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9 Years of emergency department experience in methanol poisoning: a retrospective, observational study

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Abstract

Aim: Methanol intoxication is a life-threatening condition frequently diagnosed and initially managed in emergency departments and may occur in epidemic outbreaks with high mortality rates. This study aimed to identify factors associated with mortality in patients with methanol intoxication. **Method:** This retrospective study included 80 patients diagnosed with methanol toxicity based on clinical findings and laboratory results over a 9-year period. **Results:** The mortality rate was 31.25% (n=25). Significant differences were observed between survivors and non-survivors were observed in glucose, pH, PCO2, HCO3, base excess, and creatinine levels (p=0.009, 0.001, 0.002, 0.001, 0.001, and <0.001, respectively). Patients admitted to the intensive care unit had significantly lower Glasgow Coma Scale scores and significantly altered base excess, HCO3, and potassium levels compared with those admitted to the ward (p=0.023, 0.002, 0.009, and 0.009, respectively). ROC analysis identified a pH ≤7.13 as the strongest predictor of mortality (AUC=0.831, p=0.0001). A Glasgow Coma Scale <15 was also significantly associated with mortality (AUC=0.815, p=0.0001). **Conclusion:** In addition to markers of metabolic acidosis such as pH, PCO_2, HCO_3, and base deficit, rapidly obtainable bedside parameters, including blood glucose and finger-prick oxygen saturation, may be useful for predicting prognosis and mortality in methanol intoxication.

Keywords: Alcohol poisoning, emergency department, ethyl alcohol, metabolic acidosis, methyl alcohol

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Metanol zehirlenmelerinde 9 yıllık acil servis deneyimi: retrospektif, gözlemsel bir çalışma

Öz

Amaç: Metanol zehirlenmesi, sıklıkla acil servislerde teşhis edilen ve ilk müdahalesi yapılan, hayati tehlike arz eden bir durumdur ve yüksek ölüm oranlarına sahip salgınlar şeklinde ortaya çıkabilir. Bu çalışma, metanol zehirlenmesi olan hastalarda mortalite ilişkili faktörleri belirlemeyi amaçlamıştır. **Yöntem:** Bu retrospektif çalışma, 9 yıllık bir dönemde klinik bulgular ve laboratuvar sonuçlarına göre metanol toksisitesi tanısı konulan 80 hastayı içermektedir. **Bulgular:** Mortalite oranı %31.25 idi (n=25). Ölen ve hayatta kalan hastalar arasında glukoz, pH, PCO2, HCO3, baz fazlalığı ve kreatinin düzeylerinde anlamlı farklılıklar gözlenmiştir (sırasıyla p=0.009, 0.001, 0.002, 0.001, 0.001 ve <0.001). Yoğun bakım ünitesine yatırılan hastalar, servise yatırılanlara kıyasla Glasgow Koma Skalası puanları açısından anlamlı olarak daha düşük ve baz açığı, HCO3 ve potasyum düzeyleri açısından farklılık göstermekteydi (sırasıyla p=0.023, 0.002, 0,009 ve 0,009). ROC analizi, pH ≤7.13'ü mortalitenin en güçlü öngörücüsü olarak belirledi (AUC=0.831, p=0.0001). Glasgow Koma Skorunun <15 olması da mortalite ile anlamlı bir şekilde ilişkiliydi (AUC=0.815, p=0.0001). **Sonuç:** pH, PCO_2, HCO_3 ve baz açığı gibi metabolik asidoz belirteçlerine ek olarak, kan şekeri ve parmak ucu oksijen saturasyonu gibi hızla elde edilebilen yatak başı parametreleri, metanol zehirlenmesinde prognoz ve mortaliteyi tahmin etmek için yararlı olabilir.

Anahtar Kelimeler: Alkol zehirlenmesi, acil servis, etil alkol, metabolik asidoz, metil alkol

Introduction

Methanol is a clear, colorless alcohol that tastes and smells like ethanol and can cause serious poisoning. It is found in industry as a solvent in antifreeze, windscreen washer fluid, aviation fuel, cologne, and perfumes. However, poisoning cases commonly arise as a result of illicit alcoholic beverage production.[1] Methanol is readily available worldwide in commercial products as well as in home-brewed alcoholic beverages. Poisoning cases occur following intentional or unintentional exposure. Although relatively rare in general, poisoning due to methanol metabolism may occur in epidemics and can cause serious morbidity and mortality.[2]

Methanol poisoning is a serious public health problem requiring initial intervention in emergency departments. Methanol is metabolized to formaldehyde via alcohol dehydrogenase, and formaldehyde is converted to formic acid. Formic acid is responsible for the toxicity. In the subsequent period, metabolic acidosis develops rapidly in the patient. Patients may develop visual impairment. In some cases, acute kidney injury, shock, multiple organ failure, and death may ocur.[3, 4] Mortality rates in methanol poisoning have been reported to range from 8% to 36%, and permanent blindness due to toxic optic neuropathy has been observed in 20% to 40% of patients who survive acute poisoning.[1]

The management of toxicity depends on the amount of exposure and requires close monitoring of laboratory parameters. Antidotes and extracorporeal methods are the cornerstones of treatment. The conversion of methanol to formic acid is attempted to be prevented by using ethanol or fomepizole, which are competitive inhibitors of the alcohol dehydrogenase enzyme. Hemodialysis is also an effective treatment method that directly removes toxic metabolites.[5-7] This study aimed to investigate the clinical and laboratory characteristics of patients presenting to the emergency department with methanol poisoning and the factors affecting mortality in these patients.

Methods

Eighty patients who presented to the Emergency Department of Mersin University Faculty of Medicine between 1 January 2016 and 31 December 2024 and were diagnosed with methanol toxicity were retrospectively reviewed through file analysis. Our center is a university hospital providing tertiary healthcare services. Patients aged ≥18 years who received their complete evaluation and treatment at our institution were included in the study. Patients transferred to our hospital from other centers or transferred to other centers were excluded from the study. Patients with a diagnosis of methanol intoxication confirmed by clinical and laboratory findings were included in the study. These findings can be summarized as follows: Patients who had recent alcohol consumption or exposure, presented with symptoms such as visual impairment or confusion, and had laboratory findings supporting a diagnosis of methanol poisoning, such as high anion gap metabolic acidosis, were included in the study.

The age, gender, presenting complaints, routes of alcohol exposure, Glasgow Coma Scale (GCS) score, history of chronic disease, laboratory tests, treatments administered, length of stay, and hospital outcome (hospitalization, discharge, death) of patients meeting the inclusion criteria were recorded using a standardized data collection form. .

Based on these data, factors that could influence the need for intensive care and in-hospital mortality were investigated.

Approval for the study was obtained from the Mersin University Health Sciences Research Ethics Committee on 03/12/2025, numbered 2025/01.

Statistical analysis

Normality of continuous measurements was tested using the Shapiro-Wilk test. Student's t-test was used for group comparisons of continuous measurements. Descriptive statistics are presented as mean and standard deviation values. Pearson's chi-square, likelihood ratio chi-square, and Fisher's exact chi-square tests were used to assess differences between categorical variables. Frequencies and percentages were provided as descriptive statistics. Cut-off points for parameters thought to affect mortality were obtained using ROC analysis. Cut-off points, sensitivity, specificity, and positive and negative predictive values were provided as descriptive statistics. Statistical significance was set at $p<0.05$.

Results

A total of 80 patients were included in the study. All patients included in the study were male. Seventy-five patients were diagnosed with methanol intoxication due to illicit alcohol consumption, while five patients were diagnosed with methanol intoxication due to drinking cologne for suicidal purposes. Fifty-seven (71.3%) patients had a history of alcohol use disorder. Twenty-five patients died, and the mortality rate was determined to be 31.25%. Sixty-five patients were admitted to intensive care, and 8 patients were followed up in the ward. Seven patients were discharged without being admitted to the hospital after treatment and follow-up from the emergency department. Seventy patients underwent hemodialysis. Ethyl alcohol was administered as an antidote to 65 patients in the emergency department and during follow-up. The general characteristics of the patients included in the study are shown in Table 1.

When comparing patients who died and survived methanol intoxication, the presence of chronic disease and chronic alcohol use were found to have no statistically significant effect on mortality.

However, survival rates were significantly higher in patients presenting with visual problems, and mortality was significantly higher in patients with altered consciousness ($p=0.001$)(Table 2).

Table 1. General characteristics of patients

	N	%
Comorbidity	41	51.2
Neurological	3	3.8
Cardiovascular	25	31.3
Respiratory	3	3.8
Renal	1	1.3
Hepatic-biliary	2	2.5
Diabetes	16	20
Other chronic diseases	6	7.5
Alcohol use disorder	57	71.3
Causative agent		
Illicit alcohol	75	93.8
Cologne	5	6.2
Central Imaging	44	55
Treatment	66	82.5
Ethyl Alcohol	65	81.3
Folic Acid	53	66.3
Bicarbonate	60	75
Fomepizole	4	5
Hemodialysis	70	87.5
Hospitalization		
Intensive Care Unit	65	81.3
Ward	8	10
Sequelae status	38	47.5
Sequelae location		
Eye	21	26.3
Neurological	16	20
Outcome		
Survive	55	68.75
Exitus	25	31.25

Table 2. Comparison of clinical characteristics, treatments, and outcomes between survivors and non-survivors

	Survivors		Non-Survivors		p
	N	%	N	%	
Comorbidity	29	52.7	12	48	0.880
Alcohol use disorder	37	67.3	20	80	0.295
Complaint					
Visual impairment	41	74.5	10	40	**0.001**
Altered consciousness	1	1.8	7	28	**0.001**
Cardiac arrest on admission	0	0	2	8	**0.001**
Central Imaging	26	47.3	18	72	0.069

Table 2. continiued. Comparison of clinical characteristics, treatments, and outcomes between survivors and non-survivors

	Survivors		Non-Survivors		p
	N	%	N	%	
Antidote treatment	43	76.4	24	96	0.029
Hemodialysis	46	83.6	24	96	0.160
Hospitalization					
Ward	8	14.5	0	0	0.038
Intensive Care Unit	42	76.4	23	92	

As expected, GCS was lower in non-survivors than in survivors, and this difference was statistically significant. Glucose, pH, PCO2, HCO3, base excess, and creatinine levels were laboratory parameters that differed significantly between survivors and non-survivors. (p-values were 0.009, 0.001, 0.002, 0.001, 0.001, and 0.001, respectively) (Table 3).

When patients admitted to intensive care were compared with those admitted to the ward, GCS, BE, HCO3, and potassium levels showed statistically significant differences (p-values were 0.023, 0.002, 0.009, and 0.009, respectively) (Table 3).

Table 3. Comparison of age, admission consciousness level, vital signs, and laboratory parameters between survivors and non-survivors by hospitalization location

	Non-Survivors (n=25)	Survivors (n=55)	p	ICU (n=65)	Ward (n=8)	p
Age	56.1 ± 9.1	53.1 ± 13.2	0.309[a]	55.37 ± 10.74	52.38 ± 15.53	0.482[a]
GCS	11 [3-13.5]	15 [15-15]	**<0.001[b]**	15 [9-15]	15 [15-15]	**0.023[b]**
SBP	132.12 ± 40.51	146.15 ± 29.09	0.082[a]	141.25 ± 35.67	153.38 ± 24.02	0.354[a]
DBP	82.68 ± 31.07	88.56 ± 17.55	0.383[a]	87.00 ± 24.18	89.38 ±14.28	0.787[a]
Pulse rate	99.80 ± 29.70	91.47 ± 15.87	0.197[a]	94.08 ± 21.20	93.50 ± 23.90	0.943[a]
RR	20.0 [19.5-25.5]	20.0 [20.0-22.0]	0.974[b]	20 [20-23]	20 [18.5-21.75]	0.559[b]
Temperature	36.50 ± 2.80	36.47 ± 2.51	0.641[a]	36.47 ± 0.25	36.40 ± 0.28	0.455[a]
Glucose	186.0 [126.0-289.5]	129 [108-162]	**0.009[b]**	138.0 [117.0-217.5]	136.0 [97.5-164.0]	0.406[b]
pH	6.89 ± 0.21	7.15 ± 0.17	**0.001[a]**	7.03 ± 0.19	7.28 ± 0.15	**0.001[a]**
PCO2	44.56 ± 20.41	29.81 ± 10.81	**0.002[a]**	33.34 ± 15.07	29.87 ± 11.14	0.531[a]
HCO3	8.0 [4.7-10.0]	10.3 [9.0-15.8]	**0.001[b]**	9.70 [6.15-11.45]	14.80 [9.85-24.75]	**0.009[b]**

Table 2. continiued. Comparison of age, admission consciousness level, vital signs, and laboratory parameters between survivors and non-survivors by hospitalization location

	Non-Survivors (n=25)	Survivors (n=55)	p	ICU (n=65)	Ward (n=8)	p
Be	-21.0 [-27.0-18.2]	-16.9 [-21.4-12.0]	**0.001[b]**	-19.0 [-23.5-15.9]	-10.0 [-17.7-1.8]	**0.002[b]**
Creatinine	1.3 [1.2-1.5]	0.98 [0.78-1.16]	**<0.001[b]**	1.10 [0.84-1.40]	0.825 [0.633-1.07]	**0.023[b]**
CRP	4.44 [1.1-20.3]	4 [1.63-9.72]	0.779[b]	4.00 [1.58-17.90]	5.45 [3.04-7.71]	0.744[b]
Sodium	137.24 ± 7.40	135.89 ± 4.08	0.400[a]	136.00 ± 5.66	136.37 ± 3.37	0.856[a]
Potassium	5.10 ± 1.06	4.71 ± 0.85	0.088[a]	4.99 ± 0.89	4.11 ± 0.68	**0.009[a]**

GCS: Glasgow Coma Scale, Be: Base excess, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, RR: Respiratory rate Sd: standart derivation, ICU: Intensive Care Unit [a]: p-value from the Student's t-test, with values given as mean ± standard deviation. [b]: p-value from the Mann-Whitney U test, with values given as median (IQR).

In the ROC analysis conducted for factors contributing to mortality, a pH value of 7.13 or below was identified as the parameter with the highest AUC value (p=0.0001, AUC=0.831). A GCS below 15 was also found to be significant in predicting mortality (p=0.0001, AUC=0.815). The results of the ROC analysis conducted for factors affecting mortality are shown in Figure 1 and Table 4.

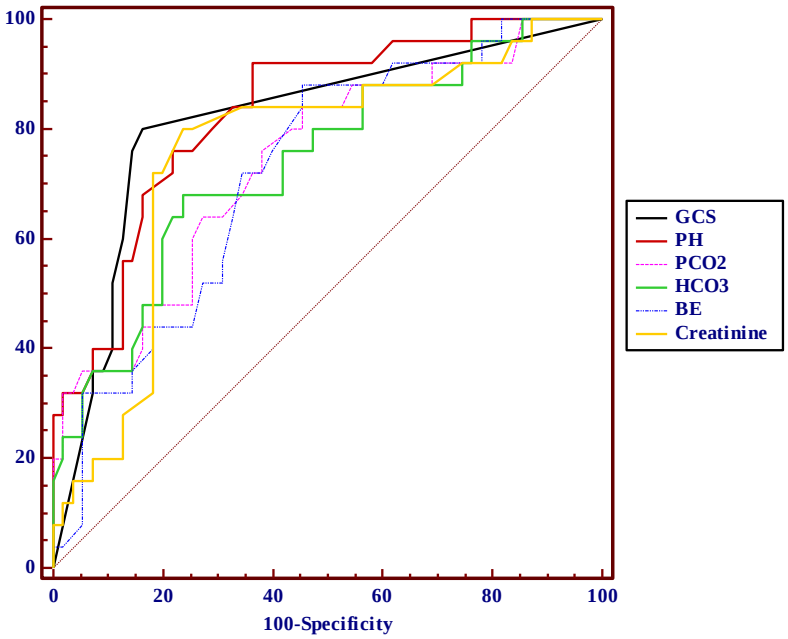

Figure 1. ROC analysis performed in terms of factors affecting mortality

Table 4. ROC analysis performed in terms of factors affecting mortality

		Cut-off	AUC (p)	Sensitivity	Specificity	PPV	NPV
Mortality	GCS	≤ 14	**0.815 (0.0001)**	80.00 (59.3 – 93.1)	83.64 (71.2 – 92.2)	69.0 (49.2 – 84.7)	90.2 (78.6 – 96.7)
	Saturation	≤ 92	**0.667 (0.0076)**	44.00 (24.4 – 65.1)	94.55 (84.9 – 98.8)	78.6 (49.2 – 95.1)	78.8 (67.0 – 87.9)
	Glucose	>178	**0.683 (0.0064)**	52.00 (31.3 – 72.2)	83.64 (71.2 – 92.2)	59.1 (36.4 – 79.3)	79.3 (66.6 – 88.8)
	Number of hemodialysis sessions	>1	**0.698 (0.0028)**	48.00 (27.8 – 68.7)	87.27 (75.5 – 94.7)	63.2 (38.4 – 83.6)	78.7 (66.3 – 88.1)
	pH	≤ 7.13	**0.831 (0.0001)**	92.00 (73.9 – 98.8)	63.64 (49.6 – 76.2)	53.5 (37.7 – 68.8)	94.6 (81.8 – 99.2)
	PCO2	>29.8	**0.741 (0.0001)**	84.00 (63.9 – 95.4)	54.55 (40.6 – 68.0)	45.7 (30.9 – 61.0)	88.2 (72.5 – 96.6)
	HCO3	≤8.9	**0,740 (0.0001)**	68.00 (46.5 – 85.0)	76.36 (63.0 – 86.8)	56.7 (37.4 – 74.5)	84.0 (70.9 – 92.8)
	BE	≤ -17.6	**0.724 (0.0001)**	88.00 (68.8 – 97.3)	54.55 (40.6 – 68.0)	46.8 (32.1 – 61.9)	90.9 (75.6 – 98.0)
	Creatinine	>1.16	**0.757 (0.0001)**	80.00 (59.3 – 93.1)	76.36 (63.0 – 86.8)	60.6 (42.1 – 77.1)	89.4 (76.9 – 96.4)

PPV: Positive prective value, NPV: Negative predictive value, AUC: Area under curve, GCS: Glasgow Coma Scale

Discussion

Methanol poisoning is a form of poisoning that can be highly fatal and requires serious medical care. Mortality rates reported in the literature range from 23% to 30%.[8, 9] In our study, the mortality rate was found to be 31.25%. While a study conducted in Estonia reported a hemodialysis application rate of 71.1% among patients, this rate was 87.5% in our study. In the same study, ethanol was administered as an antidote to 87% of patients, whereas in our study, this rate was 81.3%.[9] Although our results are generally consistent with the literature, the ease of access to hemodialysis in our hospital and the high cost and limited availability of fomepizole may have influenced the results.

Methanol exposure damages the optic nerve and retina. This damage is thought to result from the inability to meet the energy needs of the tissues. Oxidative phosphorylation is disrupted, and axonal plasma transmission is interrupted. As a result, edema occurs in the optic disc and the diameter of the optic nerve sheath increases.[10] The literature reports that visual impairment was the presenting complaint in 62.7% of methanol poisoning cases and that the rate of recovery without sequelae was higher in patients presenting with this complaint.[11] In our study, 63.7% of patients had visual impairment at the time of presentation, and the mortality rate was lower in patients with visual impairment as the primary complaint compared to patients with neurological disorders. We believe that this result may be due to visual impairment being a symptom that is more easily recognized by patients and is more bothersome. This may represent an important clinical red flag in recognizing methanol poisoning.

The literature reports that low GCS, low pH, low HCO3, high PCO2, and hyperglycemia may be predictors of poor clinical outcomes in methanol poisoning.[12-15] We obtained similar results in our study. The mean pH value at presentation was higher in survivors compared to non-survivors. PCO2 levels are also expected to be higher in non-survivors due to their impaired ability to compensate for acidosis. The decrease in HCO3 and high anion gap metabolic acidosis occur in methanol poisoning due to the increase in formic acid. Hyperglycemia may be related to the release of stress hormones or pancreatic involvement.[12-14] Our study is consistent with the literature in terms of all these results.

A multicenter study by Paasma and colleagues reported that low pH (<7), GCS <8, and inadequate hyperventilation in methanol intoxications were predictors of poor outcomes.[16] Another study found that metabolic acidosis (pH < 7.07), low GCS, and high lactate levels were associated with adverse clinical outcomes such as sequelae development and mortality. It was emphasized that mortality rates increased as the severity of metabolic acidosis increased.[11] In our study, we observed that patients admitted to intensive care had lower GCS scores and more pronounced acidosis in blood gas analysis. We concluded that pH<7.14, base deficit <-17.7, and HCO3<9 may be warning signs for mortality.

Another study conducted in Türkiye reported that low GCS, pH<7.11, high lactate levels, high blood sugar levels, and low oxygen saturation may also be poor prognostic indicators in methanol intoxications. It has been reported that low oxygen saturation in methanol poisoning may be due to the irreversible effects of methanol on the respiratory system, while hyperglycemia may be related to the adaptive stress state developed by the organism.[11,17] Another study emphasized that hyperglycaemia may be an independent prognostic factor in methanol intoxication.[18] However, in the study by Zakharov et al., although blood glucose levels differed significantly between survivors and non-survivors, hyperglycemia was not identified as an independent prognostic factor in methanol poisoning.[19] In our study, glucose levels > 178 and saturation < 93% were found to be significant in terms of mortality. This suggests that, in methanol poisoning, finger saturation and blood glucose, along with level of consciousness and degree of acidosis, are parameters that can guide clinicians in predicting patient prognosis.

Limitations

The single-center and retrospective nature of the study is a significant limitation. Although a long period of 9 years was covered, the small number of patients (80) may have affected the reliability of the data. We were unable to account for the time between the patients' presentation and the initiation of dialysis. We were unable to study lactate levels due to a lack of data. Similarly, blood methanol levels could not be included in the study, as our center did not have the facilities to measure them. This situation may have led to the inclusion of severe ethanol poisonings that mimic methanol toxicity in the study. Multicenter prospective studies are needed in this area.

Conclusion

Our findings suggest that, in addition to parameters that may be indicators of metabolic acidosis, such as pH, PCO2, HCO3, and base deficit, tests that can be performed at the bedside and provide rapid results, such as glucose and finger-prick saturation, may also be useful for predicting prognosis and mortality. These parameters may assist clinicians in managing these patients, who have a very high mortality rate and require serious medical care.

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Availability of data: The data that support the findings of this study are available from the corresponding author, AY, upon reasonable request.

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