



Clinical Severity, Treatment Characteristics, and Intensive Care Outcomes Among Patients with Organophosphate Poisoning Treated with Atropine and Pralidoxime: A Prospective Observational Study.

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Abstract

Background: Organophosphate poisoning is an important cause of morbidity and mortality, particularly in agricultural communities. Early severity assessment and timely antidotal therapy are essential for predicting respiratory complications and improving outcomes. This study evaluated clinical severity, treatment characteristics, intensive care course, and outcomes among patients with organophosphate poisoning.

Methods: This hospital-based observational study included 75 patients with organophosphate poisoning who were recruited consecutively. Sociodemographic characteristics, clinical manifestations, baseline Peradeniya Organophosphorus Poisoning (POP) score, atropine and pralidoxime therapy, complications, mechanical ventilation, ICU stay, and clinical outcomes were recorded. Associations were examined using appropriate statistical tests, with $p < .05$ considered statistically significant.

Results: The mean age was 40.28 ± 12.91 years, and 81.3% were male. Severe poisoning was identified in 36.0% of patients. Mechanical ventilation was required in 58.7%, while intermediate syndrome and aspiration pneumonia occurred in 17.3% and 13.3%, respectively. The median ICU stay was 4 days, and mortality was 17.3%. Mortality was significantly higher among patients with severe baseline POP scores than among those with non-severe poisoning (40.7% vs. 4.2%; $p < .001$). Delayed or absent POP-score reduction and mechanical ventilation were significantly associated with mortality. Early atropinisation was associated with lower mechanical ventilation requirements, shorter ICU stay, and better clinical outcomes. Atropine- and pralidoxime-dose categories were not significantly associated with POP-score reduction. **Conclusion:** Baseline POP severity, early clinical response, atropinisation status, and ventilatory requirement were important prognostic indicators. Early severity assessment, prompt atropinisation, serial POP-score monitoring, and timely respiratory support may improve outcomes in organophosphate poisoning.

Keywords: atropine; intensive care unit; mechanical ventilation; organophosphate poisoning; POP score.



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INTRODUCTION

Organophosphate poisoning remains an important cause of preventable morbidity and mortality, particularly in low- and middle-income countries where agricultural pesticides are readily accessible.[1] Exposure may occur accidentally, occupationally, or through intentional ingestion, with pesticide self-poisoning contributing substantially to the global burden of suicide. The rapid onset of toxicity, potential for respiratory failure, and requirement for prolonged critical care place considerable pressure on emergency and intensive care services. [2]

Organophosphate compounds produce toxicity primarily through inhibition of acetylcholinesterase, resulting in the accumulation of acetylcholine at muscarinic, nicotinic, and central nervous system synapses. [3] The resulting cholinergic syndrome may manifest as excessive salivation, bronchorrhoea, bronchospasm, vomiting, diarrhoea, miosis, bradycardia, muscle fasciculations, weakness, seizures, and altered consciousness. [4] Severe poisoning can lead to respiratory failure through a combination of excessive airway secretions, central respiratory depression, bronchospasm, and neuromuscular weakness. [5]

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The clinical course of organophosphate poisoning varies according to the compound involved, amount and route of exposure, time to hospital presentation, and severity at admission. Structured clinical assessment is therefore essential for early risk stratification and treatment planning. [6] The Peradeniya Organophosphorus Poisoning (POP) scale uses pupil size, respiratory rate, heart rate, fasciculations, level of consciousness, and seizures to classify poisoning severity. [7] Its simplicity makes it suitable for emergency and resource-limited settings, while higher scores have been associated with mechanical ventilation requirements and adverse outcomes. [8]

Initial management focuses on decontamination, airway stabilization, oxygenation, circulatory support, and timely administration of antidotal therapy. [9] Atropine counteracts muscarinic manifestations and is titrated according to clinical endpoints such as improvement in oxygenation, clearance of bronchial secretions, and cardiovascular stability. [10] Pralidoxime reactivates acetylcholinesterase before enzyme aging and may improve neuromuscular function. [11] However, its clinical benefit may vary according to the organophosphate compound, timing of administration, dosage regimen, and severity of poisoning. [12]

Patients with moderate or severe poisoning frequently require intensive monitoring because of the risks of respiratory failure, aspiration pneumonia, arrhythmias, seizures, intermediate syndrome, and secondary infections. [13] Mechanical ventilation is often necessary in patients with severe cholinergic manifestations or respiratory muscle weakness. [14] Baseline clinical severity, delayed improvement, failure to achieve atropinisation, mechanical ventilation, and prolonged ICU stay may identify patients at increased risk of mortality. [15] Intermediate syndrome may also prolong ventilatory dependence and hospitalization, further emphasizing the need for systematic assessment throughout the ICU course. [16]

Despite established management principles, treatment requirements and clinical outcomes vary considerably among patients with organophosphate poisoning. [17] Locally generated evidence regarding presentation, POP severity, atropine and pralidoxime use, early clinical response, complications, mechanical ventilation, ICU stay, and mortality can support early prognostic assessment and resource planning. [18] Therefore, the present study evaluated the clinical severity, treatment characteristics, ICU course, and outcomes of patients with organophosphate poisoning treated with atropine and pralidoxime and examined the associations of baseline severity, early response, atropinisation status, ventilation, and treatment-dose categories with POP-score reduction, ICU stay, and clinical outcome.

Aims and Objectives

Aim

To evaluate the clinical severity, treatment characteristics, intensive care course, and outcomes of patients with organophosphate poisoning treated with atropine and pralidoxime in the intensive care unit.

Objectives

- To describe the sociodemographic characteristics, hospital presentation, clinical manifestations, baseline POP severity, treatment profile, complications, mechanical ventilation requirement, ICU stay, and clinical outcomes of patients with organophosphate poisoning.
- To determine the association of baseline POP severity, early clinical response, early atropinisation, mechanical ventilation, and atropine and pralidoxime dose categories with POP-score reduction, duration of ICU stay, and clinical outcome.

MATERIALS AND METHODS

Study Design and Setting

A prospective observational study was conducted in the Medical Intensive Care Unit of the Department of General Medicine, Government Medical College and Hospital, Tiruppur, Tamil Nadu.

Study Population

The study population comprised adult patients with acute organophosphate poisoning admitted to the Medical Intensive Care Unit during the study period. Organophosphate poisoning was diagnosed based on a history of exposure, available information regarding the implicated compound, characteristic cholinergic manifestations, and supporting clinical evidence.

Study Duration

The study was conducted over 6 months.

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Inclusion and Exclusion Criteria

Patients aged 18 years or older who were admitted to the ICU with organophosphate poisoning and received atropine and pralidoxime were included. Patients or their legally authorised representatives were required to provide written informed consent.

Patients with a pre-existing psychiatric illness or delirium, chronic use of anticholinergic medications, or mixed poisoning involving alcohol, medicines, or other toxic substances were excluded. Patients who were discharged or died within 6 hours of ICU admission and those with incomplete essential clinical or treatment information were also excluded.

Sample Size and Sampling Technique

The sample size was calculated using the single-population proportion formula, $n = Z^2 p(1 - p)/d^2$, where n represented the required sample size, Z represented the standard normal deviate at a 95% confidence level (1.96), p represented the anticipated mortality proportion (25%) based on a previously published study, and d represented the absolute precision (10%). Accordingly, $n = (1.96)^2 \times 0.25 \times (1 - 0.25)/(0.10)^2 = 72.03$. The minimum required sample size was rounded up to 73. To account for incomplete information and potential attrition, the final sample size was fixed at 75 patients. Eligible patients admitted during the study period were recruited consecutively until the required sample size was attained.

Study Procedure

Patients admitted with suspected organophosphate poisoning were screened for eligibility. After obtaining written informed consent, information was collected using a structured case-record form. Sociodemographic information, including age and sex, was documented. Details regarding the implicated organophosphate compound, estimated quantity, route and intention of exposure, time of exposure, and time taken to reach the hospital were recorded wherever available.

Clinical assessment at admission included heart rate, blood pressure, respiratory rate, oxygen saturation, temperature, pupil size, level of consciousness, fasciculations, seizures, bronchorrhoea, bronchospasm, excessive salivation, lacrimation, gastrointestinal symptoms, and respiratory muscle weakness. The severity of poisoning at presentation was assessed using the Peradeniya Organophosphorus Poisoning scale.

Treatment information included the time to initiation of atropine and pralidoxime, initial dose, cumulative dose, duration of therapy, achievement of atropinisation, and use of mechanical ventilation or other supportive measures. Relevant haematological, biochemical, electrolyte, renal, hepatic, and arterial blood gas findings were documented when available.

Patients were monitored throughout their ICU stay. Serial clinical response and changes in the POP score were recorded. The development of intermediate syndrome, aspiration pneumonia, arrhythmias, sepsis, and other complications was documented. The requirement and duration of mechanical ventilation and length of ICU stay were recorded. The final outcome was classified as recovery or death.

Operational Definitions

Organophosphate poisoning was defined as a history or clinical suspicion of organophosphate exposure accompanied by characteristic muscarinic, nicotinic, or central nervous system manifestations.

Baseline severity was assessed using the POP scale, which evaluated pupil size, respiratory rate, heart rate, fasciculations, level of consciousness, and seizures. Scores of 0–3, 4–7, and 8–11 indicated mild, moderate, and severe poisoning, respectively. For the association analysis, mild and moderate cases were combined as the non-severe category, while scores of 8–11 were classified as severe poisoning.

Clinical atropinisation was defined by improvement in oxygenation and cardiorespiratory status, clearance of bronchial secretions, and the presence of a clear chest on auscultation. Early atropinisation was defined as the achievement of these clinical endpoints after initiating atropine. Patients who achieved these endpoints after the specified period were classified as not achieving early atropinisation. Patients who never attained the clinical endpoints were categorised as atropinisation not achieved.

Early clinical response was defined as a reduction in the POP score by Day 1 of treatment. A reduction occurring after Day 1 was considered a delayed response, while the absence of a reduction during the observation period was classified as no clinical response. Total POP-score reduction was calculated as the difference between the baseline POP score and the final documented POP score.

Mechanical ventilation was defined as the use of invasive ventilatory support at any time during the ICU stay. Intermediate syndrome was defined as the development of proximal limb, neck flexor, cranial nerve, or respiratory muscle weakness

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after the initial cholinergic phase. ICU stay was calculated from the date of ICU admission to transfer, discharge, or death. Recovery was defined as survival to discharge or transfer from intensive care after clinical stabilisation, while mortality referred to death during the hospital admission. Total atropine and pralidoxime doses were grouped into mild-, moderate-, and severe-dose categories.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics for Windows, version 26.0. Categorical variables were summarised using frequencies and percentages. Continuous variables were presented as mean and standard deviation when approximately normally distributed and as median and interquartile range when the distribution was skewed. Associations between categorical variables were examined using the chi-square test or Fisher's exact test when expected cell frequencies were less than five. Differences in continuous or ordinal outcomes between two groups were assessed using the independent-samples t test or Mann–Whitney U test, as appropriate. The Kruskal–Wallis test was used to compare ICU stay and POP-score reduction across three treatment or atropinisation categories. Spearman's rank correlation was used to examine the relationship between the baseline POP score and total POP-score reduction. All tests were two-tailed, and a p-value <0.05 was considered statistically significant.

Ethical Consideration

The study was conducted after obtaining approval from the Institutional Ethics Committee of Government Medical College and Hospital. Written informed consent was obtained from each patient or the legally authorised representative before enrolment. Patient identifiers were removed from the analytical dataset, and all collected information was maintained confidentially. Participation did not alter the standard treatment provided by the attending clinical team, and the study was conducted in accordance with the ethical principles of the Declaration of Helsinki..

RESULTS

Table 1. Sociodemographic Characteristics and Hospital Presentation of Patients with Organophosphate Poisoning (N = 75)

Characteristic	Category	n	%
Age, years, M (SD)	40.28 (12.91)	—	—
Age group	≤30 years	19	25.3
	31–49 years	34	45.3
	≥50 years	22	29.3
Sex	Male	61	81.3
	Female	14	18.7
Time to hospital arrival	≤2 hours	54	72.0
	>2 hours	21	28.0

Note. M = mean; SD = standard deviation.

The mean age of the study participants was 40.28 years (SD = 12.91). Most participants were aged 31–49 years (45.3%), followed by those aged ≥50 years (29.3%) and ≤30 years (25.3%). The study population showed a marked male predominance, with 61 (81.3%) males and 14 (18.7%) females. Most patients, 54 (72.0%), reached the hospital within 2 hours of exposure, whereas 21 (28.0%) arrived after 2 hours (Table 1).

Table 2. Clinical Manifestations and Baseline POP Score Severity of the Study Participants (N = 75)

Clinical characteristic	n	%
Gastrointestinal upset	68	90.7
Emesis	62	82.7
Bronchorrhoea	53	70.7
Bronchospasm	53	70.7
Diarrhoea	40	53.3
Excessive salivation	38	50.7
Bradycardia	20	26.7
Hypoxia	8	10.7
Seizures	6	8.0
Respiratory muscle weakness	4	5.3
Baseline POP severity		
Non-severe	48	64.0
Severe	27	36.0

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Gastrointestinal upset was the most frequently observed clinical manifestation, affecting 68 (90.7%) patients, followed by emesis in 62 (82.7%). Bronchorrhoea and bronchospasm were each present in 53 (70.7%) patients. Diarrhoea was reported in 40 (53.3%) patients, while 38 (50.7%) had excessive salivation. Bradycardia occurred in 20 (26.7%) patients. Hypoxia, seizures, and respiratory muscle weakness were less frequent, occurring in 8 (10.7%), 6 (8.0%), and 4 (5.3%) patients, respectively. Based on the baseline POP score, 48 (64.0%) patients had non-severe poisoning and 27 (36.0%) had severe poisoning (Table 2).

Table 3. Treatment Characteristics, Complications, ICU Course, and Clinical Outcomes of the Study Participants (N = 75)

Characteristic	Summary
Time to atropine administration, hours, M (SD)	2.10 (1.52)
Initial atropine dose, mg, M (SD)	1.87 (0.94)
Total atropine dose, mg, M (SD)	60.04 (47.64)
Duration of atropine therapy, days, M (SD)	3.84 (1.60)
Time to pralidoxime administration, hours, M (SD)	2.21 (1.55)
Initial pralidoxime dose, g, M (SD)	1.16 (0.43)
Total pralidoxime dose, g, M (SD)	10.65 (7.02)
Duration of pralidoxime therapy, days, M (SD)	3.69 (1.71)
ICU stay, days, median (IQR)	4 (3–5)
Mechanical ventilation, n (%)	44 (58.7)
Intermediate syndrome, n (%)	13 (17.3)
Aspiration pneumonia, n (%)	10 (13.3)
Early clinical response, n (%)	65 (86.7)
Recovered, n (%)	62 (82.7)
Death, n (%)	13 (17.3)

Note. ICU = intensive care unit; IQR = interquartile range; M = mean; SD = standard deviation.

The mean time to atropine administration was 2.10 hours (SD = 1.52), and the mean initial and total atropine doses were 1.87 mg (SD = 0.94) and 60.04 mg (SD = 47.64), respectively. Atropine therapy continued for a mean duration of 3.84 days (SD = 1.60). The mean time to pralidoxime administration was 2.21 hours (SD = 1.55), with mean initial and total doses of 1.16 g (SD = 0.43) and 10.65 g (SD = 7.02), respectively. The mean duration of pralidoxime therapy was 3.69 days (SD = 1.71). The median ICU stay was 4 days (IQR = 3–5), and 44 (58.7%) patients required mechanical ventilation. Intermediate syndrome developed in 13 (17.3%) patients, while 10 (13.3%) developed aspiration pneumonia. An early clinical response was observed in 65 (86.7%) patients. Overall, 62 (82.7%) patients recovered, while 13 (17.3%) died (Table 3).

Table 4. Association of Baseline POP Severity, Early Clinical Response, and Mechanical Ventilation with Clinical Outcome (N = 75)

Predictor	Category	Recovered, n (%)	Death, n (%)	p-value
Baseline POP severity	Non-severe	46 (95.8)	2 (4.2)	<0.001*
	Severe	16 (59.3)	11 (40.7)	
Early clinical response	POP-score reduction on Day 1	58 (98.3)	1 (1.7)	<0.001*
	Reduction after Day 1 or no reduction	4 (25.0)	12 (75.0)	
Mechanical ventilation	Not required	31 (100.0)	0 (0.0)	0.001*
	Required	31 (70.5)	13 (29.5)	

*Statistically significant

Note. POP = Peradeniya Organophosphorus Poisoning. Percentages were calculated within each predictor category. Fisher's exact test was used when the expected cell frequencies were less than five. A p-value <0.05 was considered statistically significant.

Baseline POP severity was significantly associated with clinical outcome. Among patients with non-severe poisoning, 46 (95.8%) recovered and 2 (4.2%) died, whereas among those with severe poisoning, 16 (59.3%) recovered and 11 (40.7%) died ($p < 0.001$). Early clinical response was also significantly associated with survival. Of the patients who demonstrated POP-score reduction on Day 1, 58 (98.3%) recovered and 1 (1.7%) died. In contrast, among patients who experienced a delayed reduction or no reduction, only 4 (25.0%) recovered and 12 (75.0%) died ($p < 0.001$). Mechanical ventilation was significantly associated with clinical outcome. All 31 patients who did not require ventilation recovered, whereas among the 44 patients requiring ventilation, 31 (70.5%) recovered and 13 (29.5%) died ($p = 0.001$) (Table 4).

Table 5. Association of Early Atropinisation with Mechanical ventilation and Clinical Outcome (N = 75)

Early atropinisation	n	No ventilation, n (%)	Ventilation, n (%)	Recovered, n (%)	Death, n (%)
No	44	12 (27.3)	32 (72.7)	37 (84.1)	7 (15.9)
Yes	28	19 (67.9)	9 (32.1)	25 (89.3)	3 (10.7)
Atropinisation not achieved	3	0 (0.0)	3 (100.0)	0 (0.0)	3 (100.0)
p-value		<0.001*		0.006*	

*Statistically significant

Early atropinisation status was significantly associated with the requirement for mechanical ventilation ($p < 0.001$). Among the 28 patients who achieved early atropinisation, 9 (32.1%) required mechanical ventilation, compared with 32 of the 44 patients (72.7%) who did not achieve early atropinisation. All three patients in whom atropinisation was not achieved required mechanical ventilation. Early atropinisation status was also significantly associated with clinical outcome ($p = 0.006$). Mortality occurred in 3 (10.7%) patients who achieved early atropinisation and 7 (15.9%) who did not achieve it. All three patients in whom atropinisation was not achieved died (Table 5).

Table 6. Association of Early Atropinisation and Treatment-Dose Categories with ICU Stay (N = 75)

Early atropinisation status	n	ICU stay in days, median (IQR)	p-value
Achieved early	28	3 (2–4)	0.002*
Not achieved early	44	4 (3–6)	
Atropinisation not achieved	3	5 (4–7)	

*Statistically significant

The duration of ICU stay differed significantly according to early atropinisation status ($p = 0.002$). Patients who achieved early atropinisation had a median ICU stay of 3 days (IQR = 2–4), compared with 4 days (IQR = 3–6) among those who did not achieve early atropinisation. Patients in whom atropinisation was not achieved had the longest median ICU stay of 5 days (IQR = 4–7) (Table 6).

Table 7. POP Score Reduction According to Atropinisation and Treatment-Dose Categories

Predictor	Category	n	Mean rank of POP score reduction	p-value
Early atropinisation status	Achieved early	28	29.73	0.003*
	Not achieved early	44	44.73	
	Atropinisation not achieved	3	16.50	
Total atropine-dose category	Mild	49	35.72	0.219
	Moderate	16	38.25	
	Severe	10	48.70	
Total pralidoxime-dose category	Mild	60	35.01	0.053
	Moderate	12	49.17	
	Severe	3	53.17	

Note. ICU = intensive care unit; IQR = interquartile range; POP = Peradeniya Organophosphorus Poisoning. Mean ranks were obtained from the Kruskal–Wallis test.

*Statistically significant at $p < 0.05$.

Total POP-score reduction differed significantly according to early atropinisation status ($p = 0.003$). The mean rank of POP-score reduction was 29.73 among patients who achieved early atropinisation, 44.73 among those who did not achieve early atropinisation, and 16.50 among those in whom atropinisation was not achieved. No significant difference in POP-score reduction was observed across the mild, moderate, and severe total atropine-dose categories, with mean ranks of 35.72, 38.25, and 48.70, respectively ($p = 0.219$). Similarly, POP-score reduction did not differ significantly across the mild, moderate, and severe pralidoxime-dose categories, although the mean ranks increased from 35.01 to 49.17 and 53.17, respectively ($p = 0.053$) (Table 7).

DISCUSSION

The patients in the present study had a mean age of 40.28 years, and nearly half (45.3%) were aged 31–49 years. This mean age was higher than the 34.22 years reported by Bhusal et al., although both studies indicated that organophosphate poisoning predominantly affected economically productive age groups [14]. The marked male predominance observed in the present study (81.3%) was consistent with the findings of Malaviya et al. and Kolli et al., who reported male proportions of 65.0% and 58.7%, respectively [15,18]. Conversely, Sungur and Güven reported a slight female predominance among patients requiring intensive care, suggesting that sex distributions may vary according to regional pesticide-use patterns

and the circumstances of exposure [11]. In the present study, 72.0% of patients reached the hospital within 2 hours. This comparatively early presentation was broadly consistent with the short mean presentation time reported by Bhusal et al. [14], whereas Malaviya et al. found that most patients presented within 2–6 hours [15]. Differences in presentation time may reflect geographical accessibility, referral pathways, recognition of poisoning, and availability of emergency transportation.

Gastrointestinal upset (90.7%) and emesis (82.7%) were the most frequent manifestations, followed by bronchorrhoea and bronchospasm, each occurring in 70.7% of patients. This pattern was consistent with the established muscarinic manifestations described by Peter et al., including vomiting, diarrhoea, excessive respiratory secretions, and bronchoconstriction [4]. Bhusal et al. similarly reported vomiting in 88.0% of patients, although tachycardia was more prominent in their study than in the present cohort [14]. Malaviya et al. identified vomiting and profuse respiratory secretions as common presenting features, supporting the predominance of gastrointestinal and respiratory manifestations observed in the present study [15]. By comparison, Kolli et al. reported miosis as one of the most common clinical findings, suggesting variation in the dominant manifestations across clinical settings [18]. Severe poisoning according to the baseline POP score was present in 36.0% of the current participants, slightly exceeding the 30.0% classified as severely poisoned by Kolli et al. [18], while Malaviya et al. reported no patients in the severe POP category [15]. These differences may be attributable to variations in referral practices, pesticide compounds, ingested doses, and delays before treatment.

More than half of the participants required mechanical ventilation (58.7%), indicating a substantial burden of respiratory compromise. This proportion was higher than the 21.2% reported by Sungur and Güven and the 30.0% reported by Bhusal et al., but lower than the 74.0% reported by Lee and Tai among patients specifically admitted to intensive care [5,11,14]. The intermediate syndrome occurred in 17.3% of the present cohort, which was comparable with the 19.1% reported by Sungur and Güven and the 22.0% reported by Lee and Tai [5,11]. Jayawardane et al. reported intermediate syndrome in approximately 12.8% of patients, with respiratory failure developing in half of the affected patients, emphasizing the importance of continued neuromuscular and respiratory monitoring after the initial cholinergic phase [12]. The mortality rate in the present study was 17.3%, which was higher than the 8.0% reported by Bhusal et al. and the 13.0% reported by Lee and Tai, but lower than the 27.6% reported by Sungur and Güven [5,11,14]. Such variation likely reflects differences in poisoning severity, ventilatory requirements, pesticide type, referral delay, and critical care resources.

Baseline POP severity was strongly associated with clinical outcome. Mortality was 40.7% among patients with severe poisoning compared with 4.2% among those with non-severe poisoning ($p < .001$). This finding supported the original work of Senanayake et al., in which higher POP scores were associated with mortality, ventilatory requirements, and greater atropine requirements [7]. Malaviya et al. likewise found higher mortality and mechanical ventilation requirements among patients with more severe POP classifications [15]. Kolli et al. reported mortality of 42.2% among severely poisoned patients, which was remarkably similar to the 40.7% mortality observed in the severe group in the present study [18]. Early POP-score reduction also demonstrated strong prognostic importance: mortality was only 1.7% among patients whose scores decreased on Day 1, compared with 75.0% among those with delayed or absent reduction ($p < .001$). This finding was consistent with the observations of Davies et al., who demonstrated that early clinical severity measures were useful for identifying patients at increased risk of death and intubation [6]. Mechanical ventilation was also associated with mortality ($p = .001$); however, this association should be interpreted as a marker of severe respiratory and neurological toxicity rather than evidence that ventilation itself increased mortality.

Early atropinisation was significantly associated with reduced mechanical ventilation, shorter ICU stay, and improved clinical outcome. Mechanical ventilation was required by 32.1% of patients who achieved early atropinisation, compared with 72.7% of those who did not achieve it early and all patients in whom atropinisation was not achieved ($p < .001$). Median ICU stay was also shorter among patients who achieved early atropinisation—3 days compared with 4 days among those with delayed atropinisation and 5 days among those who were never atropinised ($p = .002$). These findings were consistent with the systematic comparison by Eddleston et al., which emphasized the importance of rapid, clinically titrated atropine administration [9]. Sungur and Güven similarly highlighted early atropine therapy and respiratory support as central components of intensive care management [11]. Attari et al. reported that therapeutic strategies associated with reduced atropine requirements were accompanied by shorter hospitalization and lower mortality [17]. Lv et al. also found that interventions that shortened the time required to achieve atropine-related clinical stabilization reduced atropine exposure and improved clinical severity measures [20]. Nevertheless, the present associations may partly reflect baseline severity because patients with more profound poisoning may be more difficult to atropinise and more likely to require ventilation.

Treatment-dose categories were not significantly associated with the magnitude of POP-score reduction. The association was nonsignificant for total atropine-dose categories ($p = .219$), while the association for pralidoxime-dose categories approached but did not reach statistical significance ($p = .053$). These findings should not be interpreted as demonstrating

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an absence of treatment benefit, because higher cumulative doses were likely administered to patients with more severe or persistent toxicity. The Cochrane review by Buckley et al. concluded that available evidence was insufficient to establish a consistent clinical benefit of oxime therapy across different organophosphate compounds and treatment regimens [10]. Sungur and Güven similarly found no significant mortality difference associated with pralidoxime administration in their intensive care cohort [11]. Attari et al. reported improvements in clinical outcomes and atropine requirements with adjunctive treatment, but the effect on pralidoxime requirements was less evident [17]. Lv et al. observed reductions in atropine requirements and faster clinical improvement following adjunctive hemoperfusion, although hospital mortality was not significantly altered [20]. Collectively, these findings suggest that treatment dose is influenced by poisoning severity, treatment response, compound characteristics, and timing; therefore, cumulative dose categories should not be interpreted as independent indicators of treatment effectiveness without multivariable adjustment.

Limitations

The single-centre design limited the generalizability of the findings. The observational design and absence of adjustment for potential confounders prevented causal interpretation of the associations between treatment characteristics and clinical outcomes.

CONCLUSION

Organophosphate poisoning was associated with substantial respiratory morbidity and mortality, with more than half of the patients requiring mechanical ventilation. Severe baseline POP scores, delayed or absent early POP-score reduction, failure to achieve early atropinisation, and mechanical ventilation were significantly associated with adverse clinical outcomes. Early atropinisation was additionally associated with a shorter ICU stay and reduced requirement for mechanical ventilation, whereas cumulative atropine and pralidoxime dose categories were not significantly associated with POP-score reduction.

The POP score should be assessed at admission and monitored serially to facilitate early risk stratification and identify patients requiring intensive observation and ventilatory support. Prompt atropine administration with titration to established clinical endpoints, timely respiratory support, and close monitoring for intermediate syndrome and aspiration pneumonia are recommended. Larger multicentre prospective studies with adjustment for poisoning severity, type and quantity of organophosphate exposure, treatment delays, and antidote regimens are required to confirm these findings and define optimal atropine and pralidoxime protocols.

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