

A Case Series of Acute Methanol Poisoning from Northern Kerala

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Abstract

Background: Methanol is a cheap, potent adulterant widely used in illicit liquor. There have been several episodes of mass methanol poisoning in our country with varying mortality and morbidity rates. Serious attempts to study the profile and common variables of such patients have not been attempted from Kerala.

Aim: To study the clinical profile and prognostic factors in patients who were victims of the methanol poisoning admitted in a teaching hospital.

Methodology: Twenty four cases of methanol poisoning from adulterated toddy admitted to Calicut Medical College Hospital in September 2010 during a single outbreak of a hooch tragedy were studied.

Results: Out of total 24 admissions, 4 died and 4 developed permanent blindness. Of the 4 patients who died, 3 were brought in a critical condition, and succumbed to the toxic effects of methanol even before starting any form of treatment and the fourth died before receiving hemodialysis. Four patients with initial complete visual loss did not regain vision even after correction of acidosis and hemodialysis. Fourteen patients who had blurring of vision and other symptoms completely improved after intensive alkalisation therapy and hemodialysis. The latent period between the alleged consumption of adulterated toddy and development of symptoms was variable. Minimum fatal period was 10 hours and maximum was 2 days. All patients who were subjected to haemodialysis had short period of hospital stay.

Conclusion: This observational study shows that all those patients who received prompt and early alkalisation therapy, 10% ethanol infusion, folic acid injections and hemodialysis had striking recovery, except those who had initial complete loss of vision. The blood pH value was found to have direct correlation with visual outcome. Poor nutritional status indicated by a low BMI was suspected to have a role in increased susceptibility to toxic effects of methanol, possibly suggesting coexisting folic acid and other multiple vitamin deficiencies in such patients.

Keywords: Methanol poisoning; Metabolic acidosis; Optic neuritis

Background

Methanol, also known as wood alcohol, is commonly used as an organic solvent, the ingestion of which has severe potential toxicities. It is a constituent in many commercially available industrial solvents and in adulterated alcoholic beverages.

Toxicity usually occurs from intentional overdose or accidental ingestion and results in metabolic acidosis, neurologic sequelae, and even death. Methanol poisoning remains a common problem in many parts of the developing world, especially among members of lower socioeconomic class.

The aim of the study was to identify the clinical profile and prognostic factors among patients who were admitted with acute methanol poisoning during a single outbreak of a hooch tragedy, occurred in September 2010.

Subjects and Methods

This was an observational study done in 24 patients, admitted to Calicut Medical College, with clinical features of acute methanol intoxication. Detailed history was taken from all the patients except three who were critically ill at admission and expired soon. Special emphasis was given regarding details of the amount of alcohol intake, interval between consumption and onset of symptoms, other co morbid illnesses, diet history and their usual drinking pattern. All the cases were subjected to detailed physical examination and ophthalmological evaluation. A standard treatment protocol was followed in all the patients to start with. This was later individualized.

Results

Clinical profile

All patients in the study were males, with age ranging from 23 to 80

years, with mean age of 47 yrs. All of them belonged to low socioeconomic class. The amount of adulterated toddy consumed varied from 500 ml (4 patients) to 3000 ml (1 patient). Most of the patients had consumed between 1000-2000 ml of adulterated toddy. Amount of adulterated toddy consumed did not correlate with the symptomatology, probably because the amount of methanol in various samples (consumed from various point sources) varied which could not be assessed from history. Onset of symptoms varied from 1hr to 48 hr but most of the cases reported symptoms between 6-48 hrs. Nature of symptom was abdominal pain and blurring of vision in 3, vomiting and blurring of vision in 6, blurring of vision alone in 10, vomiting alone in 2 and other non-specific symptoms in 3. Fifteen patients were drowsy and had acidotic breathing on arrival. Absent light reflex was seen in those who presented with blindness. Others had a dilated reacting pupil. Eighteen of the patients used to consume alcohol daily and rest used to consume alcohol regularly but less often (2-3 times a week). Thirteen of them were regular toddy consumers and rest used to take various types of alcohol drinks. Fifteen patients had metabolic acidosis (<7.35). Fifteen patients underwent hemodialysis, two of them twice and one had three sittings of dialysis. Of the 24 admitted in this hospital 4 expired,

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4 developed permanent blindness and the rest 16 had an uneventful recovery and were symptom free at 1 month follow-up. Blood samples were taken for hemogram, arterial blood gas, plasma bicarbonate levels, serum electrolytes, blood urea, serum creatinine, and liver function test. Ideally serum levels of methanol and ethanol need to be assessed, but due to non-availability of methanol assay techniques, this was not done.

Management

All the 20 patients, who had clinical and laboratory findings of acidosis were given I.V. infusion of 7.5% sodium bicarbonate 100 ml fourth hourly [1-3]. 5% ethanol was given through Ryle's tube. Initially a 15 ml/kg bolus was given followed by 2-3 ml/kg/hr as maintenance infusion [4]. The amount of sodium bicarbonate and ethanol administered were guided by the clinical condition of the patient, acidosis and plasma bicarbonate levels. Ryle's tube administration of ethanol (10%) and IV sodium bicarbonate were continued till the patients recovered completely [3,4]. All were given a loading doses of folic acid 1 mg/kg (max 50 mg/dose) 6 hourly for 6 doses titrating with the patients response and ABG values. Later on it was changed to oral folic acid 15 mg 6hourly [5]. To those patients with visual symptoms, high dose of corticosteroid was given as Methylprednisolone 1 gram intravenous pulse for three days, followed by 1 mg/kg for 10 days [6,7]. Dextrose infusion supplemented with thiamin, vitamin B6 and vitamin B12 was also given [1]. All those with metabolic acidosis or visual loss and those who had blurring of vision not improving with initial therapy were taken up for hemodialysis [1-4,8]. Hemodialysis was repeated till pH was >7.35. Fomepizole [1,2,4,9], due to non-availability, could not be given. Of the 24 patients admitted, four expired; four patients who reported initially with vision loss continued to be blind in spite of vigorous treatment and the rest who presented with various manifestations like blurring of vision, vomiting, headache, drowsiness, abdominal pain etc. recovered fully on early institution of treatment (Figure 1).

All patients belonged to low socioeconomic group and 92% had poor educational status (illiterate or below matriculate). The amount of adulterated toddy consumed varied from 200 ml to 2500 ml. But there was no correlation between the amount consumed and symptoms. This may be due to the varying amounts of methanol and ethanol in various samples. Analysis of samples collected by the Excise Department from the various point sources and even different barrels from the same shop showed wide variations in the concentration of methanol. Time of onset of symptoms varied from 1hr to 48hrs which could again be explained by the above finding. BMI of all patients who survived was calculated (Table 1).

Three of the 4 patients who lost vision, had a BMI of <18.5. The fourth patient had a BMI of 25.8. The 3 patients who lost vision were aged 80, 65 and 65 yrs. The 3 patients who had a BMI <18.5 but normal visual outcome were aged 40, 23 and 46 yrs. Thus it was observed that a low BMI in an elderly patient was associated with poor visual outcome, after methanol intoxication.

Nineteen patients had metabolic acidosis. The blood pH of patients who expired was 6.89, 6.9, 6.92 and 7.12. The pH of patients who had loss of vision was 7.2, 7.27, 7.28 and 7.32. One person with a pH of 6.9 had a normal visual outcome. He had consumed rum along with toddy. In Figure 2, 15 patients were taken up for hemodialysis. Three of the 4 patients who expired had arrived in the casualty very sick and had died before institution of any specific therapy. The fourth was put on mechanical ventilator from the casualty and could not be taken up for hemodialysis. Of the 20 patients who survived, 15 had metabolic acidosis and were taken up for hemodialysis. Two of them received

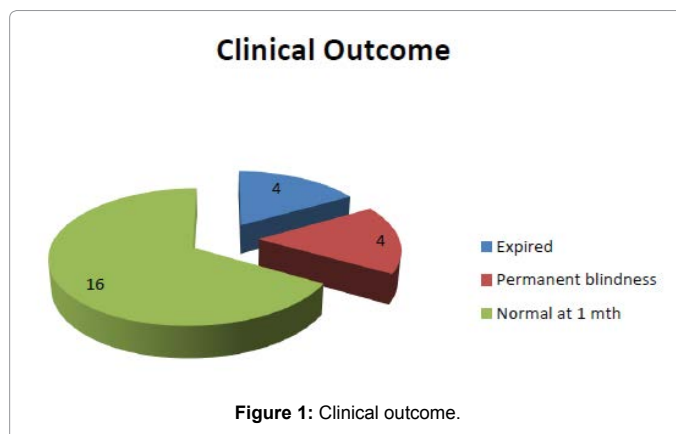


Figure 1: Clinical outcome.

BMI	Number
<18.5	6
18.6-23.5	12
>23.5	2

Table 1: Profile of BMI.

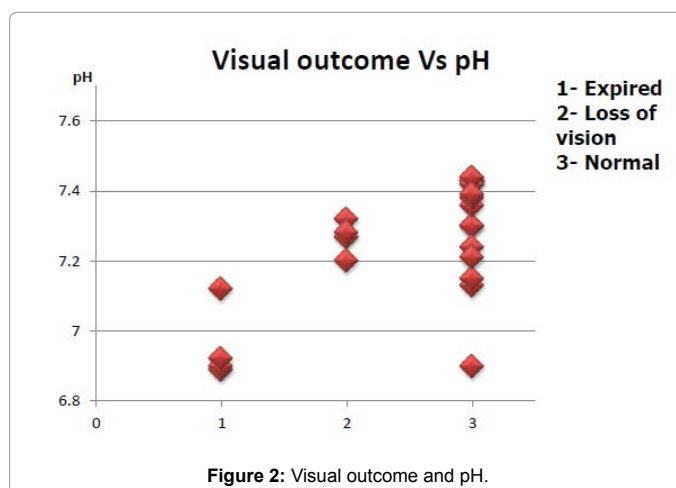


Figure 2: Visual outcome and pH.

dialysis twice and one of them was dialyzed thrice.

Discussion

Methanol poisoning is fairly common in our country. Being cheap and potent, it is the first adulterant of illicit liquors. Methanol poisoning is an acute medical emergency. If not recognised in time and treated properly, it can lead to considerable magnitude of morbidity as well as mortality. Our present study does not truly represent the mortality of this outbreak since only 4 of the total 26 deaths attributed to methanol poisoning occurred in our institution. The potentially lethal dose of methanol is variable [8,10-12]. The lowest reported is 30 ml [13]. The peculiarity of methanol poisoning is the latent period between the ingestion of the alcohol and the appearance of manifestations, which, in our study varied from 1 to 48 hrs. The latency may be related to the concomitant ingestion of ethanol which affects the metabolism of methanol and the nutritional status of the patient.

The symptoms of methanol poisoning are non-specific except for the visual disturbances. Ocular changes consist of retinal oedema, blurring of the disc margins and hyperaemia of the discs [6]. Optic atrophy is a late sequelae of poisoning. The terminal event is often respiratory arrest. The fatal period varies from 6-36 hours. Metabolic acidosis is the most striking

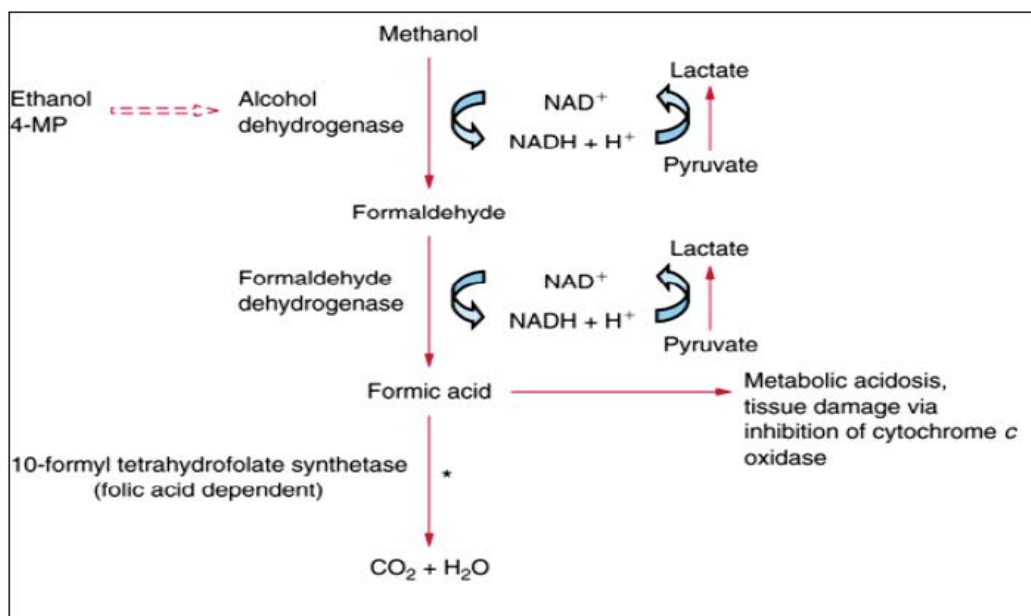


Figure 3: Pathway elucidating methanol degradation [1].

disturbance seen in methanol poisoning [1-4,10,12-14]. It is due to the accumulation of formic acid and lactic acid. The ocular changes correlated with the degree of acidosis. The accumulation of formic acid in the eye has been implicated in the ocular toxicity of methanol ingestion. Optic nerve cells possess few mitochondria and low levels of cytochrome *c* oxidase, so the optic nerve is extremely susceptible to the toxic effects of formic acid [1]. Retinal damage is also believed to be due to the inhibition of retinal hexokinase by formaldehyde, an intermediate metabolite of methanol. With correction of the acidosis, vision improves. With resolution of the acidosis there is an increase in the dissociated form of formic acid, which is unable to readily diffuse into the CNS. The accumulation of formic acid in the eye is therefore decreased in this setting (Figure 3). This pathway elucidates the role of different treatment modalities used in management of acute methanol intoxication. Ethanol and 4- methylpyrazole (Fomepizole) [1,2,4,9] competitively inhibits alcohol dehydrogenase enzyme and blocks conversion of methanol to formaldehyde (33 times more toxic than methanol).

Folic acid dependant 10 formyl THF synthetase enzyme, converts formic acid to CO₂ and water (rate limiting step) by which methanol is eliminated. This is the basis of supplementation of high dose folic acid in acute methanol intoxication [1,3-5].

Conclusions

Blood pH values were found to have direct correlation with adverse outcome (p value - 0.002). Mortality and permanent visual loss was higher in the elderly (p value - 0.026). The most important prognostic indicator of visual outcome was the visual status at presentation. Those who presented with blindness remained so even after intensive therapy. Nutritional status assessed by BMI was <18.5 in 3 out of 4 patients who lost vision. Poor nutritional status appears to be poor prognostic indicator, possibly suggesting coexisting folic acid and other multivitamin deficiency. Prompt and early alkalinisation, 10% ethanol and folic acid therapy along with hemodialysis gives striking recovery of vision, except for those who had initial complete loss of vision. Early institution of high dose steroids is also found to be beneficial in

minimizing the ocular morbidity.

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