Methyl alcohol poisoning in an 8-month-old boy: An unusual route of intoxication

André Kahn, M.D., and Denise Blum, M.D., *Brussels, Belgium*

METHYL ALCOHOL POISONING is rarely reported in the pediatric literature.[1, 2] Between 1966 and 1977 we observed eight cases in our Brussels clinic. We describe a child who died after methanol intoxication, presumably due to percutaneous resorption.

CASE REPORT

This 8-month-old boy was well until seven days before admission, when a cough developed, with low-grade fever. Acetylsalicylic acid was prescribed. The day before admission, the child's respirations became progressively rapid and shallow. A radiograph of the chest performed in another hospital was considered normal. In the afternoon the child had increasing drowsiness and did not wake up for his evening meal. The day of admission the parents were alarmed at the worsening of the dyspnea and their inability to awaken the child. On admission to our hospital he was in a coma, Stage III; the pupils were dilated and unresponsive to light; there were recent hemorrhages in both fundi and slight papillary edema. The tendon reflexes were absent. The temperature was 37.8°C, the pulse 132/minute, and the respirations were deep and 25/minute; blood pressure was 90/60 mm Hg. Slight cyanosis and some petechiae on the thorax were noted. The edge of the liver was felt 2 cm below the right costal margin. Treatment consisted of artificial ventilation and antibiotics (ampicillin, oxacillin); bicarbonate was added owing to the presence of severe metabolic acidosis (pH 6.50, CO_2 20 mEq/l, standard bicarbonate below 3 mEq/l).

The differential diagnosis of severe metabolic acidosis classically includes diabetes mellitus, acute renal failure, poisonings (methanol, salicylate, nalidixic acid, ethylene glycol), and lactic acidosis. Other causes such as abnormalities of branched-chain amino acid metabolism and fructosemia seemed unlikely in view of the patient's history.

The blood glucose concentration was 293 mg/dl, the BUN 44 mg/dl; the prothrombin time was 56% of normal, the glutamic

From the Pediatric Intensive Care Unit, Department of Pediatrics, Free University of Brussels.
Reprint address: Service de Pédiatrie, Hôpital Universitaire Saint-Pierre, Rue Haute 322, B-1000 Bruxelles, Belgium.

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oxalacetic transaminase 73 IU, the glutamic pyruvic transaminase 37 IU, the lactic dehydrogenase more than 1,000 IU, the creatine phosphokinase 103 IU, the amylase 11 IU. Blood ammonia concentration was 495 γ/dl and lactic acid 16 mEq/l. The WBC count was 24,000, with 70% neutrophils. Bacterial blood culture was negative. Serologic tests for viral infections (complement fixation and hemagglutination-inhibition) were negative. The urine (pH 6) gave a positive test for protein and glucose, and a trace positive for acetone; Phenistix was negative; the sediment contained granular casts. Chromatography of amino acids was performed on blood and urine and revealed generalized hyperaminoacidemia and exaggerated excretion of methionine and proline in urine, as can be seen in acute liver dysfunction. Toxicologic screening performed at the time of admission (including currently taken drugs like acetylsalicylic acid) was negative. On account of the severe metabolic acidosis, samples of blood and urine were also sent to the Belgian Anti-Poison Center in search of methyl alcohol. In the early afternoon, the laboratory confirmed the presence of 40 mg/dl methanol in the blood (method of measurement: oxidation by means of acid permanganate to formaldehyde, which is then determined colorimetrically).

Specific treatment was started immediately; ethyl alcohol 0.33 gm/kg iv initially, followed by 0.25 gm/kg every 4 hours, and peritoneal dialysis (the dialysing fluid containing sodium bicarbonate instead of the sodium lactate routinely used). Hemodialysis, which is the treatment of choice, was not instituted because of hemodynamic instability developing despite early administration of dopamine (5 γ/kg/minute iv). Methanol was identified in the urine and in peritoneal fluid (no quantitative estimation). The child's condition deteriorated rapidly, accompanied by hypothermia and episodes of bradycardia. An electroencephalogram was isoelectric. In the evening the arterial blood pressure fell and the child died shortly thereafter. A liver biopsy was done at the time of death and revealed diffuse fatty degeneration. A lumbar puncture performed just after the child's death revealed frank hemorrhage; bacterial culture was negative.

A new interview with the parents revealed that it is the custom of this family to "take off the cold" with warm compresses applied to the child's chest, which is previously rubbed with olive oil. First the compresses are soaked with alcohol, set on fire, and rapidly extinguished with a plate. Having no ethyl alcohol, which is traditionally used, the mother had accidentally purchased methyl alcohol; methanol soaked pads had been applied to the child's chest during the two nights preceding admission (approximate duration of application of the pads: twice 12 hours). Acetaminophen ingestion could be excluded.

DISCUSSION

Few methanol intoxications have been reported in the pediatric literature.[1,2] In our pediatric clinic, eight instances were recorded in 11 years; this represented 1% of the admissions for pediatric intoxication. The other methanol intoxications (apart from the case described above) occured in two girls and five boys. These children were poisoned after either an accidental ingestion of small amounts of pure methanol (four cases) or an ingestion of methanol containing solvents (three cases). In every instance the parents were quickly alerted and the children were brought to the hospital a half an hour to two hours after the accident; as advised by the Anti-Poison Center, four patients received oral ethanol at home. Liver function tests were normal and only one child had slight metabolic acidosis (total CO_2 17 mEq/l). The ocular fundi of all were normal. In four patients the plasma methanol level was determined. The following values were obtained: 20 mg/dl in the first and 2.5 mg/dl in the second; methanol was undetected in the last two. Six children received bicarbonate, and 0.75 ml/kg of ethyl alcohol 50% followed by 0.50 ml/kg orally every 4 hours. Recovery was uneventful in all seven children. In the patient presently reported, owing to lack of a complete history, treatment was started late, at a time when the clinical status was already desperate. Profound coma, and deep and slow respirations terminating in feeble spontaneous chest movements, have been described with this type of intoxication.[4]

Massive necrosis of various organs was probably already present on admission. Prolonged prothrombin time, moderate increase in transaminase activity, and LDH isoenzyme 5 elevation are easily accounted for by the complete hepatic necrosis, which eventually was confirmed by liver biopsy, whereas very high levels of the other ioenzymes probably reflected extended muscular lysis. The ophthalmoscopic observations and the hemorrhagic CSF are compatible with massive hemorrhagic necrosis of the brain.[4] Ethanol and methanol share common metabolic pathways (e.g., hepatic alcohol dehydrogenase). Therefore, the administration of ethanol is likely to slow the oxidation of methanol into its toxic derivatives, formaldehyde and formic acid. Some authors advocate a cautious approach to the therapeutic use of ethanol in young infants.[1] We, on the other hand, administer it as an adjunct to the treatment, as is done in adults. The doses are adjusted to obtain a blood ethanol level of 100 mg/dl. Had the child's hemodynamic and neurologic status been better, hemodialysis, instead of peritoneal dialysis, would have been instituted, because of its greater effectiveness.[3, 5]

A methyl alcohol concentration of 40 mg/dl of blood has been reported to be fatal in man[4] and in animals,[6] although data from fatalities suggest a wide variation in individual sensitivity.[3, 6, 7] Deaths have been reported with methanol levels as low as 19.4, 27.7, and 27.5 mg/dl (respectively, 48, 50, and 50 hours after intoxication).[4] At this low serum-methanol concentration, severe acidosis is evidence of advanced methanol oxidation, and might signify that the risk of death is imminent.[4]

Apart from the classical routes of intoxication, ingestion and inhalation,[6, 7] methyl alcohol has been shown experi-

mentally to cross the skin barrier.[8] Animal models were intoxicated by prolonged contact of liquid methyl alcohol with the skin,[6, 7] and monkeys died after application of methanol-soaked pads under a gas-tight cover.[7] In Argentina, in 1967, several children rubbed with methanol were reported intoxicated and some died,[2] but no specific determination of methanol in the blood was obtained.[5] In our patient, poisoning was presumably due to percutaneous absorption of the toxin, although additional inhalation cannot be excluded.

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