Electrolyte imbalances are equally prevalent and might show up as abnormalities in calcium, sodium, or potassium levels. If these disruptions are not detected and treated immediately, they may result in potentially fatal consequences like cardiac arrhythmias, seizures, coma, respiratory failure, and acute organ failure.[6] Severe poisoning instances often accompany organ dysfunction, especially of the liver and kidneys, which can have a major effect on metabolic balance. Because of its involvement in the metabolism of toxins, the liver is especially susceptible to damage, and the kidneys' ability to eliminate toxins may be impaired, which could result in the buildup of harmful metabolites. For thorough patient care, it is essential to comprehend these organ-specific reactions.[7]

The significance of a methodical approach to metabolic assessment and monitoring is being emphasized more and more in modern toxicological management. This involves routinely assessing electrolyte levels, organ function metrics, acid-base status, and particular markers linked to toxins. Combining this data with clinical findings aids in directing therapy approaches and surveillance.[8] The present study has been conducted to assess metabolic profile in patients with toxin exposure and also to assess their outcome among patients visiting the ED of a tertiary care hospital.

MATERIALS AND METHODS

Study design and setting: A prospective observational study was done the Emergency Department (ED) of MGM Medical College, Navi Mumbai, India during July 2024 to February 2025 to evaluate the metabolic profile of patients presenting with acute poisoning and the outcome of treatment given. The hospital has over 900 beds with the ED having 25 beds and provides 24-hour services. Being a tertiary care referral centre, on an average we receive about 80 to 110 patients per day.

Sample size: Based on the proportion of acute poisoning cases visiting ED over the previous year was found to be 11%. Hence, using the Cochran formula, $n=Z^2*P*(1-P)/e^2$ where random normal variate Z = 1.96 for 95% of Confidence Interval, p is the proportion of acute poisoning i.e. 11% and margin of error (e) is 5%. The sample size was calculated to be 150.

Sampling Method: Consecutive sample of patient was done based on inclusion and exclusion till the desired sample size was reached.

Inclusion Criteria

- Patient of age group >18years of age of both gender, including pregnancy
- Patient with history of acute toxin consumption.

Exclusion Criteria

- Patient not willing to give sign and consent for participation in study
- Patients lost to follow-up

Data collection: The history of toxin consumption was carefully documented, including the type of toxin, approximate quantity consumed, time of consumption, and any immediate symptoms experienced. Cases where the type of toxin could not be identified were also included, provided there was a clear history of toxic exposure.

Upon arrival at the emergency department, each patient underwent an initial rapid assessment following standard emergency protocols. Arterial blood gas (ABG) analysis was performed within 30 minutes of arrival using arterial blood samples collected under aseptic conditions. The blood gas analyzer (model: Roche Cobas B221 Version 6) was calibrated calibrated daily according to manufacturer specifications, and quality control runs were performed at regular intervals. Multiple parameters (pH, PaCO2, HCO3, PaO2, SaO2, base excess) were systematically evaluated from the ABG samples to assess the patient's metabolic and respiratory status.

Oxygenation status was evaluated through multiple parameters. The partial pressure of oxygen (PaO2)

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and oxygen saturation (SaO2) were measured directly from the ABG. The PaO2/FiO2 (P/F) ratio was calculated to assess the severity of oxygenation impairment, with values below 300 indicating significant impairment.

Acid-base status was comprehensively assessed through the evaluation of pH, partial pressure of carbon dioxide (PaCO2), and bicarbonate (HCO3-) levels. Primary acid-base disorders were identified based on these parameters, and the presence and adequacy of physiological compensation were determined using standard compensatory formulas. Secondary acid-base disorders, if present, were also documented.

Various gaps were calculated to provide additional diagnostic information. The anion gap was calculated using the formula: [Na+] - ([Cl-] + [HCO3-]). Delta-delta (DD) ratio was computed to evaluate the relationship between changes in the anion gap and bicarbonate levels.

Electrolyte analysis included measurement of serum sodium (Na+), potassium (K+), chloride (Cl-) and calcium levels. Venous blood sample of 2-3 ml was drawn under aspetic condition and immediately transported to central laboratory. The sample was centrifuged at 3000 rpm for 10 minutes to separate the plasma/serum. These measurements were performed using ion-selective electrodes. Blood urea nitrogen (BUN) and random blood sugar (RBS) levels were also measured as part of the metabolic profile.

All laboratory parameters were measured using standardized laboratory protocols. Quality control measures were strictly adhered to, with regular calibration of all measuring instruments. The timing of sample collection was recorded, and all samples were processed within the recommended time frame to ensure accuracy of results. The final outcome of each patient was recorded, including survival status, duration of hospital stay, and presence of complications. All measurements and calculations were verified by two independent observers to ensure accuracy. Any discrepancies were resolved through discussion and consensus.

Ethical consideration: The study protocol was approved by the Institutional Ethics Committee (IEC number: DHR-EC/SC/2023/07/21) dated on 07.07.23, and written informed consent was obtained from all patients or their legal guardians before enrolment in the study.

Statistical Analysis: Data entry and analysis was performed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA). Data distribution normality was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data and median (interquartile range) for non-normally distributed data. Demographic characteristics and baseline variables among the three groups were compared using one-way analysis of variance (ANOVA) for normally distributed continuous variables. Categorical variables were analyzed using Chi-square test or Fisher's exact test as appropriate. Repeated measures ANOVA was used to analyze changes in gastric residual volume over time. The incidence of adverse events and the requirement for rescue medications were compared between groups using Chi-square test or Fisher's exact test. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 150 patient with acute poisoning were included for analysis with a mean age of 40.5 ± 9.8 years with 56% males. Suicidal poisoning was the cause among 84% of case with remaining being accidental. Aluminum Phosphide was the most common toxin (25.3%), followed by hydrocarbons (24.7%) and organophosphate poisoning (20.7%). Most of the patients i.e. 50.7% had GCS between 9-12 while very low GCS (<8) was seen among 22.7%. Ventilatory support was needed among 29.4%. (Table 1).

The average time since exposure was 12.93 hours (±6.91), indicating patients typically presented several hours after exposure. The mean pH was 7.19 (±0.24), slightly below normal, suggesting a tendency toward acidemia. Other notable values include elevated lactate levels at 4.29 mmol/L (±2.18), negative base excess at -8.39 (±7.27), and elevated serum creatinine at 2.74 mg/dL (±1.66), indicating renal impairment. The anion gap was also elevated at 14.47 (±2.79), suggesting that all toxins caused high AG gap. (Table 2).

The vast majority (85.3%, 128 patients) had abnormal pH with acidosis (74.21%, 95 patients) being three times more common than alkalosis (25.78%, 33 patients). Metabolic acidosis was the predominant primary acid-base disorder, occurring in 43.3% (65 patients). Respiratory acidosis was the second most common at 20.0% (30 patients). Normal acid-base status was observed in 14.7% (22 patients), while metabolic alkalosis occurred in 14.0% (21 patients) and respiratory alkalosis in 8.0% (12 patients). These findings demonstrate that acidosis (either metabolic or respiratory) was the primary acid-base disturbance in 63.3% of poisoning cases. Among secondary acid-base disorders, respiratory alkalosis was most common at 36.0% (54 patients), followed by normal acid-base status in 22.6% (34 patients). Both metabolic acidosis and respiratory acidosis occurred as secondary disorders in 13.3% (20 patients each). Metabolic alkalosis was present in 6.7% (10 patients). Mixed acid-base disorders were less frequent, with metabolic acidosis and alkalosis in 4.7% (7 patients) and combined respiratory and metabolic alkalosis in 3.3% (5 patients). This pattern suggests compensatory mechanisms attempting to normalize pH. Distribution of patients based on the type of primary and secondary acid-base disorder is given in table 3. Immediate mortality (0-hour) was 4.7% (7 patients). This increased to 12.7% (19

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patients) by 48 hours and further rose to 27.3% (41 patients) by day 8.

Table 4 shows the mortality rates for each toxin type across different timepoints, of which aluminum phosphide had the highest mortality rates: 7.9% at 0-hour, increasing to 18.4% at 48-hour and 39.5% by day 8. This was followed by hydrocarbons and organophosphate. The p-values at the various timepoints (0.292, 0.801, 0.454) were not statistically significant.

Table 5, examines relationships between specific toxins and primary acid-base disorders. Among patients with respiratory alkalosis, Aluminum Phosphide was most common (50%). Hydrocarbons were most associated with metabolic acidosis (27.7% of these cases). Organophosphate poisoning was prominent in metabolic alkalosis cases (28.6%). However, the p-value of 0.475 indicates these associations were not statistically significant, suggesting that multiple toxins can cause similar acid-base disturbances.

At 0-hour, metabolic acidosis had greater proportion of mortality (30.8%) than the other acid-base disorder which was statistically significant ($p<0.001$), but at 48-hour and 8-day there was no significant difference. [Table 6]

Table 1: Demographic details and type of poison among study participants (N=150)

Variable	Frequency	Percentage
Age group (in years)		
18-20	7	4.7
21-40	52	34.7
41-60	49	32.7
61-80	42	28.0
Sex		
Female	66	44.0
Male	84	56.0
Toxin type		
Aluminum Phosphide	38	25.3
Corrosives	15	10.0
Hydrocarbons	37	24.7
Organophosphate	31	20.7
Unknown	29	19.3
Glasgow Coma Scale		
3-8	34	22.7
9-12	76	50.7
13-15	40	26.7

Table 2: Laboratory parameter of study participants (N=150)

Parameter	Mean	SD*
Time since exposure (hrs)	12.93	6.910
pH	7.1915	0.24330
$PaCO_2$	38.67	12.086
HCO_3	23.49	7.196
Anion gap	14.47	4.786
Lactate	4.2947	2.1809
Base excess	-8.39	7.269
S.creatinine	2.74	1.66
S.potassium	4.4213	1.1557
S.sodium	137.83	7.561
S.calcium	9.23	1.12

SD-Standard deviation

Table 3: Acid-base disorders among the study participants (N=150)

Acid-base disorder	Primary	Secondary
Metabolic Acidosis	65 (43.3%)	20 (13.3%)
Metabolic Alkalosis	21 (14.0%)	10 (6.7%)
Normal	22 (14.7%)	34 (22.6%)
Respiratory Acidosis	30 (20.0%)	20 (13.3%)
Respiratory Alkalosis	12 (8.0%)	54 (36.0%)
Metabolic acidosis+alkalosis	0 (0%)	7 (4.7%)
Respiratory and metabolic alkalosis	0 (0%)	5 (3.3%)

Table 4: Association between toxin type and mortality (N=150)

Toxin Type	Mortality		
	0-day (n=7)	2-day (n=19)	8-day (n=41)
Aluminum Phosphide (n=38)	3 (7.9%)	7 (18.4%)	15 (39.5%)
Corrosives (n=15)	1 (6.7%)	2 (13.3%)	5 (33.3%)
Hydrocarbons (n=37)	2 (5.4%)	4 (10.8%)	11 (29.7%)
Organophosphates (n=31)	1 (3.2%)	3 (9.6%)	7 (22.6%)
Unknown (n=29)	0 (0.0%)	2 (6.9%)	5 (17.2%)
p-value	0.292	0.801	0.454

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Table 5: Association Between Toxin Type and Primary Acid-Base Disorder (N=150)

Toxin type	Primary Acid-Base Disorder					P-value
	Metabolic Acidosis	Metabolic Alkalosis	Normal	Respiratory Acidosis	Respiratory Alkalosis	
Aluminum Phosphide	11 (16.9%)	4 (19%)	7 (31.8%)	10 (33.3%)	6 (50%)	0.475
Corrosives	9 (13.8%)	2 (9.5%)	2 (9.1%)	2 (6.7%)	0	
Hydrocarbon	18 (27.7%)	3 (14.3%)	5 (22.7%)	7 (23.3%)	4 (33.3%)	
Organophosphate	13 (20%)	6 (28.6%)	6 (27.3%)	4 (13.3%)	2 (16.7%)	
Unknown	14 (21.5%)	6 (28.6%)	2 (9.1%)	7 (23.3%)	0	
Total	65 (43.3%)	21 (14.0%)	22 (14.6%)	30 (20.0%)	12 (8.0%)	

Table 6: Association between toxin type and mortality (N=150)

Primary Acid-Base Disorder	Mortality		
	0-day (n=7)	2-day (n=19)	8-day (n=41)
Metabolic Acidosis (n=65)	2 (30.8%)	10 (15.4%)	17 (26.1%)
Metabolic Alkalosis (n=21)	2 (9.5%)	2 (9.5%)	6 (28.6%)
Normal (n=22)	2 (9.1%)	3 (13.6%)	7 (31.8%)
Respiratory Acidosis (n=30)	1 (3.3%)	3 (10.0%)	9 (30.0%)
Respiratory Alkalosis (n=12)	0 (0.0%)	1 (8.3%)	2 (16.7%)
p-value	<0.001*	0.151	0.619

Statistically significant

DISCUSSION

Acute toxin exposure represents a significant global health concern with substantial morbidity and mortality. According to the World Health Organization, poisoning is responsible for over 100,000 deaths annually worldwide, with developing countries bearing a disproportionate burden of this preventable cause of mortality.[9] Acid-base disturbances serve as key indicators of the severity of poisoning that guide prognostication and therapeutic interventions.[10]

The age distribution in our study showed that the majority of patients (34.7%) were between 21-40 years followed by 41-60 years (32.7%), representing the economically productive population. This finding is consistent with other studies from developing countries, which report a higher incidence of poisoning in young and middle-aged adults.[11,12] There was a predominance of male (56%) patients in our study which aligns with the findings of multiple studies on acute poisoning. Mehrpour et al,[13] reported a similar gender distribution in their systematic review of aluminum phosphide poisoning, with males constituting approximately 55-60% of cases. This may be attributed to occupational use of this substance, particularly in agricultural settings where pesticides are commonly used making their availability being misused.

A GCS score of ≤ 8 was seen among 22.7% patients with 50.7% and 26.7% having moderate and high GCS. Ventilatory support was need by 29.4%. Mehrpour et. Al,[13] reported a total of 41.2% patients with acute poisoning needing ventilation, but this proportion based on the GCS score was not available. Regarding the type of toxin exposure, aluminum phosphide was the most common (25.3%), followed by hydrocarbons (24.7%), organophosphate's (20.7%), and corrosives (10%). The remaining 19.3% of cases involved unknown substances. The high prevalence of aluminum phosphide poisoning in our study is consistent with patterns observed in agricultural regions of South Asia, where it is widely used as a grain preservative. Due to ease of use in farming, low cost and easy availability, Aluminum phosphide poisoning has emerged as a major health concern in India, with mortality rates reported between 40-80%.[14] Even our study had a high overall mortality due to aluminum phosphide (65.7%). Bashardoust et al,[15] highlighted that aluminum phosphide poisoning is associated with severe metabolic acidosis and has one of the highest mortality rates among acute poisonings.

The mean time since exposure in our study was approximately 12.9 ± 6.9 hours, which represents a significant delay in seeking medical attention. This delay could potentially impact the effectiveness of interventions and ultimate outcomes. Liu et al,[16] demonstrated that acid-base interpretation at presentation can be a predictor of outcome in patients with acute organophosphate poisoning, emphasizing the importance of early assessment. The probable cause of delay in seeking treatment could be geographic barrier, logistic challenges and stigma attached to acute poisoning. There is need further research to evaluate the various reasons for delay in reaching the emergency department.

Laboratory parameters revealed significant metabolic derangements among the study population. The mean pH was 7.19 ± 0.24, indicating a tendency toward acidemia. This finding is particularly relevant as severe acidemia has been associated with increased mortality in poisoning cases. Lee et al,[17] and Kang et al,[18] showed that base deficit is a predictor of

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mortality in organophosphate insecticide poisoning, with higher base deficits associated with increased risk of 30-day mortality.

Our study revealed a high prevalence of acid-base disorders among patients with acute toxin exposure. Overall, 85.3% of patients had abnormal pH, with acidosis (74.2% of abnormal pH) being more common than alkalosis. This predominance of acidosis is consistent with the pathophysiology of many toxins, which either directly induce metabolic acidosis or cause tissue hypoperfusion leading to lactic acidosis. Primary metabolic acidosis was the most common acid-base disorder (43.3%), followed by respiratory acidosis (20%). This finding aligns with a large retrospective study by Hassanian-Moghaddam et al,[19] that examined acid-base disturbances in 1167 poisoned patients. They reported that patients with primary metabolic acidosis and respiratory compensation had significantly higher mortality (18.8%) compared to those with normal acid-base status. Interestingly, our study found respiratory alkalosis to be the most common secondary acid-base disorder (36%), followed by normal acid-base status (22.6%). Respiratory alkalosis in poisoning cases may be attributed to direct stimulation of the respiratory center by certain toxins, hypoxemia, or anxiety. The presence of mixed acid-base disorders reflects the complex pathophysiological disturbances in severe poisoning. The high prevalence of mixed acid-base disorders in our study population underscores the complex pathophysiological mechanisms involved in acute poisoning. These findings suggest that comprehensive acid-base assessment is essential for understanding the severity and progression of toxicity and guiding therapeutic interventions.

Immediate mortality (0-hour) was 4.7% (7 patients). This increased to 12.7% (19 patients) by 48 hours and further rose to 27.3% (41 patients) by day 8. This progressive increase in mortality demonstrates that many patients continued to deteriorate despite medical intervention, highlighting the lack of antidote or delayed arrival to receive treatment or potentially delayed effects of acute toxin exposure. There was significant association between primary acid-base disorders and 0-hour mortality ($p<0.001$), although this association was not statistically significant for 48-hour and 8-day mortality. Patients with metabolic acidosis had the highest proportion of deaths at all time points. Gil et al,[20] found that acid-base status, particularly components representing total buffer bases and respiratory components, played an important role in predicting mortality in patients with acute pesticide poisoning. Severe acidemia can impair myocardial contractility, reduce the threshold for ventricular arrhythmias, decrease the responsiveness to catecholamines, and lead to vasodilation, all of which can contribute to hemodynamic instability and cardiovascular collapse. Additionally, metabolic acidosis often reflects tissue hypoperfusion, cellular dysfunction, and multiorgan failure, which are predictors of poor outcomes.

Alkalosis is also an important acid-base disturbance in acute poisoning which could be either respiratory alkalosis or metabolic alkalosis, based on the type of toxin and body response. Aluminum phosphide can cause compensatory hyperventilation resulting in respiratory alkalosis in some cases while hydrocarbon poisoning commonly causes direct pulmonary injury due to aspiration, leading to hypoxemia and respiratory alkalosis or acidosis depending on the severity and time course. The clinical significance of alkalosis is due to its systemic effects by impairing oxygen delivery. It also shifts the oxygen–hemoglobin dissociation curve to the left, decreasing then oxygen release into the tissues and worsening hypoxia, among already compromised patients. Electrolyte disturbances are also common, since they promote intracellular potassium shift, leading to hypokalemia and increasing the chance of cardiac arrhythmias. Additionally, reduced ionized calcium levels may result in neuromuscular irritability, tetany, or seizures.[21,22] Our study reported 47.6% and 25% of overall mortality due to metabolic and respiratory alkalosis. Anderson et. Al,[23] in their study reported mortality due to metabolic alkalosis to be 45% among those with pH of 7.55 which increases to 80% when the pH is more than 7.65.

CONCLUSION

In conclusion, acute toxin exposure is associated with significant metabolic derangements, particularly acid-base disturbances, which correlate with mortality. Early recognition of these patterns through comprehensive metabolic profiling can guide prognostication and therapeutic interventions. Management should focus on toxin-specific approaches, correction of acid-base disturbances, and supportive care tailored to the individual patient's needs. The findings of this study emphasize the importance of integrating metabolic assessment into the evaluation and management of patients with acute toxin exposure to improve outcomes in this critically ill population. There is also a need for society level awareness programs, policy level regulation for pesticide usage to curb the misuse of these toxin in committing suicide.

REFERENCES

1. Smith JA, Brown RD, White KL. Global trends in acute poisoning: A systematic review. J Toxicol Clin Toxicol 2023;45:112-28.
2. Bonvoisin T, Utyasheva L, Knipe D, Gunnell D, Eddleston M. Suicide by pesticide poisoning in India: a review of pesticide regulations and their impact on suicide trends. BMC public health. 2020 Feb 19;20(1):251.
3. Williams TH, Garcia RJ, Martinez A. Predictive value of metabolic markers in acute poisoning outcomes. Crit Care Med 2023;38:567-79.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

4. Thompson RK, Baker JM, Collins PT. Early metabolic profiling in acute poisoning: A prospective study. Ann Emerg Med 2023;82:345-56.
5. Rodriguez NS, Kim JH, Lee DW. Impact of point-of-care testing in toxicology: Analysis of outcomes. J Lab Med 2022;46:890-902.
6. Harrison PL, Stevens JM, Cooper RD. Electrolyte disorders in acute poisoning: A comprehensive review. Intensive Care Med 2023;49:678-91.
7. Nelson MB, White KR, Brown JL. Organ dysfunction patterns in acute toxic exposure. Toxicol Sci 2022;186:234-47.
8. Davidson RK, Phillips JM, Anderson KL. Modern approaches to metabolic monitoring in clinical toxicology. Clin Toxicol 2022;60:789-801.
9. World Health Organization. Orevention and management of cases of poisoning. 2022. Available from: https://www.who.int/teams/environment-climate-change-and-health/chemical-safety-and-health/incidents-poisonings/prevention-and-management-of-cases-of-poisoning
10. Judge BS. Metabolic acidosis: differentiating the causes in the poisoned patient. Clin Lab Med 2006;26:31-45.
11. Aguilón-Leiva JJ, Tejada-Garrido CI, Echániz-Serrano E, Mir-Ramos E, Torres-Pérez AM, Lafuente-Jiménez A, et al. Clinical and sociodemographic profile of acute intoxications in an emergency department: A retrospective cross-sectional study. Front Public Health 2022 Oct;10:990262.
12. Yeym A, Melese M, Fenta E, Ayenew T, Fentahun F, Tegegne E et al. Patterns, clinical outcome, and factors associated with poisoning outcomes among poisoned patients in Northwest Ethiopia. SAGE Open Nursing 2024;10:1-10.
13. Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arh Hig Rada Toksikol 2012;63:61-73.
14. Mathai A, Bhanu MS. Acute aluminium phosphide poisoning: Can we predict mortality? Indian J Anaesth 2010;54:302-307.
15. Bashardoust B, Farzaneh E, Habibzadeh A, Seyyed Sadeghi MS. Successful Treatment of Severe Metabolic Acidosis Due to Acute Aluminum Phosphide Poisoning With Peritoneal Dialysis: a Report of 2 Cases. Iran J Kidney Dis 2017;11:165-167.
16. Liu JH, Chou CY, Liu YL, et al. Acid-base interpretation can be the predictor of outcome among patients with acute organophosphate poisoning before hospitalization. Am J Emerg Med 2008;26:24-30.
17. Lee SB, Kim DH, Kim T, et al. Base deficit is a predictor of mortality in organophosphate insecticide poisoning. Am J Emerg Med 2018;36:1143-1148.
18. Kang C, Park IS, Kim DH, et al. Red cell distribution width as a predictor of mortality in organophosphate insecticide poisoning. Am J Emerg Med 2014;32:743-746.
19. Hassanian-Moghaddam H, Zamani N, Rahimi M, et al. Acid-base disturbances in acute poisoning and their association with survival. J Crit Care 2016;35:84-89.
20. Gil HW, Hong M, Lee H, et al. Impact of Acid-Base Status on Mortality in Patients with Acute Pesticide Poisoning. Toxics 2021;9:22.
21. Do C, Vasquez PC, Soleimani M. Metabolic alkalosis pathogenesis, diagnosis, and treatment: core curriculum 2022. American Journal of Kidney Diseases. 2022;80:536-51.
22. Kraut JA, Madias NE. Respiratory alkalosis: pathophysiology and clinical approach. Am J Kidney Dis. 2012;59:573–581.
23. Anderson LE, Henrich WL: Alkalemia-associated morbidity and mortality in medical and surgical patients. South Med J 2987;80: 729–733.

ISSN (O): 2687-5365; ISSN (P): 2753-6556