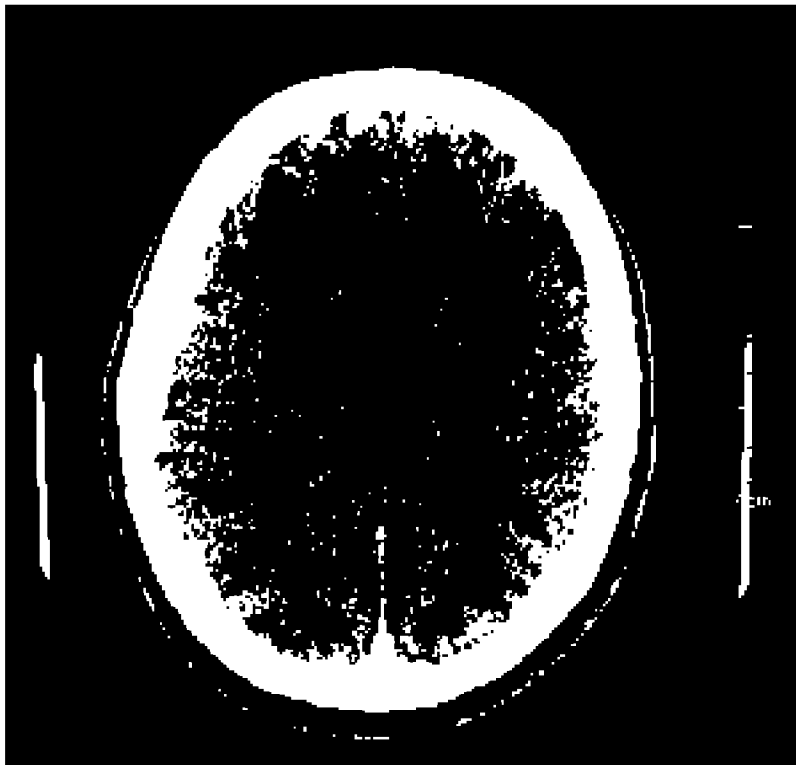


IMAGES IN CLINICAL TOXICOLOGY

Methanol Neurotoxicity

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A 46-year-old male without significant past medical history presented to our Emergency Department in an unconscious state with bilateral ocular leftward deviation. The patient was afebrile with stable vital signs and SpO₂ of 98% on room air. A fingerstick blood sugar was 112. Intravenous access was established and the patient was intubated. An emergent head CT was normal. Post-intubation arterial blood gas revealed a pH of 6.93, pCO₂ of 30 and PAO₂ of 654. An anion gap of 36 and an osmolar gap of 138 was ascertained. An ethanol level was zero and salicylates were 5.6 mg/dL. A toxic alcohol panel was sent, and aggressive treatment for methanol and ethylene

glycol ingestion was initiated, including intravenous fomepizole, thiamine, folic acid, and, eventually, hemodialysis. Approximately 6 hours after presentation, the serum methanol level was reported as 570 mg/dL. Over the next 36 hours, the patient had intermittent generalized seizure activity and, despite aggressive care, had no improvement in mental status. A repeat head CT obtained 36 hours after presentation, demonstrated bilateral putamen infarcts as well as subcortical white matter destruction (see image).

Necrosis of the putamen is the typical neuropathological finding in methanol poisoning, that may be demonstrated with neuroimaging. (1) Formic acid, the toxic metabolite of methanol, inhibits cytochrome oxidase and oxidative metabolism, leading to Na⁺/K⁺ ATPase pump failure, cerebral edema and cell death. The putamen appears to be more susceptible to this

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histotoxic hypoxia than other regions in the brain, although it is unclear as to why. One theory suggests that breakdown of the blood brain barrier results in higher levels of formic acid diffusion into the putamen. (2) Other theories postulate that a combination of high local concentrations of formic acid plus poor venous drainage result in increased histiotoxic hypoxia. In addition, the putamen may manifest higher oxygen and glucose consumption as compared with other areas of the brain thereby making this tissue more susceptible to histiotoxic hypoxia.(3)

Putamen infarcts, while suggestive of, are not pathognomonic for methanol toxicity. The differential diagnosis for this neuroradiologic finding includes Leigh's disease, Wilson's disease, Kearns-Sayre syndrome, Leber's optic atrophy, carbon monoxide poisoning as well as hypoxic and ischemic injuries. (4,5) Our patient, however, had no known episodes of hypoxia,

no exposure to carbon monoxide and a significant methanol intoxication. The brain imaging study presented correlates with a severe methanol poisoning.

REFERENCES

1. Anderson C, et al. MRI enhancing brain lesions in methanol intoxication. *Journal of Computer Assisted Tomography*. Sept/Oct 1997; Vol 21(5):834-836.
2. Onder F, et al. Acute blindness and putamenal necrosis in methanol intoxication. *International Ophthalmology*, 1999; 22:81-84.
3. Barceloux DG, et al. American academy of clinical toxicology practice guidelines on the treatment of methanol poisoning. *Clinical Toxicology*, 2002; 40(4):415-446.
4. Kuteifan K, et al. Necrosis and haemorrhage of the putamen in methanol poisoning shown on MRI. *Neuroradiology*, 1998; 40:158-160.
5. Penney Gaul H, et al. MR findings in methanol intoxication. *AJNR: American Journal of Neuroradiology*, Oct. 1995; 16(9):1783-6.

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