

IMAGES IN CLINICAL TOXICOLOGY

## Methanol poisoning with necrosis corpus callosum

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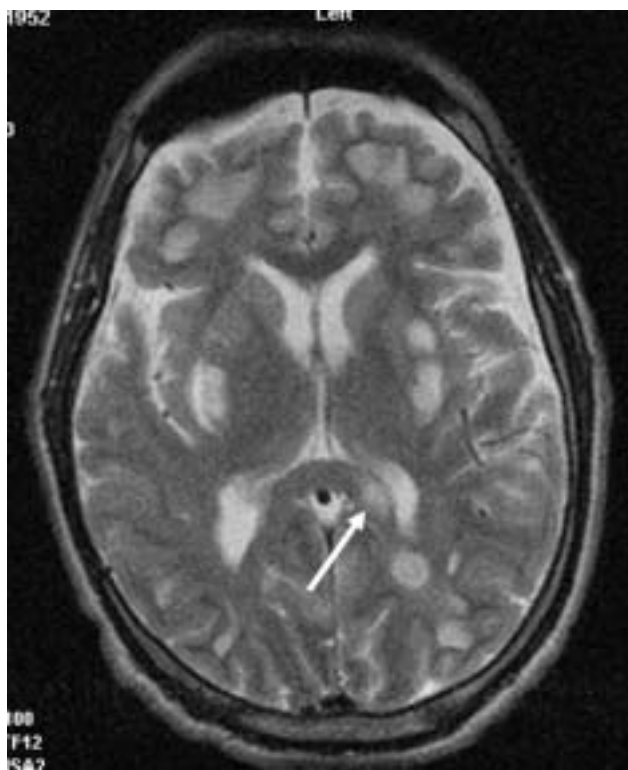
Methanol poisoning is a potentially serious event that may culminate in severe metabolic disturbances, permanent neurological dysfunction, blindness, and death. According to information from the Drug and Poison Information Center (DPIC) in Turkey (1), from 1993 to 2002 (1), sixteen patients suffering from methanol toxicity (14.1%) died, 63 (55.8%) had complete recovery and 1 (0.9%) had irreversible visual problems. Interestingly, the source for methanol in many of these cases was methanol used for producing cheap “*eu de colognes*” in Turkey.

We report a case of methanol intoxication in which the initial CT scans appeared normal. An MRI performed at 8 days post ingestion showed corpus callosum lesions consistent with methanol intoxication and peripheral white matter lesions that spared a thin rim of subcortical white matter.

This patient, a 45 year-old-man, presented with weakness and “cloudy vision”. The pupils were noted to be fixed and dilated and the fundoscopic exam revealed retinal edema with hyperemia of the optic disc. The patient was diagnosed with metabolic acidosis (blood pH 6.8) and a toxicology screening revealed a methanol level of 37 mg/dl.

Endotracheal intubation was performed for airway protection. Treatment included folic acid supplementation and HCO<sub>3</sub> and ethanol infusion with correction of the acidosis and resolution of the anion gap over the next 24 h. Following extubation, the patient continued to complain of blindness. An MRI obtained at that time demonstrated necrosis of the putamen, as well as, cerebral white matter with involvement of the corpus callosum, a previously unreported group of MRI findings in the face of methanol poisoning (Fig 1).

The most characteristic pathologic findings after methanol intoxication are discrete regions of necrosis involving the



**Fig. 1.** Axial FSE T2 image demonstrating necrosis of bilateral putamina, cerebral white matter and involvement of left side of the splenium of corpus callosum.

putamen, which may also have varying degrees of hemorrhage. These characteristic changes can be seen if the patient survives for longer than 24 hours (2). The mechanism responsible for putaminal necrosis remains unknown. It has been postulated that the necrosis results from a decreased blood flow through the basal veins of Rosenthal. However, our patient did not experience hypotension during this period, thus making decreased venous flow less likely. Alternatively, putamen necrosis may occur as a direct toxic effect of formic acid accumulating in the putamen compared to other areas of the brain (3). Discrete regions of necrosis have also been

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described in the white matter of patients surviving longer than several days.

Despite the profound metabolic acidosis and corpus callosum necrosis, this patient survived this critical illness. At follow-up one month after discharge, his cognitive function progressively improved, however the bilateral optic atrophy and blindness were permanent. Follow-up MRI showed contrast enhancement of the lesions as well as cortical involvement.

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