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Clinical Characteristics and Prognostic Factors in Methanol Intoxication: A Retrospective Study from a Tertiary Emergency Department

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Abstract

Methanol poisoning remains a major cause of toxicological mortality due to the diagnostic challenges and delayed presentation. This study aimed to evaluate the epidemiological profile, clinical features, laboratory parameters and outcomes of patients presenting to the emergency department with methanol intoxication.

Methods

This retrospective study included patients admitted to our emergency department between January 1, 2017, and January 1, 2021, with a clinical diagnosis of methanol poisoning. Inclusion criteria were: suspected methanol ingestion, supportive history and physical findings, and high anion gap metabolic acidosis (pH < 7.3, HCO_3 < 20 mEq/L, anion gap > 20 mEq/L). Demographic data, presenting symptoms, laboratory values, and outcomes were compared between survivor and non-survivor groups.

Results

Among 41 patients, 87.8% were male with a mean age of 52.2 ± 10.8 years. In-hospital mortality was 41.5%. Altered mental status at presentation was significantly more frequent in non-survivors (64.7%) than survivors (25.0%, p = 0.011). Mortality among patients with altered consciousness reached 69.2%. Survivors presented more often with visual complaints (54.2% vs. 23.5%), though the difference was not statistically significant (0.101). Non-survivors had lower mean pulse rates (84.5 ± 23.2 vs. 106.0 ± 26.6 bpm, p = 0.004), and more severe metabolic acidosis (mean pH 6.86 ± 0.23, HCO_3 6.66 ± 3.57, base deficit −23.77 ± 5.23). ROC analysis confirmed strong associations between mortality and low pH, bicarbonate, and base deficit levels, as well as elevated lactate, glucose, and anion gap values

Conclusion

Methanol poisoning diagnosis is primarily clinical, and early detection is vital to reduce morbidity and mortality. The severity of metabolic acidosis and altered mental status at admission are key indicators of poor prognosis.

Keywords: Methanol poisoning, metabolic acidosis, antidotal therapy, emergency medicine, prognostic factors

Introduction

Methanol is a toxic alcohol commonly found in various household and industrial products, such as windshield washer fluids, gas line antifreeze, carburetor cleaners, photocopier fluids, and food-warming fuels[1]. Once ingested, methanol is metabolized in the liver to formic acid, a compound responsible for high anion gap metabolic acidosis, retinal damage, and basal ganglia injury[2]. The lethal dose is estimated to range from 30 to 240 mL (approximately 1 g/kg), with as little as 30 mL causing permanent visual impairment[3]. Exposure can lead to a broad spectrum of outcomes, from mild metabolic disturbances to irreversible blindness, coma, or death, depending on dose and treatment delay[4]. Diagnostic challenges and lack of access to fomepizole or hemodialysis further compound the problem[4].

Methanol poisoning poses a public health threat in both isolated incidents and mass outbreaks, particularly in low- and middle-income countries where illicit alcohol use and poor regulation are common[5]. High mortality rates have been reported in Egypt[6], Iran[7], Turkiye[8], Saudi Arabia[9], and Eastern Europe[10], with fatality rates ranging from 10% to over 40%. During the COVID-19 pandemic, more than 5,000 people were hospitalized and over 700 died in Iran due to methanol ingestion driven by misinformation[11]. In the emergency setting, diagnosis often relies on clinical suspicion, especially when direct methanol measurement is unavailable. Key prognostic indicators such as altered mental status, low pH, bicarbonate, and elevated lactate are essential for early risk stratification.

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This retrospective study aimed to evaluate the epidemiological characteristics, clinical presentation, laboratory findings, and outcomes of patients with methanol intoxication presenting to a tertiary emergency department. By correlating initial parameters such as consciousness level, lactate, bicarbonate, and base deficit with in-hospital mortality, the study also sought to identify practical prognostic indicators and assess the effectiveness of institutional treatment protocols. This work contributes region-specific data to a field where comprehensive hospital-based analyses are valuable and needed.

Materials and Methods

Study Design and Setting

This was a retrospective, single-center study conducted in the Emergency Department of Izmir Bozkaya Training and Research Hospital, Türkiye. The study period spanned from January 1, 2017, to January 1, 2021.

Selection of Participants

Patients were included if they met the following three diagnostic criteria:

- A suspicious history of toxic alcohol ingestion (e.g., use of unlabeled products, homemade alcohol, or involvement in a cluster of affected individuals).
- Presence of compatible clinical symptoms such as visual disturbances, altered mental status, dyspnea, chest pain, nausea, or vomiting.
- High anion gap metabolic acidosis on arterial blood gas analysis (pH < 7.3, HCO_3 < 20 mEq/L, anion gap > 20 mEq/L).

Patients were excluded if they met any of the following conditions:

- Incomplete medical records or missing relevant clinical or outcome data.
- Normal acid–base status despite suspected toxic alcohol ingestion.
- Alternative diagnosis explaining the acidosis (e.g., diabetic ketoacidosis, sepsis, renal failure, or salicylate poisoning).
- Confirmed or suspected ingestion of ethylene glycol or isopropanol instead of methanol.
- Age below 18 years.
- Discharged against medical advice before full diagnostic and therapeutic evaluation.

Data Collection and Measurements

Data were collected retrospectively from the hospital's electronic medical record system. The following variables were recorded:

- Demographics: age, sex
- Vital signs: systolic and diastolic blood pressure, pulse rate, oxygen saturation
- Glasgow Coma Scale (GCS) score at admission
- Laboratory values: arterial blood gas and serum chemistry results
- Comorbidities
- Treatment modalities initiated in the emergency department
- Admission details (ward or ICU)
- In-hospital, emergency room and 30-day mortality outcomes

Outcomes

- The primary outcome was in-hospital mortality and will be mentioned as non-survivors.
- Secondary outcomes included ER mortality and 30-day mortality.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as frequencies and percentages. Continuous variables were presented as mean ± standard deviation for normally distributed data or median (min–max) for non-normally distributed data. Distribution normality was assessed using histograms and the Shapiro Wilk test. The Independent Samples t-test was used to compare normally distributed variables. The Mann Whitney U test was used for non-normally distributed variables. Categorical variables were analyzed using the Pearson Chi-square test or Fisher's exact test where appropriate. Receiver Operating Characteristic (ROC) analysis was performed to evaluate the predictive value of laboratory parameters such as pH, bicarbonate, base deficit, lactate, glucose, and anion gap for mortality. The results were presented using Area Under the Curve (AUC) values and 95% confidence intervals. A p-value of <0.05 was considered statistically significant.

Ethical Approval

The present study was initiated after obtaining approval from the Ethics Committee of the University of Health Sciences, Izmir Bozkaya Training and Research Hospital (Approval No: 2021/119, Date: 07/07/2021).Written informed consent was waived due to the retrospective nature of the study.

Results

Patient Characteristics

A total of 41 patients diagnosed with methanol intoxication were included in the analysis during the mentioned period. The mean age was 52.22 ± 10.80 years. 36 patients (87.8%) were male and 5 (12.2%) were female. Among all patients, 24 (58.5%) survived after hospitalization. All survivors were initially admitted to the intensive care unit for follow-up. Cardiopulmonary resuscitation was performed in ER in 7 patients (17.1%). The majority (61%) of patients presented within 24 hours of ingestion. The further

Table 1: Demographics and Admission Parameters of Survivors vs. Non-survivors

Variable	All Patients (n=41)	Survivors (n=24)	Non-survivors (n=17)	p-value
Number of patients	41	24	17	
Male (%)	36 (87.8%)	21 (87.5%)	15 (88.2%)	1.00
Female (%)	5 (12.2%)	3 (12.5%)	2 (11.8%)	
Mean age (years)	52.22 ± 10.80	51.87 ± 10.61	52.71 ± 11.36	0.81
Mean systolic BP (mmHg)	146.17 ± 40.43	152.83 ± 37.55	136.76 ± 43.58	0.21
Mean diastolic BP (mmHg)	82.20 ± 20.07	87.29 ± 16.84	75.00 ± 22.49	0.52
Mean pulse (bpm)	97.12 ± 26.83	106.04 ± 26.60	84.53 ± 23.23	0.004
Mean SpO2 (%)	94.73 ± 5.76	96.58 ± 4.11	92.12 ± 6.81	0.013
Median GCS (range)	15 (3–15)	15 (15–15)	12 (3–15)	0.041
Mean arterial pressure (mmHg)	103.52 ± 25.74	109.14 ± 22.19	95.59 ± 28.88	0.097
Altered Mental Status	17(41.5%)	6(25%)	11(64.7%)	0.0264
Visual disturbance	17(41.5%)	13(54.2%)	4(23.5%)	0.101

demographic and clinical characteristics of the patients are summarized in Table 1.

Presenting Symptoms and Comorbidities

The most common symptoms were visual disturbances and altered mental status, each reported in 17 patients (41.5%). Dyspnea and gastrointestinal symptoms were observed in 17.1% and 9.8% of patients, respectively. Altered mental status was significantly more frequent in non-survivors (64.7%) compared to survivors (25.0%) ($p = 0.011$). Hypertension was the most common comorbidity (31.7%), followed by COPD and coronary artery disease (each 9.8%). Notably, 58.5% of patients had no documented comorbid conditions.

Treatment and Clinical Interventions

All patients (100%) received intravenous ethanol therapy as antidotal therapy because fomepizole was not available. IV sodium bicarbonate was administered in 95.1% of cases, and 92.7% of cases underwent hemodialysis.

Laboratory Findings

Patients presented with severe metabolic derangements. The mean arterial pH was 6.99 ± 0.21, bicarbonate was 8.7 ± 3.8 mEq/L, and base deficit was −20.74 ± 5.75. Non-survivors had significantly lower pH, bicarbonate, and base deficit values, and higher creatinine, glucose, and lactate levels compared to survivors (all $p < 0.05$). Detailed comparisons of laboratory findings are shown in Table 2.

Table 2: Laboratory Parameters in Survivors vs. Non-survivors

Variable	Survivors (n=24)	Non-survivors (n=17)	p-value
Urea (mg/dL)	31.42 ± 15.78	29.12 ± 11.25	0.822
Creatinine (mg/dL)	1.11 ± 0.28	1.30 ± 0.23	0.021
Potassium (mEq/L)	4.48 ± 0.71	4.74 ± 1.18	0.542
Sodium (mEq/L)	134.65 ± 4.26	136.11 ± 3.09	0.199
Chloride (mEq/L)	100.88 ± 4.35	101.38 ± 2.75	0.491
AST (U/L)	69.50 ± 81.75	80.35 ± 73.26	0.711
ALT (U/L)	36.12 ± 27.49	42.29 ± 37.52	0.842
Glucose (mg/dL)	148.33 ± 53.56	189.53 ± 74.95	0.032
Hemoglobin (g/dL)	15.72 ± 2.31	15.63 ± 1.88	0.905
Hematocrit (%)	47.62 ± 6.77	50.26 ± 6.17	0.177
Platelets (/μL)	290,790	315,530	0.404
pH	7.08 ± 0.15	6.86 ± 0.23	0.001
pCO_2 (mmHg)	32.78 ± 11.62	38.11 ± 14.25	0.112
HCO_3 (mEq/L)	10.14 ± 3.33	6.66 ± 3.57	0.002
Lactate (mmol/L)	4.47 ± 4.24	7.46 ± 4.74	0.009
Base deficit	-18.60 ± 5.20	-23.77 ± 5.23	0.001
INR	1.01 ± 0.11	1.01 ± 0.06	0.884
aPTT (s)	32.77 ± 7.58	31.63 ± 5.06	0.594
Anion gap	23.70 ± 6.13	27.35 ± 5.81	0.063

Mortality Data

In-hospital mortality rate was 17(41.5%). Emergency department mortality rate accounted for 26.8% and 30-day mortality rates was 41.5%. The 30-day mortality rate was 44.0% among early presenters (<24 hours) and 37.5% among late presenters (>24 hours) ($p = 0.9305$). Patients with altered consciousness had significantly higher mortality (64.7%) compared to those without (25%) ($p = 0.011$). Survivors had a higher median GCS score at admission (15 vs. 12, $p = 0.041$).

Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the predictive performance of various biochemical parameters for in-hospital mortality. The Area Under the Curve (AUC) values were as follows: base deficit (AUC = 0.801), HCO_3 (AUC = 0.784), pH (AUC = 0.783), lactate (AUC = 0.741), glucose (AUC = 0.699), and anion gap (AUC = 0.673), as illustrated in Figure 1. These results indicate that base deficit, HCO_3, and pH were the most accurate predictors of mortality among the variables analyzed.

Discussion

The predominance of male patients in our study (87.8%) aligns with prior literature, where over 80% of methanol poisoning cases have been reported in males[12-14]. This gender imbalance is often attributed to higher substance use prevalence among men.

The mean patient age in our cohort (52.2 years) was higher than in reports from Malaysia[13] (32 years), Morocco[14] (39.7 years), India[15] (38.9 years) and the United States[16]

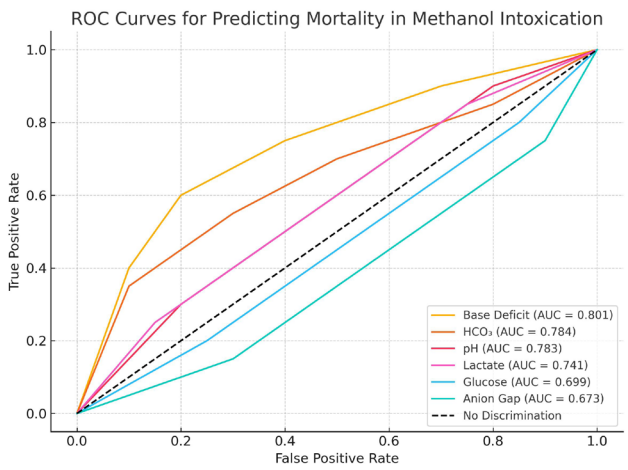

Figure 1:

(37.9 years). suggesting that methanol poisoning in Türkiye may affect an older demographic.

Timing of hospital presentation (≤24 vs. >24 hours) was not significantly associated with mortality in our study (p = 0.9305) in contrast to the findings reported by Yousefinejad et al.[17], where a significantly longer delay between methanol ingestion and hospital admission was associated with poorer outcomes (median 48 vs. 18 hours, p < 0.01).

Neurological status at admission proved to be a strong prognostic factor. Altered mental status was observed in 64.7% of non-survivors compared to 25% of survivors (p = 0.0264), with a 69.2% mortality rate among patients presenting with impaired consciousness. This is consistent with findings from Gulen et al.[18], who identified altered mental status as a key poor outcome predictor in their study. In the study by Abdelhamid et al.[19], the association between altered mental status and mortality was more clearly demonstrated. The median GCS score was 13 (IQR: 11–14) among survivors, whereas it was significantly lower in non-survivors, with a median of 4 (IQR: 3–6). These findings strongly support the link between impaired consciousness and poor prognosis in methanol poisoning and demonstrate the importance of early neurological assessment in triaging methanol-poisoned patients.

Visual complaints in our study were reported more frequently among survivors (54.2%) compared to non-survivors (23.5%), although this difference did not reach statistical significance (p = 0.101). Similarly, Sasani et al.[20] observed lower mortality rates among patients who presented with visual symptoms. This trend may be explained by the fact that visual disturbances such as blurred vision often coincide with the onset of metabolic acidosis, prompting patients to seek medical attention earlier. Consequently, earlier presentation may allow for more timely intervention, contributing to better outcomes.

Laboratory analysis revealed significantly lower pH, bicarbonate, and base excess among non-survivors, as well as elevated lactate and glucose levels. ROC analysis confirmed the prognostic utility of base deficit (AUC = 0.801), bicarbonate (AUC = 0.784), and pH (AUC = 0.783). These results agree with previous reports by Abdelhamid et al.[19] and Coskun et al.[21] who identified base deficit, lactate, and delta anion gap as significant predictors of mortality in methanol poisoning. Yurtsever et al., in a tertiary care emergency department study, similarly reported that lactate level serves as an independent predictor of mortality.[22]

In our cohort, base deficit emerged as the strongest predictor of in-hospital mortality, with an AUC of 0.801, followed by HCO_3 (AUC = 0.784), pH (AUC = 0.783), and lactate (AUC = 0.741). These findings are largely consistent with those of Abdelhamid et al., who also emphasized the prognostic value of acid-base disturbances in methanol poisoning. In their study, ROC analysis revealed an AUC of 0.816 for pH and 0.791 for serum bicarbonate (HCO_3), indicating similarly high discriminatory power. While their highest-performing variable was serum pH, in our study, base deficit slightly outperformed pH and bicarbonate. Abdelhamid et al. also incorporated these variables into a risk-prediction nomogram, underlining their clinical utility for early triage. The alignment of findings across both studies reinforces the role of metabolic derangements particularly academia as robust indicators of poor prognosis in methanol toxicity.

Our findings emphasize the importance of early detection of severe metabolic derangements particularly high anion gap acidosis and cardiovascular instability as key indicators of poor prognosis in methanol intoxication.

Limitations

This study has several limitations. First, it was a retrospective analysis based on medical records, which may be subject to documentation bias. Second, serum methanol levels could not be measured at our institution, and diagnoses were made clinically, which may have introduced diagnostic uncertainty. Third, treatment protocols were not standardized across all patients, and specific details regarding the type and duration of renal replacement therapy could not be retrieved. Fourth, all patients were treated with intravenous ethanol as the antidote, and none received fomepizole; therefore, the efficacy of fomepizole could not be evaluated in this study. Lastly, the limited sample size may have reduced the statistical power to detect subtle but potentially meaningful differences between groups.

Conclusion

Methanol intoxication continues to pose a significant clinical threat due to delayed recognition, limited diagnostic resources, and variable presentations. In this study, a high in-hospital mortality rate (41.5%) was significantly associated with altered mental status, severe metabolic acidosis, elevated lactate and glucose, and lower heart rate at

admission. Readily available parameters particularly arterial blood gas values can aid in early risk stratification and guide timely clinical decision-making, especially in settings where methanol level testing is unavailable.

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