

EDITORIAL

**ACUTE POISONING WITH METHYL ALCOHOL: A CLINICAL STUDY IN
GEZIRA STATE, SUDAN**

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ABSTRACT

14 patients were admitted in March 2005 to Wad Medani Teaching Hospital in Wad Medani, the capital of Gezira State, Sudan, with features of an acute poisoning which are described in this paper. The history taken, directly from the patients or their relatives, revealed that all of them drank a substance known among them as spirit . They consumed it as an alternative to a locally-made alcoholic beverage known as Aragi This substance was obtained from small shops which utilize it for manufacturing locally-made perfumes. These small shops are widely distributed in different towns in Sudan. This substance proved to be methanol on analysis in the Forensic and Criminology Laboratory of the Ministry of Interior (personal communication).As this substance has less obvious smell compared to that of ethanol, people who consume it could mingle with others with no easy detection of alcohol consumption. They even called it the

Social drink for the above reason while having the same mood-elating effect.All of the patients developed blindness. Thirteen of them eventually died within 30 hours from consumption of the substance; only one survived but remained blind.

No new cases were reported due to the active intervention of the Ministry of Health, Police Department and energetic warnings by television, radio and newspapers.

Key word: Acute, Poisoning, Methyl alcohol, Wadmadani, Sudan

PATIENTS

14 patients were studied. Five of them were small traders, five were labourers while the rest had no regular jobs. All of them were in bad shape; presenting with

EDITORIAL

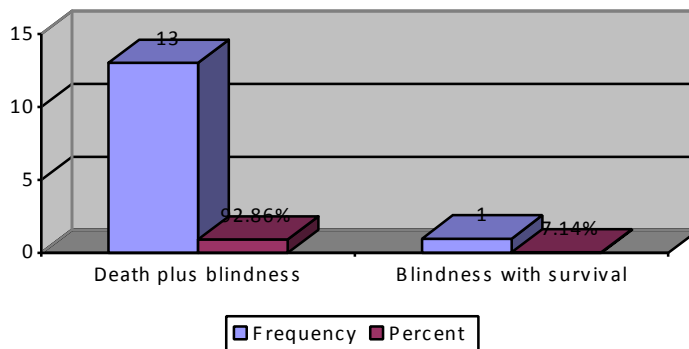
severe abdominal pain, vomiting and hypotension. In all patients acidotic breathing was profound. Blindness was universal. Dizziness and convulsions were seen in six patients. Hypotension was reported in all patients; 3 of them collapsed on admission. Deterioration of consciousness was reported in 4 patients. 93% of the patients complained of abdominal pain ranging from moderate to severe. Vomiting was a major complaint in 37% of the patients.

History taken showed that all 14 patients drank a substance known as spirit within the last 15-30 hours in amounts ranging from 150 ml to 225 ml diluted in soft drinks. Clinical features suggested an acute methyl alcohol poisoning. Symptoms and signs were compared with classical presentation in literature. Statistical analysis was done for relation between amount of alcohol consumed and the time lapsed before death occurred in these patients.

The age of the patients ranged between 20 and 36 years with a mean of 28.9 years. All of them were of low socio-economic status. 29% of them had no jobs. All patients were practically blind on arrival. This finding in addition to the marked acidotic breathing alerted the physician to the most likely diagnosis of acute methanol poisoning.

The outcome was most unfortunate: 13 out of 14 patients died; the only surviving patient remained blind (histogram).

Little help could be offered to these patients apart from intravenous infusions with dextrose/saline solutions. Intravenous ethanol infusion was not done because ethanol was not available and for legal reasons. As blood-gas analyzer was not available, no bicarbonate infusion was instituted nor haemodialysis undertaken because of the very late arrival of patients. Fomepizole treatment is not available in Sudan.



EDITORIAL

Histogram: The outcome

DISCUSSION

Methanol is a commonly used organic solvent, the ingestion of which has severe potential ramifications. It is found commonly in antifreeze, perfumes and solvents. It is used in the production of formaldehyde. Methanol production is done by steam reforming from natural gas. This source is favoured in industry to others such as coal or wood (biomass) ⁽¹⁾ .

Methanol is also known as or referred to as methyl alcohol, methyl hydrate, carbinol, wood alcohol or wood spirit ⁽²⁾ .

In Sudan, as in some other countries, methanol is a by-product of brewing of ethyl alcohol from sorghum, dates and various fruits. However, during this process, methanol has very little toxic effect because of the neutralizing effect of ethyl alcohol (ethanol). This is because ethanol is metabolized under the effect of alcohol dehydrogenase, the same enzyme needed for metabolism of methanol. This leads to methanol being eliminated without breaking down into its toxic metabolite (formic acid). Methanol is a clear, colourless liquid with a faint smell and tastes much like common alcohol (ethanol) ⁽²⁾ . It is expected to have a great potential as a source of hydrogen and fuel for vehicles with or without the addition of gasoline.

Methanol is used in hospitals as a detergent and in laboratories for fixation of slides. Traditionally methanol poisoning in Sudan used to occur in health institutions, mainly hospitals, where it was easily obtained and was mainly associated with blindness.

There are many reports of acute methanol poisoning from India ^(3, 4, 5, 6, and 7) .

A report from Kenya describes the result of deliberate addition of methanol to an illegally-made alcoholic beverage to enhance its effect resulting in many deaths ⁽⁸⁾. In March 1997, three people died in northern Ontario as a result of methanol poisoning ⁽²⁾ .

An outbreak of acute methyl intoxication with many fatalities occurred in Port Moresby, Papua New Guinea in March 1977 ⁽⁹⁾ .

Alcohol distilling, distribution and selling for the purpose of drinking are prohibited by law in accordance with Islam religion in Sudan ⁽¹⁰⁾ . However, it is licensed for various legitimate purposes.

EDITORIAL

Subjects in this report were not aware of the exact nature of the substance consumed by them. All of them were of low socio-economic status with a young age group distribution. Somehow these subjects discovered that this substance they consumed in small amounts mixed with soft drinks has the same effect of Aragi, the native illegally-brewed alcoholic beverage. They explained their preference for this substance to Aragi because it was cheaper and had a better undetectable smell. They even called it the social because they could mingle with other people without the detection of alcohol consumption by them; thus avoiding the legal and social consequences.

The problems of illegal brewery of alcohol is encountered in most countries where Alcohol-Prohibition Acts have been implemented with a consequent rise in alcohol-associated criminal activity and death from methanol poisoning⁽¹¹⁾.

The addition of methanol to ethanol (ethyl alcohol) constitutes a lesser hazard of toxicity than drinking methanol alone⁽¹²⁾. This fact might explain the severe toxicity encountered in the group described in this article as no ethanol was taken with the drink consumed. Methanol certainly qualifies as an exceptionally toxic substance even at extremely low doses^(12, 13, and 14).

A minimum single dose needed for methanol to cause death (without medical treatment) is considered to range from 300 to 1000 mg/kg body wt⁽¹⁴⁾. Some authors suggested even the lesser dose of 10 ml to cause blindness and 30 ml to be fatal⁽¹⁶⁾. The average amount of methanol consumed by this group of patients was far higher than the minimum dose described in literature. This fact explains the very high morbidity and mortality among this group.

The clinical findings in this group of patients were consistent with those described in literature: features included nausea, vomiting and abdominal pain which may be related to acute pancreatitis⁽¹⁶⁾.

Acidosis, detected clinically, was profound. We could not measure the exact degree because no blood-gas analysis machine was available. Acidosis has a significant detrimental effect on the myocardium causing myocyte degeneration and cardiac dilation⁽¹⁷⁾. In this group all patients presented with marked hypotension. Two patients had non-specific ECG changes presenting with cardiocirculatory collapse. Cardiopulmonary

EDITORIAL

toxicity is related to the degree of acidosis produced by the accumulation of formic acid, the main product of methanol biotransformation ⁽¹⁸⁾ .

All 14 patients developed blindness. Many of them showed CNS manifestations presenting with convulsions, Parkinsonian features and deterioration of consciousness degrading into deep coma. These changes are usually due to diffuse brain swelling and haemorrhagic lesions to the basal ganglia ⁽¹⁹⁾ . Death is frequently the consequence of a multiple organ failure. Symptoms of methanol toxicity are delayed for 12-24 hours after consumption leading to late recognition and delayed therapy, unless there is a high index of suspicion. This latency is due to the initial degradation of methanol to formaldehyde via the enzyme alcohol dehydrogenase (ADH) . This reaction is slower than the transformation of formaldehyde to formic acid, the main substance responsible for methanol toxicity ⁽²⁰⁾ .

The inhibition of alcohol dehydrogenase is fundamental to the treatment of methanol poisoning. This is the basis for institution of ethanol infusion to saturate alcohol dehydrogenase and to prevent the formation of formic acid. Haemodialysis is used to increase elimination of methanol and thus prevent accumulation of formic acid. Haemodialysis also normalizes acid-base disorders ⁽²¹⁾ . However, haemodialysis is only useful to eliminate methanol itself and it may be unnecessary in patients without acidosis. This is particularly so in those patients who are undergoing Fomepizole treatment. Nevertheless, haemodialysis may be warranted to prevent prolonged hospitalization of patients surviving the initial toxicity who are having very high plasma methanol levels. Fomepizole acts by inhibiting alcohol dehydrogenase and is being suggested as a safe and well effective treatment for acute methanol poisoning ⁽²²⁾ . It is not used concurrently with ethyl alcohol infusion as the latter treatment needs alcohol dehydrogenase to speed up breaking down of methanol. Fomepizole is considered advantageous over ethanol infusion as it does not cause changes in mental status, hepatotoxicity nor hypoglycaemia.

Gastric lavage is only useful if instituted early within the first 24 hours after ingestion of methanol.

EDITORIAL

Administration of high doses of bicarbonate is used to combat acidosis under strict control by blood gas analysis, when the pH is 7.3 or less. Vitamin supplements are usually given in high doses in hope of reducing the incidence of blindness, however, with little success.

In this group of patients the outcome was very bad: 13 out of 14 died. Many factors led to this sad result.

First, all patients presented to hospital very late. Second, the medical personnel receiving these patients had no experience in dealing with acute methanol poisoning. Indeed it was the consultant physician on call who was able to suggest acute methanol poisoning from the clinical picture. Thereafter information regarding the substance consumed could be obtained from the patients and their relatives. Gastric lavage was considered unuseful as too long time had already lapsed since the consumption of methanol. In addition to that, pure ethyl alcohol for infusion was not available with still legal and religious considerations to think of. Also, the pharmacokinetic characteristics of ethanol are erratic, making it difficult to maintain adequate plasma concentrations even in larger and better equipped hospitals^(23, 24). Accordingly there is a need for repeated measurements of plasma level of ethanol to ensure appropriate dose adjustments. Thirdly, no blood-gas analyzer was available to check for parameters of acid-base balance and to guide for bicarbonate infusion.

Lastly, the medical personnel had very little time to arrange for haemodialysis before the patients died. As a result only dextrose/normal saline and vitamin infusions could be offered to those patients.

This episode of acute methanol poisoning had a vast coverage by the media and a quick response by the Police Departments. In effect, no new cases appeared in response to the warnings made by T.V., radio and newspapers.

Lessons from the report:

From the tragic outcome of this episode of acute methanol poisoning some conclusions may be drawn:

1. There is a need to explain to the public the seriousness of methanol poisoning and the sources of methanol itself especially in illegal alcoholic beverages and household utility products.

EDITORIAL

2. The need to raise the awareness among medical personnel of the possibility of serious poisoning, including methanol poisoning, can not be overstressed.
3. Shortcomings of hospital equipment were made obvious by this episode. For example the blood-gas analyzer could have made some difference in management of these patients. In fact blood-gas analysis is essential for proper management of many other conditions presenting with acid-base disturbances such as diabetic keto-acidosis and renal failure, to mention some.
4. A clear-cut legislation concerning the handling of ethyl alcohol in hospitals when the need arises for ethyl alcohol infusion must be considered.

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EDITORIAL

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EDITORIAL

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