

Organophosphorus Compound Poisoning and Serum Cholinesterase Levels: An Observational Study on Clinical Severity and Outcome

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ABSTRACT

Background: Organophosphorus compound (OPC) poisoning remains one of the leading causes of hospital admission and preventable mortality in agrarian regions of the developing world. Serum cholinesterase estimation and clinical severity scoring at presentation are widely used to grade the poisoning and anticipate outcome. The objective of this study is to assess the demographic and clinical profile of patients with acute OPC poisoning, to correlate serum cholinesterase levels with clinical severity (Peradeniya Organophosphorus Poisoning score) and with outcome (death versus survival), and to identify factors associated with mortality. **Methods:** This observational study was carried out in the Department of Internal Medicine, Stanley Medical College, Chennai, over a six-month period. One hundred clinically suspected cases of OPC poisoning aged above 12 years were evaluated; patients with mixed/double poisoning, chronic medical illness, concurrent use of confounding drugs, and pregnancy were excluded. Serum cholinesterase was estimated at admission in all patients, and clinical severity was graded using the Peradeniya Organophosphorus Poisoning (POP) score. Outcome was dichotomised into a death group and a survival group and analysed with the unpaired t test, Chi-square/Fisher's exact test, and Pearson correlation, taking $p < 0.05$ as significant. **Results:** Among the analysed cohort (30 patients; 6 deaths, 24 survivors), mortality was exclusively seen in the severe category of the POP score (100% of deaths versus 4.17% of survivors, $p < 0.0001$). The mean amount of OPC consumed was significantly higher in the death group (131.67 ± 60.80 ml) than in survivors (65.63 ± 26.51 ml, $p = 0.0003$). Mean serum cholinesterase was markedly lower in the death group (336.17 ± 164.66 IU/L) compared with survivors (2732.33 ± 2415.77 IU/L, $p = 0.0235$), and fell progressively with increasing clinical severity (mild 5090.09, moderate 782.92, severe 315.29 IU/L; $p < 0.0001$, ANOVA). A strong inverse correlation was found between serum cholinesterase level and POP score (Pearson $r = -0.767$, $R^2 = 0.588$, $p < 0.0001$). Requirement of mechanical ventilation (100% versus 8.33%, $p < 0.0001$) and longer ICU stay (5.17 versus 2.79 days, $p = 0.0201$) were also significantly associated with death. Age, gender, occupation and the type of OPC consumed did not differ significantly between the two groups. **Conclusion:** Low serum cholinesterase level at admission correlates strongly with higher clinical severity, need for mechanical ventilation, prolonged ICU stay and mortality in acute OPC poisoning. Serum cholinesterase, together with the Peradeniya score, is a simple and reliable bedside tool for early risk stratification and outcome prediction.

Keywords: Organophosphorus poisoning; serum cholinesterase; pseudocholinesterase; Peradeniya score; pesticide poisoning; mortality; mechanical ventilation.

INTRODUCTION

Organophosphorus compounds (OPCs) are widely used as agricultural pesticides across South Asia because of

their low cost and easy availability, and consequently their deliberate or accidental ingestion is one of the commonest medical emergencies encountered in

hospitals of rural and semi-urban India. Self-poisoning with pesticides accounts for a substantial proportion of suicide-related deaths in the developing world, and OPCs contribute disproportionately to this burden because of their ready availability and high case fatality (1,17,18).

OPCs act by irreversibly inhibiting acetylcholinesterase at nerve synapses and neuromuscular junctions, leading to accumulation of acetylcholine and a cholinergic crisis manifesting as muscarinic (salivation, lacrimation, urination, defecation, emesis, miosis, bronchorrhoea), nicotinic (muscle weakness and fasciculation) and central nervous system effects (seizures, coma and respiratory failure) (11,16). Death in severe poisoning is usually attributable to respiratory failure arising from a combination of bronchorrhoea, bronchospasm, central respiratory depression and intermediate syndrome-related neuromuscular weakness.

Because clinical severity at presentation varies widely, several scoring systems have been developed to objectively grade severity and predict outcome. The Peradeniya Organophosphorus Poisoning (POP) scale, described by Senanayake, de Silva and Karalliedde in 1993, remains the most extensively validated and widely used bedside tool; it grades severity from clinical parameters (pupil size, respiratory rate, heart rate, fasciculations, level of consciousness and seizures) into mild (0-3), moderate (4-7) and severe (8-11) categories, and has been shown in multiple subsequent studies to correlate with the need for ventilatory support and with mortality (1,2,5,6).

Serum (pseudo)cholinesterase, synthesised in the liver and present in plasma, falls in proportion to the degree of acetylcholinesterase inhibition and is readily estimated in most clinical laboratories. A number of Indian and South Asian studies have shown that the degree of fall in serum cholinesterase correlates with clinical severity, requirement of ventilation, duration of ICU stay and mortality, making it a practical, inexpensive marker for triage and prognostication in resource-limited settings (3,4,12,13,15,19). However, the strength of this correlation, and the relative contribution of the amount of poison consumed, vital parameters and treatment requirements to outcome, continue to be examined in different populations.

This study was undertaken with the following objectives: (i) to describe the demographic and clinical profile of patients admitted with acute OPC poisoning; (ii) to grade clinical severity using the Peradeniya Organophosphorus Poisoning score and correlate it with serum cholinesterase levels at admission; (iii) to compare the death group and survival group with respect to age, gender, occupation, amount of OPC consumed, vital parameters, type of compound consumed, need for mechanical ventilation and duration of ICU stay; and

(iv) to evaluate serum cholinesterase level as a predictor of clinical outcome in acute OPC poisoning.

MATERIALS AND METHODS

This hospital-based observational study was conducted in the Department of Internal Medicine, Stanley Medical College, Chennai, a tertiary care teaching hospital, over a period of six months. A total of 100 consecutive patients clinically suspected of organophosphorus compound (OPC) poisoning were screened for eligibility. Any patient who developed muscarinic, nicotinic, or central nervous system manifestations following dermal, respiratory, or oral exposure to organophosphorus compounds such as chlorpyrifos, dimethoate, monocrotophos, or methyl parathion was considered a case of OPC poisoning, and the diagnosis was confirmed by estimation of serum cholinesterase activity. Patients of either sex aged more than 12 years with clinical suspicion of OPC poisoning were included in the study. Patients with poisoning due to double or mixed insecticides, multiple poisoning with other agents such as opioids, diazepam, or barbiturates, chronic medical illnesses, those receiving drugs that could interfere with cholinesterase activity or its clinical assessment (including aminophylline, suxamethonium, reserpine, and phenothiazine-type tranquilizers), pregnant women, and patients who had already received treatment before admission to the study hospital were excluded.

All patients underwent a detailed clinical evaluation, including history and physical examination, at the time of admission. Resuscitative measures were initiated immediately and included gastric lavage, skin decontamination, establishment of intravenous access, and administration of an intravenous bolus dose of atropine. Patients were assessed for respiratory failure, and those requiring ventilatory support were intubated and mechanically ventilated. Atropine therapy was continued and titrated according to clinical signs of atropinisation, and pralidoxime infusion was administered as specific antidotal therapy. Baseline investigations, including arterial blood gas analysis, complete blood count, random blood sugar, renal function tests, and chest radiography, were performed in all patients. Serum cholinesterase levels were measured at admission before the initiation of oxime therapy to avoid treatment-related alterations in enzyme activity. Clinical severity was assessed using the Peradeniya Organophosphorus Poisoning (POP) score and categorized as mild (0–3), moderate (4–7), or severe (8–11). Patients were followed throughout their hospital stay, and the final clinical outcome was recorded as either survival or death.

The study protocol, questionnaire, and informed consent documents were approved by the Institutional Ethics

Committee. Written informed consent was obtained from all participants or their legally authorized representatives in both English and Tamil, and confidentiality of participant information was strictly maintained. Statistical analysis was performed using SPSS version 26.0. Continuous variables were expressed as mean $\pm$ standard deviation (SD) and categorical variables as frequencies and percentages. Continuous variables were compared between the survival and death groups using the unpaired Student's t-test, while categorical variables were analysed using the Chi-square test or Fisher's exact test, as appropriate. Pearson correlation analysis and one-way analysis of variance (ANOVA) were used to assess the relationship between serum cholinesterase levels, POP score, and clinical severity. A p value of less than 0.05 was considered statistically significant.

RESULTS

Of the patients evaluated during the study period, complete outcome and laboratory data were available for analysis in 30 patients, of whom 6 (20.0%) belonged to

the death group and 24 (80.0%) belonged to the survival group.

The mean age of patients in the death group was 40.33 ± 16.93 years compared with 33.13 ± 15.51 years in the survival group; the majority of deaths occurred in the 21-30 year and 51-60 year age bands (50.0% each), while survivors were predominantly in the 21-30 year band (33.3%). This difference in age distribution was not statistically significant ($p=0.3253$, unpaired t test). Males predominated in both groups – 100.0% of the death group and 79.17% of the survival group were male and the difference in gender distribution between groups was not statistically significant ($p=0.5526$, Fisher's exact test).

Of the 100 patients screened during the study period, 70 were excluded from the final analysis because they did not meet the predefined eligibility criteria or had incomplete clinical, laboratory, or outcome data. The remaining 30 patients with complete clinical, laboratory, and outcome information were included in the final analysis.

Table 1. Age distribution of the death group and survival group

Age group (years)	Death group (n)	Survival group (n)	Death group (%)	Survival group (%)
≤ 20	0	4	0.00	16.67
21-30	3	8	50.00	33.33
31-40	0	5	0.00	20.83
41-50	0	0	0.00	0.00
51-60	3	7	50.00	29.17
Total	6	24	100.00	100.00

Mean age: Death group 40.33 ± 16.93 years vs Survival group 33.13 ± 15.51 years; $p=0.3253$ (unpaired t test, not significant).

On grading clinical severity by the Peradeniya score, all 6 patients in the death group (100.0%) belonged to the severe category, whereas in the survival group 11 patients (45.83%) had mild and 12 (50.0%) had moderate poisoning, with only 1 (4.17%) in the severe category. This difference was highly significant ($p<0.0001$, Fisher's exact test), confirming that severe

clinical grading at presentation was almost exclusively associated with a fatal outcome. The mean quantity of OPC consumed was also significantly higher among those who died (131.67 ± 60.80 ml) than among survivors (65.63 ± 26.51 ml, $p=0.0003$, unpaired t test), a difference amounting to roughly a two-fold higher intake in the death group.

Table 2. Peradeniya Organophosphorus Poisoning (POP) score and amount of OPC consumed by outcome group

Parameter	Death group	Survival group	p value
POP score – Mild (0-3), n (%)	0 (0.00)	11 (45.83)	
POP score – Moderate (4-7), n (%)	0 (0.00)	12 (50.00)	$<0.0001^*$
POP score – Severe (8-11), n (%)	6 (100.00)	1 (4.17)	
Amount consumed, mean $\pm$ SD (ml)	131.67 ± 60.80	65.63 ± 26.51	0.0003^*

*Statistically significant ($p<0.05$). POP score categorical comparison by Fisher's exact test; amount consumed compared by unpaired t test.

Patients in the death group presented with significantly lower mean pulse rate (76.00 ± 2.19 bpm vs 94.13 ± 16.20 bpm, $p=0.0117$), lower systolic blood pressure

(106.67 ± 5.16 mm Hg vs 120.42 ± 13.01 mm Hg, $p=0.0181$), and lower diastolic blood pressure (70.00 ± 0.00 mm Hg vs 78.33 ± 7.61 mm Hg, $p=0.0132$)

compared with survivors. Respiratory rate was higher in the death group (18.00 ± 4.38 vs 15.92 ± 3.36 breaths/min) but this difference did not reach statistical significance ($p=0.2109$). Body temperature was comparable between the two groups (98.50°F vs 98.79°F).

Mean serum cholinesterase level at admission was markedly and significantly lower in the death group (336.17 ± 164.66 IU/L) than in the survival group (2732.33 ± 2415.77 IU/L, $p=0.0235$, unpaired t test) – an almost eight-fold difference. On categorisation, all 6 patients (100.0%) in the death group had severe poisoning by cholinesterase level (≤ 700 IU/L), whereas in the survival group cholinesterase levels were distributed across the severe (16.67%), moderate 701-

1400 IU/L (41.67%) and latent >3500 IU/L (41.67%) categories, with none in the mild 1401-3500 IU/L band. When serum cholinesterase was analysed against the three clinical-severity strata (irrespective of outcome), the mean levels fell progressively with worsening severity: mild 5090.09 ± 1422.07 IU/L, moderate 782.92 ± 267.68 IU/L and severe 315.29 ± 160.14 IU/L ($p<0.0001$, single-factor ANOVA). Correlation analysis confirmed a strong, statistically significant, negative relationship between serum cholinesterase and the Peradeniya score (Pearson $r = -0.767$; $R^2 = 0.588$; $p<0.0001$), indicating that as serum cholinesterase level falls, clinical severity as measured by the POP score rises, with the regression model accounting for approximately 59% of the variability observed.

Table 3. Serum cholinesterase level in relation to outcome and clinical severity

Serum cholinesterase parameter	Death group	Survival group	p value
Mean $\pm$ SD (IU/L) – by outcome	336.17 ± 164.66	2732.33 ± 2415.77	0.0235*
≤ 700 IU/L – severe, n (%)	6 (100.00)	4 (16.67)	
701-1400 IU/L – moderate, n (%)	0 (0.00)	10 (41.67)	$<0.0001^*$
>3500 IU/L – latent, n (%)	0 (0.00)	10 (41.67)	

Table 4. Mean serum cholinesterase level according to clinical severity grade (POP score)

Clinical severity	Mild	Moderate	Severe
Mean serum cholinesterase (IU/L)	5090.09	782.92	315.29
SD (IU/L)	1422.07	267.68	160.14

ANOVA, $p<0.0001$. Pearson correlation between serum cholinesterase and POP score: $r = -0.767$, $R^2 = 0.588$, $p<0.0001$.

All 6 patients (100.0%) in the death group required mechanical ventilation compared with only 2 of 24 (8.33%) in the survival group, a highly significant difference ($p<0.0001$, Fisher's exact test). The mean duration of ICU stay was also significantly longer in the death group (5.17 ± 2.79 days) than in the survival group (2.79 ± 1.93 days, $p=0.0201$, unpaired t test); a third of

the death group required more than 7 days of ICU care compared with only 4.17% of survivors. The type of OPC consumed did not differ significantly between groups (chlorpyrifos being the commonest agent in both, 66.67% of deaths and 29.17% of survivors; $p=0.0843$), and occupation category also showed no significant association with outcome ($p=0.5433$).

Table 5. Mechanical ventilation requirement and ICU stay duration by outcome group

Parameter	Death group	Survival group	p value
Mechanical ventilation required, n (%)	6 (100.00)	2 (8.33)	$<0.0001^*$
ICU stay, mean $\pm$ SD (days)	5.17 ± 2.79	2.79 ± 1.93	0.0201*

*Statistically significant ($p<0.05$).

DISCUSSION

This study evaluated the demographic, clinical and biochemical profile of patients admitted with acute organophosphorus compound poisoning and examined the association of serum cholinesterase level with clinical severity and outcome. Consistent with the existing literature on pesticide self-poisoning in South Asia, most patients were young adults, and males predominated even though the difference in age and gender distribution between the death and survival groups was not statistically significant in this cohort, an

observation that mirrors the findings of Ahuja et al. and Batra et al. in Indian ICU-based poisoning cohorts, where age and sex alone were poor discriminators of mortality (7,8).

The amount of OPC consumed emerged as a strong determinant of outcome in this study, with the death group consuming approximately twice the mean volume ingested by survivors (131.67 ml vs 65.63 ml, $p=0.0003$). This dose-dependent relationship between quantity ingested and mortality has been repeatedly demonstrated and is biologically consistent with greater

acetylcholinesterase inhibition following larger exposure (13,18).

The Peradeniya Organophosphorus Poisoning score, first described by Senanayake et al., proved to be a robust discriminator of outcome in this cohort: every patient who died was graded as severe at presentation, while the majority of survivors were mild or moderate ($p < 0.0001$). This finding closely reproduces the original validation study, in which POP score at admission correlated significantly with death, need for ventilatory support and atropine requirement (1), and is corroborated by more recent Indian and Sri Lankan cohorts confirming the scale's prognostic value (2,5,6,9).

Serum cholinesterase level at admission was significantly lower in the death group and fell progressively across increasing severity strata, with a strong negative correlation with the POP score ($r = -0.767$, $p < 0.0001$). This finding is in agreement with several Indian studies that have shown an inverse relationship between serum pseudocholinesterase activity and both clinical severity and mortality, reinforcing the biochemical basis of the POP score and supporting the use of serum cholinesterase as a complementary, objective marker of severity in settings where clinical scoring alone may be influenced by inter-observer variability (3,4,12,15,19).

The requirement of mechanical ventilation and a longer ICU stay were both strongly associated with death in this study, consistent with the well-established observation that respiratory failure - resulting from a combination of bronchorrhoea, neuromuscular weakness and central respiratory depression - is the principal proximate cause of death in severe OPC poisoning (11,16,20). Vital parameters at presentation also differed significantly, with lower pulse rate and blood pressure in the death group, possibly reflecting a pattern of severe cholinergic bradycardia and hypotension or late presentation with cardiovascular compromise rather than the tachycardia and hypertension classically associated with early nicotinic and adrenergic stimulation; cardiac and autonomic dysfunction in OPC poisoning has been described in earlier studies and merits further evaluation in larger cohorts (20).

The type of compound consumed (predominantly chlorpyrifos in this cohort) did not significantly influence outcome, which is broadly consistent with reports that overall dose and time to treatment are more important prognostic determinants than the specific OPC formulation, although some individual compounds are recognised to carry higher inherent toxicity (17,19). Taken together, these findings support incorporating serum cholinesterase estimation alongside a validated clinical severity score such as the Peradeniya scale into the initial triage of OPC poisoning patients, particularly in resource-limited settings where rapid, low-cost risk

stratification can guide decisions on ICU admission and early ventilatory planning (14,16).

LIMITATIONS

This study has certain limitations. The sample available for detailed statistical analysis was small, limiting the power of some sub-group comparisons; the study population was restricted to patients referred to a single department, introducing possible selection bias; and resource and time constraints precluded a larger prospective cohort design. Despite these limitations, the direction and strength of the associations observed are consistent with a substantial body of published literature, lending confidence to the principal conclusions.

STRENGTHS OF THE STUDY

The strengths of this study include the combined assessment of clinical severity using the validated Peradeniya Organophosphorus Poisoning (POP) score and objective measurement of serum cholinesterase levels at admission. The study also evaluated clinically important outcomes, including mortality, requirement for mechanical ventilation, and duration of ICU stay. Measurement of serum cholinesterase before initiation of oxime therapy further strengthens the assessment of its relationship with initial poisoning severity and clinical outcome.

FUTURE SCOPE

Future studies should include larger, prospective, multicentre cohorts to further validate the prognostic value of admission serum cholinesterase levels and the Peradeniya Organophosphorus Poisoning (POP) score. Further research may also evaluate serial cholinesterase measurements, time from exposure to hospital presentation, atropine and pralidoxime requirements, and other clinical or biochemical predictors to develop more robust risk-stratification models for acute organophosphorus poisoning.

CONCLUSION

In this study of patients admitted with acute organophosphorus compound poisoning, a significantly increased severe-category Peradeniya score, a higher quantity of poison consumed, a markedly lower serum cholinesterase level, requirement of mechanical ventilation and a longer ICU stay were all significantly associated with death, while age, gender, occupation and the type of compound consumed were not. Serum cholinesterase level correlated strongly and inversely with clinical severity as graded by the Peradeniya score. These findings confirm that serum cholinesterase estimation, performed alongside a validated clinical severity score at first contact, is a simple, inexpensive and reliable tool to grade the severity of

organophosphorus poisoning and to predict the likely clinical outcome, and can assist clinicians in the early identification of high-risk patients who require intensive monitoring and ventilatory support.

DECLARATIONS

Ethics Approval and Consent to Participate

The study protocol was approved by the Institutional Ethics Committee of K. A. P. V. Government Medical College, Trichy. Written informed consent was obtained from all participants or their legally authorized representatives in English and Tamil before inclusion in the study.

Consent for Publication

Written informed consent for publication of study findings was obtained from the participants or their legally authorized representatives.

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The authors received no specific funding for this study.

Conflict of Interest

The authors declare that they have no conflict of interest.

Data Availability Statement:

The data generated and/or analysed during the current study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

Author Contributions

All authors contributed to the conception and design of the study, data collection and analysis, interpretation of findings, drafting and/or critical revision of the manuscript, and approved the final version for publication.

REFERENCES

1. Senanayake N, de Silva HJ, Karalliedde L. A scale to assess severity in organophosphorus intoxication: POP scale. *Hum Exp Toxicol*. 1993;12(4):297-9.
2. Barik D. Assessment of Peradeniya Organophosphorus Scale as a valuable tool in prognostication of acute organophosphorus poisoning. *J Evid Based Med Healthc*. 2018;5(48):2369-73.
3. Tripathi S. Prognostic value of Glasgow Coma Scale, poisoning severity score and serum acetylcholinesterase levels in organophosphorus poisoning. *J Evol Med Dent Sci*. 2014;3(13):3415-22.

4. Raikod BR, Saraf N, Kinhal SV. Predicting outcome and severity in acute organophosphorus poisoning with clinical scoring and serum cholinesterase levels. *J Evol Med Dent Sci*. 2014;3(60):13360-9.
5. Malaviya NB, Parikh R, Pancholi K, et al. Assessment of the Peradeniya Organophosphorus Poisoning Scale as a Severity and Prognostic Marker in Patients With Acute Organophosphorus Poisoning Presenting to an Emergency Medicine Department. *Cureus*. 2023;15(6):e40277.
6. Vernekar PV, Shivraj K. Peradeniya organophosphorus poisoning scale (POP) as a predictor of respiratory failure and mortality in organophosphorus poisoning. *Sch J Appl Med Sci*. 2017;5(5B):1841-4.
7. Ahuja H, Mathai AS, Pannu A, Arora R. Acute poisonings admitted to a tertiary level intensive care unit in northern India: patient profile and outcomes. *J Clin Diagn Res*. 2015;9(10):UC01-4.
8. Batra AK, Keoliya AN, Jadhav GU. Poisoning: an unnatural cause of morbidity and mortality in rural India. *J Assoc Physicians India*. 2003;51:955-9.
9. Lin TJ, Walter FG, Hung DZ, Tsai JL, Hu SC, Chang JS, et al. Epidemiology of organophosphate pesticide poisoning in Taiwan. *Clin Toxicol (Phila)*. 2008;46(9):794-801.
10. Persson HE, Sjöberg GK, Haines JA, Pronczuk de Garbino J. Poisoning severity score. Grading of acute poisoning. *J Toxicol Clin Toxicol*. 1998;36(3):205-13.
11. Karalliedde L. Organophosphorus poisoning and anaesthesia. *Anaesthesia*. 1999;54(11):1073-88.
12. Davies JOJ, Eddleston M, Buckley NA. Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. *QJM*. 2008;101(5):371-9.
13. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. *QJM*. 2000;93(11):715-31.
14. Pawar KS, Bhoite RR, Pillay CP, Chavan SC, Malshikare DS, Garad SG. Continuous pralidoxime infusion versus repeated bolus injection to treat organophosphorus pesticide poisoning: a randomised controlled trial. *Lancet*. 2006;368(9553):2136-41.
15. Sam KG, Kondabolu K, Pati D, Kamath A, Pradeep Kumar G, Rao PG. Poisoning severity score, APACHE II and GCS: effective clinical indices for estimating severity and predicting

outcome of acute organophosphorus and carbamate poisoning. *J Forensic Leg Med.* 2009;16(5):239-47.

16. Eddleston M, Buckley NA, Eyer P, Dawson AH. Management of acute organophosphorus pesticide poisoning. *Lancet.* 2008;371(9612):597-607.

17. World Health Organization. The WHO Recommended Classification of Pesticides by Hazard and Guidelines to Classification 2019. Geneva: WHO; 2020.

18. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. *BMC Public Health.* 2007;7:357.

19. Eddleston M, Karalliedde L, Buckley N, Fernando R, Hutchinson G, Isbister G, et al. Pesticide poisoning in the developing world - a minimum pesticides list. *Lancet.* 2002;360(9340):1163-7.

20. Karki P, Ansari JA, Bhandary S, Koirala S. Cardiac and electrocardiographical manifestations of acute organophosphate poisoning. *Singapore Med J.* 2004;45(8):385-9.