ORIGINAL ARTICLE

# Acute methanol poisoning in Tunisia: clinical features, biological and associated factors for mortality

## Intoxication aiguë au méthanol en Tunisie : Caractéristiques cliniques, biologiques et facteurs de mortalité

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### 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 Eid al- Fitr) corresponding to May 24, 2020.
**Results:** We included 65 male victims of MP. The median [interquartile] age was 28.0 [21.0 – 35.0] years with extremes ranging from 17 to 75 years. The median [interquartile] 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%). Four patients remained visually impaired and 8 patients (12.3%) had died. The univariate analysis reported an association between mortality and age, amount of methanol ingested, co-ingestion of cannabis, delay to consultation, neurological distress, seizures, lower systolic and diastolic blood pressure, metabolic acidosis, lower levels of potassium, higher levels of sodium, hematocrit, glycemia, creatinine, anion gap, and high Acute Physiology and Chronic Health Evaluation II score.
**Conclusion:** Mortality rate following MP was high and was associated with several factors.

**Keywords:** Epidemiology; Methanol; Poisoning; Prognosis; North Africa

### Résumé

**Contexte :** L'intoxication aiguë au Méthanol est rare mais potentiellement grave.
**Objectifs :** Étudier les caractéristiques cliniques et biologiques de l'intoxication aigue au methanol et ses facteurs associés à la mortalité.
**Méthodes :** Nous avons mené une étude transversale incluant une série de cas d'intoxication aigue au méthanol qui a eu lieu à Kairouan, Tunisie. Les cas ont commencé à consulter les urgences un jour festif (1er jour d'Aïd al-Fitr) correspondant au 24 mai 2020.
**Résultats:** Nous avons inclus 65 hommes victimes d'intoxication au méthanol. La médiane [interquartile] de l'âge était de 28,0 [21,0 – 35,0] ans avec des extrêmes allant de 17 à 75 ans. La médiane [interquartile] du entre l'ingestion de méthanol et la consultation médicale était de 48,0 [24,0 – 50,0] heures. A l'admission, la majorité des patients ont décrit des symptômes neurologiques (98,4%) et gastro intestinaux (51,4%). Quatre patients restaient malvoyants et 8 patients (12,3 %) ont été décédés.
L'analyse univariée a révélé une association entre la mortalité et l'âge, la quantité de méthanol ingérée, la co-ingestion de cannabis, le délai de consultation, le coma, les convulsions, la baisse de la pression artérielle systolique et diastolique, l'acidose métabolique, aux taux faibles de potassium, aux taux élevés de sodium, d'hématocrite, de glycémie, de créatinine et de trou anionique , et à un score élevé d'«*Acute Physiology and Chronic Health Evaluation II*".
**Conclusion :** Le taux de mortalité suite à l'intoxication par le méthanol s'est avéré élevé et associé à plusieurs facteurs.

**Mots clés :** Epidémiologie; intoxication; méthanol; Pronostic; Afrique du nord

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## INTRODUCTION

Methanol (CH3OH), formerly known as wood alcohol because it was obtained by distillation of wood, is now prepared by synthesis (1). Acute Methanol Poisoning (MP) is most often results from accidental ingestions due to distillation, fermentation errors and its use as an unlisted ingredient in supposedly alcohol-based products (2).

The clinical presentation of acute MP may mislead clinicians depending on the nature of the toxic alcohol, exposure time, and coingestion of ethanol (3,4). The toxicity of these products is not related to the molecules ingested but to their metabolites (3). In Tunisia, there is no record of MP and epidemiological and clinical data are missing on this subject.

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

## METHODS

We conducted a cross-sectional study including victims of a collective acute MP which took place in Hajeb el Ayoun, Kairouan, Tunisia. This city belongs to the governorate of Kairouan in the center of Tunisia. Its area is 6,712 km² with 581,300 inhabitants, i.e. 86.6 inhabitants/km². This governorate suffers from several socio-economic problems. It is placed in the 22nd position on the national scale of poverty and is also known for its high rates of suicide, school drop outs, illiteracy and unemployment (5).

The victims started consulting the Emergency Room (ER) after drinking locally treated cologne sold by a wholesaler for drinking purposes on a festive day (1st day of Eid al-Fitr) corresponding to May 24, 2020. The last case was recorded on May 27, 2020 at 6 p.m. We did not include two patients who died at home before arriving at the ER and three who escaped from the hospital.

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 a standard neurological examination and complete ocular examination with standard ophthalmological tests and a magnetic resonance imaging (MRI) was performed in case of neurological and ophthalmological abnormalities. We conducted laboratory analyses on blood samples already drawn for treatment purposes. Methanol was measured by a gas chromatographic method with flame ionization detection and a direct injection with internal, limit of detection 6 mg/dL(1.9 mmol/L). Calibrators and controls were made by dilution of methanol. According to the literature, a methanol concentration of less than 0.5 mg/l is physiological (6). Above 0.5g/l the intoxication is serious and the prognosis is life-threatening (7).The severity of the intoxication was assessed by the Acute Physiology and Chronic Health Evaluation II (APPACHE II) score which is used in intensive care units to assess the degree of severity of illness based on clinical and biological features (8).

All patients were managed following standard protocols (3,9,10) using available treatments.

According to the outcome, patients were divided into 2 groups: group I, patients who survived and group II, patients who died.

All statistical analyses were performed using SPSS Version 20.0. The categorical data were expressed as numbers and percentages and the quantitative data as median with an interquartile range [IQR], a appropriate. For comparisons of data between groups, non-parametric tests such as Mann Whitney U test and Kruskal-Wallis 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 baseline characteristics of cases are presented inTable 1.The study population consisted of 65 methanol poisoned patients. All the included subjects were males having a median [IQR] age of 28.0 [21.0 – 35.0] years with extremes ranging from 17 to 75 years. All were smokers and 64.6% were alcoholic. The median [IQR] amounts 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.Twenty seven cases(46.1%) co-ingested other substances concomitantly with methanol such as cannabis (15.3%), organochlorine (3%), Parkizol (3%) and ethanol (1.5%).

**Table 1.** Socio-demographic and methanol consumption characteristics of methanol –poisoned patients (n=65) in Kairouan/Tunisia, 24 may 2020

| Socio-demographic and methanol consumption characteristics | | Total (n = 65) |
|---|---|---|
| | | 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) |
| | Loborer | 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 [IQR]** |
| **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] |

IQR: interquartile range

The median [IQR] delay between methanol ingestion and medical consultation was 48.0 [24.0 – 50.0] hours with extremes ranging from 7 to 72 hours.

Tables 2 and 3 demonstrate clinical manifestations, laboratory data, funduscopy and imagery results of the methanol poisoned patients on admission.

**Table 2.** Clinical presentation of methanol –poisoned patients (n=65) in Kairouan/Tunisia, 24 may 2020.

| | Total (n =65) |
|---|---|
| | 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) |
| Unreactive mydriasis | 3 (4.6) |
| Seizures | 8 (12.3) |
| Signs of shock | 9 (13.8) |
| | Median [IQR] |
| SBP (mmHg) | 120.0 [110.0 – 130.0] |
| DBP (mmHg) | 70.0 [60.0 – 80.0] |
| HR (bpm) | 90.0 [80.0 – 106.5] |
| RR (bpm) | 20.0 [18.0 – 22.0] |
| $SpO^2$ (%) | 98.0 [96.0 – 99.0] |

DBP: diastolic blood pressure, HR: heart rate, IQR: interquartile range RR: respiratory rate, SBP: systolic blood pressure, $SpO^2$: oxygen saturation measured by pulse oximeter.

**Table 3.** Biological and radiological features of methanol – poisoned patients (n=65) in Kairouan/Tunisia, 24 may 2020

| | Total (n=65) |
|---|---|
| | n (%) |
| Dose of methanolemia >0.5g/l | 11 (16.9) |
| Toxic optic neuritis | 4 (6.1) |
| Toxic encephalopathy | 4 (6.1) |
| | Median [IQR] |
| 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] |
| Glycaemia (mmol/l) | 6.0 [5.6 – 6.6] |
| Creatinemia (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] |
| APACHE II 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] |

APACHE II: Acute Physiology and Chronic Health Evaluation II, IQR: interquartile range, $PaCO^2$: partial pressure of carbon dioxide, $PaO^2$: partial pressure of oxygen, PH: Potential hydrogen

On admission, the majority of patients presented with neurological (98.4%) and gastrointestinal symptoms (51.4 %). Headache was the most common neurological symptom (87.7%) and abdominal pain was the most gastrointestinal presenting sign (32.3%). Seven patients were comatose on admission (20%) and 8 presented seizures (12.3%). Visual disturbances were present in 41.5% of cases with blurred vision in 27 patients and visual impairment in 6 patients. The visual acuity test was abnormal in 43.4 % of cases and the pupillary light reflex showed a mydriasis in 9.3%of cases. The rest of the ophthalmic evaluation demonstrated an optic neuritis which was confirmed by an MRI (12.3%). Half of patients with optic neuritis had associated signs of toxic encephalopathy on imagery (6.1%).

Concerning blood tests, more than half of patients presented with a metabolic acidosis (57.3%) and hadhypokalemia (52%). Hyper-osmolarity exceeding 290 mmol was noted in 41.5% of cases. The anion gap was higher than 20 mmol/l in 24.6% of patients with a median of 32.0 [28.5 – 40.2]mmol/l.. Laboratory investigations showed other biological disorders such as acute renal failure (15.4%) and acute pancreatitis (18.5%) with hypoglycemia (15.4%). Methanol serum level was higher than 0.5 g/l in 16.9% of patients. The median APACHE II score was 2.0 [0.0 – 7.0].

As for immediate resuscitation, only 20% of patients required mechanical ventilation and 70.7% needed oxygen. All patients received hydration with saline and eight patients (12.3%) required the administered to 33 patients (50.7%) and folic acid to only 5 patients (7.7%).

The majority of patients (75.3%) were referred to specialized departments, 43% were transferred to an intensive care unit, 18.4% were transferred to the Department of Intensive Care Medicine and Clinical Toxicology in Tunis, 13.8% were transferred to the medical department and 18.4% were kept in the ER due to the lack of hospital beds. The median length of stay in the ER was 24.0 [8.0 – 60.0] hours with extremes of 2 and 96 hours.

Among the 65 victims, 53 made a complete recovery (81.5%), 4 kept a visual impairment (6.15%) and 8 died (12.3%).

A univariate analysis evaluating the associated factors of mortality in patients with MP showed a significant association between mortality and certain features as shown in table 4.

We found that age, amount of methanol ingested, quantity of water diluting methanol, co-ingestion of cannabis, and delay between ingestion and consultation were significantly associated with mortality.

On admission, those who died had a significantly higher frequency of coma compared to survivors.

However, survivors had a significantly higher prevalence of headache and dizziness compared to the deceased.

Mortality was also associated with lower systolic and diastolic blood pressure, metabolic acidosis, high APACHE II score, lower levels of potassium and higher levels of sodium, hematocrit, glycemia, creatinine, plasma osmolarity and anion gap.

**Table 4.** Associated factors with mortality among methanol –poisoned patients (n=65) in Kairouan/Tunisia, 24 may 2020.

| | | Survivors (n = 57) | Deceased (n=8) | p |
|---|---|---|---|---|
| | | 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 |
| 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) | 0.040 |
| Unreactive 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) | $<10^{-3}$ |
| Dose of methanolemia >0.5g/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 |
| | | Median [IQR] | Median [IQR] | |
| 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] | $<10^{-3}$ |
| 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 (bpm) | | 18.0 [18.0 – 21.0] | 26.0 [22.0 – 28.0] | $<10^{-3}$ |
| $SpO^2$ (%) | | 98.0 [97.0 – 99.0] | 93.0 [90.0 – 95.7] | 0.938 |
| 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 |
| Glycaemia (mmol/l) | | 6.0 [5.5 – 6.4] | 8.0 [7.0 – 10.5] | $<10^{-3}$ |
| Creatinemia (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] | 0.350 |
| APACHE II 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] | $<10^{-3}$ |

APACHE II Score: Acute Physiology and Chronic Health Evaluation II, DBP: diastolic blood pressure, HR: heart rate, IQR: interquartile range, $PaCO^2$: partial pressure of carbon dioxide, $PaO^2$: partial pressure of oxygen, PH: Potential hydrogen, RR: respiratory rate, SBP: systolic blood pressure, $SpO^2$: oxygen saturation measured by pulse oximeter.

**Table 5.** mortality rates related to methanol poisoning in different countries from 1998 to 2020.

| Country | Year | Number of cases | Number and rate of deceased cases n (%) |
|---|---|---|---|
| **Canada (25)** | 1998 | 50 | 18(36.0) |
| **United States (26)** | 2000 | 24 | 8 (33.3) |
| **Norway (4)** | 2005 | 51 | 9 (17.6) |
| **Iran (27)** | 2007 | 25 | 12 (48.0) |
| **Tunisia (17)** | 2007 | 16 | 3 (19.0) |
| **India (28)** | 2012 | 63 | 20 (31.7) |
| **Iran (29)** | 2013 | 42 | 17 (40.5) |
| **Czechia (13)** | 2014 | 121 | 41 (33.9) |
| **Taiwan (19)** | 2014 | 32 | 11 (34.4) |
| **Canada (9)** | 2015 | 55 | 1 (1.8) |
| **Libya (30)** | 2016 | 1066 | 101 (9.5) |
| **Kenya (30)** | 2016 | 467 | 126 (26.9) |
| **Czechia (15)** | 2017 | 106 | 23 (21.7) |
| **Uganda (12)** | 2017 | 15 | 12 (80.0) |
| **Taiwan (21)** | 2018 | 50 | 14 (28.0) |
| **China (14)** | 2019 | 52 | 2 (3.8) |
| **Tunisia *** | 2020 | 65 | 8(12.3) |

*Results found in our current study, IQR: interquartile range

## DISCUSSION

The number of poisoned subjects recorded was 65. The median age was 28.0 [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 presented with neurological (98.4%) and gastrointestinal symptoms (51.4%). Mortality rate was 12.3%. We found an association between mortality and age, amount of methanol ingested, amount of water diluting methanol, co-ingestion of cannabis, and delay between ingestion and consultation and neurological distress.

MP stands as a challenge for healthcare providers. It is mainly collective, occurring during festive days (3). Despite improvements in care, morbidity and mortality remain high (7). Studies reporting mass MP are scarce (2,3). The number of cases of MP reported in the literature was variable (11).

For instance, 15 victims of MP were reported in Uganda (12), 26 cases in Morocco (7) and 121 cases in Czech Republic (13).

All patients were males. Our results are in accordance with results from the literature showing a male predominance in methanol intoxication (7,12-15). This could be explained by the particularity of the Tunisian population where only a minority of women consume alcohol due to socio-cultural norms.

Concerning sources of methanol, the outbreak was caused by drinking a low priced and locally treated cologne sold by a wholesaler for drinking purposes in a festive context. 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%) (16).

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 (9,14,17). For instance,

Brahmi et al. (17) found a delay of 36 hours (range between 6 and 48 hours). In Canada, the authors reported a delay of 38 ± 1.51 hours (range between 15 and 85 hours) (9). In China, the delay was estimated to 41.52 ± 0.72 hours (14). This delay could be explained by the fact that many patients were alcoholic 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 (4).

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 frequently reported in 18 to 67% of cases (4,10,18). Dyspnea was reported in 8 to 25% of cases (4,10,18). Visual disturbances were present in 29 to 64% of cases (4,10) and neurological symptoms, especially coma, was reported in 10.7% to 36% of cases (7,17,19-21). Neurological deterioration generally occurs gradually and coincides with advanced stages of intoxication (19). The severity of central nervous system (CNS) damage is directly related to the degree of metabolic acidosis caused by the accumulation of formic acid (22,23). The CNS is a main target of methanol intoxication, especially the brain and the visual pathways which are sensitive to formic acid (22,23). 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 (22,23). These lesions can also affect the cerebellum and the hypothalamus (22).

Systemic toxicity was also described in the literature such as hemolysis or rhabdomyolysis with secondary renal failure, pancreatitis and acute hepatitis (22). Formic acid causes metabolic acidosis with a high anion gap by inhibiting oxidative reactions which promote anaerobic metabolism generating lactic acid and pyruvic acid worsening acidosis (17,19,22). According to Nazir et al. (24) the triad of metabolic acidosis, plasma hyperosmolarity and a high anion gap points towards methanol intoxication.

Our mortality rate is considered high, which is consistent with results from other studies (17,22). We noted a big

variance in mortality rates between different studies as shown in table 5. It ranged from 1.8% in a study in Canada (9) to 80% in Uganda (12).

Despite the improvement in treatment, morbidity and mortality following MP remains high. This may be explained by the delay in diagnosis and therapeutic management (3,12,17). Furthermore, our analysis of associated factors for mortality concluded that the quantity of methanol ingested, the quantity of water diluting methanol, the delay of consultation, metabolic acidosis and some clinical and biological features were significantly associated with mortality. Our results are in accordance with those from other studies (4,11,14,20). Severe metabolic acidosis was the most described predictor of death. In a study conducted by Meyer et al. (26) the most important predictor of mortality was acidosis with blood pH of 7.0. A study conducted in Norway also revealed that severe metabolic acidosis (pH of 6.9) was a strong predictor of death (4). Coulter et al. (31) analyzed the literature data and concluded that a low pH of 7.22 was associated with increased mortality and that pH had the highest predictive value. In Estonia, it was shown that the outcome was related to the degree of metabolic acidosis (32). In a multicenter study of Paasma et al. (11), low pH (pH of 7) was among the strongest predictors of poor outcome.

However, our study had some limitations. 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 clinical 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 laboratory equipment. In addition, the hospital was unable to provide all antidotes for all patients including fomepizole which is not available in Tunisia. Despite these limitations, our study summarizes MP clinical and biological features. In accordance with previous studies, it demonstrates a large number of predictive factors for MP-related mortality.

Acute MP remains serious as it is not only life-threatening but also responsible for damage of several organs and may lead to blindness as well as irreversible damage to the central nervous system (23). Mortality rate following MP was high and associated with several factors. Some of these factors can be modified by effective actions if implemented at the population level such as educational campaigns about MP and the enforcement of laws pertaining to alcohol use.

The early identification of this intoxication and rapid management are essential to improve the prognosis (22).We also recommend timely intravenous administration of ethanol to victims of MP.

In addition, fomepizole should be included

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