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Original Article

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Methyl Alcohol Poisoning: An Analysis of 18 Consecutive Cases

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Abstract

Objective: This study aimed to investigate the importance of a multidisciplinary approach by reflecting real-life data in methanol intoxication.
Materials and Methods: A total of 18 patients, treated between January 2018 and January 2020 for methanol poisoning, were included in this retrospective study. The patients were stratified as non-survivors and survivors. Systemic findings and laboratory parameters of patients during admission and follow-up in intensive care were compared.
Results: A total of 7 patients in the non-survivor group (NG) and 11 patients in the survivor group (SG) were included in the study. There was no difference between the groups in terms of age and sex. The most common findings were gastrointestinal symptoms (72%), followed by dyspnea (61%), and visual impairment (61%). The serum bicarbonate levels were significantly lower, whereas serum lactate, base deficit, serum creatinine, and hemodialysis durations were significantly higher in NG patients than in SG patients (p=0.035, p=0,020, p=0.027, p=0.003, and p=0.002, respectively). There was a strong correlation between survival and creatinine level, hypotension, dyspnea, the need for invasive mechanical ventilation, and hemodialysis duration (r=0.692, p=0.001; r=-0.798, p<0.001; r=-0.636, p=0.005; r=-0.892, p<0.001; r=0.721, p=0.001, respectively)
Conclusion: Despite effective management in methanol intoxication, mortality and morbidity rates were high in the sample. The treatment of methanol poisoning requires a multidisciplinary approach.
Keywords: Methanol, intoxication, treatment, acidosis, antidote

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INTRODUCTION

Methyl alcohol poisoning is a clinical condition that can cause multiple organ injuries and can be fatal. It may occur either as bulk or individual cases. The bulk poisoning cases are seen in countries with higher alcohol tax rates resulting in illegal alcohol production. It can also occur following consumption of substances such as cologne or ethyl alcohol by individuals with alcohol dependence when access to alcohol is limited. The poisoning most commonly occurs via the oral route; however, it may result from alcohol exposure via inhalation and, in rare instances, the transdermal route (1). Alcohol distribution is equal across all organs and tissues, regardless of the route of exposure.

Methyl alcohol is an aliphatic alcohol (CH_3OH), which is used as a solvent and denaturing agent in cosmetic formulations (2). It is a toxic substance, which is used as a raw material in the industrialized production of several products, such as paints, wax, cleansing liquids, and defrosters (3).

Methanol poisoning can lead to a wide range of clinical manifestations varying from severe metabolic acidosis, total loss of vision, permanent neurological dysfunction, or eventual death (4). Methanol is converted to formaldehyde in the liver and oxidized to formic acid. Formic acid is toxic for the central nervous system, causing hypoxia at the cellular level and axonal cell death (5). Thus,

it exerts potential depressant effects on the central nervous system following acid exposure via ingestion, inhalation, or the transdermal route (6-8).

An anion gap >30 mEq/L and/or a base deficit <-15 mEq/L, impaired vision, renal failure, and worsening or refractory metabolic acidosis (pH<7.25) are indications for prompt hemodialysis after methanol or ethylene glycol exposure (9). The mortality increased with the increasing severity of metabolic acidosis.

The treatment modalities include prevention of toxic metabolite formation, alkalinization to correct acidosis, clearance of toxic metabolites and acidosis, intravenous folinic acid, oral or intravenous ethanol, fomepizole, and hemodialysis (10). Fomepizole is a competitive inhibitor of alcohol dehydrogenase enzyme and is used to treat methanol poisoning in adult patients (11).

This is a real-life study conducted to demonstrate the importance of a multidisciplinary approach in methanol poisoning.

MATERIALS AND METHODS

Study Population and Design

This is a single-center, retrospective, and observational study. The study was approved by the Ethics Committee of Kayseri City Hospital (Approval Date: July 14, 2020; Approval Number: 40) and conducted in accordance with the tenets of the Helsinki Declaration. The study included 26 patients who were treated for methanol poisoning by the same team in the medical intensive care unit of Kayseri City Hospital between January 2018 and January 2020. A total of 5 patients were excluded as methyl alcohol poisoning could not be ascertained, whereas 3 were excluded owing to incomplete data. In all the patients, data were retrospectively extracted from patient charts, the clinical data sheet, and the hospital database. The patients were classified as non-survivors and survivors.

A detailed history was obtained directly from the patients or, when that was not possible, from their relatives. Symptoms of patients with low Glasgow coma scale (GCS) were obtained from their relatives at the time of the first admission. However, no definitive information could be gathered regarding the time to presentation after methyl alcohol ingestion despite these efforts. Comprehensive neurological and ophthalmologic examinations were performed on alternate days and before discharge. Baseline laboratory studies included with the analyses were obtained at presentation. However, we did not include methanol level in the analyses, as methanol levels could not be studied in our facility. Thus, only patients with definite methanol ingestion based on history were included in the study. The laboratory studies at presentation and during follow-up were included in the analyses.

Main Points

- Methanol poisoning can lead to a wide range of clinical manifestations varying from severe metabolic acidosis, total loss of vision, permanent neurological dysfunction, or eventual death.
- The study included 18 patients who were followed for methyl alcohol poisoning. Of these, 7 patients were non-survivors (NG), whereas 11 patients were survivors (SG).
- The treatment of methanol poisoning requires a multidisciplinary approach. The severity of acidosis, the clinical picture at presentation, and the time to presentation are factors influencing mortality.
- Methanol poisoning should be considered when treating metabolic acidosis with an extremely high anion gap.

Treatment

All patients were treated in accordance with the guidelines of the American Academy of Clinical Toxicology and the European Association of Poison Centers and Clinical Toxicologists. The treatment was guided by blood gas analysis and the clinical picture. Sodium bicarbonate infusion was given as a buffer in cases where there was a delay in hemodialysis (HD). It was also given to patients with severe or refractory acidosis despite long-term HD. Ethanol (10% solution) infusion was given at a loading dose of 7.5-8.0 mL/kg for over 1 hour; followed by infusion of 1.0-2.0 mL/kg during follow-up or 2.5-3.0 mL/kg during HD. The ethanol infusion was set to maintain a serum ethanol level above 100 mg/dL. No patient received oral ethanol therapy. No fomepizole was given to patients as antidote because it was unavailable. Calcium folinate (Leucovorin®) therapy was given to all patients.

All the patients underwent HD. Blood gas analysis was performed to determine acidosis at the end of HD, which was prolonged in patients with persistent acidosis. Additional HD sessions were performed if metabolic acidosis was present during blood gas monitoring.

Laboratory Assays

Arterial blood gas analysis was performed before, during, and after HD, and thereafter, at 2- or 4-hour intervals until recovery from acidosis. Ethanol assays were studied in venous blood samples (2 mL) drawn into 2-mL vacuum tubes (Sodium fluoride-disodium EDTA (NaF-Na2EDTA); closed collection system) at presentation and at 2- to 4-hour intervals thereafter. Methanol and formic acid levels could not be analyzed as they are not studied in our laboratory.

Statistical Analysis

All calculations were performed using the Statistical Package for the Social Sciences version 22.0 (IBM Corp.; Armonk, NY, USA). Normal distribution was assessed using the Shapiro-Wilk test. Data with skewed distribution were presented as median (25-75 percentile), whereas categorical variables are presented as a percentage (%). The chi-squared test was used to compare categorical data between groups, whereas the Mann-Whitney U

test was used to compare data with skewed distribution. Spearman's correlation analysis was used to assess relationships among variables. In all tests, a p value <0.05 was considered as statistically significant.

RESULTS

The study included 18 patients who were followed for methyl alcohol poisoning. Of the 18 patients, 16 were men and 2 women, with mean age of 38 years. Their acute physiology and chronic health evaluation (APACHE) II score was 25, and their GCS was 13. The most common symptoms were gastrointestinal symptoms (72%), followed by dyspnea (61%) and visual impairment (61%). Of the patients, 11.1% used ethyl alcohol along with methyl alcohol. For treatment, 44.4% needed mechanical ventilation and 38.9% needed a vasopressor. The demographic data are summarized in Table 1.

Of these patients, 7 were in the NG, whereas 11 were in the SG. There were 5 men in the NG and 11 in SG, indicating no significant difference in sex between the groups (p=0.137). Demographic, clinical, and laboratory parameters between surviving patients and the patients who died are compared in Table 2. Median length of intensive care unit stay was 4 days (1-12); however, there was no significant difference between groups (p=0.659). The median GCS score at presentation was 3 and 14 in the NG and the SG, respectively (p<0.001). The median APACHE II score at presentation was 35 and 16 in the NG and SG, respectively (p<0.001). There was no difference between the groups in terms of blood glucose levels (p=0.246), but there was a significant difference between the 2 groups in terms of systolic, diastolic, and mean blood pressures (p<0.001, p<0.001, and p<0.001; respectively). Vasopressor use, the need for invasive mechanical ventilation, dyspnea, and sodium bicarbonate therapy were significantly more common in NG than in the SG (p<0.001, p<0.001, p=0.013, and p=0.002; respectively).

Table 1. Demographic, clinical, and laboratory findings of all the patients

Sex (men/women)	16/2
Age (years)	38 (20.75-50.5)
APACHE II score	25 (16-35)
Glasgow coma scale	13 (3-15)
Symptoms	
Gastrointestinal symptoms	13 (72.2)
Dyspnea	11 (61.1)
Visual impairment	10 (55.6)
Need for mechanical ventilation	8 (44.4)
Intensive care follow-up (day)	4 (2-4.25)
Need for the vasopressor agent	7 (38.9)
Ethyl alcohol with methyl alcohol	2 (11.1)

Values are presented as number (%) of patients or median (25^{th}-75^{th} percentile)
APACHE II: Acute Physiology and Chronic Health Evaluation II

Table 3 presents the extent and direction of correlation between clinical parameters and survival. The serum bicarbonate level was significantly lower in SG patients, whereas the serum lactate, base deficit, serum creatinine, GGT (gamma glutamyl transferase), and hemodialysis duration were significantly higher in NG patients than in SG patients (p=0.035, p=0.020, p=0.027, p=0.003, p=0.035, and p=0.002; respectively). There was a strong correlation between survival and APACHE II scoring, GCS, creatinine level, hypotension, dyspnea, the need for invasive mechanical ventilation, and hemodialysis duration (r=0.848, p<0.001; r=-0.865, p<0.001; r=0.692, p=0.001; r=-0.798, p<0.001; r=-0.636, p=0.005 r=-0.892, p<0.001; r=0.721, p=0.001, respectively), whereas there was a moderate correlation with base deficit, bicarbonate level, lactate, and GGT (r=0.527, p=0.024; r=-0.516, p=0.028; r=0.550, p=0.018; r=0.506, p=0.032, respectively).

DISCUSSION

In our study, there were significant differences in GCS, APACHE II, serum bicarbonate, lactate, creatinine and GGT levels, base deficit, duration of hemodialysis, the need for invasive mechanical ventilation, vasopressor need, the need for bicarbonate infusion, and dyspnea between non-survivors and survivors.

Consistent with previous studies, our patients were mainly young to middle-aged male individuals (8, 12). However, no significant correlation was found between mortality and demographic characteristics such as age and sex. This may be owing to the fact that irregular and uncontrolled alcohol intake is more common in this age group.

Methanol poisoning can lead to the onset of symptoms that reflect impairment in several systems because of disorders in a number of organ systems and tissues. In this study, patients presented to the emergency department with several symptoms, with gastrointestinal symptoms being the most common in both groups. Visual impairment was among the most common presenting complaints, and its incidence was comparable between the 2 groups. However, dyspnea, another common presenting complaint, was a clinical symptom showing significant differences between the groups in agreement with the study by Paasma et al (8).

All types of alcohol cause intoxication in a dose-dependent manner, and methanol has the lowest intoxicant effect. Because of the relatively lower intoxicant effect of methanol, no significant reduction was observed in GCS in patients not requiring intubation. The low GCS score in non-survivors might be owing to severe metabolic acidosis or high-dose methanol

Table 2. Comparison of demographic, clinical, and laboratory parameters between living patients and patients who died

Conditions and interventions	Study group		p
	NG (non-survivor group) (n=7)	SG (survivor group) (n=11)	
Age	47 (36-54)	22 (20-50)	0.151
Sex (Male)	5 (71)	11 (100)	0.137
GCS	3 (3-3)	14 (14-15)	<0.001
APACHE II score	35 (34-42)	16 (14-24)	<0.001
Intensive care follow-up (day)	2 (1-6)	4 (3-4)	0.659
Glucose (mg/dL)	112 (94-123)	125 (98-213)	0.246
Systolic blood pressure (mm Hg)	119 (102-120)	78 (70-80)	<0.001
Diastolic blood pressure (mm Hg)	75 (70-76)	40 (35-40)	<0.001
Mean blood pressure (mm Hg)	88 (84-90)	53 (47-53)	<0.001
pH	6.77 (6.76-7.33)	7.19 (7.10-7.28)	0.126
HCO_3 (mmol/L)	5.8 (5.6-10.7)	10.9 (9.5-14.9)	0.035
pCO_2 (mm Hg)	38.5 (18.0-53.8)	30 (25.3-36.6)	0.930
Lactate (mmol/L)	12 (10.5-16.0)	3 (1.5-11.0)	0.020
Base deficit (mmol/L)	28 (20.9-30.0)	16.5 (13.6-21.0)	0.027
Blood Urea Nitrogen (mg/dL)	9 (6.0-1.7)	11.1 (9-16)	0.536
Creatinine (mg/dL)	1.31 (1.21-2.15)	0.84 (0.77-1.10)	0.003
Chlorine (mmol/L)	101 (100-105)	104 (101-105)	0.479
Aspartate aminotransferase (U/L)	50 (23-172)	22 (15-35)	0.069
Alanine aminotransferase (U/L)	25 (15-81)	15 (8-24)	0.104
Gamma glutamyl transferase (U/L)	45 (34-100)	15 (12-25)	0.035
Alkaline phosphatase (U/L)	96 (75-141)	91 (66-100)	0.536
Hemodialysis time (hour)	7 (5-10)	3 (2-4)	0.002
Need for vasopressor agent	7 (100)	0 (0)	<0.001
Need for mechanical ventilation	7 (100)	1 (9)	<0.001
Imaging finding	5 (71)	6 (54.5)	0.637
Gastrointestinal symptoms	6 (86)	7 (64)	0.596
Dyspnea	7 (100)	4 (36)	0.013
Bicarbonate treatment	6 (86)	1 (9)	0.002
Visual impairment	6 (86)	5 (45)	0.151

Values are presented as number (%) of patients or median (25^{th}-75^{th} percentile)
GCS: Glasgow Coma Scale; APACHE II: Acute Physiology and Chronic Health Evaluation II; HCO_3: bicarbonate; CO_2: carbon dioxide

Table 3. Correlation between clinical parameters and survival

	r	p
Age (years)	0.365	0.136
Sex	-0.443	0.065
APACHE II score	0.848	<0.001
GCS	-0.865	<0.001
Creatinine (mg/dL)	0.692	0.001
pH	-0.385	0.115
Base deficit (mmol/L)	0.527	0.024
HCO_3 (mmol/L)	-0.516	0.028
Lactate (mmol/L)	0.550	0.018
CO_2 (mm Hg)	0.033	0.897
Hypotension	-0.798	<0.001
Dyspnea	-0.636	0.005
Need for mechanical ventilation	-0.892	<0.001
Hemodialysis time (hour)	0.721	0.001
Gamma glutamyl transferase (U/L)	0.506	0.032

Spearman correlation test.
GCS: Glasgow Coma Scale; APACHE II: Acute Physiology and Chronic Health Evaluation II; HCO_3: bicarbonate; CO_2: carbon dioxide

ingestion. The negative correlation between GCS and mortality in our study was in agreement with the previous literature (13, 14). In addition, there was a positive correlation between mortality and APACHE II score. The most important reasons for this are the increase in metabolic acidosis and neuronal damage because of late admission to the hospital. Deep acidosis and neuronal damage can develop as a result of increased exposure to toxic end products owing to late admission to the hospital (6, 7).

In this study, a minority of patients had ingested ethyl alcohol together with methanol. Unlike the study by Hovda Ke et al. (13), there was no significant correlation between ethyl alcohol ingestion and mortality rate. This might be the result of a smaller sample size and the limited number of patients who had ingested ethyl alcohol in this study.

In this study, there was a strong correlation between mortality rate and the need for invasive mechanical ventilation. In previous studies, partial carbon dioxide pressure was not high in non-survivors. This might be because of the provision of elective early intubation and better sedation (13). Unlike some studies, partial carbon dioxide pressures were not high in the patient group who died. This may be because of elective early intubation and well-secured sedation of the patients.

Methanol poisoning should be suspected when there is a concomitant increase in serum osmolarity and anion gap (15). Metabolic acidosis with an elevated anion gap is a characteristic feature of toxic alcohol poisoning. Formic acid and lactate account for the development of acidosis in methanol poisoning. In agreement with the literature, increased serum lactate level and base deficit were significantly more common in the NG. Consistent with many previous studies, there was a positive correlation between mortality and severity of acidosis or elevated base deficit (16-18). In our study, the duration of hemodialysis was longer in the NG. Thus, the need for sodium bicarbonate infusion to correct acidosis was also higher in the NG.

A literature review shows that the severity of methanol poisoning is associated with the level of acidosis rather than the methanol level (9). However, no such comparison could be made as methanol testing is unavailable in our hospital. Many markers are used to evaluate toxic alcohol poisoning owing to problems in measurement and interpretation of serum concentrations. Thus, appropriate treatment should be initiated early based on history, clinical presentation, laboratory findings, and the severity of acidosis even if methanol levels cannot be studied.

The definitive treatment in toxic alcohol poisoning is HD. In this study, HD was performed in the presence of anion gap and metabolic acidosis regardless of symptom severity in patients in whom methanol poisoning was suspected. We think that this approach significantly decreased the mortality rate. Zakharov et al. (19) applied intermittent HD, extended daily dialysis, and continuous renal replacement therapy to the patients with methanol poisoning and showed that the intermittent HD regimen was superior. In our study, intermittent HD was performed on all the patients.

Formic acid, a methanol metabolite, is a mitochondrial enzyme that inhibits cytochrome oxidase. Neurons at basal ganglia are highly susceptible to formic acid toxicity. By the action of 10-formyl tetrahydrofolate synthase followed by its oxidation to carbon dioxide catalyzed by 10-formyltetrahydrofolate dehydrogenase, folates improve formic acid metabolism by transforming it into 10-formyl tetrahydrofolate. The presence of a folate derivative improves formic acid oxidation by preventing the production of the enzyme catalyst deficient metabolic pathways (20). High doses of folate or folinic acid can facilitate the conversion of formic acid to carbon dioxide and water. Characteristic bilateral basal ganglia lesions of methanol poisoning can be seen on a computed tomography scan and on magnetic resonance imaging (6, 7). In this study, there was no significant difference in imaging findings between groups, and calcium folinate was given to all the patients with methanol poisoning to prevent and reverse end-organ injury. In survivors, recovery was reported in visual impairment in control visits after disposition.

This study had some limitations, including the retrospective and single-center design as well as a lack of methanol level measurements and fomepizole use as an antidote. Future studies with larger sample sizes can provide more comprehensive outcomes.

CONCLUSION

Despite effective management, morbidity and mortality rates are high in methanol poisoning, the treatment of which requires a multidisciplinary approach. The severity of acidosis, the clinical picture at presentation, and the time to presentation are all factors influencing mortality. Methanol poisoning should be considered when treating metabolic acidosis with an extremely high anion gap.

Ethics Committee Approval: Ethics committee approval for this study was received from the Ethics Committee of Kayseri City Hospital (Approval Date: July 14, 2020; Approval Number: 40).

Informed Consent: Informed consent was not obtained due to the nature of this study.

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