ORIGINAL RESEARCH

Organophosphate Poisoning in a Paediatric Intensive Care Unit: A Retrospective Analysis Based on Ten Years of Experience

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Background: Organophosphate (OP) poisoning is one of the most common etiologies of poisoning in the pediatric age group.

Objective: This study aimed to evaluate the demographic characteristics, clinical features, clinical course, and outcomes of children with toxicity from organophosphates admitted to the pediatric intensive care unit.

Methods: A retrospective review of hospital medical records of all children aged 14 years and younger who were admitted to the PICU with a provisional diagnosis of organophosphate poisoning at King Fahad Hospital of the University (KFHU), Alkhobar, Saudi Arabia, between January 1, 2008, and December 31, 2018, was conducted. Patients with incomplete medical record information or with suspicion or evidence of one or more agents other than organophosphate were excluded from the study.

Results: Thirty-one patients were enrolled in the study. The median age of the study population was 2 years, and 19 (61%) were males. The majority of patients (68%) had more than one route of organophosphate exposure. Skin exposure was reported in 26 patients (84%). Only three patients (10%) had suicidal organophosphate exposure from organophosphates, while the majority (28 patients; 90%) had accidental poisoning. Bronchorrhea was the most prevalent presenting feature, reported in 28 patients (90%). 17 patients (55%) were treated with intravenous atropine and (45%) were used a combination of pralidoxime with atropine for treatment. Five patients (16%) developed acute respiratory distress syndrome. Twelve patients (39%) needed endotracheal intubation and mechanical ventilation secondary to respiratory failure.

Conclusion: The presenting features of organophosphate poisoning differ widely in children. Risk factors for mortality for PICU patients with organophosphate poisoning include delayed hospital arrival by more than 1 hour, inhalational route of exposure, need for mechanical ventilation, and high lactate levels in the first 24 hours post-exposure.

Keywords: organophosphate, poisoning, toxicology, pediatric, intensive care

Introduction

Pediatrics poisoning is a major public health issue that causes significant morbidity and mortality (number of deaths during the period of the study) worldwide.[1,2] In Saudi Arabia, a retrospective study including 1013 children admitted to hospital showed that 49 patients (4.8%) were admitted to the pediatric intensive care unit (PICU) and 3 patients (0.3%) died after presenting with acute poisoning. Organophosphate(OP) poisoning constitutes 1.68–2.9% of acute poisoning in pediatric patients in Saudi Arabia.[3,4] However, organophosphates were the most common cause of poisoning in children 6 years and older and were found to be the fourth agent involved in admission to PICU.[5] Furthermore, Among a cohort study conducted by Alruwaili et al, 2019, mortality in children presenting with acute poisoning were related to organophosphate exposure due to abuse at home.[5] Organophosphates act as irreversible cholinesterase inhibitors because the organophosphate-cholinesterase bond is not spontaneously reversible without pharmacological intervention. This inhibition leads to accumulation of acetylcholine and elevations in synaptic acetylcholine levels, which in turn leads to excessive stimulation of both

muscarinic and nicotinic acetylcholine receptors, leading to the cholinergic toxidrome.[6,7] Additional pathways, including oxidative stress, lead to overstimulation of cholinergic and glutamatergic nervous systems, which is followed by generation of reactive species and oxidative damage in many tissues. The presenting clinical features, which include lacrimation, bronchospasm, bronchorrhea, diarrhea, abdominal pain, vomiting, arrhythmia, fasciculation, and respiratory failure, are the results of enhanced muscarinic and nicotinic overstimulation.[8–10] Anxiety, restlessness, convulsion, miosis, insomnia, coma, and Cheyne-Stokes breathing are all neurological manifestations.[11] Atropine, an anticholinergic drug, remains the mainstay of treatment worldwide. Pralidoxime and other drugs in the oxime class reactivate acetylcholinesterase inhibition by organophosphorus.[10] Despite the beneficial effects of pralidoxime first noted with parathion poisoning, its effectiveness in organophosphate poisoning has been much debated.[12] Assessing of the mortality related to organophosphate poisoning and the lag time, amount of organophosphate consumed, clinical severity, pseudocholinesterase levels, acute renal failure, and duration of ventilator support may addressed the organophosphate Poisoning. Therefore, the aim of this study was to evaluate the demographic characteristics, clinical features, clinical course, and outcomes of children with toxicity from organophosphates admitted to the pediatric intensive care unit at a teaching hospital based on ten years of experience in Khobar, Eastern Province of Saudi Arabia.

Methods

This retrospective study aimed to evaluate the demographic characteristics, clinical features, clinical course, and outcomes of children with organophosphate poisoning admitted to the pediatric intensive care unit (PICU) at a tertiary teaching hospital. King Fahad Hospital medical records of all children aged 14 years and younger who were admitted to the PICU with a provider diagnosis of organophosphate poisoning at King Fahad Hospital of the University (KFHU), Alkhobar, Saudi Arabia, between January 1, 2008 and December 31, 2018 were screened for eligibility. The Institutional Review Board (IRB) of Imam Abdulrahman Bin Faisal University approved this study (IRB-2022-01-140). The IRB waived the need to obtain informed consent because of the retrospective nature of the study. Furthermore, data confidentiality was ensured following the Declaration of Helsinki principles. Inclusion criteria included a history of organophosphate exposure 24 hours before hospital presentation, patients with clinical characteristics of organophosphate poisoning, and clinical improvement after treatment using atropine and/or oximes. Patients with incomplete medical record information or with suspicion or evidence of one or more poisoning agents other than organophosphate were excluded from the study. Our PICU during the study period comprised a total of 8–12 beds, and the PICU receives a mean of 711 admissions annually. A predetermined set of variables was adopted. The extracted data included demographics (eg, age, sex, time of hospital arrival), basic information related to circumstances of poisoning (eg, reason of exposure and route of exposure [ingestion, inhalation, skin contact]), clinical features, vital signs, mental status, duration of hospital and PICU length of stay (LOS), therapeutic options, and clinical outcomes (eg, complications, ventilatory support, and mortality). The Glasgow Coma Scale (GCS) was used to assess the patient's level of consciousness. The final collected data consisted of minimal non-outcome missing variables, including ten values (32%) of missing blood pressure measurement and one value (3%) of missing respiratory rate. The assumption of missing completely at random (MCAR) was made. Little's MCAR test failed to reject the null hypothesis while demonstrating that the missing data were MCAR (P = 0.601). The missing data was estimated using the expectation-maximization (EM) algorithm and the maximum likelihood method. The descriptive statistics were performed. A Kolmogorov–Smirnov test was used to test the data normality, in which skewed data was defined as a P-value of 0.05. Metric data were shown as the median and the 25th–75th interquartile range (IQR), while categorical data were shown as frequencies and percentages. Categorical data was compared using Fisher's exact test. On the other hand, continuous data was compared using the Mann–Whitney U-test. A two-tailed p-value of 0.05 was considered statistically significant. The statistical analysis was conducted by the IBM Statistic Package for Social Sciences (SPSS) version 26 (IBM, Armonk, NY, USA).

Results

Thirty-one patients were admitted to the PICU with a history of organophosphate exposure and clinical characteristics of organophosphate and were treated with atropine and pralidoxime. Although atropine only affects muscarinic receptors,

pralidoxime should also be used to influence nicotinic receptors in organophosphate toxicity. By attaching to the organophosphate, pralidoxime reactivates phosphorylated AChE. However, it must be administered within 48 hours of the poisoning in order to be effective. The drug does not cause respiratory depression and can be used with atropine. The median age of the study population was 2 years (range, 7 months to 14 years), and 19 (61%) were males. The majority of patients (68%) had more than one route of organophosphate exposure. Skin exposure was reported in 26 patients (84%), ingestion was reported in 23 patients (74%), and inhalation was reported in 18 patients (58%). Only three patients (10%) had suicidal organophosphate poisoning, while the majority (28 patients; 90%) had accidental (ie, unintentional) poisoning. Table 1 show the demographic and baseline characteristics. Most patients (68%) presented to the hospital within the first hour after organophosphate exposure. In our cohort, bronchorrhea was the most prevalent presenting feature, reported in 28 patients (90%). Twelve patients (39%) were drowsy at hospital presentation. Figure 1 illustrates the presenting signs and symptoms. In addition, the median GCS was 14 (IQR, 9–15). Of the included patients, 17 patients (55%) were treated with intravenous atropine and 14 (45%) were used a combination of pralidoxime with atropine for treatment. Table 2 shows the subjects' clinical features, vital signs, laboratory findings, and management. Furthermore, five patients (16%) developed acute respiratory distress syndrome (ARDS), and only one patient (3%) developed a seizure secondary to organophosphate poisoning. Twelve patients (39%) needed endotracheal intubation and mechanical ventilation secondary to respiratory failure. Our patients had a median PICU stay of 2 (IQR, 1–5) days and a median hospital stay of 4 (3–9) days. The overall in-hospital mortality rate was 26% (8/31 patients). Table 3 summarizes the subjects' complications, length of stay, and in-hospital mortality. One patient aged 10 years and two patients aged 14 years were exposed to organophosphate due to suicidal intention. Compared to accidental exposure, older pediatric age was significantly associated with suicide (P = 0.005). In addition, these suicidal patients presented significantly later than patients with accidental exposure (P 0.027). Moreover, all suicidal exposures required mechanical ventilation, and the subjects died subsequently (n = 3). The need for mechanical ventilation and mortality were significantly associated with suicidal organophosphate poisoning (P = 0.049 and P = 0.012, respectively). Table 4 presents characteristics and statistical comparisons between suicidal and accidental organophosphate exposure patients. We have found that patients with inhalational exposure to organophosphate are significantly associated with the need for mechanical ventilation (P 0.001). Furthermore, those with more than one route of organophosphate exposure were significantly associated with the need for mechanical ventilation (P 0.004). Late arrival to the hospital after the exposure cut-off of 1 hour was significantly associated with the need for mechanical ventilation (P 0.001). Additionally, patients

Table 1 Demographics and Baseline Characteristics

Study Variables:	Total Population:
Age, median (IQR)	2 (1–8) years
Gender:	
Male, n (%)	19 (61%)
Female, n (%)	12 (39%)
Route of exposure:	
Skin contact, n (%)	26 (84%)
Ingestion, n (%)	23 (74%)
Inhalation, n (%)	18 (58%)
More than 1 route of exposure, n (%)	21 (68%)
Reason of exposure:	
Accidental, n (%)	28 (90%)
Suicidal, n (%)	3 (10%)
Hospital arrival time:	
30 minutes after exposure, n (%)	13 (42%)
30 minutes – 1 hour after exposure, n (%)	8 (26%)
1–3 hours after exposure, n (%)	3 (10%)
3–6 hours after exposure, n (%)	7 (23%)

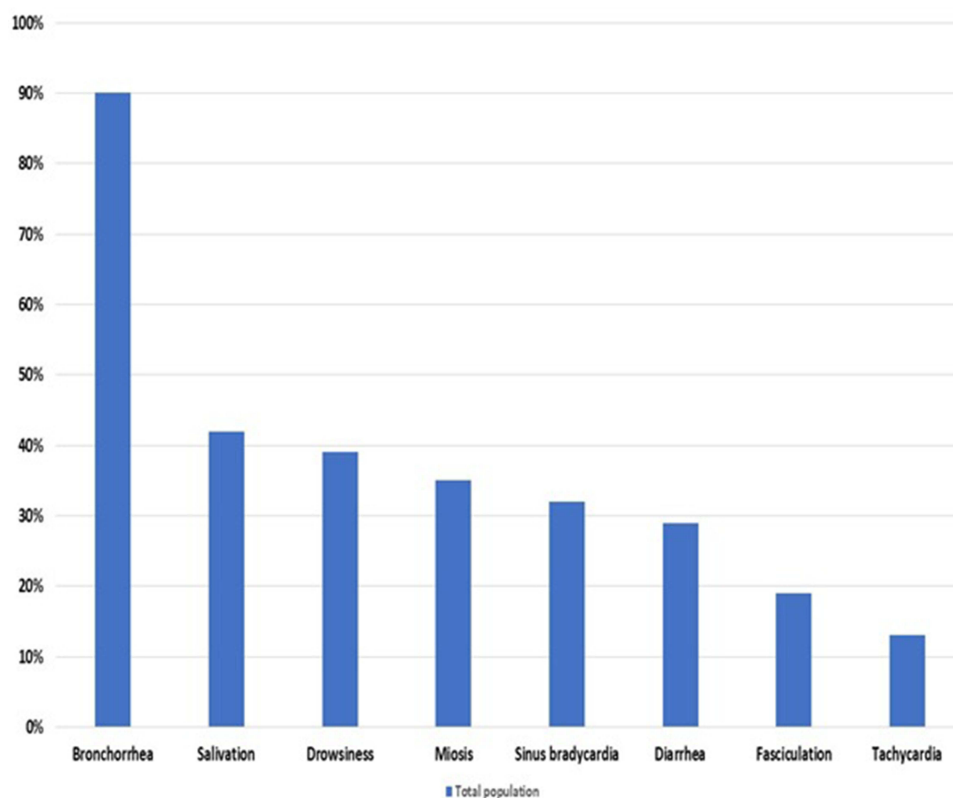**Abbreviations**: IQR, interquartile range; n, number.

Figure 1 Bar chart illustrating the presenting symptoms and signs.
Notes: The bronchorrhea was the most prevalent presenting feature, reported in 28 patients (90%). Twelve patients (39%) were drowsiness at hospital presentation.

who were on mechanical ventilation had significantly lower initial GCS (8 versus 15; P 0.001). Lactate levels at hospital arrival, after 6 hours of exposure, and after 24 hours of exposure were all significantly higher in patients who needed mechanical ventilation at some point in their PICU course (P = 0.014, P 0.001, and P 0.001, respectively). All patients who developed ARDS (n = 5) required mechanical ventilation (P 0.001). The mortality rate was 67% among those who required ventilator support (8/12 mechanically ventilated patients). Four patients (33%) were intubated successfully and discharged uneventfully. All patients who died (n = 8) were on mechanical ventilation (P 0.001). Table 5 presents the comparison between patients who required mechanical ventilation and those who did not. Specific routes of organophosphate exposure had a significant association with mortality. Organophosphate exposure via inhalation was associated with a higher mortality rate (P = 0.010). In addition, being exposed to organophosphate via more than one route of entry had a significant correlation with mortality as well (P = 0.032). Later hospital arrival of more than 1 hour after exposure correlated significantly with death (P 0.001). Multiple presenting clinical features were also significantly associated with death, including miosis (P = 0.001), salivation (P = 0.043), diarrhea (P = 0.027), fasciculation (P = 0.002), tachycardia (P = 0.043), and drowsiness (P = 0.001). Lactate levels at hospital arrival, after 6 hours of exposure, and after 24 hours of exposure were significantly higher in patients who died (P = 0.010, P 0.001, and P 0.001, respectively). All patients who died required treatment with atropine (P = 0.003). The mortality rate among patients who developed ARDS post exposure was 80% (4/5). Table 6 presents the comparison between survived and deceased subjects.

Discussion

Organophosphate intoxication is a common reason for PICU admissions around the world.[13] In Saudi Arabia, pesticides are commonly used for agricultural and environmental pest control. The popularity of its use as a pesticide has increased the incidence of accidental and suicidal poisoning in children. Most cases of organophosphate poisoning in the pediatric age group are unintentional.[3,4] Multiple routes of OP poisoning exist, including oral ingestion, inhalation, and skin

Table 2 Clinical Features, Vital Signs, Laboratory Findings, and Management

Study Variables: Clinical features:	Total Population:
Bronchorrhea, n (%)	28 (90%)
Salivation, n (%)	13 (42%)
Drowsiness, n (%)	12 (39%)
Miosis, n (%)	11 (35%)
Sinus bradycardia, n (%)	10 (32%)
Diarrhea, n (%)	9 (29%)
Fasciculation, n (%)	6 (19%)
Tachycardia, n (%)	4 (13%)
Vital signs:	
Heart rate, median (IQR)	120 (110–134) beats per minute
Respiratory rate, median (IQR)*	38 (33–45) breaths per minute
Systolic blood pressure, median (IQR)*	110 (101–111) mmHg
Diastolic blood pressure, median (IQR)*	55 (47–59) mmHg
Glasgow Coma Scale, median (IQR)	14 (9–15)
Lactate level:	
At hospital arrival, median (IQR)	1 (1–1) mmol/L
6 hours after hospital admission, median (IQR)	1 (1–3) mmol/L
24 hours after hospital admission, median (IQR)	1 (1–2) mmol/L
Treatment with Atropine, n (%)	17 (55%)

Note: *Missing details were estimated using expectation maximization.
Abbreviations: n, number; IQR, interquartile range.

Table 3 Complications, Length of Stay, and in-Hospital Mortality

Study Variables:	Total Population
ARDS, n (%)	5 (16%)
Seizure, n (%)	1 (3%)
Requirement for endotracheal intubation and mechanical ventilation, n (%)	12 (39%)
PICU length of stay, median (IQR)	2 (1–5) days
Hospital length of stay, median (IQR)	4 (3–9) days
In-hospital mortality, n (%)	8 (26%)
Causes of death	
Multiple organ failure, n (%)	3 (10%)
Tachyarrhythmia, n (%)	2 (7%)
Unknown/not documented in the patient's chart, n (%)	3 (10%)

Abbreviations: ARDS, acute respiratory distress syndrome n, number; PICU, pediatric intensive care unit; IQR, inter-quartile range.

absorption. In children, it has been reported that oral ingestion is the most common route for organophosphate toxicity.[14,15] However, most of the organophosphate intoxications in our study occurred due to accidental skin exposure and were commonly associated with other forms of exposure. A plausible explanation is that organophosphate-containing solutions are used commonly by some Saudi Arabian families as a treatment for head lice, as described by Alruwaili et al.[5] A similar finding was also described in Egypt by Abdel Baseer et al, where skin absorption and inhalation were the common routes of OP intoxication. Similarly, they also found that the second most common method of OP poisoning in their patients was via home-made lotions using organophosphate compounds for treating head lice, with the most common being direct inhalation from farm fields sprayed with pesticides.[16] Muscarinic effects were the most common clinical features in the present study, with bronchorrhea and respiratory distress being the most common

Table 4 Characteristics and Statistical Comparison Between Suicidal vs Accidental Organophosphate Exposure Patients

Study Variables	Overall Population [n=31]	Suicidal [n=3]	Accidental [n=28]	P-value
Age, range	14–0.5 years	14–10 years	14–0.5 years	0.005**ª
Hospital arrival time:				
30 minutes after exposure, n	13	0	13	–
30 minutes – 1 hour after exposure, n	8	0	8	
1–3 hours after exposure, n	3	0	3	
3–6 hours after exposure, n	7	3	4	
Arrived within 1 hours of exposure, n	21	0	21	0.027**ᵇ
Arrived after 1 hour of exposure, n	10	3	7	
More than 1 exposure, n	21	3	18	0.533
Seizure, n	1	0	1	1
ARDS, n	5	0	5	1
Requirement for endotracheal intubation and mechanical ventilation, n	12	3	9	0.049**
Mortality, n	8	3	5	0.012**

Notes: **Significant at P ≤ 0.05 level. ªP-value was calculated using the Mann Whitney *U*-test. ᵇP-value was calculated using fisher-exact test.
Abbreviations: n, number; ARDS, acute respiratory distress syndrome.

clinical manifestations. This finding is different from Abdel Baseer et al,[16] Banday et al,[12] and Banerjee et al,[17] where the most common muscarinic effect was miosis in all three studies. These autonomic signs were also the predominant manifestation of OP poisoning in 37 infants and children reported by Zwiener and Ginsburg.[18] In contrast, several studies have shown that neurological manifestations, including seizures and reduced levels of consciousness, are more common in pediatrics than the classical muscarinic and nicotinic features.[14,19–21] this variation of the clinical features from our study to others may be related to the route of exposure, the quantity absorbed, and the type of the organophosphate involved. The mortality rate in children exposed to organophosphate poisoning ranged from 4% to 30%.[12] Alruwaili et al reported that the mortality rate in children in Saudi Arabia's central region from organophosphate poisoning ranged from 1.5% to 4.6%. 5. The mortality rate in our study was 26%. This higher rate in our study reflects the severe presentations in the present study group requiring ICU management as compared to other studies, which included mild to moderate organophosphate poisoning in addition to severe OP intoxication. The documented causes of death in our patients were attributed to multiple organ failure in three cases and tachydysrhythmias in two. Our data has shown that delayed presentation to the hospital was a significant risk factor for mortality following OP exposure. Our finding is consistent with previous data showing that the mean time to presentation in OP poisoning was significantly higher in non-survivors.[15] The mortality rate was also higher in patients who required mechanical ventilation than in others. Our data indicates that two-thirds of the patients who were ventilated died. Compared to our study, 67% of the patients who developed respiratory failure and required ventilation in El-Naggar et al expired. In our study, nearly 40% of the patients required mechanical ventilation; in contrast, only 12% in El-Naggar et al were ventilated.[15] One possible explanation for this variation is the differences in the routes of exposure between the two studies (predominantly inhalational and dermal in our data, compared to oral ingestion in 86% of their patient group). Another difference is that only 36% of the 47 patients described by El-Naggar et al were admitted to the ICU, compared to 100% in our study. Of note, the serum lactate level in the mortality group was significantly higher than the survival group at hospital arrival, at 6 hours, and 24 hours post-exposure.[15] These data suggest that monitoring serum lactate levels during critical care in acute organophosphate intoxication might aid in the prognostication of pediatric patients presenting with severe organophosphate poisoning. To the best of our knowledge, no previous studies have investigated the clinical usefulness of serum lactate level as a prognostic factor in pediatric patients with acute OP intoxication. We found one study describing the usefulness of serum lactate level as a predictor of successful discontinuation of atropine infusion in adult patients with severe organophosphate poisoning.[22] The strength of this study includes being one of the few studies that

Table 5 Comparison Between Patients Who Required Mechanical Ventilation and Patients Who Did Not Require Mechanical Ventilation

Study Variables	Overall Population [n=31]	Not Intubated [n=19]	On Mechanical Ventilation [n=12]	P-value
Age, median (IQR)	2 (1–8) years	2 (1–8) years	2 (1–9) years	I
Route of exposure:				
Ingestion, n	23	12	11	0.108
Inhalation, n	18	6	12	<0.001**c
Skin contact, n	26	14	12	0.128
More than 1 exposure, n	21	9	12	0.004**b
Hospital arrival:				
30 minutes after exposure, n	13	11	2	–
30 minutes – 1 hours after exposure, n	8	7	1	
1–3 hours after exposure, n	3	1	2	
3–6 hours after exposure, n	7	0	7	
Arrived within 1 hours of exposure, n	21	18	3	<0.001**b
Arrived after 1 hour of exposure, n	10	1	9	
Vital signs:				
HR, median (IQR)	120 (110–134) beats per minute	116 (111–127) beats per minute	128 (82–142) beats per minute	0.617
RR, median (IQR)*	38 (33–45) breaths per minute	35 (32–39) breaths per minute	46 (34–53) breaths per minute	0.018
SBP, median (IQR)*	110 (101–111) mmHg	110 (101–114) mmHg	106 (100–110) mmHg	0.326
DBP, median (IQR)*	55 (47–59) mmHg	55 (50–59) mmHg	52 (45–63) mmHg	0.509
Glasgow Coma Scale, median (IQR)	14 (9–15)	15 (14–15)	8 (8–9)	<0.001**a
Lactate levels:				
Lactate at hospital arrival, median (IQR)	1 (1–1) mmol/L	1 (1–1) mmol/L	1.5 (1–2) mmol/L	0.014**a
Lactate 6 hours after hospital admission, median (IQR)	1 (1–3) mmol/L	1 (1–1) mmol/L	4 (3–4.75) mmol/L	<0.001**a
Lactate 24 hours after hospital admission, median (IQR)	1 (1–2) mmol/L	1 (1–1) mmol/L	2 (1.25–3) mmol/L	<0.001**a
Seizure, n	1	0	1	0.387
ARDS, n	5	0	5	0.005**b
Mortality, n	8	0	8	<0.001**b

Notes: *Missing details were estimated using expectation maximization. **Significant at P ≤ 0.05 level. [a]P-value was calculated using the Mann Whitney U-test. [b]P-value was calculated using Fisher's exact test. [c]P-value was calculated using chi-square test.
Abbreviations: IQR, interquartile range; n, number; ARDS, acute respiratory distress syndrome.

described the patients' characteristics and the risk factors for severe organophosphate poisoning in Saudi Arabia. One limitation of this study is its retrospective nature, based on information from the patients' medical records, with some data missing, including the type of OP agent involved. Another limitation is the small number of patients in the study over a long period of time, and the standard of care in the intensive care setting may have changed during the 10-year period.

In conclusion, the demographic characteristics, clinical features, clinical course, and outcomes of children hospitalized in the pediatric intensive care unit with organophosphate toxicity were characterized by presenting symptoms of organophosphate poisoning that differed greatly in children. Independent risk factors for mortality in PICU patients with OP poisoning include more than 1 hour of delayed hospital arrival; inhalational mode of exposure; mechanical ventilation; and high lactate levels in the first 24 hours post-exposure.

Table 6 Comparison Between Patients Who Survived and Patients Who Died

Study Variables	Overall Population [n=31]	Survivors [n=23]	Fatalities [n=8]	P-value
Age, median (IQR)	2 (1–8) years	2 (1–6) years	2.5 (1–13)	0.611[a]
Male, n	19	15	4	0.676[b]
Female, n	12	8	4	
Ingestion, n	23	15	8	0.076[b]
Inhalation, n	18	10	8	0.010**[b]
Skin contact, n	26	18	8	0.291[b]
More than 1 route of exposure, n	21	13	8	0.032**[b]
Suicidal, n	3	0	3	0.012**[b]
Arrived within 1 hours of exposure, n	21	21	0	<0.001**[b]
Arrived after 1 hour of exposure, n	10	2	8	
Bronchorrhea, n	28	20	8	0.550[b]
Sinus bradycardia, n	10	6	4	0.381[b]
Miosis, n	11	4	7	0.001**[b]
Salivation, n	13	7	6	0.043**[b]
Diarrhea, n	9	4	5	0.027**[b]
Fasciculation, n	6	1	5	0.002**[b]
Tachycardia, n	4	1	3	0.043**[b]
Drowsy, n	12	4	8	<0.001**[b]
Seizure, n	1	0	1	0.258[b]
HR, median (IQR)	120 (110–134) beats per minute	116 (110–127) beats per minute	133 (89–150) beats per minute	0.339[a]
RR, median (IQR)*	38 (33–45) breaths per minute	35 (32–39) breaths per minute	46 (37–53) breaths per minute	0.034**[a]
SBP, median (IQR)*	110 (101–111) mmHg	110 (102–113) mmHg	104 (92–109) mmHg	0.187[a]
DBP, median (IQR)*	55 (47–59) mmHg	55 (50–59) mmHg	51 (45–62) mmHg	0.550[a]
Lactate at hospital arrival, median (IQR)	1 (1–1) mmol/L	1 (1–1) mmol/L	2 (1–2) mmol/L	0.010**[a]
Lactate 6 hours after hospital admission, median (IQR)	1 (1–3) mmol/L	1 (1–1) mmol/L	4 (4–5) mmol/L	<0.001**[a]
Lactate 24 hours after hospital admission, median (IQR)	1 (1–2) mmol/L	1 (1–1) mmol/L	2.5 (2–3.75) mmol/L	<0.001**[a]
Treatment with atropine, n	17	9	8	0.003**[b]
Glasgow Coma Scale, median (IQR)	14 (9–15)	15 (13–15)	8 (7–9)	<0.001**[a]
ARDS, n	5	1	4	0.010**[b]
Requirement for endotracheal intubation and mechanical ventilation, n	12	4	8	<0.001**[b]
PICU length of stay, median (IQR)	2 (1–5) days	2 (1–4) days	7 (6–12) days	<0.001**[a]
Hospital length of stay, median (IQR)	4 (3–9) days	3 (3–6) days	9 (6–12) days	0.001**[a]

Notes: *Missing details were estimated using expectation maximization. **Significant at p ≤ 0.05 level. [a]P-value was calculated using the Mann Whitney *U*-test. [b]P-value was calculated using fisher-exact test.
Abbreviations: n, number; IQR, interquartile range; ARDS, cute respiratory distress syndrome; PICU, pediatric intensive care unit.

Ethical Approval

The study received ethical approval from Imam Abdulrahman Bin Faisal University's Institutional Review Board (IRB-2022-01-140). Because of the retroactive nature of the study, the IRB decided not to collect informed consent.

Acknowledgments

Author Contributions

All authors contributed equally to this analysis and significantly to its design, information management, or data collection and analysis; participated in its drafting or critically revised it for important intellectual content; agreed to submit it to the current journal; gave final approval of the version to be published; and agreed to be fully responsible for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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