

MRI findings in methanol intoxication: a report of three cases

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Abstract

Methanol is a highly toxic substance and acute methanol poisoning produces severe metabolic acidosis and serious neurological symptoms, including severe visual impairment, extrapyramidal signs and coma. We present three cases of accidental methanol intoxication and discuss the MRI findings.

Keywords: Magnetic Resonance Imaging, Central Nervous System, Methanol Intoxication.

Introduction

Methanol is a clear, colorless, highly toxic liquid. It is an uncommon but life threatening poison. It causes toxicity by absorption through the skin, inhalation or ingestion.¹ Methanol poisoning affects the optic nerve and the central nervous system with a predilection for basal ganglia, resulting in symptoms of visual disturbances, blindness drowsiness, seizures and coma.² CT and MR imaging are able to demonstrate toxic effects of methanol in the CNS. Putaminal necrosis with or without haemorrhage are the most frequent reported findings. Other affected areas that are reported in the literature are subcortical white

matter, hippocampus, optic nerve, tegmentum, cerebral gray matter and cerebellum.³

Case Report

Case-1:

A 51 year old male patient with a history of Acute Renal Failure (ARF) and chronic alcoholism accidentally ingested methanol mistaking it for alcohol. He was referred to the emergency department with complaints of blurred vision and diplopia. Laboratory findings were as follow: Arterial blood gas investigation showed (pH: 7.2, BICARB: 18.9, PCO₂: 40.9, PO₂: 77.2). BUN: 77, Cr: 3.7, Na: 140, K: 5.4. Methanol blood level was not done.

His MRI brain was done which showed abnormal signal intensity in the white matter involving subcortical and deep white matter in the frontal and occipital lobes bilaterally. Abnormal signal areas were also seen in the corpus callosum, putamen and cerebellum bilaterally. These changes showed iso to hypointense signal on T1-weighted and hyperintense signal on T2-weighted images. Based on these findings the diagnosis of Methanol Intoxication was suggested. The patient was in a comatose state and was

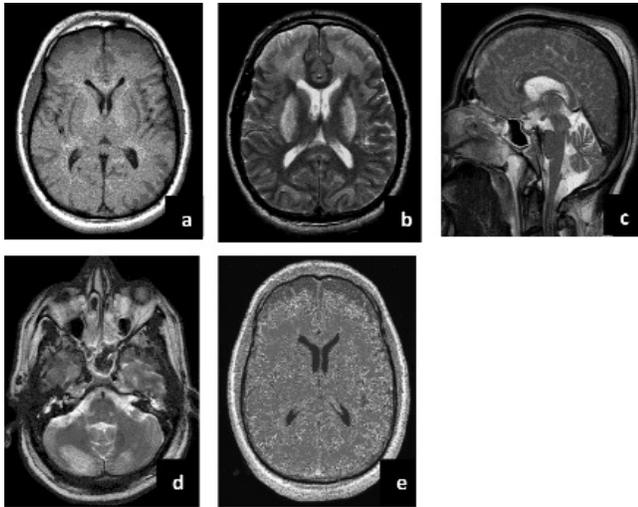


Figure-1: Methanol Intoxication. (a) T1-W Axial Image shows hypointense signal in putamina and cerebral white matter in frontal region bilaterally (b) T2-W Axial Image shows hyperintense signal in putamina and cerebral white matter in frontal region bilaterally. (c) T2-W Sagittal Image show hyperintense signal in corpus callosum (d) T2W Axial Image shows hyperintense signal in cerebellum bilaterally. (e) Post contrast T1-W Axial Image shows no abnormal enhancement.

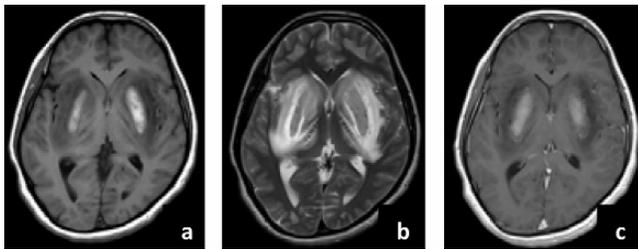


Figure-2: Methanol Intoxication. Haemorrhagic necrosis of the putamina bilaterally. These are associated with surrounding oedema. (a) T1-W Axial Image shows hyperintense signal in putamina bilaterally. (b) T2-W Axial Image show isointense signal. (c) T1-Post contrast Axial Image shows no enhancement.

intubated and taken to the ICU. Unfortunately the patient did not survive.

Case-2:

A 23 year old male who was a habitual alcoholic, presented in the emergency in a critical unconscious state with Glasgow's coma scale of 3/15 and bilateral non-reactive pupils. Shortness of breath, drowsiness and generalized abdominal pain for past one day following the ingestion of about two glasses of locally brewed alcohol (desi sharab). On presentation his vitals were: heart-rate=120 beats per minute, B.P.=70/35 mm/Hg, Respiratory rate=40 and temperature=36°C. His arterial blood gas investigation showed (pH: 7.5, PCO₂:25.5, PO₂: 150.7, BICARB: 20.2), BUN:7, Cr: 0.5, Na: 130, K: 3.5, Cl: 100.

He underwent MRI brain which showed bilaterally symmetrical subacute haemorrhage noted in the bilateral

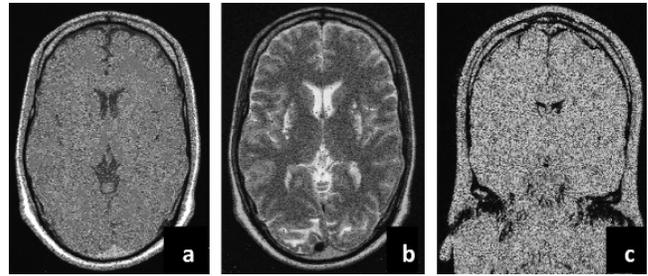


Figure-3: Methanol Intoxication. Bilateral putaminal lesions (a) T1-weighted axial image show hypointense signal. (b) T2-weighted axial image show hyperintense signal. (c) Coronal FLAIR image show hyperintense signal. These changes are suggestive of non haemorrhagic necrosis.

putamina. These appeared hyperintense on T1-weighted and isointense on T2-weighted images, which were associated with surrounding oedema. Based on these findings a most probable diagnosis of Methanol Intoxication was suggested.

Case-3:

A 32 years old male patient with no significant medical history presented with complaints of unconsciousness for four days. When he regained consciousness he had visual changes and was unable to see at a distance. His vision had reduced to finger counting. On examination he was conscious and oriented. Fundoscopy showed normal bilateral optic disc. On presentation his vitals were heart-rate=135 beats per minute, B.P.=140/80 mm/Hg, Respiratory rate=28 and temperature=34°C. Arterial blood gas and blood methanol level were not done. He underwent MRI brain which showed bilateral symmetrical areas of abnormal signal intensity in the putamen and occipital lobes bilaterally. These showed iso to hypointense signals on T1-weighted, hyperintense signals on T2-weighted and FLAIR images. All these changes represented non haemorrhagic necrosis. No abnormal signals were identified in the optic nerves. Based on these findings along with the clinical history he was diagnosed as a case of Methanol Intoxication.

Discussion

Susceptibility to methanol poisoning varies greatly. Methanol intoxication can cause severe metabolic acidosis, visual defects, permanent neurological dysfunction and death.⁴ A latent period of 12-24 hours often follows methanol ingestion. The latent period most likely corresponds to the time period in which methanol is metabolized into formaldehyde and formic acid.⁵ Most patients note visual disturbances ranging from blurred vision to permanent blindness secondary to optic nerve necrosis or demyelination, as one of the first symptoms. Central nervous system symptoms are common and include nausea, vomiting, headache, dizziness, weakness, and

malaise. Large amount of methanol ingestion can result in seizures, stupor, coma and sometimes death.³ The diagnosis is based on the presence of severe metabolic acidosis with high anion and osmolar gap and high serum methanol levels.³ Neuroradiological findings in methanol poisoning have occasionally been described in the literature. The most characteristic MR findings in methanol toxicity are bilateral putaminal necrosis with or without haemorrhage. On the other hand, putaminal changes may also be seen in Wilson's disease, Leigh's disease, Kearns-Sayre syndrome, carbon mono-oxide inhalation, hypoxic-ischaemic injury, trichloroethane poisoning and acute cyanide intoxication.⁶ Cerebral and intraventricular haemorrhage, diffuse cerebral oedema, cerebellar necrosis, and abnormalities of basal ganglia, optic nerve and pontine tegmentum are the other MRI findings of methanol intoxication.⁷ When a large amount of methanol is ingested, death usually occurs within three days.⁸ Treatment is by drug elimination (e.g. haemodialysis) and inhibition of the metabolism of methanol to toxic formic acid by competitive inhibition of the enzyme alcohol dehydrogenase (with ethyl alcohol or fomepizole).⁹

Our report illustrates the usual effects of methanol intoxication on the nervous system. The lesion site may be restricted to the putamen, as in our second and third case and in most similar reports in the literature, or may involve the putamen, white matter, cerebellum and corpus callosum as seen in our third case.

In previous studies MR imaging appearances were established in methanol poisoning. In one report patient did not have a documented blood methanol level but did have necrotic changes in the putamen bilaterally.¹⁰ Another case of acute methanol intoxication presented with weakness, blurred vision, mildly reactive bilateral mydriasis and

progressive decrease in the level of consciousness. The CT and MR imaging in that patient showed bilateral putaminal haemorrhagic necrosis and subcortical white matter lesions.³

Conclusion

In conclusion when symmetrical lesions are detected in the basal ganglia and white matter along with sudden visual disturbances, there can be a long list of differential but correct diagnosis could be reached if history of methanol ingestion is available. Since early diagnosis may improve the prognosis in acute phase, methanol intoxication should be considered in the differential diagnosis of such lesions on MRI examinations.

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