Methanol poisoning

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A 22-year-old male presented with acute onset vomiting, diarrhea, abdominal pain and altered sensorium. He had a history of acute alcohol intake (locally made with solvent) a day prior to the presentation. Biochemical investigations revealed severe metabolic acidosis. Diagnosis of methanol poisoning was made on the basis of history and biochemical abnormality. Magnetic resonance imaging (MRI) done on fifth day revealed hemorrhagic putaminal necrosis, left occipital, corpus callosum and cerebellar cortical lesions [Figures 1-4].

Acute methanol intoxication can occur as accidental or suicidal ingestion. Patients present acutely with acute neurological, visual and gastrointestinal symptoms.[1]

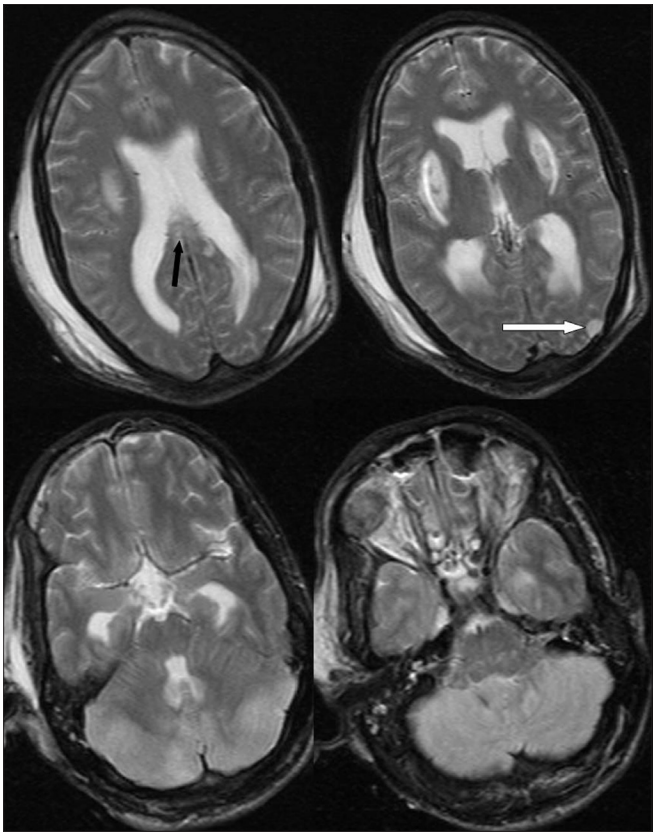

Figure 1: Axial T2 weighted images showing lesions in the putamina, left occipital cortex (white arrow), bilateral posterior cerebellar cortex, and in splenium of corpus callosum (black arrow)

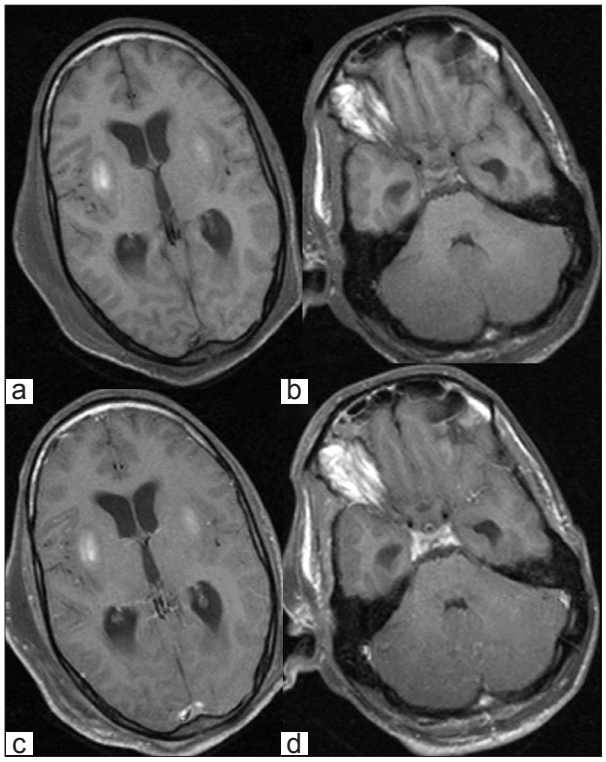

Figure 2: Axial T1 weighted pre-contrast (a and b) and post contrast (c and d) images showing non-enhancing hemorrhagic lesions in the putamina and hypointense lesions in bilateral posterior cerebellar cortex

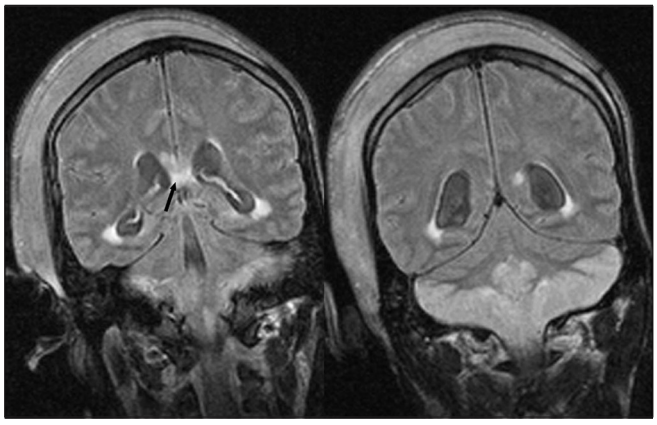

Figure 3: Coronal FLAIR images showing the lesions in the cerebellar cortex and in splenium of corpus callosum (black arrows)

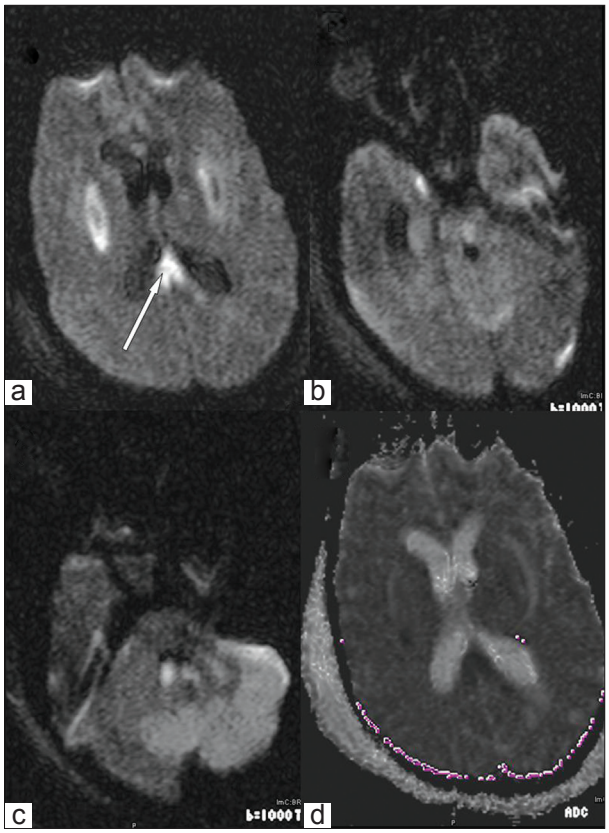

Figure 4: Diffusion weighted (a, b and c) and ADC (d) images showing diffusion restriction in the putamina, left occipital cortex, posterior cerebellar cortex and in splenium of corpus callosum (white arrow)

Neuroimaging helps in establishing the clinical diagnosis of the methanol poisoning. MRI findings in methanol poisoning are characteristic and include hemorrhagic putaminal necrosis (most characteristic), subcortical and deep white matter lesions, cerebral and cerebellar cortical lesions, and midbrain lesions.[1-4] Basal ganglia involvement is likely due to direct effect of metabolites of methanol as well as selective vulnerability of the basal ganglia to acidosis, as compared to rest of brain. Selective basal ganglia and white matter lesions are not specific to methanol intoxication and can be seen in hepatolenticular degeneration, carbon monoxide poisoning, hypoxic-ischemic insult and Leigh's disease.[4] Optic nerve lesions are considered to be due to myelinoclastic effect of formic acid and due to axonal loss.[3] Hemorrhage in methanol poisoning is seen in up to 14% of patients and diffusion restriction may be seen in the involved areas.[4] Index case showed almost entire spectrum of the MRI finding seen in the methanol poisoning.

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