Methyl alcohol poisoning causing putamen necrosis

Sir,

Admissions due to methanol poisoning are common among our poverty-ridden population, mostly due to consumption of country liquor or exposure with toxic products like antifreeze agent etc.^[1] We are reporting a patient of Methanol poisoning subsequently developing Putamen necrosis.

A 50-year-old male patient presented to the emergency department with chief complaints of giddiness, vomiting, abdominal pain, and blurred vision, since 4 h. His history was negative except for consumption of country liquor, 6 h back. On examination, patient was irritable, in altered sensorium had labored breathing, heart rate was 70/min, noninvasive blood pressure (BP) was 84/50 mm Hg, respiratory rate was 40/min, and pupils were dilated and sluggishly reacting. Systemic examination was within normal limits. Arterial blood gas (ABG) analysis revealed severe high anion gap metabolic acidosis with co-exiting respiratory acidosis and mild hypoxemia. In view of the suggestive history, chief complaints, and the ABG reports, we considered it as a methanol poisoning, in the absence of definitive diagnostic facilities.

He was rehydrated with isotonic saline (1L intravenous [IV]) over 20 min and with continued rehydration his BP returned to 118/82 mm Hg within 30 min. Invasive positive pressure ventilation was started (synchronized intermittent mandatory ventilation), gastric lavage was done and ethyl alcohol (10%, 10 mg/kg, IV) was infused over 10 min and thereafter continued on a maintenance dose of 2 mL/kg/h IV, for the next 36 h. Midazolam (0.05 mg/kg/h)and fentanyl (2.0 μ g/kg/h) infusion was started. Soda-bicarbonate (100 ml IV) was given, and ABG samples were repeated every 2 hourly. Oral folic acid (1 mg/kg) was started and administered 4 hourly, for the next 7 days. On the follow-up, acidosis got corrected over 24 h, but patient remained unconscious, so magnetic resonance imaging was planned. It revealed bilateral basal ganglia hyper-intensity lesion, extensive edema over fronto-temporal region and right basal ganglia hemorrhage [Figure 1]. Thereafter, injection mannitol (20% 100 mL, three times daily), syrup glycerine (30 mL, 3 times daily) and tablet acetazolamide (one tab twice daily) were started to decrease the intracranial edema. Gradually, the patient regained full sensorium and was extubated on seventh Intensive Care Unit day. His cardio respiratory parameters were within normal limits but had blurred vision and residual motor weakness in all the limbs. Thereafter patient remained stable and was shifted to the respective ward after 14 days.

Country liquor contain many impurities and varying amount of methyl alcohol. After ingestion, methyl alcohol is metabolized by alcohol dehydrogenize to formaldehyde and subsequently to formic acid. Toxic effects of these metabolites usually manifest after 12–24 h of ingestion. Initial symptoms are nausea, vomiting,

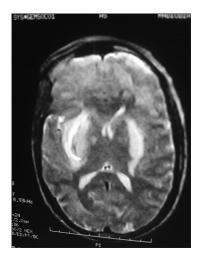


Figure 1: Computed tomography scan of head showing putamen necrosis

headache, dizziness and visual disturbances ranging from blurred vision to permanent blindness.^[2] Later manifestations include severe metabolic acidosis, brain edema/necrosis, and cerebral hemorrhage.^[3,4] Putaminal necrosis or hemorrhage could be due to these metabolic effects or as a result of direct toxicity from metabolic end products of methyl alcohol.^[5] Thus, persistent weakness or unconsciousness in a patient with methanol poisoning should always point toward the possibility of an intracranial complication while managing such cases.

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