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Original Research Article

PERADENIYA ORGANOPHOSPHORUS POISONING SCORE, EARLY ATROPINISATION, AND INTENSIVE CARE OUTCOMES IN ACUTE ORGANOPHOSPHATE POISONING: A PROSPECTIVE OBSERVATIONAL STUDY FROM SOUTH INDIA

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ABSTRACT

Background: Organophosphate (OP) poisoning is a major preventable cause of death in agricultural India. We assessed severity, treatment and intensive care outcomes in acute OP poisoning and examined factors associated with mortality.

Materials and Methods: This prospective observational study enrolled 75 consecutive adults with acute OP poisoning treated with atropine and pralidoxime in a tertiary care intensive care unit (ICU) in Tamil Nadu over 6 months. Severity was graded with the Peradeniya Organophosphorus Poisoning (POP) scale at admission and serially. Ventilation, ICU stay and in-hospital outcome were analysed using chi-square, Fisher's exact and Kruskal–Wallis tests.

Results: Mean age was 40.3 ± 12.9 years; 81.3% were men and 36.0% had severe poisoning. Mechanical ventilation was required in 58.7%. Median ICU stay was 4 days and mortality 17.3%. Mortality was higher with severe than non-severe baseline POP scores (40.7% vs. 4.2%; $p < 0.001$) and with delayed or absent versus Day-1 POP-score reduction (75.0% vs. 1.7%; $p < 0.001$). Early atropinisation was associated with less ventilation (32.1% vs. 72.7%; $p < 0.001$) and shorter ICU stay (3 vs. 4 days; $p = 0.002$). Cumulative antidote dose was not associated with POP-score reduction.

Conclusions: A severe admission POP score and failure of the score to fall by Day 1 identified patients at highest risk of death. Serial POP scoring and prompt atropinisation aid early risk stratification.

Keywords: Atropine; Intensive care units; Organophosphate poisoning; Pralidoxime compounds; Respiration, artificial; Severity of illness index.

INTRODUCTION

Organophosphate (OP) poisoning remains an important cause of preventable morbidity and mortality in low- and middle-income countries, where agricultural pesticides are readily accessible.[1] Exposure may be accidental, occupational or intentional, and pesticide self-poisoning contributes substantially to the global burden of suicide. Rapid onset of toxicity, the risk of respiratory failure and the need for prolonged critical care place considerable pressure on emergency and intensive care services.[2]

Organophosphates inhibit acetylcholinesterase, causing acetylcholine to accumulate at muscarinic, nicotinic and central nervous system synapses.[3]. The resulting cholinergic syndrome includes salivation, bronchorrhoea, bronchospasm, vomiting, diarrhoea, miosis, bradycardia, fasciculations, weakness, seizures and altered consciousness.[4] Respiratory failure in severe poisoning results from a combination of excessive airway secretions, bronchospasm, central respiratory depression and neuromuscular weakness.[5]

The clinical course varies with the compound, the amount and route of exposure, the time to presentation and severity at admission, making structured early assessment essential for risk stratification.[6] The Peradeniya Organophosphorus Poisoning (POP) scale grades severity using pupil size, respiratory rate, heart rate, fasciculations, level of consciousness and seizures.[7] It requires no laboratory support, which suits resource-limited settings, and higher scores have been associated with the need for mechanical ventilation and adverse outcomes.[8]

Management centres on decontamination, airway and circulatory support and timely antidotal therapy.[9] Atropine reverses muscarinic effects and is titrated to clinical endpoints such as clear chest, adequate oxygenation and cardiovascular stability.[10]

Pralidoxime reactivates acetylcholinesterase before enzyme ageing and may improve neuromuscular function,[11] although its benefit varies with the compound, timing, dose regimen and severity of poisoning.[12] Patients with moderate or severe poisoning require intensive monitoring because of the risk of respiratory failure, aspiration pneumonia, arrhythmias, seizures, intermediate syndrome and secondary infection,[13] many need mechanical ventilation.[14] Baseline severity, delayed clinical improvement, failure to achieve atropinisation and the need for ventilation may identify patients at increased risk of death,[15] while intermediate syndrome can prolong ventilator dependence and hospitalisation.[16]

Despite established management principles, treatment requirements and outcomes vary widely between settings,[17] and locally generated data on severity, antidote use, early response and ICU outcomes can inform prognostic assessment and resource planning.[18] We therefore aimed to describe the clinical severity, treatment characteristics, ICU course and outcomes of adults with acute OP poisoning treated with atropine and pralidoxime, and to examine the association of baseline POP severity, early clinical response, early atropinisation, mechanical ventilation and antidote dose categories with POP-score reduction, length of ICU stay and in-hospital mortality.

MATERIALS AND METHODS

Study design and setting

This 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, India, over 6 months.

Participants

Adults (≥18 years) admitted to the ICU with acute OP poisoning who received atropine and pralidoxime were eligible. The diagnosis was based on a history of exposure, information on the implicated compound where available, and characteristic cholinergic manifestations. Written informed consent was obtained from each patient or a legally authorised representative.

Patients with pre-existing psychiatric illness or delirium, chronic use of anticholinergic drugs, or mixed poisoning involving alcohol, medicines or other toxic substances were excluded, as were those discharged or deceased within 6 hours of ICU admission and those with incomplete essential clinical or treatment data.

Sample size

Sample size was calculated using the formula for a single proportion, $n = Z^2p(1 - p)/d^2$, with $Z = 1.96$ (95% confidence), an anticipated mortality (p) of 25% from a previous study,[3] and an absolute precision (d) of 10%. This yielded 72.03, rounded up to 73; allowing for incomplete data, 75 patients were enrolled. Eligible patients were recruited consecutively until this number was reached.

Data collection

Data were recorded on a structured case-record form. Sociodemographic details, the implicated compound, estimated quantity, route and intent of exposure, time of exposure and time to hospital arrival were documented where available. Admission assessment included heart rate, blood pressure, respiratory rate, oxygen saturation, temperature, pupil size, level of consciousness, fasciculations, seizures, bronchorrhoea, bronchospasm, salivation, lacrimation, gastrointestinal symptoms and respiratory muscle weakness, and severity was graded with the POP scale. Treatment data comprised time to initiation, initial and cumulative dose and duration of atropine and pralidoxime, achievement of atropinisation, and the use of mechanical ventilation and other supportive measures. Haematological, biochemical, renal, hepatic and arterial blood gas results were recorded when available.

Patients were followed throughout their ICU stay, with serial recording of the POP score and clinical response. Intermediate syndrome, aspiration pneumonia, arrhythmias, sepsis and other complications, the need for and duration of mechanical ventilation, and the length of ICU stay were documented. The outcome was recorded as recovery or death.

Operational definitions

Severity: POP scores of 0–3, 4–7 and 8–11 denoted mild, moderate and severe poisoning, respectively.[7] For analysis, mild and moderate cases were combined as non-severe.

Atropinisation: clear chest on auscultation with clearance of bronchial secretions and improvement in oxygenation and cardiorespiratory status. Early atropinisation was defined as reaching these endpoints; patients reaching them later were classified as not achieving early atropinisation, and those who never reached them as atropinisation not achieved.

Early clinical response: any reduction in the POP score by Day 1 of treatment; reduction after Day 1 was termed delayed response and no reduction during

observation, no response. Total POP-score reduction was the difference between the baseline and the last recorded POP score.

Other definitions: mechanical ventilation was any invasive ventilatory support during the ICU stay. Intermediate syndrome was proximal limb, neck flexor, cranial nerve or respiratory muscle weakness developing after the initial cholinergic phase. ICU stay was counted from ICU admission to transfer, discharge or death. Recovery was survival to discharge or transfer from the ICU after stabilisation; mortality was death during the hospital admission. Cumulative atropine doses were categorised as mild, moderate and severe, and cumulative pralidoxime doses as mild, moderate and severe.

Statistical analysis

Data were entered in Microsoft Excel and analysed with IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as frequency and percentage, and continuous variables as mean ± standard deviation (SD) or median and interquartile range (IQR), according to distribution. Categorical associations were tested with the chi-square test or, when any expected cell count was below five, Fisher's exact test. Two-group comparisons of continuous or ordinal variables used the independent-samples t test or Mann–Whitney U test, and three-group comparisons of ICU stay and POP-score reduction used the Kruskal–Wallis test. The correlation between baseline POP score and total POP-score reduction was assessed with Spearman's rank correlation. All tests were two-tailed, with $p < 0.05$ considered significant.

Ethics

The study was approved by the Institutional Ethics Committee, Government Medical College and Hospital, Tiruppur and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained before enrolment. Participation did not alter the standard treatment given by the attending team, and the analytical dataset was de-identified.

RESULTS

Patient characteristics and clinical presentation

Of the 75 patients, 61 (81.3%) were men, with a mean age of 40.3 ± 12.9 years; 34 (45.3%) were aged 31–49 years. Fifty-four (72.0%) reached hospital within 2 hours of exposure (Table 1). The most frequent features were gastrointestinal upset (90.7%) and emesis (82.7%), followed by bronchorrhoea and bronchospasm (70.7% each). Seizures (8.0%) and respiratory muscle weakness (5.3%) were uncommon. At admission, 27 patients (36.0%) had severe poisoning. [Table 2]

Table 1: Sociodemographic characteristics and hospital presentation (N = 75)

Characteristic	Category	n	%
Age (years), mean ± 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

SD: Standard deviation

Table 2: Clinical manifestations and baseline POP severity (N = 75)

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 (0–7)	48	64.0
Severe (8–11)	27	36.0

POP: Peradeniya Organophosphorus Poisoning scale

Treatment, ICU course and outcome

Atropine was started at a mean of 2.10 ± 1.52 hours after arrival and pralidoxime at 2.21 ± 1.55 hours. The mean cumulative atropine dose was 60.04 ± 47.64 mg over 3.84 ± 1.60 days, and the mean cumulative pralidoxime dose was 10.65 ± 7.02 g over 3.69 ± 1.71 days. Forty-four patients (58.7%) required mechanical ventilation, 13 (17.3%) developed intermediate syndrome and 10 (13.3%) developed aspiration pneumonia. Median ICU stay was 4 days (IQR 3–5). Sixty-two patients (82.7%) recovered and 13 (17.3%) died. [Table 3]

Table 3: Treatment characteristics, complications, ICU course and outcome (N = 75)

Characteristic	Value
Time to atropine (hours), mean ± SD	2.10 ± 1.52
Initial atropine dose (mg), mean ± SD	1.87 ± 0.94
Total atropine dose (mg), mean ± SD	60.04 ± 47.64
Duration of atropine therapy (days), mean ± SD	3.84 ± 1.60
Time to pralidoxime (hours), mean ± SD	2.21 ± 1.55
Initial pralidoxime dose (g), mean ± SD	1.16 ± 0.43
Total pralidoxime dose (g), mean ± SD	10.65 ± 7.02
Duration of pralidoxime therapy (days), mean ± 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 (POP reduction by Day 1), n (%)	[65 (86.7)]
Recovered, n (%)	62 (82.7)
Died, n (%)	13 (17.3)

ICU: Intensive care unit; IQR: Interquartile range; POP: Peradeniya Organophosphorus Poisoning; SD: Standard deviation

Factors associated with mortality

Mortality was 40.7% with a severe baseline POP score compared with 4.2% with a non-severe score ($p < 0.001$). Patients whose POP score fell by Day 1 had a mortality of 1.7%, compared with 75.0% among those with delayed or no reduction ($p < 0.001$). No patient who avoided mechanical ventilation died, whereas mortality among ventilated patients was 29.5% ($p = 0.001$). [Table 4]

Table 4: Association of baseline POP severity, early clinical response and mechanical ventilation with outcome (N = 75)

Variable	Category	Recovered, n (%)	Died, n (%)	*p*
Baseline POP severity	Non-severe (n = 48)	46 (95.8)	2 (4.2)	<0.001*
	Severe (n = 27)	16 (59.3)	11 (40.7)	
Early clinical response	Reduction by Day 1 (n = 59)	58 (98.3)	1 (1.7)	<0.001*
	Delayed or no reduction (n = 16)	4 (25.0)	12 (75.0)	
Mechanical ventilation	Not required (n = 31)	31 (100.0)	0 (0.0)	0.001*
	Required (n = 44)	31 (70.5)	13 (29.5)	

Percentages are row percentages. Fisher's exact test was used where any expected cell count was <5. ∗Statistically significant ($p < 0.05$). POP: Peradeniya Organophosphorus Poisoning

Early atropinisation, ventilation and ICU stay

Early atropinisation was achieved in 28 patients (37.3%), achieved later in 44 (58.7%) and not achieved in 3 (4.0%). Mechanical ventilation was needed in 32.1% of patients with early atropinisation, 72.7% of those with later atropinisation and all three who were never atropinised ($p < 0.001$). Mortality was 10.7%, 15.9% and 100%, respectively ($p = 0.006$); the difference was driven largely by the never-atropinised group (Table 5). Median ICU stay was 3, 4 and 5 days in the three groups, respectively ($p = 0.002$). [Table 5]

Table 5: Association of early atropinisation with mechanical ventilation and outcome (N = 75)

Atropinisation status	n	Not ventilated, n (%)	Ventilated, n (%)	Recovered, n (%)	Died, n (%)
Early	28	19 (67.9)	9 (32.1)	25 (89.3)	3 (10.7)
Not early (later)	44	12 (27.3)	32 (72.7)	37 (84.1)	7 (15.9)
Not achieved	3	0 (0.0)	3 (100.0)	0 (0.0)	3 (100.0)
p		<0.001*		0.006*	

Percentages are row percentages. ∗Statistically significant ($p < 0.05$)

Table 6: Length of ICU stay by atropinisation status (N = 75)

Atropinisation status	n	ICU stay (days), median (IQR)	*p*
Early	28	3 (2–4)	0.002*
Not early (later)	44	4 (3–6)	
Not achieved	3	5 (4–7)	

Kruskal–Wallis test. ∗Statistically significant ($p < 0.05$). ICU: Intensive care unit; IQR: Interquartile range

POP-score reduction by atropinisation status and antidote dose

Total POP-score reduction differed by atropinisation status ($p = 0.003$), with the highest mean rank among patients atropinised later (44.73), followed by those atropinised early (29.73) and those never atropinised (16.50). POP-score reduction did not differ across cumulative atropine-dose categories ($p = 0.219$) or pralidoxime-dose categories ($p = 0.053$), although mean ranks rose with increasing pralidoxime dose. [Table 7]

Table 7: Total POP-score reduction by atropinisation status and cumulative antidote dose (N = 75)

Variable	Category	n	Mean rank	*p*
Atropinisation status	Early	28	29.73	0.003*
	Not early (later)	44	44.73	
	Not achieved	3	16.50	
Cumulative atropine dose	Mild	49	35.72	0.219
	Moderate	16	38.25	
	Severe	10	48.70	
Cumulative pralidoxime dose	Mild	60	35.01	0.053
	Moderate	12	49.17	
	Severe	3	53.17	

Kruskal–Wallis test; a higher mean rank indicates a greater POP-score reduction. ∗Statistically significant ($p < 0.05$). POP: Peradeniya Organophosphorus Poisoning

DISCUSSION

In this prospective ICU cohort, more than half of the patients required mechanical ventilation and nearly one in six died. A severe admission POP score and, in particular, failure of the score to fall within the first day of treatment were strongly associated with death, while early atropinisation was associated with less ventilator use and a shorter ICU stay.

Our patients were predominantly men in the economically productive age group. The mean age (40.3 years) was higher than the 34.2 years reported by Bhusal et al,[14] and the male predominance (81.3%) exceeded that reported by Malaviya et al. (65.0%) and Kolli et al. (58.7%).[15,18] Sungur and Güven, in contrast, reported a slight female predominance among ICU patients,[11] suggesting that sex distribution reflects regional patterns of pesticide use and the circumstances of exposure. Nearly three-quarters of our patients arrived within 2 hours, similar to Bhusal et al,[14] and earlier than in the series of Malaviya et al., where most presented at 2–6 hours,[15] such differences probably reflect geography, referral pathways and access to emergency transport.

The predominance of gastrointestinal and respiratory muscarinic features is consistent with the classical description by Peter et al,[4] and with the findings of Bhusal et al., who reported vomiting in 88.0% of patients,[14] and Malaviya et al.[15] Kolli et al. reported miosis as a leading sign,[18] indicating that the dominant features vary between settings. Severe poisoning was present in 36.0% of our patients, slightly more than the 30.0% reported by Kolli et al,[18] whereas Malaviya et al. recorded no patients in the severe POP category.[15] Referral practices, the compounds involved, the quantity ingested and pre-hospital delay are likely explanations.

The ventilation rate of 58.7% was higher than the 21.2% and 30.0% reported by Sungur and Güven and Bhusal et al., but lower than the 74.0% reported by Lee and Tai in an exclusively ICU population.[5,11,14] Intermediate syndrome (17.3%) occurred at a frequency comparable with that reported by Sungur and Güven (19.1%) and Lee and Tai (22.0%).[5,11] Jayawardane et al. observed intermediate syndrome in about 12.8% of patients, half of whom developed respiratory failure,[12] and serial nerve-conduction studies suggest that it involves two distinct processes at the neuromuscular junction,[19] underlining the need for continued neuromuscular and respiratory monitoring beyond the cholinergic phase. Our mortality of 17.3% lies between the 8.0% and 13.0% reported by Bhusal et al. and Lee and Tai and the 27.6% reported by Sungur and Güven,[5,11,14] variation that likely reflects case mix, pesticide type, referral delay and critical care resources.

The tenfold difference in mortality between severe and non-severe baseline POP scores (40.7% vs. 4.2%) supports the original validation by Senanayake et al., who linked higher scores to death, ventilation and greater atropine requirements,[7] and the findings of Malaviya et al.[15] Mortality in our severe group was almost identical to the 42.2% reported by Kolli et al.[18] The early trajectory of the score was even more discriminating: only 1 of 59 patients whose score fell by Day 1 died, against 12 of 16 whose score did not. This agrees with Davies et al., who showed that early clinical severity measures identify patients at risk of intubation and death,[6] and suggests that repeating the POP score at 24 hours is a simple way to identify patients who need escalation of care. The association between mechanical ventilation and mortality should be read as a marker of severe respiratory and neurological toxicity, not as an effect of ventilation itself.

Early atropinisation was associated with less need for ventilation (32.1% vs. 72.7%) and a shorter ICU stay (median 3 vs. 4 days), in line with the emphasis by Eddleston et al. on rapid, clinically titrated atropine dosing,[9] and with ICU management principles described by Sungur and Güven.[11] Attari et al. and Lv et al. similarly found that strategies which reduced atropine requirement or shortened the time to clinical stabilisation were accompanied by better outcomes.[17,20] However, mortality differed little between early and later atropinisation (10.7% vs. 15.9%), and the overall association with mortality was driven by the three patients who were never atropinised. Because more severely poisoned patients are harder to atropinise and more likely to need ventilation, these associations may partly reflect baseline severity. The smaller POP-score reduction among early-atropinised patients is likely explained in a similar way: patients who stabilise quickly tend to have lower admission scores, leaving less room for reduction.

Cumulative atropine and pralidoxime dose categories were not associated with the magnitude of POP-score reduction, although the trend for pralidoxime approached significance ($p = 0.053$). This should not be interpreted as an absence of treatment benefit, because higher cumulative doses are given to patients with more severe or persistent toxicity. The Cochrane review by Buckley et al. found insufficient evidence of a consistent benefit of oximes across compounds and regimens,[10] and Sungur and Güven found no mortality difference with pralidoxime.[11] Attari et al. reported a reduced atropine requirement with adjunctive therapy but a less clear effect on pralidoxime use,[17] and Lv et al. reported faster improvement without a significant change in mortality.[20] Cumulative dose is therefore better regarded as a marker of severity and response than as an independent measure of treatment effectiveness.

Strengths and limitations

The prospective design, consecutive enrolment, standardised severity scoring and serial reassessment are strengths of this study. Its limitations include the single-centre setting and modest sample size, which limit generalisability and statistical power; the small number of deaths precluded multivariable analysis, so the reported associations are unadjusted and cannot be interpreted causally. The implicated compound and quantity were not available for all patients, preventing compound-specific analysis, and plasma cholinesterase activity was not included in the analysis. Outcomes were limited to the hospital stay, without post-discharge follow-up.

CONCLUSION

Acute OP poisoning in this ICU cohort carried a substantial burden of respiratory failure and a mortality of 17.3%. A severe admission POP score and failure of the score to fall by Day 1 were strongly associated with death, and early atropinisation was associated with less need for ventilation and a shorter ICU stay. Cumulative antidote dose did not reflect clinical improvement. The POP score should be recorded at admission and repeated at 24 hours to guide escalation of care, alongside prompt atropine titrated to clinical endpoints and close monitoring for intermediate syndrome and aspiration pneumonia. Larger multicentre studies with adjustment for severity, compound, treatment delay and antidote regimen are needed to confirm these findings and refine antidote protocols.

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