Acute bilateral putaminal haemorrhagic necrosis in methanol poisoning

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DESCRIPTION
A 30-year-old man without significant past illness was admitted to the neurology intensive care unit with a 2-day history of altered level of consciousness, vomiting and vision loss. History revealed that he had consumed unknown amounts of unbranded alcohol 4 days earlier. The clinical examination revealed a Glasgow Coma Scale of E2V2M4, bilaterally dilated pupils, optic disc oedema, normal deep tendon reflexes and flexor plantar responses bilaterally. Arterial blood gas analysis showed severe metabolic acidosis (pH 7.11). Further evaluation revealed high anion (40 mEq/L; normal 8–16 mEq/L) and osmolar gap (31 mOsm/L; normal <10 mOsm/L water). Brain CT scan without contrast (figure 1) showed bilateral putamen hypodensities. Based on the history, clinical findings and investigations, a diagnosis of methanol poisoning was made. The patient underwent one cycle of haemodialysis, following which the level of consciousness improved. MRI on day 11 after alcohol consumption revealed bilateral putaminal lesions (figure 2A–C). These lesions were haemorrhagic as evidenced by their hyperintense appearance on T1 (figure 2A) and blooming on gradient echo sequence (figure 2D). The patient’s sensorium recovered completely on discharge, but his vision did not improve. His vision was finger counting at 1 ft at the time of discharge. Methanol is metabolised to formic acid which is responsible for most of its toxic manifestations such as vision loss, abdominal pain, vomiting, altered sensorium, seizures and coma. The very few aetiologies that have been described to cause haemorrhagic necrosis of the putamen include methanol ingestion, carbon monoxide poisoning, cyanide poisoning, Leigh’s disease and deep venous sinus thrombosis.12

Learning points

▸ MRI of the brain can assist in the diagnosis of methanol poisoning due to its characteristic findings.
▸ The combination of visual loss, acidosis and bilateral putaminal haemorrhagic necrosis on imaging is helpful in making a diagnosis of methanol poisoning as a history of methanol ingestion may not always be forthcoming.

Figure 1  Non-contrast CT image of the brain showing bilateral hypodensity of the putamen.
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Competing interests None.

Patient consent Obtained.

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Figure 2 Brain MRIs without contrast showing bilateral hyperintense putaminal lesions in: (A) sagittal T1-weighted sequence; (B) coronal T2-weighted sequence; and (C) axial fluid-attenuated inversion-recovery sequence. (D) Gradient-echo sequence shows bilateral blooming in the putaminal region suggestive of haemorrhages.

REFERENCES
