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Effect of Alcohol Co-Ingestion on Clinical Severity and Outcomes in Acute Organophosphorus Poisoning: An Observational Cohort Study.

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Abstract

Background:
Organophosphorus (OP) compound poisoning continues to impose a substantial health burden in many developing regions where pesticide access is high and timely medical care is inconsistent.

Aim:
To compare the clinical severity and outcomes of OP poisoning among adults with and without alcohol co-ingestion.

Methods:
This hospital-based observational cohort study included 120 adults presenting with acute OP poisoning. Participants were classified into Group A (OP poisoning with alcohol co-ingestion; n = 48) and Group B (OP poisoning without alcohol; n = 72). Severity at admission was quantified using the Peradeniya Organophosphorus Poisoning Scale (POPS). Key outcomes assessed were ventilator requirement, major complications such as aspiration pneumonia, duration of hospitalization, and mortality.

Results:
The cohort had a mean age of 34.2 ± 10.6 years, and 74.1% were men. Patients with alcohol co-ingestion demonstrated higher clinical severity (mean POPS: 4.9 ± 1.8 vs. 3.8 ± 1.6; p = 0.002). The need for ventilatory support was almost twice as frequent in Group A (41.7% vs. 22.2%; p = 0.02). Aspiration pneumonia occurred more often among those who consumed alcohol (25% vs. 11.1%; p = 0.04). Hospital stay was significantly longer in the co-ingestion group (8.7 ± 3.6 vs. 6.4 ± 2.9 days; p = 0.01). Mortality was also higher (16.7% vs. 6.9%; p = 0.04).

Conclusion:
Alcohol co-ingestion intensifies the clinical severity of OP poisoning and increases the likelihood of complications, prolonged hospitalization, and death. Early identification of alcohol intake is essential to guide risk stratification and ensure timely, aggressive supportive care.

Recommendations:
Clinicians should routinely assess alcohol use in all suspected OP poisoning cases and classify such patients as high-risk at presentation. Early airway protection, rapid initiation of antidotes, and close hemodynamic and respiratory monitoring are advised.

Keywords: *Organophosphorus poisoning, alcohol co-ingestion, Peradeniya Organophosphorus Poisoning Scale (POPS), ventilator requirement, cholinesterase inhibition, aspiration pneumonia, mortality.*
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Introduction
Organophosphorus (OP) compounds are among the most widely used pesticides globally, particularly in agricultural regions of low- and middle-income countries. Owing to their easy availability and high toxicity, OP compounds constitute a major cause of deliberate self-

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harm and poisoning-related mortality in South and Southeast Asia [1,2]. OP toxicity occurs primarily through irreversible inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at synapses and neuromuscular junctions. This results in excessive cholinergic stimulation manifesting as muscarinic, nicotinic, and central nervous system effects [3].

Alcohol consumption represents another major public health concern in the same regions where OP poisoning is highly prevalent. Both acute and chronic alcohol intake can significantly influence the pharmacokinetics of toxic substances by altering hepatic metabolism, impairing immune function, and exerting direct toxic effects on multiple organ systems [4]. In cases of deliberate self-harm, co-ingestion of alcohol with OP compounds is frequently observed, either due to habitual alcohol use or intentional intake prior to pesticide consumption.

The interaction between alcohol and OP compounds within the human body is complex and not fully understood. Ethanol has been reported to enhance gastrointestinal absorption and increase the lipid solubility of OP compounds, potentially intensifying toxicity [5]. Conversely, some evidence suggests that acute ethanol exposure may competitively inhibit the metabolic activation of certain OP agents, thereby exerting a paradoxical protective effect. Clinical outcomes among patients with combined OP and alcohol ingestion remain inconsistent across studies. While some reports indicate a worse prognosis characterized by increased respiratory failure, prolonged hospitalization, and higher mortality, others have found no significant difference in outcomes [6].

Given these conflicting findings, evaluating the impact of alcohol co-ingestion on the clinical course and outcome of OP poisoning is crucial for early risk stratification and optimized management. The present study was therefore designed to compare the clinical profile and outcomes of patients presenting with OP poisoning with and without alcohol co-ingestion.

Material and Methods

Study Design and Setting

This hospital-based prospective observational study was conducted at Konaseema Institute of Medical Sciences and Research Foundation (KIMS & RF), Amalapuram, Andhra Pradesh, India. KIMS & RF is a tertiary care teaching hospital affiliated with Dr. YSR University of Health Sciences and serves as a referral center for coastal Andhra Pradesh, catering to both urban and rural populations. The institution has a fully equipped emergency department, intensive care unit, and toxicology management facilities. The study was carried out over a 12-month period from January 2024 to December 2024.

Study Population

All consecutive adult patients (≥18 years) presenting to the emergency department with a clinical diagnosis of organophosphorus (OP) poisoning during the study period were screened for eligibility. Diagnosis was based on history of exposure, characteristic clinical features (miosis, salivation, bronchorrhea, fasciculations), and supportive laboratory findings where available.

Participants were selected using a consecutive sampling method to minimize selection bias. Patients were divided into two groups based on a history of alcohol co-ingestion at the time of poisoning. History was obtained from the patient, relatives, or accompanying persons. Patients with mixed poisoning (other than alcohol), chronic liver disease, or incomplete records were excluded.

Bias

Efforts were made to minimize potential sources of bias. Selection bias was reduced by enrolling consecutive eligible patients during the defined study period. Information bias was limited by using standardized clinical assessment protocols and uniform criteria for diagnosing organophosphorus poisoning. Severity assessment was performed using the Peradeniya Organophosphorus Poisoning Scale (POPS), applied uniformly at admission by trained clinicians. Data were recorded prospectively in a structured proforma to reduce recall errors and incomplete documentation.

Confounding due to differences in baseline characteristics was addressed through appropriate statistical comparison between groups.

Study Size

The sample size of 120 participants was determined based on the average number of organophosphorus poisoning cases admitted annually to the institution, which is approximately 110–130 cases per year. Considering the defined study duration of 12 months and applying inclusion and exclusion criteria, all eligible cases presenting during the study period were included. Therefore, the study employed a census-based sampling approach rather than a priori sample size calculation.

Inclusion criteria:

Patients with documented ingestion of OP compounds within the last 24 hours.

Age ≥18 years.

Willingness to provide informed consent (self or relative).

Exclusion criteria:

Mixed poisoning with agents other than alcohol.

Chronic alcohol dependence without acute alcohol ingestion.

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Patients with significant comorbidities such as chronic liver disease, chronic kidney disease, or pre-existing neurological disorders could confound outcome assessment.

Grouping of Patients

Based on the history of alcohol consumption at the time of poisoning, patients were divided into two groups:

Group A (Co-ingestion group): Patients with OP poisoning who had co-ingested alcohol within 6 hours of OP intake.

Group B (Non-alcohol group): Patients with OP poisoning without alcohol co-ingestion.

The history of alcohol ingestion was obtained from the patient or reliable informants and corroborated by clinical evidence of acute intoxication (odor of alcohol, altered sensorium, or breath alcohol analysis where available).

Data Collection

A structured proforma was used to record demographic details, amount and type of OP compound ingested, alcohol intake history, clinical features at presentation, vital parameters, and laboratory investigations.

Baseline investigations: Complete blood count, renal and liver function tests, serum electrolytes, random blood glucose, and arterial blood gases.

Toxicological investigations: Plasma pseudocholinesterase levels were measured at admission and repeated after 24–48 hours, wherever feasible.

Severity Assessment: The severity of OP poisoning was graded using the Peradeniya Organophosphorus Poisoning Scale (POPS) at admission.

Clinical outcome parameters: Need for atropine and oxime therapy, requirement and duration of mechanical ventilation, length of hospital stay, complications (respiratory failure, intermediate syndrome, aspiration pneumonia, seizures), and in-hospital mortality.

Treatment Protocol

All patients received standard treatment according to WHO guidelines for the management of OP poisoning, including airway management, gastric decontamination (if within 1 hour of ingestion), intravenous atropine titrated to response, and oxime therapy with pralidoxime. Supportive management was provided as required.

Statistical Analysis

Data were analyzed using SPSS software version 13. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range), while categorical variables were expressed as frequencies and percentages. Comparisons between groups were made using the Student's t-test. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee, Konaseema Institute of Medical Sciences, Amalapuram, Andhra Pradesh, India. All procedures were performed in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration.

Results

Participants

During the study period (January 2024 to December 2024), a total of 138 patients with suspected organophosphorus poisoning presented to the emergency department. Of these, 130 were assessed for eligibility. Eight patients were excluded at the initial stage due to mixed poisoning with substances other than alcohol (n = 5) or incomplete clinical documentation (n = 3).

Among the 130 eligible patients, 10 were excluded due to pre-existing chronic liver disease (n = 4) or refusal of consent by relatives (n = 6). Finally, 120 patients fulfilled the inclusion criteria and were enrolled in the study. All enrolled participants completed in-hospital follow-up and were included in the final analysis (n = 120).

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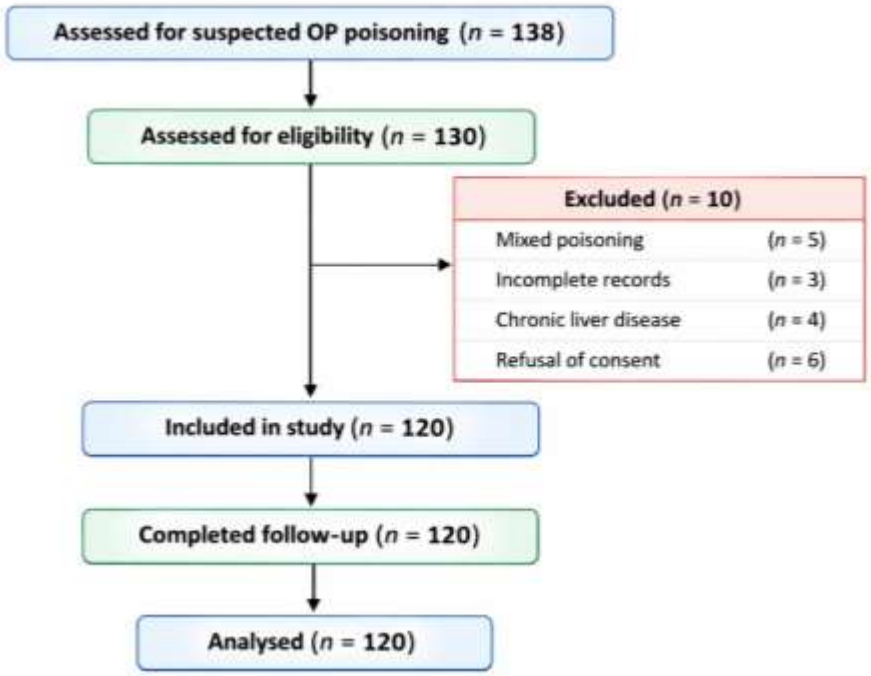

Figure 1: Participant Flow Diagram

Baseline Characteristics

A total of 120 patients with acute OP poisoning were enrolled during the study period. Of these, 48 patients (40%) had co-ingested alcohol (Group A), and 72 patients (60%) had consumed OP compounds alone (Group B).

The mean age of the study population was 34.2 ± 10.6 years (range: 18–65 years), with a male predominance (74.1%). The mean quantity of OP compound ingested did not differ significantly between the two groups (p = 0.42). Baseline demographic and clinical features are summarized in Table 1.

Table 1. Baseline characteristics of patients

Variable	Group A (OP + Alcohol) n=48	Group B (OP only) n=72	*p* value
Mean age (years)	35.6 ± 9.8	33.3 ± 11.1	0.28
Male: Female ratio	3.5: 1	2.1: 1	0.04*
Mean quantity of OP ingested (ml)	42.5 ± 15.3	39.7 ± 14.8	0.42
Time to hospital arrival (hrs)	3.6 ± 1.8	3.2 ± 1.6	0.31
Baseline pseudocholinesterase (U/L)	1800 ± 620	2100 ± 590	0.08

**Statistically significant*

Severity of Poisoning

The mean Peradeniya Organophosphorus Poisoning Scale (POPS) score was significantly higher in the co-ingestion group (4.9 ± 1.8) compared with the non-alcohol group (3.8 ± 1.6; p = 0.002). Patients in Group A demonstrated a significantly higher incidence of central nervous system (CNS) depression and respiratory distress at presentation compared to Group B. CNS depression was observed in 38.3% of patients in Group A versus 20.8% in Group B ($\chi^2 = 4.87$, p = 0.027). Similarly, respiratory distress was more frequent among patients with alcohol co-ingestion (41.7% vs. 23.6%; $\chi^2 = 5.12$, p = 0.023).

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Regarding overall severity, the mean Peradeniya Organophosphorus Poisoning Scale (POPS) score was significantly higher in Group A (4.9 ± 1.8) compared to Group B (3.8 ± 1.6) (independent t-test = 3.12, p = 0.002), indicating more severe clinical presentation among those with alcohol co-ingestion.

Treatment and Complications

The need for atropine and oxime therapy was similar in both groups; however, ventilator requirement was significantly higher in Group A (41.7% vs. 22.2%; p = 0.02). Patients with alcohol co-ingestion also had a higher incidence of aspiration pneumonia and intermediate syndrome (Table 2).

Table 2. Clinical course and complications

Parameter	Group A (OP + Alcohol)	Group B (OP only)	*p* value
Mean POPS score	4.9 ± 1.8	3.8 ± 1.6	0.002*
Atropine dose (mg)	62.5 ± 18.9	59.7 ± 20.4	0.41
Oxime therapy received (%)	93.7%	95.8%	0.64
Ventilator requirement (%)	41.7%	22.2%	0.02*
Duration of ventilation (days)	4.8 ± 2.1	3.1 ± 1.4	0.01*
Aspiration pneumonia (%)	25%	11.1%	0.04*
Intermediate syndrome (%)	14.6%	6.9%	0.18

Outcome: -

The mean hospital stay was significantly longer in Group A (8.7 ± 3.6 days) compared with Group B (6.4 ± 2.9 days; p = 0.01). Overall mortality was higher in the alcohol co-ingestion group (16.7% vs. 6.9%), which was statistically significant (p = 0.04).

Table 3. Outcome of patients

Outcome	Group A (OP + Alcohol)	Group B (OP only)	*p* value
Mean hospital stay (days)	8.7 ± 3.6	6.4 ± 2.9	0.01*
Mortality (%)	16.7%	6.9%	0.04*

Discussion

In this observational study, clinical outcomes were compared among patients with acute organophosphorus (OP) poisoning with and without alcohol co-ingestion. Alcohol co-ingestion was associated with a more severe clinical course. Patients in the co-ingestion group had higher poisoning severity scores (mean POPS 4.9 ± 1.8 vs. 3.8 ± 1.6; p = 0.002) and presented more frequently with CNS depression (38.3% vs. 20.8%; $\chi^2 \approx 4.01$, $p \approx 0.045$) and respiratory distress (41.7% vs. 23.6%; $\chi^2 \approx 4.40$, $p \approx 0.036$). Clinically relevant outcome differences were also observed: ventilator requirement was higher with alcohol co-ingestion (41.7% vs. 22.2%; p = 0.02), aspiration pneumonia occurred more often (25.0% vs. 11.1%; p = 0.04), hospital stay was longer (8.7 ± 3.6 vs. 6.4 ± 2.9 days; p = 0.01), and mortality was higher (16.7% vs. 6.9%; p = 0.04).

These findings require cautious interpretation. Given the observational design, the associations do not establish causality, and unmeasured factors—such as the quantity of alcohol consumed, time since ingestion, baseline comorbidities, and variability in the specific OP compound—may have contributed to the observed differences. Nevertheless, the consistent direction of effect across severity measures, respiratory compromise, complications, and hard outcomes suggests that alcohol co-ingestion may identify a higher-risk subgroup warranting early intensive monitoring.

The pattern of increased severity with alcohol co-ingestion is biologically plausible and has been described in earlier clinical work. Acute alcohol exposure can depress consciousness, impair airway reflexes, and increase aspiration risk, thereby contributing to respiratory failure, a key driver of morbidity in OP poisoning [3,7]. Alcohol may also enhance systemic absorption of lipophilic OP compounds through effects on gastrointestinal permeability and solubility, potentially increasing toxic burden [6]. The higher frequency of respiratory distress at presentation and greater need for mechanical ventilation are consistent with this mechanism [8,9].

The higher frequency of aspiration pneumonia in the co-ingestion group further supports a respiratory-complication pathway (25.0% vs. 11.1%; p = 0.04). Prior studies similarly report that alcohol co-ingestion predisposes to early respiratory depression and prolonged

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ventilator dependence [10,11]. Although intermediate syndrome was numerically higher in the co-ingestion group (14.6% vs. 6.9%), this difference was not statistically significant (p = 0.18), suggesting that not all complications differed consistently in this sample.

Earlier experimental suggestions of a potential "protective" effect of alcohol through inhibition of metabolic activation of certain OP compounds have not been consistently supported in clinical settings [12]. Subsequent evidence indicates that any theoretical biochemical interaction may be outweighed by the adverse physiological effects of intoxication, including altered sensorium, aspiration risk, delayed recognition, and delays in timely treatment [13,14]. The observed higher severity at presentation and worse outcomes among alcohol co-ingesters are aligned with these contemporary clinical observations.

Mortality was significantly higher among patients with alcohol co-ingestion (16.7% vs. 6.9%; p = 0.04), consistent with prior studies reporting increased fatality in the presence of alcohol [15,16]. A plausible explanation is that alcohol may blunt early symptom recognition and delay escalation of atropinization and respiratory support in rapidly deteriorating patients. Collectively, these findings support closer early respiratory monitoring and lower thresholds for ICU triage when alcohol co-ingestion is suspected in OP poisoning.

Generalizability

The findings of this hospital-based observational study are applicable to similar tertiary care settings managing acute organophosphorus poisoning, particularly in regions where alcohol consumption and pesticide availability are common. The results may be generalizable to adult populations presenting with intentional OP ingestion in low- and middle-income countries. However, caution is required when extrapolating these outcomes to pediatric populations, rural primary care facilities, or settings with different patterns of pesticide use and emergency care infrastructure.

Conclusion

Alcohol co-ingestion in cases of organophosphorus poisoning is associated with a markedly more severe clinical course when compared with OP poisoning alone. Patients with concurrent alcohol intake exhibit higher poisoning severity, increased need for ventilatory support, greater incidence of complications, prolonged hospitalization, and significantly higher mortality. These findings underscore the adverse impact of alcohol on the physiological response to OP toxicity. Alcohol consumption should therefore be recognized as a key prognostic factor in patients presenting with OP poisoning. Early identification of alcohol co-ingestion at the time of admission can facilitate timely risk stratification, prompt initiation of intensive supportive measures, and more accurate prediction of clinical outcomes, ultimately contributing to improved patient management and survival.

Strengths and Limitations

The strengths of this study include prospective data collection, use of an objective poisoning severity score, and comprehensive assessment of clinically relevant outcomes. Nevertheless, certain limitations must be acknowledged. Alcohol intake was partially based on patient history and attendant reporting, which may be subject to recall bias. The single-center design limits generalizability, and heterogeneity in the types of OP compounds ingested could have influenced toxicity patterns, although subgroup analysis was not feasible due to sample size constraints.

Recommendations

Routine assessment for alcohol co-ingestion should be incorporated into the initial evaluation of all patients presenting with organophosphorus poisoning. Early identification of alcohol intake can aid in prompt risk stratification and guide decisions regarding intensive monitoring and ventilatory support. Patients with suspected or confirmed alcohol co-ingestion should be managed with heightened vigilance, including early airway protection, aggressive supportive care, and close observation for respiratory and neurological complications. Standard treatment protocols may be strengthened by integrating alcohol history as a prognostic indicator. Further multicentric studies with larger sample sizes are recommended to explore dose–response relationships, biochemical interactions, and to develop validated risk prediction models for improved clinical decision-making.

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Abbreviations

OP – Organophosphorus
POPS – Peradeniya Organophosphorus Poisoning Scale
CNS – Central Nervous System

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ICU – Intensive Care Unit
MV – Mechanical Ventilation
GCS – Glasgow Coma Scale

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Conflict of interest
The authors declare no conflict of interest.

Author contributions
AA-Concept and design of the study, results interpretation, review of literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript.**TJ**-Concept and design of the study, results interpretation, review of literature, preparing the first draft of the manuscript, and revision of the manuscript. **VVKO-**Review of literature and preparing the first draft of the manuscript. Statistical analysis and interpretation.

Data availability
Data available on request

Author Biography
Dr. Anand Acharya, MBBS, MD (Pharmacology), currently serves as Dean and Professor, Department of Pharmacology, at the Konaseema Institute of Medical Sciences & Research Foundation (KIMS&RF), Amalapuram, Andhra Pradesh, India. A distinguished academician, researcher, and medical education leader, he has been pivotal in transforming KIMS&RF from its formative phase into a premier medical institution with over 200 undergraduate and 100 postgraduate seats.

With more than 18 years of teaching and administrative experience, Dr. Acharya has held several leadership positions, including Vice Principal, Principal, Chief Warden, Member Secretary of Institutional Ethics and Animal Ethics Committees, and is an approved PhD Guide under Dr. NTR University of Health Sciences, Vijayawada. His visionary leadership has significantly enhanced the institution's academic quality, clinical exposure, research infrastructure, and postgraduate training standards.

He has successfully completed prestigious national faculty development programs such as the Revised Basic Course Workshop (rBCW), Advanced Course in Medical Education (ACME), and National Teacher Training Course (NTTC, JIPMER, Puducherry). He also serves as Coordinator for Pharmacovigilance and Materiovigilance Programs under IPC–PvPI and MoHFW, Government of India, contributing actively to national drug safety and regulatory initiatives.

A prolific academician, Dr. Acharya has authored and co-authored more than 100 scientific publications in reputed national and international indexed journals. His wide-ranging research covers toxicology, pharmacovigilance, antimicrobial resistance, endocrinology, neuropharmacology, and clinical pharmacology. His recent studies include long-term analyses of pyrethroid, paraquat, and chlorpyrifos poisoning, investigations into antimicrobial resistance trends, and predictive models for treatment outcomes in dermatological and toxicological emergencies.

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