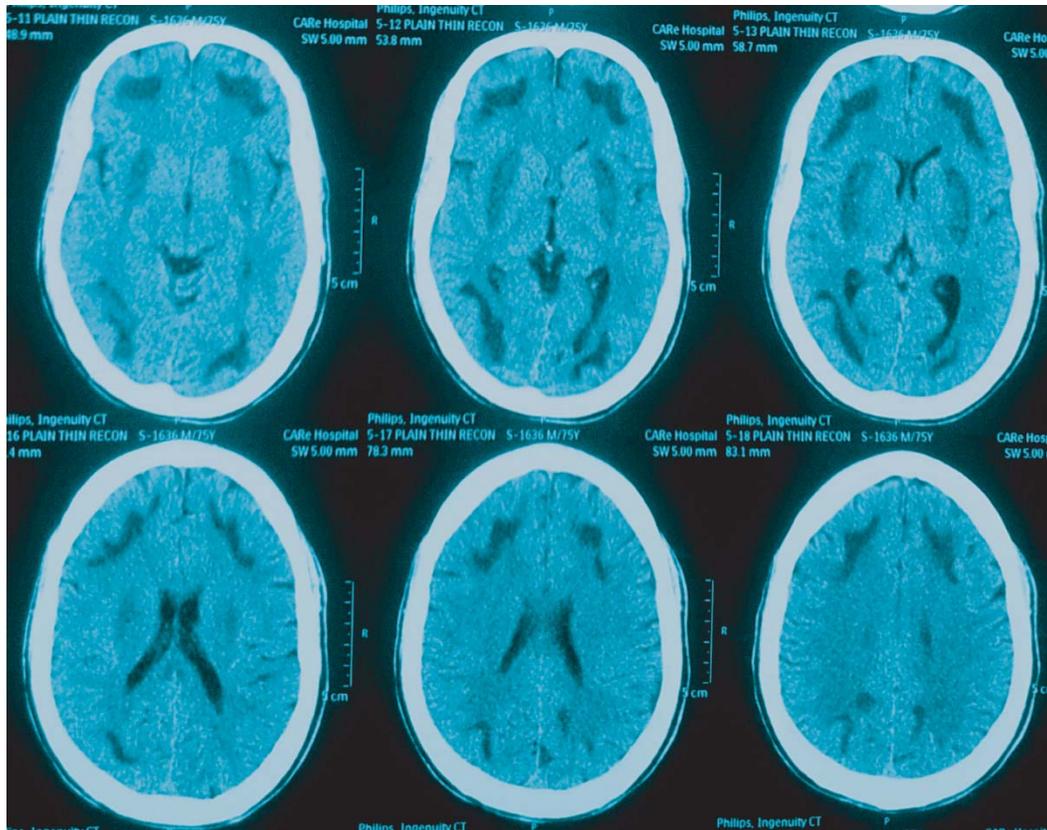


## Putaminal and White Matter Necrosis due to Methanol Intoxication



Head CT scans of a 65 year old man, after regaining consciousness level, taken 48 hours after admission into hospital

A 65-year-old man with history of chronic alcoholism developed acute vomiting and abdominal pain following a binge of country made liquor. He developed shortness of breath and progressive loss of consciousness on the next day. With these complaints he got admitted into an intensive care unit of Dhaka city 2 days after ingestion.

On admission, he was comatose with Glasgow Coma Score 8 (E4M3V1). He had deep shallow respiration with respiratory rate 35 per minute, hypotension (blood pressure 70/40 mmHg) and fundoscopic examination did not show papilloedema. Arterial

blood gas analysis showed metabolic acidosis with high anion gap (pH 7.07,  $\text{PCO}_2$  14 mmHg,  $\text{PO}_2$  62 mmHg,  $\text{HCO}_3^-$  4.1 mmol/L, BE -24 mmol/L). With a presumed diagnosis of methanol intoxication, he was treated with sodium bicarbonate, folic acid, high dose Vitamin B<sub>12</sub> and thiamine in injectable form and noradrenaline infusion for hypotension.

Three hours following sodium bicarbonate administration metabolic acidosis was corrected (pH 7.52,  $\text{PCO}_2$  29 mmHg,  $\text{PO}_2$  71 mmHg,  $\text{HCO}_3^-$  23.7 mmol/L, BE 1.9 mmol/L) without haemodialysis or giving ethanol; subsequently blood pressure norma-

lized and noradrenaline was gradually tapered off and stopped on next day. Consciousness level gradually improved. On next day of admission, he was found conscious, haemodynamically stable with loss of vision in both eyes. High dose steroid (Injection methylprednisolone) was started along with injection thiamine, vitamin B<sub>12</sub> and folic acid. His visual acuity allowed for visualizing hand motion only. Initial intraocular pressure was within normal limits in both eyes. Fundus examination showed optic disc swelling in both eyes. CT scans of the brain obtained 48 hours after admission into hospital showed symmetric areas of low attenuation in the peripheral white matter of both cerebral hemispheres bordered by the grey white junction, involving frontal, parietal, occipital and temporal lobes (Fig 1, small arrows). Symmetric hypointensities were also noted in both lentiform nuclei, centered within the putamen and extending into the external capsules (Fig 1, **bold** arrow).



Fig 1. CT scan showing symmetrical areas of low attenuation in the peripheral white matter (small arrows) and symmetrical hypointensities in both lentiform nuclei (**bold** arrow).

Methanol poisoning is due to accidental or suicidal intent, or adulteration of alcoholic beverages and leads to neurological sequelae of variable severity.<sup>1-3</sup> Visual loss is one common manifestation and is

thought to be due to myelinotoxic effect of methanol or its formic acid metabolite on the optic nerve.

The most characteristic brain CT finding consists of bilateral putaminal necrosis, other changes include white matter necrosis and subarachnoid haemorrhage; these changes occur as a consequence of toxemia and acidosis.<sup>1,3</sup>

However, certain other conditions must be kept in mind in the differential diagnoses, such as carbon monoxide intoxication,<sup>4</sup> striatonigral degeneration, hypoxic ischaemic encephalopathy and Wilson's disease.<sup>5</sup> Although bilateral involvement of the basal ganglia on CT scans is a characteristic finding of methanol intoxication, it is infrequently seen in clinical practice.

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