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**Case Series**

# Our experience with methyl alcohol poisoning at SLBSGMCH Mandi at Nerchowk

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## ABSTRACT

Methyl alcohol poisoning is an extremely hazardous poisoning commonly occurs via oral ingestion of illicit or adulterated liquors or as ethanol substitution. Toxicity results in gastrointestinal manifestations, metabolic acidosis, neurologic squeal and even death. Rapid recognition, early and aggressive management have been emphasized to prevent hazardous outcome. We hereby present a case series of 16 cases who presented to our hospital as methyl alcohol poisoning. Demographic, clinical and biochemical data were collected retrospectively from the hospital record section of the tertiary care hospital in the state of Himachal Pradesh. Out of 16 patients admitted, 12 patients had metabolic acidosis and treated with sodium bicarbonate. Four patients underwent hemodialysis for the improvement of symptoms. Five patients with severe metabolic acidosis, hypotension and altered sensorium did not be recover and succumbed.

**Keywords:** Methyl alcohol, Formic acid, Metabolic acidosis

## INTRODUCTION

Acute methanol poisoning is global crisis. Many such out breaks have been reported in the past not only from India but also from all over the world. These out breaks mainly affect poor and vulnerable strata of the society.[1-3]

Wood alcohol (Methyl alcohol) is a colorless, odorless, highly toxic organic solvent. which is a component in many industrial solvents, photocopying fluids, washing fluids, paints removers, antifreeze formulations and perfumes.[4] Since methanol is available and cheap, it is used in the production of illegal alcoholic beverages in many developing countries causing high morbidity and mortality in many methanol poisoned patients.[5] Methanol does not cause toxicity by itself and its pathological effects are caused by the accumulation of its metabolites formic acid which affects central nervous system, gastrointestinal tract and eyes causing metabolic acidosis and cellular dysfunction.[6]

Depending on the presence or absence of food, peak absorption occurs within 30-60 minutes. Similar to other organic solvents, methanol is relatively well absorbed through the skin and also by inhalation. It is estimated that the pulmonary absorption fraction is 65-75%. Moreover, methanol is water soluble and has an apparent volume of distribution of 0.6-0.7 L/kg.[7]

## CASE SERIES

A total of 16 patients of methanol poisoning, all male (100%) patients, were admitted in tertiary care hospital of SLBSGMC Mandi at Nerchowk. The demographic, clinical, and biochemical data were collected retrospectively from the hospital record section. The data was extracted retrospectively from patient charts and hospital records. A detailed history was obtained from the patients, relatives and prehospital personnel. Detailed clinical examination including vital signs, mental status and ophthalmological screening was done. After

immediate primary assessment airway, breathing and circulation were taken care of in all the patients. Intubation was done in indicated patients. Hypotension was treated with intravenous crystalloid fluids.

Routine blood investigations like complete blood count, RFTs, electrolytes, complete urine examination and Arterial blood gases were done for all the patients. In patients with pH <7.3, intravenous sodium bicarbonate was given as per calculated dose. Hemodialysis was done for patients who did not improve with bicarbonate therapy.

All of them were between 34 and 56 years of age with mean age of 42.87 years. Most of the patients were brought to the hospital after 12 hours of methanol intake (Table 1). Gastrointestinal symptoms like nausea, vomiting, and abdominal pain were observed in all cases followed by neurological symptoms in the form of altered sensorium in 5 patients (31%) (Table 2). Out of 5 altered sensorium patients, 2 patients had Glassgo coma scale (GCS) less than 5 score and they died immediately after admission. Visual symptoms like blurring of vision were seen in 2 (12.5%) patients.

**Table 1: Patient characteristics, (n=16).**

| Variable | Patients |
|---|---|
| **Age (Year)-mean** | 42.87 (34-56) |
| **Sex** | Male (100%) |
| **Route of poisoning** | Ingestion (100%) |
| **Mode of poisoning** | Intentional (100%) |
| **Habitual alcohol consumption** | 7 (44%) |
| **Delay time, hours (lag period)** | 12 to 48 |
| **Type of poisoning** | Acute poisoning (100%) |
| **Profession** | Laborer |
| **Source of alcohol** | Country made liquor shop |
| **Consumed unknowingly?** | Yes |
| **Quantity** | Not known |

**Table 2: Clinical profile, (n=16).**

| Clinical profile | Patients, n (%) |
|---|---|
| **Gastrointestinal symptoms** | 16 (100) |
| **Altered sensorium** | 5 (31) |
| **Breathlessness** | 3 (19) |
| **Visual symptoms** | 2 (12.5) |

The mean values of arterial pH and bicarbonate levels were 6.97±0.85 and 12.3±7.1 mmol/l, respectively. The mean value of hematocrit was 42.5±2.8 (Table 3). 12 patients (75%) of the patients received intensive alkali therapy and 4 patients (25%) were dialyzed once. Acute kidney injury (37.5%), sepsis (43.7%) and shock (43.7%) were common complications in patients with acute methanol poisoning during hospital stay. Five patients (31%) succumbed to death due to multiple complications (Table 4).

**Table 3: Biochemical profiles.**

| Biochemical variable | Mean ± SD |
|---|---|
| **Arterial pH** | 6.97±0.85(6.34-7.4) |
| **Bicarbonate (mmol/L)** | 12.3±7.1 |
| **Anion gap** | 24±4.2 |
| **Lactate (mmol)** | 2.0±0.8 |
| **Base deficit (mmol/L)** | 15.2±3.8 |
| **PCV (%)** | 42.5±2.8 |

**Table 4: Complications.**

| Complication | N (%) |
|---|---|
| **Acute kidney injury** | 6 (37.5) |
| **Sepsis** | 7 (43.7) |
| **Hypotension** | 7 (43.7) |
| **Outcome (death)** | 5 (31.2) |

## DISCUSSION

Methanol poisoning is one of the common poisoning outbreaks in India, affecting mainly lower and uneducated strata of society. It is a cheap substitute used as an adulterant in the locally sourced liquors. Methanol poisoning in many places still have a poor outcome because of the late arrival and diagnosis of the patients in addition to the unavailability of blood methanol level in many hospitals and the unavailability of antidote.[8]

In our study the mean age of presentation of patients with methyl alcohol poisoning was 42.8 years and all were male. Male are commonly affected, which is consistent with the local culture, where females are less frequently consuming alcohol. This also signified that such outbreaks are affecting the earning members of society. The age of presentation is comparable to a study done by Jarwani et al from Gujarat.[9]

Symptoms usually appear within 12-24 hours after ingestion, but can be significantly delayed if ethanol is ingested simultaneously. In our study most of the patients were brought to the hospital between 12-48 hours of methanol intake. Clinical features are nonspecific, gastrointestinal symptoms like pain abdomen, nausea, and vomiting are the most common symptoms followed by dyspnoea, chest pain and hyperventilation dominating along with visual disturbances.[10] In our study all patients had some form of gastrointestinal symptoms (100%) followed by neurological symptoms (31%). The degree of the visual disturbance varies from blurred vision, decreased visual acuity, photophobia, and the “feeling of being in a snow field” to complete blindness. In our study, visual symptoms were seen in 2 (12.5%) patients of methyl poisoning. Previous studies have also high lighted similar observations.

A study by Kumar et al from Uttarakhand, visual symptoms were seen in 12 (12.9%) patients, of which 9 (9.6%) patients had complaints of blurred vision, while 3 (3.2%) patients suffered from visual loss. In our study, acute kidney injury observed in 6 patients (37.5%). The incidence of acute kidney injury is variable among different studies. Chang et al study on 50 methanol patients showed acute kidney injury in 33 patients (66%) and concluded as a useful predictor of hospital mortality. In a study by Verhelst et al acute renal injury was found in 15 of 25 (60%) patients. However, nephrotoxic effect of methanol is still uncertain.

***Diagnosis of methanol poisoning***

Specific laboratory analyses are most often not available where the poisonings occur. Biochemically, typical findings during methanol intoxication are metabolic acidosis, increased anion gap (AG) and osmolal gap (OG) (where testing available), increased serum (S)-methanol, and increased S-formate (as determined with a new and simple diagnostic approach with limited, yet increasing availability) (Hovda et al).[11] The measurement of arterial blood gases (ABGs) is thus the most commonly available laboratory analysis that helps to diagnose methanol poisoning. Metabolic acidosis is the most striking disturbance seen in methanol poisoning. It is probably due to the accumulation of formic acid and lactic acid. In our study, metabolic acidosis was present in more than 80% of cases of methanol poisoning. All the 5 patients succumbed to death had severe metabolic acidosis at the time of admission.

Criteria of diagnosing of methanol toxicity:[12] Documented plasma methanol concentration > 20 mg/dl (>200 mg/L), documented recent history of ingesting toxic amounts of methanol and osmolal gap >10 mOsm/kg, history or strong clinical suspicion of methanol poisoning with at least two of the following criteria: Severe metabolic acidosis i.e., Arterial pH<7.3, serum bicarbonate <20 meq/L (mmol/L), osmolal gap >10 mOsm/ and any of the three.

***Treatment of methanol poisoning***

Unlike many other poisonings, methanol poisoning needs both specific therapeutic modalities and general supportive treatment. The first treatment is to secure and maintain airway, breathing and circulation. The treatment of methanol poisoning consists of a buffer to correct metabolic acidosis, an antidote (ethanol or fomepizole) to block alcohol dehydrogenase from producing toxic formic acid, and hemodialysis (HD) to remove methanol and its toxic metabolites and to correct the metabolic acidosis. Finally, the ingestion of folinic acid is recommended to increase the endogenous formate metabolism. Bicarbonate treatment also decreases the amount of non-dissociated formic acid, which easily accesses the central nervous system (CNS) and thereby causes toxicity.

The recommended therapeutic blood S-ethanol level is approximately 22 mmol/L (100 mg/dL).[13] The recommended dose of fomepizole (4-methyl-pyrazole) is 15 mg/kg as a loading dose, followed by 10 mg/kg every 12 hours (Hovda et al). The antidote treatment can be discontinued when the methanol level decreases to below 6 mmol/L (20 mg/dL) if there is no acidosis or visual disturbances.

Methanol is easily removed by dialysis because of its low molecular weight (MW) (32 g/mol), lack of protein binding, and low volume of distribution (0.6-0.7 L/kg) by Hovda et al.[14] Similarly, formate has a low MW 46 g/mol, lack of protein binding, and low volume of distribution (approximately 0.5 L/kg) (Hovda et al).[11]

All patients were treated in accordance with the guidelines of the American academy of clinical toxicology and the European association of poison centers and clinical toxicologists. The treatment was guided by blood gas analysis and the clinical picture. All patients accordingly treated with intravenous fluids, thiamin, folinic acid, Sodium bicarbonate infusion, hemodialysis (HD). No patient received oral ethanol therapy. Fomepizole and intravenous Ethanol was not given to patients as antidote because of unavailability in local market.

## CONCLUSION

Methanol outbreaks represent a significant challenge most often occurring in the developing world, frequently affecting poor and vulnerable populations. Despite effective management, morbidity and mortality rates are high in methanol poisoning, the treatment of which requires a multidisciplinary approach. High case fatality rates for methanol poisonings in these contexts are likely secondary to severe metabolic acidosis, multiple other complications, diagnostic limitations and treatment constraints and late presentation to hospital. Rapid recognition, early and aggressive management has been emphasized to prevent the hazardous outcome.

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