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ARTICLE

What Are the Adverse Effects of Ethanol Used as an Antidote in the Treatment of Suspected Methanol Poisoning in Children?[#]

Michel Roy, M.D., F.R.C.P.C.,[1] Benoit Bailey, M.D., M.Sc., F.R.C.P.C.,[1,2,*] Dominic Chalut, M.D., F.R.C.P.C.,[3] Pierre-Etienne Senécal, M.D., F.R.C.P.C.,[3] and Pierre Gaudreault, M.D., F.R.C.P.C.[2]

[1]Division of Emergency Medicine and [2]Division of Clinical Pharmacology and Toxicology, Department of Pediatrics, Hôpital Ste-Justine, Université de Montréal, Montréal, Quebec, Canada
[3]Montreal Children's Hospital, McGill University Health Centre, Montréal, Quebec, Canada

ABSTRACT

Background. Ethanol used as an antidote is said to have various adverse effects, particularly in children. The rate of these adverse effects is not known.

Methods. Twenty-one-year retrospective chart review (1980–2000) from suspected methanol poisoning patients treated with ethanol in two large pediatric tertiary care centers.

Results. A total of 60 children (median age of 24 months) received ethanol for suspected methanol poisoning: 39 orally and 21 intravenously. Median initial methanol level was 4.16 mmol/L (13.3 mg/dL) (range 0 to 87.5 mmol/L or 0 to 280 mg/dL). Median duration of ethanol treatment was 16 hours (range 1.5 to 72 hours). None [0% (95% CI 0–5%)] of the 60 patients developed symptomatic hypoglycemia. Of the 50 patients that had a glucose level measured, none [(0% [95% CI 0–6%)] had a serum glucose concentration <2.78 mmol/L (<50 mg/dL). Eight patients [16% (95% CI 8–30%)] had at least one serum glucose concentration between 2.78–3.61 mmol/L (50–65 mg/dL), but none of those had symptoms compatible with hypoglycemia. A total of 42 patients [84% (95% CI 70–92%)] had all their serum glucose concentrations >3.61 mmol/L (>65 mg/dL). There was no identifiable difference in the glucose intake between the serum glucose concentration groups. Six out of the 60 patients [10% (95% CI 4–21%)] were described as more drowsy after ethanol but none was comatose or needed intubation. No child showed signs of hypothermia [0/40 (95% CI 0–8%)] (rectal temperature $<35°C$), hepatotoxicity (0/12) (AST or ALT >100 U/L) or even thrombophlebitis (0/21). None of the 22 patients with toxic levels of methanol (≥ 6.2 mmol/L– ≥ 20 mg/dL) died or had ethanol-induced morbidity despite wide variation in ethanol levels.

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[*]Correspondence: Benoit Bailey, M.D., M.Sc., F.R.C.P.C., Hôpital Ste-Justine, 3175 Chemin de la Côte-Ste-Catherine, Montréal, Quebec, Canada, H3T 1C5; Fax: (514) 345-4823; E-mail: baileyb@med.umontreal.ca.

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Conclusion. The rate of clinically important adverse effects related to ethanol used as an antidote to treat methanol poisoning in children was either absent or low in a tertiary care pediatric hospital setting. There was no morbidity or mortality associated with ethanol when it was used despite wide variation in ethanol levels. These results suggest that with appropriate monitoring and intravenous glucose intake in a controlled environment such as a pediatric intensive care unit, ethanol therapy does not carry as many risks as currently believed.

Key Words: Methanol; Poisoning; Child; Ethanol; Treatment; Adverse effects.

INTRODUCTION

Ethanol has been used as an antidote for methanol poisoning for many decades because it inhibits the alcohol dehydrogenase enzyme that leads to the production of the toxic metabolite of methanol, formic acid (1,2). It was the sole antidote available for that purpose until fomepizole became available in the past few years in North America (2). Fomepizole is promoted as having several advantages over ethanol including slower rate of elimination and predictable pharmacokinetics, thus facilitating its administration, stronger potency, and a lesser potential for adverse effects (2,3). Its disadvantages include cost and availability.

However, there has been no published study that evaluates the rate of adverse effects associated with the use of ethanol as an antidote. Purported adverse effects include hypoglycemia, CNS depression, hypotension, and thrombophlebitis (3). Thus, the main objective of this study was to determine the incidence of hypoglycemia and other adverse effects when ethanol was used as an antidote in the treatment of suspected methanol poisoning in a pediatric population. Another objective was to document the clinical outcome of the more significant methanol poisonings when treated with ethanol over the last 21 years in two pediatric tertiary care hospitals.

METHODS

Study Population

A retrospective chart review of all admissions for methanol and ethylene glycol poisonings over a 21-year period (January 1980 to December 2000) was done in the two pediatric tertiary care centers in one city (combined annual pediatric emergency visits of 150,000 patients). These hospitals are the reference hospitals for a population of approximately 5 million. Charts were identified using the ICD-9 code 980.1 (methanol), and 982.8 and 987.8 (ethylene glycol). All patients 0 to 18 years old with suspected methanol poisoning who received at least one dose of ethanol were enrolled. Patients who did not receive ethanol were excluded. Because there was only one patient with either confirmed or suspected ethylene glycol poisoning that received ethanol, this study focused on the larger group of methanol poisoning and excluded the ethylene glycol case. Charts were reviewed by one investigator in each center using a standardized form and a priori definitions. Age, weight, sex, and the nature of the toxic substance swallowed were all noted. Bolus and maintenance doses of ethanol used were recorded. Parenteral glucose intake was calculated by summing up the amount of glucose received via peripheral IV by unit of time. Finally, symptoms were reviewed by careful reading of all nursing and medical notes.

Adverse Effects

Blood glucose levels were divided in three groups according to the lowest serum glucose concentration recorded: 1) at least one glucose level < 2.78 mmol/L (< 50 mg/dL), 2) at least one serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL) and 3) all serum glucose concentration > 3.61 mmol/L (> 65 mg/dL). Hypoglycemia was defined a priori as at least one serum glucose concentration < 2.78 mmol/L (< 50 mg/dL) or at least one serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL) with the presence of symptoms compatible with hypoglycemia (diaphoresis, sudden altered behavior, or somnolence). To assess the impact of duration of ethanol treatment on incidence of hypoglycemia, cases were additionally divided and analyzed in two groups of patients: those who received ethanol as bolus and maintenance doses (BM group—mainly patients with methanol levels ≥ 6.2 mmol/L– ≥ 20 mg/dL) and those who received ethanol only as bolus dose (B group—mainly consisting of suspected methanol poisoning cases with methanol levels < 6.2 mmol/L– < 20 mg/dL). We also evaluated if there was a correlation between the total ethanol dose given and the lowest

glucose concentration observed for each patient. Furthermore, the proportion of patients five years or younger with all serum glucose concentration $\geq$ 3.61 mmol/L ($\geq$65 mg/dL) and those with at least one serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL) was compared using a Fisher exact test. Glucose intake between serum glucose concentration groups were compared using the Mann-Whitney Rank Sum test. We also correlated serum glucose concentration with glucose intake using Spearman correlation. Difference in ethanol levels (highest, lowest, and variation between highest and lowest) between serum glucose concentration groups was compared using either a student t-test or a Mann-Whitney Rank Sum test, whenever appropriate.

The effect of ethanol on the level of consciousness was studied using two clinical parameters: 1) any decrease in the Glasgow coma scale or 2) any mention of drowsiness in the patient's chart. Results were then categorized as follows: "more drowsy after ethanol," "comatose after ethanol," or "needed intubation after ethanol."

The effect of ethanol on the blood pressure was also evaluated. Hypotension was defined as a measure of blood pressure below two standard deviations for age (below 95th percentile) anytime after initiation of ethanol therapy (4). Furthermore, hepatotoxicity and hypothermia were evaluated. Hepatotoxicity was defined as an elevation of liver enzymes (AST or ALT $>$ 100 U/L). Hypothermia was defined as a rectal temperature below 35°C (95°F). Mention of clinically suspected thrombophlebitis, gastritis, or pancreatitis were also noted.

Outcome of Patients with Toxic Methanol Level

To further describe our study group, methanol poisoning outcome was detailed for patients with a toxic methanol level (methanol $\geq$ 6.2 mmol/L– $\geq$ 20 mg/dL) and for patients with any methanol level with acidemia on arrival (serum bicarbonates $<$ 20 mmol/L– $<$ 20 meq/L). Studied outcome variables were initial methanol level, delay between ingestion and presentation, initial presence of acidemia (defined as serum bicarbonate $<$ 20 mmol/L– $<$ 20 meq/L), progression of acidemia [defined as a decrease of more than 2 mmol/L (2 meq/L) in serum bicarbonates], necessity of hemodialysis, hospital stay [duration of stay in the pediatric intensive care unit (PICU) and general pediatric ward], visual impairment, other morbidity, and mortality.

Results are expressed as mean $\pm$ standard deviation or median (range), whenever appropriate. Level of significance was set at $p < 0.05$.

RESULTS

During the study period, 60 children (39 boys and 21 girls) received ethanol for suspected methanol poisoning. Sources of the ingested methanol are given in Table 1. The median age of patients was 24 months (range 6 months to 18 years): 45 were 5 years old or younger. Median initial methanol levels was 4.16 mmol/L (13.3 mg/dL) (range 0 to 87.5 mmol/L or 0 to 280 mg/dL). Twelve patients had undetectable methanol levels.

Thirty-nine patients received oral (PO) ethanol. Median initial PO dose was 0.74 g/kg (range 0.15 to 1.33 g/kg); a second PO bolus was given in 7 cases with a median dose of 0.74 g/kg (range 0.16 to 1.28 g/kg). Of the 39 that received PO boluses, 28 also received PO maintenance for a median of 0.38 g/kg (range 0.09 to 0.82 g/kg) every 4 hours for a median of 4 doses (range 1 to 18 doses).

Twenty-one patients received intravenous (IV) ethanol. Initial median IV bolus dose was 0.77 g/kg (range 0.40 to 0.90 g/kg); a second IV bolus was given in 4 cases at a dose of 0.49 $\pm$ 0.18 g/kg. Of the 21 patients that received IV boluses, 19 received IV maintenance with a median dose of 0.13 g/kg/h (range 0.06 to 0.22 g/kg/h) for a median of 16 hours (1.5 to 64 hours).

Overall, the patients received an average of 2.4 $\pm$ 1.5 g/kg of ethanol (range 0.2 to 19.1 g/kg).

Adverse Effects

None of the 60 patients developed symptomatic hypoglycemia (95% CI 0–5%). A total of 50 patients had serum glucose concentration measured during ethanol treatment. There were 3.8 +/− 2.5 serum glucose concentration determinations per patient with a median of 3 levels per patient (range 1 to 13). None of those 50 patients (95% CI 0–6%) had serum glucose concentration measured $<$ 2.78 mmol/L ($<$50 mg/dL). Eight of

Table 1. Source of ingested methanol.

Source	n (%)
Windshield washer	18 (30)
Fondue fluid	13 (22)
Gasoline de-icer	4 (7)
Paint remover	4 (7)
Polish remover	2 (3)
Others	10 (17)
Unknown	9 (15)

the 50 patients or 16% (95% CI 8–30%) had at least one serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL). None of those patients had symptoms suggestive of hypoglycemia. A total of 42/50 patients or 84% (95% CI 70–92%) had all serum glucose concentration measured >3.61 mmol/L (>65 mg/dL). There was no difference, in the proportion of patients aged 5 years or younger, between groups of patients with all serum glucose concentration >3.61 mmol/L (>65 mg/dL), and those with at least one serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL): 32/42 vs. 7/8, respectively ($p = 0.66$).

The majority of patients (50/60) had an IV dextrose solution during treatment: 40 patients had a dextrose 5% solution and 10 patients had a dextrose 10% solution. Serum glucose concentration results were similar in the patients that received a bolus and maintenance doses of ethanol and those who only received a bolus of ethanol (Table 2). There was no significant difference in glucose intake in these two groups (Table 2). Furthermore, there was no correlation between serum glucose concentration and intravenous glucose intake ($r_s = 0.14$, $p = 0.34$).

There was no difference in highest and lowest serum ethanol concentration measured in the groups of patients with all serum glucose concentration >3.61 mmol/L (>65 mg/dL) and those with at least one serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL): highest serum ethanol concentration measured: 16.3 ± 14.8 vs. 16.7 ± 15.2 mmol/L (75 ± 68 vs. 77 ± 70 mg/dL), respectively, $p = 0.95$; lowest serum ethanol concentration measured: 2.0 mmol/L (range 0–24.8) vs. 1.0 mmol/L (range 0–21.7) [9 mg/dL (range 0–114) vs. 4.8 mg/dL (range 0–100)], respectively, $p = 0.80$. Furthermore, there was no difference in the variation between highest and lowest serum ethanol concentration measured: 16.1 ± 11.5 vs. 18.0 ± 14.8 mmol/L (74 ± 53 vs. 83 ± 68 mg/dL), respectively, $p = 0.74$. Also, there was no correlation between the total ethanol dose given and the lowest serum glucose concentration ($r = 0.15$, $p = 0.30$).

The rate of the other adverse effects is given in Table 3. In the six patients found more drowsy after ethanol, three had nontoxic methanol levels (0, 0, 2.5 mmol/L; 0, 0 and 8 mg/dL). They respectively had a peak ethanol level of 0.6, 8.0, and 25.6 mmol/L (3, 37 and 119 mg/dL). The other 3 patients had methanol levels of 9.6, 38.4, and 86.8 mmol/L (31, 124, and 280 mg/dL) with peak ethanol levels of 34.4, 48.4, and 25.2 mmol/L, respectively (160, 225, and 117 mg/dL). The maximum time to recover from the drowsiness was 8 hours in a patient that coingested codeine and received diazepam as treatment. It lasted 4 hours in one patient and less than 2 hours in the other four patients. After initiation of ethanol therapy, one patient had a blood pressure below two standard deviations for age. This particular patient arrived in the emergency department 72 hours postingestion of methanol with a severe metabolic acidosis (pH 6.76). On arrival, he was dehydrated and already hypotensive before any treatment was even started.

One patient had a clinically suspected erosive gastritis and was treated with ranitidine. This patient was treated exclusively with intravenous ethanol. No amylase or lipase result could be found in the chart for this patient or any other.

Table 2. Serum glucose concentration and parenteral glucose intake in patients who received ethanol either as bolus only (B group) or as bolus and maintenance (BM group).

	B group (n = 9)	BM group (n = 41)	Median glucose intake* (g/kg/h)
All [glucose] ≥ 3.61 mmol/L (65 mg/dL)	8	34	0.18 (0–0.84)
At least one [glucose] 2.78–3.61 mmol/L (50–64 mg/dL)	1	7	0.22 (0.11–0.34)
At least one [glucose] < 2.78 mmol/L (<50 mg/dL)	0	0	—
Median glucose intake (g/kg/h)**	0.22 (0–0.84)	0.16 (0.04–0.70)	

*p = 0.93.
**p = 0.33.

Table 3. Rate of other adverse effects associated with ethanol as an antidote in children.

	n (%)	[95% CI]
More drowsy after ethanol	6/60 (10)[a]	4–21
Comatose after ethanol	0/60 (0)	0–5
Needed intubation after ethanol	0/60 (0)	0–5
Hypotension	1/30 (3)[b]	0.2–19
Hypothermia	0/40 (0)	0–8
Hepatotoxicity	0/12 (0)	
Thrombophlebitis	0/21 (0)	

[a]See text for description.
[b]Patient was hypotensive before initiation of ethanol.

Outcome of Patients with Toxic Methanol Level

In the subgroup of 22 patients with a toxic methanol level (all with methanol $\geq$ 6.2 mmol/L– $\geq$ 20 mg/dL), the initial median methanol level was 11.9 mmol/L (range 6.2 to 87.5) [38 mg/dL (range 20 to 280 mg/dL)]. Median presentation time was 3 hours postingestion (range 1 to 72).

Seven out of the 22 patients had acidemia on arrival (serum bicarbonate 13.8 $\pm$ 6.8 mmol/L or mEq/L; range 2.0–19.7). Five of those seven patients received IV bicarbonate. There was no further decrease of serum bicarbonate during ethanol treatment. However three patients, with normal serum bicarbonate on arrival, developed mild acidemia during treatment with ethanol (Fig. 1). None of these three patients received bicarbonate. Despite a lowering in serum bicarbonate upon discharge, one patient had a methanol level of 0 mmol/L (0 mg/dL) at that time and was completely asymptomatic. No control of the serum bicarbonate was requested in this patient.

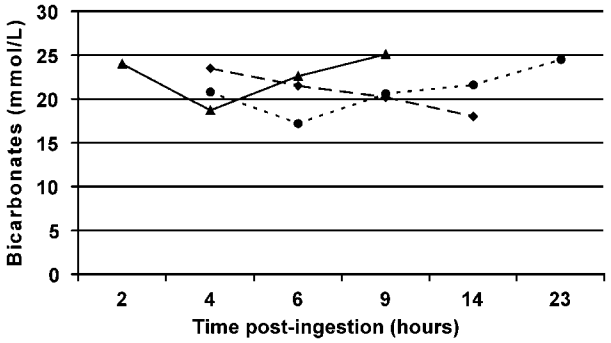

Figure 1. Evolution of bicarbonate in the three patients who arrived with no acidemia but developed it later during treatment.

In the 22 patients with toxic methanol levels, 9 were hemodialysed: 8 with methanol level > 16.6 mmol/L (>50 mg/dL); and one with methanol level of 11.9 mmol/L (38 mg/dL) and bicarbonate of 18.5 mmol/L (18.5 mEq/L).

Nine of the 22 patients were admitted to the pediatric intensive care unit for a median length of stay of 24 hours (range 12–48 hours). Patients were then transferred to a pediatric ward for a median stay of 31 hours (range 0–96 hours). The other 13 patients were admitted directly to a pediatric ward for a median stay of 48 hours (range 13–216 hours). No patient had visual impairment either on admission or at discharge. Nineteen out of 22 patients were evaluated by an ophthalmologist. There was no mortality and no morbidity.

DISCUSSION

Since the recent approval of fomepizole as a new antidote for ethylene glycol poisonings and more recently for methanol poisonings, ethanol has been criticized with numerous adverse effects (5–7). However, the American Academy of Clinical Toxicology 1999 practice guidelines on ethylene glycol poisoning and more recently the 2002 practice guidelines on methanol poisoning underline that there are few data on the complications of ethanol infusion, especially in children (3,8).

According to some, an important advantage of the new antidote in the pediatric age group is that it eliminates the concern for ethanol-induced hypoglycemia (7). Although ethanol-induced hypoglycemia has never been studied when ethanol is used as an antidote, hypoglycemia secondary to acute alcohol intoxications has been emphasized in case reports and case series in children (9–20). In fact, in case series of children less than 18 years of age, hypoglycemia occurred in anywhere from 0 to 40% of children intoxicated with ethanol (15–21). In the few case series of children less than 7 years of age, the incidence of hypoglycemia after ethanol intoxication varied from 0 to 19% (17,19). In our study, there was no hypoglycemia (95% CI 0–6%) noted when ethanol was used as an antidote. The difference in incidence between the two situations, intoxication vs. treatment, may well be related to the presence of a continuous dextrose infusion in the majority of patients in the treatment group. In ethanol intoxication, hypoglycemia was usually reported on presentation at the hospital prior to intravenous dextrose administration and not during their hospital stay, suggesting that ethanol-induced hypoglycemia is preventable with intravenous glucose intake. Also there was theoretically

a protective effect by monitoring the blood glucose levels during the treatment with ethanol—although actually, no adjustment of glucose infusion rate was done because of lowering blood glucose in our study.

Anecdotal reports have suggested that children under 5 years of age develop hypoglycemia more often than adolescents when exposed to ethanol (9). It has been suggested that younger children are more vulnerable and sensitive to ethanol-induced hypoglycemia (22). Previously published case series of children intoxicated with ethanol do not support this (17,19). This study does not support the assertion that younger children would be at greater risk for hypoglycemia from ethanol if used as an antidote.

When studying serum glucose concentration levels, clinically significant hypoglycemia should be predefined. According to Haymond, hypoglycemia must be defined in the context of the clinical setting (22). Any child or infant with a quantitative serum glucose concentration <2.78 mmol/L (<50 mg/dL) must be carefully observed; and at concentrations lower than 2.22 mmol/L (<40 mg/dL), the child should be considered to be hypoglycemic and diagnostic and therapeutic interventions initiated (22). In this study the level of 2.78 mmol/L (50 mg/dL) was our lower limit for normoglycemia. To be more conservative, patients who had at least one serum glucose concentration value between 2.78 and 3.61 mmol/L (50 and 65 mg/dL) and presented hypoglycemia compatible symptoms were also considered to be hypoglycemic. However, none of the eight patients that had serum glucose concentration between 2.78 and 3.61 mmol/L (50 and 65 mg/dL) had symptoms compatible with hypoglycemia.

Central nervous system depression is another concern with ethanol therapy in children. The most common presenting finding of ethanol intoxication in children is sleepiness (15). In a study of acute ethanol intoxications in children, 61% of patients over 8 years old were found to be more somnolent (15). In another study on acute ethanol intoxications, conscious level was normal in 12% of patients while 26% of patients were in coma (16). In our study, 10% (95% CI 4–21%) of patients were noted to be more drowsy after initiation of ethanol therapy. More importantly, however, none were comatose or needed intubation. However, in at least one case, drowsiness could have been caused by either coingestant (codeine) or cotherapy (diazepam). Moreover, ethanol level was low (0 to 8 mmol/l–0 to 38 md/dL) in two patients and the drowsiness lasted 2 hours or less in four patients. Among patients screened for other adverse reactions, no case of hepatotoxicity, hypothermia, or thrombophlebitis could be found. One patient was treated with ranitidine for clinically suspected gastritis. It is unlikely that the suspected gastritis could have been secondary to ethanol since it was given parenterally. Hantson and Mahieu reported an adult series of pancreatic injury following methanol poisoning and suggested that ethanol therapy may complicate the pancreatic injury (23). In our study, only the patient with the clinically suspected gastritis could have had symptoms compatible with pancreatic injury but no amylase or lipase levels were requested.

Heterogeneous pharmacokinetics of ethanol therapy complicates its dosage and the targeting of the therapeutic concentration of 21.8 mmol/L (100 mg/dL) (3). This has been illustrated in a review carried in a poison control center in which serum ethanol concentrations were above the 21.8 mmol/L (100 mg/dL) level consistently in only 2/17 patients (24). Similar difficulties were encountered in our population as illustrated in Fig. 2. However, despite wide variation in ethanol levels, there was no impact on outcome. This may be due to the fact that the target level of 21.8 mmol/L (100 mg/dL) is not evidence-based and that the therapeutic level may actually be lower. Since our population presented early (a median of 3 hours postingestion), it is unclear what would have been the impact of such variations in the targeted level of ethanol if the delay of presentation was longer. Our results may suggest that it is not the actual ethanol level that is important for outcome but, more importantly, how long after methanol ingestion ethanol is actually started.

Limitation of our study includes its retrospective nature. Not all of our patients had regular measures of serum glucose concentration. As monitoring of patients was not to the present standard of care in the earlier days of the study (patients admitted to a general pediatric ward

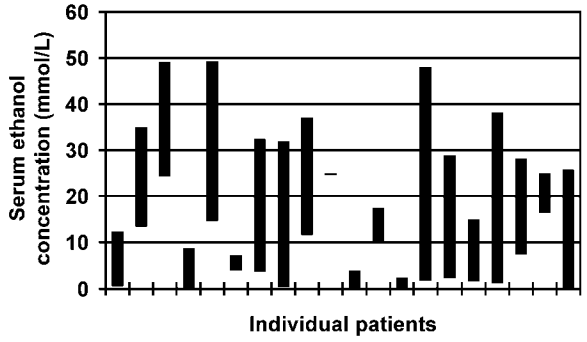

Figure 2. Highest and lowest ethanol levels measured for each patient with methanol ≥ 6.2 mmol/L (≥ 20 mg/dL). Upper-end of boxes = highest ethanolemia measured; Lower-end of boxes = lowest ethanolemia measured.

with no or few glucose levels monitoring), it is unclear what the impact of optimal monitoring would have had on the study results. However, it is reassuring that despite such monitoring, there was no documented adverse effects and no morbidity associated with ethanol treatment. This further suggests that with appropriate monitoring in a controlled environment such as a pediatric intensive care unit, ethanol therapy does not carry as many risks as currently believed. Also, only parenteral glucose intake was evaluated. We have not attempted to evaluate oral intake of glucose; this extra glucose intake could have had a protective effect against hypoglycemia. Furthermore, most patients received ethanol for a short period of time either because they did not ingest methanol (suspected cases) or because they had low methanol level (nontoxic cases). Therefore, it is unclear if a longer period of treatment could have resulted in more adverse effects. Also, it is unclear what was the impact of the wide variations of ethanol level observed during treatment on the incidence of adverse effects.

Thus, our results suggest that the incidence of adverse effects is at most low when ethanol is used as an antidote in children in a tertiary care hospital setting. Furthermore, our study also illustrates that children treated with ethanol for methanol poisoning usually have a good prognosis despite the wide variation in ethanol levels. Future studies should focus on evaluating what is the required therapeutic level of ethanol and comparing its efficacy and cost-effectiveness with that of fomepizole.

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