



# Factors Predicting Mortality in Methyl Alcohol Intoxication: A Retrospective Clinical Trial

## Metil Alkol İntoksikasyonunda Mortaliteyi Öngören Faktörler: Retrospektif Bir Klinik Çalışma

Murat Aslan, Deniz Özel Bilgi

University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital, Clinic of Anesthesiology and Reanimation, İstanbul, Türkiye

### ABSTRACT

**Objective:** Methyl alcohol, which is quite cheap compared to ethyl alcohol, can be used to produce imitated alcohol. For this reason, cases of methyl alcohol intoxication can be observed from time to time. Although it is rare, it is an emergency that needs to be diagnosed and treated early because of its high mortality.

**Methods:** In this study, 22 patients aged  $\geq 18$  years who were admitted to the intensive care unit (ICU) due to methyl alcohol intoxication between 2015 and 2022 were included. The patients were divided into 2 groups, survivor and non-survivor, and compared retrospectively in terms of factors predicting mortality.

**Results:** Except for one of the 22 patients included in the study, all patients developed methyl alcohol intoxication because of the use of imitated alcohol. Only one patient had a history of using perfume for suicidal purposes. Of the patients who developed methyl alcohol intoxication, 13 (59%) were in the non-survivor group and 9 (41%) were in the survivor group. While the rate of invasive mechanical ventilation needed before the ICU was 55.6% in the survivor group, it was 100% in the non-survivor group, and there was a statistically significant difference between them ( $p < 0.017$ ). In the non-survivor patient group, blood  $\text{HCO}_3^-$  and pH levels were found to be significantly lower after ICU admission ( $p = 0.002$ ,  $p = 0.008$ ). At the same time, blood creatinine, potassium, and total bilirubin levels were significantly higher ( $p = 0.002$ ,  $p = 0.007$ ,  $p < 0.035$ ). Acute physiology and chronic health evaluation-II and sequential organ failure assessment scores after ICU admission were also significantly higher in the non-survivor group than in the survivor group ( $p = 0.005$ ,  $p = 0.035$ ).

**Conclusion:** It was determined that in patients who died due to methyl alcohol intoxication, deeper metabolic acidosis and irreversible multi-organ failure developed during the period until ICU admission. The acute physiology and chronic health evaluation-II and sequential organ failure assessment scores were both effective in predicting mortality.

**Keywords:** Methyl, alcohol, intoxication, mortality

### ÖZ

**Amaç:** Etil alkolle göre oldukça ucuz olan metil alkol, dolandırıcılar tarafından sahte alkol üretiminde kullanılabilir. Bu nedenle zaman zaman metil alkol zehirlenmesi olguları görülebilmektedir. Nadir görülmekle birlikte mortalitesinin yüksek olması nedeniyle erken teşhis ve tedavisi gereken acil bir durumdur.

**Gereç ve Yöntem:** Bu çalışmada 2015-2022 yılları arasında metil alkol intoksikasyonu nedeniyle yoğun bakım ünitesine (YBÜ) yatırılan  $\geq 18$  yaş 22 hasta çalışmaya dahil edildi. Hastalar sağ kalan ve sağ kalmayan olmak üzere 2 gruba ayrılarak mortaliteyi öngören faktörler açısından retrospektif olarak karşılaştırıldı.

**Bulgular:** Çalışmaya dahil edilen 22 hastadan biri dışında tüm hastalarda sahte alkol kullanımı nedeniyle metil alkol intoksikasyonu gelişti. Sadece bir hastada intihar amaçlı parfüm kullanma öyküsü vardı. Metil alkol intoksikasyonu gelişen hastaların 13'ü (%59) sağ kalmayan grupta, 9'u (%41) sağ kalan grupta yer aldı. Sağ kalan grubunda YBÜ öncesi invaziv mekanik ventilasyon ihtiyacı oranı %55,6 iken, sağ kalmayan grupta %100'dü ve aralarında istatistiksel olarak anlamlı fark vardı ( $p = 0,017$ ). Sağ kalan grupta YBÜ'ye yatış sonrası kan  $\text{HCO}_3^-$  ve pH düzeyleri istatistiksel olarak anlamlı derecede düşük bulundu ( $p = 0,002$ ,  $p = 0,008$ ). Aynı zamanda kan kreatinin, potasyum ve total bilirubin düzeyleri istatistiksel olarak anlamlı

**Address for Correspondence:** Murat Aslan, University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital, Clinic of Anesthesiology and Reanimation, İstanbul, Türkiye  
Phone: +90 541 714 10 60 E-mail: aslmurat@hotmail.com ORCID ID: orcid.org/0000-0002-0499-6859

**Cite as:** Aslan M, Özel Bilgi D. Factors Predicting Mortality in Methyl Alcohol Intoxication: A Retrospective Clinical Trial.  
Med J Bakirkoy 2024;20:48-54

**Received:** 11.01.2023  
**Accepted:** 29.05.2023

derecede yüksekti ( $p=0,002$ ,  $p=0,007$ ,  $p<0,035$ ). YBÜ'ye kabulü sonrası akut fizyoloji ve kronik sağlık değerlendirmesi-II ve sıralı organ yetmezliği değerlendirmesi skorları da sağ kalmayan grupta sağ kalan gruba göre istatistiksel olarak anlamlı derecede yüksek bulundu ( $p=0,005$ ,  $p=0,035$ ).

**Sonuç:** Metil alkol intoksikasyonu nedeniyle mortal seyreden hastalarda YBÜ kabulüne kadar geçen sürede daha derin metabolik asidoz ve geri dönüşümsüz çoklu organ yetmezliği geliştiği belirlendi. Akut fizyoloji ve kronik sağlık değerlendirmesi-II ve sıralı organ yetmezliği değerlendirmesi skorlarının her ikisi de mortaliteyi öngörmede etkilidir.

**Anahtar Kelimeler:** Metil, alkol, intoksikasyon, mortalite

## INTRODUCTION

Although methyl alcohol intoxication is a rare condition, it should be diagnosed and treated early because of its high mortality. Even 8-10 mL of methanol taken from the body is toxic. Approximately 25-30 mL of methanol can lead to intoxication that can cause permanent blindness, and ingestion of 1 mL/kg or 100 mL of methanol is fatal (1,2). Methyl alcohol is a product that is generally used for industrial purposes, and it often causes poisoning by taking it for suicidal purposes or by accident. Windshield washer fluid, gas line antifreeze, carburetor cleaner, copier fluid, perfumes, and many other industrial substances contain methyl alcohol (3). In addition, because it is cheap, it can be used by fraudsters in the production of imitated alcohol, as is seen in our country, and can cause intoxication cases from time to time. Because methyl alcohol is a colorless and odorless substance, it is not possible to distinguish it from ethyl alcohol when taken orally.

Methyl alcohol intoxication often occurs after oral ingestion but can also occur through inhalation and skin absorption. Methyl alcohol is quickly absorbed from the gastrointestinal tract after oral administration and reaches peak blood concentrations within 30-60 min. Once absorbed, methyl alcohol has a volume of distribution similar to that of body water. They are then metabolized in the liver or excreted renally. The metabolism of methyl alcohol to formic acid leads to increased anion gap metabolic acidosis. Afterwards, formic acid ultimately inhibits oxidative phosphorylation, which then leads to anaerobic respiration and thus an increase in lactate levels (2,4-6). Although the clinical signs and symptoms related to methyl alcohol intoxication may start in as little as 40 minutes, they can last up to 72 hours. This period depends on the type of exposure, amount, and taking it together with its antidote, ethanol (2,7).

Although blood levels should be checked for a definitive diagnosis of methyl alcohol intoxication, this is not possible in every center. Visual impairment, hyperosmolarity, and increased anion gap metabolic acidosis are the most important findings of methyl alcohol intoxication. Along with these findings, a history of consumption of products containing methyl alcohol and the exclusion of other

factors likely to cause metabolic acidosis with an increased anion gap can be used in the diagnosis of methyl alcohol intoxication (4,5). For treating methyl alcohol intoxication, the administration of ethyl alcohol or fomepizole is the first basic step of treatment. The affinity of ethyl alcohol to the alcohol dehydrogenase enzyme is 10-20 times higher than that of methyl alcohol, and 100 mg/dL blood level of ethyl alcohol almost stops the metabolism of methyl alcohol (8). The half-life of methyl alcohol, which is 14-30 h alone, can be extended to 43-96 h with ethyl alcohol (4). However, although fomepizole has 500-1000 times more affinity for alcohol dehydrogenase than ethyl alcohol, its cost is high and cannot be obtained in every center. Slowing down the metabolism of methyl alcohol is important in terms of slowing down the rate of increase in the serum level of toxic metabolites that will occur and allowing them to be removed by hemodialysis without irreversible damage to the body. All alcohols can be easily removed from the body by hemodialysis because they have low molecular weight, low body distribution volume, and are not bound to proteins (5,7,9).

In our study, we aimed to examine the factors predicting mortality in patients hospitalized in the intensive care unit (ICU) due to methyl alcohol intoxication as a primary cause. Second, we aimed to look at the change in the annual number of ICU admissions and the length of stay in the ICU due to methyl alcohol intoxication. In addition, we aimed to show the pathognomonic findings of methyl alcohol intoxication.

## METHODS

### Design and Study Population

The study was conducted retrospectively on patients over the age of 18 with a diagnosis of methyl alcohol intoxication admitted to the ICU between 01.10.2015 and 01.10.2022. Other toxic alcohol intoxications, patients under 18 years of age, and pregnant women were excluded from the study (Figure 1).

The study was conducted in full accordance with local Good Clinical Practice guidelines and current legislation. Ethical approval was obtained from the Ethics Committee of the

University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital (decision number: 2022-21-14, date: 07.11.2022).

### Data Collection

Study data were obtained retrospectively from the 'IcmdSoft-Metavision/qlinICU Clinical Decision Support Software' system. A history of visual impairment, cardiac arrest, need for invasive mechanical ventilation (IMV), and intermittent hemodialysis (IHD) application were recorded before ICU admission. Then, demographic (gender, age, weight, height, body mass index), comorbidity, clinical findings, complications, laboratory, in-hospital mortality and other data of the patients after ICU admission were obtained from the decision support system and recorded. Acute physiology and chronic health evaluation-II (APACHE-II), sequential organ failure assessment (SOFA) and Charlson comorbidity index (CCI) scores were calculated using the patients' available data at ICU admission (Supplement Table).

### Protocol

Patients who apply to our emergency department because of the suspicion of methyl alcohol intoxication and whose general clinical condition is poor are immediately consulted. Because the blood methyl alcohol level and routine blood osmolarity could not be measured in our center, the diagnosis of the patients who applied to our emergency department with the suspicion of methyl alcohol intoxication was as follows:

- A history of substance intake that may contain methyl alcohol before coming to the emergency department and strong clinical suspicion.
- Increased anion gap metabolic acidosis (pH <7.30,  $\text{HCO}_3^- < 15$ , anion gap >15, base deficit <-3) during follow-up to the emergency department.

- It was established by excluding other possible causes of increased anion gap metabolic acidosis (ethylene glycol intoxication, salicylic acid intoxication, paracetamol intoxication, lactic acidosis, diabetic ketoacidosis, other).

After the patients came to the emergency room, blood and urine toxic panels were studied to differentiate other possible toxic causes. Afterwards, an intravenous (IV) infusion of 10% ethyl alcohol at 1-2 mL/kg/h (followed by 7.5-8 mL/kg IV loading in 1 hour) was administered. The target blood level of ethyl alcohol was more than 100 mg/dL. In addition, 1-2 mL/kg  $\text{NaHCO}_3$  and 1000-2000 cc crystalloid IV bolus administration were applied to correct metabolic acidosis (in those with pH <7.30).

It was thought that urgent hemodialysis should be applied in cases with increased anion gap (>30 mEq/L) or base deficit (<-15), visual impairment, renal failure, and refractory metabolic acidosis (pH <7.25). If these patients did not need IMV support because of hemodynamic instability and coma, ICU was accepted after IHD was applied first and continuous renal replacement therapy (CRRT) support was continued. Otherwise, ICU was accepted without applying IHD, and CRRT application was initiated. Ethanol infusion was increased to 2-3 mL/kg/h to maintain a serum ethanol level more than 100 mg/dL during hemodialysis. Hemodialysis treatment was continued until metabolic acidosis improved.

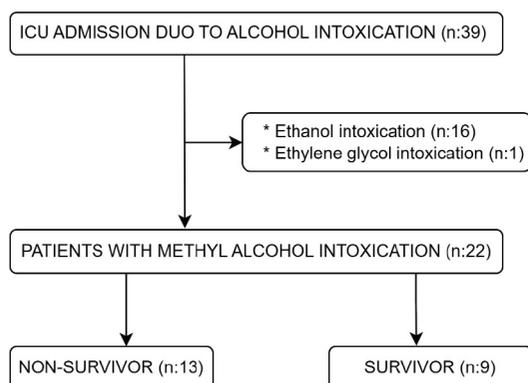
To increase the metabolism of formic acid, 50 mg/day folic acid was administered enterally until metabolic acidosis resolved.

### Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test was used to determine whether the data were normally distributed. Categorical variables are given as frequency (n) and percentage (%), numerical variables mean  $\pm$  standard deviation or median with interquartile range. Independent-samples t-test was used to compare the quantitative variables with normal distribution between the two groups. Mann-Whitney U test was used for comparisons between two groups of quantitative variables that did not show normal distribution. Fisher's Exact test was used to compare categorical variables. Statistical significance was set as  $p < 0.05$ .

## RESULTS

Between 2015 and 2022, 22 patients admitted to the ICU because of methyl alcohol intoxication were evaluated. The majority of these patients [59% (n=13)] were intoxication cases accepted in 2021 (Figure 2). One of these patients



**Figure 1.** Study flowchart  
ICU: Intensive care unit

developed intoxication because of voluntarily ingesting perfume. All the other patients were intoxicated because of imitated alcohol consumption.

One-month in-hospital mortality was 59% (n=13) in 22 patients included in the evaluation (Table 1). The patients were analyzed in terms of factors predicting mortality in 2 groups as survivor (n=9) and non-survivor (n=13). There was no statistically significant difference between the two patient groups in terms of demographic data and CCI score (Table 1).

The need for IMV support before ICU was 55.6% (n=5) in the survivor group and 100% in the non-survivor group, with a statistically significant difference between them (p=0.017). Again, 3 patients (23.1%) in the non-survivor patient group and 1 patient (11%) in the survivor patient group had a history of cardiac arrest before ICU admission, but the difference between them was statistically insignificant. However, no statistically significant difference was observed in terms of the development of visual impairment and IHD use before ICU (Table 1).

APACHE-II and SOFA mortality scores after ICU admission in the non-survivor group were significantly higher than those in the survivor group (p=0.005, p=0.035). At the same time both blood creatinine, potassium, and total bilirubin levels were higher and blood pH and HCO<sub>3</sub> levels were lower in the non-survivor group (p=0.002, p=0.007, p<0.035). There was no statistically significant difference between the two groups in terms of other laboratory parameters (Table 1).

Total ICU hospitalization time was found to be statistically lower in the non-survivor patient group than in the survivor group [3 (2-5.5), 6 (4.5-15.5)] (Table 1).

Bilateral basal ganglia bleeding, which is a pathognomonic finding for methyl alcohol intoxication, was detected in one patient from the non-survivor group (Figure 2).

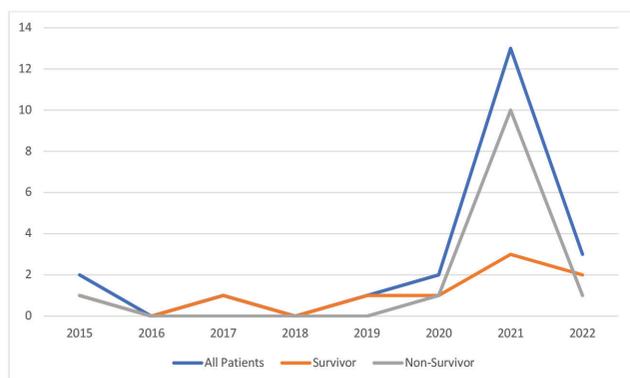


Figure 2. The number of methyl alcohol intoxication cases in the last 8 years

Table 1. Demographic and clinical characteristics of patients

	Non-survivor (n=13)	Survivor (n=9)	p-value
Age	47±13	48±13	0.863
Male, n (%)	8 (61.5)	7 (77.8)	0.648
Body mass index	24.3±2.3	25.5±3.7	0.373
CCI	1 (0-2)	1 (0-2)	0.7
<b>Before ICU, n (%)</b>			
Invasive MV need	13 (100)	5 (55.6)	0.017*
Cardiac arrest	3 (23.1)	1 (11)	0.61
Defect of vision	5 (38.5)	5 (55.6)	0.666
IHD use	1 (7.7)	4 (44)	0.116
SOFA score	9 (8.5-10)	8 (6-9)	0.035*
APACHE-II	28±5	20±6	0.005*
Urea (mg/dL)	36 (22-57)	21 (16-35.3)	0.089
Creatinine (mg/dL)	1.8±0.7	0.94±0.34	0.002*
Total bilirubin (mg/dL)	1.2 (0.85-1.5)	0.6 (0.44-2.1)	0.035*
AST (U/L)	66 (41-230)	135 (33-196)	0.815
ALT (U/L)	41 (25-110)	33.6 (11.3-66)	0.367
Na (mmol/L)	139±9	137±5	0.511
K (mmol/L)	5.3±1.2	4±0.7	0.007*
Cl (mmol/L)	110±7	110±5	0.725
Procalcitonin (ng/mL)	0.14 (0.06-0.34)	1.3 (0.22-2.6)	0.160
CRP (mg/L)	3.3±2.5	9±7.5	0.057
pH	6.78 (6.71-6.9)	7.11 (6.85-7.27)	0.008*
PCO <sub>2</sub> (mmHg)	36±14	29±10	0.171
PO <sub>2</sub> (mmHg)	179±64	148±74	0.3
SO <sub>2</sub> (%)	96 (96-98)	98 (88-99)	0.316
HCO <sub>3</sub> (mmol/L)	5 (4.5-6.2)	9.1 (6.5-13.5)	0.002*
Base deficit (mmol/L)	-27.9±9.9	-21.7±7	0.124
Lactate (mmol/L)	9.6±4.7	5.8±4.7	0.077
Glucose (mg/dL)	198±133	106±76	0.079
WBC (x10 <sup>9</sup> /L)	24.5±9.5	19.5±10.8	0.264
Hematocrit (%)	44±6	40.1±8.5	0.221
Platelet (x10 <sup>9</sup> /L)	289±86	220±86	0.097
ICU time (day)	3 (2-5.5)	6 (4.5-15.5)	0.034*

ICU: Intensive care unit, CCI: Charlson comorbidity index, MV: Mechanical ventilation, IHD: , SOFA: Sequential organ failure assessment, APACHE-II: Acute physiology and chronic health evaluation-II, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, CRP: C-reactive protein, WBC: White blood cell Fisher's Exact test [n(%)], independent-samples t-test (mean ± standard deviation), Mann-Whitney U test [median (interquartile range)], \*p<0.05

## DISCUSSION

The mortality rate of our study was found to be slightly higher than that of other studies. In various studies on methyl alcohol intoxication, the mortality rate varies between 20% and 55%, depending on factors such as the amount of methyl alcohol intake of the patient, the duration of admission to the hospital, concomitant ethanol intake, and different geographical regions (10-14). As in many centers, because blood methyl alcohol level was not measured in our center, its relationship with mortality could not be evaluated. The critical period between the onset of symptoms and hospitalization may be too extended because the unconsciousness and other symptoms that develop due to methyl alcohol intoxication after imitated alcohol intake are similar to the symptoms that develop after ethanol intake. In a retrospective study conducted on 383 people who died due to methyl alcohol intoxication between 2002 and 2010 in Türkiye, 64.7% (n=248) of these deaths died at home, 7.5% (n=29) died in open areas, 9.9% (n=38) died in other areas, and only 12.8% (n=49) died in the hospital (15). In our patients, although it could not be detected clearly, they had a history of methyl alcohol intake 1 day or more, and their admission to the hospital was quite delayed. Therefore, we believe that our mortality rate is higher. The fact that the need for IMV on arrival at the hospital is so high and that some of them even reached the level of cardiac arrest shows that these patients are quite late for treatment.

Visual impairment, which is a finding specific to methyl alcohol intoxication and can be permanent, was observed in 5 patients in each group. In fact, the number of patients with visual impairment may be higher than this because many patients who come to our emergency department have severe unconsciousness and because routine eye examinations are not performed by an ophthalmologist. In a retrospective study conducted in 2022, the rate of visual impairment was found to be 70% in patients with methyl alcohol intoxication who applied to the emergency department (16).

Although the number of patients who underwent IHD before ICU admission was higher in the survivor group, it did not make a statistically significant difference. The lack of statistical difference may be due to the low sample size. IHD application before ICU could be performed in patients who did not need IMV and who were hemodynamically stable. Hemodialysis removes both methanol and toxic metabolites (formic acid) from the blood and thus corrects the acid-base disorder (17). Even though we performed CRRT immediately in all our patients admitted to the ICU,

IHD is the first choice as it provides a shorter time to remove methyl alcohol and toxic metabolites (18). The late arrival to the hospital after methyl alcohol intake may have caused us to not benefit sufficiently from hemodialysis.

The APACHE-II and SOFA scores, which are the scores used to predict mortality in critical illnesses, are higher in the non-survivor group in methyl alcohol intoxication and can be used to predict mortality. Blood creatinine, potassium, and total bilirubin levels measured at hospital admission are also higher in the non-survivor group and can be used to predict mortality. Although the blood potassium level was higher in the non-survivor group, it was within the upper limits of normal ( $5.3 \pm 1.2$ ). This elevation may have resulted from renal failure or metabolic acidosis. These results show how severe the toxic picture is during ICU admission in the non-survivor patient group. Formic acid is formed because of methyl alcohol intoxication; it has a direct toxic effect on all organs, mainly on the eyes, brain, kidneys, and liver (2,17).

In the non-survivor patient group, blood  $\text{HCO}_3^-$  and pH values were quite low, and there was deeper metabolic acidosis. In a retrospective study, it was found that a pH value of  $\leq 6.9$  was strongly associated with mortality (19). We believe that both blood  $\text{HCO}_3^-$  and pH values should be used to predict mortality.

The total length of stay in the ICU was found to be statistically shorter in the non-survivor group than in the survivor group. At the same time, the median value of the total hospitalization period in the non-survivor patient group is as short as '3 days', which shows how fast death due to methyl alcohol intoxication occurs.

Bilateral basal ganglia hemorrhage, a pathognomonic finding due to methyl alcohol intoxication, was detected in the non-survivor group (Figure 3) (20). However, clear statistical data could not be obtained because not all patients underwent routine cranial imaging.



**Figure 3.** Bilateral basal ganglia bleeding in a methyl alcohol intoxication patient

The most important limitations of our study are that it was single-centered, the sample size was small, and the blood levels of methyl alcohol, which is the gold standard for diagnosis, could not be measured. In addition, in cases of methyl alcohol intoxication due to imitated alcohol intake, the timing of methyl alcohol intake could not be precisely determined because of late detection and admission to the hospital. However, a positive aspect of our study was that the necessary tests could be performed to detect other possible toxic or non-toxic causes that may cause metabolic acidosis with increased anion gap in these patients. Apart from this, the use of the 'ImdSoft-Metavision/QliniCU Clinical Decision Support Software' decision support system was another advantage for the security of research data.

## CONCLUSION

The depth of metabolic acidosis, the need for an invasive mechanical ventilator in the early period, acute renal injury, hyperbilirubinemia, and high SOFA and APACHE-II scores are the most important factors predicting mortality in patients who develop methyl alcohol intoxication.

In patients who develop methyl alcohol intoxication due to imitated alcohol use, the possibility of irreversible organ damage in terms of survival is high when the intoxication is recognized. Therefore, it is crucial for state administrators to take measures to prevent fraudsters from producing imitated alcohol and to increase consumers' awareness of this issue.

## ETHICS

**Ethics Committee Approval:** Ethical approval was obtained from the Ethics Committee of the University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital (decision number: 2022-21-16, date: 07.11.2022).

**Informed Consent:** Retrospective study.

## Authorship Contributions

Surgical and Medical Practices: M.A., D.Ö.B., Concept: M.A., Design: M.A., D.Ö.B., Data Collection or Processing: M.A., Analysis or Interpretation: M.A., Literature Search: M.A., Writing: M.A., D.Ö.B.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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

## REFERENCES

1. 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.
2. Taşkın Ö, Açıkalın A, Dişel R. Methyl Alcohol Intoxications. *Anatolian J Emerg Med* 2022;5:37-42.
3. Ashurst JV, Nappe TM. Methanol Toxicity. [Updated 2022 Jun 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK482121/>
4. Kraut JA, Kurtz I. Toxic alcohol ingestions: clinical features, diagnosis, and management. *Clin J Am Soc Nephrol* 2008;3:208-25.
5. Barceloux DG, Bond GR, Krenzelok EP, Cooper H, Vale JA; American Academy of Clinical Toxicology Ad Hoc Committee on the Treatment Guidelines for Methanol Poisoning. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. *J Toxicol Clin Toxicol* 2002;40:415-46.
6. Barceloux DG, Krenzelok EP, Olson K, Watson W. American Academy of Clinical Toxicology Practice Guidelines on the Treatment of Ethylene Glycol Poisoning. Ad Hoc Committee. *J Toxicol Clin Toxicol* 1999;37:537-60.
7. Kruse JA. Methanol and ethylene glycol intoxication. *Crit Care Clin* 2012;28:661-711.
8. Chu J, Wang RY, Hill NS. Update in clinical toxicology. *Am J Respir Crit Care Med* 2002;166:9-15.
9. Brent J. Current management of ethylene glycol poisoning. *Drugs* 2001;61:979-88.
10. Çetinkaya A, Aslan Sirakaya H, Aydın K. Methyl Alcohol Poisoning: An Analysis of 18 Consecutive Cases. *Turkish J Nephrol* 2021;30:57-62.
11. Morteza Bagi HR, Tagizadieh M, Moharamzadeh P, Pouraghaei M, Kahvareh Barhagi A, Shahsavari Nia K. Epidemiology of Alcohol Poisoning and Its Outcome in the North-West of Iran. *Emerg (Tehran)* 2015;3:27-32.
12. Moghaddam SAB, Pajoumand A. Prognostic Factors including clinical manifestation and Paraclinic finding in sever methanol toxicity. *Life Sci J* 2014;11:11-4.
13. Shadnia S, Rahimi M, Soltaninejad K, Nilli A. Role of clinical and paraclinical manifestations of methanol poisoning in outcome prediction. *J Res Med Sci* 2013;18:865-9.
14. Manning L, Kowalska A. Illicit alcohol: Public health risk of methanol poisoning and policy mitigation strategies. *Foods* 2021;10:1625
15. 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.
16. Şahin T, Bol O, Altuntaş M. Retrospective Analysis of Methyl Alcohol Poisonings Admitted to the Emergency Department. *Konuralp Medical Journal* 2022;14:503-11.
17. 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.
18. Zakharov S, Pelclova D, Navratil T, Belacek J, Kurcova I, Komzak O, et al. Intermittent hemodialysis is superior to continuous venovenous hemodialysis/hemodiafiltration to eliminate methanol and formate during treatment for methanol poisoning. *Kidney Int* 2014;86:199-207.
19. Kute VB, Godara SM, Shah PR, Gumber MR, Goplani KR, Vanikar AV, et al. Hemodialysis for methyl alcohol poisoning: A single-center experience. *Saudi J Kidney Dis Transpl* 2012;23:37-43.
20. Aisa TM, Ballut OM. Methanol intoxication with cerebral hemorrhage. *Neurosciences (Riyadh)* 2016;21:275-7.

**Supplement Table**

---

**1. Charlson comorbidity indexes of the patients;** It was calculated by entering patient data from the <https://www.mdcalc.com/calc/3917/charlson-comorbidity-index-cci> website.

---

**2. SOFA scores of the patients;** It was calculated by entering patient data from the <https://www.mdcalc.com/calc/691/sequential-organ-failure-assessment-sofa-score> website.

---

**3. APACHE-II scores of the patients;** It was calculated by entering patient data from the <https://www.mdcalc.com/calc/1868/apache-ii-score> website.

---