ISSN: 2690-263X

LOGHMARI. Int Arch Subst Abuse Rehabil 2022, 4:016
DOI: 10.23937/2690-263X/1710016
Volume 4 | Issue 1
Open Access

International Archives of
Substance Abuse and Rehabilitation

ORIGINAL ARTICLE

Acute Methanol Poisoning in Tunisia: Clinical Features, Biological and Associated Factors for Mortality

*Dorra LOGHMARI**

Faculty of Medicine of Sousse, Emergency Medical Services, Sahloul University Hospital, Sousse, Tunisia

***Corresponding author:** *Dorra LOGHMARI, Faculty of Medicine of Sousse, Emergency Medical Services, Sahloul University Hospital, Sousse, Tunisia*

Abstract

Background: Acute Methanol Poisoning (MP) is rare but potentially serious.

Objectives: To study the clinical and biological characteristics of acute MP and its associated factors of mortality.

Methods: We conducted a cross-sectional study including case series of MP which took place in Kairouan. Tunisia. Cases started consulting the emergency room on a festive day (1st day of Aid el Feter) corresponding to May 24, 2021.

Results: We included 65 victims of MP, they were all males. The median age was 28.0 [21.0-35.0] years with extremes ranging from 17 to 75 years. The median time between the ingestion of methanol and the medical consultation was 48.0 [24.0-50.0s] hours.

On admission, the majority of patients described neurological (98.4%) and gastrointestinal symptoms (51.4%). Four patients remained visually impaired, and 8 patients (12.3%) had died.

The univariate analysis showed an association between mortality and age, the amount of methanol ingested, the amount diluting methanol, the co-ingestion of cannabis and the delay between ingestion and consultation, neurological distress, hemodynamic instability, seizures, hypernatremia, hypokalemia, high level of hematocrit, acute kidney failure, hyperglycemia, metabolic acidosis, plasma hyperosmolarity, high anion gap and APACHII score.

Conclusion: Acute MP remains serious not only it can be life-threatening but also is responsible of several organ damages and may lead to blindness and irreversible damage to the central nervous system. The realization of campaigns promoting awareness about MP and the enforcement of laws governing alcohol manipulation are keys for MP prevention. In addition, early identification of this intoxication and rapid management are essential to improve the prognosis.

Keywords

Epidemiology, Methanol, Poisoning, Prognosis, Tunisia

Introduction

Methanol (CH_3OH), formerly, was known as wood alcohol because it was obtained by distillation of wood, now, is prepared by synthesis [1]. Acute Methanol Poisoning (MP) is most often due to accidental ingestions due to distillation, fermentation errors and its use as unlisted ingredient in supposedly alcohol-based products [2].

The clinical presentation may mislead clinicians depending on the nature of the toxic alcohol, time of exposure, and coingestion of ethanol. The toxicity of these products is not related to the molecules ingested but to their metabolites [3]. In Tunisia, there is no record of methanol poisoning. Epidemiological and clinical data are missing on this subject.

We aimed to describe the epidemiological, clinical and therapeutic features of the collective acute MP that took place in the governorate of Kairouan in Tunisia and to study the associated factors for mortality.

Population and Methods

We conducted a cross-sectional study on victims of a collective acute MP which took place in Hajeb el Ayoun, Kairouan, Tunisia. This city belongs to the governorate of Kairouan which is in the center of Tunisia. Its area is 6,712 km^2, populated by 581,300 inhabitants, i.e. 86.6 inhabitants/km^2. This governorate suffers from

Citation: LOGHMARI D (2022) Acute Methanol Poisoning in Tunisia: Clinical Features, Biological and Associated Factors for Mortality. Int Arch Subst Abuse Rehabil 4:016. doi.org/10.23937/2690-263X/1710016

Accepted: December 20, 2022: **Published:** December 22, 2022

several socio-economic problems. It is placed in the 22nd position on the national scale of poverty. It is known by its high rates of suicide, school dropouts, illiteracy and unemployment [4].

Cases started consulting the Emergency Room (ER) on a festive day (1st day of Aid el Feter) corresponding to May 24, 2021. The last case was recorded on May 27, 2021 at 6 p.m.

The Two patients who died at home before arriving in the ER and the three who escaped from hospital were not included in our study.

Socio-demographic data, including age, sex, educational level, profession, alcohol addiction, amount of methanol ingested, amount of water diluting methanol, time interval from ingestion to hospital admission, and outcome of the patients were recorded. The clinical examination included standard neurological examination and complete ocular examination with standard ophthalmologic tests. An MRI was performed to symptomatic patients. Severity was assessed by the Acute Physiology and Chronic Health Evaluation (APACHE II). We performed laboratory analyzes on admission from blood samples already drawn for treatment purposes.

All patients received appropriate treatment according to standard protocols. They received ethanol as antidote, folic acid and hemodialysis. Patients were divided into 2 groups, according to the outcome: Group I, patients who survived and group II, patients who died.

All statistical analyses were performed using SPSS Version 20.0. Qualitative data were expressed as numbers and percentages and quantitative variables as median with an interquartile range [Q25-Q75], as appropriate. For comparisons of data between groups, non-parametric–tests such as Mann Whitney U test and Kruskal-Wall is test were employed to compare quantitative data. Chi-squared test and Fisher's exact test were used to compare qualitative data. A p-value less than 0.05 was considered statistically significant.

Results

The study population consisted of 65 patients. All the included subjects were male. The median age was 28.0 [21.0-35.0] years with extremes ranging from 17 to 75 years. The majority of patients had a primary educational level (86.1%). All of the patients were smokers and 64.6% of them were alcoholics. The median amount of methanol ingested was 1000.0 [750.0-1500] ml i.e., 972.0 [850.0-1300.0] g with a minimum of 500 ml i.e., 324 g and maximum of 3000 ml i.e., 1950 g. The median amount of water used to dilute methanol product was 1000.0 [1000.0-2000.0] ml with amounts ranging between 500 and 3000 ml.

The ingestion of methanol was associated to other substances in 46.1% of cases. The illicit substances used were cannabis (15.3%), organochlorine (3%), Parkizol (3%) and ethanol (1.5%).

The median delay between the ingestion of methanol and the medical consultation was 48.0 [24.0-50.0] hours with a minimum of 7 hours and a maximum of 72 hours (Table 1).

On admission, the majority of patients described neurological (98.4%) and gastrointestinal symptoms (51.4%). Headache was the most common neurological symptom (87.7%) and abdominal pain was the most gastrointestinal described sign (32.3%). Visual disturbances were present in 41.53% of cases with blurred vision (27 cases) and visual impairment (6 cases). On first examination, 12.3% of patients presented hypotension and the median Systolic Blood Pressure (SBP) was 12.0 [11.0-13.0] mmHg. The median

Table 1: Socio-demographic and methanol consumption characteristics in Kairouan, Tunisia in 2020.

Socio-demographic and methanol consumption characteristics		Total n (%)
Educational level	Analphabet	3 (46.0)
	Primary	56 (86.2)
	Secondary	6 (9.2)
Alcohol addiction		42 (64.6)
Profession	unemployed	60 (92.3)
	laborer	5 (7.7)
Co-ingestion of substance		27 (41.5)
Co-ingestion of Cannabis		10 (15.4)
Co-ingestion of Ethanol		16 (24.6)
		Median [Q25-Q75]
Age (year)		28.0 [21.0-35.0]
Amount of methanol ingested (ml)		1000.0 [750.0-1500.0]
Amount of water diluting methanol (ml)		1000.0 [1000.0-2000.0]
Consultation delay (h)		48.0 [24.0-50.0]

respiratory rate was 20.0 [18.0-22.0] with a range between 16 and 30 cpm, only 36.9% of patients were polypneic and 30.8% of them presented a desaturation.

Only 20% of patients arrived in coma and 12.3% presented seizures. A mydriasis was found in 9.3% of cases. The visual acuity test was abnormal in 43.4% of cases. The fundus copy showed an optic neuritis in 12.3% of patients which was confirmed by the MRI. Half of these patients with optic neuritis had associated signs of toxic encephalopathy (6.1%) (Table 2).

Table 2: Clinical features characteristics according to MP in Kairouan, Tunisia in 2020.

	Total n (%)
Headache	57 (87.6)
Dizziness	56 (86.1)
Dyspnea	4 (6.1)
Coma	7 (10.7)
Sleepiness	9 (13.8)
Isoreactive mydriasis	6 (9.3)
Reactive mydriasis	3 (4.6)
Seizures	8 (12.3)
Signs of shock	9 (13.8)
	Median [Q25–Q75]
SBP (mmHg)	12.0 [11.0-13.0]
DBP (mmHg)	70.0 [60.0-80.0]
HR (bpm)	90.0 [80.0-106.5]
RR (cpm)	20.0 [18.0-22.0]
SaO_2 (%)	98.0 [96.0-99.0]

Table 3: Biological features according to MP in Kairouan Tunisia, in 2020.

	Total
	Median [Q25–Q75]
Sodium (mmol/L)	142.0 [141.0-144.0]
Potassium (mmol/L)	3.4 [2.9-3.9]
Hematocrit	43.0 [40.0-49.0]
Blood sugar (mmol/l)	6.0 [5.6-6.6]
Creat (mmol/l)	76.0 [62.0-100.0]
Urea (mmol/l)	4.0 [3.2-4.8]
PH	7.3 [7.2-7.4]
Bicarbonate (mmol/L)	12.4 [7.5-20.0]
$PaCO_2$ (mmHg)	26.0 [22.0-35.0]
PaO_2 (mmHg)	100.0 [80.0-125.0]
APACHII Score	2.0 [0.0-7.0]
Anion gap (mmol/l)	32.0 [28.5-40.2]
Plasma osmolarity (mosmol/kg)	100.0 [80.0-125.0]
	n (%)
Dose of methanolemia > 0.5 g/l	11 (16.9)
Toxic optic neuritis	4 (6.1)
Toxic encephalopathy	4 (6.1)

Concerning blood tests, we found an acute renal failure in 15.4% of cases. Arterial blood gas showed metabolic acidosis in 57.3% of cases. Plasma osmolarity was higher than 290 mmol in 41.5% of cases. Half of the patients had hypokalemia (52%). The anion gap was higher than 20 mmol/l in 24.6% of cases and the median anion gap was 32.0 [28.5-40.2] mmol/l with extremes ranging between 17 and 51. Acute pancreatitis was noted in 18.5% of cases and hypoglycemia in 15.4%. Methanol serum level was higher than 0.5 g/l in 16.9% of patients. The median APACHII score was 2.0 [0.0-7.0] (Table 3).

All patients received hydration with saline. The antidote which is ethanol was administered to 33 patients, i.e., 50.7%. Five patients (7.7%) received folic acid.

Hemodialysis was performed in 33 patients (50.7%). Among the eight deceased patients, 6 cases had a 2-hour hemodialysis session. Oxygen was administered to 46 patients (70.7%). Mechanical ventilation was performed to 13 patients (20%). Eight patients (12.3%) required the use of vascular drugs.

Concerning the orientation of patients, 43% of patients were transferred to an intensive care unit 18.4% were transferred to the CAMU center, 13.8% were transferred to a medical department and 18.4% were kept in the emergency room due to lack of places.

Among the 65 victims, 53 patients (81.5%) had a total recovery after therapeutic care, 4 patients (6.15%) kept a visual impairment and 8 cases died (12.3%).

The median length of stay in the ER was 24.0 [8.0-60.0] hours with extremes of 2 and 96 hours.

The mortality rate reached was 12.3%. An analysis of the predictive factors of mortality in patients with MP showed a significant association between mortality and some clinical and biological features. In the univariate analysis, we found that age ($p = 0.04$), the quantity of methanol ingested, the quantity of water diluting methanol, the co-ingestion of cannabis and the delay between ingestion and consultation were significantly associated with mortality.

Concerning clinical and biological features, mortality was associated with neurological distress, hemodynamic instability, seizures, hypernatremia, hypokalemia, high level of hematocrit, acute kidney failure, hyperglycemia, metabolic acidosis, plasma hyperosmolarity, high anion gap and APACHII score (Table 4).

Discussion

MP has represented a challenge for healthcare providers. It is mainly collective, occurring during festive days [3]. Despite improvement in care, morbidity and mortality remain high [5]. The number of poisoned subjects recorded was 65. The median age was 28.0

Table 5: Comparison of mortality rate between different studies.

Country	Year	n (%)
Canada [23]	1998	50 (36.0)
UNITED STATES [24]	2000	24 (33.3)
Norway [13]	2005	51 (17.6)
Iran [25]	2007	25 (48.0)
Tunisia [11]	2007	16 (19.0)
India [26]	2012	63 (31.7)
Iran [27]	2013	42 (40.5)
Czechia [7]	2014	121 (33.9)
Taiwan [16]	2014	32 (34.4)
Canada [12]	2015	55 (1.8)
Libya [28]	2016	1066 (9.5)
Kenya [28]	2016	467 (26.9)
Czechia [9]	2017	106 (21.7)
Uganda [6]	2017	15 (80.0)
Taiwan [18]	2018	50 (28.0)
China [8]	2019	52 (3.8)
Tunisia	2020	65 (12.3)

[21.0-35.0] years. The median time between the ingestion of methanol and the medical consultation was 48.0 [24.0-50.0] hours. On admission, the majority of patients described neurological (98.4%) and gastrointestinal symptoms (51.4%). The mortality rate was 12.3%. We found an association between mortality and age, the amount of methanol ingested, the amount diluting methanol, the co-ingestion of cannabis and the delay between ingestion and consultation and neurological distress.

Studies reporting mass MP are scarce. The number of cases of MP reported in the literature was variable. For instance, 15 victims of MP were reported in Uganda [6], 26 cases in Morocco [5] and 121 cases in Czech Republic [7].

All the patients were males. Our results are congruent with results from the literature showing a masculine predominance in methanol intoxication [5-9]. This could be explained by the particularity of the Tunisian population where only a minority of women consumes alcohol due to socio-cultural norms.

Concerning sources of methanol, our outbreak was caused by drinking locally treated cologne sold by a wholesaler for drinking purposes. Cologne was also the main source of methanol in Turkey with a rate of 72.6% followed by spirits (10.6%) then antifreeze (2.7%) [10].

In our series, the delay between methanol ingestion and ER consultation was long and significantly associated with mortality. Similar results were found in several other studies. For instance, Brahmi and al found a delay of 36 hours (range between 6 and 48 hours) [11]. In Canada, authors reported a delay of 38 hours ± 1.51 hours (range between 15 and 85 hours) [12]. In China, the delay was estimated to 41.52 ± 0.72 hours [8]. This delay could be explained by the fact that many of the patients were alcoholics and may have misinterpreted symptoms of MP as alcohol withdrawal, others might have drunk a mixture of methanol and ethanol (antidote) which would delay the onset of symptoms [13].

All the patients were symptomatic upon admission and the most frequent clinical features reported were neurological and gastrointestinal symptoms. In the literature, gastrointestinal disorders were reported frequently in 18 to 67% of cases [13-15]. Dyspnea was reported in 8 to 25% of cases [13-15]. Visual disturbances were present in 29 to 64% of cases [13,14] and neurological symptoms, especially coma, was reported in 10.7% to 36% of cases [5,11,16-18].

Neurological deterioration generally occurs gradually and coincides with advanced stages of intoxication [16]. The severity of central Nervous System (CNS) damage is directly related to the degree of metabolic acidosis caused by the accumulation of formic acid [19,20]. The CNS is a main target of methanol intoxication, especially the brain and the visual pathways which are sensitive to formic acid. The neurological lesions caused by MP are characterized by the presence of a bilateral and symmetrical inflammation of the optic nerves, ischemic and hemorrhagic ranges of the grey nuclei as well as necrosis of the Putamen generally complicated of a hemorrhage with edema, a demyelination of the surrounding white matter and neuronal destruction. These lesions can also affect the cerebellum and the hypothalamus [19,20].

Systemic toxicity was also described in the literature such as hemolysis or rhabdomyolysis with secondary renal failure, pancreatitis and acute hepatitis [19]. Formic acid causes metabolic acidosis with a high anion gap by inhibiting oxidative reactions which promotes anaerobic metabolism generating lactic acid and pyruvic acid worsening acidosis [11,16,19]. According to Nazir, et al. the triad of metabolic acidosis, plasma hyperosmolarity and a high anion gap points towards methanol intoxication [8,21-28].

Our mortality rate is considered to be high, which is consistent with results from other studies [11,19,22]. We noted a big variance in mortality rates between different studies as shown in Table 4. It ranged from 1.8% in a study in Canada [12] to 80% in Uganda [6] (Table 5).

Despite the improvement in treatment, morbidity and mortality following MP remain high. This may be explained by the delay in diagnosis and therapeutic management [3,6,11]. Furthermore, our analysis for associated factors for mortality concluded that the quantity of methanol ingested, the quantity of water diluting methanol, the delay of consultation, metabolic

Table 4: Associated factors with mortality in Kairouan, Tunisia, in 2020.

		Survivors (n = 57)	Deceased (n = 8)	
Sociodemographic characteristics &methanol consumption n (%)			**n (%)**	
Educational level	Analphabet	3 (5.3)	0 (0)	0.480
	Primary	48 (84.2)	8 (100)	
	Secondary	6 (10.5)	0 (0)	
Alcohol addiction		34 (59.6)	8 (100)	0.040
Profession	Unemployed	52 (91.2)	8 (100)	0.380
	Laborer	5 (8.8)	0 (0)	0.507
Co-ingestion of substance		20 (35.1)	7 (87.5)	0.015
Co-ingestion of Cannabis		3 (5.3)	7 (87.5)	$< 10^{-3}$
Co-ingestion of Ethanol		16 (28.1)	0 (0)	0.089
		Median [Q25-Q75]	**Median [Q25-Q75]**	
Age (year)		27.0 [21.0-33.5]	35.0 [29.7-51.0]	0.040
Amount of methanol ingested (ml)		1000.0 [500.0-1500.0]	1750.0 [1500.0-2000.0]	$< 10^{-3}$
Amount of water diluting methanol		1000.0 [1000.0-2000.0]	1750.0 [1500.0-2000.0]	$< 10^{-3}$
Consultation delay (h)		48.0 [24.0-50.0]	24.0 [24.0-24.0]	$< 10^{-3}$
Symptoms		**n (%)**	**n (%)**	
Headache		54 (94.7)	3 (37.5)	$< 10^{-3}$
Dizziness		53 (93.0)	3 (37.5)	$< 10^{-3}$
Dyspnea		3 (5.3)	1 (12.5)	0.417
Coma		2 (3.5)	5 (62.5)	$< 10^{-3}$
Sleepiness		7 (12.3)	2 (25.0)	0.305
Isoreactive mydriasis		1 (1.8)	2 (25.0)	0.04
areactive mydriasis		0 (0)	3 (37.5)	$< 10^{-3}$
Seizures		2 (3.5)	6 (75.0)	$< 10^{-3}$
Signs of shock		2 (3.5)	7 (87.5)	$< 10^{-3}$
		Median [Q25-Q75]	**Median [Q25-Q75]**	$< 10^{-3}$
SBP (mmhg)		120.0 [110.0-130.0]	90.0 [80.0-112.0]	$< 10^{-3}$
DBP (mmhg)		70.0 [60.0-80.0]	50.0 [40.0-70.0]	$< 10^{-3}$
HR (bpm)		90.0 [80.0-100.0]	115.0 [110.0-120.0]	$< 10^{-3}$
RR(cpm)		18.0 [18.0-21.0]	26.0 [22.0-28.0]	$< 10^{-3}$
SaO_2 (%)		98.0 [97.0-99.0]	93.0 [90.0-95.7]	0.938
Blood test		**Median [Q25-Q75]**	**Median [Q25-Q75]**	
Sodium (mmol/L)		142.0 [140.0-144.0]	145.0 [143.0-146.0]	0.025
Potassium (mmol/L)		3.4.0 [3.0-3.9]	2.4 [1.8-3.4]	$< 10^{-3}$
Hematocrit		43.0 [39.5-46.0]	50.0 [44.5-50.7]	0.044
Blood sugar (mmol/l)		6.0 [5.5-6.4]	8.0 [7.0-10.5]	$< 10^{-3}$
Creat (mmol/l)		74.0 [61.5-89.5]	167.5 [134.0-205.0]	$< 10^{-3}$
Urea (mmol/l)		4.0 [3.1-4.4]	6.7 [3.7-16.0]	0.150
PH		7.3 [7.2-7.4]	7.0.0 [6.8-7.3]	0.024
Bicarbonate (mmol/L)		13.0 [8.0-21.0]	4.0 [2.6-11.7]	0.007
$PaCO_2$ (mmHg)		27.0 [22.0-35.0]	22.5 [16.5-33.2]	0.344
PaO_2 (mmHg)		100.0 [80.0-125.5]	120.0 [105.0-197.5]	0.35
APACHII Score		1.0 [0.0-5.0]	23.0 [15.5-27.0]	$< 10^{-3}$
Anion gap (mmol/l)		31.0 [28.0-40.5]	36.0 [36.0-36.0]	0.018
Plasma osmolarity (mosmol/kg)		289.0 [285.5-292.5]	306.5 [314.0-301.0]	$< 10^{-3}$
		n (%)	**n (%)**	
Dose of methanolemia > 0.5 g/l		3 (5.3)	8 (100)	$< 10^{-3}$
Toxic optic neuritis		3 (5.3)	1 (12.3)	0.417
Toxic encephalopathy		2 (3.5)	2 (25.0)	0.071

acidosis and some clinical and biological features were significantly associated with mortality. Our results are congruent with those from other studies [8,13,17,29]. Severe metabolic acidosis was the most described predictor of death. In a study conducted by Meyer, et al. the most important predictor of mortality was acidosis with blood pH of 7.0 [24]. A study conducted in Norway also revealed that severe metabolic acidosis (pH of 6.9) was a strong predictor of death [13]. Coulter, et al. analyzed the literature data and concluded that low pH of 7.22 was associated with increased mortality and that pH had the highest predictive value [30].

In Estonia, it was shown that the outcome was related to the degree of metabolic acidosis [31]. In a multicenter study of Paasma, et al. low pH (pH of 7) was also found to be among the strongest predictors of poor outcome [29].

Limit and Strength

Concerning the limitations of the study, it was a cross-sectional study and the number of cases was relatively small which provided insufficient data confirming the association between MP poor income and certain clinic and laboratory parameters. Furthermore, concentrations of other components in the alcohol-based fuels were not detected and formic acid and ethanol were not measured, due to a lack of available laboratory equipment. In addition, the hospital was unable to provide all antidotes for all patients including fomepizole which is not available in Tunisia.

In spite of these limitations, our study summarizes the clinic and biological features of MP. In accordance with previous studies, it demonstrates a large number of predictive factors for mortality after MP.

Acute MP remains serious not only it can be life-threatening but also is responsible of several organ damages and may lead to blindness and irreversible damage to the central nervous system [20]. Early identification of this intoxication and rapid management are essential to improve the prognosis [19]. We recommend timely intravenous administration of ethanol to victims of MP, in addition, fomepizole should be included in the list of necessary antidotes. We also recommend campaigns promoting awareness about MP and the enforcement of laws governing alcohol manipulation.

Conflict of Interest

We have no conflict of interest to declare.

References

1. (2022) Methanol | Properties, Production, Uses, & Poisoning | Britannica [Internet].
2. Mégarbane B, Brahmi N, Baud F (2001) Intoxication aiguë par les glycols et alcools toxiques: Diagnostic et traitement. Reanimation 10: 426-434.
3. Kraut JA (2016) Approach to the Treatment of Methanol Intoxication. Am J Kidney Dis 68: 161-167.
4. Kairouan (2022) In: Wikipédia [Internet]. 2022.
5. Essayagh S, Bahalou M, Essayagh M, Essayagh T (2020) Epidemiological profile of methanol poisoning, El Hajeb, Morocco. East Mediterr Health J 26: 1425-1429.
6. Doreen B, Eyu P, Okethwangu D, Biribawa C, Kizito S, et al. (2020) Fatal Methanol Poisoning Caused by Drinking Adulterated Locally Distilled Alcohol: Wakiso District, Uganda. J Environ Public Health 2020: 5816162.
7. Zakharov S, Pelclova D, Urban P, Navratil T, Diblik P, et al. (2014) Czech mass methanol outbreak 2012: Epidemiology, challenges and clinical features. Clin Toxicol (Phila) 52: 1013-1024.
8. Maoxia R, Ying L, Zhang L, Wu W, Lin J (2019) Clinical features, treatment, and prognosis of acute methanol poisoning: experiences in an outbreak. Int J Clin Exp Med 12: 5938-5950.
9. Rulisek J, Balik M, Polak F, Waldauf P, Pelclova D, et al. (2017) Cost-effectiveness of hospital treatment and outcomes of acute methanol poisoning during the Czech Republic mass poisoning outbreak. J Crit Care 39: 190-198.
10. Kalkan S, Cevik AA, Cavdar C, Aygoren O, Akgun A, et al. (2003) Acute methanol poisonings reported to the Drug and Poison Information Center in Izmir, Turkey. Vet Hum Toxicol 45: 334-337.
11. Brahmi N, Blel Y, Abidi N, Kouraichi N, Thabet H, et al. (2007) Methanol poisoning in Tunisia: Report of 16 cases. Clin Toxicol (Phila) 45: 717-720.
12. Lachance P, Mac-Way F, Desmeules S, De Serres SA, Julien AS, et al. (2015) Prediction and validation of hemodialysis duration in acute methanol poisoning. Kidney Int 88: 1170-1177.
13. Hovda KE, Hunderi OH, Tafjord AB, Dunlop O, Rudberg N, et al. (2005) Methanol outbreak in Norway 2002-2004: epidemiology, clinical features and prognostic signs. J Intern Med 258: 181-190.
14. Mégarbane B, Borron SW, Trout H, Hantson P, Jaeger A, et al. (2001) Treatment of acute methanol poisoning with fomepizole. Intensive Care Med 27: 1370-1378.
15. Chen WY, Jeng GY, Yen TS, Hsieh BS, Kuo TL, et al. (1978) Studies on acute methanol intoxication. Taiwan Yi Xue Hui Za Zhi 77: 97-102.
16. Lee CY, Chang EK, Lin JL, Weng CH, Lee SY, et al. (2014) Risk factors for mortality in Asian Taiwanese patients with methanol poisoning. Ther Clin Risk Manag 10: 61-67.
17. Eghbali H, Mostafazadeh B, Ghorbani M, Behnoush B (2015) Neurologic complications of methanol poisoning: A clinicoepidemiological report from poisoning treatment centers in Tehran, Iran. Asia Pac J Med Toxicol 4: 47-50.
18. Chang ST, Wang YT, Hou YC, Wang IK, Hong HH, et al. (2019) Acute kidney injury and the risk of mortality in patients with methanol intoxication. BMC Nephrol 20: 205.
19. Brasseur E, Lemineur T, Leonard P, Richardy M, Lambermont B (2001) Le cas clinique du mois. Intoxication aiguë au méthanol. Rev Med Liege 56: 7-10.
20. Ayé YD, Ayé YM, Babo CJ, Bouh KJ, Amonkou AA (2012) Atteintes putaminales au cours d'une intoxication à l'alcool frelaté: à propos d'un cas. Rev Afr Anesthesiol Med Urg 17: 86-91.
21. Nazir S, Melnick S, Ansari S, Kanneh HT (2016) Mind the gap: a case of severe methanol intoxication. BMJ Case Rep 2016: bcr2015214272.

22. Korabathina K (2021) Methanol Toxicity. [En Ligne].

23. Liu JJ, Daya MR, Carrasquillo O, Kales SN (1998) Prognostic factors in patients with methanol poisoning. J Toxicol Clin Toxicol 36: 175-181.

24. Meyer RJ, Beard ME, Ardagh MW, Henderson S (2000) Methanol poisoning. N Z Med J 113: 11-13.

25. Hassanian-Moghaddam H, Pajoumand A, Dadgar SM, Shadnia Sh (2007) Prognostic factors in methanol poisoning. Hum Exp Toxicol 26: 583-586.

26. Shah S, Pandey V, Thakore N, Mehta I (2012) Study of 63 cases of methyl alcohol poisoning (hooch tragedy in Ahmedabad). J Assoc Physicians India 60: 34-36.

27. Sanaei-Zadeh H, Emamhadi M, Farajidana H, Zamani N, Amirfarhangi A (2013) Electrocardiographic manifestations in acute methanol poisoning cannot predict mortality. Arh Hig Rada Toksikol 64: 79-85.

28. Rostrup M, Edwards JK, Abukalish M, Ezzabi M, Some D, et al. (2016) The methanol poisoning outbreaks in Libya 2013 and Kenya 2014. PLoS One 11: e0152676.

29. Paasma R, Hovda KE, Hassanian-Moghaddam H, Brahmi N, Afshari R, et al. (2012) Risk factors related to poor outcome after methanol poisoning and the relation between outcome and antidotes-a multicenter study. Clin Toxicol (Phila) 50: 823-831.

30. Coulter CV, Farquhar SE, McSherry CM, Isbister GK, Duffull SB (2011) Methanol and ethylene glycol acute poisonings - predictors of mortality. Clin Toxicol (Phila) 49: 900-906.

31. Paasma R, Hovda KE, Tikkerberi A, Jacobsen D (2007) Methanol mass poisoning in Estonia: Outbreak in 154 patients. Clin Toxicol 45: 152-157.