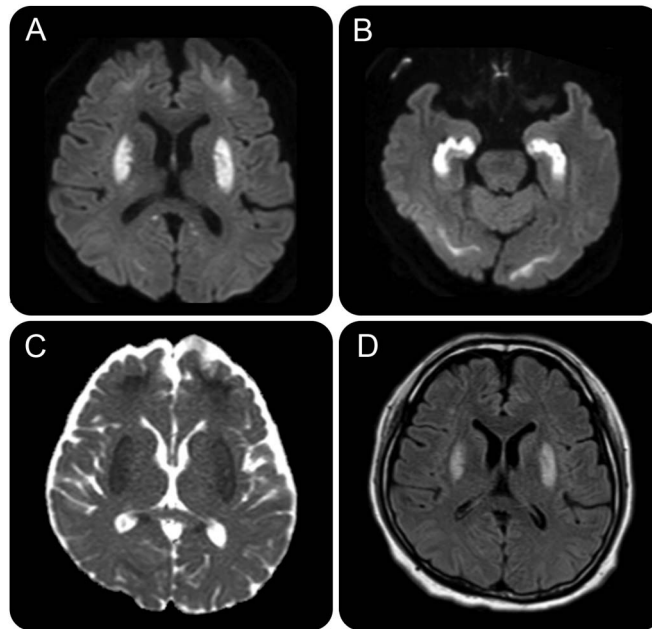


# Acute hippocampal and chronic diffuse white matter involvement in severe methanol intoxication

**Figure 1** MRI on admission



Representative images of brain MRI on admission, including diffusion-weighted imaging (A, B), the apparent diffusion coefficient map (C), and fluid-attenuated inversion recovery image (D), show hyperintense lesions in the bilateral putamen, hippocampi, and subcortical white matter.

A 42-year-old man presented with coma from methanol intoxication (178 mg/dL; its metabolite, formic acid, 860  $\mu\text{g}/\text{mL}$ ). Continuous hemodiafiltration improved his disturbance of consciousness, but he subsequently developed cognitive dysfunction and parkinsonism, which were finally alleviated by rehabilitation and amantadine therapy. Brain MRI revealed acute basal ganglionic and hippocampal lesions and chronic developmental white matter lesions (figures 1 and 2).

Formic acid disrupts mitochondria, resulting in cytotoxic hypoxia and oxygen free radical production, which might cause acute putaminal and hippocampal injuries.<sup>1</sup> Oxygen free radicals contribute to the development of white matter lesions.<sup>2</sup> In our case, toxic encephalopathy likely resulted from diverse mechanisms.

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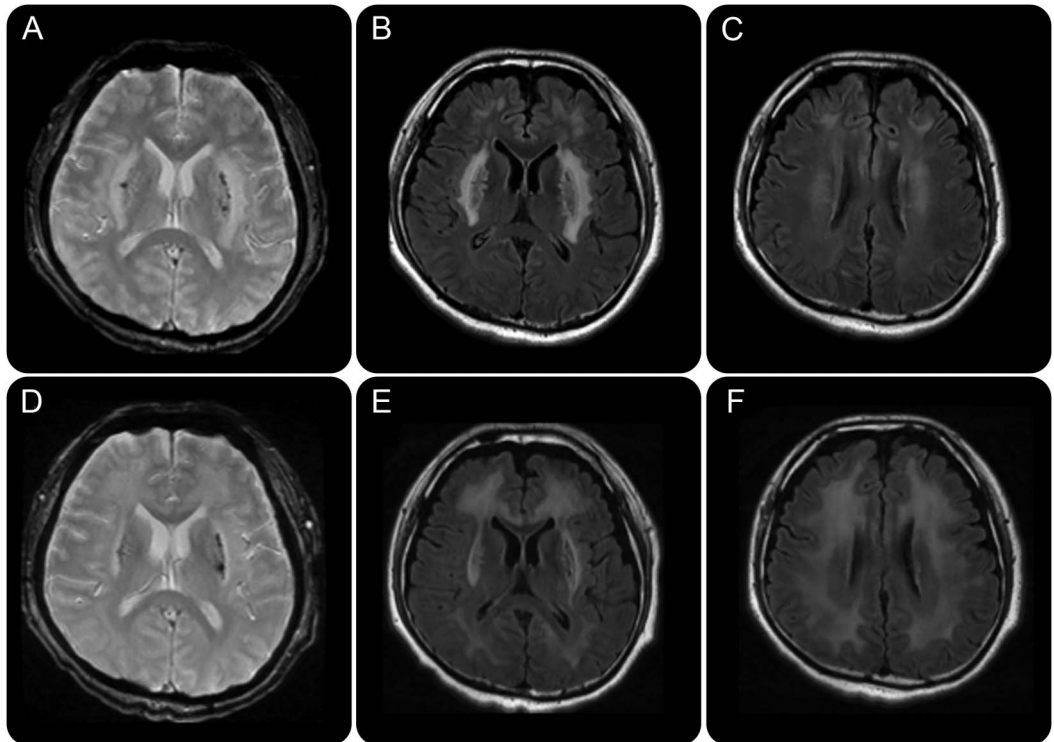
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Figure 2 Repeat MRI



Representative images of repeat MRI on days 14 (A–C) and 35 (D–F) after admission; T2\*-weighted images (A, C) show scattered microbleeds in the bilateral putamen while fluid-attenuated inversion recovery images (B, C, E, F) show putaminal lesions and chronic development of vast white matter lesions.

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