North Clin Istanb 2026;13(5):523–532
doi: 10.14744/nci.2026.59852

Original Article ANESTHESIOLOGY & REANIMATION

Clinical and laboratory characteristics associated with in-hospital mortality in methanol poisoning: A 5-year retrospective cohort study

Yigit Sahin,[1] Iskender Aksoy[2]

[1]Department of Anesthesiology and Reanimation, Giresun University Faculty of Medicine, Giresun, Turkiye
[2]Department of Emergency Medicine, Giresun University Faculty of Medicine, Giresun, Turkiye

ABSTRACT

OBJECTIVE: To compare the demographic, clinical, and laboratory characteristics of survivors and non-survivors presenting to the emergency department with methanol poisoning and to identify variables associated with in-hospital mortality.

METHODS: This single-center retrospective observational cohort study included all consecutive adults (≥18 years) diagnosed with methanol poisoning at the emergency department of Giresun Training and Research Hospital, Türkiye, between September 01, 2021, and May 01, 2026. Patients were categorized as survivors and non-survivors. Demographic characteristics, clinical findings, laboratory parameters, severity scores (Glasgow Coma Scale [GCS], Acute Physiology and Chronic Health Evaluation II [APACHE II], and Sequential Organ Failure Assessment [SOFA]), and treatment variables were compared using univariable analyses.

RESULTS: A total of 26 patients were included, and 9 (34.6%) died. At emergency department presentation, loss of consciousness was more frequent among non-survivors (88.9% vs. 11.8%; p<0.001). Non-survivors also had lower GCS scores (median 4 vs. 15; p<0.001) and admission pH (7.00 vs. 7.11; p=0.011), together with higher lactate (7.1 vs. 2.9 mmol/L; p<0.001), creatinine (1.52 vs. 1.00 mg/dL; p=0.001), glucose (194 vs. 123 mg/dL; p=0.029), and anion gap levels (27.0 vs. 20.9 mmol/L; p=0.018). Within the intensive care unit (ICU) subgroup (n=20), ICU non-survivors (n=9) had higher APACHE II and SOFA scores than ICU survivors (n=11) (p=0.002 and p<0.001, respectively). During the subsequent hospital course, all non-survivors required invasive mechanical ventilation compared with none of the survivors (100% vs. 0%; p<0.001); this finding was considered a marker of greater clinical severity rather than a baseline prognostic characteristic.

CONCLUSION: In this small exploratory cohort, non-survivors showed greater neurological impairment and a general pattern of more severe metabolic derangement at emergency department presentation. Within the selected ICU subgroup, ICU non-survivors had higher APACHE II and SOFA scores than ICU survivors. These findings are hypothesis-generating and may help identify clinical features warranting heightened concern; however, they should not be interpreted as validated prognostic criteria or as a risk-stratification tool.

Keywords: Emergency department; metabolic acidosis; methanol; methanol poisoning; mortality.

Cite this article as: *Sahin Y, Aksoy I. Clinical and laboratory characteristics associated with in-hospital mortality in methanol poisoning: A 5-year retrospective cohort study. North Clin Istanb 2026;13(5):523–532.*

Methyl alcohol (methanol) is a colorless solvent found in glass cleaning solutions, antifreeze, paint thinners, photocopier fluids, and many industrial products. Because methanol is substantially less expensive than ethanol, it may be illegally substituted for ethanol in counterfeit alcoholic beverages [1, 2]. Although methanol itself is not particularly toxic, its metabolites—formaldehyde and formic acid, which are

Received: July 31, 2026 *Revised:* August 11, 2026 *Accepted:* August 26, 2026 *Online:* October 19, 2026

Correspondence: Yigit SAHIN, MD. Giresun Universitesi Tip Fakultesi, Anesteziyoloji ve Reanimasyon Anabilim Dali, Giresun, Turkiye.
Tel: +90 543 298 37 93 e-mail: yigit_sahin1988@hotmail.com

Istanbul Provincial Directorate of Health - Available online at www.northclinist.com

produced through the action of the alcohol dehydrogenase enzyme—inhibit cellular respiration, leading to metabolic acidosis, vision loss, central nervous system damage, and death [3, 4]. Due to its low cost compared to ethyl alcohol, methanol is frequently used in the production of counterfeit and illicit alcoholic beverages; in Türkiye, it has also occasionally caused individual and mass poisoning outbreaks [5, 6].

The clinical course of methanol poisoning ranges widely, from asymptomatic cases to multiple organ failure and death. Nausea and vomiting, headache, blurred vision, complete vision loss, altered consciousness, tachypnea, and severe metabolic acidosis are the primary symptoms [4, 7]. The inability to measure blood methanol levels at every center and the non-specific nature of presenting symptoms make diagnosis difficult; therefore, the diagnosis is typically made based on the history, the presence of metabolic acidosis with a high anion gap, and the exclusion of other causes [8, 9]. The cornerstone of treatment consists of administering ethanol or fomepizole to inhibit the formation of toxic metabolites, correcting acidosis, providing folic/folinic acid supplementation, and performing hemodialysis to remove toxic metabolites [10, 11]. Despite advances in treatment options, mortality rates remain high due to late hospital presentation and delayed diagnosis [12, 13].

In patients presenting to emergency departments with methanol poisoning, early recognition of clinical and laboratory features associated with poor outcomes may facilitate timely clinical assessment, appropriate monitoring, and management [14]. Although the literature reports that low pH, high anion gap, severe base deficit, high lactate, low Glasgow Coma Scale (GCS), elevated creatinine, and vision loss are associated with mortality [9, 15, 16], regional differences, access to healthcare, and patient profiles limit the generalizability of these findings. In particular, there are a limited number of retrospective analyses based on real-world data that focus on parameters at the time of emergency department presentation.

In this study, we aimed to describe and compare the clinical and laboratory characteristics of survivors and non-survivors among adult patients presenting to the emergency department with methanol poisoning and to evaluate their unadjusted associations with in-hospital mortality. The study further sought to identify exploratory clinical features at emergency department presentation that may warrant heightened clinical concern.

Highlight key points

- Real-world data identified variables associated with mortality in methanol poisoning
- Low GCS, severe acidosis, and elevated lactate were linked to poor outcomes.
- ICU non-survivors had substantially higher APACHE II and SOFA scores.
- Clinical findings at emergency department presentation may support early risk stratification.
- The findings provide practical evidence for the early management of methanol poisoning.

MATERIALS AND METHODS

Study Design and Population

This single-center, retrospective, observational cohort study included all consecutive patients aged ≥18 years who presented to the emergency department of Giresun Training and Research Hospital with a diagnosis of methanol poisoning between September 01, 2021, and May 01, 2026. The study was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study and the use of anonymized data, the requirement for informed consent was waived by the Ethics Committee. Patients younger than 18 years, pregnant women, patients with exposure to other toxic alcohols (e.g., ethylene glycol or isopropyl alcohol), and those with incomplete medical records were excluded.

Data Collection and Definitions

Clinical data were retrospectively extracted from the hospital information management system and electronic medical records using a standardized data collection form. The following variables were recorded: age, sex, season of presentation, Charlson Comorbidity Index, presenting symptoms, cause of poisoning (counterfeit/homemade alcohol or cologne), GCS score at presentation, intensive care unit (ICU) admission, requirement for invasive mechanical ventilation, hospital and ICU length of stay, blood gas and biochemical parameters obtained at emergency department presentation, brain imaging findings, treatments administered, and in-hospital mortality. For all patients, the blood gas and biochemical parameters analyzed in this study were derived from the first blood samples obtained at emergency department presentation, before the initiation of specific treatment for suspected methanol poisoning, including sodium bicarbonate administration, ethanol antidote therapy, he-

modialysis, and invasive mechanical ventilation. When multiple laboratory measurements were available, only the first pretreatment values were included in the analysis; subsequent measurements obtained after therapeutic interventions were not analyzed. For patients admitted to the ICU, Acute Physiology and Chronic Health Evaluation II (APACHE II) and Sequential Organ Failure Assessment (SOFA) scores were calculated using data obtained at the time of ICU admission.

Diagnosis and Treatment Protocol

Patients were identified from the hospital information management system and electronic medical records among adult emergency department presentations recorded with a clinical diagnosis of methanol poisoning during the study period. Because serum methanol measurements were unavailable at our institution, biochemical confirmation of methanol exposure was not possible. For inclusion in the study, patients were required to have a documented credible history of suspected methanol-containing alcohol ingestion, compatible clinical manifestations, and high-anion-gap metabolic acidosis, together with a final clinical diagnosis of methanol poisoning established during routine clinical care by the treating emergency physician and/or clinical toxicologist. Alternative causes of high-anion-gap metabolic acidosis were evaluated on the basis of the available medical history, physical examination, routine biochemical and blood gas findings, and the overall clinical context. Specifically, the medical records were reviewed for evidence of diabetic or starvation ketoacidosis, severe lactic acidosis attributable to an alternative critical illness, advanced renal failure/uremia, salicylate toxicity, and exposure to ethylene glycol or other substances capable of producing high-anion-gap metabolic acidosis. These alternative diagnoses were considered unlikely when they were not supported by the available clinical and laboratory findings and the overall presentation was judged by the treating physician and/or clinical toxicologist to be consistent with methanol poisoning. The diagnosis used for study inclusion was therefore the clinical diagnosis documented during the index hospitalization rather than a diagnosis assigned retrospectively solely for study purposes. Nevertheless, because serum methanol concentrations and osmolal gap measurements were unavailable, diagnostic misclassification cannot be completely excluded.

The anion gap at presentation was calculated using the standard formula: anion gap = $Na^+ - (Cl^- + HCO_3^-)$, based on serum electrolyte and bicarbonate values obtained at admission. Serum albumin was not routinely measured, so an albumin-corrected anion gap could not be calculated; serum and urine osmolality were not routinely measured either, so the osmolal gap could not be calculated.

Intravenous ethanol infusion was administered as an antidote when clinically indicated, and sodium bicarbonate was used for the correction of severe metabolic acidosis. Emergency hemodialysis was initiated in patients with severe or refractory metabolic acidosis, visual disturbances, renal impairment, or other clinical indications determined by the treating team. The general institutional approach to the management of methanol poisoning remained unchanged throughout the study period. Folic acid supplementation was administered to only three patients. Due to the retrospective nature of the study, the reasons for its non-administration in the remaining patients could not be reliably determined from the medical records. Treatment decisions were made by the treating clinical team as part of routine care and were not standardized by the study protocol; therefore, treatment variables were evaluated descriptively and were not interpreted as comparative measures of treatment effectiveness.

Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences version 21.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables with non-normal distributions are presented as median (interquartile range [IQR]) and were compared using the Mann–Whitney U test. Categorical variables are presented as frequencies (percentages) and were compared using the chi-square test or Fisher's exact test, as appropriate. For continuous variables, between-group effect estimates were additionally reported as Hodges-Lehmann location-shift estimates with 95% confidence intervals (CIs), calculated as non-survivors minus survivors. The 95% CIs were obtained using 10,000 bootstrap resamples. These estimates were reported to provide information on the magnitude and precision of the observed between-group differences rather than relying solely on p-values. A two-sided $p<0.05$ was considered statistically significant. All analyses were univariable (unadjusted). Due to the limited sample size (n=26) and the small number of in-hospital deaths (n=9), multivariable regression analyses were not performed; therefore, the reported variables should be interpreted as factors

associated with in-hospital mortality rather than independent predictors or risk factors. Given the exploratory nature of the study and the limited sample size, no adjustment was made for multiple comparisons. Therefore, individual P values were interpreted cautiously and were not considered confirmatory evidence, particularly in the context of multiple unadjusted comparisons. The reported effect estimates and their 95% CIs should likewise be interpreted as exploratory and unadjusted. Overall, the findings should be regarded as hypothesis-generating and require confirmation in larger prospective studies.

RESULTS

Patient Characteristics

During the study period, 26 adult patients with a clinical diagnosis of methanol poisoning were identified and screened for eligibility. None were excluded due to age <18 years, pregnancy, incomplete medical records, or exposure to another toxic alcohol. Therefore, all 26 eligible patients were included in the final analysis. Of these, 9 patients (34.6%) were non-survivors and 17 (65.4%) were survivors. The vast majority of patients were male (24/26; 92.3%), and the median age of the entire population was similar across groups (non-survivors: 51 [49.5–56.5]; survivors: 54 [48–59]; p=0.387). In terms of seasonal distribution, presentations were most common in spring and fall; no significant difference in seasonal distribution was observed between the groups (p=1.000). The cause of poisoning was counterfeit/homemade alcohol in 61.5% of cases and cologne in 38.5%, with no significant difference between groups (p=1.000) (Table 1).

Clinical Findings at Emergency Department Presentation

Loss of consciousness at emergency department presentation was significantly more frequent among non-survivors (88.9% vs. 11.8%; p<0.001). Confusion and visual disturbances were numerically more frequent among survivors, although these differences did not reach statistical significance (Table 1).

Subsequent Hospital Course and Treatment

During the subsequent hospital course, all non-survivors required invasive mechanical ventilation compared with none of the survivors (100% vs. 0%; p<0.001). All non-survivors were admitted to the ICU, compared with 64.7% of survivors. Hemodialysis and ethanol antidote therapy were also more frequently administered to non-survivors, although these differences did not reach statistical significance (Table 1). Because these variables occurred after emergency department presentation and were influenced by clinical severity and treatment decisions, they should be interpreted as descriptors of the subsequent clinical course and management rather than as baseline prognostic characteristics.

Severity Scores

The GCS score at emergency department presentation was lower among non-survivors than survivors in the overall cohort (median 4.0 [IQR, 3.5–5.5] vs. 15.0 [14.0–15.0]; Hodges-Lehmann difference, -10.0; 95% CI, -11.0 to -9.0; p<0.001). APACHE II and SOFA scores were available only for the 20 patients admitted to the ICU and were therefore analyzed exclusively within this subgroup. ICU non-survivors (n=9) had higher APACHE II scores than ICU survivors (n=11) (25.0 [20.5–25.5] vs. 11.0 [3.0–14.0]; Hodges-Lehmann difference, 14.0; 95% CI, 9.0–20.0; p=0.002) and higher SOFA scores (12.0 [9.0–12.0] vs. 4.0 [4.0–6.0]; Hodges-Lehmann difference, 6.0; 95% CI, 5.0–8.0; p<0.001) (Table 2).

Laboratory Findings

At emergency department presentation, non-survivors had lower pH than survivors (7.00 [6.88–7.08] vs. 7.11 [7.01–7.33]; Hodges-Lehmann difference, -0.17; 95% CI, -0.30–-0.03; p=0.011). Non-survivors also had higher lactate (7.1 [6.8–8.7] vs. 2.9 [1.9–5.1] mmol/L; Hodges-Lehmann difference, 4.3; 95% CI, 3.0–6.0; p<0.001), creatinine (1.52 [1.42–1.85] vs. 1.00 [0.83–1.23] mg/dL; Hodges–Lehmann difference, 0.56; 95% CI, 0.36–0.83; p=0.001), glucose (194 [137–219] vs. 123 [98–151] mg/dL; Hodges-Lehmann difference, 56; 95% CI, 10–88; p=0.029), and anion gap (27.0 [24.6–31.2] vs. 20.9 [14.6–27.8] mmol/L; Hodges-Lehmann difference, 7.3; 95% CI, 2.6–12.7; p=0.018). No statistically significant between-group differences were observed for the remaining laboratory parameters (Table 3). Given the exploratory nature of the analyses and the absence of adjustment for multiple comparisons, these findings should be interpreted cautiously.

Neuroimaging Findings

Cranial imaging was performed in 16 of the 26 patients (61.5%) based on clinical indications. Among the 16 pa-

Table 1. Baseline characteristics, clinical presentation, and subsequent hospital course according to survival status

Variables	Non-survivors (n=9)	Survivors (n=17)	p
A. Baseline demographic and clinical characteristics			
Age	51.0 (49.5–56.5)	54.0 (48.0–59.0)	0.387
Sex			
Female	0 (0.0)	2 (11.8)	0.529
Male	9 (100.0)	15 (88.2)	
Season			
Winter	2 (22.2)	4 (23.5)	1.000
Spring	3 (33.3)	6 (35.3)	
Summer	1 (11.1)	1 (5.9)	
Autumn	3 (33.3)	6 (35.3)	
Charlson Comorbidity Index	1.0 (0.5–2.5)	1.0 (1.0–2.0)	0.936
Hypertension	1 (11.1)	6 (35.3)	0.357
Coronary artery disease	0 (0.0)	2 (11.8)	0.529
COPD	1 (11.1)	1 (5.9)	1.000
Cerebrovascular disease	1 (11.1)	2 (11.8)	1.000
Liver cirrhosis	1 (11.1)	1 (5.9)	1.000
Cause of poisoning			
Counterfeit/homemade alcohol	6 (66.7)	10 (58.8)	1.000
Cologne	3 (33.3)	7 (41.2)	
Use of other substances/Medications			
None	9 (100.0)	14 (82.4)	0.686
Medication	0 (0.0)	2 (11.8)	
Medication+substance	0 (0.0)	1 (5.9)	
B. Clinical findings at emergency department presentation			
Visual disturbance	1 (11.1)	8 (47.1)	0.098
Loss of consciousness	8 (88.9)	2 (11.8)	**<0.001**
Confusion	0 (0.0)	6 (35.3)	0.063
Dyspnea	0 (0.0)	3 (17.6)	0.529
Nausea/vomiting	0 (0.0)	2 (11.8)	0.529
C. Subsequent hospital course and treatment			
ICU admission	9 (100.0)	11 (64.7)	0.063
ICU length of stay (days) (n=9/n=11)	3.0 (1.5–14.0)	3.0 (2.0–5.0)	0.729
Hospital length of stay (days)	3.0 (1.5–14.0)	4.0 (2.0–6.5)	0.913
Need for invasive mechanical ventilation	9 (100.0)	0 (0.0)	**<0.001**
Hemodialysis	9 (100.0)	12 (70.6)	0.129
Ethanol (antidote) therapy	9 (100.0)	10 (58.8)	0.058
Bicarbonate therapy	8 (88.9)	11 (64.7)	0.357
Folic acid	0 (0.0)	3 (17.6)	0.529
Pulse steroid therapy	0 (0.0)	3 (17.6)	0.529
Symptomatic treatment	0 (0.0)	5 (29.4)	0.129

Data are presented as median (interquartile range) and n (%). COPD: Chronic obstructive pulmonary disease; ICU: Intensive care unit. Fisher's exact test (2×2 comparisons) or the Freeman-Halton extension of Fisher's exact test (r×c comparisons: Season; Use of other substances/medications) and Mann-Whitney U test were used, given the small expected cell counts throughout this table. A $p<0.05$ was considered statistically significant.

Table 2. Clinical severity scores according to survival status

Score	Non-survivors (n=9)	Survivors	HL difference (95% CI)	p
GCS (n=9/n=17)	4.0 (3.5–5.5)	15.0 (14.0–15.0)	-10.0 (-11.0– -9.0)	<0.001
APACHE-II (n=9/n=11)	25.0 (20.5–25.5)	11.0 (3.0–14.0)	14.0 (9.0–20.0)	0.002
SOFA (n=9/n=11)	12.0 (9.0–12.0)	4.0 (4.0–6.0)	6.0 (5.0–8.0)	<0.001

Data are presented as median (Interquartile range). GCS was assessed at emergency department presentation in the overall cohort (n=26). APACHE II and SOFA scores were calculated only for patients admitted to the intensive care unit (n=20) and were compared between ICU non-survivors (n=9) and ICU survivors (n=11). Between-group effect estimates are presented as Hodges-Lehmann location-shift estimates with 95% confidence intervals (CIs), calculated as non-survivors minus survivors. The 95% CIs were obtained using 10,000 bootstrap resamples. p-values were calculated using the two-sided Mann–Whitney U test. All analyses were unadjusted and exploratory. GCS: Glasgow Coma Scale; APACHE II: Acute Physiology and Chronic Health Evaluation II; SOFA: Sequential Organ Failure Assessment; CI: Confidence interval.

Table 3. Pretreatment biochemical and blood gas parameters at emergency department presentation according to survival status

Parameter	Non-survivors (n=9)	Survivors (n=17)	HL difference (95% CI)	p
WBC ($\times 10^9$/L)	13.51 (8.56–21.50)	12.67 (10.29–16.26)	-0.19 (-3.93–7.65)	0.978
Hemoglobin (g/dL)	14.3 (11.8–15.2)	15.4 (13.6–16.2)	-1.2 (-2.7–0.8)	0.196
Hematocrit (%)	46.6 (37.7–49.2)	45.7 (41.7–50.2)	-1.8 (-7.9–4.9)	0.608
Platelet count ($\times 10^9$/L)	291 (105–446)	270 (207–328)	3 (-131–171)	0.978
Glucose (mg/dL)	194 (137–219)	123 (98–151)	56 (10–88)	**0.029**
Urea (mg/dL)	30.0 (23.5–66.0)	26.0 (21.5–30.5)	6 (-3–42)	0.215
Creatinine (mg/dL)	1.52 (1.42–1.85)	1.00 (0.83–1.23)	0.56 (0.36–0.83)	**0.001**
ALT (U/L)	48 (24–66)	31 (18–50)	13 (-4–34)	0.118
AST (U/L)	60 (30–180)	39 (30–66)	16 (-6–170)	0.269
Potassium (mmol/L)	5.5 (4.3–6.0)	4.6 (4.0–5.3)	0.8 (-0.6–1.5)	0.160
Sodium (mmol/L)	137 (135–141)	138 (136–140)	0 (-3–4)	0.704
Chloride (mmol/L)	101 (91–106)	102 (98–106)	0 (-8–3)	0.417
CRP (mg/L)	9.87 (3.32–119.48)	4.31 (1.34–9.87)	6.73 (-0.88–182.96)	0.195
pH	7.00 (6.88–7.08)	7.11 (7.01–7.33)	-0.17 (-0.30– -0.03)	**0.011**
pO_2 (mmHg)	42 (34–78)	39 (28–54)	7 (-7–33)	0.434
sO_2 (%)	47 (36–95)	65 (54–78)	-4 (-27–24)	0.808
pCO_2 (mmHg)	40 (36–50)	35 (25–46)	6 (-1–16)	0.124
HCO_3^-	9.5 (9.1–11.9)	11.8 (9.1–22.2)	-2.3 (-9.7–0.4)	0.246
Base deficit (mmol/L)	-19.3 (-21.6–14.0)	-17.9 (-22.8–1.4)	-2.8 (-13.7–3.1)	0.403
Lactate (mmol/L)	7.1 (6.8–8.7)	2.9 (1.9–5.1)	4.3 (3.0–6.0)	**<0.001**
Anion gap (mmol/L)	27.0 (24.6–31.2)	20.9 (14.6–27.8)	7.3 (2.6–12.7)	**0.018**

Data are presented as median (Interquartile range). Effect estimates are Hodges-Lehmann location-shift estimates calculated as non-survivors minus survivors, with 95% CIs obtained using 10,000 bootstrap resamples. p-values were calculated using the two-sided Mann–Whitney U test. All analyses are unadjusted and exploratory, and no correction for multiple comparisons was applied. CI: Confidence interval; WBC: White blood cell; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CRP: C-reactive protein.

tients who underwent imaging, 3 (18.8%) demonstrated bilateral basal ganglia abnormalities considered compatible with methanol toxicity. Because neuroimaging was performed selectively rather than routinely in the entire cohort, these findings were evaluated descriptively, and no between-group statistical comparison was performed.

DISCUSSION

In this small exploratory cohort, several variables available at emergency department presentation differed between survivors and non-survivors. Non-survivors had substantially lower GCS scores and admission pH and higher lactate, creatinine, glucose, and anion gap levels. These findings should be interpreted as unadjusted associations rather than independent prognostic factors. In contrast, ICU admission, invasive mechanical ventilation, hemodialysis, and antidotal or supportive treatments occurred after the initial presentation and were influenced by disease severity and clinical decision-making. Accordingly, these subsequent variables should be considered descriptors of the clinical course and management intensity rather than baseline prognostic characteristics.

In our study, the mortality rate was found to be 34.6%. In the literature, mortality from methanol poisoning ranges from approximately 20–55%, depending on the amount of methanol ingested, time to presentation, concomitant ethanol intake, and geographic region [5, 15, 17]. In an outbreak series in Istanbul, the mortality rate was 45.5%, while in a series of 116 cases reported from Çukurova, it was approximately 23% [16, 17]. The fact that our rate falls within this range can be explained by our status as a tertiary care center, where patients with severe clinical conditions present, and by delayed presentation due to the consumption of counterfeit alcohol.

About 92.3% of our entire population was male, and the median age was around 50 years, which is consistent with studies reporting that methanol poisoning particularly affects middle-aged men [2, 6, 18]. Age and Charlson comorbidity index did not differ significantly between survivors and non-survivors in this cohort; however, given the limited statistical power, the absence of statistically significant differences should not be interpreted as evidence that age or underlying comorbidity was unrelated to mortality.

Regarding clinical presentation, loss of consciousness was substantially more frequent among non-survivors than survivors, whereas confusion and visual disturbances were numerically more frequent among survivors but did not differ significantly between groups. Given the small sample size and multiple unadjusted comparisons, these findings should be interpreted cautiously. Nevertheless, the marked difference in loss of consciousness, together with the substantially lower GCS scores observed among non-survivors, suggests greater neurological impairment in patients with fatal outcomes, consistent with previous reports [7, 9, 19]. Within the selected subgroup of patients admitted to the ICU (n=20), ICU non-survivors had substantially higher APACHE II and SOFA scores than ICU survivors, consistent with previous reports demonstrating associations between higher illness severity scores and poor clinical outcomes in methanol poisoning [13, 20]. However, these findings should be interpreted strictly as an exploratory ICU subgroup comparison. Because APACHE II and SOFA scores were unavailable for patients who were not admitted to the ICU, and because ICU admission itself was related to disease severity, these results cannot be generalized to the overall cohort or interpreted as evidence that these scores independently predicted mortality.

Among the blood gas and biochemical parameters, non-survivors had lower admission pH and higher lactate and anion gap levels, whereas HCO_3^- and base deficit did not differ significantly between groups. These acid-base variables are biologically and mathematically interrelated and should therefore not be interpreted as independent biological or prognostic signals. Rather, the overall pattern suggests greater metabolic derangement among non-survivors, consistent with more severe poisoning. Previous studies have similarly reported associations between greater acid-base disturbance and adverse outcomes in methanol poisoning [15, 16, 21]. Although formate-mediated inhibition of mitochondrial oxidative metabolism may contribute to lactate accumulation, elevated lactate is not specific to methanol metabolism or formate toxicity and may also reflect circulatory compromise, tissue hypoxia, seizures, adrenergic stress, or other concurrent physiological disturbances in critically ill patients [3, 4]. Accordingly, the higher lactate levels observed among non-survivors should be interpreted as part of the overall metabolic and physiological derangement rather than as evidence of a specific pathogenic mechanism or an independent prognostic contribution. The higher anion gap observed among non-survivors should likewise be interpreted cautiously because it was derived from an unadjusted univariable comparison, was not corrected for serum albumin, and osmolal gap data were unavailable. Given the small sample size, multiple unadjusted comparisons, and the potential collinearity among pH, lactate, bicarbonate, base deficit, and anion gap, the present study cannot determine the independent prognostic contribution of any individual acid-base parameter. Creatinine and glucose levels were also higher among non-survivors; however, these findings should similarly be regarded as exploratory and require confirmation in larger studies [12, 22].

From a treatment perspective, all non-survivors required invasive mechanical ventilation, and nearly all underwent hemodialysis and received ethanol antidote therapy; these rates were numerically higher than in the survivors, in which dialysis and ethanol therapy were also administered to the majority of patients (70.6% and 58.8%, respectively), and the between-group differences did not reach statistical significance, likely reflecting the limited sample size. This pattern is consistent with greater overall clinical severity among non-survivors; however, the markedly higher use of invasive mechanical ventilation, hemodialysis, and ethanol antidote therapy should not be interpreted as evidence of adverse prognostic effects of these interventions. These treatments were initiated in response to greater poisoning severity and are therefore inherently susceptible to confounding by indication. In particular, the observation that all non-survivors required invasive mechanical ventilation whereas none of the survivors did most likely reflects profound neurological and physiological deterioration rather than a causal relationship between mechanical ventilation and mortality. The cornerstones of methanol poisoning treatment are alcohol dehydrogenase inhibition (with ethanol or fomepizole), aggressive correction of acidosis, and hemodialysis to remove toxic metabolites [10, 11]. Although fomepizole has a higher enzyme affinity and a lower side-effect profile compared to ethanol, its cost and limited availability mean it is not available at every center; consequently, ethanol treatment is preferred at our center, as is the case in Türkiye [10, 11]. Among the 16 patients who underwent cranial imaging, bilateral basal ganglia abnormalities compatible with methanol toxicity were identified in three patients (18.8%). Because imaging was performed selectively according to clinical indications, this finding should be interpreted descriptively and cannot be used to estimate the prevalence of neuroimaging abnormalities in the overall cohort.

When interpreting these findings in relation to previous studies, several differences in study setting and case mix should be considered. Although the associations of greater neurological impairment and more severe metabolic derangement with mortality are broadly consistent with previous reports [9, 15, 16, 19, 21], direct comparisons should be made cautiously. Our cohort was small, derived from a single tertiary-care center, and included only patients with a clinical diagnosis of methanol poisoning without biochemical confirmation. Differences across studies in referral patterns, severity at presentation, availability and timing of antidotal therapy and hemodialysis, diagnostic criteria, and time from ingestion to hospital presentation may contribute to variation in reported mortality rates and associated clinical or laboratory findings. In addition, the relatively high mortality observed in our cohort may partly reflect the clinical severity and referral characteristics of patients treated at our institution rather than the outcomes of methanol poisoning in the broader population. Therefore, the present findings should be viewed as context-specific exploratory observations and should not be assumed to be directly generalizable to other institutions, regions, or patient populations.

From a clinical perspective, greater neurological impairment and more severe metabolic derangement at presentation may identify patients warranting heightened clinical concern and close monitoring. However, the present findings should not be interpreted as validated prognostic criteria or used to define treatment thresholds, given the small sample size, unadjusted analyses, and absence of predictive-model validation. Treatment decisions, including antidotal therapy, correction of metabolic acidosis, and hemodialysis, should therefore continue to be based on established clinical indications rather than on the associations observed in this study. At the population level, effective policies aimed at preventing the production and consumption of counterfeit or illicit alcohol remain important for reducing methanol poisoning and related mortality [5, 6].

Study Limitations

The major limitations of our study include its retrospective, single-center design, the relatively small sample size, and, importantly, the absence of biochemical confirmation of methanol exposure. Serum methanol concentrations were unavailable, and serum and urine osmolality were not routinely measured; therefore, the osmolal gap could not be calculated. Although study inclusion required a compatible exposure history, clinical presentation, high-anion-gap metabolic acidosis, and a clinical diagnosis of methanol poisoning after consideration of alternative causes, the possibility of diagnostic misclassification cannot be completely excluded.

The small sample size (n=26) and limited number of in-hospital deaths (n=9) precluded reliable multivariable modeling and limited the statistical power and precision of the analyses. Accordingly, multivariable logistic regression and receiver operating characteristic analyses were not performed, and the present study cannot establish

independent prognostic factors, predictive performance, or clinically applicable cutoff values. All reported associations are unadjusted and exploratory. Because multivariable adjustment was not feasible, confounding by differences in baseline clinical severity, comorbidities, presentation delay, and other measured or unmeasured factors cannot be excluded. Consequently, the observed associations should not be interpreted as independent effects of individual variables on mortality. Although effect estimates with 95% CIs were additionally reported to characterize the magnitude and precision of between-group differences, these estimates remain unadjusted and should be interpreted within the same exploratory framework. Moreover, because multiple comparisons were performed without adjustment for multiplicity, individual P values should be interpreted cautiously, and some statistically significant findings may represent chance observations (type I error). The findings should therefore be considered hypothesis-generating and require confirmation in larger, adequately powered prospective studies.

APACHE II and SOFA scores were available only for patients admitted to the ICU and were therefore analyzed exclusively within this selected subgroup. Because ICU admission itself was related to disease severity, these comparisons may be affected by selection bias and cannot be generalized to the overall cohort or interpreted as demonstrating independent prognostic effects of these scores. Similarly, treatment-related variables, including invasive mechanical ventilation, hemodialysis, and antidotal or supportive therapies, occurred after initial presentation and were determined by clinical severity and physician decision-making. These variables are therefore susceptible to confounding by indication and should be interpreted as descriptors of subsequent clinical management rather than baseline prognostic characteristics or comparative treatment-effect data.

Another important limitation was the inability to reliably determine the interval between methanol ingestion and hospital presentation in a substantial proportion of patients. Because many exposures involved counterfeit or homemade alcoholic beverages, the timing and duration of consumption were frequently uncertain or inadequately documented. Presentation delay therefore represents an important unmeasured confounder, as patients presenting later may have had more advanced neurological and metabolic toxicity, potentially contributing simultaneously to lower pH, higher anion gap, greater impairment of consciousness, increased treatment requirements, and higher mortality. Consequently, the observed unadjusted associations cannot be separated from the potential influence of delayed presentation.

Finally, cranial imaging was performed selectively according to clinical indications in only 16 of 26 patients rather than systematically in the entire cohort. Therefore, the observed frequency of bilateral basal ganglia abnormalities among imaged patients should not be interpreted as their prevalence in the overall cohort. Ophthalmological examinations were also not systematically available for all patients, which may have resulted in underestimation of visual complications.

Conclusion

In this small retrospective cohort of patients with clinically diagnosed methanol poisoning, non-survivors showed greater neurological impairment and a general pattern of more severe metabolic derangement at emergency department presentation. These findings represent unadjusted associations and should not be interpreted as independent prognostic effects of individual clinical or laboratory variables. Within the selected ICU subgroup, ICU non-survivors had higher APACHE II and SOFA scores than ICU survivors, but these findings cannot be generalized to the entire cohort. Treatment-related variables reflected subsequent clinical management and disease severity rather than baseline prognostic characteristics. Overall, these exploratory observations may help identify clinical features warranting heightened concern but do not constitute validated prognostic criteria or a risk-stratification tool. Larger prospective studies with multivariable modeling and appropriate validation are required.

Ethics Committee Approval: This study was approved by The Giresun University Medical Research Ethics Committee (Approval No. 15; June 11, 2026).

Informed Consent: The requirement for informed consent was waived by the ethics committee due to the retrospective nature of the study and the use of anonymized data.

Conflict of Interest: The authors declare that there is no conflict of interest.

Financial Disclosure: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: The authors used ChatGPT (OpenAI) exclusively to improve the language, grammar, and readability of the manuscript.

Authorship Contributions: Conception – YS, IA; Design – YS, IA; Supervision – YS, IA; Materials – YS; Data Collection and/ or Processing – YS, IA; Analysis and/ or Interpretation – YS, IA; Literature Review – YS; Critical Review – YS, IA.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Sarıbaş K, Akpınar AA, Özşeker PE, Taşkın Ö, Dişel NR, Devecioğlu GF. Methanol poisoning in the emergency department: methanol blood levels, prognosis, and sequelae outcomes. Ir J Med Sci 2025;194:1461-70. [CrossRef]
2. Arslan K, Şahin AS. Clinical characteristics of patients with methyl alcohol intoxication followed up in the intensive care unit and factors affecting mortality. Bagcilar Med Bull 2023;8:222-9. [CrossRef]
3. Pohanka M. Toxicology and the biological role of methanol and ethanol: Current view. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 2016;160:54-63. [CrossRef]
4. Nekoukar Z, Zakariaei Z, Taghizadeh F, Musavi F, Banimostafavi ES, Sharifpour A, et al. Methanol poisoning as a new world challenge: A review. Ann Med Surg (Lond) 2021;66:102445. [CrossRef]
5. Manning L, Kowalska A. Illicit alcohol: public health risk of methanol poisoning and policy mitigation strategies. Foods 2021;10:1625. [CrossRef]
6. Aslan M, Özel Bilgi D. Factors predicting mortality in methyl alcohol intoxication: a retrospective clinical trial. Med J Bakirkoy 2024;20:48-54. [CrossRef]
7. Taşkın Ö, Açıkalın A, Dişel R. Metil alkol zehirlenmeleri. Anatolian J Emerg Med. 2022;5:37-42. [Article in Turkish] [CrossRef]
8. Bozkurt Babuş S, İraöz G, Köse A, Orekici Temel G, Tiftik RN, Toker İ. Acil serviste metil alkol zehirlenmelerinin retrospektif analizi. Tepecik Eğit. ve Araşt. Hast. Dergisi 2019;29:235-40. [Article in Turkish]
9. Ahmadi S, Ostadi A, Chitsazi H, Alikhah H. Clinical and laboratory prognostic factors associated with methanol toxicity outcomes in patients at Tabriz Sina Hospital: A retrospective study. Hum Exp Toxicol 2025;44:9603271251358632. [CrossRef]
10. Kraut JA. Approach to the Treatment of Methanol Intoxication. Am J Kidney Dis 2016;68:161-7. [CrossRef]
11. Zakharov S, Pelclova D, Navratil T, Belacek J, Komarc M, Eddleston M, et al. Fomepizole versus ethanol in the treatment of acute methanol poisoning: Comparison of clinical effectiveness in a mass poisoning outbreak. Clin Toxicol (Phila) 2015;53:797-806. [CrossRef]
12. Abdelhamid WG, El-Sarnagawy GN, Sobh ZK. Outcome assessment of acute methanol poisoning: A risk-prediction nomogram approach for in-hospital mortality. Toxicol Rep 2024;13:101817. [CrossRef]
13. Gulen M, Satar S, Avci A, Acehan S, Orhan U, Nazik H. Methanol poisoning in Turkey: Two outbreaks, a single center experience. Alcohol 2020;88:83-90. [CrossRef]
14. Çetinkaya A, Aslan Sırakaya H, Aydın K. Methyl alcohol poisoning: an analysis of 18 consecutive cases. Turk J Nephrol 2021;30:57-62. [CrossRef]
15. Aksu N, Doğan E, Çelik C, Karagöz Güzey M. Predictive Value of Base Excess for Mortality in Methanol Intoxication. Glob Emerg Crit Care 2023;2:96-101.
16. Karacabey S, Korgan MB, Efeoğlu Saçak M, Sanrı E, Denizbaşı A. Alkol zehirlenmeleri olgularının incelenmesi: retrospektif demografik analiz. Anatolian J Emerg Med 2023;6:117-20. [Article in Turkish] [CrossRef]
17. Şahin T, Bol O, Altuntaş M. Retrospective Analysis of Methyl Alcohol Poisonings Admitted to the Emergency Department. Konuralp Med J 2022;14:503-11. [CrossRef]
18. Kurtas O, Imre KY, Ozer E, Can M, Birincioglu I, Butun C, et al. The evaluation of deaths due to methyl alcohol intoxication. Biomed Res 2017;28:3680-7.
19. Aisa TM, Ballut OM. Methanol intoxication with cerebral hemorrhage. Neurosciences (Riyadh) 2016;21:275-7. [CrossRef]
20. Dikme O, Dikme O, Kurt E, Sadillioglu S, Erdede MO. Predictors of in-hospital mortality after a methanol poisoning outbreak in Istanbul. BMC Emerg Med 2026;26:182. [CrossRef]
21. Ermete Güler E, Bora ES, Efgan MG, Bilgin S. Beyond lactate: prognostic value of metabolic and biochemical parameters in methanol poisoning - a retrospective observational study. BMC Emerg Med 2026;26:71. [CrossRef]
22. Ağaçkıran İ, Ağaçkıran M. Lactate-to-albumin ratio and albumin-corrected anion gap as predictors of outcome in methanol poisoning: A retrospective observational study. PLoS One 2025;20:e0336382. [CrossRef]