

Clinical Image

Methanol Poisoning: An Unusual Case of White Matter Changes on MRI

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A 47-year old Bangladeshi gentleman presented with the complaints of sudden onset bilateral blurring of vision for 3 days with aggressive behaviour. The visual loss was acute, and the patient had become rather drowsy, with incoherent speech and tremors. There was no preceding history of trauma, fever, neck rigidity, limb weakness, seizures or other comorbidities requiring toxic drugs. On query, his relatives gave a history of chronic alcohol intake for over 10 years, possibly including methanol due to adulteration of ethyl alcohol. The patient had taken an alcoholic binge the night before to onset of symptoms. He was hypertensive, well controlled on amlodipine-atenolol combination. On examination, GCS was 15/15 (i.e. conscious) but he was delirious and extremely aggressive. Pulse was 96 beats/min. Blood pressure was 95/60mmHg. Pupils were slightly dilated bilaterally with sluggish reaction to light. Visual acuity was no perception of light (NPL). Ophthalmoscopy revealed bilateral optic atrophy (Figure 1). There was no nystagmus but extraocular muscles could not be tested owing to NPL. Other cranial nerves were intact and there was no focal neurological deficit, although higher psychic function was severely impaired. He also had retrograde amnesia. There were no abnormalities on other systemic examinations. Random blood sugar was 6.1mmol/L so hypoglycaemia as a cause for visual loss and delirium were effectively excluded. Serum electrolytes: Na⁺ 141mmol/L. K⁺ 5 mmol/L. Cl⁻ 95mmol/L. TCO₂ 12mmol/L. Plasma anion gap: 37mmol/L. Complete blood count revealed: haemoglobin: 15.5g/L (MCV 101.5fl; MCH 35pg) , WBC: 7000/cumm (neutrophils: 80%; lymphocytes 15%), Platelets: 190,000/cumm and ESR: 6mm/1st hour. Serum creatinine, liver and thyroid function tests were within normal limits. Urine routine examination, ECG and chest X ray revealed no abnormality.

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MRI of brain revealed lesions of subcortical white matter of both frontal lobes showing increased signal intensity signifying restricted diffusion on diffusion-weighted imaging (DWI), while on FSET2 and T2 FLAIR the same lesions showed hyperintensity. These findings were consistent with toxic myelinosis (Figure 2 a & b). Corpus callosum, optic chiasma, basal ganglia including the putamen, cerebellum, thalami, pituitary and para-sellar areas appeared normal in signal characteristics and morphology.



Fig.-1: Fundus photography showing bilateral optic atrophy.

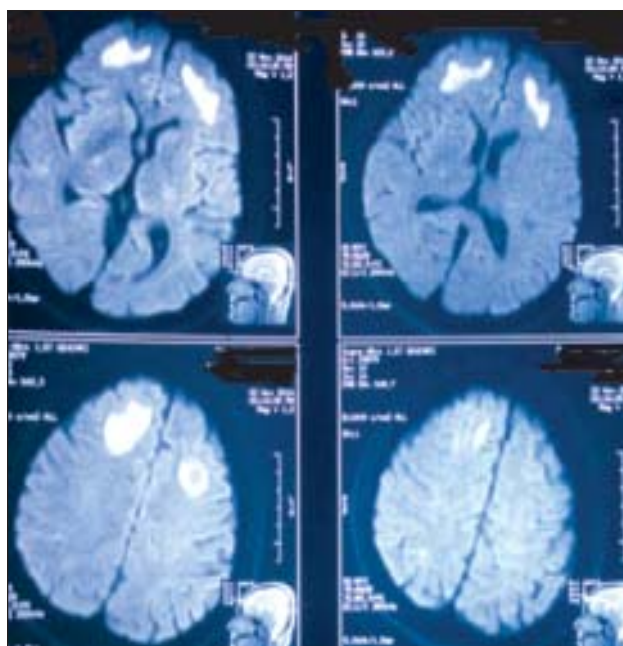


Fig. 2(a): MRI showing toxic myelinosis involving frontal lobe on DWI

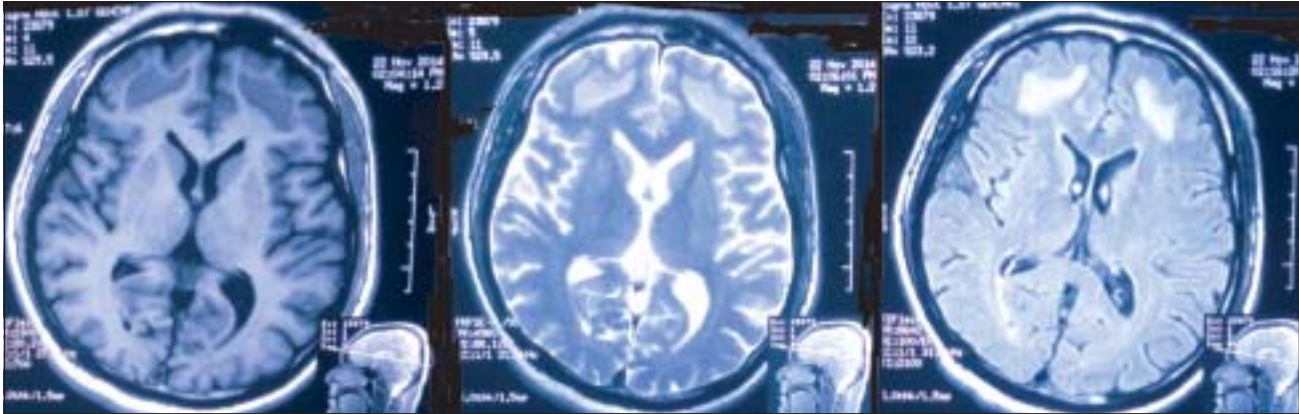


Fig-2 (b): MRI showing toxic myelinosis on T_1 , T_2 and FLAIR imaging respectively

The patient was instituted a trial of Injectable methylprednisolone for 3 consecutive days to treat the toxic optic neuropathy, but his visual acuity remained NPL at discharge. He was also given anti-psychotic medication for acute management of the delirium, in addition to injectable thiamine. Typically treatment should have been instituted with the antidote fomepizole, an alcohol dehydrogenase inhibitor, but could not be administered in our case due to poor economic solvency of the patient.

Discussion:

Methanol, also known as wood alcohol, is a clear, colourless toxic liquid commonly used as an organic solvent in anti-freeze solutions, varnishes, paint, and fuel. In the developing world however, methanol toxicity is a common problem chiefly owing to the fact that it is a commonly found constituent of poorly adulterated alcoholic beverages.¹

As it both tastes and smells similar to ethanol,¹ alcoholics are inadvertently prone to its toxicity, mostly among the lower socioeconomic classes. Methanol toxicity occurs due to its metabolism into the toxic compounds formaldehyde and formic acid in the liver, which accounts for the latent period of around 12-24 hours, between its consumption and the onset of symptoms.^{2,3} Most patients have a decreased level of consciousness at presentation, making an adequate history unobtainable, therefore it is important to involve the patients' bystanders in a thorough history-taking.

The clinical presentation of methanol toxicity varies from patient to patient, but its onset is usually characterized by visual disturbances, secondary to optic nerve necrosis or demyelination in most cases.^{1,2} Optic atrophy is a common finding in methanol intoxication but is not specific to it and other causes need to be excluded. Central nervous system symptoms are common and include headache, dizziness, weakness and malaise, with larger doses resulting in seizure,

stupor, coma and sometimes death.^{1,2} Biochemically, the diagnosis is based on the presence of severe metabolic acidosis with high anion and osmolar gap and high serum methanol levels.¹

CT and MR imaging demonstrate toxic effects of methanol in the central nervous system, and they have frequently been described in the literature.¹⁻⁸ The most well known CT and MRI finding of methanol intoxication is bilateral necrosis of the basal ganglia, primarily the putamen with or without haemorrhage.¹⁻⁶ Lesions can also extend into the corona radiata, centrum semiovale, hippocampus, optic nerve, tegmentum, cerebral grey matter and cerebellum,¹ optic nerves⁴ and subcortical white matter,^{1,5,6} produce diffuse cerebral oedema or separate necrotic lesions in the cerebral white matter.^{1,2,5} The basis for the selective vulnerability of these regions remains unknown. However, it has been postulated that direct toxic effects of methanol metabolites also were responsible for the subcortical and putaminal lesions.⁴ Optic nerve demyelination secondary to myelinoclastic effect of formic acid has been suggested as responsible for optic nerve damage with or without axonal loss.⁴

White matter changes without putamen involvement are, however, a less well recognized complication of methanol intoxication and not many cases have been described previously,⁴⁻⁶ much less so for frontal lobe lesions as found in our patient. However, authors have previously reported typical MRI lesions on DWI, FLAIR and T2 sequences, corroborating with the lesions in our case, which involved the white matter. Server et al. reported that conventional MRI usually depicts symmetrical, bilateral increased signal in the involved areas; DWI shows decreased diffusion, probably reflecting cytotoxic edema; FLAIR and T2-weighted images showed hyperintensity in the involved areas, characteristic of post-necrotic changes.⁵ MRI images of our case reported similar findings.

Conclusion:

Our report illustrates the fact that, in addition to the typical radiological findings of putaminal necrosis, methanol toxicity may also present with other MRI changes of white matter lesions. A wide differential diagnosis, including multiple sclerosis, ADEM (Acute Disseminate Encephalomyelitis), Progressive Multifocal Leukoencephalopathy (PML) and Subacute Sclerosing Panencephalitis (SSPE) need to be borne in mind.⁹ Thus, a careful history is paramount, and will be able to identify methanol intoxication early, thus enabling timely life-saving treatment.

Conflict of Interest : None**References:**

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