MRI BRAIN, FEATURING METHANOL INTOXICATION: A RARE CASE REPORT

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ABSTRACT

Methanol poisoning although a rare entity,but has life threatening outcomes due to its highly toxic effects on body. It may be ingested accidentally or may be with suicidal intentions as well as habitual. Methanol intoxication leads to metabolic acidosis, neurological damage resulting in extrapyramidal symptoms, blindness and coma. According to our knowledge the MRI findings of methanol poisoning have been described in only few literatures. Therefore we present a case of one such patient who presented to emergency department with altered level of consciousness, drowsiness and vomiting. His MRI Brain revealed typical features of methanol poisoning.
Key words: methanol intoxication,Putamen Hemorrhage, Magnetic Resonance Imaging, neurological damage.

Introduction

Methanol is a highly toxic liquid with a taste and odor similar to ethanol. It is commercially available as a component of antifreeze, varnishes, perfumes, gasoline enamel, plastics, film, textiles, wood alcohol etc. It has an unpleasant taste when impure however after purification it becomes edible.[1-4] It may be available as homebrew liquor (Kachi sharab).

Intestinal tract, skin and respiratory systems are the routes of absorption.[2] It can also cross the placenta[1] the toxicity of methanol is mainly due to its metabolite formate.[3]

Clinical manifestations of Methanol poisoning include altered level of consciousness, blurring of vision, severe metabolic acidosis, drowsiness, seizures,and coma and in severe cases there may be permanent CNS dysfunction, blindness or death.[2-3,7]

Imaging modalities like CT and MR have demonstrated the toxic effects of methanol in brain. Methanol most frequently targets the basal ganglia, causing necrosis and hemorrhage of putamen. Less commonly involving the subcortical whitematter, hippocampus, optic nerve, tegmentum, cerebral graymatter and cerebellum.[7]

There are various treatment options including dialysis, gastric lavage and drug therapies. (1-7)

Case Report

A 47 year old male patient addicted to alcohol and smoking from last 18 years presented to Emergency Department with complaints of altered level of consciousness, drowsiness and vomiting and with a positive history of homebrew liquor ingestion. His GCS was 4/15.

On arrival his vitals were: pulse-rate=76 beats per minute, B.P=140/80 mm/Hg, respiratory rate=40 and temperature=98F. His arterial blood gas investigation showed (pH: 7.25, PCO2:30, PO2: 97, BICARB: 10), Cr: 1.97, Na: 130, K: 3.5, Cl: 100, RBS: 56. His blood methanol levels were 160mg/dl. Patient was intubated in E.D.

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He underwent an MRI of brain which showed bilateral symmetrical abnormal signal intensity areas in putamen with surrounding perilesional edema. These appearto be isointense to hyper intense on T1 and isointense to hypo intense on T2W and FLAIR images. His positive ingestion history, blood methanol levels, acidotic state of metabolism and MRI findings are all suggestive of methanol intoxication.

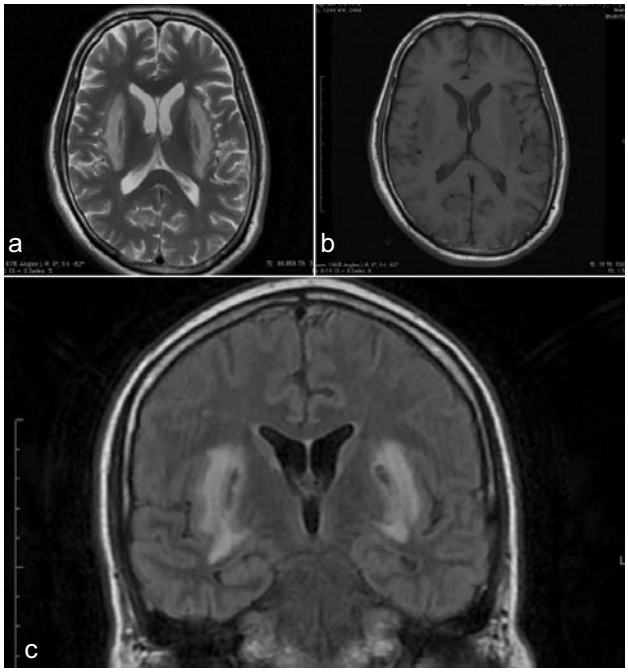

Figure 1: Shows Hemorrhagic necrosis of putamen bilaterally associated with surrounding edema. **(a)** T2w axial image shows hyper intense signals in bilateral basal ganglia representing edematous changes. **(b)** T1w axial image shows iso to hyper intense signals in basal ganglia. **(c)** Coronal FLAIR image shows hyper intense signals in bilateral basal ganglia representing edematous changes.

Discussion

Methanol is highly poisonous and its ingestion can be accidental as well as suicidal. Its toxic effects include brain damage often permanent neurological impairment, optic nerve impairment, severe metabolic impairment and also death.[7] Methanol is metabolized in the liver to formaldehyde and later to formic acid. This process itself takes 12-24 hours resulting in the latent period before symptoms appear.[7]

As less as 4ml of methanol causes blindness, approximately 15ml- 30ml of methanol and blood levels of 1500mg\l are sufficient to cause death.[1]

Early manifestations of methanol intoxication are mostly due to the necrosis or demyelination of the optic nerve resulting in blurring of vision as well as blindness. Headache, weakness, drowsiness, nausea and vomiting are the common neurological symptoms. In severe cases this may lead to seizures, coma and death.[7]

It can be diagnosed by evaluation of the anion gap, osmole gap and serum methanol levels, which all appear to be raised in such cases.[6,7] The gold standard for the diagnosis of methanol poisoning is methanol analysis by gas chromatography. It is however not a good indicator of level of toxicity ultimately the prognosis of the patient. Hence in our case the gold standard was the strong positive history of ingestion and blood methanol levels, later supported by the typical MRI findings.

On the basis of Radiological imaging the most striking characteristic feature includes bilateral necrosis of basal ganglia. Both hemorrhagic and non-hemorrhagic damage of the putamen can occur. Involvement of frontal and occipital lobe is common. Our patient showed the characteristic findings of bilateral putminal hemorrhage with perilesional edema. The findings of putaminal necrosis are evident on both CT and MRI however MRI is considered a superior modality due to the better anatomical details.[1]

Other differentials for putaminal damage may include Wilson's disease, Leigh's disease, Kearns-Sayre syndrome, carbonmono-oxide inhalation, hypoxic-ischemic injury, trichloroethane poisoning and acute cyanide poisoning. These causes were excluded in our case considering the positive methanol ingestion history, age of the patient (Leigh's disease, Wilson's disease and Kearns-Sayre syndrome present in early ages), raised blood methanol levels.

Methanol poisoning may rarely show involvement of cerebral and cerebellar hemispheres, intraventricular hemorrhage, diffuse cerebral edema and optic nerve necrosis.[4,6,7] There are reports of rare cases of sub arachnoid hemorrhage also.[4]

According to literature there are various reasons for the predominant putaminal involvement including the regional high metabolic demand, others suggested that it is related to decreased blood flow through the veins of Rosenthal secondary to hypotension or direct toxic effects of formic acid to the brain.[1,5,6] Patients surviving the acute attacks may show resorption of hemorrhagic and infarcted putaminal tissue and may

show formation of cystic cavities within it on follow up imaging.[1,5,6,7]
Treatment options include ethanol therapy, gastric lavage and dialysis.[1]

Conclusion

Methanol poisoning may be a rare entity but whenever a patient presents with symmetrical lesions involving the basal ganglia, methanol intoxication should also be included in differential. A positive ingestion history may support the diagnosis. Unfortunately despite improved treatment, mortality remains high due to delayed diagnosis. So it is necessary for clinicians and as well as for radiologists to be aware of clinical presentation and radiological features of methanol intoxications for early diagnosis and better prognosis.

Conflict of Interest: None to declare.

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