

SHORT COMMUNICATION

Transdermal Methyl Alcohol Intoxication: A Case Report

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Methyl alcohol (methanol) is a colourless, odourless and bitter substance found in solvents, paint removers, varnishes, antifreezes, cologne and grain alcohol (1). Methanol is a central nervous system depressant that is potentially toxic after its ingestion, inhalation or transdermal exposure (1–4). Most of the patients have headache, nausea, vomiting, weakness and vision loss during this period. As a result of high intake, the patient presents with stupor, coma and even death. Although methanol intoxication is most frequently reported due to oral intake, cases of inhalation and transdermal methanol intoxication are reported as well (5). In this paper we report a rare case of transdermal methanol intoxication. We suggest that transdermal intoxication should be considered and questioned whilst taking the medical history of a patient.

CASE REPORT

A 57-year-old man without previous history of any disease had soaked his jumper with 1 l of methanol and wore it to treat his ongoing muscle pain. To increase its efficacy, he covered his jumper with a nylon material and lied down for approximately 1 h and 30 min for extensive exposure of his skin to methanol. Two h after he took off his wet jumper, he had nausea, blurred vision and colour blindness. The patient then visited a medical centre; however, he did not mention his exposure to methanol and was therefore sent home with palliative medication for nausea. Next evening, the patient came to our emergency room with increasingly severe and persistent complaints of nausea, vomiting, and visual disorder, which suggested that he had transdermal methanol intoxication after he mentioned his exposure to transdermal methanol. His arterial blood gas analysis demonstrated the presence of metabolic acidosis. Routine complete blood count and blood biochemistry tests revealed normal test results. Serum bicarbonate was 15 mEq/l, methanol was 15 mOsm/kg, formic acid was 25.1 mg/dl, (reference 0–13 mg/dl), and the patient had metabolic acidosis (pH: 7.11, anion gap: 25). After a detailed examination in our emergency room, the patient was referred to our intensive care unit with a diagnosis of methanol intoxication. The patient was given bicarbonate infusion at a rate of 10 mEq/h. In addition, oral methyl prednisolone (60 mg/day), and vitamin B1 (150 mg/day) were given as replacement therapy. The physical examination of the eyes was normal. As assessed by ophthalmologists, the patient's

visual disorder gradually improved during the period he was observed in the intensive care unit. His cranial imagings revealed no abnormality. Bicarbonate infusion was discontinued after 24 h as metabolic acidosis had normalised. After 72 h of monitoring, the patient was discharged after stabilisation.

DISCUSSION

After oral intake methanol is converted to formaldehyde in the liver and oxidised to formic acid. Formic acid is toxic for central nervous system and as a result, histological hypoxia, which is caused by axonal cell death occurs (6). Percutaneous exposure of methanol is possible when clothes are contaminated with a large amount of methanol. After 30 h of the first exposure, serious metabolic acidosis develops and the plasma osmolarity of bicarbonate increases while the plasma levels decrease. Predominant symptoms of methanol intoxication are headache, lethargy, vertigo, vomiting, blurred vision and at the later phases, loss of vision (1, 2, 6, 7). Hyperpnoea, delirium, convulsion may occur and if untreated, patients may experience convulsive paralysis, coma or even death. In conditions resulting in death, blood methanol levels are between 150 and 300 mg/dl. When our patient was presented to our unit, it had been 36 h after methanol exposure. He had visual symptoms and metabolic acidosis. Formic acid levels were high. Following bicarbonate perfusion to treat metabolic acidosis in addition to steroid and vitamin B1 replacement treatment, health condition of our patient was stabilised after 72 h of follow-up and he was then discharged. Treatment of methanol intoxication includes several options such as antidote treatment that blocks methanol metabolism via alcohol dehydrogenase, cofactor treatment, or dialysis treatment that aims to eliminate methanol and methanol metabolites from the circulation. In our case, we did not use ethyl alcohol as the patient was asymptomatic, and his vital signs were stable. Besides, metabolic acidosis is treated using bicarbonate infusion and/or haemodialysis. It is believed in some regions of Eastern Europe that ethyl or methanol is a remedy for muscle pain. As indicated in a previous report, a patient who had applied methanol transdermally to relieve muscle pain had progressive bilateral optical atrophy (8). It has been revealed that particularly in some geographic regions, methanol is commonly applied on the skin to relieve pain. For that

reason, transdermal toxication should be considered and questioned whilst taking the medical history of a patient.

The authors declare no conflict of interest.

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