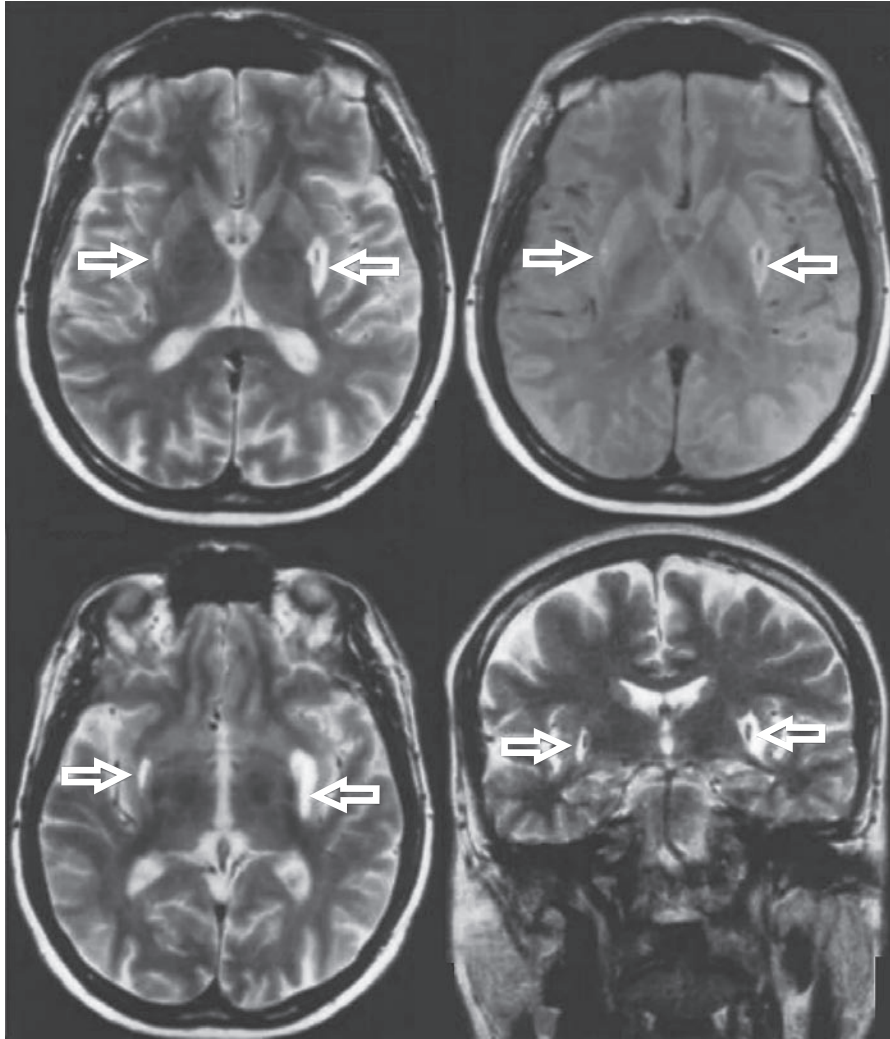


## IMAGES IN CLINICAL MEDICINE

## Putaminal Necrosis



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**A** 51-YEAR-OLD MAN PRESENTED WITH ACUTE-ONSET BILATERAL BLINDNESS THAT HAD DEVELOPED DURING the previous 4 days. He reported having consumed cheap, unbranded whisky in a bout of binge drinking. This episode was followed by severe breathlessness that lasted for 48 hours. He then noticed dimness in his vision, with pain at the back of both eyes that was exacerbated by eye movement. On physical examination, visual acuity was reduced to perception of light, and the pupils were bilaterally dilated, with afferent pupillary defect. Eye movements were painful. The optic disks were edematous bilaterally, and the margins were blurred, with pallor extending into the peripapillary regions of the retinal nerve fiber. The neurologic examination was otherwise normal. There was metabolic acidosis; no methanol was detected in the serum.

Four weeks later, T<sub>2</sub>-weighted magnetic resonance imaging of the brain showed bilateral, symmetric hyperintense putaminal lesions, suggesting hemorrhagic necrosis (arrows). Direct toxicity of formic acid (an end product of methanol metabolism), ischemic injury, and acidosis are postulated mechanisms of putaminal injury. In this case, survival was probably related to the low amount of methanol ingested and its dilution with ethanol, which may have had a protective effect by inhibiting the metabolism of methanol. At follow-up 2 years later, the patient's vision had not improved.

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