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Prognostic Factors for Mortality and Visual and Neurological Sequelae in Acute Methanol Poisoning: An Integrative Review

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Abstract

Methanol poisoning, usually caused by adulterated spirits, leads to severe metabolic acidosis, blindness, and basal ganglia injury. This integrative review, with systematic search methods and narrative synthesis, examined evidence published from 2021 to 2026 on factors associated with in-hospital mortality and with visual and neurological sequelae in adults with acute methanol poisoning. We searched MEDLINE/PubMed and Europe PubMed Central (PMC) on November 9, 2026. Of 234 records, 128 were screened by a single reviewer and 33 primary studies were included, none from Brazil; only two databases were searched, and the 33 studies include overlapping populations from the same outbreaks or poison centers. No study was judged at low risk of bias. Mortality ranged from 0% to 48.6%. The factors most consistently associated with death were the severity of metabolic acidosis (pH, bicarbonate, lactate, and anion gap), depressed consciousness, and organ failure, each reported in at least three apparently independent cohorts. The pH cut-offs ranged from 6.89 to 7.11, were derived in small cohorts, and are not interchangeable. Inflammatory indices, electrocardiographic findings, and all prediction models were exploratory, and none was externally validated; the accuracy of one nomogram fell from an apparent area under the curve of 0.978 to 0.778 after correction for optimism. Visual sequelae were reported in widely differing proportions, but definitions, denominators, and follow-up periods differed, so the figures are not comparable. Acidosis and delay in admission or treatment were associated with visual sequelae in several cohorts, and serum methanol gave inconsistent results. Evidence on neurological and cognitive sequelae was limited, and standardized follow-up was rare. Where methanol assays are unavailable, blood gas analysis and level of consciousness may support triage, but their thresholds require external validation. Prospective multicenter cohorts with standardized follow-up of vision and cognition are needed.

Categories: Neurology, Emergency Medicine, Ophthalmology
Keywords: blood gas analysis, integrative review, metabolic acidosis, methanol poisoning, mortality, optic neuropathy, prognostic factors, serum lactate

Introduction And Background

Methanol is a toxic alcohol found in solvents, antifreeze, and fuel, and it enters the food chain when distilled beverages are adulterated. Methanol itself causes little harm. The damage begins when the liver enzyme alcohol dehydrogenase converts it to formaldehyde and then to formic acid. Formic acid inhibits mitochondrial respiration [1] and, as it accumulates, causes a severe metabolic acidosis [2]. The tissues most affected are the retina, the optic nerve, and the putamen, a deep brain structure involved in movement control [1,3]. Patients who survive may be left blind or with movement disorders [2].

Poisoning often occurs in clusters. In October 2025, Brazil faced an outbreak caused by adulterated beverages. As of October 8, 2025, there were 24 confirmed cases and five confirmed deaths, with 235 further notifications under investigation, and most services could not measure serum methanol, which was run in reference laboratories [2]. Earlier outbreaks in Norway [4] and the Czech Republic [5] showed how quickly a cluster can overwhelm emergency departments and intensive care units, and an international consensus has set out how to approach patients in such a setting [6]. Treatment combines supportive care, correction of acidosis, an antidote that blocks alcohol dehydrogenase, and hemodialysis in selected patients [6,7].

Two questions follow from this. First, when dialysis and intensive care beds are limited, which patients are at highest risk of death and should be treated first? Second, which patients are likely to keep a visual or neurological deficit? The two outcomes are related but not identical: death may reflect the overall severity of acidosis and organ failure, whereas blindness and basal ganglia injury reflect damage to specific tissues, so a

marker that predicts one may not predict the other. Answering these questions depends on admission data that are available in most hospitals, such as acidosis, level of consciousness, and routine laboratory tests, because serum methanol is often unavailable.

Recent reviews have addressed magnetic resonance imaging findings [3], kidney outcomes [8], optic neuropathy [1], and antidotes [7]; to our knowledge, none has brought together the factors that predict death and sequelae. Many cohorts and several prediction models have been published since 2021, but they differ in setting, outcome definition, and analysis, and some come from the same outbreaks. This integrative review therefore asked which clinical, laboratory, and treatment factors have been reported as associated with in-hospital mortality and with visual and neurological sequelae in adults with acute methanol poisoning, how consistent these associations are across cohorts, and how reliable the underlying evidence is.

Review

Materials and methods

Review Framework and Reporting

We conducted an integrative review with systematic search methods and a narrative synthesis, following the five stages described by Whittemore and Knafl [42]: problem identification, literature search, data evaluation, data analysis, and presentation. It is not a systematic review with meta-analysis, because the studies were not pooled (see Synthesis). We report the review against the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement [43] for the items that apply to a review without pooled estimates, and against PRISMA-S [44] for the search. The protocol was not registered. The eligibility criteria are those of the first version of this review, with one clarification added during revision: only peer-reviewed reports were eligible, so preprints were excluded.

Research Question and Eligibility Criteria

The question followed the population, exposure, comparison, and outcome (PECO) format. The population was adults with acute methanol poisoning; the exposures, clinical, laboratory, imaging, and treatment factors; the comparison, absence of the factor or lower values; and the outcomes, short-term (in-hospital) mortality and visual, neurological, or cognitive sequelae. Mortality and sequelae were kept apart throughout, because the variables that identify patients at risk of dying in the first days are not necessarily those that predict the damage carried by survivors.

We included peer-reviewed primary studies published from 2021 to 2026 with at least 10 adults with methanol poisoning and an analysis of factors associated with death or sequelae. Methanol poisoning had to be established by the study authors from the history together with clinical or biochemical findings, or by a methanol assay. We excluded reviews, letters, editorials, and corrigenda; case reports and case series of fewer than 10 patients; experimental, laboratory, and animal studies; forensic series that included only fatal cases; studies restricted to children; samples without confirmed methanol poisoning or dominated by other alcohols; and preprints.

The 2021 to 2026 window was chosen to describe prognostic evidence from contemporary practice (point-of-care blood gas and lactate measurement, and current availability of dialysis and antidotes) and from the outbreaks of this period, including those during the COVID-19 pandemic in Iran and Turkey and the 2025 outbreak in Brazil [2]. Earlier landmark series, such as the Norwegian outbreak of 2002 to 2004 [4] and the Czech outbreak of 2012 [5], fall outside the window. They are cited for context but were not extracted or appraised, which is a limitation.

Information Sources and Search Strategy

The first version of this review searched PubMed alone on October 15, 2026 (74 records, restricted to the MeSH term Humans). For this revision, the search was repeated and extended on November 9, 2026, in two sources: MEDLINE/PubMed (123 records) and Europe PubMed Central (PMC) (111 records). Europe PMC was added because it indexes MEDLINE, PubMed Central, and preprints, and it returns records that have not yet received MeSH indexing; for the same reason, the Humans filter was removed. No language restriction was applied. Both searches combined methanol terms with prognostic terms in the title or abstract and were limited to publication dates from January 1, 2021, to November 9, 2026. The complete strings are given in Appendix A. We did not search Embase, Scopus, or Web of Science, to which we had no access, and we did not search reference lists, trial registers, or grey literature.

Study Selection

Records were screened in two stages (title and abstract, then full report) against the criteria above, and the reason for each exclusion was recorded. For the updated search, screening was carried out by one reviewer; independent duplicate screening was not performed, and this is acknowledged as a limitation. Duplicates

between PubMed and Europe PMC were removed by PubMed identifier and by title. Reports that could not be retrieved are listed in Appendix C. An artificial intelligence language model (Claude, Anthropic, San Francisco, CA) assisted with the classification of records and with data extraction, under the supervision of the authors, who are responsible for the final decisions.

Data Extraction

Data were extracted with a standardized form: country, setting and period, design (prospective or retrospective), sample size, how methanol poisoning was defined, the factors studied, the definition and assessment of each outcome (in-hospital mortality; visual, neurological, or cognitive sequelae), duration of follow-up, effect estimates with 95% confidence intervals (CIs) and whether they were adjusted or unadjusted, areas under the receiver operating characteristic curve (AUCs) with CIs, the cut-off and the way it was derived, and whether any internal or external validation was done. The full text was read for 21 of the 33 included studies, including six that had first been assessed from abstracts and were verified against the full publication during revision [10,11,17,18,39,40]; for the other 12, the full text could not be accessed, extraction was limited to the abstract, and these studies are marked in Table *1*. Values not reported in the source were recorded as not reported, not imputed.

Risk of Bias

Risk of bias was assessed with the Quality in Prognostic Studies (QUIPS) tool [45] for studies of prognostic factors (domains: study participation, attrition, prognostic factor measurement, outcome measurement, study confounding, and statistical analysis and reporting) and with the Prediction model Risk Of Bias Assessment Tool (PROBAST) [46] for the three studies that developed prediction models [9,29,33]. Overall risk was rated high when any domain was rated high and moderate when no domain was high but at least one was moderate. For studies read only as abstracts, domains that could not be judged were rated unclear and the overall rating relied on the domains that could be judged. These judgements were made by one reviewer and are provisional. We did not grade the certainty of the evidence.

Synthesis

The synthesis was narrative and organized by outcome domain. We did not undertake pool estimates, because the studies differed in design, in the definition of exposures and outcomes, in follow-up, and in cut-offs that were derived separately in each sample; because several cohorts probably share patients; and because most estimates were unadjusted or minimally adjusted, so a pooled estimate would suggest a precision the data do not have. Where findings conflicted, we gave more weight to adjusted than to unadjusted estimates, to prospective than to retrospective designs, to larger samples, and to objectively ascertained outcomes. A factor was called consistent when the association had the same direction in at least three non-overlapping cohorts, at least one of which reported a multivariable analysis. A factor reported in one or two cohorts, only with unadjusted estimates, or only with cut-offs derived and tested in the same sample was called exploratory. Because the review reports published results and does not analyze individual data, it did not require ethical approval, and no separate statistical analysis was undertaken.

Results

Study Selection

The searches identified 234 records (PubMed 123, Europe PMC 111). After removing 106 duplicates (105 records shared by the two sources and one preprint of an article that was also published), 128 records were screened by title and abstract and 92 were excluded: 22 reviews, letters, editorials, or corrigenda; 31 case reports or case series of fewer than 10 patients; 13 experimental, laboratory, or animal studies; seven forensic series of fatal cases only; two studies not in adults; three preprints; and 14 that did not analyze factors associated with death or sequelae, mixed several poisonings, or addressed other outcomes such as intensive care admission or intubation. Of 36 reports sought for full-text assessment, 34 were assessed. One of the studies was excluded because methanol exposure was not confirmed in every patient (the optic nerve sheath diameter study [47]). Two more studies could not be retrieved (a Vietnamese cohort [48], whose abstract was incomplete and whose full text was inaccessible, and a 2022 electrocardiographic report from Shiraz with neither abstract nor full text available; Appendix C). Thirty-three studies were included (Figure *1*).

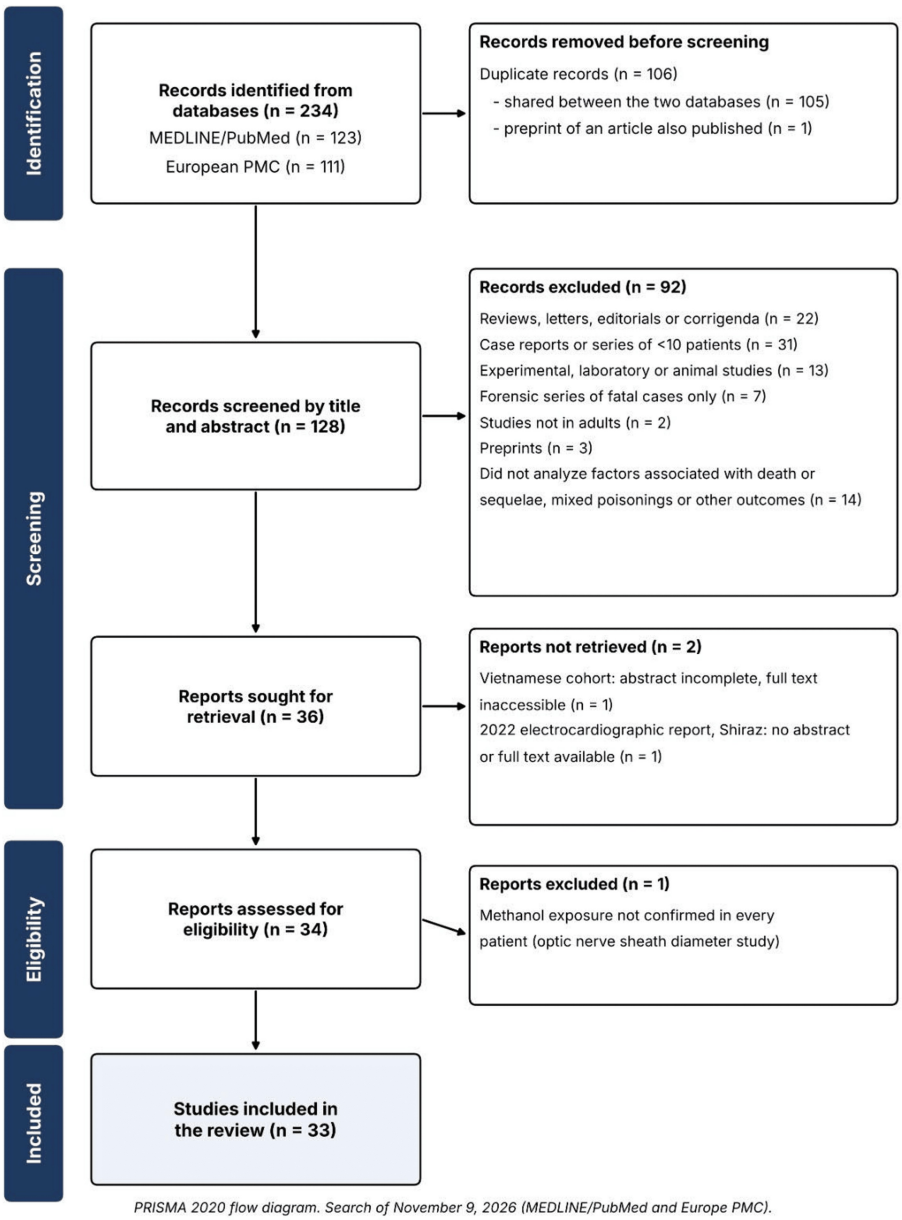


FIGURE 1: PRISMA chart

PRISMA 2020 flow diagram of the search of November 9, 2026 (MEDLINE/PubMed and Europe PMC). PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

Characteristics of the Included Studies

The 33 studies were published between 2021 and 2026 (2021, seven; 2022, four; 2023, four; 2024, seven; 2025, eight; 2026, three). They came from Iran (11), Turkey (eight), Egypt (three), Saudi Arabia, the United States, the Czech Republic, and Tunisia (two each), and Canada, Morocco, and China (one each); none came from Brazil. Three were prospective cohorts [28,36,39], one was a longitudinal follow-up of survivors [27], two analyzed national hospital databases [24,25], one was a multicenter linkage study [22], and the remainder were retrospective, cross-sectional, or case-series analyses of one or two centers. Sample sizes ranged from 14 to 897 patients; one abstract did not state the number of patients. Many studies were set in outbreak or pandemic-period contexts: the Istanbul outbreak [9], a Turkish series from the COVID-19 period [12], Iranian studies from the same period [16,22,35,37,39], a Moroccan outbreak [21], outbreaks in Riyadh [30] and Tunisia [31,40], and the Czech outbreak [27,28]. Data on long-term cognitive and dopaminergic outcomes came only from the Czech cohorts [27,28]. Table *1* summarizes the studies by the main outcome domain.

Study (first author, year: study name) and reference	Country	Design	Patients	Outcome domain	Main findings	Risk of bias
Dikme et al., 2026: predictors of in-hospital mortality after the Istanbul outbreak [9]	Turkey	Retrospective cohort	55	Mortality	Mortality 45.5% (25 of 55); pH ≤6.89 (AUC 0.969); per 0.1-unit fall in pH, OR 2.78 (95% CI 1.34 to 5.76); in a model without blood gases, lactate OR 1.60 (1.23 to 2.08) and Glasgow Coma Scale OR 1.38 (1.07 to 1.78) per unit; two-variable models (25 events); no external validation	High
Yaşar et al., 2025: clinical indicators of hemodialysis and mortality [10]	Turkey	Retrospective cross-sectional	64	Mortality	Mortality 29.7% (19 of 64) at 30 days; pH ≤7.08 (AUC 0.894); bicarbonate ≤8.45 (AUC 0.820); lactate ≥3.67 mmol/L and anion gap ≥27.25 also associated with death; univariable ROC analysis only	High
Ahmadi et al., 2025: prognostic factors at Tabriz Sina Hospital [11]	Iran	Retrospective (2019 to 2021)	242	Mortality; visual	Mortality 6.6% (16 of 242); visual sequelae 14.5%; adjusted OR for death: coma 8.0 (95% CI 3.2 to 19.9), arrhythmia 5.5 (2.0 to 15.1), pH <7.1 4.2 (2.0 to 8.9), opiate co-use 2.8 (1.2 to 6.5), ethanol delay >3 h 2.3 (1.1 to 4.8); 16 events	Moderate
Güler and Üçöz Kocaşaban, 2024: home-distillation outbreak in Turkey during the COVID-19 pandemic [12]	Turkey	Retrospective case-control	43	Mortality	Mortality 46.5%; pH <7.11; lactate >4.50 mmol/L	High
Sasani et al., 2024: prognosis of methanol poisoning in a developing setting [13]	Iran	Hospital-based cohort	123	Mortality; neurological (imaging)	Level of consciousness (OR 42.2; 95% CI 2.35 to 756.5) and pH associated with death; abnormal head CT in 34.3%, OR for death 14.05 (3.78 to 52.34) unadjusted and 4.25 (0.91 to 19.95) adjusted	Moderate
Ermete Güler et al., 2026: prognostic value of metabolic and biochemical parameters beyond lactate [14]	Turkey	Retrospective observational	82	Mortality	Mortality 11.0%; lactate >8.5 mmol/L (AUC 0.785)	High
Ağaçkıran and Ağaçkıran, 2025: lactate-to-albumin ratio and albumin-corrected anion gap [15]	Turkey	Retrospective	42	Mortality	Mortality 35.7%; anion gap ≥29.4 (AUC 0.942; 95% CI 0.870 to 1.000); albumin-corrected anion gap ≥25.6 and lactate-to-albumin ratio ≥1.24 associated with death	High
Simani et al., 2022: brain lesions during the COVID-19 methanol outbreak [16] †	Iran	Retrospective	516 (40 with CT)	Neurological (imaging)	Necrosis, hemorrhage, and edema more frequent among deaths	High
Abdelwahab et al., 2022: neutrophil-to-lymphocyte versus platelet-to-lymphocyte ratio [17]	Egypt	Cross-sectional	109	Mortality; visual	Mortality 31% (34 of 109); visual loss 27.5%; NLR AUC 0.991 (95% CI 0.95 to 1.0) for death at a cut-off >6.6 (sensitivity 100%, specificity 94.7%) derived in the same sample; PLR AUC 0.734 for visual loss	High
Arslan et al., 2021: red cell distribution width and in-hospital mortality [18]	Turkey	Retrospective	42	Mortality	Mortality 21.4% (9 of 42); RDW AUC 0.778 (95% CI 0.567 to 0.988), cut-off 16.2; Glasgow Coma Scale AUC 0.956	High
Şahin et al., 2025: systemic immune-					Mortality 48.6%; SII HR 2.44 (95% CI	

inflammation index and pan-immune-inflammation value [19]	Turkey	Retrospective, two centers	76	Mortality	1.05 to 5.67), AUC 0.750 (0.649 to 0.865); cut-off 665.56 gave sensitivity 50% and specificity 46%	Moderate
Sarıbaş et al., 2025: methanol blood levels, prognosis, and sequelae in the emergency department [20]	Turkey	Retrospective	116	Mortality; visual	Mortality 23.3%; methanol level not correlated; visual sequelae 33.7%; neurological 6.2%	High
Zniber et al., 2025: northwestern Moroccan outbreak treated with hemodialysis [21] †	Morocco	Not stated	202 (22 dialyzed)	Visual	Overall mortality 5%; no deaths among 22 dialyzed patients; three developed permanent blindness, with higher blood methanol in those patients (p=0.02)	High
Mahdavi et al., 2022: multicenter linkage study of admissions and mortality in Iran [22]	Iran	Cross-sectional linkage, multicenter	795	Mortality; treatment	Mortality 10.6%; hemodialysis within about 60 min: sequelae 11.4% vs 19.6%	Moderate
Sharif et al., 2021: predictors of poor outcome with emphasis on the SOFA score [23]†	Saudi Arabia	Multicenter	37	Visual (unfavorable outcome)	No deaths; SOFA >4.5 (AUC 0.955); optic neuropathy and blindness 29.7%	High
Thongprayoon et al., 2021: acute kidney injury in the National Inpatient Sample [24]†	United States	National database	603	Mortality	Acute kidney injury 22.4%, associated with in-hospital death	Moderate
Kaewput et al., 2021: inpatient burden and mortality in the United States [25]†	United States	National database	603	Mortality	Mortality 6.5%; risk rose with number of organ failures	Moderate
AlSamh and Kramer, 2024: neurologic complications in critically ill patients (Alberta) [26]†	Canada	Retrospective cohort	55 (methanol)	Composite (death or persistent deficit)	Death or persistent cognitive or visual deficit 24%; no favorable recovery with anion gap ≥28	Moderate
Bukacova et al., 2021: longitudinal cognitive changes after methanol exposure [27]†	Czech Republic	Longitudinal	Not stated	Neurological (cognitive)	MMSE decline over time (p=0.007); 43% to 60% below 1.5 SD in memory	Moderate
Kotikova et al., 2021: ioflupane SPECT, 6-year prospective study [28]†	Czech Republic	Prospective	42	Neurological (dopaminergic imaging)	Posterior putamen ioflupane binding correlated with pH (r=0.574)	Moderate
Rahimi et al., 2024: machine learning prediction of prognosis [29]†	Iran	Retrospective, machine learning	897	Mortality; sequelae	AUC 0.943 to 0.947 (internal validation only); 90 deaths; 234 with sequelae	High
Alhusain et al., 2024: outbreaks in Riyadh, Saudi Arabia [30]	Saudi Arabia	Retrospective case series	22 arrived alive	Mortality; visual	Mortality 17.4%; vision impairment in seven of 12 assessed	High
Ketata et al., 2022: acute methanol poisoning in Tunisia [31]†	Tunisia	Cross-sectional	65	Mortality	Mortality 12.3%; age, delay to consultation, acidosis; four visually impaired	High
Sadeghi et al., 2023: five-year retrospective study in northern Iran [32]†	Iran	Retrospective	101	Mortality; treatment	Mortality 18.8%; consciousness and acidosis; corticosteroids associated with lower mortality	High
Abdelhamid et al., 2024: risk-prediction nomogram	Egypt	Retrospective	180	Mortality;	Mortality 23.9%; blindness 28.9%; nomogram (blindness, mean arterial pressure, bicarbonate, creatinine) AUC 0.978 apparent and 0.778 after bootstrap	High

for in-hospital mortality [33]		cohort		visual	correction; for blindness, delayed admission OR 1.039 (95% CI 1.010 to 1.069) and anion gap OR 1.053 (1.007 to 1.101)	
Nafea et al., 2025: leukocyte glucose index and outcomes [34]	Egypt	Retrospective	82	Mortality; visual	Mortality 26.8% (22 of 82); leukocyte glucose index AUC 0.808 for death; no discrimination for visual outcome	High
Esmaeilian et al., 2023: clinical, laboratory, and brain imaging findings during the COVID-19 pandemic [35]	Iran	Retrospective cross-sectional	306	Neurological (imaging); mortality	Putaminal hypodensity 11.1%, cerebellar nuclei 8.2%, diffuse edema 7.5%, hemorrhage 1.6%; poor outcome related to blindness, opium addiction, chronic alcohol use, hyperglycemia, and abnormal CT (p<0.001); cerebellar hypodensity, edema, and hemorrhage most predictive	High
Gheshlaghi et al., 2024: follow-up of visual defects [36]	Iran	Prospective cohort	39	Visual	Reduced visual acuity at admission predicted long-term acuity and field defects and optic disc atrophy; blurred vision alone did not; 14 of 36 dialyzed patients had visual impairment at follow-up	High
Nikoo et al., 2021: Brugada-like electrocardiographic pattern [37]	Iran	Observational	356	Mortality	Pattern in 20 patients (5.6%), associated with death, very low Glasgow Coma Scale score, higher glucose, and lower pH	High
Shen et al., 2023: visual damage in inhalational poisoning [38]	China	Retrospective	14	Visual	Visual prognosis related to duration of exposure, delayed admission, and degree of acidosis; acuity stabilized after the fifth month	High
Shirvani et al., 2025: visual outcomes and retinal nerve fiber layer changes [39]	Iran	Prospective cohort	61 (survivors)	Visual	Lower pH and bicarbonate and higher anion gap associated with retinal nerve fiber layer thinning and worse visual outcome at 12 months (p<0.001); unadjusted analyses (Spearman correlation, univariable regression); patients who died were excluded	High
Rym et al., 2023: ocular manifestations in a Tunisian outbreak [40]	Tunisia	Case series	21	Visual	Ocular abnormalities in 81.8% of patients with ocular symptoms; optic neuropathy in 7 (63.6%); blood methanol higher in patients without ocular symptoms (p=.03); examined a mean of 4 days after admission; 14 patients lost to follow-up	High
Majidi et al., 2026: five-year series with ocular outcomes [41]†	Iran	Observational	124	Mortality; visual	Died 19.4%; blind 13.7%; reduced acuity 6.5%; pH and bicarbonate lower in deaths; older age and later referral in those who died or lost vision	High

TABLE 1: Characteristics, main findings and risk of bias of the 33 included studies

AUC: area under the receiver operating characteristic curve; CI: confidence interval; CT: computed tomography; HR: hazard ratio; MMSE: Mini-Mental State Examination; NLR: neutrophil-to-lymphocyte ratio; OR: odds ratio; PLR: platelet-to-lymphocyte ratio; RDW: red cell distribution width; SII: systemic immune-inflammation index; SOFA: Sequential Organ Failure Assessment.

†Data were extracted from the abstract only because the full text could not be accessed; the full text was read for the other studies. Outcome domain is the main outcome analyzed.

Risk of bias is a provisional judgement by one reviewer (Quality in Prognostic Studies (QUIPS), or Prediction model Risk Of Bias Assessment Tool (PROBAST) for studies that developed prediction models); no study was at low risk. The number of patients is as stated in each source and refers to patients with methanol poisoning unless indicated. Studies are identified by first author, year, and study name; the number in brackets is the reference number.

Predictors of In-hospital Mortality

Acid-base and metabolic markers: Acidosis at admission was the factor most often associated with death. Reported cut-offs for arterial pH were ⩽6.89 (area under the receiver operating characteristic curve (AUC) 0.969; 55 patients, 25 deaths) [9], ⩽7.08 (AUC 0.894; 64 patients) [10], <7.1 (odds ratio 4.2) [11], and <7.11 [12]. In the Istanbul cohort, each 0.1-unit fall in pH was associated with 2.78 times the odds of death (95% confidence interval (CI) 1.34 to 5.76) [9]. pH was also associated with death in a cohort of 123 patients [13], pH and bicarbonate were lower in the patients who died in the Urmia series [41], and in a two-center cohort lower pH, lower bicarbonate, and a larger base deficit were each associated with death after adjustment for age and sex, although with wide intervals (for pH, hazard ratio 0.20; 95% CI 0.05 to 0.88) [19]. Lactate showed a similar pattern: above 8.5 mmol/L (AUC 0.785) [14], above 4.50 mmol/L [12], and, per 1 mmol/L, an odds ratio of 1.60 (95% CI 1.23 to 2.08) in the Istanbul model that excluded blood gas variables [9]. A lactate-to-albumin ratio of 1.24 or more and an albumin-corrected anion gap of 25.6 or more were also associated with death in 42 patients; the AUC of the uncorrected anion gap (cut-off 29.4) was 0.942, with a 95% CI of 0.870 to 1.000 [15]. Acid-base variables were therefore consistent across cohorts, but the cut-offs were derived and tested in the same small samples and are not interchangeable.

Consciousness, circulation, and organ failure: In a multivariable model in 242 patients from Tabriz (16 deaths), coma at admission (adjusted odds ratio 8.0; 95% CI 3.2 to 19.9), arrhythmia (5.5; 2.0 to 15.1), pH below 7.1 (4.2; 2.0 to 8.9), and opiate co-use (2.8; 1.2 to 6.5) were independently associated with death, although the small number of events limits the stability of the model [11]. In the cohort of 123 patients, level of consciousness had an odds ratio of 42.2 with a 95% CI of 2.35 to 756.5, an interval so wide that it shows imprecision rather than effect size, and all five hypotensive patients died [13]. In Istanbul, each point lower on the Glasgow Coma Scale carried an odds ratio of 1.38 (95% CI 1.07 to 1.78) [9]. Consciousness was also associated with death in Sadeghi et al. [32], mean arterial pressure and serum creatinine entered a nomogram [33], and mechanical ventilation (hazard ratio 13.15; 95% CI 1.76 to 98.5) and lower mean arterial pressure were associated with death after adjustment for age and sex in the two-center cohort [19]. In United States hospital data, acute kidney injury occurred in 22.4% of admissions and was associated with death [24], and mortality of 6.5% depended mainly on the number of failing organs [25]. A Brugada-like electrocardiographic pattern was present in 20 of 356 patients (5.6%) in the Shiraz outbreak and was associated with death, very low Glasgow Coma Scale scores, and lower pH [37]; this finding comes from a single cohort and is exploratory.

Neuroimaging: Abnormal head computed tomography (CT) was present in 34.3% of the 123 patients in one cohort; the odds ratio for death was 14.05 (95% CI 3.78 to 52.34) before adjustment and 4.25 (95% CI 0.91 to 19.95) after adjustment, so the adjusted estimate is compatible with no association [13]. Necrosis, hemorrhage, and edema were more frequent among patients who died in a cohort of 516 patients, 40 of whom had CT [16]. In 306 patients from two Iranian hospitals, hypodensity of the cerebellar nuclei, diffuse cerebral edema, and intracranial hemorrhage were the imaging findings most related to a poor outcome, which was also related to blindness, opium addiction, chronic alcohol use, and hyperglycemia [35]. Abnormal CT is therefore associated with death in the direction expected, but the adjusted evidence is imprecise.

Inflammatory indices: The neutrophil-to-lymphocyte ratio had an AUC of 0.991 for death in 109 patients, and the platelet-to-lymphocyte ratio an AUC of 0.734 for visual loss [17]; in the full report the 95% CI was 0.95 to 1.0, and the cut-off of 6.6, chosen by the Youden index in the same sample, gave a sensitivity of 100% and a specificity of 94.7%, so the near-perfect AUC is an apparent estimate that has not been validated. Red cell distribution width had an AUC of 0.778 (95% CI 0.567 to 0.988) in 42 patients with nine deaths, lower than that of the Glasgow Coma Scale in the same cohort (AUC 0.956) [18]. The systemic immune-inflammation index had a hazard ratio of 2.44 (95% CI 1.05 to 5.67) and an AUC of 0.750 (95% CI 0.649 to 0.865), but the cut-off of 665.56 gave a sensitivity of 50% and a specificity of 46% [19]. The leukocyte glucose index had an AUC of 0.808 for death and no discrimination for visual outcome [34]. Each index was tested in a single cohort, so all are exploratory.

Serum methanol: Blood methanol did not correlate with mortality in 116 patients [20]. Among 22 patients treated with hemodialysis in Morocco, higher levels were associated with blindness (p=0.02) [21], whereas in a Tunisian series of 21 patients the levels were higher in those without ocular symptoms (p=0.03) [40]. A Vietnamese cohort states in its title that methanol concentration predicts severe outcomes [48], but only an incomplete abstract was available. The findings on serum methanol are therefore inconsistent.

Prediction models: Three studies built multivariable or machine learning models. The Istanbul analysis limited each logistic model to two variables because it had 25 events [9]. A four-variable nomogram (blindness, mean arterial pressure, bicarbonate, creatinine) had an apparent AUC of 0.978 in 180 patients, which fell to 0.778 after bootstrap correction for optimism [33]. A machine learning model in 897 patients reached an AUC of 0.943 to 0.947 with internal validation only [29]. None was validated in an external population.

Predictors of Visual Sequelae

Visual sequelae were reported in 14.5% of patients in Tabriz [11], as visual loss in 27.5% [17], 29.7% [23], and 33.7% [20] of patients, blindness in 28.9% [33] and 13.7% (with a further 6.5% with reduced acuity) [41], and visual impairment in seven of 12 patients assessed [30]. These figures are not comparable, because the denominators (all admissions, survivors, dialyzed patients, or patients assessed), the definitions (blindness, reduced acuity, any ocular finding), and the time of assessment (discharge to 12 months) differ. In a prospective cohort of 61 survivors (patients who died during hospitalization were excluded), lower pH, lower bicarbonate, and a higher anion gap were associated with retinal nerve fiber layer thinning and worse visual acuity at 12 months; these associations came from Spearman correlation and univariable regression, without adjustment for confounders [39]. Delayed admission (odds ratio 1.039; 95% CI 1.010 to 1.069) and a higher anion gap (odds ratio 1.053; 95% CI 1.007 to 1.101) were risk factors for blindness in 180 patients [33]. Duration of exposure, delayed admission, and degree of acidosis were related to visual prognosis in 14 patients with inhalational poisoning [38], and patients who lost their vision were older and had been referred later in the Urmia series [41]. In a prospective cohort of 39 patients, reduced visual acuity at admission predicted visual field defects and optic disc atrophy at follow-up, whereas blurred vision alone did not [36]. A Sequential Organ Failure Assessment (SOFA) score above 4.5 predicted an unfavorable outcome (AUC 0.955; 37 patients) [23]. Acidosis and delay to treatment were thus associated with visual sequelae in several independent cohorts, whereas inflammatory indices [17,34] and serum methanol [21,40] were not consistent.

Neurological and Cognitive Sequelae

Neurological sequelae were rarely the primary outcome. They were reported in 6.2% of 116 patients [20], and in Alberta 24% of 55 critically ill patients died or had persistent cognitive or visual deficits, with no favorable recovery when the anion gap was 28 or more [26]. Among survivors of the Czech outbreak, global cognitive performance (Mini-Mental State Examination) declined over repeated assessments from 2013 to 2019 and 43% to 60% scored more than 1.5 standard deviations below the norm in memory tests [27]; ioflupane (dopamine transporter) binding in the posterior putamen correlated with arterial pH (r about 0.574) in 42 survivors [28]. These two reports concern the same outbreak and probably overlapping survivors, and they are interpreted separately from the acute predictors of death. No study validated an early marker against long-term cognitive or dopaminergic outcome.

Treatment-Related Factors

All treatment associations came from observational data and are confounded by severity at presentation. A delay in ethanol therapy of more than three hours was independently associated with death in Tabriz (adjusted odds ratio 2.3; 95% CI 1.1 to 4.8) [11]. In a multicenter linkage study, hemodialysis within about 60 minutes coincided with sequelae in 11.4% of patients in East Azerbaijan, compared with 19.6% in other provinces (adjustment is not reported in the abstract) [22]. Systemic corticosteroids were associated with lower mortality in northern Iran [32], and early erythropoietin and corticosteroids were proposed as neuroprotective in the Shiraz prospective cohort [39]; neither study was designed to test treatment. In the Isfahan cohort, 14 of 36 dialyzed patients (38.9%) had visual impairment at follow-up [36], which reflects the severity of the patients selected for dialysis and not a treatment effect.

Risk of Bias and Overlapping Populations

No study was at low overall risk of bias. Nine were rated moderate [11,13,19,22,24-28] and 24 high (Table *1* and Appendix B). After full-text review during revision, the prospective visual cohort [39] was reclassified from moderate to high risk because its analyses were unadjusted and patients who died were excluded. The most frequent problems were retrospective single-center designs, no or minimal adjustment for severity, cut-offs derived and tested in the same sample, few events per variable, nonstandardized outcome assessment, and attrition that was not described. In PROBAST, the analysis domain was rated high for all three model-development studies [9,29,33]. Judgements for the 12 studies read only as abstracts are provisional.

Several studies probably share patients. The two US analyses used the same 603 patients [24,25]. The machine learning study [29] used a hospital dataset of 897 patients that was also used by two machine learning reports we excluded for other outcomes. Studies from the Shiraz outbreak of 2020 [35,37] and from the Czech outbreak [27,28] probably share patients, as may the Egyptian poison-center cohorts [17,33,34] and other Iranian [11,13,16,22,32,41] and Turkish [12,19] series from the same hospitals or periods. We counted each study once and did not treat agreement between overlapping cohorts as independent confirmation.

Discussion

Principal Findings: Established and Exploratory Predictors

In 33 studies published from 2021 to 2026, the factors most consistently associated with in-hospital death were the severity of metabolic acidosis (pH, bicarbonate, lactate, and anion gap), depressed consciousness, and organ failure. Each was reported in at least three apparently independent cohorts, at least one with a multivariable analysis. The same variables predicted death in the large outbreak series that fall outside our window: in Norway, respiratory arrest, coma, and a pH below 6.90 were strong predictors of poor outcome [4], and in the Czech outbreak in 2012 a pH below 7.0, coma, and a negative serum ethanol were the only independent predictors of death [5]. The finding fits the toxicology, because formic acid inhibits mitochondrial respiration and pH, bicarbonate, and lactate reflect the accumulated acid load [1]. These variables indicate severity and do not replace early empirical treatment during outbreaks [6].

For the visual outcome, acidosis and delay to admission or treatment were associated with sequelae in several cohorts [33,38,39,41]. Abnormal head CT was associated with death in the expected direction, but the only adjusted estimate had a confidence interval that included 1 [13]. We classify as exploratory the inflammatory indices (each tested in a single cohort), the Brugada-like electrocardiographic pattern, and all prediction models, none of which was externally validated. Serum methanol gave inconsistent results across cohorts [20,21,40], and a Vietnamese cohort that reports an association [48] could not be appraised.

Discrepancies, Thresholds, and the Limits of Reported Accuracy

The pH cut-offs ranged from 6.89 to 7.11 and are not interchangeable. They were derived by different methods in different populations, with different times from ingestion to arrival, and with small numbers of events; the Istanbul analysis had 25 deaths [9]. Reported accuracy should be read with the same caution. The AUC of 0.969 for pH came from 55 patients with no external validation, the AUC of 0.991 for the neutrophil-to-lymphocyte ratio came from a cut-off chosen in the same 109 patients without validation [17], and the nomogram AUC fell from 0.978 to 0.778 once optimism was corrected [33]. The machine learning AUCs of 0.943 to 0.947 reflect internal validation in a single hospital dataset [29]. An AUC can also hide poor performance at the chosen cut-off, as the systemic immune-inflammation index shows: an AUC of 0.750 with a sensitivity of 50% and a specificity of 46% [19]. Estimates from very small groups are unstable; the confidence interval of the odds ratio for consciousness ran from 2.35 to 756.5 [13], and the interval of the anion gap AUC reached 1.000 [15]. Mortality ranged from 0% in selected samples to 48.6%, and an earlier systematic review estimated 24% [8]; the variation probably reflects case mix, delay to arrival, access to dialysis and antidotes, and whether deaths outside hospital were counted.

Mortality and sequelae should not be merged. The leukocyte glucose index discriminated death but not visual outcome [34], and the studies of cognitive and dopaminergic damage came only from survivors of the Czech outbreak [27,28], so there is no evidence on whether an admission marker predicts long-term cognitive deficit. The percentages of visual sequelae are not comparable across studies for the reasons given in the Results, and we do not give a single range.

Biological Plausibility

Methanol itself is relatively non-toxic; the harm follows its conversion to formic acid, which inhibits mitochondrial respiration and produces metabolic acidosis [1,3]. The retina, optic nerve, and putamen are especially vulnerable [3], and the included studies show the corresponding consequences: retinal nerve fiber layer thinning associated with acidosis [39], necrosis, hemorrhage, and edema on cranial imaging in patients who died [16,35], and dopamine transporter binding in the putamen that correlated with pH [28]. A possible reason why serum methanol is a weaker marker than acid-base status is that its concentration depends on the time since ingestion and on treatment already given, whereas acidosis reflects metabolic damage already established. This explanation is a hypothesis, and no included study tested it.

Implications for Triage and Clinical Practice

Variables available in most emergency departments carry prognostic information. In Brazil, where most services could not measure serum methanol during the 2025 outbreak, the Ministry of Health advised treatment on clinical suspicion without waiting for laboratory confirmation [2]. In that setting, pH, bicarbonate, lactate, the anion gap, and the Glasgow Coma Scale are the tools at hand, and in Istanbul lactate and level of consciousness kept their prognostic value in a model without blood gas parameters [9]. They are best used to identify patients who need intensive care, early dialysis, and ophthalmological and neurological follow-up, not to decide whether antidotal treatment starts [6]. Because the cut-offs are not validated, we do not recommend any single threshold as a decision rule. The associations with treatment timing [11,22] and with corticosteroids [32] are observational and may reflect severity at presentation, so they do not support a change in management.

Strengths and limitations

The strengths of this review are the separation of mortality from sequelae, explicit synthesis rules, the reporting of confidence intervals and validation status, and an explicit account of overlapping populations. Its limitations are as follows.

Search and Selection

We searched two sources, PubMed and Europe PMC, and not Embase, Scopus, or Web of Science, so eligible studies indexed only there may be missing. The 2021 to 2026 window excludes landmark series [4,5] that were not appraised. The protocol was not registered, and for the updated search screening was done by one reviewer with the assistance of a language model, without independent duplicate screening, which may have led to errors in selection. Two reports could not be retrieved, including one Vietnamese cohort [48], and one study was excluded because methanol exposure was not confirmed in all patients [47].

Data Extraction and Appraisal

Full texts were read for 21 of the 33 studies; for the remaining 12, which could not be accessed, we may have missed adjustment, confidence intervals, and outcome definitions that appear only in the full report. Risk-of-bias ratings were made by one reviewer and are provisional, and certainty of evidence was not graded.

Primary Studies

Most studies were retrospective and from one or two centers. Outcome definitions, time of assessment, and follow-up differed. Hospital-based samples exclude people who died before arrival (in the Czech outbreak, 20 of 121 patients died outside hospital [5]) and so can underestimate mortality and bias the associations. Populations overlap, and 19 of 33 studies came from Turkey and Iran, with none from Brazil, so extrapolation to Brazilian services requires caution.

Synthesis

We did not pool results and did not involve a statistician in the synthesis. The confidence intervals and AUCs are reproduced from the sources, and the classification of factors as consistent or exploratory follows the rules in the Methods, which are a judgement and not a statistical test.

Directions for future research

First, visual and neurological sequelae need standardized definitions and assessments at predefined intervals in prospective cohorts, ideally with a minimum follow-up of 12 months. Second, the pH, lactate, and inflammatory-index cut-offs should be tested in large multicenter cohorts with external validation, and prediction models should be reported according to PROBAST [46] with calibration and optimism-corrected performance. Third, studies should record the interval between ingestion and treatment, the use of ethanol or fomepizole, and the timing of dialysis in a standardized way, so that treatment effects can be separated from severity at presentation. Fourth, studies from the same outbreak or hospital should cite one another and state overlap. Finally, prospective cohorts or registries from the 2025 Brazilian outbreak [2], with predefined follow-up of vision and cognition, would show whether the thresholds reported elsewhere apply to Brazilian services. Future syntheses should use a registered protocol, independent duplicate screening, at least three databases, and a full-text risk-of-bias assessment.

Conclusions

In studies published between 2021 and 2026, the most consistent predictors of in-hospital death in adults with acute methanol poisoning were the severity of metabolic acidosis, depressed consciousness, and organ failure. These markers identify patients at high risk, but the pH thresholds differ between cohorts and should not be applied as fixed cut-offs. Acidosis and delay in admission or treatment were associated with visual sequelae in several cohorts, whereas evidence on neurological and cognitive sequelae is limited. Inflammatory indices, electrocardiographic findings, and all prediction models remain exploratory because none was externally validated, and some cohorts overlap.

The evidence comes mostly from retrospective single-center cohorts, and no study was judged to be at low risk of bias. Because only two databases were searched and a single reviewer screened the records, relevant studies may have been missed. Prospective multicenter cohorts that record time to presentation, define sequelae in a standard way, and validate prediction models externally are needed before any score can guide triage.

Appendices

Appendix A: Search strategies

MEDLINE/PubMed (searched on November 9, 2026; 123 records): ("Methanol/poisoning"[MeSH Terms] OR "methanol poisoning"[Title/Abstract] OR "methanol intoxication"[Title/Abstract] OR "methanol toxicity"[Title/Abstract]) AND (prognos*[Title/Abstract] OR predict*[Title/Abstract] OR mortality[Title/Abstract] OR outcome*[Title/Abstract] OR sequela*[Title/Abstract] OR survival[Title/Abstract] OR "risk factor*"[Title/Abstract] OR death[Title/Abstract]) AND 2021/01/01:2026/11/09[Date - Publication].

Europe PMC (searched on November 9, 2026; 111 records): (TITLE_ABS:"methanol poisoning" OR TITLE_ABS:"methanol intoxication" OR TITLE_ABS:"methanol toxicity") AND (TITLE_ABS:prognos* OR TITLE_ABS:predict* OR TITLE_ABS:mortality OR TITLE_ABS:outcome* OR TITLE_ABS:sequela* OR TITLE_ABS:survival OR TITLE_ABS:"risk factor*" OR TITLE_ABS:death) AND (FIRST_PDATE:[2021-01-01 TO 2026-11-09]).

No language filter was applied. Of the 234 records, 105 were shared by both databases and one was a preprint of a published article, leaving 128 records for title and abstract screening.

Appendix B: Risk of bias of the included studies

Prognostic-factor studies were appraised with the Quality in Prognosis Studies (QUIPS) tool [45] and model-development studies [9,29,33] with the Prediction model Risk Of Bias ASsessment Tool (PROBAST) [46]. Ratings were made by a single reviewer and are provisional. No study was rated at low risk of bias.

Moderate risk of bias (nine studies): [11,13,19,22,24-28].

High risk of bias (24 studies): all other included studies, including [39], which was reclassified from moderate to high after full-text review because its analyses were unadjusted and patients who died were excluded. The main reasons were retrospective single-center design, small numbers of events, incomplete adjustment for confounders, unclear handling of missing data, and cut-offs derived and tested in the same sample. Studies [16,21,23-29,31,32,41] were appraised from abstracts only because their full texts could not be accessed; studies [10,11,17,18,39,40], first appraised from abstracts, were re-appraised from the full text during revision.

Appendix C: Reports not retrieved or excluded after full-text assessment

Not retrieved (two): a Vietnamese cohort on serum methanol and severe outcomes [48], for which the abstract was incomplete and no full text was accessible; and a 2022 electrocardiographic report from Shiraz (PubMed ID 35646209; DOI 10.5847/wjem.j.1920-8642.2022.032), for which neither abstract nor full text could be obtained.

Excluded after full-text assessment (one): [47], because methanol exposure was not confirmed in all patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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