CLINICAL AND METABOLIC FEATURES OF ETHANOL-METHANOL POISONING IN CHRONIC ALCOHOLICS

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Summary The kinetics of methanol were examined in 84 chronic alcoholics admitted after drinking a cleansing solution containing 90% ethanol and 5% methanol. On admission, the average blood methanol concentration was 20 mmol/l (64 mg/dl) and blood ethanol concentration was 39 mmol/l (179 mg/dl). Although these patients were not treated with ethanol, and methanol concentrations remained high as blood ethanol concentrations fell to zero, no acidosis or other signs of classic methanol poisoning developed. The rate of metabolism of methanol was correlated to the initial ethanol concentration. To avoid unnecessary invasive therapy, treatment of methanol poisoning should be based on the case history, clinical signs, and laboratory features—not solely on blood methanol concentrations.

Introduction

METHANOL poisoning can produce a life-threatening condition characterised by formate and lactate acidosis.[1] The patient often presents with nausea and vomiting and epigastric pain, and may be confused and ataxic. As the acidosis develops, visual symptoms progress and coma deepens. Early diagnosis and treatment—with ethanol, large quantities of buffer base, and dialysis—are essential for a successful outcome.[2]

In Sweden, before 1978, methanol poisoning was not uncommon among alcoholics, who supplemented their regular intake with cleansing solutions containing up to 80% methanol. In 1978, the methanol concentration in these solutions was limited by statute to 5%, mixed with at least 90% ethanol. These solutions are widely consumed by alcoholics, often in drinking bouts lasting one to two weeks, by the end of which both methanol and ethanol concentrations in the blood may be high. As the blood ethanol is metabolised, the blood methanol concentration remains high—up to 50 mmol/l (160 mg/dl). Patients of this sort—chronic alcoholics with high initial ethanol concentrations—may have no signs of classic methanol poisoning on admission: although not treated with ethanol, they do not develop signs of a formate-induced metabolic acidosis.[3,4] The usual approach to the diagnosis and treatment of methanol poisoning is based on the blood methanol concentration alone,[1] which in this group of patients would have led to unnecessarily aggressive and expensive therapy. We have studied the kinetics of methanol in these patients, in relation to the development of the classic signs of methanol poisoning.

Subjects and Methods

This study included 84 chronic alcoholic patients who were admitted to Lillhagens Psychiatric Hospital, and who had both methanol and ethanol in their blood, due to cleansing solution abuse. A conventional psychiatric history was taken and a physical examination was performed. Venous blood gases were analysed and heart rate, blood pressure, respiratory rate, coma grade, and abdominal status were assessed every 4 h. Patients with a base excess (BE) of less than −6 mmol/l were referred to a medical emergency department. Blood methanol and ethanol concentrations were measured every 12 h until the blood methanol concentration was below 15 mmol/l (48 mg/dl).

The elimination rate of methanol is expressed as the average decrease in methanol concentration in mmol/hour. Since elimination follows zero order kinetics, values were calculated in all patients with two or more concentrations.

Correlation analysis between the different variables was performed with a non-parametric method (Pitman's test). A p level of less than 0·05 was considered significant. Linear regression was used to plot the curves of ethanol and methanol concentrations.

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Variable	n	Range	Mean	SD
Age (yr)	84	26–69	44·72	8·22
Methanol conc on admission (mmol/l)	81	12–64	22·78	8·47
Ethanol conc on admission (mmol/l)	80	1–125	50·59	24·36
Minimum base excess (BE) (mmol/l)	72	−13–+6	−0·15	3·33
Methanol conc at minimum BE (mmol/l)	82	1–60	20·13	8·67
Ethanol conc at minimum BE (mmol/l)	82	1–101	39·37	28·6
Methanol:ethanol ratio at minimum BE	82	0·13–27	3·47	6·45
Admission respiratory rate (breaths/min)	77	8–28	16·19	3·86
Respiratory rate at lowest BE (breaths/min)	75	8–30	16·51	3·7
Heart rate on admission (beats/min)	78	70–150	99·41	16·19
Systolic BP on admission (mm Hg)	82	110–195	142·44	16·9
Diastolic BP on admission (mm Hg)	82	70–110	90·61	10·28
Elimination rate of methanol (mmol/l/h)	78	0·169–3·2	0·98	0·54

n = no of patients available for analysis.

Results

84 patients (82 male and 2 female), average age 45 years, were entered into the study. The mean, range, and standard deviation for 13 variables are shown in the table. Not all of these are available for every patient: 3 patients, although without symptoms, were referred to a medical emergency department because of a base excess of less than −6 mmol/l. One of these had a methanol concentration of 30 mmol/l, and was treated with ethanol, although no more acidosis developed.

Most patients had drunk 1–2 l of cleansing solution over several days, and had an average blood methanol concentration of more than 20 mmol/l and an average blood ethanol concentration of more than 50 mmol/l (230 mg/dl) on admission. Most patients had higher ethanol than methanol levels, but in some patients the opposite occurred. No patient had measurable concentrations of ethanol in the blood at 12 h. The elimination rate of methanol averaged 0·98 mmol/l per h. Two or more blood gas analyses were performed in 72 patients: the base excess increased or remained unchanged in 59 and decreased in only 13.

There was no relation between the initial methanol and ethanol concentrations. The methanol concentration on admission was, however, inversely related to the ethanol concentration existing when the lowest base excess value was measured ($p < 0·01$). This initial methanol concentration was also inversely correlated to the systolic blood pressure on admission ($p < 0·02$).

The elimination rate of methanol was inversely related to the ethanol concentration on admission, and the

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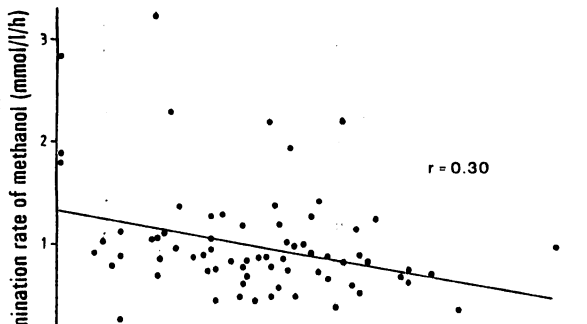

Elimination rate of methanol in relation to the ethanol concentration on admission.

Coefficient of correlation 0·304; standard error of estimate 0·53.

concentration at the minimum base excess ($p < 0.01$ and 0·05, respectively). The higher the ethanol concentration, the lower the rate of elimination of methanol (fig), although there is a considerable standard error. The rate of elimination of methanol was positively correlated to age ($p < 0.02$). Age was also related to the presence of neurological symptoms ($p < 0.05$).

Discussion

In classic methanol poisoning there is a delay of 12–24 h before a metabolic acidosis develops and symptoms occur. There is no known relation between the blood concentration of methanol and the symptoms, suggesting that it is the metabolites which cause the toxicity. Methanol is oxidised to formate within minutes,[5,6] by several different metabolic routes.[1] Formate is oxidised to carbon dioxide, possibly by the folate biochemical pathway, since the rate of formate oxidation can be limited by the amount of tetrahydrofolate in the liver.[7] Lactate levels also increase in advanced methanol poisoning because of tissue hypoxia, but in the early stages the major component of metabolic acidosis is formate alone.[8]

In this report, chronic alcoholics ingesting mixtures of methanol/ethanol solutions do not suffer the serious sequelae associated with classic methanol poisoning, although many patients had only methanol in their blood after the ethanol had been metabolised.

Competitive inhibition of alcohol dehydrogenase during binge drinking of alcohol can cause accumulation of small quantities of endogenous methanol and other volatiles.[9] Majchrowicz and Mendelson[10] found blood concentrations of endogenous methanol of up to 0·24 mmol/l with blood ethanol concentrations between 32·6–97·8 mmol/l. It is unlikely that endogenous methanol accounted for a significant proportion of the methanol found in this study, because the initial methanol concentrations in our patients were much higher. We found no increase in respiratory rate, and no acidosis developed even with methanol levels up to 64 mmol/l. Lactate and formate levels were not measured: since no acidosis developed, these levels cannot have been grossly raised. These patients may not metabolise methanol to formate, or the formate may be metabolised faster and not accumulated. This suggestion is supported by a study in a similar group of chronic alcoholic patients,[3] where the rate of metabolism of methanol was also 1 mmol/l per h in patients who were not simultaneously treated with ethanol. In that study, formate levels increased slightly from 1 to 3 mmol/l, which suggests that metabolism does occur, but with no acidosis or clinical sequelae. Two other publications report patients presenting with high methanol concentrations without symptoms or acidosis. In one, a patient presented 24 h after ingesting 400 ml methanol;[11] in the other, methanol concentrations of between 31–41 mmol/l were found in 2 patients after taking methylated spirits containing 5% methanol.[12]

There was no relation between the initial methanol and ethanol concentrations. This is to be expected since the time between stopping drinking and admission to hospital varied. The rate of elimination of methanol was inversely related to the ethanol concentration on admission, and there seems to be a linear relation between these variables, although there was a large standard error of estimate. Some patients metabolised over 2 mmol/l/h methanol, with no acidosis, despite ethanol concentrations of 50–75 mmol/l. In these cases, methanol metabolism was not blocked by a concentration of 21·7 mmol/l (100 mg/dl) ethanol—which is the level usually accepted as being adequate to inhibit methanol and ethylene glycol oxidation.[13] Age was related to the rate of elimination of methanol and this may indicate enzyme induction after a longer period of alcohol abuse. Diffuse neurological symptoms were also related to age, but may be just the result of many years of heavy drinking.

The diagnosis and treatment of methanol poisoning is often based on blood concentrations alone, dialysis being recommended in all patients with high blood methanol levels.[1,2] There appear to be two groups of patients—those with classic acute methanol intoxication, and chronic alcoholics who misuse methanol/ethanol mixtures. With conventional treatment, many of our patients in this second group would have been subjected to unnecessary therapy, perhaps even dialysis. The diagnosis and treatment of combined methanol and ethanol poisoning should be based on the case history, the clinical signs, and the presence of a metabolic acidosis—not on blood methanol concentrations alone.

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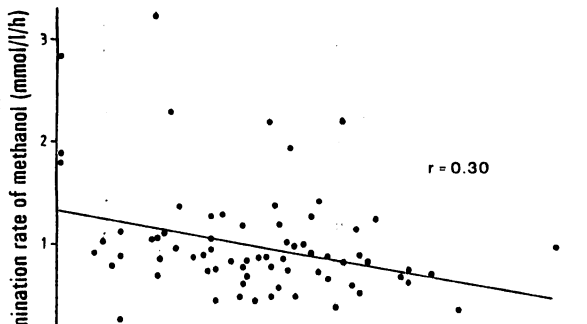

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