



Methanol-Induced Encephalopathy: a Case Report

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Case Report

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A characteristic imaging finding in cases of methanol intoxication is putaminal necrosis, but its presence is usually not suspected due to its rarity. Methanol intoxication generally produces serious neurological symptoms that include visual disturbances and diminished consciousness, characteristically with metabolic acidosis. We reported the case of a 59-year-old man who was admitted to the hospital with diminished consciousness. Acute methanol intoxication was determined as the cause. Laboratory tests revealed high anion gap metabolic acidosis. Diffusion-weighted MRI indicated diffuse symmetric diffusion restriction lesions in the subcortical white matter of both cerebral hemispheres.

Keywords: Methanol intoxication; Diffuse symmetric subcortical white matter lesion; Magnetic resonance imaging

INTRODUCTION

Methanol is a clear, colorless, highly toxic liquid with an odor similar to that of ethanol. It is a common component of solvents, perfumes, paint removers, and windshield washer fluids. It can be accidentally or intentionally ingested, inhaled, or transdermally absorbed (1). Once ingested, methanol is metabolized to formic acid and lactic acid, leading to severe metabolic acidosis with high anion and osmolar gaps. Methanol intoxication affects the central nervous system (CNS), including the optic nerves, deep gray matter, and cerebral white matter, resulting in visual disturbances, seizures, and diminished consciousness. Computed tomography (CT) and magnetic resonance imaging (MRI) are able to identify bilateral putaminal necrosis resulting from methanol toxicity in the CNS (2). Imaging studies have reported effects in the subcortical white matter, hippocampus, optic nerves, tegmentum, cerebral gray matter, and cerebellum (3, 4). Herein, we described the CT and MRI findings of acute methanol intoxication in a 59-year-old man who developed diffuse symmetric diffusion restriction lesions in the subcortical white matter of both cerebral hemispheres with absence of putaminal necrosis.

CASE REPORT

A 59-year-old man who was a habitual alcoholic and living a usual daily life without past illness presented to the emergency room with diminished consciousness. The patient was scored 7 on the Glasgow Coma Scale. Initial laboratory tests indicated the absence of significant abnormalities, with normal blood glucose and ammonia levels; in addition, the arterial blood oxyhemoglobin saturation was within normal limits. The patient had no history of significant illnesses such as chronic liver disease, hepatic encephalopathy, or diabetes mellitus.

Brain CT scans showed minor chronic subdural hemorrhage with low attenuation in the left cerebral convexity and a small old lacune in the left basal ganglia (data not shown) that were not considered as significant under the clinical circumstance. The CT scans revealed the absence of other acute traumatic lesions in the brain. Diffusion-weighted MRI revealed diffuse symmetric diffusion restriction lesions in the subcortical white matter of both cerebral hemispheres (Fig. 1). Magnetic resonance angiography (MRA) showed no abnormalities.

Initially, these were considered as non-specific findings; however, the diffuse symmetric subcortical white matter lesions suggested the possibility of toxic or acquired metabolic encephalopathy, since the patient had a normal health status before he abruptly became stuporous (5). However, it was difficult to diagnose a specific metabolic or toxic encephalopathy based on the imaging findings alone. Among the several acquired metabolic encephalopathies known to affect white matter (5–7), we could not identify a suitable diagnosis for this case based on the white matter involvement pattern and the clinical circumstances. A review of relevant literature (3, 4, 6–8) indicated that several toxic encephalopathies, especially methanol- or ethylene glycol-related CNS changes, produced subcortical white matter lesion patterns remarkably similar to those in our case; However, we were not confident of this, especially due to the lack of putaminal necrosis, which is a characteristic MRI finding in methanol-induced encephalopathy (2, 3, 7). However, the patients' medical record revealed unexplained severe metabolic acidosis (pH 6.8; pCO₂ 29.7; HCO₃ 4.6; base excess -29.7) with a high anion gap (40) and a high osmolar gap (56); without significant increases in lactic acid or ketone bodies. Since methanol or ethylene glycol intoxication is characterized by severe metabolic acidosis (8), we considered the possibility of toxic metabolic encephalopathy due to methanol consumption.

Ten days later, his family informed us of his history of alcoholism and that they had prevented the patient from abusing alcohol during the previous week. In addition, they told us that he had complained of blurred vision, which is a characteristic symptom of methanol intoxication (7), just four hours before he was found unconscious. Because we suspected that he had toxic encephalopathy, through tenacious history taking and a search for the causative agent, his family found windshield washer fluid in the refrigerator, whose principal component is methanol. It was assumed that the patient ingested it, mistaking it for ethanol. However, his blood levels of methanol were not measured. Despite treatment including renal replacement therapy in an intensive care unit, the patient died due to progressive cerebral edema, leading to increased intracranial pressure and uncal herniation.

DISCUSSION

We reported the case of a patient with methanol-induced encephalopathy who was admitted to hospital with stupor. MRI revealed diffuse symmetric subcortical white matter lesions. He also had unexplained metabolic acidosis and complained of visual disturbances.

Methanol intoxication is a rare life-threatening condition that may result from ingesting even a small quantity of methanol (1, 6). It can result from accidental ingestion of methanol-containing products, which are mistaken for ethanol-containing products due to their similar odor (3). Once ingested, methanol is metabolized to formaldehyde, and then to formic acid and formate in the liver. This pathway is easily saturable. The blood formic acid level rapidly increases after ingestion of even a small amount of methanol (6). This intermediate acidic metabolite leads to characteristic, severe high anion gap metabolic acidosis.

Most patients with methanol exposure characteristically complain of visual disturbances, ranging from blurred vision to permanent blindness (3, 9). The visual symptoms of methanol intoxication are thought to result from optic nerve necrosis or demyelination secondary to the myelinoclastic effect of formic acid (9). Occupational sub-chronic inhalation or dermal exposure is reported to mainly result in visual disturbances without an altered mental state (10). As little as one swallow of pure methanol can result in toxic encephalopathy, and its principal symptoms are diminished consciousness and seizure (8). Ingestion of 30 mL of pure methanol usually results in death (7).

Bilateral putaminal necrosis with or without hemorrhage

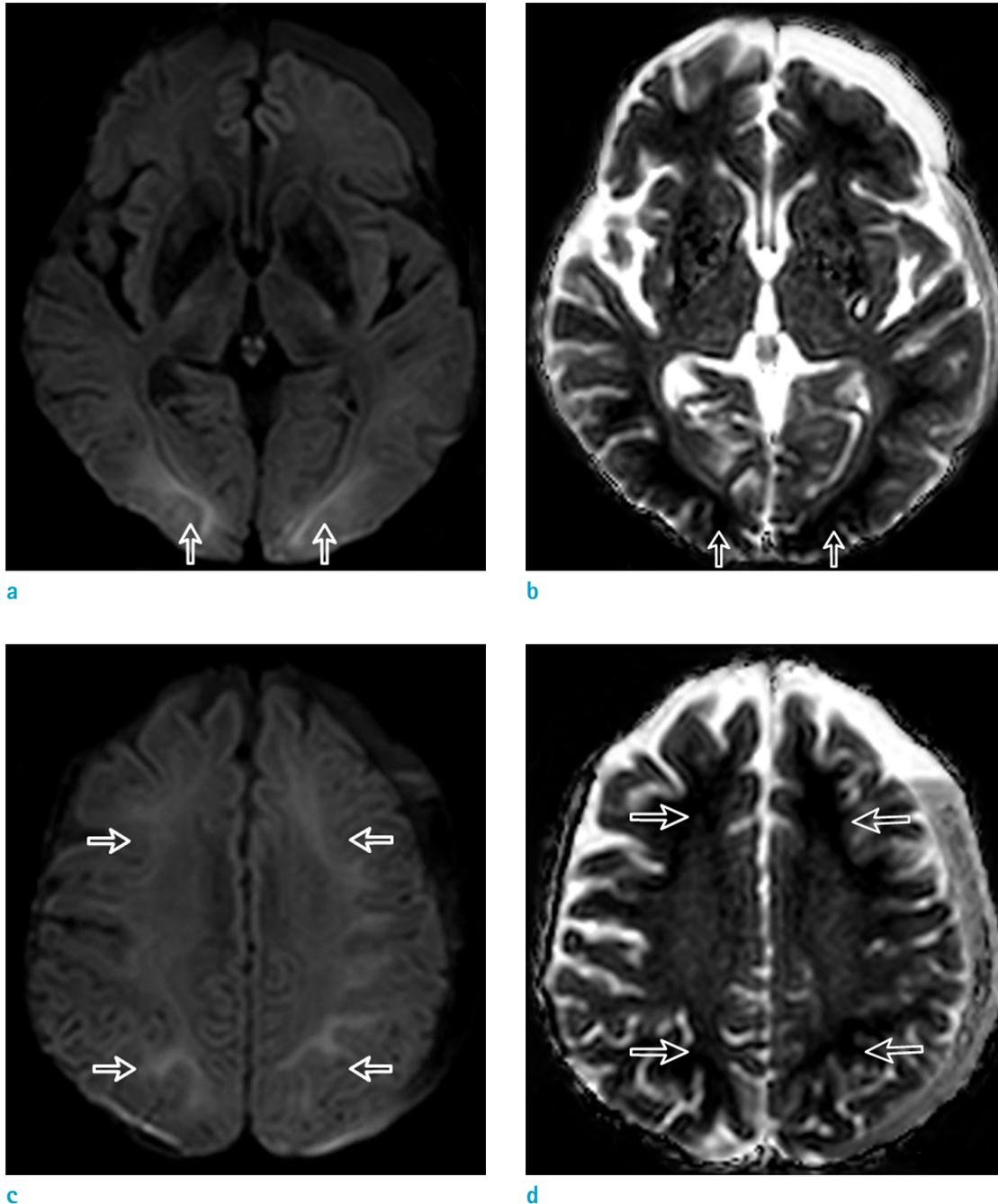


Fig. 1. 59-year-old man with diminished consciousness. (a-d) Diffusion-weighted MRI shows bilateral diffuse symmetric subcortical white matter lesions with a high signal intensity and decreased apparent diffusion coefficient values (white arrows). The lesions were evenly and peripherally distributed, except for the slight predominance in both occipital lobes. These lesions did not affect the overlying cortex. There was no evidence of putaminal necrosis or hemorrhage in this patient.

is accepted as the most characteristic radiological sign of methanol intoxication (3, 7, 9). The detailed mechanism responsible for putaminal necrosis is unknown. It has been postulated that the necrosis results either from direct toxic effects of methanol metabolites and metabolic acidosis on

the basal ganglia, or from decreased blood flow through the basal veins of Rosenthal secondary to hypotension (9).

Subcortical white matter lesions in cases with methanol intoxication have also been reported (3, 5, 7), but these cases had co-occurring putaminal lesions (3). Because

our patient showed bilateral symmetric subcortical white matter lesions without bilateral putaminal necrosis, we were unsure whether accidentally ingested methanol could account for his encephalopathy. However, we suspected an acquired metabolic or toxic encephalopathy because despite the patients' good health status and a history of no significant past illness except for alcohol abuse, he showed abruptly diminished consciousness as a result of the bilateral diffuse symmetric subcortical white matter lesions with reduced apparent diffusion coefficient (5, 7). Several acquired metabolic encephalopathies that can affect bilateral white matter were ruled out as inconsistent with the patient's radiological pattern and clinical circumstances. Considering the patient's visual disturbances, the unexplained severe metabolic acidosis with high anion and osmolar gaps, and the methanol-based windshield washer fluid found in the refrigerator, methanol was considered the most likely causative agent of the toxic encephalopathy (8).

High anion gap metabolic acidosis is usually caused by lactic acidosis or ketoacidosis due to various underlying illnesses or clinical circumstances. Generally, in cases of metabolic acidosis without increased levels of lactate or ketone bodies, methanol or ethylene glycol poisoning is considered as the cause (8). Thus, methanol almost certainly was the causative agent in this case; in addition, the unexplained metabolic acidosis with high anion and osmolar gaps provide a helpful differential point for future clinical practice.

Methanol is the main component of windshield washer fluid, with ethylene glycol sometimes added in small amounts. Ethylene glycol can also cause high anion gap metabolic acidosis and result in toxic encephalopathy with radiological patterns similar to those of methanol intoxication (6); however, since windshield washer fluid contains much larger amounts of methanol than ethylene glycol, although un-confirmed, methanol is the highly likely cause in this case. In daily practice, ethylene glycol should be considered as a rare alternative cause after methanol, because it is usually difficult to differentiate between them.

In conclusion, when diffuse symmetric lesions of the subcortical white matter are detected in a patient with sudden confusion or visual disturbances and severe metabolic acidosis, toxic encephalopathy by methanol should be considered in the differential diagnosis even in the absence of bilateral putaminal necrosis. In this case, tenacious and intentional pursuit of determining potentially ingested toxic material proved helpful.

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