

CASE REPORTS

BILATERAL PUTAMEN HAEMORRHAGIC NECROSIS DUE TO METHANOL INTOXICATION

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INTRODUCTION

Methanol toxicity presents as an emergency because of its preferential and early involvement of basal ganglia and optic pathways. Although history might help in early clinical diagnosis but here we present a rare case of chronic alcoholic patient, who presented as a diagnostic dilemma from a local hospital.

CASE REPORT

A 35-year-old man with a history of chronic alcoholism was referred to the emergency department with weakness of all four limbs and blurred vision. Patient was initially treated at local hospital with normal base line investigations i.e. blood complete picture, urine routine examination, chest X-ray, blood sugar and ECG. Patient was admitted for further evaluation as well as for brain CT scan and MRI. Fundoscopic examination did not show papilledema. Arterial gasometry showed systemic metabolic acidosis (pH, 7.08; pCO₂ 28; HCO₃, 7.6; BE-27) with high anion gap and high

blood methanol level determination. Methanol blood level was 50 mg/dL. The patient underwent continuous venovenous hemodiafiltration for 96 hours after residual blood methanol levels were found. His Glasgow Coma Scale (GCS) score was normal with weakness of all four limbs as well as visual disturbance. A cranial CT scan performed on day 5 showed low attenuation confluent lesions in superficial white matter and putamina consistent with acute toxic edema with high attenuation foci in the putamina consistent with hemorrhage (Figure-A).

Figure-B and C show central putamen haemorrhage which is hyperdense on CT and hyperintense on T1WI (hypointense on T2WI) alongwith peripheral oedema. MRI brain also showed multi-focal subcortical demyelination in cerebral and cerebellar cortices as well. Patient condition in terms of weakness and visual disturbance remained same with no significant improvement during hospitalization.

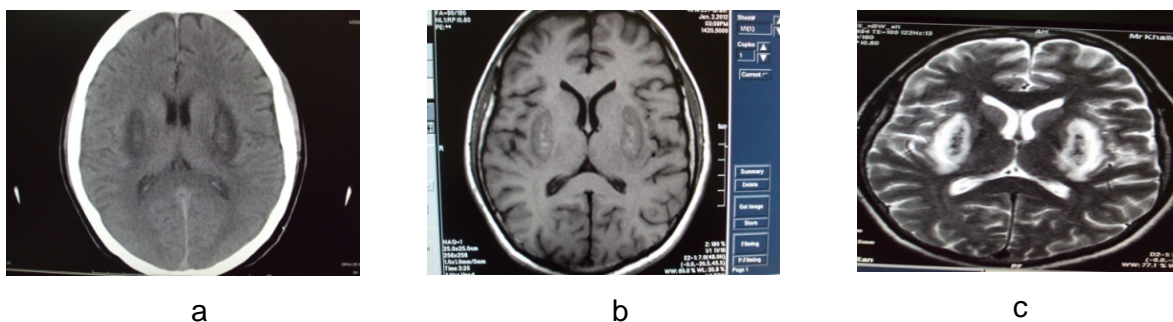


Figure: (a) Bilateral putamen central hyperdensity with surrounding oedema (haemorrhagic necrosis), (b) T1W sequence shows central hyperintense linear area (haemorrhage) within hypointense putamen (c) Bilateral putamen hyperintensity with central hypointensity.

osmolar gap (40 mEq/L). With a presumed diagnosis of methanol intoxication, bicarbonate and ethanol were administered while awaiting

Further follow-up was lost.

DISCUSSION

Acute methanol poisoning is a rare accidental or suicidal intoxication. It has also been described as a result of fraudulent adulteration of alcoholic drinks. As in our case, this case occurred in wake of new year of 2012

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and number of intoxications were reported all over the country. The clinical presentation of methanol intoxication can vary greatly from patient to patient. A latent period of 12–24 hours often follows methanol ingestion¹. The latent period most likely corresponds to the time period in which methyl alcohol is metabolized into formaldehyde and formic acid, 2 chemicals that are more toxic than methanol².

Susceptibility to methanol poisoning varies greatly. Most patients note visual disturbances, secondary to optic nerve necrosis or demyelination, as one of the first symptoms³. Central nervous symptoms are common and include headache, dizziness, weakness, and malaise. Large amounts of methanol ingestion can result in seizure, stupor, coma, and sometimes death. Gastrointestinal symptoms are common. The diagnosis is based on the presence of severe metabolic acidosis with high anion and osmolar gap and high serum methanol levels⁴. In acute methanol intoxication, to prevent the conversion of methanol into toxic metabolites, ethanol is administered because its affinity for alcohol dehydrogenase enzyme is 10–20 times greater than that of methanol. Other therapeutic procedures include gastric lavage, correction of acidosis with sodium bicarbonate, folic acid, and secondary detoxication with hemodialysis. The ultimate survival after methanol poisoning depends on how much of the poison was swallowed and how soon was the treatment received³⁻⁵.

The most characteristic MR findings in methanol toxicity are bilateral putaminal necroses, which may have varying degrees of hemorrhage. This finding is by no means specific to methanol toxicity but is seen also in a variety of conditions, including Wilson disease and Leigh disease. Putaminal necrosis and hemorrhage probably result from the direct toxic effects of methanol metabolites and

metabolic acidosis in the basal ganglia⁶. Cerebral and intraventricular hemorrhage, cerebellar necrosis, diffuse cerebral edema, bilateral subcortical white matter necrosis or edema, and optic nerve necrosis all have been described in severe methanol intoxication⁷. It is possible that direct toxic effects of methanol metabolites also were responsible for the subcortical and putaminal lesions. It has also been suggested that putamen is particularly at risk to various pathologic processes because of its high metabolic demand and because it lies in the boundary zones of vascular perfusion⁸. The basis for the selective vulnerability in these regions remains unknown. It is probably a combination of factors, including cerebral microvascular anatomy and direct toxic effects of methanol metabolites, that causes the characteristic distribution of pathologic findings, including severe alterations of subcortical white matter and central gray matter alteration with sparing of peripheral gray matter⁹.

CONFLICT OF INTEREST

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

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