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ORIGINAL RESEARCH

Clinico-Epidemiological Prediction of Adverse Outcomes in Acute Pediatric Poisoning: A Risk Prediction Nomogram Approach

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Purpose: Globally, acute pediatric intoxication is a serious health concern with a significant burden. Differentiation between pharmaceutical and non-pharmaceutical poisoning is crucial for promoting early diagnosis and implementing effective preventive strategies.

Patients and Methods: This three-year retrospective cohort study investigated 1328 exposed children, aiming to develop risk prediction nomograms to identify patients in need of pediatric intensive care unit (PICU) admission and those at risk of mortality.

Results: With a mean age of 8.21±6.64 years, a mortality rate of 1.7% and a PICU admission rate of 1.3%, more than 99% of infants and preschool children were exposed unintentionally, and intentional exposure was observed in about 88% of adolescents ($p<0.001$). Aluminum phosphide (AlP) was a leading cause of mortality and PICU admission. Non-pharmaceutical poisoning was associated with more severe clinical presentations and was exclusively linked to mortality. A predictive model for mortality with an overall accuracy of 99% underscores the role of receiving prehospital treatment in increasing the likelihood of mortality. Exposure to AlP contributed to PICU admission with a notably high odds ratio (50.596). Significant predictors of PICU need were rapid admission and leucocytosis. A model predicting PICU admissions, with a Nagelkerke pseudo-R^2 of 0.710, encompassed mutual factors contributing to mortality and PICU need, including age, sex, and blood pressure.

Conclusion: The obtained findings highlight critical differences in poisoning characteristics and outcomes across pediatric age groups and exposure types, emphasizing a need to implement preventive strategies through proper family education, increased social awareness, and the provision of psychological support for at-risk individuals.

Keywords: pharmaceutical, non-pharmaceutical, poisoning, pediatric, mortality: pediatric intensive care unit

Introduction

Globally, acute pediatric intoxication is still a serious health concern with a significant burden on emergencies.[1] In 2019, the Institute for Health Metrics and Evaluation observed 2.2 million poisoning episodes among pediatric age groups.[2] Annually, the World Health Organization (WHO) estimates 45,000 fatalities from acute children and adolescent poisoning.[3] Accordingly, acute poisoning is one of the five chief mortality causes in pediatrics.[4] While global data highlight the significant burden of pediatric poisoning, epidemiological profiles differ from country to country.[5] Precise data about the problem of pediatric poisoning in Egypt are scarce and contradictory. While some studies reported no fatalities[6] or very low mortality rate (0.3%),[5] others described higher case fatality rates reaching approximately 6%.[7] This discrepancy hinders estimating the size of the problem and impedes the development of targeted preventive strategies and evidence-based clinical guidelines.

Various studies have documented that all pediatric age groups are highly vulnerable to poisoning, showing bimodal peak age distribution.[8] Substantially, children below five years of age have behavioral tendencies to explore the

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surrounding environment orally, being more susceptible to accidental ingestion.[9] On the other hand, the various psychological stresses and emotional dysregulations in adolescence increase the probability of intentional poisoning.[10] A broad spectrum of pharmaceutical and non-pharmaceutical substances is involved in acute pediatric intoxications.[11] Regarding pharmaceutical exposures, specific drug classes such as analgesics (paracetamol) and psychotropic medications are frequently implicated in pediatric poisoning, often leading to significant morbidity and mortality.[12] Reports from the United States and United Kingdom mentioned that paracetamol poisoning was the leading cause of hepatic failure in pediatrics, accounting for 14% of all cases.[13] Poisoning with psychotropics and antidepressants was associated with cardiotoxicity and higher fatalities than other pharmaceuticals.[12] On the other hand, non-pharmaceutical substances showed diverse outcomes, ranging from complete cure to severe morbidities. Besides the caustic agents, pesticides and heavy metals were the most common agents associated with a significant need for hospitalization and intensive care in poisoned pediatrics.[3]

The effect of pediatric poisoning extends beyond acute morbidity and mortality, often leading to significant long-term structural, neurological, developmental, and psychosocial consequences for affected children and their families. Substantially, delayed neurologic sequelae and behavioural changes are recorded as long-term complications following acute pediatric poisoning with organophosphorus and carbon monoxide.[14] Toxic types of mushrooms can cause fulminant hepatic failure.[15] It was reported that about 5% of children exposed to caustic agents suffered from esophageal damage, other organ damage, and a long-term disability.[15,16] Adolescents diagnosed with self-poisoning were at higher risk of future death from accidents.[17] Additionally, the impact of pediatric poisoning-related exposure not only has devastating outcomes but also bears an economic burden on healthcare systems.[4] These enduring sequelae underscore the critical need for effective prevention and early intervention strategies.

Beyond the causative agent, the epidemiology of pediatric poisoning is profoundly influenced by several factors. Socioeconomic disparities and living in rural areas are some of these factors.[18] The parental education, proximity to medical facilities, and varying access to healthcare, as well as cultural practices, can dictate harmful practices, delay in presentation, and ultimately, worsen patient outcomes.[19] Additionally, the trends of acute pediatric poisoning vary according to geographical distribution, social concepts, and substance availability.[20]

Since pediatric poisoning has a global nature variation, the preventive policies should be regionally specific, including drug storage regulations and packaging, as well as education programs targeting caregivers and adolescents.[11,21] Furthermore, the impact of effective preventive strategies and educational programming for alleviating pediatric poisoning incidence is still limited.[2] Unsafe indoor storage of medications and household substances, which increase the likelihood of unintentional childhood poisoning.[22] Alternatively, easy access to highly toxic pesticides and prescriptions accounts for adolescent intentional exposures.[23] Although cosmetics, alcohol, and illicit substances are the most prevalent poisoning in developed countries, intoxications with pesticides, hydrocarbons, and traditional medications are usually observed in developing countries.[24] Accordingly, updating the epidemiological data in each locality facilitates the design of suitable protocols, optimizes resource utilization and prevention approaches, and raises public awareness.[25]

Previous studies considered age as one of the factors affecting the poisoning severity, where pediatric patients are more vulnerable to severe outcomes.[7] While various risk stratification tools exist in adult toxicology, objective predictive models specifically tailored for pediatric poisoning outcomes remain scarce, with no objective predictive models that assess several pediatric poisoning outcomes. Existing approaches often rely on subjective clinical judgment or simplified scoring systems that may not fully capture the complex interplay of factors influencing outcomes in children.[7] A risk prediction nomogram is a statistical regression model integrating significant determinants for potential outcomes.[26] Nomograms have been developed in various toxicological settings, either in distinct substances or poisoning categories, tailoring risk estimations upon hospital admissions.[27–30] Nomograms, as visual and intuitive predictive models, are particularly well-suited for poisoning settings due to their ability to integrate multiple significant determinants into a user-friendly tool, offering individualized risk estimations that can aid clinicians in rapid decision-making.[26] However, embedding this tool in the assessment of pediatric poisoning has not yet been utilized.

Given the unique characteristics of pharmaceutical and non-pharmaceutical classes regarding pattern, course, and implications, differentiation between both categories is crucial for promoting early diagnosis and implementing effective therapeutic strategies.[24] However, the emerging course and outcome prediction in the pediatric age are often challenging

due to the disclosure inability and delayed admissions, together with immature body systems potentiating unfavorable outcomes, especially with highly toxic substances.[2] Timely and accurate identification of pediatric poisoning patients requiring pediatric intensive care unit (PICU) admission is paramount. Early recognition and transfer to specialized care can significantly reduce the risk of severe morbidity and mortality by enabling prompt advanced life support, specific antidotal therapies, and close monitoring.[31]

Though various public health campaigns and educational initiatives aimed at reducing pediatric poisoning, there is a gap in the literature evaluating the effectiveness of these interventions, particularly in diverse socioeconomic and cultural contexts, particularly in resource-limited settings.[32] Therefore, our study aims to perform a comparative analysis between pharmaceutical and non-pharmaceutical pediatric poisoning, focusing on disparities in patterns and outcomes, and to develop a risk prediction nomogram to identify patients in need for PICU admission and those at risk of mortality based on integrating clinical, epidemiological, and laboratory parameters.

Materials and Methods

Study Design and Setting

This contemporary 3-year retrospective cohort study included all pediatric patients admitted to Tanta University Poison Control Centre (TUPCC) with acute poisoning with either pharmaceutical or non-pharmaceutical substances from January 2021 to December 2023. The retrospective nature of this study was designed to ensure a high volume of hospital-based data ready for all analyses. Tanta University Poison Control Centre is a tertiary-care center in the core of Nile delta, Egypt, offering supportive medical care to acutely poisoned patients, serving principally El-Gharbia residents, as well as the nearby governorates.

Sampling and Sample Size Calculation

In this study, we deployed convenience sampling technique where all patients fulfilled the inclusion criteria were included. The sample size was calculated for the derivation cohort using the R Statistical language (version 4.5.0),[33] and the "pmsampsize package" (version 1.1.3).[34] Based on the results of a previous study, we assumed an incidence of 2.84% for intensive care unit admission and a mortality rate of 2.13%.[24] We also assumed a 0.05 acceptable difference in apparent and adjusted R-squared, a 0.05 margin of error in estimation of intercept, a c-statistic of 0.9 at least, and the potential use of eight model parameters. A c-statistic of 0.9 was assumed to represent a strong but hypothetical model performance for sample size estimation purposes. This value was selected arbitrarily due to the absence of prior validated prediction models for pediatric non-pharmaceutical poisoning and does not reflect an expected area under the curve (AUC) of the final model. The minimum sample size required for new model development based on these assumptions for mortality was 1121, with 24 events and an event per predictor parameter (EPP) = 2.99. As for PICU admission, the minimal sample size was 860 (with 25 events, EPP = 3.05). The decision was to adopt the higher sample size (n = 1121) as the minimal number to include.

Inclusion and Exclusion Criteria

This study involved all pediatric patients of either sexes aged 18 years or younger presented with acute pharmaceutical or non-pharmaceutical poisoning admitted to TUPCC over three years (2021–2023). The documented diagnosis in hospital files was principally based on the medical interviews of parents/guardians, documented verified container/substance identification, distinctive clinical findings, and the addressable toxicological and laboratory investigations and other radiological or electrocardiographic (ECG) workup. As Figure 1 demonstrates, pediatric patients were excluded from this study if they had co-ingested substances, combined poisoning with trauma, or chronic pre-existing disorders such as liver, kidney, or heart disease. Additionally, other exclusion criteria involved cases with doubtful unclear diagnosis of toxic exposure, those transferred to another health facility, deaths on arrival, and those with incomplete health records.

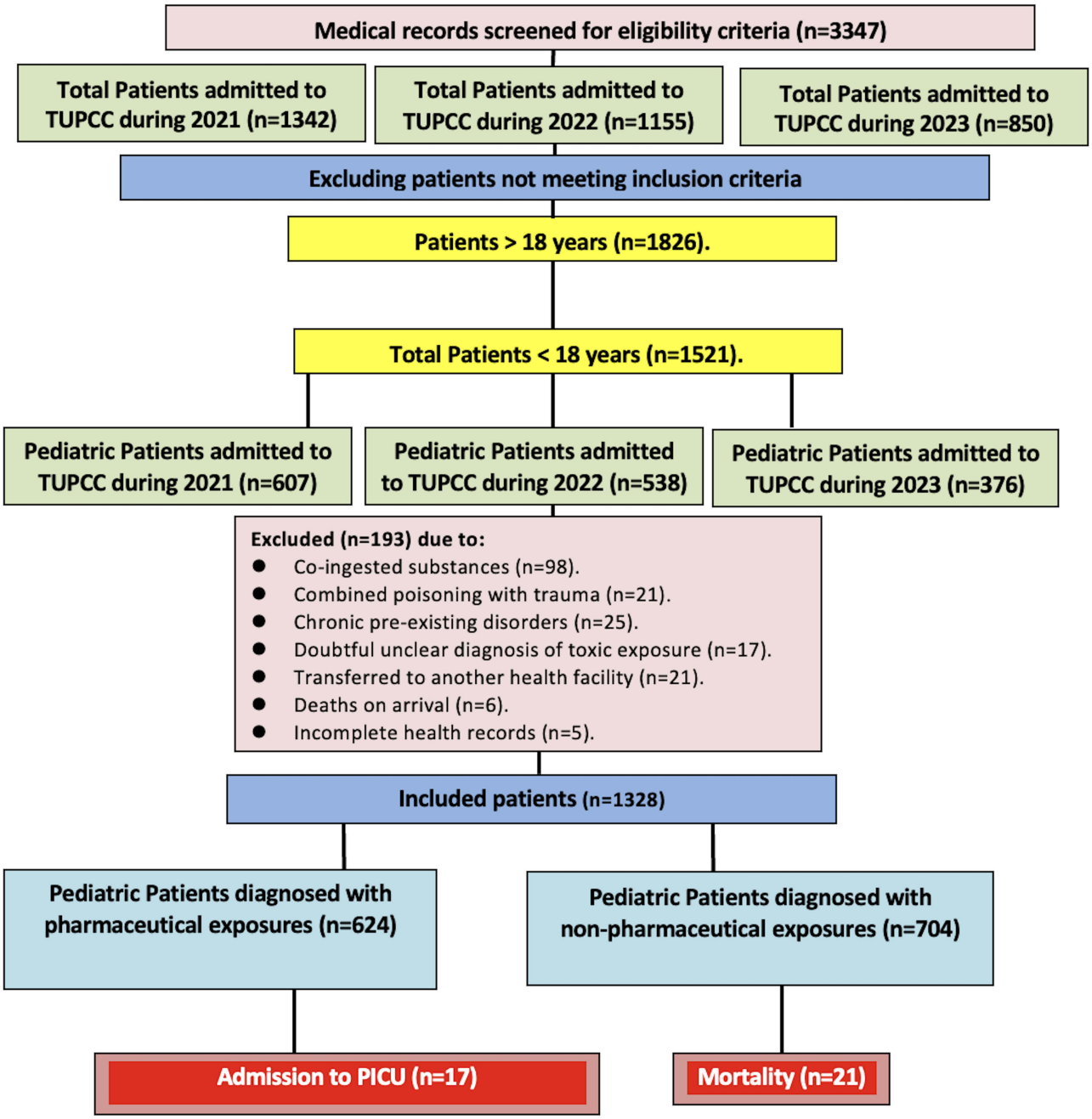

Figure 1 Flow chart of included and excluded patients showing recruitment of the pediatric patients poisoned with pharmaceuticals and non-pharmaceuticals during the study period.

Ethical Consideration

This study was approved by the Research Ethics Committee of the Faculty of Medicine, Tanta University (Approval number: 36264PR975/12/24) according to the Helsinki Declaration principles. To maintain data security and confidentiality, the patients' data were de-identified by making a password code for each patient. To ensure data security and confidentiality, all patient data were de-identified, where direct personal identifiers were removed and replaced with unique, non-identifiable study codes. The key linking the codes to patient identifiers was stored separately on a secure, encrypted server accessible only to the principal investigator. All electronic data was stored on password-protected computers with restricted access. Physical records were stored in a locked cabinet in a secure location. Since this study

was observational and retrospective, involving only the retrieval of de-identified data from archived medical records, the research ethics committee waived obtaining informed consent from the patient's parents or guardians.

Data Collection Tool

Demographics and Toxicological Data

Two clinical toxicologists involved in data collection were trained to carefully evaluate the reliability of the data and to exclude any records with inconsistencies or uncertainties. Data abstractors were supervised by a senior researcher. Data were abstracted from the archived medical records using a standardized data collection form designed specifically for this study. The forms included clear definitions for each variable and specific instructions for data entry. The used forms included predefined fields for all clinic-epidemiological, sociodemographic, toxicological, clinical, and laboratory variables to ensure consistency and minimize the risk of data extraction bias.

The obtained sociodemographic details included the age, sex, and residence. The pediatric patients were classified into four age groups: infant (less than 1 year), preschool (1 to less than 6 years), school-age (6 to less than 13 years), and adolescents (13 to less than 18 years).[8] The obtained toxicological data included any history of addiction, manner of poisoning (unintentional and intentional). Unintentional poisoning included accidental consumption of a substance without adult supervision or by mistake, like giving a substance due to a parent's or guardian's mistake. Additionally, intentional ingestion of a substance is suicidal attempt that aimed at ending one's life. Furthermore, we recorded the exposure route (dermal, inhalation, injection or oral) and poisoning type (pharmaceutical/non-pharmaceutical), including the broad category and distinct agent names. Other toxicological data included the place of exposure (home or outside), the time from exposure until hospital admission in hours, and the received prehospital therapy.[1]

Initial Clinical Examination and Investigation Data

The initial vital signs of all patients were recorded, including heart rate, systolic blood pressure (SBP) and diastolic blood pressure (DBP), respiratory rate, axillary temperature in Celsius, and Oxygen saturation percentage. The consciousness was assessed using the conventional Glasgow Coma Scale (GCS) (3–15), while the pediatric GCS was used for those aged less than 5 years.[35] Also, the Poisoning Severity Score (PSS), according to Persson et al was used to evaluate the severity of poisoning: grade 0 indicates no symptoms or signs; grade 1 (minor) refers to self-limiting symptoms; grade 2 (moderate) involves marked symptoms; and grade 3 (severe) reflects symptoms that may endanger one's life.[36] Additionally, pupil size was assessed if normal, constricted, dilated, or pinpoint.

Results of baseline laboratory investigations were documented, including the White blood cells (WBCs) count, arterial blood gases analysis (pH, $PaCO_2$, and HCO_3), serum electrolytes (sodium, potassium), random blood sugar (RBS), liver transaminases [Serum Glutamic Pyruvic Transaminase (SGPT) and Serum Glutamic-Oxaloacetic Transaminase (SGOT)], and kidney function tests, including serum creatinine and urea. Electrocardiographic abnormalities were also recorded (yes or no abnormalities).

Therapeutic Regimens

The treatment of acute pharmaceutical and non-pharmaceutical poisoning in children was performed in conformity with the TUPCC protocol and was supervised by a skilled toxicologist together with pediatric consultations. All acutely poisoned pediatric patients received emergency and supportive treatment. Gastric lavage was allowed for eligible patients presented within 1–2-hour post-ingestion. Furthermore, specifically accessible antidotes were administered. Specific antidotes were administered based on the identified toxin and clinical indications, following established institutional and international guidelines.[37]

Referral to the PICU was conducted based on the attending physician decision, where the main indications of PICU admission were unstable vital data, respiratory failure, severe hypovolemia, or persistent unconsciousness.[38] Any encountered complications were reported, including cardiogenic shock, chest infection, acid-base disturbance, electrolyte disturbance, dehydration, gastrointestinal stricture, delayed neurological sequel (DNS), skin infection/disfigurement, and oral candidiasis. The length of stay (LOS) at hospital for all enrolled patients was also documented in hours.

Grouping and Outcomes

Exposed patients were sub-classified, based on the type of their exposure, into two groups: either pharmaceutical exposed or non-pharmaceutical exposed groups (primary outcome). Pharmaceutical substances were defined according to the United States Food and Drug Administration (FDA) guidelines and the European Medicines Agency. They are agents primarily intended for therapeutic, curative, prophylactic, or diagnostic use in humans.[39,40] This category includes both prescription and over-the-counter medications that are manufactured, distributed, and regulated by the Egyptian Ministry of Health (MoH). Non-pharmaceutical substances encompassed all other agents not approved by MoH for therapeutic, prophylactic, or diagnostic use, including household products, industrial chemicals, pesticides, and pure illicit substances (alcohols). Besides, the previously mentioned four pediatric age groups were compared as regards their sociodemographic data, and type of exposure. Secondary outcomes included comparing the patients of different types of exposure regarding some adverse outcomes (mortality, PICU admission, and complications).

Data Analysis

Analyses were conducted using R Statistical language version 4.4.2.[41] Categorical variables were presented as counts and percentages. Counts below 5 were reported grouped to prevent re-identification of individual. The association between categorical variables was assessed using appropriate statistical tests. Specifically, Pearson's Chi-squared test was employed for associations between two nominal variables or when no specific trend was hypothesized. For associations involving an ordinal variable where a monotonic relationship was expected, the Chi-squared test for trend in proportions was utilized. Additionally, Fisher's exact test was used when expected cell counts were low. The distribution of continuous numerical variables was assessed using the Shapiro–Wilk test for normality and the Q-Q plots. Numerical variables following a normal distribution were presented as the mean and standard deviation (SD), and comparisons between groups were made using the Two-Sample *T*-Test. Numerical variables that did not follow a normal distribution were summarized as the median and interquartile range (IQR; 25^{th}–75^{th} percentiles), and comparisons were made using the Wilcoxon rank sum test (for two groups) or the Kruskal–Wallis rank sum test (for more than two groups).

Regarding comparisons adjusted for age, the least-squares adjusted mean difference was used. When the number of outcome events was sufficient, we conducted univariate and multivariate logistic regression analyses were conducted to assess the independent variables contributing significantly to the categorical outcomes (mortality and PICU admission) after adjusting for age and sex. The performance of these models was estimated by Nagelkerke pseudo R^2 and Hosmer-Lemeshow Goodness-of-Fit Test was used to show model fitness. We have rigorously assessed the model's performance through internal validation via bootstrapping, to assess the risk of overfitting. Receiver operating characteristic (ROC) curves, calibration curves, and a Kattan-style risk prediction nomogram were created for the models. The nomogram shows three scales: one for each predictor, a total score scale, and a probability scale. To utilize the nomogram, values for each patient were compared to the scales corresponding to each predictor to obtain scores. The total score was then determined by summing the individual scores for all predictors. Finally, the probability of the predicted outcome—such as the need for PICU admission, or mortality was assessed using the probability scale, which was aligned with the total score obtained in the previous step.[26] A p-value of 0.05 and a 95% confidence interval (CI) were chosen to interpret the results of the statistical tests.

Results

The current study was conducted with 1328 exposed patients, having a mean age of 8.21±6.64 years, comprising 21 infants, 699 preschool children, 123 school children, and 485 adolescents. Table 1 shows that the non-pharmaceutical exposure exceeded the pharmaceutical (53% versus 47%), with no significant differences among age groups. However, females constituted more than half of the studied cases and outnumbered males significantly, especially in adolescents ($p<0.001$). More than 99% of infants and preschool children were exposed unintentionally, and intentional suicidal exposure was observed in about 88% of adolescents ($p<0.001$). Oral ingestion constituted the primary route of exposure in all age groups. The need for PICU admission and mortality were significantly observed in adolescents compared to other age groups ($p<0.001$). However, prolonged hospitalization was significantly recorded in school children ($p<0.001$).

Table 1 Comparison Between Different Age Group Distribution Regarding Sociodemographic, Poisoning Data, and Investigated Outcomes

Characteristics	Age Groups				Overall N = 1,328	p-value
	Infants N = 21	Pre-school Children N = 699	School Children N = 123	Adolescents N = 485		
Sex, n (%)						<0.001*[b]
Female	9 (43%)	315 (45%)	54 (44%)	368 (76%)	746 (56%)	
Male	12 (57%)	384 (55%)	69 (56%)	117 (24%)	582 (44%)	
Residence, n (%)						0.439[b]
Rural	6 (29%)	239 (34%)	41 (33%)	175 (36%)	461 (35%)	
Urban	15 (71%)	460 (66%)	82 (67%)	310 (64%)	867 (65%)	
History of addiction, n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	7 (1.4%)	7 (0.5%)	0.009*[c]
Manner, n (%)						<0.001*[c]
Unintentional	21 (100%)	698 (99.9%)	96 (78.0%)	59 (12.2%)	874 (65.8%)	
Intentional	0 (0.0%)	<5	27 (22.0%)	427 (87.8%)	≥454 (≥34.2%)	
Route, n (%)						0.446[c]
Oral	18 (85.7%)	685 (98.0%)	110 (89.4%)	469 (96.7%)	1,282 (96.5%)	
Non-oral[#]	3 (14.3%)	14 (2.0%)	13 (10.6%)	16 (3.3%)	46 (3.5%)	
Toxic agent, n (%)						0.088[b]
Non-pharmaceutical	10 (48%)	363 (52%)	54 (44%)	277 (57%)	704 (53%)	
Pharmaceutical	11 (52%)	336 (48%)	69 (56%)	208 (43%)	624 (47%)	
PICU admission	<5	<5	<5	14 (2.9%)	≥14 (≥1.1%)	<0.001*[c]
Mortality	<5	<5	<5	20 (4.1%)	≥20 (≥1.5%)	<0.001*[c]
Length of hospital stay (hr)						<0.001*[d]
Median (IQR)[a]	6.0 (4.0–10)	8.0 (4.5 −16)	12 (6.0 −21)	10 (5.5–21)	9.0 (5.0–18)	
Range	2.0 - 77	0.5 - 288	1 – 56	0.5 - 360	0.5 - 360	
Any complication	<5	48 (6.9%)	5 (4.1%)	31 (6.4%)	≥84 (≥6.3%)	0.760[b]
Major complications	<5	18 (2.6%)	<5	17 (3.5%)	≥35 (≥2.7%)	0.295[c]
Metabolic complications	<5	18 (2.6%)	<5	14 (2.9%)	≥32 (≥2.4%)	0.894[c]
Other complications	<5	12 (1.7%)	<5	<5	≥12 (≥0.9%)	0.009*[c]

Notes: *Significance at p value <0.05; n: Number; [a]IQR: Interquartile range (25th - 75th percentiles); [b]Chi-squared Test for Trend in Proportions; [c]Fisher's exact test; [d]Wilcoxon rank sum test; [#]The "Non-oral route" category combines dermal, inhalation, and injection routes. This aggregation was performed to protect patient confidentiality by preventing the re-identification risks associated with presenting small cell counts; Major complications include cardiogenic shock and chest infection; Metabolic complications include acid-base disturbance and electrolyte disturbance; Other Complications include dehydration, gastrointestinal stricture, delayed neurological sequelae, skin infection, and oral candidiasis.

Abbreviation: PICU, Pediatric intensive care unit admission.

Although there was comparable complication incidence among age groups, dehydration, gastrointestinal strictures, delayed neurological sequelae, skin infections, and oral candidiasis were significantly higher among pre-school children (p=0.009).

The present study shows that exposure to Organophosphorus compounds (OPCs) was the most frequently reported category of poisoning, representing 12% of total admissions, followed by corrosive (9.7%), antipsychotic (8.7%), zinc phosphide (8.3%) and Aluminum phosphide (AlP) (7.8%). Supplementary 1 depicts the distribution of the exposure type according to the age group, showing that in pharmaceutical poisoning, preschool children showed significantly more frequent exposure to non-steroidal anti-inflammatory drugs (NSAIDs), antihistamines, oral anticoagulants, and Iron, in contrast to the infants, where these types of exposure were less encountered ($p<0.05$). Regarding the non-pharmaceuticals, exposure to phosphides and carbamates was considerably higher in adolescents, while preschool children showed higher presentation with corrosive and pyrethroid poisoning.

Table 2 depicts that pharmaceutical poisoning was significantly higher among females and substantially associated with urban settings and oral route ($p=0.041$, $p=0.042$, and $p=0.004$, respectively). Although non-pharmaceutical poisoned

Table 2 Comparison Between Pharmaceutical and Non-Pharmaceutical Pediatrics Poisoning Regarding Sociodemographic, Poisoning Data, and Initial Clinical Characteristics

Characteristics	Poison			p-value
	Non-pharmaceutical N = 704	Pharmaceutical N = 624	Overall N = 1,328	
Age (years)				0.758[b]
Median (IQR)[a]	5.0 (2.0–16)	4.0 (2.5–15)	4.5 (2.0–16)	
Range	0.2–18	0.2–18	0.2–18	
Sex, n (%)				0.041*[c]
Female	377 (54%)	369 (59%)	746 (56%)	
Male	327 (46%)	255 (41%)	582 (44%)	
Residence, n (%)				0.042*[c]
Rural	262 (37%)	199 (32%)	461 (35%)	
Urban	442 (63%)	425 (68%)	867 (65%)	
Place of exposure, n (%)				0.618[c]
Home	694 (99%)	613 (98%)	1,307 (98%)	
Outside	10 (1%)	11 (2%)	21 (2%)	
Route				0.004*[c]
Oral	670 (95.2%)	612 (98.1%)	1,282 (96.5%)	
Non-oral[#]	34 (4.8%)	12 (1.9%)	46 (3.5%)	
Delay time (hr)				<0.001*[b]
Median (IQR)	2.0 (1.0–4.0)	3.3 (1.0–6.0)	2.5 (1.0–5.0)	
Range	0.5 - 72	0.3 - 48	0.3 - 72	
Prehospital management, n (%)	302 (43%)	152 (24%)	454 (34%)	<0.001*[c]
Glasgow Coma Scale (GCS)				<0.001*[b]
Median (IQR)	15 (15–15)	15 (15 −15)	15 (15–15)	
Range	3.0 - 15	3.0 - 15	3.0 - 15	

(*Continued*)

Table 2 (Continued).

Characteristics	Poison			p-value
	Non-pharmaceutical N = 704	Pharmaceutical N = 624	Overall N = 1,328	
Poison Severity Score (PSS), n (%)				0.002*[c]
0	247 (35%)	268 (43%)	515 (39%)	
1	241 (34%)	197 (32%)	438 (33%)	
2	166 (24%)	138 (22%)	304 (23%)	
3	50 (7%)	21 (3%)	71 (5%)	
Heart rate (b/min)				0.747[d]
Mean ± SD	112 ± 23	113 ± 22	112 ± 23	
Range	45 - 199	56 - 201	45 - 201	
Systolic blood pressure (mmHg)				0.334[d]
Mean ± SD	108 ± 18	109 ± 16	109 ± 17	
Range	12 - 230	60 - 180	12 – 230	
Diastolic blood pressure (mmHg)				0.523[d]
Mean ± SD	65 ± 14	65 ± 13	65 ± 13	
Range	30 - 130	30 - 110	30 - 130	
Respiratory rate (cycle/min)				<0.001*[d]
Mean ± SD	23 ± 6.8	22 ± 5	23 ± 6.0	
Range	6.0 - 70	12 - 55	6.0 - 70	
Temperature (Celsius)				0.066[d]
Mean ± SD	37 ± 0.5	37 ± 0.4	37 ± 0.5	
Range	35 - 40	36 - 40	35 – 40	
O_2 saturation				<0.001*[e]
Mean ± SD	97 ± 5.5	98 ± 3.0	97 ± 4.5	
Range	30 - 100	42 - 100	30 - 100	
Pupil condition, n (%)				0.020*[c]
Normal	612 (86.9%)	522 (83.7%)	1,134 (85.4%)	
Constricted	48 (6.8%)	71 (11.4%)	119 (9.0%)	
Pinpoint pupil	11 (1.6%)	5 (0.8%)	16 (1.2%)	
Dilated	33 (4.7%)	26 (4.2%)	59 (4.4%)	

Notes: *Significance at p value <0.05; n: Number; [a]IQR: Interquartile range (25th - 75th percentiles); [b]Wilcoxon rank sum test; [c]Pearson's Chi-squared test; [d]Least-squares adjusted mean difference (adjusted for age); [e]Two Sample *t*-test; [#]The "Non-oral route" category combines dermal, Inhalation, and injection routes. This aggregation was performed to protect patient confidentiality by preventing the re-identification risks associated with presenting small cell counts.
Abbreviation: SD, Standard deviation.

cases significantly received prehospital management, a substantial delay was recorded in pharmaceutical poisoning ($p<0.001$ in each). Additionally, patients with pharmaceutical poisoning had significantly lower GCS, while non-pharmaceutical poisoning was significantly associated with severe PSS. Accordingly, patients with non-pharmaceutical poisoning had significantly lower O_2 saturation and higher respiratory rates than those with pharmaceutical poisoning, with no significant difference in other vital signs.

Table 3 conveys that patients exposed to non-pharmaceutical compounds exhibited significantly higher leucocytic count and lower levels of pH and HCO_3 than those exposed to pharmaceutical agents. Mortality was an exclusive finding linked to non-pharmaceutical exposure. Furthermore, non-pharmaceutical poisoning was significantly associated with a higher incidence of complications ($p=0.014$) and the need for PICU utilization ($p<0.001$). Supplementary 2

Table 3 Comparison Between Pharmaceutical and Non-Pharmaceutical Pediatric Poisoning Regarding Laboratory Workup Characteristics

Characteristics	Non-pharmaceutical N = 704	Pharmaceutical N = 624	Overall N = 1,328	p-value
White blood cells count (cell*10^9/L)				0.003*[b]
Mean ± SD	8.5 ± 3.5	7.9 ± 3.1	8.2 ± 3.4	
Range	1.4–36	3.4–35	1.4–36	
pH				0.003*[b]
Median (IQR) [a]	7.4 (7.4–7.4)	7.4 (7.4–7.5)	7.4 (7.4–7.5)	
Range	6.6–7.6	7.2–7.6	6.6–7.6	
$PaCO_2$ (mmHg)				0.104[b]
Mean ± SD	36 ± 7.8	37 ± 7.1	36 ± 7.5	
Range	3.7 - 81	4.3 - 76	3.7 - 81	
HCO_3 (mEq/L)				0.003*[b]
Mean ± SD	23 ± 4.4	23 ± 3.9	23 ± 4.2	
Range	1.5 - 48	7.8 - 39	1.5 - 48	
Na (mmol/L)				0.108 [b]
Mean ± SD	140 ± 4.4	140 ± 4.6	140 ± 4.5	
Range	116 - 159	115 - 159	115 - 159	
K (mmol/L)				0.874[b]
Mean ± SD	3.9 ± 0.5	3.9 ± 0.5	3.9 ± 0.5	
Range	2.1–5.9	2.1–5.9	2.1–5.9	
Random blood sugar (mg/dL)				0.110[b]
Mean ± SD	114 ± 35	111 ± 28	113 ± 32	
Range	45 - 460	12 - 300	12 – 460	
Serum Glutamic Pyruvic Transaminase (U/L)				0.622[b]
Median (IQR)	18 (15–23)	19 (15–23)	19 (15–23)	
Range	8.0 - 369	10 - 84	8.0 - 369	

(*Continued*)

Table 3 (Continued).

Characteristics	Non-pharmaceutical N = 704	Pharmaceutical N = 624	Overall N = 1,328	p-value
Serum Glutamic-Oxaloacetic Transaminase U/L)				0.434[b]
Median (IQR)	21 (18–29)	22 (18–30)	22 (18–29)	
Range	10 - 295	10 - 85	10 - 295	
Creatinine (mg/dL)				0.538[b]
Mean ± SD	0.7 ± 0.2	0.7 ± 0.2	0.7 ± 0.2	
Range	0.2–1.6	0.2–2.2	0.2–2.2	
Urea (mg/dL)				0.075 [b]
Mean ± SD	23 ± 7.8	23 ± 8.0	23 ± 7.9	
Range	2.0 - 55	0.0 - 93	0.0 - 93	
Electrocardiographic abnormality, n (%)				0.196[c]
No	637 (91%)	551 (88%)	1,188 (90%)	
Yes	67 (9%)	73 (12%)	140 (10%)	
PICU admission, n (%)	16 (2.3%)	<5	≥16 (≥1.2%)	<0.001*[c]
Mortality, n (%)	21 (3%)	<5	≥21 (≥1.6%)	<0.001*[c]
Length of hospital stay (hr)				0.052[d]
Median [IQR]	9.0 (5.3–20)	9.0 (4.5–16)	9.0 (5.0–18)	
Range	0.5 - 360	0.5 - 288	0.5 - 360	
Any complication, n (%)	56 (8.0%)	29 (4.6%)	85 (6.4%)	0.014*[e]
Major complications, n (%)	29 (4.1%)	7 (1.1%)	36 (2.7%)	<0.001*[e]
Metabolic complications, n (%)	15 (2.1%)	20 (3.2%)	35 (2.6%)	0.222[e]
Other complications, n (%)	12 (1.7%)	<5	≥12 (≥0.9%)	0.014*

Notes: *Significance at p value <0.05; n: Number; [a]IQR: Interquartile range (25th - 75th percentiles); SD: Standard deviation; [b]Least-squares adjusted mean difference (adjusted for age and sex); [c]Pearson's Chi-squared test; [d]Wilcoxon rank sum test; [e]Fisher's exact test; Major complications include cardiogenic shock and chest infection; Metabolic complications include acid-base disturbance and electrolyte disturbance; Other complications include dehydration, gastrointestinal stricture, delayed neurological sequelae, skin infection, and oral candidiasis.

Abbreviation: PICU, Pediatric intensive care unit admission.

demonstrates that AlP was the leading cause of mortality (16%) and PICU admission (15%). Additionally, the highest proportion of non-pharmaceutical complications was observed in corrosives (n = 22, 17%) and AlP (n = 14, 14%).

Potential individual predictors of mortality in patients exposed to non-pharmaceutical compounds were female sex, older age, suicidal intent, AlP exposure, receiving prehospital treatment, lower GCS and higher PSS on admission, as well as the presence of ECG abnormalities. However, hypotension (low SBP or DBP), tachypnea, hypothermia, a higher leukocytic count, lower pH, HCO_3, and $PaCO_2$ were other significant predictors of mortality, in addition to high random blood glucose and creatinine levels (Table 4). Indeed, a proposed model using multivariate forward stepwise regression analysis incorporates seven elements (age, sex, prehospital treatment, SBP, DBP, WBC count, and creatinine level) that could significantly predict mortality; it explains 62.5% of the variance in mortality among patients exposed to non-pharmaceutical compounds. The prehospital treatment was the most significant elements in this model. Visualization of

Table 4 Univariate Logistic Regression and Multivariate Forward Stepwise Regression Analyses Showing the Potential Predictors of Mortality Among Pediatrics Poisoned with Non-Pharmaceuticals

Univariate Logistic Regression Analysis			
Characteristics	**OR**	**95% Confidence Interval**	**p-value**
Age (years)	1.24	1.12–1.44	<0.001*
Sex			0.007*
Female	1.00	Reference	
Male	0.22	0.05–0.69	0.019*
Residence			0.882
Rural	1.00	Reference	
Urban	0.93	0.36–2.56	0.882
Intentional manner			<0.001*
No	1.00	Reference	
Yes	33.27	6.77–601.27	<0.001*
Aluminum phosphide	55.08	15.33–352.23	<0.001*
Delay time (hr)	0.93	0.72–1.05	0.436
Prehospital treatment			0.002*
No	1.00	Reference	
Yes	4.84	1.72–17.20	0.006*
Glasgow Coma Scale (GCS)	0.80	0.70–0.94	0.010*
O_2 saturation	0.93	0.89–0.96	<0.001*
Poisoning Severity Score (PSS)	9.78	4.68–24.32	<0.001*
Electrocardiographic abnormality			
No	1.00	Reference	
Yes	8.50	3.14–22.38	<0.001*
The following variables were adjusted for age and sex			
Pulse (b/min)	0.99	0.97–1.02	0.615
Systolic blood pressure (mmHg)	0.92	0.88–0.95	<0.001*
Diastolic blood pressure (mmHg)	0.88	0.83–0.93	<0.001*
Respiratory rate (cycle/min)	1.15	1.08–1.22	<0.001*
Temperature (Celsius)	0.35	0.12–1.00	0.046*
White blood cells count (cell*10^9/L)	1.11	0.99–1.22	0.043*
pH	0.00	0.00–0.01	<0.001*
$PaCO_2$ (mmHg)	0.88	0.83–0.93	<0.001*
HCO_3 (mEq/L)	0.83	0.77–0.91	<0.001*
Na (mmol/L)	1.09	0.97–1.21	0.146

(*Continued*)

Table 4 (Continued).

K (mmol/L))	0.50	0.18–1.44	0.191
Random blood glucose (mg/dL)	1.01	1.00–1.02	0.009*
Serum Glutamic Pyruvic Transaminase (U/L)	1.00	0.98–1.01	0.793
Serum Glutamic-Oxaloacetic Transaminase (U/L)	1.00	0.97–1.02	0.830
Creatinine (mg/dL)	149.29	17.22–1,715.60	<0.001*
Urea (mg/dL)	1.02	0.96–1.08	0.619
Multivariate forward stepwise regression analysis			
Intercept	1.25	0.01–204.95	0.932
Age (years)	1.13	1.01–1.27	0.052
Sex			1.000
Female	Reference	Reference	
Male	0.37	0.07–2.03	0.250
Prehospital management			<0.001*
No	Reference	Reference	
Yes	34.28	3.26–360.67	0.003*
Systolic blood pressure (mmHg)	0.91	0.86–0.97	0.688
Diastolic blood pressure (mmHg)	0.96	0.89–1.04	1.000
White blood cells count (cell*10^9/L)	1.12	1.00–1.26	0.469
Creatinine (mg/dL)	14.31	0.54–378.43	0.804

Notes: *Significance at p value <0.05; OR: Odds Ratio; In multivariate model: Nagelkerke pseudo R^2 = 0.625; Hosmer-Lemeshow Goodness-of-Fit Test (p-value) = 0.999. Variables entered in first step: age, sex, Aluminum phosphide, prehospital treatment (yes/no), GCS, systolic and diastolic blood pressure, respiratory rate, temperature, O2 saturation, pH, HCO_3, PCO_2, White blood cells count, random blood sugar level, creatinine, ECG abnormality. Manner was not included due to high variance inflation factor.

this model was designed in the form of a user-friendly nomogram where the overall points a patient obtained indicate the probability of death. A real patient estimation mortality risk is shown in Figure 2, showing an example of estimating the risk of mortality in an admitted poisoned child. The ROC curve analysis of the proposed model demonstrated excellent performance, as indicated by an AUC (95% CI) of 0.981 (0.96–1), sensitivity of 55%, specificity of 99%, and an overall accuracy of 99% (Figure 3a). The calibration plot (Figure 3b) illustrates the agreement between predicted and observed probabilities of mortality. The *apparent* calibration curve (dotted line) demonstrates good initial model fit. After 5000 bootstrap repetitions, the *bias-corrected* line (solid) shows a slight downward deviation at higher predicted probabilities, indicating mild overestimation of mortality risk in that range. The overall agreement between predicted and observed probabilities remains excellent, as reflected by the low mean absolute error (0.005).

Regarding the PICU admission in non-pharmaceutical poisoning, Table 5 shows that exposure to AlP was a significant contributor to PICU admission, denoted by the extremely high odds ratio (102.27). Additionally, older children, suicidal exposure, Oxygen desaturation, less delay time, lower GCS, higher PSS, and the presence of ECG abnormalities were other significant predictors of PICU admission ($p<0.05$). Among the laboratory investigations, respiratory rate, WBC count, random blood glucose level, SGOT, and creatinine were significant positive predictors of PICU admission in pediatric patients exposed to non-pharmaceutical compounds. Nonetheless, SBP, DBP, pH, HCO_3, and $PaCO_2$ were significant negative predictors. However, when all factors are considered together, less delay and higher

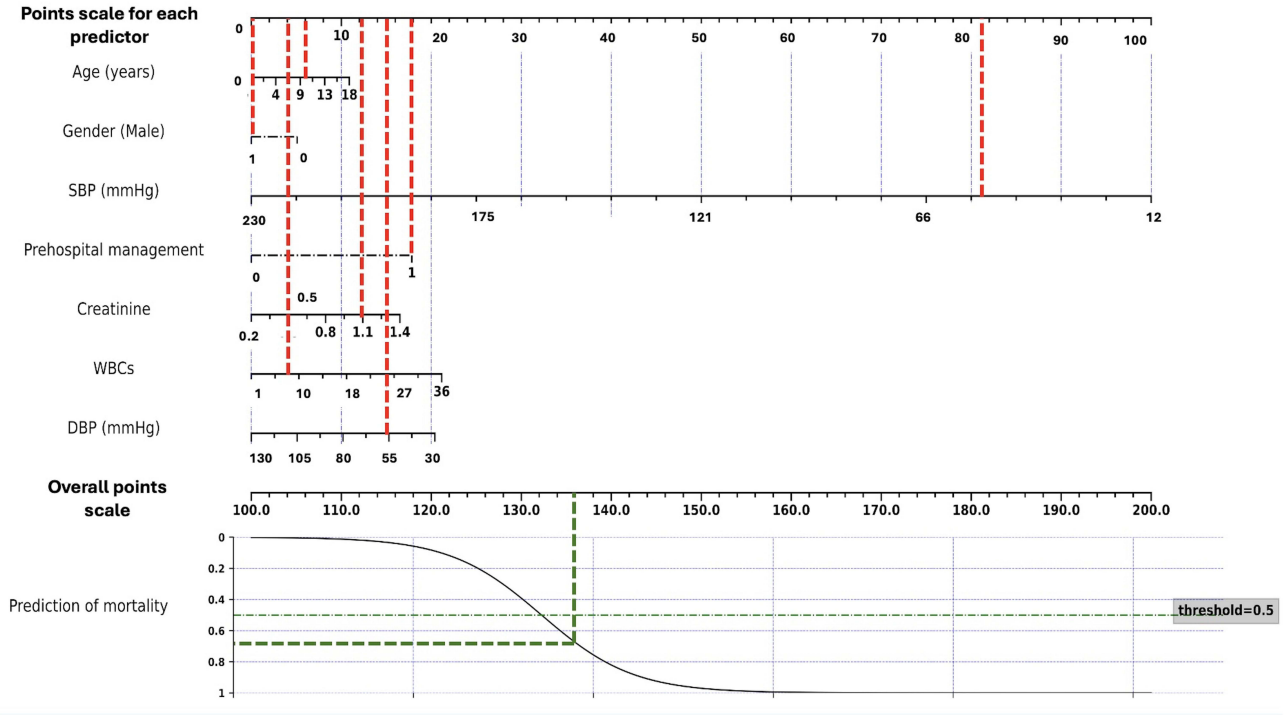

Figure 2 Nomogram for predicting the probability of mortality among pediatric patients poisoned with non-pharmaceuticals. An example of calculating the probability of mortality in a patient presented with acute exposure to a non-pharmaceutical compound is shown. In this case, the probability of mortality could be assessed, where the red dotted lines represent the real data of the patient. The child aged 10 years old corresponds to 6 points, being male gives a score of 1, which equals 0 points, systolic blood pressure of 55 mm Hg accounts of 81 points, receiving prehospital treatment accounts for 18 points, creatinine level of 1.1 increases the total points by 12, leucocytic count of 7.5 thousand collects 4 points, and a diastolic blood pressure of 55 mm Hg gave 15 points. The dotted green lines relate the overall points with the probability. Overall points= 6 + 81 + 18 + 12 + 4 + 15 = 136 and probability of death = 70%. The dotted green line indicates a threshold from the logistic regression model for the outcome of interest to take place (0.5).

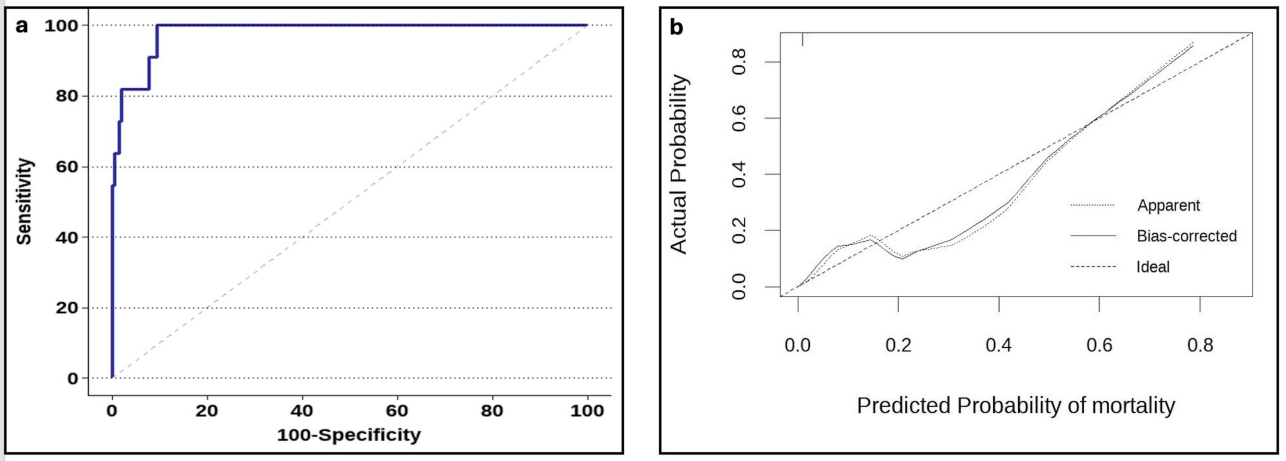

Figure 3 (**a**) Receiver operating characteristic (ROC) curve analysis of the multivariate model predicting the mortality following exposure to non-pharmaceutical agents. This model shows an AUC (95% CI) of 0.981 (0.96–1), sensitivity of 55%, specificity of 99%, and an overall accuracy of 99% (TP=6, TN=679, FP=1, FN=5). The blue line represents the proposed mortality prediction model. (**b**) Calibration curve for the predicted probability of mortality among pediatric patients exposed to non-pharmaceutical poisoning.

leukocytic counts remain significant predictors of PICU admission. Exposure to AlP and ECG abnormalities are other contributors to the proposed model, as indicated by the borderline p-value, considering the reported higher odds ratios of 50.60 and 4.78, respectively.

Table 5 Univariate Logistic Regression and Multivariate Model Backward Elimination Regression Analyses Showing the Potential Predictors of Pediatric Intensive Care Unit Admission Among Pediatrics Poisoned with Non-Pharmaceuticals

Univariate Logistic Regression Analysis			
Characteristics	**OR**	**95% Confidence Interval**	**p-value**
Age (years)	1.19	1.09–1.36	<0.001*
Sex			0.211
Female	1.00	Reference	
Male	0.52	0.16–1.44	0.225
Residence			0.293
Rural	1.00	Reference	
Urban	0.59	0.21–1.61	0.290
Intentional manner			<0.001*
No	1.00	Reference	
Yes	13.49	3.73–86.40	<0.001*
Aluminum phosphide	102.27	20.35–1,860.86	<0.001*
Delay time (hr)	0.74	0.47–0.996	0.049*
Prehospital treatment			0.563
No	1.00	Reference	
Yes	1.34	0.49–3.68	0.563
Glasgow Coma Scale (GCS)	0.80	0.70–0.95	0.015*
O2 saturation	0.94	0.90–0.97	0.002*
Poisoning Severity Score (PSS)	5.40	2.87–11.45	<0.001*
Electrocardiographic abnormality			<0.001*
No	1.00	Reference	
Yes	24.83	8.71–81.15	<0.001*
The following variables were adjusted for age and sex			
Pulse (b/min)	0.99	0.96–1.01	0.312
Systolic blood pressure (mmHg)	0.93	0.90–0.96	<0.001*
Diastolic blood pressure (mmHg)	0.89	0.84–0.94	<0.001*
Respiratory rate (cycle/min)	1.14	1.07–1.21	<0.001*
Temperature (Celsius)	0.40	0.14–1.21	0.098
White blood cells count (cell*10^9/L)	1.12	1.004–1.23	0.025*
pH	0.001	0.00–0.04	<0.001*
$PaCO_2$ (mmHg)	0.87	0.82–0.92	<0.001*
HCO_3 (mEq/L)	0.79	0.72–0.86	<0.001*

(*Continued*)

https://doi.org/10.2147/RMHP.S550232

Table 5 (Continued).

Na (mmol/L)	1.01	0.91–1.13	0.854
K (mmol/L)	0.56	0.19–1.72	0.305
Random blood glucose (mg/dL)	1.02	1.01–1.03	<0.001*
Serum Glutamic Pyruvic Transaminase (U/L)	1.01	0.99–1.02	0.133
Serum Glutamic-Oxaloacetic Transaminase (U/L)	1.01	1.00–1.03	0.038*
Creatinine (mg/dL)	56.14	6.57–543.63	<0.001*
Urea (mg/dL)	1.02	0.95–1.08	0.576
Multivariate model backward elimination regression analysis			
Intercept	0.04	0.00–8.67	0.243
Age (years)	1.07	0.88–1.30	1.000
Sex			1.000
Female	Reference	Reference	
Male	1.11	0.18–7.01	0.911
Aluminum phosphide poisoning	50.60	1.98–1,290.93	0.058
Delay time (hr)	0.28	0.10–0.74	0.005*
Diastolic blood pressure (mmHg)	0.93	0.86–1.00	0.136
White blood cells count (cell*10^9/L)	1.24	1.06 −1.46	0.045*
Electrocardiographic abnormality			0.549
No	Reference	Reference	
Yes	4.78	0.98–23.27	0.053

Notes: *Significance at p value <0.05; OR: Odds Ratio; In multivariate model, Nagelkerke pseudo R^2 = 0.710; Hosmer-Lemeshow Goodness-of-Fit Test (p-value) = 0.999; Variables entered in first step: age, sex, manner, Aluminum phosphide, delay, GCS, systolic and diastolic blood pressure, respiratory rate, temperature, O_2 saturation, pH, HCO_3, PCO_2, white blood cells count, random blood sugar level, creatinine, serum Glutamic-Oxaloacetic Transaminase, and electrocardiographic abnormality.

Figure 4 depicts the visualization of the proposed model, where we plotted a seven-element nomogram to estimate the probability of PICU admission. This nomogram comprises age, sex, AlP exposure, delay time, DBP, WBC count, and ECG abnormalities. This model exhibited high performance (R2 of 71% and good fitness with a Hosmer-Lemeshow Goodness-of-Fit Test p-value of 0.999. Receiver operating characteristic curve analysis of the multivariate model predicting PICU admission following exposure to non-pharmaceutical agents is shown in Figure 5a, showing exceptional AUC (95% CI) of 0.994 (0.99–1), sensitivity of 56%, specificity of 99%, and an overall accuracy of 99% Calibration plot of PICU admission predicting model indicates good fit with a localized but systematic overestimation in the low-risk range (approximately 0.1 to 0.3 predicted probability), where the model's predictions modestly exceeded the observed ones (Figure 5b). Regarding the internal validation of the proposed models, Supplementary 3 demonstrates that in bootstrapping based on 10000 samples, both models maintain high AUCs above 0.98 with fair bias-corrected Nagelkerke pseudo R^2 values.

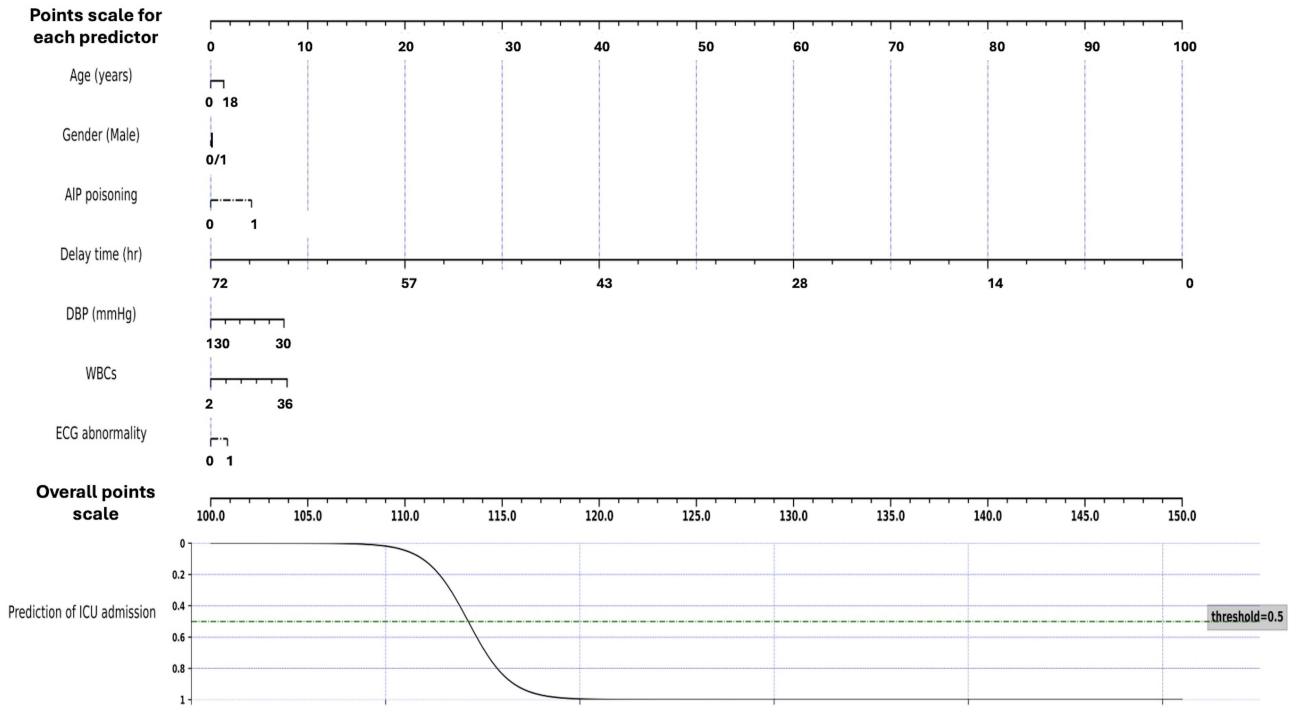

Figure 4 Nomogram for predicting the probability of pediatric intensive care unit admission among patients poisoned with non-pharmaceuticals. Males and females receive the same value for sex = zero. The dotted green line indicates a threshold from the logistic regression model for the outcome of interest to take place (0.5).

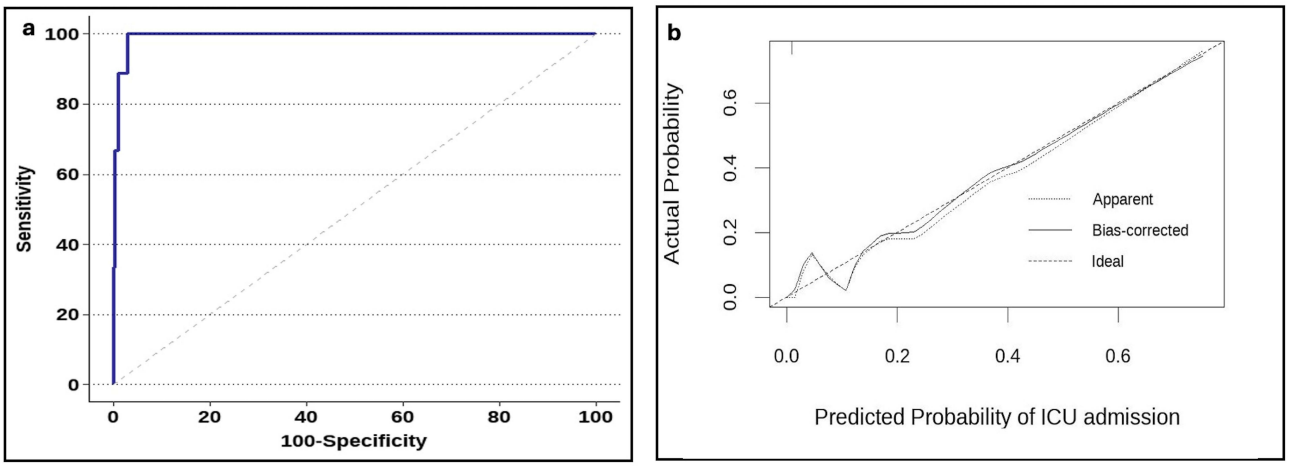

Figure 5 (**a**) Receiver operating characteristic (ROC) curve analysis of the multivariate model predicting pediatric intensive care unit (ICU) admission following exposure to non-pharmaceutical agents. This model shows an AUC (95% CI) of 0.994 (0.99–1), sensitivity of 56%, specificity of 99%, and an overall accuracy of 99% (TP=5, TN=652, FP=1, FN=4). The blue line represents the proposed ICU admission predicting model. (**b**) Calibration curve for predicted probability of pediatric intensive care unit admission among pediatric patients exposed to non-pharmaceutical poisoning.

Discussion

The present study aimed to explore the clinical and epidemiological patterns of acute poisoning with pharmaceutical and non-pharmaceutical substances in children, assessing their significant characteristic variations. This study provides a comprehensive clinico-epidemiological evaluation of pediatric poisoning, revealing distinct patterns of exposure and outcomes across different age groups. Our findings indicate that non-pharmaceutical exposures were more common than pharmaceutical ones, with accidental poisonings predominantly affecting infants and preschool children, while suicidal intent was a significant concern among adolescents. Notably, adolescents experienced higher rates of PICU admission

and mortality, with AlP emerging as a particularly hazardous agent contributing substantially to severe outcomes. Poisoning is a potentially life-threatening and frightening incident for both parents and children.[1] Recognizing the predictive factors of mortality and PICU may help decision makers to identify patients at risk.[42] We identified some significant predictors of mortality in pediatric poisoning cases including increasing age and prehospital treatment. For PICU admission, key predictors were rapid admission, elevated white blood cell count, AlP exposure, and the presence of cardiac dysrhythmias.

The observed higher poisoning with non-pharmaceuticals agrees with WHO reports, which indicate that that pesticides, fuel and lighting chemicals in developing countries are considered the primary source of poisoning for children in these countries, compared to pharmaceuticals in developed countries.[43] The current study reported a mortality rate of 1.7% and an PICU admission rate of 1.3%, which differs from what has been reported in the literature. Marano et al reported higher rates of PICU (8%) than the current study.[1] While some studies reported no mortality at all in pediatrics following toxic exposure,[1] others reported higher rates. The reported mortality rates ranged between 1% in developed countries and 3–5% in developing countries.[44] An earlier study carried out in Taiwan reported a mortality rate of 5.7%.[45] However, variations in the study contexts explain discrepancies in the reported rates of PICU, where in some developed countries, only vital signs monitoring is the main indication for the majority of PICU admissions.[1] Generally, the outcomes in pediatrics are different from adults, showing much lower fatality rates as the majority of admissions are accidental, and the ingested dose is low mainly to induce severe intoxication.[1] Nonetheless, exposure in pediatrics should be treated seriously considering the low body mass, which might result in increased toxicity and contribute to increased risk of morbidity and mortality.[43] Concurrently, the risk of mortality was higher after exposure to non-pharmaceuticals.[24]

Consistent with the obtained findings, more male patients, of relatively older ages were exposed to with non-pharmaceutical, compared to females who tend to take pharmaceuticals.[24] Miller et al attributed the higher exposure of females to pharmaceuticals to some cultural considerations that matter about the role of females as caregivers, resulting in increased exposure to pharmaceutical substances through medication management responsibility.[46] It is noteworthy to mention that the obtained findings considered that female sex was a significant predictor of mortality in non-pharmaceutical poisoning which is in line with El-Sarnagawy et al.[8] Contradicting the current study, male sex was a significant predictor of mortality in a previous study conducted in Iran. The observed discrepancy is attributed to variations in the demographics of the investigated sample, including its geographical location and age range.[24]

The obtained findings raised a serious problem: suicide intent in children. We observed that 88% of reported exposure in adolescents and 22% of reported exposure in school children were suicidal attempts. However, in non-pharmaceutical exposure, suicidal exposure was a mutual individual predictor of PICU admission and mortality. The present work demonstrated that more than half of adolescents were exposed to non-pharmaceutics, with a higher prevalence of inhalational exposure in this drug category. Usually, pharmaceutical poisoning happens intentionally using over-the-counter (OTC) medications, while non-pharmaceutical exposures occur accidentally using household products or pesticides.[47,48] These correlated findings could be explained considering previous studies where non-pharmaceutical poisoning was significantly associated with psychiatric disorders and addiction.[24] Indeed, suicide is considered the second leading cause of death in adolescents.[49] Marano et al found that intentional exposure due to suicidal attempts or inebriation purposes was associated with older age and significant hospital admission.[1] Nevertheless, an earlier study carried out in Turkey reported that more than 30% of suicides among children and adolescents were attributed to pharmaceutical poisoning.[50]

Contradicting the present study results, where increasing age contributed to mortality and PICU admission, Hamid et al reported that infants below six months had higher mortality rates compared to older children up to 15 years.[51] Others noted that about 80% of deaths in pediatrics because of poisoning occurred in children under five.[52] These studies investigated only pharmaceutical pediatric poisoning, which might justify the observed discrepancy between them and the current research. There are other social, cultural, and behavioral factors contributing to the observed exposure pattern. Aligned with the obtained findings, non-pharmaceutical self-poisoning was reported to be more common in rural areas among young individuals.[53,54]

The obtained findings revealed significant age-related variations in the type of toxicity; there is no consensus about the most prevalent type of toxic exposure. An earlier study reported that insecticide poisoning is the most prevalent type

of acute exposure in developing countries, which agrees with the current observations.[55] In contrast, previous studies reported that toxicities with alcohol and its derivatives were the most common cause of admission.[56–58] Central nervous system-related medications were the second reported pharmaceutical poisoning in a recent study conducted in Iran.[24] In partial agreement with the current study, where antipsychotics were among the frequently reported toxicities, Hadeiy et al mentioned that narcotics and psychotropic drugs were the second reported drugs after antiepileptic, sedative, and antiparkinsonian medicines.[59]

Focusing on specific age groups, the current study found that preschool children exhibited significantly higher exposure to non-pharmaceutical substances, particularly corrosives. Moreover, this age group showed a higher percentage of exposure to OTC medications, including NSAIDs, antihistamines, and Iron supplements. Following the industrial, domestic, and cosmetic products, analgesic drugs constituted the commonly reported agents in exposed pediatrics reported to the poison control call center. Nevertheless, corrosive exposure was the leading cause of ED admission. Consistent with the present study, which found that preschool children constituted more than half of the investigated patients, a recent Italian study reported that 74.1% of exposed children belonged to this age category.[1] Most admissions involving household products were from preschool children below the age of 5 years.[60] The significant involvement of this age group is attributed to the curiosity and exploratory behavior children at this age exhibit, as well as their limited hazard awareness.[61] Imitation of adult behaviors is another contributing factor, particularly in pharmaceutical poisonings.[61]

Several studies attributed the children's poisoning to the unsafe storage of medications and non-pharmaceuticals at home and the lack of paternal supervision.[62–64] Medications of bright colors and sugary coats are another factor precipitating pediatric poisoning, which warrants a need to readjust this formula and educate those tablet recipients.[65] Moreover, policymakers should enhance the regulation of OTC medication sales, organize training workshops to assess and monitor community drug prevention programs, update existing rules, and establish an effective oversight system.[24]

The current study showed that exposure to non-pharmaceuticals was significantly associated with a more severe presentation characterized by lower GCS, higher PSS, and greater deterioration in vital signs (tachypnea and desaturation). Aligned with these findings, Eizadi-Mood et al found that the level of consciousness and abnormal gastrointestinal and respiratory system manifestations characterized patients with non-pharmaceutical poisoning.[24] As the respiratory system is the primary route of absorption for some non-pharmaceutical household products, including corrosives and pesticides, these agents tend to induce direct toxic effects on the respiratory system.[66,67] Cardiovascular embarrassments were another significant feature of non-pharmaceutical poisoning, which was agreed earlier.[24] Abnormal ECG was a mutual contributor to mortality and the need for PICU. While drugs like digoxin and some antihypertension drugs induce cardiovascular toxicity,[28] non-pharmaceutical toxicity, particularly with AlP, and other pesticides are known for their associated severe cardiovascular toxicities.[68–70] The observed organ affection in non-pharmaceutical poisoning is attributed to the high prevalence of pesticides, including phosphides and OPC, which are known to induce multiple organ affection.[26,67,71] Pesticide exposure is associated with high mortality rates.[72–76]

Aluminum phosphide is a major contributor to poisoning-related fatalities in Egypt owing to its low cost and wide availability for agricultural or even household use. Clinical and epidemiological data retrieved from various Egyptian poison centers report very high case-fatality rates (often 30–100%), resulting in rising total numbers of AlP admissions in recent years. These epidemiological patterns strongly mandate that prevention must go beyond clinical care and require coordinated regulatory, supply-chain, as well as public-health actions.[77] The current observations underscore the necessity to find a solution to mitigate the increasing use of pesticides, particularly the AlP, to attempt suicide in adolescents. AlP resulted in 15% and 16% of PICU admissions and deaths, respectively in the investigated patients. Exposure to phosphide induces respiratory tract irritation and gastrointestinal manifestations. Once phosphine gas is absorbed, it causes an inevitable circulatory collapse, arrhythmias, and multiple organ failure, including hepatotoxicity and nephrotoxicity. Death typically happens within 24 hours of exposure.[68] Phosphine gas also induces metabolic acidosis and pulmonary edema.[78]

In the context of pesticide-regulatory frameworks it is advised that AlP should only be purchased and used under strict supervision and licensing from the Ministry of Agriculture as well as traceable sales records to mitigate its widespread availability. Regulations should be settled to ban its open sale, while authorities work together to identify

newer and safer alternatives. In addition, community education programs and campaigns, especially farmers, as well as the power of social media, should raise public awareness about proper handling and storage.[79,80]

In accordance with our study, El-Sarnagawy et al highlighted the highest discriminatory power of leucocytosis in predicting all adverse sequelae in pediatric age groups.[8] Likewise, Sharif and colleagues found that leucocytosis was a significant predictor of PICU admission and subsequent mortality in non-pharmaceutical poisoning.[81] The latter study was conducted among AlP-intoxicated adults, and the observed leucocytosis was attributed to oxidative stress-inducing inflammatory responses, as proven by the dramatic response to antioxidant therapy described in other studies.[82,83] This classical presentation justifies the observed association between hemodynamic instability, metabolic disturbances, a surge in creatinine levels, and worsened outcomes, given that 30.2% of non-pharmaceutical exposure in the present study was due to AlP.

Regarding pesticides, OPC constituted the most common type of exposure in the present study (12%). Easy accessibility made OPC a common tool to attempt suicide in adolescents. There is an urgent need to establish a suicide awareness program in schools where students should be oriented with the risks of toxic exposure. Furthermore, public efforts should be integrated to restrict access to pesticides, particularly phosphides and OPC. School employees should be trained in reporting suicide incidents and implementing crisis response plans and teams in school.[24]

The significant association between prehospital management and the higher risk of mortality is noteworthy. Some caregivers attempt harmful first-aid interventions at home before accessing emergency services.[64] A recent study showed that the time interval from poisoning with non-pharmaceutical to death was shorter, indicating higher severity than in pharmaceutical poisoning.[24] In their systematic review, Mottla et al concluded that significant delay after toxic exposure to pharmaceuticals is associated with higher mortality in pediatrics, especially in developing countries.[2] One precipitating factor of delay is the inability or fear to disclose exposure in children.[43] Another contributing factor to the delay and worsened outcomes (mortality) is the lack of healthcare facilities and geographic barriers to approaching emergency services.[84] Inadequate caregiver knowledge about the toxicity and hazards of exposure is another undeniable factor.[64]

The paradoxical finding that prehospital management was associated with higher mortality may reflect confounding by severity, as more critically ill patients are likely to receive early intervention before hospital arrival. The observed association may indicate inappropriate initial management due to faulty caregiver knowledge or reliance on traditional remedies prior to hospital presentation, especially non-pharmaceutical poisoning.[8] Similarly, the higher hospitalization rates among older adolescents may relate to both increased exposure opportunities and a lower clinical threshold for admission in this age group. Families of school-attending children may be more readily identify poisoning signs and more likely to seek hospital care, or children are more likely to be brought to the hospital by school guardians.[85–87]

The sex differences in exposure patterns could be influenced by gender-specific roles and behaviors, as well as differential healthcare-seeking behavior or supervision. Gender-related differences in household roles, behavioral tendencies, or social expectations influence both exposure risk and care-seeking behavior. Boys and girls are different in play activities, locations of unsupervised time, or home responsibilities that alter exposure risk.[88] There may be a substantial cultural difference in seeking medical care differently for boys versus girls as regards timing and level of urgency, which may adversely affect the outcome. Also, gender norms may influence the declaration of intentional versus accidental poisoning.[89] These findings highlight the complex interplay between cultural, behavioral, and healthcare system determinants that merit further study.

The present study is privileged to include all predictors of PICU admission and mortality in a bedside nomogram, which could be used early on admission to treat the exposed children according to their risk of developing these adverse outcomes. The proposed nomograms included factors that were incorporated in previously established nomograms in other contexts.[26] Age, blood pressure, creatinine level, and leukocytic count were the most reported predictors of adverse outcomes in previous studies.[26]

While the proposed models are highly effective at correctly identifying survivors and those who will not require PICU admission, the high accuracy and AUC values might result from the low event rate, which inflates accuracy despite limited sensitivity. The small number of PICU admissions and mortality events may have contributed to model overfitting or instability, despite an apparently satisfactory Hosmer–Lemeshow test. Furthermore, these models may miss a notable

proportion of patients at risk of death or those in need of PICU admission. So, the proposed models should be interpreted carefully. We must keep in mind that these models represent internal performance only, without external validation; future studies using more balanced samples are needed to confirm their predictive utility. Future research should also focus on refining the proposed models to improve sensitivity without unduly compromising specificity, and external validation is essential to assess their performance in different clinical contexts.

Limitations and Recommendations

The absence of confirmatory measurements identifying the nature of exposure is the primary limiting factor, which is attributed to the resource-constrained nature of the study setting. Though including a large sample, the single-center nature of our study is a significant limitation of this study. The patients' characteristics, treatment protocols, and data collection practices may vary across institutions. The retrospective design, where the data integrity relies on archived records and caregiver interviews carries potential for recall bias, missing data, and misclassification of poisoning agents. Another limitation of this retrospective design was the potential inter-rater variability in clinical assessments such as the GCS and PSS. Though these scores were performed by the attending physicians on duty, they were not evaluated and could introduce a degree of measurement error.

Additionally, the adopted convenience sampling and low reported rates of some outcomes, including PICU admission and mortality might affect statistical power, introduce potential selection bias and increase the risk of overfitting in predictive models limiting their robustness. We recommend considering oversampling rare outcomes in future model development. Likewise, both developed models exhibited a relatively low sensitivity (~55%), which may fail to identify a substantial proportion of high-risk patients. Therefore, we recommend future studies exploring alternative modelling techniques or additional predictors to improve the sensitivity of the proposed models.

Indeed, some confounding factors such as socioeconomic status, parental education, and mental health history were not included in this study, which might limit the understanding of contextual risk factors, especially for poisoning with suicidal intention. Excluding poly-intoxications, patients with incomplete records and deaths on arrival might not reflect all patterns of pediatric poisonings and underestimate true severity and outcomes. Therefore, we recommend future studies using more comprehensive clinical and contextual data to allow for investigating these patterns of intoxications and multivariable adjustment and assessment of residual confounding factors. Future studies with larger and more heterogeneous populations are warranted to confirm associations of different outcomes with intent of exposure, age, or residence which could further refine the understanding of model performance. Lack of follow-up period after discharge is another limiting factor that needs to be addressed in the upcoming work.

While the predictive models demonstrated high accuracy and AUC values with good calibration, the small number of PICU admissions and mortality events may have contributed to model overfitting or instability, despite an apparently satisfactory Hosmer–Lemeshow test. The model's event per variable which is below the ideal threshold and the absence of an independent external validation cohort for our risk prediction nomogram are undeniable limitations. While internal validation methods were employed during model development to assess its robustness and predictive performance within our dataset, the generalizability of the nomogram to other populations in different geographic regions or healthcare system settings remains to be fully established. Additionally, we were unable to develop separate predictive models for mortality and PICU admission for patients intoxicated with pharmaceuticals, because the number of these outcome events was extremely limited. This prevented reliable model estimation and validation. Future research should prioritize prospective studies with diverse external validation cohorts to confirm the broader applicability and clinical utility of the proposed nomogram.

This study highlights areas for future research to investigate the underlying causes of observed differences in intoxication patterns in pediatrics. Due to the significant impact of pesticide intoxication, particularly in developing countries, it is recommended that careful and effective preventive strategies be implemented. The observed pattern of poisoning indicates that the occurrence of poisonings can be significantly lowered through proper family education, increased social awareness, and the provision of psychological support for at-risk individuals. Validation of the proposed nomograms in other toxic exposure settings would allow generalizability of the obtained findings.

Conclusion

Our study highlights distinct differences in pediatric poisoning profiles, with unintentional exposures dominating in younger children and intentional self-harm being more prevalent among adolescents. A key finding is the significantly higher morbidity and mortality associated with non-pharmaceutical agents, particularly AlP. Rapid admission and leucocytosis were significant predictors of mortality, and receiving prehospital treatment was a significant indicator of PICU need. The developed nomograms encompassed other mutual factors contributing to mortality and PICU need, such as increasing age, female sex, and low blood pressure. The developed nomograms provide a practical tool for clinicians to rapidly identify patients at high risk for mortality or the need for PICU admission. In clinical practice, these models can guide early management, optimize resource allocation, and inform crucial conversations with families. Nonetheless, these findings should be interpreted considering the acknowledged limitations.

Our results also carry significant public health implications. They underscore the urgent need for targeted preventive strategies, including enhancing public awareness about the dangers of common household agents, improving storage practices for toxic substances, and establishing robust psychological support systems for adolescents at risk of self-harm. By integrating these clinical and public health approaches, we can work towards reducing the burden of pediatric poisoning and improving outcomes for this vulnerable population.

Institutional Review Board Statement

This study was approved by the Research Ethics Committee of the Faculty of Medicine, Tanta University (Approval number: 36264PR975/12/24) according to the Helsinki Declaration principles. To maintain data security and confidentiality, the patients' data were dealt with anonymously by making a password code for each patient.

Abbreviations

AUC, Area under curve; AlP: Aluminum phosphide; CI, Confidence interval; DBP, Diastolic blood pressure; DNS, Delayed neurological sequel; EEP, Event per predictor parameter; ECG, Electrocardiographic; GCS, Glasgow Coma Scale; LOS, Length of stay; MoH, Ministry of health; NSAIDs, Non-steroidal anti-inflammatory drugs; OPC, Organophosphorus compounds; OTC, Over-the-counter; PICU, Pediatric intensive care unit; PSS, Poisoning Severity Score; RBS, Random blood sugar; ROC, Receiver operating characteristic; SBP, Systolic blood pressure; SD, Standard deviation; SGOT, Serum Glutamic-Oxaloacetic Transaminase; SGPT, Serum Glutamic Pyruvic Transaminase; TUPCC, Tanta University Poison Control Centre; WHO, World Health Organization; WBC, White blood cells.

Data Sharing Statement

The data analyzed in the current study are available upon reasonable request from the corresponding author.

Informed Consent Statement

Since this study was observational and retrospective, involving only the retrieval of anonymized data from archived medical records, the research ethics committee waived obtaining informed consent from the patient's parents or guardians.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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