Neuroradiology (1997) 39: 192–194

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Optic atrophy and cerebral infarcts caused by methanol intoxication: MRI

Received: 2 February 1996
Accepted: 1 March 1996

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Abstract We present the MRI findings of cerebral and optic pathway damage in the acute and subacute stages of methanol intoxication. In the acute stage, CT and MRI showed bilateral haemorrhagic necrosis of the corpus striatum and infarcts in the anterior and middle cerebral arterial territories. MRI in the subacute stage demonstrated atrophy of the optic chiasm and prechiasmatic optic nerves in addition to the cerebral infarcts. The patient survived, with total blindness.

Key words Brain · Toxic encephalopathy · Methanol intoxication · Magnetic resonance imaging

Introduction

Patients with methanol intoxication usually present with acute encephalopathy and blindness as a result of selective toxicity to the optic nerve and necrosis of the basal ganglia [1–3]. We report a patient with methanol intoxication whose MRI showed findings not previously reported.

Case report

A 20-year-old man ingested approximately 0.6 l of a methanol-containing beverage in a suicide attempt. Thirty-six hours before admission, he suffered from symptoms including severe nausea, vomiting, blurred vision and eventually became unconscious. On admission, he was comatose, with generalised hypotonia, nonreactive pupils and hyperventilation. Fundoscopy disclosed oedema of the optic discs. The blood pressure was 120/60 mm Hg, pulse rate 100/min, respiration rate 30/min, and temperature 36.8 °C. A chest radiograph, urinalysis, haemoglobin value, white blood cell count, electrolytes, and blood glucose concentrations were normal. Abnormal laboratory findings included an arterial blood pH of 7.15 and carbon dioxide tension of 30 mm Hg; a serum bicarbonate of 12 meq/l, and an anion gap of 37 mEq/l. The blood methanol on admission was 98 mg/ml. The patient was treated with intravenous sodium bicarbonate and ethanol. He regained consciousness and reacted to verbal commands after 24 h in hospital. Repeat examination showed bilateral blindness and extrapyramidal dysfunction. CT 4 h after admission demonstrated bilateral, symmetrical low density in the caudate nuclei, putamen, median cortex of frontal lobes and insular cortex. MRI performed immediately after the CT showed haemorrhage in the left putamen (Fig. 1) and high signal bilaterally in the corpus striatum, and medial frontal and insular cortex on T2-weighted images.

One month later the patient remained completely blind with optic atrophy on fundoscopy and had a moderate bilateral extrapyramidal syndrome. MRI 32 days after admission showed atrophy of the optic chiasm and prechiasmatic optic nerves in addition to high-signal blood products in both putamina. There was contrast enhancement of the cortex of the medial frontal lobes and insula (Fig. 2).

Discussion

Formic acid, the metabolite of methyl alcohol, is significant toxic to the central nervous system in persons who ingest methylated spirits. Symptoms of methanol poisoning are usually delayed for 12–18 h, this latent period

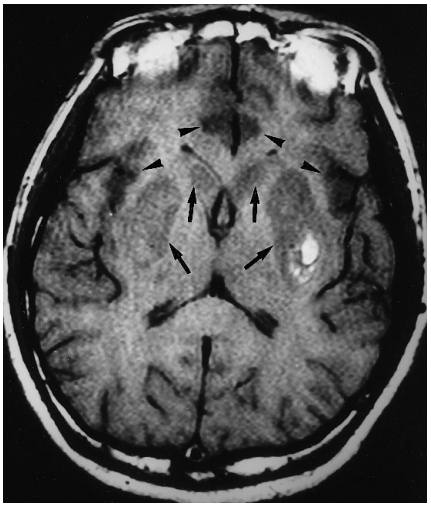


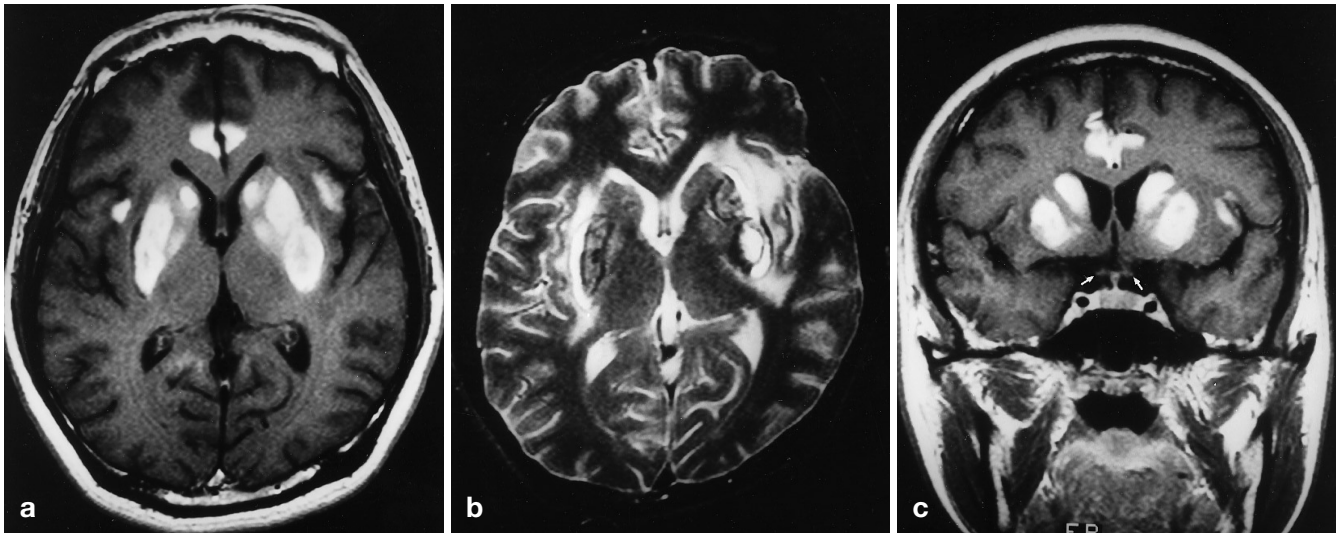
Fig. 1 T1-weighted MRI shows bilaterally symmetrical low signal lesions in the basal ganglia *(black arrows)*, medial cortex of frontal lobes and insular cortex *(black arrowheads)*. There is also central high signal, suggestive of haemorrhage, in the left putamen

corresponding to the time required for methanol to be oxidised to the more toxic formate. Drowsiness, headache, nausea, vomiting, abdominal pain and blurring of vision are the usual presenting symptoms and may be followed by blindness, coma, and cardiac arrest if a large amount has been comsumed [2, 4]. The diagnosis is based on the presence of a high serum methanol level.

Autopsies in patients who succumb to methanol poisoning (lethal dose 60–240 ml of 40 % methanol) have shown typical haemorrhagic necrosis of both putamina, white matter oedema, retrolaminar demyelinating optic neuropathy and optic atrophy [1–5]. Bilateral necrosis of the putamen, often recognised radiographically, is characteristic of severe methanol intoxication [2, 4–6]. This part of the basal ganglia is particularly at risk to various pathological processes, because of its high metabolic demand and because it lies in the boundary zones of vascular perfusion. Accumulation of higher concentrations of methanol in these areas has also been suggested [2]. What is interesting in our case is that not only the corpus striatum but also the cortex of the medial frontal lobes and insula showed MRI changes consistent with infarcts. This coincides with the hypothesis that the selective neurotoxicity of methanol is due to anoxia, as a result of inhibition of cytochrome oxidase by formic acid [7]. The same mechanism is also thought to be the cause of the optic neuropathy, involving particularly the retrolaminar segment of the optic nerve [1]. The inital MRI of our patient did not reveal any obvious abnormality in the optic pathways. MRI in the subacute stage did show significant atrophy of the optic chiasm and prechiasmatic optic nerves. The intraorbital optic nerves were not well delineated, although follow-up fundoscopy showed atrophy of the optic discs.

Bilateral putaminal lesions can be found on MRI in a variety of conditions including Leigh's syndrome [8, 9], Wilson's disease [10], hypoxic-ischaemic insults [11], encephalitis and certain types of metabolic disorders. We suggest that the combination of haemorrhagic necrosis of the corpus striatum, along with symmetrical cortical infarcts and optic atrophy, may be unique to subacute methanol intoxication.

Fig. 2 **a** Follow-up MRI shows contrast enhancement of the infarcted cortex and basal ganglia. **b** On T2 weighting areas of low signal in the basal ganglia represent subacute haemorrhage. **c** Coronal contrast-enhanced image demonstrates marked atrophy of the optic chiasm *(arrows)*



References

1. Sharpe JA, Hostovsky M, Bilbao JM, Rewcastle NB (1982) Methanol optic neuropathy: a histopathological study. Neurology 32: 1093–1100
2. McLean DR, Jacobs H, Mielks BW (1980) Methanol poisoning: a clinical and pathological study. Ann Neurol 8: 161–167
3. Glazer M, Dross P (1993) Necrosis of the putamen caused by methanol intoxication: MR findings. AJR 160: 1105–1106
4. Phang PT, Passerini L, Mielke B, Berendt R, King EG (1988) Brain hemorrhage associated with methanol poisoning. Crit Care Med 16: 137–140
5. Pelletier J, Habib MH, Khalil R, Salamon G, Bartoli D, Jean P (1992) Putaminal necrosis after methanol intoxication [letter]. J Neurol Neurosurg Psychiatry 55: 234–235
6. Koopmans RA, Li DKB, Paty DW (1988) Basal ganglia lesions in methanol poisoning: MR appearance. J Comput Assist Tomogr 12: 168–169
7. Nicholls P (1975) Formate as an inhibitor of cytochrome oxidase. Biochem Biophys Res Commun 67: 610–616
8. Davis PC, Hoffman JC, Braun IF, et al (1987) MR of Leigh's disease (subacute necrotizing encephalomyelopathy). AJNR 8: 71–75
9. Medina L, Chi TL, De Vivo DC, et al (1980) MR findings in patients with subacute necrotizing encephalomyelopathy (Leigh syndrome): correlation with biochemical defect. AJR 154: 1269–1274
10. Nazer H, Brismar J, Al-Kawi MZ, et al (1993) Magnetic resonance imaging of the brain in Wilsons's disease. Neuroradiology 35: 130–133
11. Barkovich AJ (1992) MR and CT evaluation of profound neonatal and infantile asphyxia. AJNR 13: 959–972