Journal of Medical and Health Studies (JMHS)
ISSN: 2710-1452
DOI: 10.32996/jmhs
Journal Homepage: www.al-kindipublisher.com/index.php/jmhs

JMHS
AL-KINDI CENTER FOR RESEARCH AND DEVELOPMENT

| RESEARCH ARTICLE

Admission Blood Glucose as a Predictor of Mortality and Severe Outcomes in Acute Organophosphorus Poisoning: A Systematic Review and Meta-Analysis

Saeed Ali Alzahrani[1], Hesham Zuhair Ajaj[2], Duaa Mubarak Abu Salim[3], Abdulrahman Mustafa Toonisi[3]✉, Yasir Mousa Alhusayni[3], Fatma Mustafa Toonisi[4]

[1]Senior Registrar & Deputy Director of The Forensic Medicine Training Program, Forensic Medicine Services Center, Ministry of Health, Riyadh, Saudi Arabia
[2]Consultant of Emergency Medicine, Department of Emergency Medicine, King Fahad Armed Forces Hospital, Jeddah, Saudi Arabia
3Medical Intern, Faculty of Medicine, University of Tabuk, Tabuk, Saudi Arabia
[4]Medical Student, Faculty of Medicine, University of Tabuk, Tabuk, Saudi Arabia
Corresponding Author: Abdulrahman Mustafa Toonisi, **E-mail**: dr.toonisi@gmail.com

| ABSTRACT

Acute organophosphorus poisoning remains a major toxicologic emergency and cause of morbidity and mortality, especially in agricultural and resource-limited settings. Early identification of patients at risk of deterioration is essential. Admission blood glucose has been proposed as a simple prognostic marker, but the evidence has not been synthesized in a focused review. This study aims to evaluate the association between admission blood glucose and mortality and severe clinical outcomes in acute organophosphorus poisoning. A systematic review and meta-analysis was conducted using PubMed and a supplementary Google Scholar search. PubMed yielded 157 records, and the first 200 Google Scholar records sorted by relevance were also screened. After title and abstract screening, full-text review, and exclusion of ineligible reports, 5 observational studies were included in the qualitative synthesis and 3 studies in the quantitative synthesis. The primary outcome was in-hospital mortality. Secondary outcomes included mechanical ventilation or respiratory failure and poisoning severity. Odds ratios (ORs) with 95% confidence intervals (CIs) were pooled using the Mantel-Haenszel random-effects model. Results from the included studies showed that elevated admission glucose was associated with significantly increased odds of mortality (OR 5.68, 95% CI 3.55-9.07; I^2 = 0%) and mechanical ventilation or respiratory failure (OR 4.46, 95% CI 3.02-6.59; I^2 = 0%). Narrative synthesis of the remaining studies also supported the association between higher admission glucose and greater poisoning severity. These findings point towards admission glucose being a simple and clinically useful early prognostic marker, although the available evidence is limited by observational design and variation in glycemic cut-offs.

| KEYWORDS

Acute organophosphorus poisoning; hyperglycemia; blood glucose; glycemic status; mortality; mechanical ventilation; systematic review; meta-analysis

| ARTICLE INFORMATION

ACCEPTED: 21 March 2026 **PUBLISHED:** 09 April 2026 **DOI:** 10.32996/jmhs.2026.7.6.5

1. Introduction

Acute organophosphorus poisoning is a major global toxicologic problem and continues to account for substantial morbidity and mortality, particularly in rural and agricultural regions of low- and middle-income countries (Buckley et al., 2005; Gunnell et al., 2007; Rambabu et al., 2019). Although the clinical syndrome is primarily driven by acetylcholinesterase inhibition and cholinergic excess, case fatality remains considerable despite treatment with atropine, oximes, and supportive care (Buckley et al., 2005; Eddleston et al., 2008; Moon et al., 2016). Respiratory failure is one of the main determinants of poor outcome in acute organophosphorus poisoning. It may occur early because of central respiratory depression, bronchorrhea, bronchospasm, and respiratory muscle weakness, or later as part of intermediate syndrome and delayed neuromuscular dysfunction (Karalliedde, 1999; Giyanwani et al., 2017). Early recognition of patients at high risk of respiratory deterioration remains central to

management. A number of prognostic indicators have been explored in organophosphorus poisoning, including clinical severity scores, cholinesterase levels, acid-base abnormalities, electrocardiographic changes, and intensive care scoring systems (Senanayake et al., 1993; Davies et al., 2008; Gündüz et al., 2015; Sagah & Elhawary, 2021). Some of these measures require more complex assessment, are not uniformly available, or show inconsistent predictive value across settings. Blood glucose is different. It is inexpensive, rapidly measurable, and routinely available, even in low-resource hospitals. Disturbance of glucose homeostasis after organophosphorus exposure has been described in both experimental and clinical studies. Proposed mechanisms include catecholamine excess secondary to sustained cholinergic stimulation, increased ACTH release, enhanced glycogenolysis and gluconeogenesis, oxidative stress, and pancreatic dysfunction (Panda et al., 2015; Rambabu et al., 2019; Bhat et al., 2021). Clinical studies have reported hyperglycemia, hypoglycemia, glycosuria, and, less commonly, diabetic ketoacidosis in patients with acute poisoning (Panda et al., 2015; Sagah & Elhawary, 2021; Bhat et al., 2021). Several observational studies have suggested that elevated admission glucose or abnormal glycemic status is associated with increased poisoning severity, need for mechanical ventilation, and mortality in acute organophosphorus poisoning (Moon et al., 2016; Gündüz et al., 2015; Sagah & Elhawary, 2021; Bhat et al., 2021). The evidence is scattered, glucose thresholds vary across studies, and no focused synthesis has clarified the consistency and magnitude of this association. This study aimed to systematically review and quantitatively synthesize the available evidence on the prognostic value of admission blood glucose or glycemic status in acute organophosphorus poisoning.

2. Methodology

This study was conducted as a systematic review and meta-analysis of observational studies evaluating the association between admission blood glucose or glycemic status and adverse outcomes in acute organophosphorus poisoning. The review was prepared in accordance with the PRISMA 2020 statement. Search reporting was informed by PRISMA-S recommendations (Page et al., 2021; Rethlefsen et al., 2021).

2.1. Information Source and Search Strategy

A systematic literature search was performed in PubMed and supplemented by Google Scholar. PubMed was searched using a predefined strategy combining terms related to organophosphorus poisoning and blood glucose or glycemic status. This search yielded 157 records. To improve sensitivity and identify potentially relevant studies not captured through PubMed indexing, the first 200 Google Scholar records sorted by relevance were also screened. Reference lists of potentially eligible studies were reviewed manually to identify additional relevant reports.

2.2 Eligibility Criteria

Studies were eligible if they included adult patients with acute organophosphorus or organophosphate poisoning, measured admission blood glucose, random blood sugar, random plasma glucose, or glycemic status at presentation or on admission, reported at least one clinically relevant outcome, and used an observational design. Studies were excluded if they were case reports, case series, review article, animal studies, if they involved chronic or occupational exposure rather than acute poisoning, mixed intoxications without separable organophosphorus data, non-organophosphorus toxicants, conference proceedings or abstract-only publications, or exposures not centered on admission glucose or glycemic status.

2.3 Study Selection

Titles and abstracts were screened first, followed by full-text review of potentially eligible studies. From the PubMed search, 13 records were judged potentially relevant at title and abstract screening. Screening of the first 200 Google Scholar records identified 29 additional potentially relevant reports. After removal of duplicate and clearly ineligible records, 15 full-text articles were assessed for eligibility. Ten articles were excluded because of wrong exposure definition, wrong population, wrong toxic agent, mixed anticholinesterase exposure not separable to organophosphorus poisoning, conference-only publication, or insufficient alignment with the review question. Five studies were included in the final qualitative synthesis. Disagreements in study selection and data extraction were resolved by discussion and review of the full text.

2.4 Data Extraction

Data were extracted into a standardized spreadsheet in Microsoft Excel. Extracted variables included first author, year, country, study design, sample size, patient characteristics, exclusion of diabetes mellitus, admission glucose metric, glucose cut-off values or exposure categories, and reported outcomes. For studies eligible for quantitative synthesis, raw event data were extracted to construct 2 × 2 tables for mortality and mechanical ventilation or respiratory failure.

2.5 Outcomes

The primary outcome was in-hospital mortality. Secondary outcomes included mechanical ventilation or respiratory failure, poisoning severity, and markers of severe clinical course where reported.

2.6 Risk of Bias Assessment

Risk of bias was assessed according to study design. Retrospective cohort or case-series type studies were appraised using domains derived from the Newcastle-Ottawa Scale, whereas prospective observational and analytical cross-sectional studies were assessed using the Joanna Briggs Institute critical appraisal framework (Wells et al., n.d.; Moola et al., 2020). Overall judgments were categorized as low, low-to-moderate, moderate, or high risk of bias.

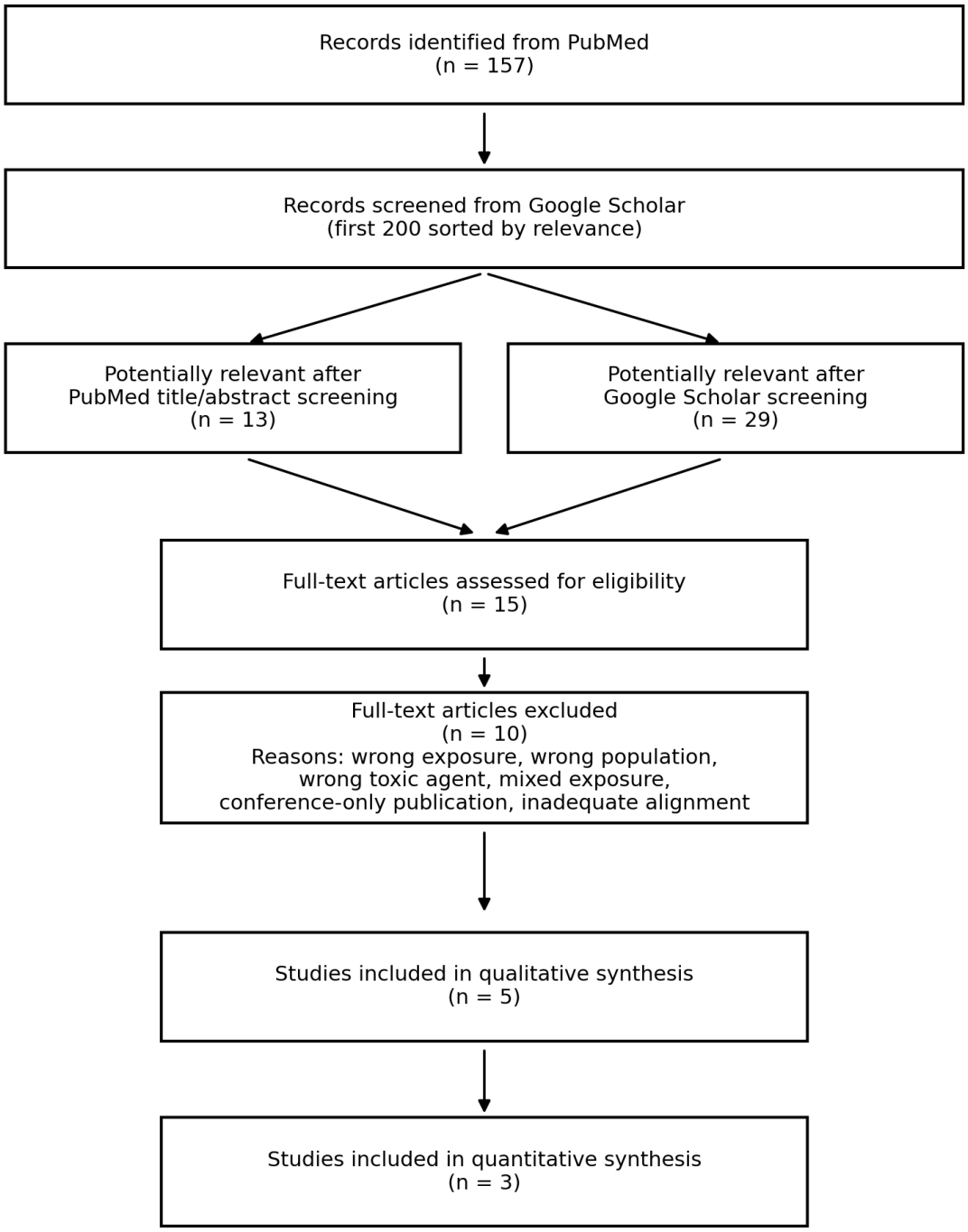
2.7 Statistical Analysis

A meta-analysis was undertaken only when at least three studies reported sufficiently comparable categorical data for the same outcome. Because the included studies used different definitions of hyperglycemia and different glucose categorizations, the primary quantitative synthesis was restricted to studies reporting categorical hyperglycemia or high admission glucose versus lower or reference glucose. Studies reporting only continuous glucose effects or non-comparable severity-only data were synthesized narratively. Meta-analysis was performed using standard Mantel-Haenszel methods under a random-effects model, reproducing the same analytical framework commonly used in Review Manager (RevMan) for dichotomous outcomes. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for dichotomous outcomes. A random-effects model was selected a priori because of the anticipated clinical and methodological heterogeneity across studies, including differences in glucose cut-offs, study design, and patient populations. Statistical heterogeneity was assessed using the Chi-square test and quantified with the I^2 statistic. Publication bias was not formally assessed because fewer than 10 studies were included in the quantitative synthesis. Because the included studies used different thresholds for hyperglycemia, the quantitative synthesis pooled the closest reported categorical high-glucose versus lower or reference comparison from each eligible study. No protocol was registered.

3. Results

The search identified 157 records from PubMed. A supplementary Google Scholar search was then performed, and the first 200 records sorted by relevance were screened. After title and abstract screening, 13 PubMed records and 29 Google Scholar records were considered potentially relevant. Duplicate and clearly irrelevant records were removed. Fifteen full-text articles were assessed for eligibility. Ten full-text articles were excluded for reasons related to exposure, population, toxic agent, publication type, or lack of alignment with the review question. Five studies were included in the qualitative synthesis. Three studies were included in the quantitative synthesis (Figure 1).

Figure 1. PRISMA flow diagram of study selection



3.1 Study Characteristics

The review included five observational studies published between 2015 and 2021. They were conducted in South Korea, Turkey, Egypt, and India. Sample size ranged from 90 to 400 participants. Two studies used a retrospective design. Three were prospective. Admission glycemic status was reported as venous glucose, blood glucose, random blood sugar, or random plasma glucose measured at presentation. The definition of hyperglycemia was not uniform across studies. Two studies used >140 mg/dL, one used ≥200 mg/dL, one used four glucose strata, and one treated glucose as a continuous predictor. The main outcomes were mortality, mechanical ventilation or respiratory failure, and poisoning severity (Table 1).

Table 1. Characteristics of studies included in the systematic review and meta-analysis.

Study	Country	Design	N	Diabetes excluded	Glucose measure	Glucose definition	Main outcomes	Role in synthesis
Moon et al. (2016)	South Korea	Retrospective observational	184	Yes	Initial venous glucose	<140, 140-200, 200-300, ≥300 mg/dL	Mortality; respiratory failure; ICU stay; MV duration	Quantitative
Panda et al. (2015)	India	Prospective observational	102	Yes	Random plasma glucose	Hyperglycemia >140 mg/dL; POP severity groups	Severity; atropine requirement; cholinesterase; glycosuria	Narrative
Gündüz et al. (2015)	Turkey	Retrospective cohort	296	Not clearly stated	Blood glucose at presentation	Continuous	Mortality	Narrative
Sagah & Elhawary (2021)	Egypt	Prospective cross-sectional	90	Yes	Random blood sugar	<70, 70-140, >140 mg/dL	Mortality; ventilation; severity	Quantitative
Bhat et al. (2021)	India	Prospective observational	400	Yes	Random plasma glucose	<70, 70-199, ≥200 mg/dL	Mortality; ventilation; HDU; severity	Quantitative

3.2 Risk of Bias Within Studies

Overall, the included studies were judged to be at low-to-moderate to moderate risk of bias. The main concerns were observational design, single-center recruitment, and limited adjustment for confounding. The strongest studies were those by Sagah and Elhawary (2021) and Bhat et al. (2021) because glucose was measured on admission before treatment and the outcome data were clearly reported. Moon et al. (2016) and Gündüz et al. (2015) were limited by retrospective design. Panda et al. (2015) was useful for severity analysis, but not for the primary mortality synthesis because deaths after admission were excluded from the analyzed cohort (Table 2).

Table 2. Risk of bias assessment of the included studies.

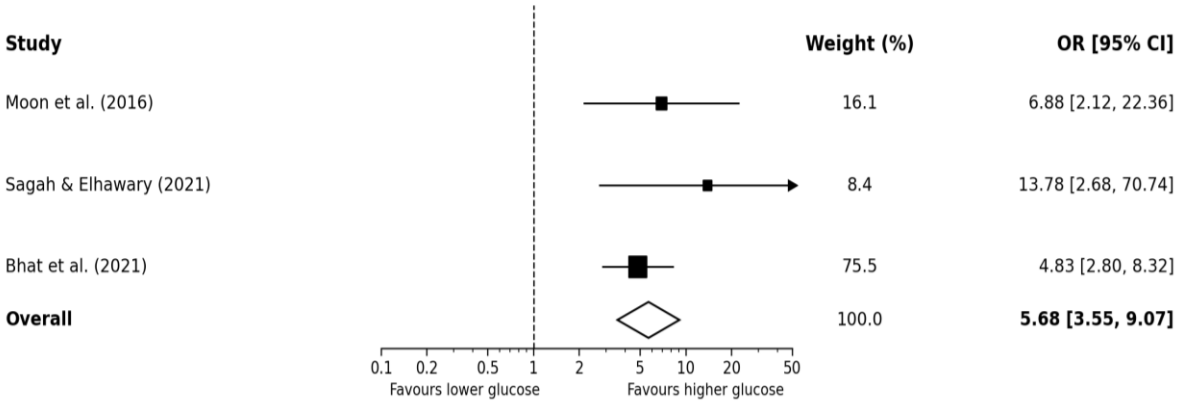
Study	Design	Key strengths	Main limitations	Overall judgment
Moon et al. (2016)	Retrospective	Glucose measured before treatment; clear mortality outcome; multivariable analysis	Single-center retrospective design; limited generalizability	Moderate risk of bias
Panda et al. (2015)	Prospective	Prospective design; non-diabetic cohort; pre-treatment glucose measurement	Deaths after admission excluded from analytic cohort	Moderate risk of bias

Study	Design	Key strengths	Main limitations	Overall judgment
Gündüz et al. (2015)	Retrospective	Large cohort; mortality clearly reported; logistic regression	Retrospective design; diabetes handling unclear; no poolable glucose categories	Moderate risk of bias
Sagah & Elhawary (2021)	Prospective	Clear eligibility criteria; raw outcome data; glucose measured before medication	Single-center study; limited confounder adjustment	Low-to-moderate risk of bias
Bhat et al. (2021)	Prospective	Large sample; clear glycemic definitions; detailed outcomes	Observational design; limited adjustment for confounding	Low-to-moderate risk of bias

3.3 Admission Blood Glucose and Mortality

Three studies contributed data to the mortality meta-analysis. All three showed the same direction of effect. Higher admission glucose was linked to a higher risk of death. In Moon et al. (2016), mortality rose across glucose strata, from 3.2% in patients with glucose <140 mg/dL to 27.3% in those with glucose ≥300 mg/dL. In Sagah and Elhawary (2021), 8 of 26 hyperglycemic patients died, compared with 2 of 64 non-hyperglycemic patients. In Bhat et al. (2021), mortality was 35% in the hyperglycemic group and 9.9% in the non-hyperglycemic group. The pooled analysis showed that elevated admission glucose was associated with significantly higher odds of mortality (OR 5.68, 95% CI 3.55-9.07). Heterogeneity was not observed (Chi^2 = 1.56, df = 2, p = 0.46; I^2 = 0%) (Table 3, Figure 2).

Figure 2. Forest plot of the association between elevated admission glucose and mortality in acute organophosphorus poisoning.



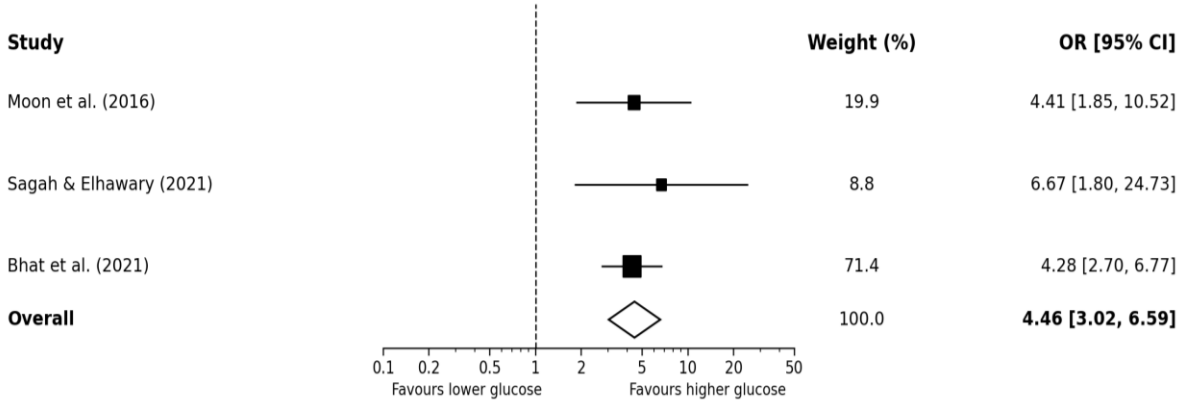
3.4 Admission Blood Glucose and Mechanical Ventillation or Respiratory Failure

In Moon et al. (2016), respiratory failure became more frequent as glucose increased and occurred in 100% of patients with admission glucose ≥300 mg/dL. In Sagah and Elhawary (2021), mechanical ventilation was required in 8 of 26 hyperglycemic patients and 4 of 64 non-hyperglycemic patients. In Bhat et al. (2021), ventilatory support was needed in 80 of 118 hyperglycemic patients and 93 of 282 non-hyperglycemic patients. The pooled analysis showed significantly higher odds of mechanical ventilation or respiratory failure among patients with elevated admission glucose (OR 4.46, 95% CI 3.02-6.59). Heterogeneity was absent (Chi^2 = 0.39, df = 2, p = 0.82; I^2 = 0%) (Table 3, Figure 3).

Table 3. Summary of quantitative synthesis for mortality and mechanical ventilation/respiratory failure.

Outcome	Studies	Pooled OR	95% CI	Chi^2	df	p-value	I^2
Mortality	Moon 2016; Sagah 2021; Bhat 2021	5.68	3.55-9.07	1.56	2	0.46	0%
Mechanical ventilation / respiratory failure	Moon 2016; Sagah 2021; Bhat 2021	4.46	3.02-6.59	0.39	2	0.82	0%

Figure 3. Forest plot of the association between elevated admission glucose and mechanical ventilation or respiratory failure in acute organophosphorus poisoning.



3.5 Narrative Synthesis

Two studies were not included in the pooled analysis. Gündüz et al. (2015) reported glucose as a continuous predictor and found that it remained independently associated with mortality, with an odds ratio of 1.022 per mg/dL increase. Panda et al. (2015) found that hyperglycemia and higher admission random plasma glucose were associated with greater poisoning severity, but the study was not suitable for the mortality meta-analysis because deaths after admission were excluded from the analyzed sample (Table 4).

Table 4. Narrative synthesis of studies not included in the pooled analyses.

Study	Exposure format	Main finding	Reason not pooled
Gündüz et al. (2015)	Continuous glucose	Glucose independently predicted mortality, OR 1.022 per mg/dL increase	No categorical 2 × 2 comparison for pooling
Panda et al. (2015)	Hyperglycemia >140 mg/dL; severity-based analysis	Hyperglycemia was more frequent in severe poisoning	Mortality dataset unsuitable because deaths after admission were excluded

Table 5. Raw 2 × 2 data used in the quantitative synthesis.

Study	Exposure definition	Exposed events	Exposed non-events	Control events	Control non-events	Outcome
Moon et al. (2016)	Glucose ≥200 mg/dL vs <200 mg/dL	12	51	4	117	Mortality
Sagah & Elhawary (2021)	Hyperglycemia >140 mg/dL vs non-hyperglycemia	8	18	2	62	Mortality
Bhat et al. (2021)	Hyperglycemia ≥200 mg/dL vs non-hyperglycemia	41	77	28	254	Mortality
Moon et al. (2016)	Glucose ≥200 mg/dL vs <200 mg/dL	56	7	78	43	Respiratory failure
Sagah & Elhawary (2021)	Hyperglycemia >140 mg/dL vs non-hyperglycemia	8	18	4	60	Mechanical ventilation
Bhat et al. (2021)	Hyperglycemia ≥200 mg/dL vs non-hyperglycemia	80	38	93	189	Mechanical ventilation

4. Discussion

This review found a consistent association between elevated admission glucose and poor outcome in acute organophosphorus poisoning. Patients with higher glucose at presentation had higher odds of death and mechanical ventilation or respiratory failure. The studies that could not be pooled pointed in the same direction and supported an association with greater poisoning severity. Admission glucose appears to have practical prognostic value. It is fast, inexpensive, and available in almost any emergency setting. That matters in poisoning care, especially where access to intensive care resources is limited. The association remained stable across studies despite differences in glucose cut-offs, study design, and setting. Several mechanisms may explain this pattern. Organophosphorus poisoning can produce sustained cholinergic stimulation, catecholamine release, and increased ACTH secretion. That can lead to stress hyperglycemia. Increased glycogenolysis and gluconeogenesis may also contribute. Oxidative stress and pancreatic dysfunction have been proposed as additional mechanisms of disturbed glucose homeostasis in acute poisoning (Panda et al., 2015; Rambabu et al., 2019; Bhat et al., 2021). The present findings are in line with prior reports that describe respiratory failure as a major determinant of adverse outcome in organophosphorus poisoning (Giyanwani et al., 2017). They also fit with the broader treatment literature, which shows that mortality remains substantial despite the continued use of atropine, oximes, and supportive care (Buckley et al., 2005; Eddleston et al., 2008). In that setting, a simple bedside marker such as admission glucose may help identify patients who need closer monitoring and earlier escalation of care. Admission glucose should not replace clinical assessment. It may still help with early triage. Patients with marked hyperglycemia may need closer observation, earlier respiratory monitoring, and lower threshold for intensive care referral. This may be especially useful in low-resource settings where advanced prognostic tools are not always available. This review addressed a focused clinical question and combined quantitative and narrative synthesis. The pooled analyses showed consistent effects with no observed statistical heterogeneity. That strengthens the signal. The evidence base still has limits. All included studies were observational. Hyperglycemia was not defined in the same way across studies. Adjustment for confounding was inconsistent. Most studies were single-center. The small number of quantitative studies also meant that formal assessment of

publication bias was not appropriate. One study of clear clinical interest could not contribute to the mortality synthesis because deaths after admission were excluded from the analytic cohort (Panda et al., 2015).

5. Conclusion

Elevated admission glucose was associated with higher odds of mortality and mechanical ventilation or respiratory failure in acute organophosphorus poisoning. The available evidence also supports an association with greater poisoning severity. Admission glucose appears to be a useful early prognostic marker. More prospective multicenter studies with standardized glucose definitions are still needed.

Funding: Please add: This research received no external funding.
Conflicts of Interest: The authors declare no conflict of interest.
Publisher's Note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers.

References

[1] Bhat, M. H., Bhat, S. Q., Dar, I. H., & Bhat, J. A. (2021). Glycemic status at the presentation and its role as marker of severity and outcome in patients with organophosphorus poisoning. *Romanian Journal of Diabetes Nutrition and Metabolic Diseases*, 28(4), 369-375. https://doi.org/10.46389/rjd-2021-1054

[2] Buckley, N., Eddleston, M., & Szinicz, L. (2005). Oximes for acute organophosphate pesticide poisoning. *Cochrane Database of Systematic Reviews*, CD005085. https://doi.org/10.1002/14651858.CD005085

[3] Davies, J. O. J., Eddleston, M., & Buckley, N. A. (2008). Predicting outcome in acute organophosphorus poisoning with a poison severity score or the Glasgow coma scale. QJM, 101(5), 371-379. https://doi.org/10.1093/qjmed/hcn014

[4] Eddleston, M., Buckley, N. A., Eyer, P., & Dawson, A. H. (2008). Management of acute organophosphorus pesticide poisoning. *The Lancet*, 371(9612), 597-607. https://doi.org/10.1016/S0140-6736(07)61202-1

[5] Eddleston, M., Szinicz, L., Eyer, P., & Buckley, N. A. (2002). Oximes in acute organophosphorus pesticide poisoning: A systematic review of clinical trials. *QJM*, 95(5), 275-283.

[6] Giyanwani, P. R., Zubair, U., Salam, O., & Zubair, Z. (2017). Respiratory failure following organophosphate poisoning: A literature review. *Cureus*, 9(9), e1651. https://doi.org/10.7759/cureus.1651

[7] Gunnell, D., Eddleston, M., Phillips, M. R., & Konradsen, F. (2007). The global distribution of fatal pesticide self-poisoning: Systematic review. *BMC Public Health*, 7, 357. https://doi.org/10.1186/1471-2458-7-357

[8] Gündüz, E., Dursun, R., Icer, M., Zengin, Y., Güllü, M. N., Durgun, H. M., & Gökalp, O. (2015). Factors affecting mortality in patients with organophosphate poisoning. *Journal of Pakistan Medical Association*, 65(9), 967-972.

[9] Karalliedde, L. (1999). Organophosphorus poisoning and anaesthesia. *Anaesthesia*, 54(11), 1073-1088.

[10] Konradsen, F., Dawson, A. H., Eddleston, M., & Gunnell, D. (2007). Pesticide self-poisoning: Thinking outside the box. T*he Lancet*, 369(9557), 169-170. https://doi.org/10.1016/S0140-6736(07)60085-3

[11] Moola, S., Munn, Z., Tufanaru, C., Aromataris, E., Sears, K., Sfetcu, R., Currie, M., Lisy, K., Qureshi, R., Mattis, P., & Mu, P.-F. (2020). *Chapter 7: Systematic reviews of etiology and risk. In E. Aromataris & Z. Munn (Eds.), JBI Manual for Evidence Synthesis. JBI*. https://doi.org/10.46658/JBIMES-20-08

[12] Moon, J. M., Chun, B. J., & Cho, Y. S. (2016). Hyperglycemia at presentation is associated with in hospital mortality in non-diabetic patient with organophosphate poisoning. *Clinical Toxicology*, 54(3), 252-258. https://doi.org/10.3109/15563650.2015.1128544

[13] Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., et al. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, 372, n71. https://doi.org/10.1136/bmj.n71

[14] Panda, S., Nanda, R., Mangaraj, M., Rathod, P. K., & Mishra, P. K. (2015). Glycemic status in organophosphorus poisoning. *Journal of Nepal Health Research Council*, 13(31), 214-219.

[15] Rambabu, L., Megson, I. L., & Eddleston, M. (2019). Does oxidative stress contribute to toxicity in acute organophosphorus poisoning? A systematic review of the evidence. *Clinical Toxicology*. https://doi.org/10.1080/15563650.2019.1693589

[16] Rethlefsen, M. L., Kirtley, S., Waffenschmidt, S., Ayala, A. P., Moher, D., Page, M. J., Koffel, J. B., et al. (2021). PRISMA-S: An extension to the PRISMA statement for reporting literature searches in systematic reviews. *Systematic Reviews*, 10, 39. https://doi.org/10.1186/s13643-020-01542-z

[17] Sagah, G. A., & Elhawary, A. E. (2021). Glycemic status as a predictor for the outcomes in patients with acute organophosphorus pesticides poisoning. Egyptian Journal of Forensic Sciences and Applied Toxicology, 21(1), 129-137.

[18] Senanayake, N., de Silva, H. J., & Karalliedde, L. (1993). A scale to assess severity in organophosphorus intoxication: POP scale. *Human & Experimental Toxicology*, 12(4), 297-299. https://doi.org/10.1177/096032719301200407

[19] Wells, G. A., Shea, B., O'Connell, D., Peterson, J., Welch, V., Losos, M., & Tugwell, P. (n.d.). Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. *Ottawa Hospital Research Institute*. https://ohri.ca/en/who-we-are/core-facilities-and-platforms/ottawa-methods-centre/newcastle-ottawa-scale

[20] Yurumez, Y., Yavuz, Y., Saglam, H., Durukan, P., Ozkan, S., Akdur, O., et al. (2009). Electrocardiographic findings of acute organophosphate poisoning. *Journal of Emergency Medicine*, 36(1), 39-42.

[21] Yamashita, M., Yamashita, M., Tanaka, J., & Ando, Y. (1997). Human mortality in organophosphate poisonings. *Veterinary and Human Toxicology*, 39(2), 84-85.