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MR Enhancing Brain Lesions in Methanol Intoxication [Neuroradiology]

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Outline

Graphics

Fig. 1

Abstract

Methanol intoxication can cause necrosis of the putamen and subcortical white matter that is evident on neuroimaging. We report a 47-year-old man with significant methanol intoxication who had enhancing lesions in the caudate nuclei, putamina, hypothalamus, and subcortical white matter by MRI. This case demonstrates that contrast enhancement of brain lesions can be observed after methanol poisoning.

Methanol poisoning causes an acute confusional state, seizures, and coma.
Survivors may have visual loss, parkinsonian signs, and cognitive deficits (1-3). Bilateral necrosis of the putamen and hemispheric white matter lesions are the most common findings on CT (2,4-14) and MRI (8-14). To our knowledge, contrast enhancement of these lesions has not been reported.

We present the case of a man who had a brain MR scan performed 2 weeks after a significant methanol ingestion. Lesions that enhanced with gadolinium were present in the caudate nuclei, putamina, hypothalamus, and subcortical white matter.

CASE REPORT

A 47-year-old man was admitted after ingesting an unknown amount of methanol. At presentation to the emergency department, he was confused and had nausea, vomiting, and headache. The patient stated that he had been drinking antifreeze.
It is not known how many hours had passed from the time of ingestion to his arrival in the emergency room. His mental status deteriorated until he was in a coma. He had metabolic acidosis, and toxicology screening revealed a serum methanol level of 250 mg/dl. An alcohol screen was negative for other alcohols.
He was intubated and treated with sodium bicarbonate, ethanol, folinic acid, and hemodialysis. Seven days after ingesting the methanol, he was transferred to a psychiatric facility. He had a confusional state that lasted for 6 weeks and visual loss that was permanent. Examination at 3 months showed 20/60 vision in the right eye and 20/400 in the left eye. His past medical history was significant for chronic paranoid schizophrenia and a prior methanol ingestion 1 year before this admission.

An MR study of the brain on the 12th day after admission showed lesions in the caudate, hypothalamus, and putamen bilaterally and in the subcortical white matter of the frontal and occipital lobes (Fig. 1). A thin rim of subcortical white matter immediately subjacent to the cortex was spared. These lesions were hyperintense on T2-weighted images. All of the lesions enhanced with gadolinium.
The putamina had linear regions of hypointensity on the precontrast T1-weighted scan.

FIG. 1. A: T2-weighted coronal scan through the frontal lobes demonstrates hyperintensity in the peripheral white matter that spares the U fibers. B:
Postcontrast T1-weighted section through the same region as in A demonstrates enhancement of the peripheral white matter lesions. C: T2-weighted scan through the basal ganglia demonstrates hemorrhagic and hyperintense lesions involving the caudate heads and hyperintensity involving the hypothalamus, putamina, and peripheral white matter. D: Postcontrast T1-weighted coronal section at the same level as in C demonstrates enhancement of the caudate, hypothalamus, putamina, and peripheral white matter lesions.

DISCUSSION

Antifreeze containing methanol or ethylene glycol can be consumed for its intoxicating effect, and our patient was found to have ingested a large amount of methanol. The initial presentation of poisoning with both alcohols is similar, featuring intoxication and the laboratory finding of an anion and osmolal gap metabolic acidosis. The two substances can be identified by toxicological screening, however, and each produces a characteristic clinical course. Ethylene glycol initially causes inebriation followed by progressive obtundation, seizures, and cardiopulmonary and renal complications (15). In contrast, individuals who consume methanol become acutely intoxicated and then, after a latent period, develop visual disturbances, nausea, vomiting, and mental status alterations. With large ingestions, patients can progress to seizures and coma. Pancreatitis and hemorrhagic gastritis may develop in addition to the central nervous system effects (3).

Methanol is metabolized to formaldehyde and formic acid. The initial intoxication is due to the methanol itself, but this alone does not cause lasting neurologic sequelae. The latent period corresponds to the time period in which the methanol is converted to formic acid, the metabolite responsible for the acidosis and the toxic effects (16).

Optic atrophy related to loss of myelin in the optic nerves is perhaps the best known neuropathologic change after methanol poisoning (17). In the cerebrum, the typical neuropathologic finding is necrosis of the putamen and subcortical white matter, which can be hemorrhagic. The most peripheral components of hemispheric white matter, the short association arcuate or U fibers, are spared. Cortical, cerebellar, and brainstem lesions have also been described (18).

These changes can be seen with CT and MRI (2,4-14). We are unaware, however, of prior descriptions of these lesions enhancing with contrast agent. Reviewing the literature, we found six reports of patients with methanol poisoning who received contrast agent, all with CT scanning, and none of them showed enhancement (4-6,8-10). In the reported cases, the CT scans were performed within 10 days or >2 months after the time of ingestion. Our patient demonstrated enhancement 12 days after ingestion.

Contrast enhancement of methanol-associated lesions in the subacute phase of poisoning is consistent with what is known about the pathophysiology of this alcohol. Methanol, metabolized to formic acid, causes toxicity by inhibiting the cytochrome oxidase complex of the mitochondrial respiratory chain. This blockade of aerobic metabolism has been called "histotoxic hypoxia" and may lead to cell death (16). Metabolic dysfunction and cellular injury involving endothelial cells of the central nervous system are a potential mechanism explaining the disruption of the blood-brain barrier represented by contrast enhancement. The rapidity and extent of the brain injury vary between patients and are related to the rate of conversion of methanol to formic acid, folic acid stores, and the concomitant consumption of ethanol. Some patients do not survive long enough to manifest neuroimaging changes. Because of the clinical variability of methanol poisoning, the

time period when contrast enhancement and other imaging changes are visible may be missed if imaging is performed too early or too late. This may be similar to the imaging of brain lesions in carbon monoxide poisoning and cerebral ischemic events. Two reports described contrast-enhancing lesions in the basal ganglia and dentate nucleus of the cerebellum on CT done 8 days (19) and 2 weeks (20) after carbon monoxide poisoning, while CT scans done 6 weeks did not show contrast enhancement (21). Carbon monoxide, like methanol, may cause tissue injury by interfering with mitochondrial function (22). Stroke may be another analogous situation. Contrast enhancement of brain infarction usually appears around 7 days after the event, peaks at 2-3 weeks, and then declines.
Enhancement is rare after 2 months (23).

The contrast-enhancing brain lesions in this case of methanol intoxication are reminiscent of lesions due to carbon monoxide poisoning and stroke that enhance only after a latent period. This finding is also consistent with the postulated mechanism of metabolic inhibition leading to cellular dysfunction and cell death with subsequent breakdown of the blood-brain barrier.

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