

CT head findings in methanol poisoning

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Keywords: CT head, methanol poisoning



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A 73 years old male presented to the emergency department with the history of binge drinking of methanol 4 hours back. He initially complained of sudden onset of diminished vision, followed by drowsiness. Patient had high anion gap severe metabolic acidosis. Patient was electively intubated for low Glasgow Coma Scale (GCS) of 7 and underwent hemodialysis. Patient was also managed with ethanol and folinic acid.¹ Fomepizole was not used as it was not available. Plain CT scan of head performed on the next day revealed symmetrical hypodensities with interspersed areas of hyperdensities within bilateral lentiform nucleus. There were also confluent symmetrical hypodensities involving deep and subcortical white matter of bilateral frontal lobes (Fig. 1). Patient underwent tracheostomy for prolonged mechanical ventilation due to poor GCS and succumbed on day 18 of admission due to sepsis.



Fig. 1. The CT head findings show symmetrical hypodensities with interspersed areas of hyperdensities within bilateral lentiform nucleus and confluent symmetrical hypodensities involving deep/subcortical white matter of bilateral frontal lobes. The findings are typical of toxic leukoencephalopathy.

Bilateral putaminal necrosis is a characteristic feature of methanol toxicity.^{2,3} Putaminal necrosis and hemorrhage result from the direct toxic effects of methanol metabolites and metabolic acidosis in the basal ganglia. These pictures marked A and B both clearly demonstrate CT findings that are typical in a patient suffering from methanol intoxication. There are many theories that have been proposed to explain the specificity of the damage to the putamen, one of which suggests that it may be due to a high concentration of formic acid in the putamen partly due to poor venous drainage in the lenticular nucleus from the veins of Rosenthal, or inadequate arterial flow.^{3,4} These factors suggest there is a vulnerability of the putamen to histotoxic hypoxia due to formic acid accumulation.

Conflicts of Interest: None

Funding: None

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