MRI of the Brain in Methanol Intoxication*

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Introduction
Poisoning with methanol is rare but leads to severe multiorgan damage. Methanol is metabolized in the liver to formaldehyde and then to formic acid [1]. The resulting metabolic acidosis can cause irreversible damage and death. The spectrum of neurologic symptoms ranges from inebriation and drowsiness to seizures and coma [2–6]. The damage to the optic nerves leads to visual disturbances or even permanent blindness [6–9].

Bilateral necroses of the putamen and hemispheric white matter are the most common findings in MRI and CT [1, 2, 4, 5].

Methanol can be found in many substances of daily use like various solvents, antifreeze, varnish and as a common additive to gasoline. Intoxication is possible, accidentally or with suicidal intent by oral ingestion, inhalation or absorption through the skin [3–5, 10, 11].

This article reports on a patient with cerebral lesions due to methanol intoxication.

Case Report
A 43-year-old man was admitted as an emergency for MRI because he was suspected of thrombosis of the basilar artery. The day before, his wife had noticed progressive drowsiness. At first, she was able to awaken him. Since his condition worsened, he was brought to the regional hospital the next morning. At presentation there, his pupils were mydriatic, without any signs of reaction to light. He was restless, but pathologic reflexes, meningism and elevated temperatures were not observed. 1 week earlier, he had had an influenza virus infection. The month before, he had been to Egypt on vacation. In the laboratory, only a leukocytosis was detected. An initial CT scan showed no intracranial abnormalities. In consequence of progressive respiratory insufficiency, but without cessation of breathing, he had been intubated. No abuse of medicaments or drugs was known. Because of these symptoms, an MRI was needed and therefore the patient was sent to our hospital.

After the MRI examination, the patient had first been admitted to the neurologic clinic and in the evening of the same day to the intensive care unit of our hospital because of renal failure and electrolyte imbalances. The low pH of 6.7 was a sign of severe metabolic acidosis. The base excess could not be measured. The potassium level was 7.2 mmol/l. Continuous venovenous hemodialysis (CVVHD) for detoxication and bicarbonate replacement therapy to improve metabolic acidosis were started. The body temperature had fallen to 34.5 °C. C-reactive protein and cerebrospinal fluid (CSF) were inconspicuous. Without any sedation, the patient was comatose, his pupils now were miotic, still without reaction to light. It was not possible to evoke reflexes of the brain stem; there were no signs of hemiparesis.

Only after admission to the intensive care unit was it found out by asking his wife that he had assembled tiles in their home the day before. She discounted attempted suicide. In his job, he worked with metals, galvanization, alkalines, and solvents. Serious former illnesses could not be ascertained.

Laboratory findings were as follows: serum methanol level 1,200 mg/l, massive elevations of liver enzymes (GOT = AST [aspartate transaminase] > 5,000 U/l, GPT = ALT [alanine aminotransferase] > 900 U/l, γ-GT [γ-glutamyltransferase] 250 U/l), total bilirubin 12.4 mg/dl,
LDH [lactate dehydrogenase] > 2,000 U/l, AP [alkaline phosphatase] > 500 U/l. The creatinine level increased to 5.4 mg/dl. Rhabdomyolysis developed with highly increased levels for CK (creatine kinase, > 140,000 U/l!), CK-MB (> 2,600 U/l), and myoglobin (> 1,400 µg/l).

A suspicion of barbiturate poisoning could not be verified.

The patient died 2 weeks after admission to our hospital.

The MRI (1.5 T) showed bilateral DWI (diffusion-weighted imaging) hyperintensities in the inferior parts of the cerebellar hemispheres (Figure 1a) and in the posterior part of the medulla oblongata (Figures 1a and 1d), in the tegmentum of midbrain and pons (Figure 1d). These signal changes were hypointense in the apparent diffusion coefficient (ADC; Figures 1b and 1e) and presented less clearly in the T2-weighted (T2w) images (Figures 1c and 1f). Corresponding bilateral signal changes were also found supratentorially with accentuation in the putamina including the head of the caudate nucleus (i.e., corpus striatum = striate body) and appeared as small spots in both thalami (Figures 1f and 2a to 2c). Nearly the whole white matter showed significant, but less expressive hyperintensity in DWI and hypointensity in ADC (Figures 1e and 2b). The T1-weighted (T1w) images without contrast media only revealed little bilateral hypointensities in the striate body (Figure 2d). The cortex was presented in the T2w sequences (FLAIR [fluid-attenuated inversion recovery], T2-TSE) with an overaccentuation (Figures 1f and 2c),