Methanol Toxicity in Eastern Province: An Emergency Department and Toxicologist Dilemma - Case Series with a Review of the Literature

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Abstract

Methanol is a clear, colorless, highly toxic alcohol widely used as paint, varnish removers, automotive radiators, and washer fluid. Methanol intoxication incidence can be due to accidental, occupational, suicidal exposure or following an adulterated liquor's ingestion. Methanol is not toxic itself but will be metabolized to the exceedingly poisonous formaldehyde and formic acid. Methanol ingestion ends in wide anion-gap metabolic acidosis and devastating neurological complications, including drowsiness to coma and devastating intracranial hemorrhages. Severe toxicities can occur with an intake of 0.25 ml/kg of 100% methanol. The blood level of methanol above 25 mg/dl is considered highly toxic. Methanol reaches its peak plasma levels within an hour. Treatment guidelines include antidotal therapy, hemodialysis, and metabolic acidosis correction. Due to methanol's poisoning severity and grave outcome of the affected cases, we had to present 4 case series for methanol intoxication, each with its clinical manifestations and laboratory findings, to enlighten the medical practitioners about possible causes, presentations, and treatment modalities of such severe toxicity.

Four methanol poisoning case series with different presentations were discussed in the current work. A questionnaire about methanol poisoning awareness was distributed through the ER physicians' and nurses' mail to investigate their ability to diagnose and treat methanol poisoning cases.

More training of ER medical staff on toxicological presentations is highly recommended. Clinical and analytical toxicologists should have a more prominent role in the management of such cases. More light and international publicity should be shed upon the methanol toxicity outbreaks encountered globally, with frequently updating the diagnostic and therapeutic guidelines following an evidence-based approach are highly recommended.

Key Words: *Methanol poisoning, toxicity, hemodialysis, occupational exposure, coma.*

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Objectives

To study Methanol poisoning case series with different presentations admitted to hospitals and go through exposure, diagnosis, and treatment guidelines. A questionnaire about methanol poisoning awareness was distributed through the ER physicians' and nurses' mail to investigate their ability to diagnose and treat methanol poisoning cases.

Background

Methanol intoxication outbreaks pose several difficulties in health care systems globally. Hence, the late hospital presentation delayed case diagnosis and the non-specific clinical features at the time of hospital admission make it a real medical and toxicological dilemma, especially for young physicians or in the absence of a toxicologist. Moreover, the social stigma behind presenting a son or a relative to the hospital with a history of alcoholic beverage ingestion makes the hospital presentation so late, and the statistical recording so far from reality. This adds more to the already established difficult management and poor outcome of the affected cases. In many parts of the world, the diagnostic and therapeutic services in methanol poisoning in several hospitals are sometimes inadequate, with absent or ineffective evidence of comparative clinical efficacy and defined therapeutic guidelines.

Moreover, complications, increased mortality rate, and long-term health problems among the survivors of methanol poisoning make the clinical outcomes of such patients very low.

Frequently there is a declared methanol mass poisoning outbreak in one place or another with high numbers of poisoned subjects and even fatalities reaching thousands of patients. [1,2]

Methanol poisoning leads to a severe medical emergency due to the rapid blockade of the enzyme alcohol dehydrogenase system, an essential pathway to abort the formation of methanol's toxic metabolites. Formaldehyde and formic acid are the metabolic products of methanol produced by alcohol dehydrogenase and acetaldehyde dehydrogenase on methanol. Both metabolites are significantly harmful to the Central Nervous System, retina, optic nerve, and renal system. [3-6] Peak levels of poisonous metabolites are reached about 30 to 90 minutes post-ingestion. These two metabolites are highly toxic and lead to severe wide anion-gap metabolic acidosis, ocular and vicious neurological manifestations. Most toxicity incidents occur after oral ingestion, but rapid absorption of methanol is known to occur through the lungs or skin. The methanol metabolism products, namely formate, have a robust cytotoxic outcome by inhibiting the mitochondrial cytochrome c oxidase activity producing histotoxic hypoxia. [2,7]

After mild toxicity, the serum half-life is 14 to 20 hours, increasing to 24 - 30 hours in severe toxicity. The liver removes methanol (90 to 95%), while renal excretion accounts for 3 to 5 percent only, and pulmonary excretion is trivial. Due to its characteristic delayed metabolism, its metabolic products might be produced in the body up to 7 days post-ingestion. Toxic methanol brain insults can be ischemic, necrotic, or hemorrhagic. [8-11]

Bilateral Putamen necrosis, with or without hemorrhage and subcortical white matter lesions, are the most frequent outcomes in methanol intoxicated patients. Probable explanations include a direct toxic effect of methanol or its metabolites, injury secondary to acidosis, and anoxia. Anaerobic glycolysis and lactic acidosis will lead to superoxide anions and hydroxyl radicals' generation, ending in a state of cellular and membranous damage. Calcium influx into the cells will follow, producing mitochondrial dysfunction, cellular death, and cerebral vasospasm. [10]

Formic acid accumulation will lead to different grades of rapidly progressive metabolic acidosis. Metabolic acidosis will be accompanied by basal ganglia and optic nerve damage, leading to visual impairment, significantly when its concentration rises to 0.9 –1.1 mmol/L. (12-16) The optic nerve neurons are selectively susceptible to histotoxic hypoxia as its fibers, and their myelin sheaths have fewer mitochondria and little reserves of cytochrome oxidase being of low metabolic requirements. (15,16) Many formate toxicity-induced morphologic and biochemical changes are observed in retinal photoreceptors, retinal glial cells, and the subretinal pigment epithelium.(3,17)

A timely diagnosis of the disease and the start of the proper treatment to minimize the severity of the metabolic disturbances is the most crucial determinant of morbidity and mortality due to methanol poisoning. During treatment, ethanol might be used as an antidote to saturate the alcohol dehydrogenase system to prevent the production of toxic metabolites. Sodium bicarbonate is needed to treat the severe metabolic acidosis encountered in patients with methanol poisoning. Folic and folinic acid is needed to increase formic acid biotransformation and to prevent its accumulation. Hemodialysis in severely poisoned patients is a crucial treatment line augmented by the administration of these medicines. Folates augment the metabolism of formates transforming it into 10-formyl tetrahydrofolate by the action of 10-formyl tetrahydrofolate synthase followed by its oxidation to carbon dioxide catalyzed by 10-formyl-tetrahydro folate dehydrogenase, thus getting rid of the toxic metabolites of methanol. (5,9,18-20) A folate derivative augments formate oxidation by preventing the development of enzyme catalyst short metabolic pathways. (21) Because of this, and several experimental studies, the folic or folinic acid administration to the methanol-poisoned patients is routinely recommended. (22) Although folinic acid is preferred to folic acid since it does not necessitate its metabolic reduction, folic acid is well thought out as a suitable alternative. (11)

Methods

Analysis of blood and urine samples for methanol, ethanol, and abused drug detection is not available as a routine analysis in most hospitals. Toxicology consultation was performed in all cases by the treating physician. The toxicologist on call gave treatment recommendations and requested blood and urine sample collection for toxicological analysis. All samples were referred to the Regional Poison Control Centre in Dammam for analysis and result interpretation. Analysis of the collected samples was performed using ARCHITECT system c4000, model i1000 SR by Abbott laboratories to test for the presence or absence of ethanol, amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, tramadol, and opiates. Confirmation for any positive result was confirmed by Gas chromatography Mass Spectrometry (GC-MS-QP2010, Shimadzu, Japan). Volatile analysis for detecting ethanol, methanol, ethylene glycol was done using gas chromatography – headspace (GC ultra-model K0C33B730000000, Milano, Italy) following the technique adopted by **Sahar Y. Issa** et al. (1). A questionnaire about methanol poisoning awareness was distributed to a total of 250 ER physicians' and nurses' via their emails to investigate their ability to diagnose and treat methanol poisoning cases.

Results

As seen in figure (1-A), only 124 respondents from medical personnel (56 physicians and 68 nurses) completed the survey and sent it back to our team. Male physicians constituted about 26% of respondents, while female nurses were about 42%. The females (62%) surpassed the males (38%) in our surveyed group of medical personnel (Figure 1-B).

Most of the responses (54%) came from medical personnel working in governmental hospitals (Figure 1-C). Figures (2-A and 2-B) emphasized that most of the respondents had working experience ranging from <1 -5 years.

As seen in figure (2-C), less than half of the investigated groups (43%) demonstrated their familiarity with the clinical picture of methanol Toxicity. Only 28% of them had an established policy for the management of methanol poisoning in their Workplace. A quarter of the investigated group expressed their ability to train others about the diagnosis and management of methanol poisoning. 56% of the studied groups gave proper answers regarding methanol poisoning treatment guidelines, antidotal regimens, and arterial blood gases interpretation in cases of methanol poisoning. 32% had managed methanol poisoning cases before. 19% of the respondents confirmed having full access to digital libraries, books, or a supervisor consultation that might help them diagnosing methanol poisoning cases. 92% of the respondents found it more manageable and rapid to get toxicological consultations through electronic systems or phone calls with professional toxicologists.

Case Studies

First Methanol Poisoning Case:

A 42-year-old Indian male with a known history of alcohol abuse was transferred to the emergency department after being discovered by his roommate unconscious in his bed. His vitals, laboratory Fundoscopic, and radiological findings are displayed in table (1) in detail.

On the Emergency room (ER) presentation, his neurologic examination revealed a Glasgow Coma Score (GCS) of 3 with dilated fixed pupils in a gasping state with SpO_2 65%. The cardiovascular examination did not reveal any abnormal findings. Shortly after arrival, immediate intubation and admission to the intensive care unit were made. The toxicologist on-call was contacted, and the suspicion for being a case of suspected methanol poisoning was raised, and both blood and urine samples were requested for alcohol and illicit drugs analysis. Initial arterial blood gas measurement following endotracheal intubation revealed severe metabolic acidosis: bicarbonate 9.2 mmol/L; pH 6.39; and PCO_2 52 mm Hg. Laboratory tests showed an Osmolar gap of 143 mmol/L and an anion gap metabolic acidosis of 31. Serum ethanol concentration was 127mg/dl, while serum methanol levels were 53 mg/dl. Urine was examined for the presence of calcium oxalate crystals and proved negative. Urine drug screen for drugs and substances of abuse and other drugs was negative. CT scan of the brain revealed extensive brain edema.

Shortly after hospital admission, the patient developed seizures that were controlled by benzodiazepines. The toxicology report diagnosed the patient as a case of acute methanol poisoning, and hemodialysis was requested. Later, the patient's roommate gave a history of ingestion of facial wash purchased from the market two days before the incident, and he brought one of the bottles consumed by his colleague. On inspection of this facial wash bottle, the ingredients label showed 55% wood alcohol as an ingredient. Toxicological analysis of the facial wash indicated the presence of methanol in a concentration of about 98%. Sodium bicarbonate infusion was already started to correct the severe metabolic acidosis. The antidote 4-methyl pyrazole, besides the intravenous folinic acid, was shortly started.

Hemodialysis was performed to support methanol and formic acid elimination. Treatment improved metabolic disorders, and methanol serum concentrations started to decline. However, there was no improvement in his neurologic condition, and the patient remained in a comatose status. At 48 hours post-admission, brain death was declared and documented by EEG. The likelihood of family approval for organ donation was requested, and the patient's agreement was obtained, and the patient was treated as a possible organ donation candidate.

Second Methanol Poisoning Case Study:

A 29-year-old Saudi male with a history of psychiatric illness and depression presented to the emergency department with his family in an unconscious status suspecting a suicidal attempt by their son by ingesting an unknown chemical. On examination, his vital signs indicated tachypnea (30 breaths/min), low blood pressure 79/45 mm Hg, tachycardia (109 beats/min), and temperature of 36.4°C.

Pulmonary and cardiac examinations revealed bilaterally equal air entry, with regular rhythm and heart sounds. Neurological examination revealed a Glasgow coma scale of 3/15 with bilaterally dilated and non-reactive pupils and absent reflexes. The patient was admitted to the medical intensive care unit, where mechanical ventilation was started.

The examination and investigations result on admission are displayed in detail in table (2). These laboratory and radiological tests main findings were plasma creatinine: 1.9 mg/dl, blood urea nitrogen: 22 mg/dl; arterial blood gases pH: 6.82, $PaCO_2$: 61 mm Hg, bicarbonate: 9.6 mmol/l, potassium: 4.1 mmol/l, sodium: 139 mmol/l and chloride of 123 mmol/l. The anion gap was 29 mmol/l with an Osmolar gap of 121 mmol/L. Random blood sugar was 112 mg/dl; renal functions were greatly impaired, showing BUN of 34 mg/dl and creatinine of 3.6 mg/dl. The drug Toxicology report showed a methanol serum level of 148 mg/dl, absent ethanol level, and positive urine screen for Fluoxetine. The patient underwent a CT scan of the brain that showed extensive pontine hemorrhage (Figure3-I). The treating physician immediately requested hemodialysis after consulting the on-call nephrologist. Post hemodialysis, methanol level became 12 mg/dl. The patient was given an infusion of sodium bicarbonate to manage severe metabolic acidosis and, Fomepizole as a methanol antidote was started. On the second day in ICU patient was declared dead following cardiorespiratory arrest with failed resuscitation attempts by the medical attendant.

Third Methanol Poisoning Case Study:

A 21-year-old Saudi male was admitted to an Emergency Department in a private hospital with a history of visual impairment and breathing difficulty in the last one day following ingestion of liquor with his colleagues working in a perfume selling shop. Shortly after he arrived at the ER, he started to deteriorate, especially visual impairment, with decreased urine output. Due to the unavailability of a toxicology screen in that private hospital, the patient was referred to a Governmental Central Hospital, where the ER physician requested blood gases examination, urine, and blood toxicology screen, in addition to serum ethanol and methanol levels. His clinical examination shortly after arrival demonstrated tachycardia (120 beats/min), tachypnea (37 breaths/min), average temperature (37.2 ^{0}C), and hypotension (78/56 mm Hg). He had dilated, mildly reactive pupils, and a fundoscopic examination revealed a disc pallor, optic disc edema, and blurred disc margins (figure 3-II).

Blood gases showed severe wide anion gap metabolic acidosis. His methanol blood level was 113 mg/dl, whereas his serum ethanol level was about 15 mg/dl. The patient reported that about 36 hours before, he drank alcohol-based juices prepared locally by his colleagues in a perfume selling shop. Antidote therapy with Fomepizole was started together with sodium bicarbonate intravenous infusion to treat the metabolic acidosis. Hemodialysis was initiated shortly after his admission to the intensive care unit. The patient recovered from metabolic acidosis, but his visual acuity, after an initial improvement, worsened significantly to 18/90 on the right eye and 6/120 on the left eye, and this condition was considered questionable for future improvement by the treating ophthalmologist. Renal functions showed initial deterioration but got improved with hemodialysis and medical care. Detailed radiological, laboratory and clinical examination data are given in table (3).

Fourth Methanol Poisoning Case:

A 34-year-old Indian male working in a petrochemical plant in Jubail was transferred in an unconscious and desaturated state to the emergency department after falling in a methanol tank while performing service maintenance to the tubing system. All his clothes were soaked with methanol. All clothing was discarded, and thorough skin decontamination was performed. A chest x-ray performed shortly after admission showed evidence of aspiration pneumonitis and pulmonary edema. His vitals, laboratory findings are displayed in table (4) in detail. Soon after ER's presentation, the patient was admitted to the intensive care unit, and emergency hemodialysis was started to correct his wide anion gap metabolic acidosis. Given the history and clinical manifestations confirming methanol's intoxication, whose level was requested immediately, it was chosen to start bicarbonate, leucovorin sodium, fomepizole infusions a high flow hemodialysis session for about 8 hours was performed. The patient's condition did not improve, and by early morning on the next day, he was arrested and declared dead despite the executed resuscitation attempts.

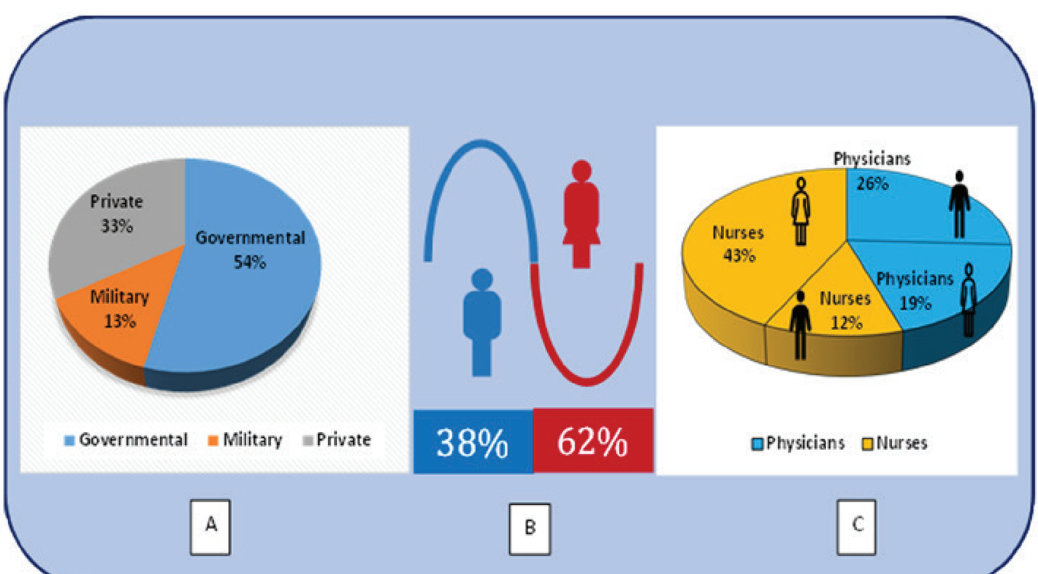

Figure (1): (1-A): Investigated Medical Team, (1-B): Gender Distribution Among Investigated Medical Professionals, (1-C): Types of Medical Facilities of Surveyed Medical Personnel

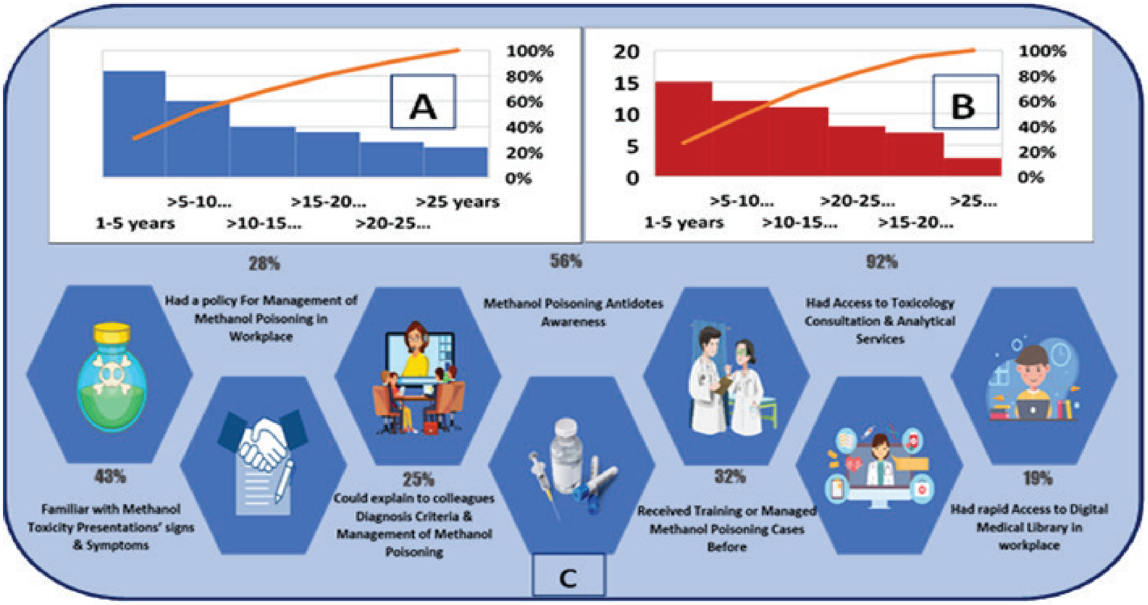

Figure (2): (2-A) Years of Experience Among the Investigated Physicians, (2-B): Years of Experience Among the Investigated Nurses, (2-C) Proportions of Different responses among surveyed Medical Personnel about Methanol Poisoning

Figure (3): (3-I) CT Brain Showing Pontine Hemorrhage, (3-II): Fundoscopic Examination revealing bilateral (A: Right, B: left) optic disc edema with blurred disc margins.

Table (1): Clinical/Laboratory/and Radiological data for the first Methanol Poisoning case

Examination/Investigation	Day 1	Day 2	Day 3
Vital Signs			
Blood Pressure	112/63	101/60	74/52
Heart Rate (beat/min)	102	95	64
Respiratory rate (Breath/min)	36	On mechanical ventilator	
Temperature (0 C)	35.4	36	35.2
Consciousness Level			
Glasgow Coma Scale (GCS)	3	3	3
Toxicology Screen			
Serum Methanol Level (mg/dl)	53	23	1.7
Serum Ethanol Level (mg/dl)	127	17	0
Drugs of abuse	None detected		
Other Drugs	None detected		
Blood Gases			
pH	6.39	6.52	7.14
CO2 (mmHg)	52	18	24
HCO3 (mmol/l)	9.2	13.6	28.7
Osmolar gap (mmol/l)	143	136	141
Anion Gap (mEq/l)	31	25.4	18.3
Chemistry			
Random blood sugar (mg/dl)	162	154	128
BUN (mg/dl)	25	18	20
Creatinine (mg/dl)	2.4	1.6	1.35
Potassium (mEq/l)	4.7	3.9	2.4
Sodium (mEq/l)	142	156	180
Chloride (mEq/l)	115	129	163
Urine calcium oxalate crystals	Negative		
Fundoscopic Examination	Bilateral optic disc hyperemia and venous engorgement		
CT Brain	Brain edema		

Table (2): Clinical/Laboratory/and Radiological data for the second case

Examination/Investigation	**Day 1**	**Day 2**
Vital Signs		
Blood Pressure	79/45	84/50
Heart Rate (beat/min)	109	92
Respiratory rate (Breath/min)	30	On mechanical ventilator
Temperature (0 C)	36.4	37
Consciousness Level		
Glasgow Coma Scale (GCS)	3	3
Toxicology Screen		
Serum Methanol Level (mg/dl)	148	12
Serum Ethanol Level (mg/dl)	0	0
Drugs of abuse	**None detected**	
Other Drugs	**Fluoxetine**	
Blood Gases		
pH	6.82	7.1
CO2 (mmHg)	61	14
HCO3 (mmol/l)	9.2	13.6
Osmolar gap (mmol/l)	121	142
Anion Gap (mEq/l)	29	12.1
Chemistry		
Random blood sugar (mg/dl)	112	114
BUN (mg/dl)	34	17
Creatinine (mg/dl)	3.6	1.4
Potassium (mEq/l)	4.1	3.7
Sodium (mEq/l)	139	144
Chloride (mEq/l)	123	121
Urine calcium oxalate crystals	Negative	
Fundoscopic Examination	Bilateral severe optic disc hyperemia	
CT Brain	Pontine Hemorrhage	

Table (3): Clinical/Laboratory/and Radiological data for the third case

Examination/Investigation	Day 1	Day 2	Day 3
Vital Signs			
Blood Pressure	78/56	110/62	117/81
Heart Rate (beat/min)	120	89	82
Respiratory rate (Breath/min)	37	On mechanical ventilator	
Temperature (0 C)	37.2	36.9	36.7
Consciousness Level			
Glasgow Coma Scale (GCS)	9	10	14
Toxicology Screen			
Serum Methanol Level (mg/dl)	53	23	1.7
Serum Ethanol Level (mg/dl)	127	17	0
Drugs of abuse	None detected		
Other Drugs	None detected		
Blood Gases			
pH	6.89	6.32	7.27
CO2 (mmHg)	52	18	24
HCO3 (mmol/l)	9.2	13.6	28.7
Osmolar gap (mmol/l)	143	136	141
Anion Gap (mEq/l)	31	25.4	18.3
Chemistry			
Random blood sugar (mg/dl)	162	154	128
BUN (mg/dl)	25	18	20
Creatinine (mg/dl)	2.4	1.6	1.35
Potassium (mEq/l)	4.7	3.9	2.4
Sodium (mEq/l)	142	156	180
Chloride (mEq/l)	115	129	163
Urine calcium oxalate crystals	Negative		
Fundoscopic Examination	Bilateral optic disc hyperemia and venous engorgement		
CT Brain	Brain edema		

Table (4): Clinical/Laboratory/and Radiological data for the third case

Examination/Investigation	Day 1	Day 2
Vital Signs		
Blood Pressure	64/48	70/52
Heart Rate (beat/min)	78	84
Respiratory rate (Breath/min)	On mechanical ventilator	
Temperature (0 C)	36.8	37.4
Consciousness Level		
Glasgow Coma Scale (GCS)	3	3
Toxicology Screen		
Serum Methanol Level (mg/dl)	403	185
Serum Ethanol Level (mg/dl)	0	0
Drugs of abuse	None detected	
Other Drugs	None detected	
Blood Gases		
pH	6.84	7.16
CO2 (mmHg)	57	19
HCO3 (mmol/l)	9.3	11.2
Osmolar gap (mmol/l)	289.3	293.5
Anion Gap (mEq/l)	29.7	23.6
Chemistry		
Random blood sugar (mg/dl)	188	127
BUN (mg/dl)	34	28
Creatinine (mg/dl)	2.1	1.9
Potassium (mEq/l)	3.9	3.6
Sodium (mEq/l)	138	127
Chloride (mEq/l)	98	102
Urine calcium oxalate crystals	Negative	
Fundoscopic Examination	Bilateral optic disc edema and congestion	
CT Brain Chest x-ray	Brain edema Aspiration pneumonitis & pulmonary edema	

Discussion and Review of the Literature

Methanol is a commercial and industrial solvent that is widely available and is a potentially fatal poison. While toxicity after skin absorption or inhalation is possible, the main route of poisoning is ingestion. Ingestion is usually encountered in cases involving intake of cheap alcohol or accidental ingestion in children. Methanol or "wood alcohol" outbreaks of poisoning from adulterated liquor sources have also been observed. Methanol can be fatal following the ingestion of as small as 60 to 240 mL methanol or even 15 to 30 mL of the more concentrated types. (21) Methanol blood levels peak 30 to 90 minutes after ingestion, with a distribution volume of 0.6 to 0.7 L/kg. (23-25) Only 5% of ingested methanol is excreted unchanged in the urine, but the main elimination pathway is hepatic and renal biotransformation into formaldehyde and then formic acid. Methanol's metabolites are primarily responsible for its toxicity much more than their parent compound. (26)

Inebriation and drowsiness are early signs of intoxication. Secondary to local conversion to formaldehyde, delayed symptoms may include blurred vision, dilated pupils, and retinal toxicity (optic disc hyperemia and possible blindness). Vomiting, diarrhea, vertigo, cold and clammy extremities, bradycardia, delirium, agitation, and decreased urine output with a formaldehyde odor are other delayed symptoms. Kussmaul respiration, coma, inspiratory apnea, convulsions, and death are all possible outcomes of severe intoxication. Necrosis of the putamen and pontine hemorrhages are two severe side effects of methanol poisoning. (27) At autopsy, pancreatitis was also discovered. (21)

As the methanol metabolism is relatively slow (about 8 mg/dL/h) (28), symptomatic acidosis will develop as late as 15–20 hours post-ingestion. If the ethanol were co-ingested with the methanol as the case in most adulterated liquors, the antidote ethanol would increase the periods before symptomatic ill-health is manifested in the affected subjects as 96 hours. (29) During most of the methanol outbreaks, there is a co-ingestion of a mixture of methanol in ethanol, increasing the latency period even longer than 96 hours. Diagnosis based on history is, therefore, hard to attain. (28)

Early in intoxication, the laboratory results show elevated osmolality due to the unmetabolized methanol, followed by a formate retention-induced wide anion gap metabolic acidosis in addition to low bicarbonate. Other findings may include increased sugar level and elevated serum amylase. (29)

Gastric lavage should be performed on patients with suspected methanol toxicity and early symptoms to remove any remaining gastric methanol. Because the symptoms are frequently delayed, treatment entails preventing and removing toxic metabolites. Alcohol dehydrogenase (ADH), an enzyme with higher affinity and ethanol efficiency, is required for the enzymatic oxidation of methanol to formaldehyde. To prevent formaldehyde conversion, patients should be given ethanol or another ADH inhibitor, 4-methyl pyrazole (4-MP). Folic and folinic acid also aids in the transformation of formate to water and carbon dioxide. (30) If 4-MP is available from hospital pharmacies, most medical regimens recommend it as soon as the diagnosis is considered. (31-34)

Hemodialysis is an excellent way to eliminate methanol and its toxic metabolites while also correcting metabolic acidosis. Patients with levels above 50 mg/dL, severe symptoms, or refractory acidosis should consider it. Dialysis should be continued until plasma concentrations fall below 20 mg/dL, with the rebound of plasma concentrations being monitored. If ethanol is given, it can be mixed into the dialysate or substituted after the dialysis is completed. [35-37] Since methanol metabolism has a long half-life, treatment with 4-MP alone is not recommended because it may result in prolonged intensive care stays. If this is the case, hemodialysis can be used to reduce methanol's half-life. [37-38] Dialysis also removes 4-MP, and dose adjustment is necessary. [39, 40] Poisoning from a wide range of medicines and chemical products, including methanol and other toxic alcohols, can lead to many metabolic disorders through a wide range of mechanisms. Some metabolic problems are life-threatening, but they all need close evaluation, supervision, and treatment attention. Rapid diagnosis, treatment, and toxicological consultation will lower the risk of morbidity and mortality. [41] Treatment can be tailored to the particular poisoning and be time-sensitive or adopt general and acute medicine methods. It would be difficult for every doctor to have an in-depth knowledge of all poisons they might encounter in their career, so it's vital to learn the basic principles of poison management and to consult toxicologists when necessary. [42] Toxicologists had a better understanding of methanol poisoning, and hence they typically propose a complicated approach to interpreting methanol level based on a consciousness level, acid-base status, end-organs toxicity, and serial acid-base analysis. Methanol concentrations are often not readily available, whereas coma and acidosis are the more appropriate for the first triage and priority of care in methanol poisoning cases. [43] To our knowledge, this work is the first of its kind to evaluate the medical personnel's awareness about methanol poisoning. Further investigations are highly recommended.

Conclusions

The current work might offer some required updated background about some unusual instances of methanol poisoning that need a high index of suspicion for correct diagnosis and treatment in previously non-diseased healthy human subjects, which might be a significant contribution for diagnosis of possible methanol toxicity sources. These results would permit more straightforward future findings' interpretation in such clinically bizarre cases. More training of ER medical staff on toxicological presentations is highly recommended. Clinical and analytical toxicologists should have a more prominent role in the management of such cases. More light and international publicity should be shed upon the methanol toxicity outbreaks encountered globally, with frequently updating the diagnostic and therapeutic guidelines following an evidence-based approach are highly recommended.

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