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Acute Blindness Following Methanol Poisoning

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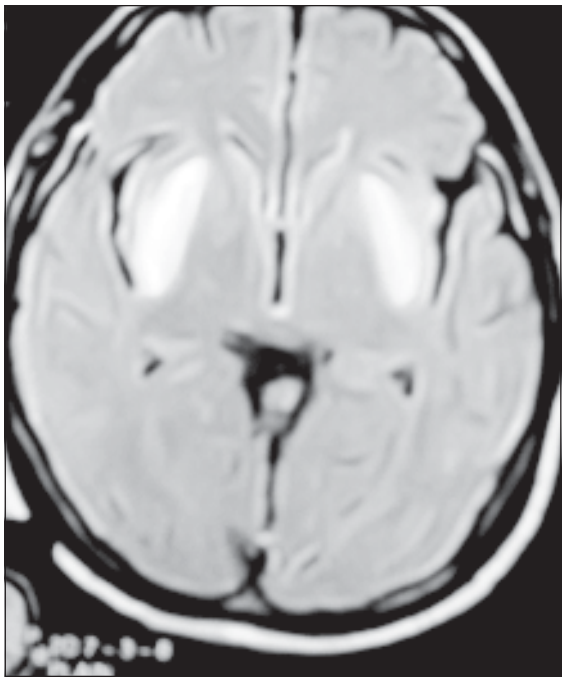
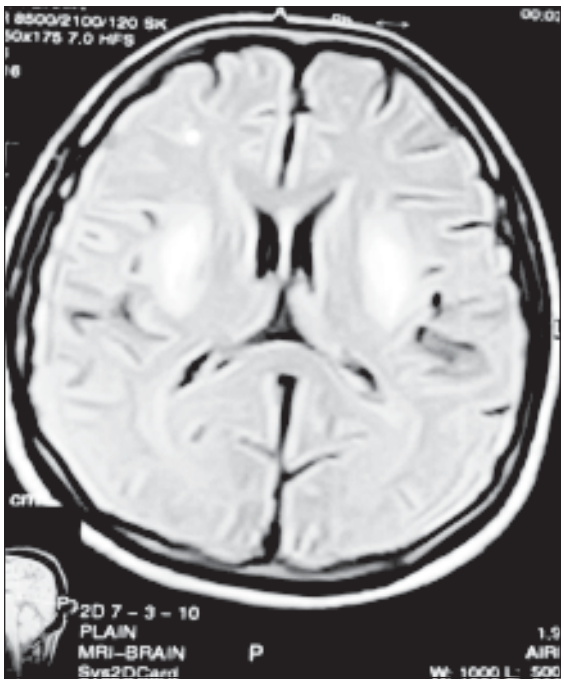
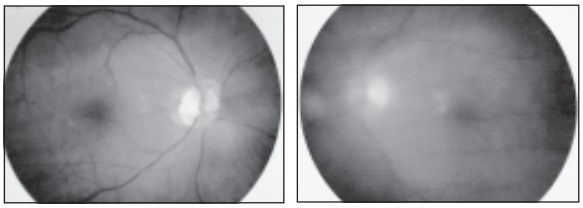


Hyperintensities in both lentiform nucleus.



Hyperintensities in both lentiform nucleus.

A 42 years old, male muslim, married, normotensive, non diabetic, non-smoker was admitted to DMCH with the history of disorientation for 4 days, headache and blurring of vision for 3 days and respiratory distress for 2 days. There is no history of trauma, head injury, fever, cough, vomiting, convulsion, weakness of limbs, bleeding episodes or bowel and bladder incontinence. On query, he confessed that he was taking methanol regularly for last 8 years, but intake was large before the day of admission. Examination revealed patient was mildly anaemic. His pulse was 90/min, BP 90/60 mm Hg, respiratory rate 40 breaths/min, GCS 15/15,



Fundus phorograph: Bilateral Optic atrophy

bilaterally fixed and dilated pupils, absent pupillary light reflex without ptosis and nystagmus. Fundoscopic examination revealed bilateral optic atrophy. Other cranial nerves were intact. He had no signs of meningeal irritation and other systemic examination revealed no abnormalily.

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Investigation findings:

CBC with ESR	Hb-10.7g/dl, WBC-11500/cmm(N-93.5%),Platelet-247000/cmm MCV-96fl,ESR-42 mm in 1st hour
RBS	7.9 mmol/l
S.Creatine	1.22mg/dl
S.Urea	44mg/dl
S.Electrolytes	Na-135 mmol/l ,K-5.8mmol/l, Cl:121 mmol/l, Tco2: 14mmol/L
S. Alcohol	<10mg/dl
Urine R/E	Pus cells-1-4 /HPF, RBC-nil, Albumin-(+)
ECG	Normal
MRI of Brain with contrast	T2 W1 and FLAIR hyperintensities in both lentiform nucleus. No enhancement seen after contrast adminstration. No midline shifting. Ventricles, extraventricular CSF spaces, sellar, suprasellar and parasellar regions appear normal .Normal flow seen in major cerebral blood vessels **Comment: Toxic Encephalopathy**
Arterial blood gas	Metabolic acidosis

Discussion :

Susceptibility to methanol poisoning varies greatly. 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.[1] Methanol intoxication can cause severe metabolic acidosis, visual defects, permanent neurological dysfunction and death.[2] 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. Neuroradiological findings in methanol poisoning have occasionally been described in the literature. These imaging findings reflect a variety of pathologic processes that can be acute, subacute, or chronic in nature and have concomitantly affected the white matter of the brain. **Multiple sclerosis** (MS) is a common white matter disease process that affects young adults. **Autoimmune/idiopathic diseases -** *Acute disseminated encephalomyelitis (ADEM), Subacute sclerosing panencephalitis (SSPE), Neuromyelitis optica* ,**Infection -** *Lyme neuroborreliosis, Progressive multifocal leukoencephalopathy (PML),* **Vascular -** *Central nervous system vasculitis, CADASIL , Susac's syndrome and* **Acquired conditions-***Radiation necrosis, Osmotic demyelination syndrome, Diffuse axonal injury can cause deep white matter lesion in MRI finding.*[4]

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.[5] 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.[6] Our report illustrates the usual effects of methanol intoxication on the nervous system . 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] When a large amount of methanol is ingested, death usually occurs within three days.[7]

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.

References:

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