Case Report

Diffusion Weighted Image (DWI) findings in Methanol Intoxication

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Abstract

Methanol is a toxic substance with extremely devastating effects upon exposure. The case described suffered from such kind of poisoning. MRI brain demonstrated necrosis bilaterally in the Putamen areas which is a classic hallmark finding. Additional Diffusion weighted imaging showed abnormal signal bilaterally in the putamen areas along with Diffusion positive bilateral lesions (possibly infarctions) in both the frontal and occipital lobes that were not evident on MRI with or without contrast.

Introduction

Methanol/ Methyl Alcohol/ Wood Alcohol / Wood spirit is obtained from wood distillation. It is used in a variety of materials for example varnishes, enamel, plastics, film, textiles, dyes, windshield cleaners etc. The crude form of methanol is not palatable due to the presence of impurities. After the purification process it can become ingestible which greatly increases the chances for accidental or deliberate poisoning. Pure methanol is a colorless liquid that has a slight difference in odour from ethanol. It can be absorbed through the gastrointestinal tract, respiratory system or the skin. The danger from methanol poisoning is due to the formation of its metabolite formate that is the main culprit for causing the damage.

The minimal recorded amount required to kill is 15ml. and the highest recorded is equal to 500ml. The metabolism is via oxidation by aldehyde dehydrogenase to formaldehyde, formic acid and carbon-dioxide which can be excreted through sweat urine and expired air.[1]

Case Report

This case is of 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 un-reactive pupils. His family stated that the patient had been complaining of shortness of breath, drowsiness and generalized abdominal pain for the past one day following the ingestion of about two glasses of locally brewn 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^{o}C. His initial investigations were as follows:

Complete Blood Count — Hemoglobin=14.5gm/dl, WBC=31.8 x 10^9/L with Neutrophils at 83.9% and Platelets=442 x 10^9/L.

Serum Chemistry — Na=130meq/L, K=6.9meq/L, Cl=89meq/L, Bicarbonate=4.4meq/L, BUN=27mg/dl, Cr=1.6mg/dl, Random Blood Sugar=531mg/dl and Blood Alcohol level=136mg/dl.

Arterial Blood Gases — pH=6.89, pO_2=40.6, pCO_2=80, HCO3=7.9, O_2 saturation-83%.

These reports showed that he had developed severe metabolic acidosis. He was intubated immediately and started on treatment for methanol poisoning with I/V ethanol and NaHCO3 replacement. He was shifted to the Intensive Care Unit and followed vigilantly. Only one session of hemodialysis was carried out that reduced the Blood Alcohol level to 66mg/dl.

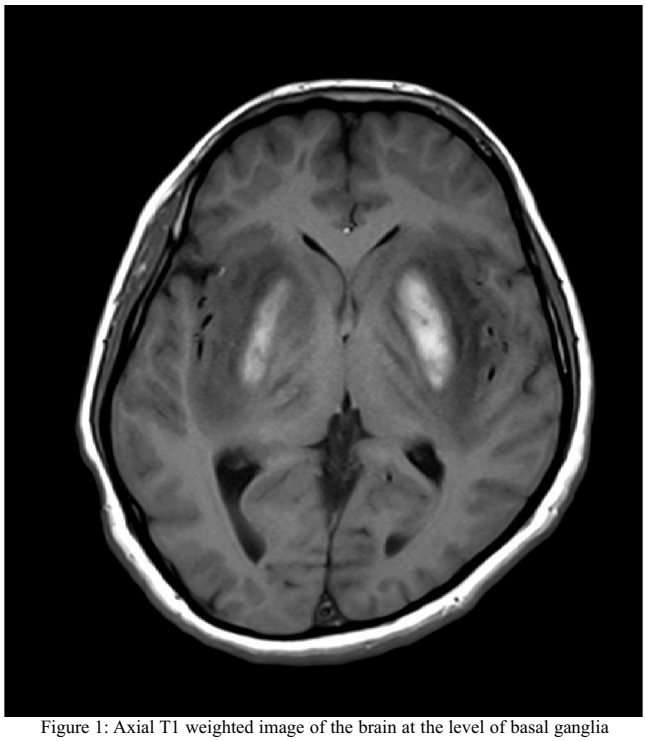

Figure 1: Axial T1 weighted image of the brain at the level of basal ganglia showing bilateral Putamen hemorrhages.

MRI of brain revealed haemorrhage bilaterally in the putamen areas with surrounding edema hyper-intense on T1 and iso-intense on T2 (Figure 1).

Diffusion weighted imaging demonstrated abnormal signal variation bilaterally in the putamen regions alongwith bilateral abnormal signals (Diffusion positive) in both the Frontal and Occipital lobes suggestive of acute infarctions (Figure 2).

Additional Electroencephalogram analysis reported

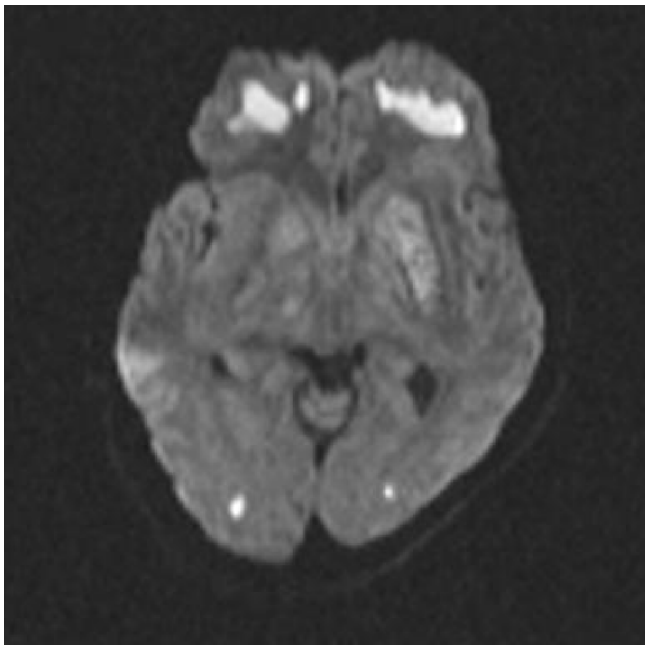

Figure 2: Axial Diffusion weighted image of the brain at the level of basal ganglia, showing abnormal signal bilaterally in the putamen with diffusion positive subcortical infarctions in the frontal and occipital regions.

neuronal dysfunction due to moderate to severe encephalopathy of hypoxic/toxic origin.

He was managed supportively. Extubation was carried out on the 3rd day of hospital admission. For longterm nutritional support the Nasogastric tube was replaced with a percutaneous endoscopic gastrostomy tube (PEG) on the 5th admission day and this was followed by a tracheostomy on the 8th admission day.

The patient improved steadily but his visual capacity decreased dramatically to point of irreversible blindness. After a total duration of nine days in the Intensive Care Unit, he was moved out to the Special Care Unit. On the 12th admission day he developed fever spikes which was investigated to be due to tracheal colonization of Acinetobacter species that was sensitive to Polymyxin-B. Furthermore he also developed left sided basal atelactasis that settled after starting Amikacin I/V. After resolution of the infection the family was counseled for discharge inorder to prevent the patient form risk of acquiring any other nosocomial infection. Homecare nursing services were arranged and the patient was discharged alongwith the percutaneous endoscopic gastrostomy tube (PEG) and tracheostomy to be followed further onwards in the clinic.

Discussion

The ultimate survival after methanol poisoning depends on how much of the poison was swallowed and how soon was the treatment received. The exact rates for mortality and morbidity have not been reported however prognosis is correlated with the degree of metabolic acidosis.

The effects of methanol in relation to visual damage have been extensively studied upon. The main area effected is the retro-laminar optic nerve as a result of intra-axonal swelling and organelle destruction. Similarly severe poisoning causes Putamen haemorrhagic/non-haemorrhagic damage resulting in a dystonic clinical picture. The reason for affecting the Putamen is due to the sensitivity of striatal neurons to the toxic metabolites (Formate).

MRI findings of bilateral Putamen necrosis have been well documented.[2,3] However in our case Diffusion weighted imaging showed bilateral abnormal signals in the Frontal and Occipital lobes that were not evident on the MRI. This can be explained in part that methanol is implicated in causing sub-cortical infarction aside from the classic findings as described earlier.[4,5]

Apart from these manifestations methanol poisoning can also result in intraventricular haemorrhage, cerebellar necrosis, diffuse cerebral oedema, optic nerve demyelination or necrosis.[6,7] All these can present with different variations on standard neuroimaging and the reason for involvement of these areas is still largely unknown.

Conclusion

Methanol toxicity is a very acute and dangerous emergency that can cause intense damaging effects to the CNS and should be dealt with urgent and prompt treatment. Although methanol poisoning causes a wide variety of CNS effects, all such findings cannot be always demonstrated on routine neuroimaging alone. Advance techniques might be required to reveal additional changes if present however the classic hallmarks of optic nerve demyelination and Putamen necrosis are easy to pick up on MRI brain and the requirement to further investigate should depend on the need if it arises.

References

1. Chinard FP, Frisell WR. Methanol intoxication: biochemical and clinical aspects. J Med Soc NJ 1976; 73:712-9.
2. Bessell-Browne, Bynevelt M. Two cases of methanol poisoning: CT and MRI features. Australas Radiol 2007; 51:175-8.
3. Server A, Hovda KE, Nakstad PH, Jacobsen D, Dullerud R, Haakonsen M. Conventional and diffusion-weighted MRI in the evaluation of methanol poisoning: Acta Radiol 2003; 44: 691-5.
4. McLean DR, Jacobs H, Mielke BW. Methanol poisoning: a clinical and pathological study. Ann Neurol 1980; 8:161-7.
5. Blancoa M, Casadob R, Vázqueza F, Pumara JM. CT and MR imaging findings in methanol intoxication. AJNR Am J Neuroradiol 2006; 27:452-4.
6. Halavaara J, Valanne L, Setälä K. Neuroimaging supports the clinical diagnosis of methanol poisoning. Neuroradiology 2002; 44:924-8.
7. Gaul HP, Wallace CJ, Auer RN, Fong TC. MR findings in methanol intoxication. AJNR Am J Neuroradiol 1995; 16:1783-6.