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 funduscopic examination did not show papilloedema. Arterial blood gas analysis showed metabolic acidosis with high anion gap (pH 7.07, PCO₂ 14 mmHg, PO₂ 62 mmHg, HCO₃⁻ 4.1 mmol/L, BE -24 mmol/L). With a presumed diagnosis of methanol intoxication, he was treated with sodium bicarbonate, folinic acid, high dose Vitamin B₁₂ and thiamine in injectable form and noradrenaline infusion for hypotension.

Three hours following sodium bicarbonate administration metabolic acidosis was corrected (pH 7.52, PCO₂ 29 mmHg, PO₂ 71 mmHg, HCO₃⁻ 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 B12 and folinic 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).

Methanol poisoning is due to accidental or suicidal intent, or adulteration of alcoholic beverages and leads to neurological sequelae of variable severity.\(^1\)\(^-\)\(^3\) 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 toxaemia and acidosis.\(^1\)\(^,\)\(^3\)

However, certain other conditions must be kept in mind in the differential diagnoses, such as carbon monoxide intoxication,\(^4\) striatonigral degeneration, hypoxic ischaemic encephalopathy and Wilson’s disease.\(^5\) 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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References