Methanol Related Deaths in Jordan

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The aim of this study is to examine methanol poisoning cases from the medico-legal point of view. The records of the National Institute of Forensic Medicine (NIFM)-Jordan were reviewed retrospectively for all methanol poisoning from January 2007 to January 2008. It was found that methanol poisoning comprise 0.92% of all forensic autopsies (n: 16), 87.5% of the cases were males, the mean age of victims was 35, range 14-56 years, the largest age group was 30-39 years accounted for (31.25%, 5 cases) followed by the age group 40-49 years (25%, 4 cases). The methanol blood concentrations ranged widely from 56 to 400 mg/dL. There were 12 cases (75%) with the methanol blood concentrations over 100mg/dL. Fifteen cases were found dead and one was admitted to hospital before death. The main cause of death in all cases was due to methanol intoxication as underlining cause of death

Keywords: Methanol, Jordan, medico legal cases.

Introduction

Methanol (wood alcohol, methyl alcohol, Columbian spirits, colonial spirit, wood naphtha) is the alcohol in its simplest structure. Numerous studies over the years have investigated its toxicity potential, as well as the most beneficial means for treating acute toxicity (Goessel and Bricker, 1990). Methanol is widely used in industry and at home as a general solvent labeled as methyl hydrate and in such products as paint thinner, antifreeze, fluid for desk 'Spirit' duplicating machines and source of heat for fondue burners (e.g., Sterno). Occasionally, at concentrations up to 5%, methanol is used as a denaturant for ethanol that is not intended for human consumption. It is called 'wood' alcohol because formerly its primary source was from the distillation of wood (Goessel and Bricker, 1990; Hodgson, 2000). Most reports of methanol toxicity are because of the ingesting of methanol itself, or a Methanol-containing product, although poisonings have been reported following absorption through the skin and inhalation of air that contained as little as 0.2% (Forney and Harger, 1971). The actual cause of poisoning was from methanol which was added to the substance in order to increase the quantity of product that may be sold (Goessel and Bricker, 1990). Accidental ingestions also occur because methanol closely resembles ethanol in appearance and odor, is readily available, tax-free (thus cheaper), and, in general, is not known by the public as more dangerous than ethanol. Methanol poisoning continues to be a worldwide (although regional) problem associated with high mortality and morbidity (Roe, 1982; Goessel and Bricker, 1990). Sporadic or epidemic accidental use of methyl alcohol is one of the basic problems that forensic medicine professionals have to face in the whole world. Alcoholics know that ethanol may have harmful effects they intentionally substituted methanol containing substances.

In addition to sporadic cases several large epidemics have been reported (Naraqi *et al*., 1979; *Kane et al*., 1968; Benton and Calhoun, 1953; Swartz *et al*., 1981). When we look at the list of methanol poisoning outbreaks in the world, we see the outbreaks in 14 countries. Most events have come into existence in India from 1976 to 2015 and more than 1900 people died(Wikipedia, 2015). Three major methanol outbreaks in Libya (2013) and Kenya (May and July 2014) occurred (Rostrup *et al*., 2016). Four outbreaks were registered from Turkey and they were 21 deaths in 2004, 23 deaths in 2005, 5 deaths in 2011 and 32 deaths in 2015 people (Wikipedia, 2015). The 2012 Czech Republic methanol poisonings occurred in September 2012 in the Czech Republic, Poland, and Slovakia. Over the course of several days, 38 people in the Czech Republic and four people in Poland died as a result of methanol poisoning and several tens of others were taken to hospital (Dziennik, 2012). In 2013, as a result of methanol mass poisoning in Iran 694 people were hospitalised in the city of Rafsanjan. Eight people were reported dead due to severe intoxication (Moghaddm *et al*., 2014). In December 2016, 72 people died in a mass methanol poisoning in Irkutsk, Siberia. The poisoning was precipitated by drinking counterfeit surrogate alcohol actually scented bath lotion that was marked as not safe for consumption. Named *Boyaryshnik* ("Hawthorn"), it was described by the Associated Press as being counterfeit *(*Isachenkov, 2016*)*. In September 2018, 37 people died of methanol poisoning from drinking fake liquor. In this incident, the methanol content was up to 50 times more than the permissible amount. Cases of toxic alcohol poisoning have been reported in Selangor, the Federal Territories, Perak and Negeri Sembilan. The deaths comprised two Bangladeshi, two Indian nationals, eleven Myanmar nationals, eight Nepalese, ten Malaysians and four others of unknown nationality.

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Three Indian nationals, believed to be responsible for the distribution of cheap counterfeit liquor to retailers around Selayang and Desa Jaya which led to the methanol poisoning incident have now been arrested (Selangor, 2018). Review of Jordanian medical literature regarding fatal alcohol toxicity revealed two articles addressing the fatal poisoning of alcohols and drugs with no data regarding methanol fatalities (Hadidi *et al*., 1998; Abu-ragheb and Hadidi, 1999) with just only one data regarding methanol fatalities that reported at the National Institute of Forensic Medicine in 2006 for the first time in Jordan(Abdallat *et al*., 2009). However, we need to continue the surveillance of alcohol poisoning deaths in Jordan to develop an evidence base in order to project the magnitude of the problem on the health of the population.

The aim of this study is to investigate the prevalence and concentration of methanol in medico-legal autopsies in the National Institute of Forensic Medicine during one year from January 2007 to January 2008. As the level of Toxicity of Methanol increases the results will improve. Several examples of artifacts in real EEG signals depend on the functions of the Toxicity of Methanol (Al-Kasasbeh and Lvov 2005; Al Kasasbeh *et al*., 2011). According to (Al-Kasasbeh and Lvov 2005, Al-Kasasbeh and Lvov 2006, Al Kasasbeh *et al*., 2012, Korenevskiy *et al*., 2013), mathematical models were applied for the interaction of the internal and biological active points of meridian structures. It is shown that the use of fuzzy logic decision-making yields good results for the prediction and early diagnosis of diseases depending on the reaction energy of biologically active points (acupuncture points). It is worth mentioning that the above results are related to the level Toxicity of Methanol.

1 Materials and Methods

1.1 Toxic kinetics of methanol

Methanol and 1-propanol have been used throughout the study. They have been chosen for high quality. Glasses (10x22x46mm) headspace vials with Teflon septa and magnetic seal caps, beakers, flasks and micro-pipettes, and other glassware have been selected of high-quality Pyrex, and are available at the toxicology laboratory in the NIFM. Methanol is the simplest alcohol. It is a light, volatile, colorless, flammable liquid with a distinctive odor that is very similar to, but slightly sweeter than, ethanol "Drinking alcohol"(Wikipedia, 2010).

Methanol is absorbed and distributed throughout the body in a manner similar to ethanol (Goessel and Bricker, 1990). Methanol is metabolized by oxidation via alcohol dehydrogenase (ADH) and aldehyde dehydrogenase **Figure 1.** Although ADH metabolizes all alcohols, enzyme-binding affinities vary; the affinity of ADH for ethanol is four times greater than its affinity for methanol. These variable affinities have significant therapeutic importance although peak methanol concentrations are achieved rapidly. They occur about 30 to 90 minutes after ingestion (Gideon, 2002; Goldfrank *et al*., 2002). Metabolism may not be evident until 24 hours (range, 1-72 hours), delaying the development of acidosis and toxic symptoms. In the liver, ADH metabolizes approximately 75-85% of methanol to formaldehyde which is then oxidized by aldehyde dehydrogenase to formic acid in the presence of foliate; formic acid is converted to carbon dioxide and water (Goldfrank *et al*., 2002). Metabolic conversion is not the sole means for elimination. Because of its volatility, methanol can be eliminated unchanged by the lung (10-20%), and about 3% is excreted unchanged via the kidney. However, metabolism is the major reaction (Goessel and Bricker, 1990, Goldfrank *et al*., 2002).

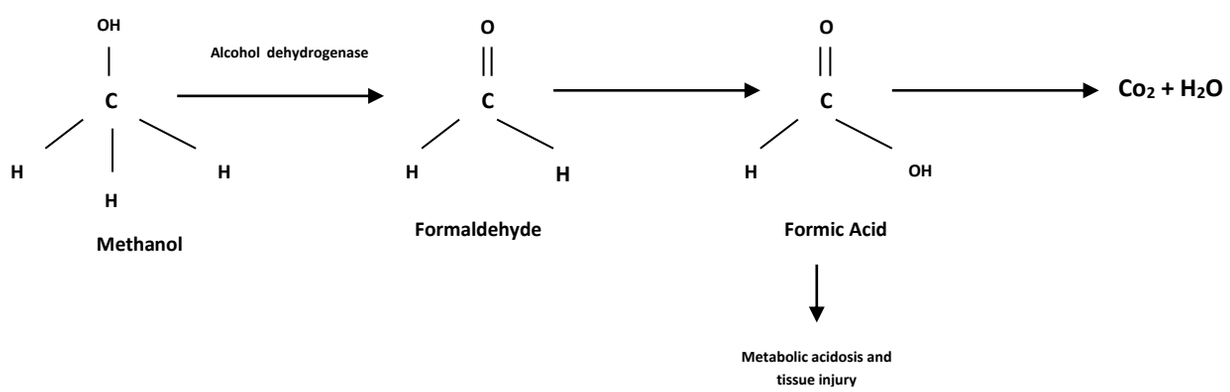

Fig. 1 Biochemical pathway for methanol metabolism (Brent *et al*., 2001).

1.2 Distribution of Methanol in Body Fluids and Tissue

Methanol, like ethanol, is distributed throughout the body in proportions equivalent to the amount of water in various tissues (Goessel and Bricker, 1990). Postmortem analysis of methanol concentrations in body tissues has reported fatal human cases of methanol poisoning, and has revealed high concentration of methanol in cerebrospinal fluid (CSF), vitreous humor and bile. Methanol concentration in these fluids has been higher than blood concentration. The highest concentration has been found in the tissues of brain, kidney, lung and spleen, with lower concentration in skeletal muscle, pancreas, liver and heart (Fishbein and FairfaxL, 1997).

1.3 Toxicity of Methanol

The major toxicological concerns following an acute methanol exposure are metabolic acidosis, visual disturbances, and permanent blindness. Central nervous system depression due to methanol causes mild inebriation. The severe metabolic sequelae and other toxic effects are caused by complications of acidosis (Goessel and Bricker, 1990). When methanol is oxidized to formaldehyde and formic acid, there is increased conversion of NAD^+ to NADH, the excess NADH favors reduction of pyruvate to lactate. Thus, acidosis associated with methanol poisoning is caused by formation and accumulation of both formic acid and lactic acid, and decreases in serum bicarbonate concentration. Consequently, there is an increased anion gap (difference between total cations and total anions). The normal anion gap is calculated as $[Na^{+}{}^{+}\ K^+]$-$[Cl^-$+ $HCO_3^-]$, the value of anion gap may be two or more times above normal following methanol intoxication (Goessel and Bricker, 1990). If visual symptoms develop, they usually develop within 24 hours after methanol exposure. The patient may complain of blurred or (snowfield) vision. This is the result of formic acid-mediated retinal toxicity (Goldfrank *et al*., 2002). Thus the target organ of methanol within the eye is the retina, specifically the optic disk and optic nerve. Optic disk edema and hyperemia are seen, along with morphological alterations in the optic nerve head and the intraorbital portion of the optic nerve. Both axons and glial cells exhibit altered morphologies. Accumulating evidences indicate that Muller cells, neuroglia that function in the maintenance of retinal structure and in intra-and intercellular transport, are early targets of methanol. Rods and cones, the photoreceptors of the retina, are also altered functionally and structurally. There are indications of mitochondria disruption in Muller and photoreceptor cells. This is consistent with the long-held view that cytochrome oxidase activity in mitochondria is inhibited, resulting in a reduction in ATP. This mechanism would explain, at least in part, the selective toxicity to photoreceptors and other highly metabolically active cells (Klaassen, 2001). Ocular symptoms appear above 100mg/dL, and fatalities in untreated patients occur in the range of 150-200mg/dL (Ellenhorn and Barceloux, 1988).

1.4. Methodology

This study has been carried out at the National Institute of Forensic Medicine (NIFM) at AL-Basheer Hospital, Ministry of Health. The study has been implemented through three main stages. Firstly, biological samples have been collected from medico-legal autopsies from January 2007 to January 2008. Secondly, these samples have been prepared for systemic toxicological analysis by (headspace-GC) with FID detector in the toxicology laboratory at the National Institute of Forensic Medicine (NIFM). Thirdly, a manual research of the autopsy records of all the cases has been carried out to evaluate the cases in terms of age, sex, time and manner of death, specific cause of death. Methanol analyses have been carried out by headspace gas chromatography (GC-2010 SHIMADZU) equipped with flame ionization detector and a meta wax column (30m X 0.25mm I.D, film thickness 0.25μm). Headspace injections have been performed using an automatic sampler cycle compose PAL1-(AOC-5000 auto-injector). The obtained results of methanol concentration in biological samples have been calculated in mg/dL.

1.4.1 Postmortem Cases and Specimens Sampling

Postmortem cases have been performed at the National Institute of Forensic Medicine (NIFM). All death certificates files for this period (from January 2007 to January 2008) have been manually searched. Then all autopsies, in which alcohol tests have been performed, are documented and considered in this study. Information included in autopsy reports has been classified in a set of variables with pre-defined values. The inclusion criterion defining the medico-legal autopsy data set used in this study is Senior citizen who died during the period January 2007 to January 2008 in the National Institute of Forensic Medicine (NIFM). This data set has been analyzed according to the following variables: age, sex, manner of death, specific cause of death, concentration of methanol analyses in all cases. All blood samples have been obtained in tubes with 1% sodium fluoride as preservative and transferred to the laboratory on the same day and stored at (4° C) until analyzed.

1.4.2 Determination of Methanol

A 200μL of specimen has been placed into a 10 mL headspace vial with 2mL of internal standard (1- propanol) the vials have been sealed with Teflon septa and magnetic seal caps. The specimens have been incubated at 70°C for 3min. prior to injection. Pre-incubation agitator speed has been set at 500 rpm. The syringe fill speed has been set to (100μL/s), and the injection speed is

(500μL/s). The prepared injection has been split at (170ºC) the column temperature has been set for (50ºC) to max temp (320ºC). The temperature of the detector has been set for 250ºC, Helium (He) has been used as carrier gas with a linear velocity of 20.4cm/sec Diazo dye [C.I. Acid Red 97(AR97), λ max: 498 nm] supplied by Techno Color Corporation (Bombay, India) was used as principal adsorbate. Chemical structure of dye is shown in Fig. 1.

2 Results and Discussion

The total number of medico-legal autopsies carried out from January 2007 to January 2008 was 1731; methanol has been detected in the blood of sixteen cases (0.92%). Fourteen of the cases were males (87.5%) and two of them were females (12.5%). The age distributions are present in **Table 1**. Their age varies from (14 to 56) years and most of them were between 30 and 49 years old, but there is one case with unknown age. Fifteen cases (93.75%) were found dead on the scene, one case (6.25 %) was admitted to the hospital in a coma state and died later. Blood methanol concentrations are represented in **Table 2**, the concentrations ranged widely. The lowest was 56mg/dL, and the highest was 400mg/dL, mean 220.7 mg/dL. In 12 cases (75%) methanol concentrations was over 100mg/dL. No ethanol or other drugs of abuse were detected. **Figure 2** shows the frequency distribution of sex and concentrations of methanol in blood samples in deaths. The intoxication due to different toxic materials constitutes an important part of legal medicine practice (Derya, 2006). In our study, various aspects of death among methanol poisonings in Jordan were evaluated and compared with the data in the literature on the subject. It is a well-known fact that the availability of methanol is associated with an increase of methanol poisonings (Derya, 2006). In our society, methanol and related products are obtained easily because they are cheap and sold everywhere. In our study, a comprehensive analysis of fatal methanol intoxication cases was conducted. Methanol fatalities constituted (0.92 %) of all medico-legal autopsies from January 2007 to January 2008. About(87.5 %) of the cases are males, while (12.5 % females, n=2). The existence of females in our study is represented in few cases; it shows that alcohol consumption is no more limited to males only. Studies suggest that women are less likely to be identified and diagnosed with harmful and hazardous drinking while other reports suggest that alcoholism has been the third leading cause of morbidity and mortality among women (Zaidan *et al*., 2007; Svikis and Reid-Quinones, 2003).

Table 1 Age Distribution of Methanol Victims

Age (years)	No. of cases	%
10-19	1	6.25
20-29	3	18.8
30-39	5	31.25
40-49	4	25.0
≥50	2	12.5
Unknown	1	6.25
Total	16	100.0

Table 2 Blood Methanol Concentrations

BAC (mg/dL)	No. of cases	%
10-49	0.0	0.0
50-99	4	25.0
100-149	2	12.5
150-299	5	31.25
≥300	5	31.25
Total	16	100.0

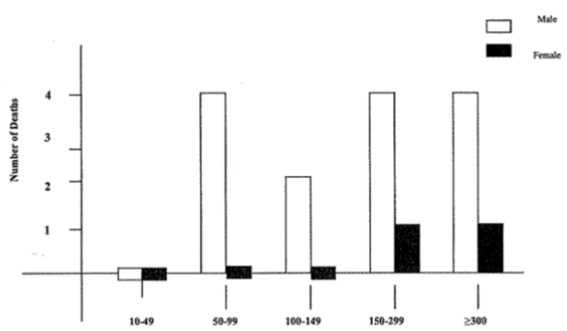

Fig. 2 Distribution of Sex and Blood Methanol Concentrations of Deaths Attributed to Methanol poisoning.

The highest number of cases occurred in this age group 30-39 years (5 cases, 31.25%) followed by the age group 40-49 years (4 cases, 25%). This result is similar to a previous study in Jordan (Abdallat *et al*., 2009). They have reported that the highest age group for methanol fatalities is (21-40) years, so this shows high prevalence of methanol consumption among middle-aged males. It is known that Jordanian society disapproves alcohol intake due to religious and social reasons. Therefore, their experience with alcohols is very limited and when they start drinking in their early twenties they are most likely unaware of the danger and adulteration of spirits, and

as a result of a cheap source of alcohol is quite a common cause of methanol fatalities especially to those who have low income (Abdallat *et al*., 2009). The case for methanol poisoning definition corresponds generally to the underlying cause of death definition used in death certification. The lethal dose of methanol in humans shows pronounced individual differences (Yayci *et al*., 2003). The lethal blood level in an untreated methanol poisoning has been generally estimated at 80mg/dL (Ellenhorn and Barceloux, 1988). In this study the methyl alcohol blood concentrations range widely from 56-400 mg/dL (n=16, 0.92%). Consumed methanol is almost exclusively metabolized by alcohol dehydrogenase. Yet in the case that blood alcohol concentrations exceed (20-50mg/dL), methanol can not be metabolized and accumulates (Derya, 2006). Patients with methyl alcohol concentration less than 20mg/dL are usually asymptomatic, but effects on the central nervous system from methanol poisoning appear when blood methanol levels are above 20mg/dL (Gideon, 2002). In our study, the methanol poisoning victims have methanol levels above 20mg/dL; the lowest concentration is 56mg/dL. Blood methanol levels more than 50mg/dL usually leads to acidosis. Victims with levels more than 100mg/dL have visual symptoms, and fatalities have been associated with levels more than 150mg/dL (Gideon, 2002). In our study, only four victims have been reported with methyl alcohol concentration above 50mg/dL from 50-99mg/dL, while 12 cases with more than 100 mg/dL. Fatalities have occurred in untreated patients with initial methanol levels range from 150-200mg/dL (Ellenhorn and Barceloux, 1988). Only one case with 150-200mg/dL has been reported in our study, but nine cases have been accounted for more than 200mg/dL from 201 to 400mg/dL. In our study, there is only one case died at the hospital (n=1, 6.25%). The case was brought to the hospital in a coma and died later, while other cases have been found dead or died on arrival to the hospital. Victims did not feel the symptoms while drinking or even after, this might be explained as being related to the presence of the latent period is responsible for not having medical treatment immediately. The people who had taken the alcoholic beverages containing methanol had an asymptomatic period approximately 12-24h, while the usual latent period is 12 to 18 hours (Derya, 2006). The major reason for this latent period in which the patient is asymptomatic is due to insufficient metabolism which produces signs of serious acidosis and toxic symptoms. They occurred because the affinity of ADH for ethanol is four times greater than its affinity for methanol (Goldfrank *et al*., 2002). So a person continues to consume alcoholic beverages, and when he/she realizes the intoxication symptoms, time then is too late for hospital admission (Derya, 2006). This can explain the fatality rate among methanol poisoning fatalities in different hospitals which comprises (22.5%), while the high percentage (77.5%) died before arriving to hospital in turkey, as revealed in a Turkish study reporting 8 years(Yayci *et al*., 2003). Another related study reported that deaths before admission to hospitals are 16.6% (Meyer *et al*,.2000). The highest ratio of death at home can be explained with the latent period of methanol, which is similar to the findings of (Yayci *et al*., 2003).

Conclusions

Fatalities due to methyl alcohol intoxication in our country have been proceeding on a certain level among postmortem samples in this material. The risk of methyl alcohol-related deaths is noticed higher among males than females. Most of methyl alcohol in the blood of the deaths is consistent with toxic and fatal ranges.

Nomenclature

ADH	=Alcohol Dehydrogenase	[-]
CSF	=Cerebrospinal Fluid	[-]
Λ	=Wavelength	[nm]
NAD	=Nicotinamide adenine	[-]
NADH	=Nicotinamide Adenine Dinucleotide	[-]
NIFM	=National Institute of Forensic Medicine	[-]

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