

Clinical Image

Methanol Intoxication Causing Bilateral Putaminal Necrosis

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A 30-year-old lady without significant past medical illness was admitted to an ICU in Dhaka with the complaints of altered level of consciousness and blurring of vision for 1 day. She had no history of fever, headache, vomiting, convulsion, head trauma. History revealed that she had consumed unknown amounts of unbranded alcohol 2 days earlier.

Clinical examination revealed a Glasgow Coma Scale score of E1V1M4, bilaterally equally dilated (6 mm) pupils, normal deep tendon reflexes and bilateral flexor plantar responses. Immediate arterial blood gas analysis showed severe metabolic acidosis (pH 6.96, pCO₂ 27 mm Hg, HCO₃⁻ 6.3 mEq/L). A non-contrast CT scan of head done 2 hours after admission which revealed bilateral hypodensities in the putamen (Figure 1). Based on these, with a presumed diagnosis of putamen necrosis secondary to methanol intoxication, bicarbonate and ethanol were administered. Immediately she received one session of haemodialysis following which her level of consciousness improved. Eventually a non-contrast MRI of brain done 4 days after methanol intoxication, which also revealed bilateral putaminal necrosis (Figure 2A-D).

6 days later, the patient was transferred out of ICU in stable condition. At the time of discharge, her sensorium recovered completely, but her vision was finger counting at 1 ft.



Figure 1: Non-contrast CT brain axial image showing bilateral putaminal hypodensity.

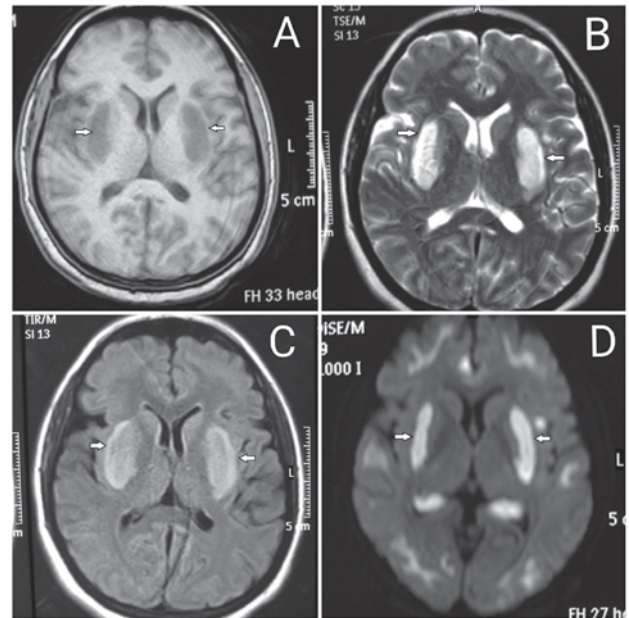


Figure 2: Brain MR Image without contrast showing bilateral symmetrical changes in the putamen. Hypointensity in T1W axial image (2A), hyperintense signal changes in T2W axial image (2B), FLAIR axial image (2C) & diffusion restriction in DW axial images (2D).

Discussion:

Methanol is a clear, colorless, highly toxic liquid with a smell and taste similar to alcohol. It is readily available as a component of many household products like antifreeze, paint removers, windshield washer fluid, various solvents and cleaners and gasoline mixtures^{1,3,5,6}. It has also been used in fraudulent adulteration of alcoholic drinks⁶. Accidental or intentional ingestion leads to severe metabolic acidosis with associated serious neurological manifestations. Signs and symptoms of toxicity include nausea, vomiting, headache, dizziness and visual disturbances ranging from blurred vision to permanent blindness. As the metabolic acidosis worsens, respiratory failure and coma ensue^{1,5}. Symptoms are usually observed 12-24 hour after methanol consumption. This latent period corresponds to the time period in which methanol is metabolized by alcohol dehydrogenase into formaldehyde and then to formic acid. It is the metabolite that lead to the toxic manifestations of methanol poisoning^{4,6}.

History, neuro-ophthalmological symptoms, findings of metabolic acidosis with a high anion and osmolar gap are the diagnostic basis of methanol poisoning and high serum methanol levels further support the diagnosis⁵.

Neuroimaging is characteristic and diagnostic. Both CT and

MRI give similar results but the latter yields better anatomical detail. Bilateral necrosis of the putamen with or without hemorrhage is the most characteristic radiological feature. Additional brain lesions including necrosis of the subcortical white matter, cerebellar cortical lesions, bilateral tegmental necrosis, diffuse cerebral edema, subarachnoid hemorrhage, cerebral hemorrhage and optic nerve necrosis have been described occasionally^{1,2,5,6}.

Differential diagnosis of bilateral putamen necrosis includes Wilson's disease, hypoxic-ischemic insults, encephalitis, Leigh disease, Kearns-Sayre syndrome, trichloroethane poisoning, striatal degeneration associated with Leber's optic atrophy, certain types of metabolic disorders, carbon monoxide inhalation and hypoxic-anoxic injuries such as near-drowning^{2,7}. History of recent consumption of adulterated or non-standard alcohol is the main clue to proper diagnosis. Predilection to putamen not caudate helps to differentiate methanol poisoning from other conditions that tends to involve mainly the caudate nucleus⁷.

Treatment consists of inhibition of metabolism of methanol to toxic formic acid by competitive inhibition of the enzyme alcohol dehydrogenase by ethyl alcohol or 4-methyl pyrazole (fomepizole), alkalization by administration of sodium bicarbonate, the use of cofactors such as folate and drug elimination (for example, haemodialysis)^{3,5}.

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