CASE REPORTS

ROLE OF NEUROIMAGING IN METHANOL TOXICITY - A CASE REPORT

Bushra Muzaffar Khan, Wasim Alamgir, Taqdees Fatima*

Pak Emirates Military Hospital/National University of Medical Sciences (NUMS) Rawalpindi Pakistan, *Combined Military Hospital/National University of Medical Sciences (NUMS) Rawalpindi Pakistan

ABSTRACT

Methanol is a frequently used organic solvent[1]. However toxic doses of methanol may lead to serious metabolic acidosis and neurologic side effects including subarachnoid hemorrhage[2] and sometimes even death. The case described suffered from such poisoning. Diagnosis was made on MRI brain that demonstrated bilateral putamen necrosis[3] a neuro-radiological hallmark of methanol toxicity.

Keywords: Imaging, Methanol, Toxicity.

INTRODUCTION

Methanol toxicity is relatively uncommon but life threatening. It is being used as a substitute for ethyl alcohol at many illegal alcoholic beverage markets[4] due to its low cost. Due to impurities, the impure form of methanol is non palatable. The purification process makes it tasteless and odorless thus increasing the chances of accidental or deliberate poisoning. Methanol is metabolized to formic acid which is the main toxic end product leading to all the signs and symptoms of toxicity[5].

CASE REPORT

A 58 years old male presented to emergency department with complaints of acute onset nausea, vomiting, central chest pain and altered sensorium for 12 hours after attending a late night dinner. On examination he had Glasgow Coma Scale of 10/15. He was afebrile with blood pressure of 80/50 mm Hg, pulse was regular with rate of 136 beats/min, respiratory rate 28/min and oxygen saturation of 88% at room air. Chest auscultation revealed bilateral harsh breath sounds and scattered crackles. ECG showed sinus tachycardia. Pupils were normally reactive to light with no papilledema on fundoscopy. There was no focal neurological deficit. Initial investigations showed normal blood biochemistry except for high anion gap metabolic acidosis having pH of 7.1. His urine and blood samples were immediately sent for toxicology screening which later on confirmed methanol intoxication. MRI Brain was advised. Symptomatic treatment was started in intensive care unit because of acidosis. CSF examination showed normal study and MRI brain revealed bilateral hemorrhagic necrosis of putamen and posterior limb of right internal capsule with edema in the deep white matter. On the basis of characteristic MRI brain changes and metabolic acidosis a presumptive diagnosis of methanol toxicity was made. He was treated with sodium bicarbonate 50 ml four hourly to reduce acidosis along with mega doses of co-factors like folic acid, pyridoxine and thiamine in the form of folic acid 5mg by nasogastric tube and five ampules Neurobion injections containing 100mg of pyridoxine and thiamine and 10 mg of cyanocobalamin each, thrice a day. He regained consciousness the next day but complained of bilateral blindness, and on questioning he accepted that he had consumed adulterated alcoholic beverage prior to losing consciousness. He made a steady recovery over the next week, but the blindness did not improve and he is being followed up in ophthalmology department.

DISCUSSION

Methanol poisoning does not produce any remarkable symptoms in early stages except visual disturbances. In one case report presented

Correspondence: Dr Bushra Muzaffar Khan, Department of Dermatology, Pak Emirates Military Hospital Rawalpindi Pakistan
Email: bushra.muzaffar1@gmail.com
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by Gadodia *et al*[6] in India, the presenting symptoms were same as in our case except chest pain and the diagnosis was made on CT scan and MRI findings. Their patient also developed these symptoms after ingesting locally made alcohol. In another case report by Blanco *et al*[7], the initial presentation was chest pain and autonomic dysfunction with history of chronic alcoholism and diagnosis was made by CT scan brain. The patient was managed with ethanol and bicarbonate.

When ingested methanol is absorbed from the gastrointestinal system rapidly. It is metabolized in the liver converting methanol first into formaldehyde and then into formic acid via the enzymes alcohol dehydrogenase and aldehyde dehydrogenase, respectively.

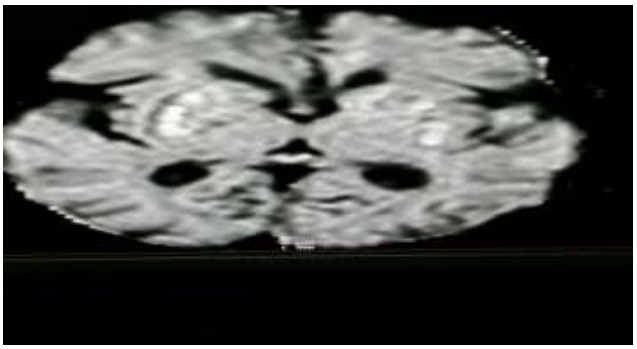

Figure-1: DWI Axial image showing bright signals in putamen signifying necrosis.

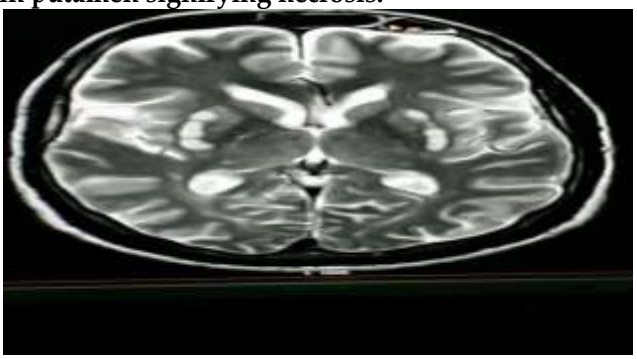

Figure-2: T2W Scan at the level of lateral ventricles showing bright signals in putamen.

Formic acid is oxidized to carbon dioxide and water with the help of the enzymes 10-formyl tetrahydrofolate synthetase and dehydrogenase[8]. This last step is very slow thus resulting in accumulation of formic acid in the body which leads to life threatening metabolic acidosis. Another postulated mechanism is the low hepatic levels of these enzymes in humans leading to methanol intoxication.

Methanol toxicity damages the optic nerve thus causing acute blindness. It selectively damages the retro laminar optic nerve myelin sheath as seen on histopathology[9]. Early changes include hyperemia and swelling of the optic disc leading to a papilledema like appearance.

Chronic methanol poisoning leads to Parkinsonian motor impairment postulated to be due to formic acid accumulation in the putamen[10].

Neurological imaging investigations are helpful in diagnosis[11] and show bilateral basal ganglia necrosis. Other findings include cerebral edema, necrosis of subcortical white and gray matter, cerebellar cortical lesion, subarachnoid hemorrhage, bilateral tegmental necrosis and intra cerebral hemorrhage. High concentrations of formic acid in the putamen lead to its necrosis due to increased propensity of the striatal neurons to accumulate the toxic metabolites of methanol[12] (fig-1 & II).

The management of acute methanol toxicity includes gastric lavage, treating metabolic acidosis, administration of ethanol which leads to competitive inhibition of methanol oxidation and hemodialysis to remove both formate and methanol.

The amount of methanol ingested and the subsequent severity of metabolic acidosis determines the prognosis in methanol poisoning.

This patient was received in Combined Military Hospital (CMH) Quetta with a vague history of alcohol ingestion but altered sensorium and typical MRI findings were highly suggestive of methanol toxicity. Since antidotal treatment is available so early recognition of this poisoning and prompt institution of specific therapy decreased the morbidity and mortality associated with this form of poisoning.

CONCLUSION

Methanol toxicity is commonly seen in underdeveloped countries especially the lower socioeconomic lining of society. With increasing availability of various neuroimaging modalities in larger hospitals of our country, many of these patients are investigated with MRI Brain.

Health care providers especially physicians working in A & E and acute medicine need to be well informed about the characteristic MRI findings so that the diagnostic haze is cleared early and appropriate management is expeditiously instituted.

CONFLICT OF INTEREST

This study has no conflict of interest to be declared by any author.

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