Acknowledgment

The authors thank J.J. Bot and M.A.A. van Walderveen for referring patients, and B. Young for valuable help with preparing the manuscript.

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Figure. CT scan at day 3 (A); MRI at day 9 (B); and postmortem specimen at day 10 (C). All three show diffuse subcortical and putamenal necrosis with right putamenal hemorrhage. The sparing of centrum semiovale is a characteristic feature.

Severe white matter injury and bilateral putamenal necrosis with hemorrhage in methanol ingestion

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A 48-year-old white man ingested an unknown quantity of methanol, resulting in severe metabolic acidosis. He was deeply comatose at 72 hours and died at 10 days.

Nonenhanced computed tomography of the brain showed low attenuation, primarily in the subcortical white matter of both cerebral hemispheres, as well as abnormal hypodensity of the putamen. Additional focus of hemorrhage involved the right putamen (figure A). MRI flair sequence demonstrated an increased signal, especially in the subcortical white matter of both cerebral hemispheres. The brighter signal involving the putamen represents

edema, as well as coexistent blood products on the right (figure B). This was consistent with primarily subcortical white matter infarction and bilateral putamenal necrosis with right-sided hemorrhage, documented on postmortem examination (figure C).

The presence of subcortical white matter (with relative sparing of centrum semiovale) and putamenal injury is typical of severe methanol toxicity.¹ This is likely related to methanol metabolism to formates, hypoxemia, severe acidosis, and coexistent circulatory depression.²

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Neurology 2000;54:1239
DOI 10.1212/WNL.54.6.1239

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