Role of Imaging in Acute Methanol Intoxication - A Case Series

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Abstract:
Methanol is a clear, colorless, highly toxic volatile liquid with an odour and taste similar to ethanol. Acute methanol poisoning produces sever metabolic acidosis and serious neurological symptoms. CT (Computed Tomography) and MRI (Magnetic Resonance Imaging) are able to demonstrate toxic effects of methanol intoxication in the CNS (Central Nervous System) including putaminal necrosis with or without hemorrhage and pathologies involving subcortical white matter, hippocampus, optic nerve, tegmentum, cerebral gray matter and cerebellum. Here we present three cases demonstrating different CNS changes demonstrated in MRI.

Keyword: *Computed tomography, Magnetic Resonance Imaging, Central Nervous system, Methanol Intoxication.*

Introduction:

Methanol is a clear, colorless, highly toxic volatile liquid with an odour and taste similar to ethanol. It is used as a constituent of a large number of commercially available solvent. Acute methanol intoxication appears after accidental, suicidal oral ingestion of industrial solvent and cleaning antifreeze liquid or occasionally is due to fraudulent adulteration of wine or other alcoholic beverage[1]. Acute methanol poisoning produces sever metabolic acidosis and serious neurological symptoms, including sever visual impairment, extrapyramidal signs and coma[2]. Optic neuropathy and putaminal necrosis are the two main complication of this poisoning, generally occurring in combinations after sever intoxication. Surviving patients usually show permanent sequelae like residual visual or consist of bilateral blindness, motor dysfunction including rigidity, hypokinesia and other parkinsonian like signs[3]. CT and MR imaging are able to demonstrate toxic effects of methanol in the CNS. Putaminal necrosis with or without hemorrhage are the most frequent reported findings. Other affected areas that reported in the literature are subcortical white matter, hippocampus, optic nerve, tegmentum, cerebral gray matter and cerebellum[1].

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Case Reports:

Case-1

A 35 years old male patient with altered consciousness was brought in to emergency department with history of acute alcohol intake few hours prior to presentation. On neurological examination, the Glasgow Coma Scale was 10. An arterial blood gas analysis that showed high anion gap metabolic acidosis. Provisional diagnosis was alcoholic intoxication. Patient was admitted in ICU. Initially CT scan of brain was done, which was normal. As patient condition was not improving, MRI of brain was done after 2 days. MRI showed putaminal iso to hypointense signal on T1-weighted and hyper intense signal on T2-weighted image representing putaminal necrosis. Based on these findings most probable diagnosis of Methanol intoxication was suggested. Blood sample was sent for measuring the methanol level, which was high.

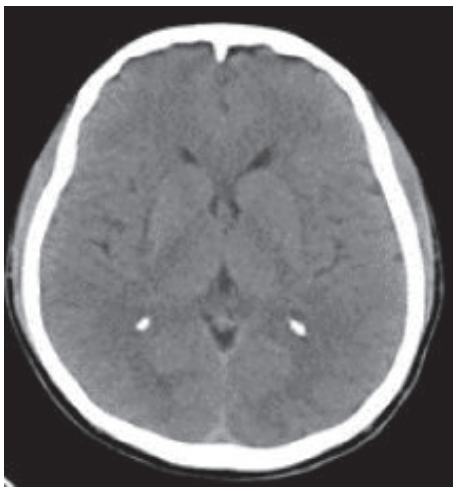

Fig -1: *Normal CT scan of brain*

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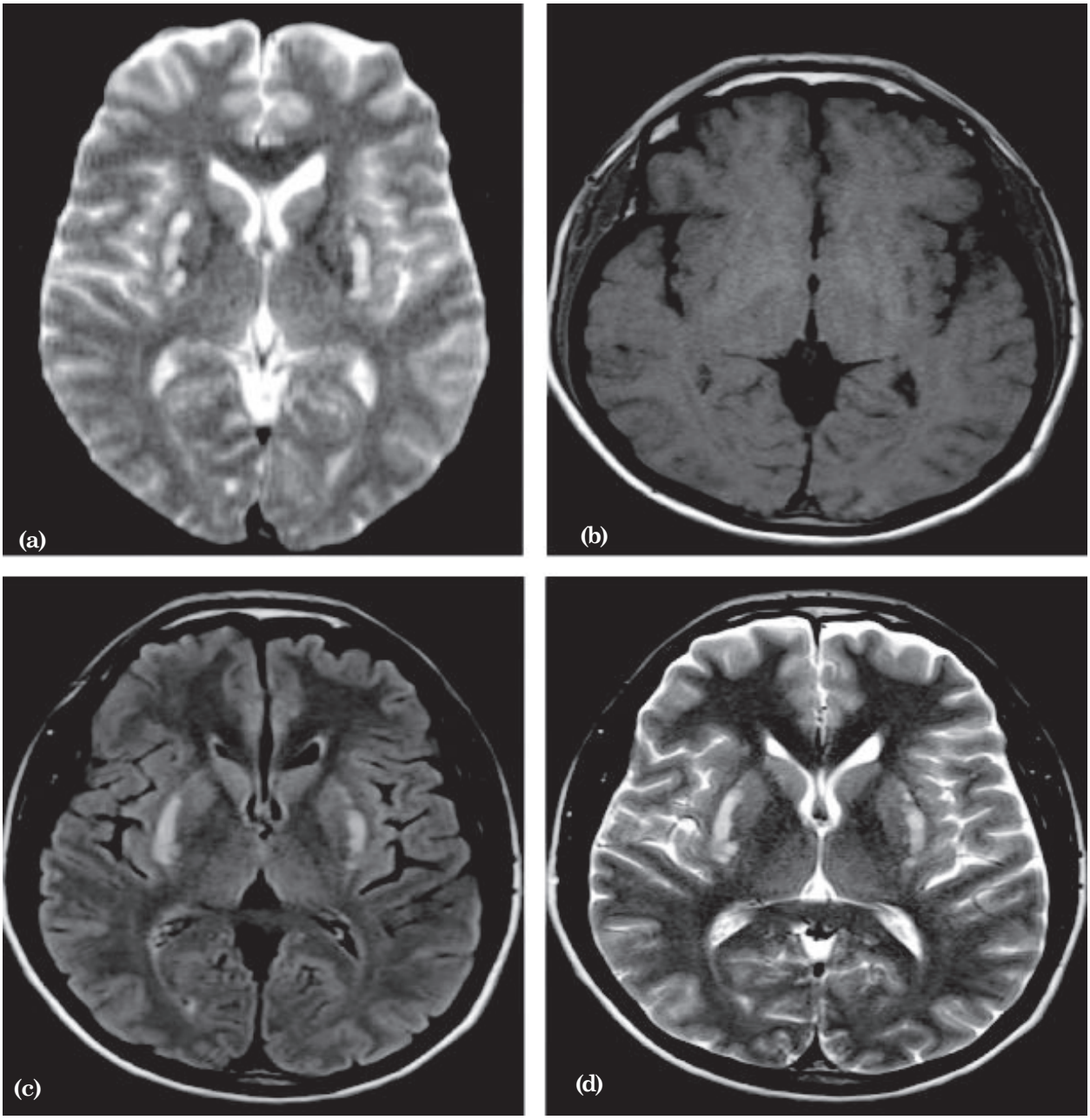

Fig.-2: a, b, c & d: *Axial ADC, T1, FLAIR & T2W images showing bilateral necrosis of putamen.*

Case-2

A 38 years old male patient presented at emergency with weakness and blurred vision with accidental intoxication. CT was normal and arterial gasometry showed systemic metabolic acidosis with high anion gap. The patient was in a comatose state and was intubated and taken to the intensive care unit. Provisional diagnosis was methanol intoxication. Glasgow Coma Scale score was 7 on 5th day. CT scan done for further evaluation which showed confluent low attenuation in subcortical white matter and basal ganglia representing toxic edema. Later MRI was done which showed almost symmetrical subcortical white matter and basal ganglion involvement which was hypo intense on T1 and hyper intense on T2-weighted image consistent with edema. On 20th day patient was discharged with family counselling and education for patient care.

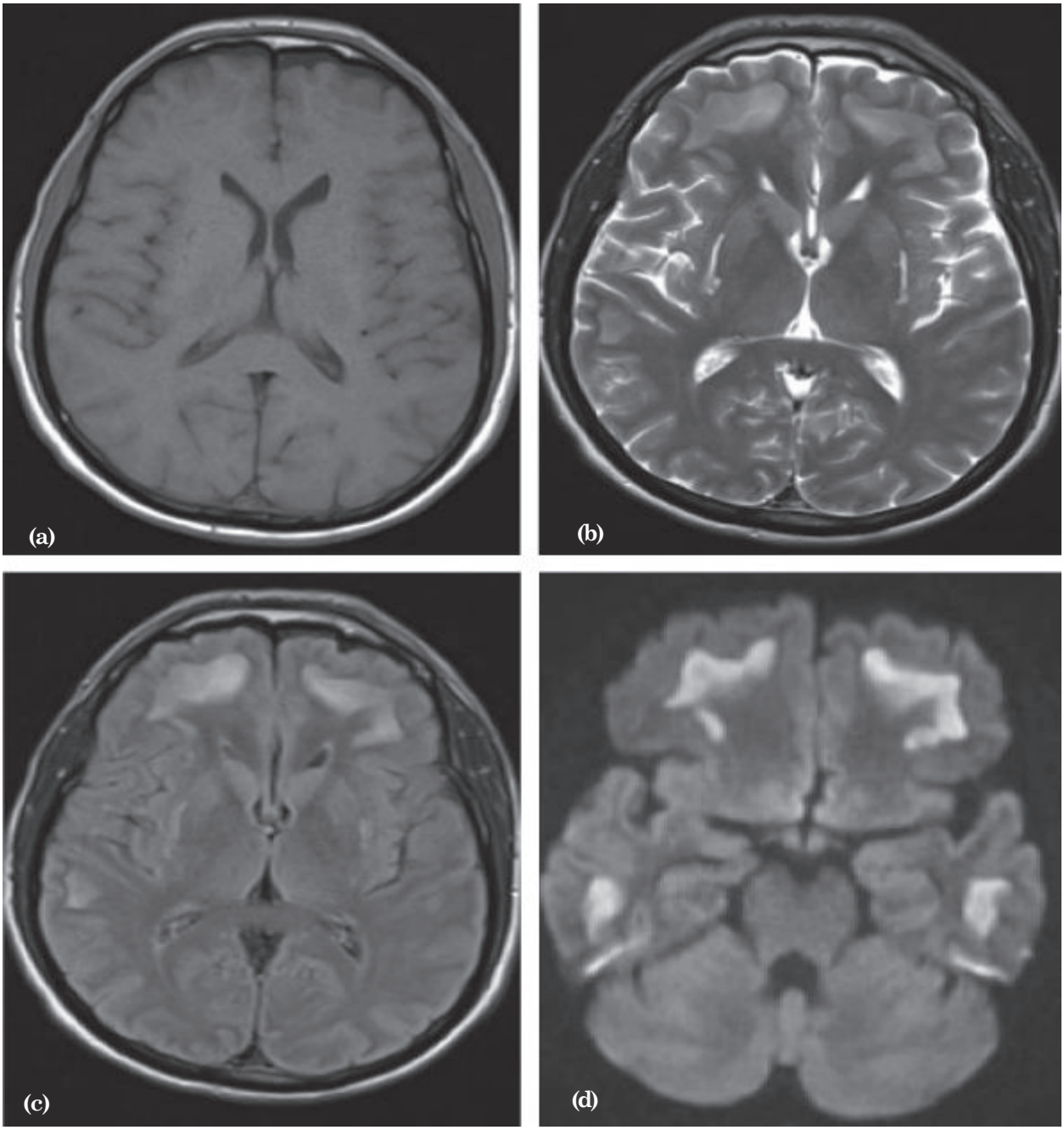

Fig 3: *(a, b, c & d) - Axial T1, T2, FLAIR and DWI showing subcortical white matter change with putaminal early necrosis.*

Case-3

A 39 years old man with history of habitual alcoholism presented with drowsy consciousness in the emergency department. He gave history of excess alcohol intake 1(One) day back and complained of suffering from dizziness, severe headache and also about blurring of vision after 24 hours of excessive alcohol intake. On admission, he was in coma with Glasgow Coma Scale score of 7. Arterial blood gas analysis showed systemic metabolic acidosis (pH-6.7, pCO_2-34 mm Hg; HCO_3-5.8 mmol/L) with a high anion gap (34mmol/L). He was intubated and transferred to the intensive care unit. After bicarbonate replacement therapy and hemodialysis, systemic acidosis persisted as

before. MRI was done on 8th day which showed Bilateral symmetrical putaminal hyperintensity in T2 and hypointensity in T1 weighted images. On 18th day CT scan was done which showed hypodensity in basal ganglia and subcortical white matter. CT findings were further reconfirmed by MRI on next day, that appeared as bilateral symmetrical T2 hyperintensity and T1 hypointensity in basal ganglia and confluent of subcortical white matter edema presented as T2 hyperintensity, involving the frontal, parietal, temporal, occipital lobes and also the cerebellum. After month of rehabilitation program was started for the patient which included exercise for muscle strengthening and functional task training. He was discharged on 45th day when he could sit, stand up independently and able to walk with a walker.

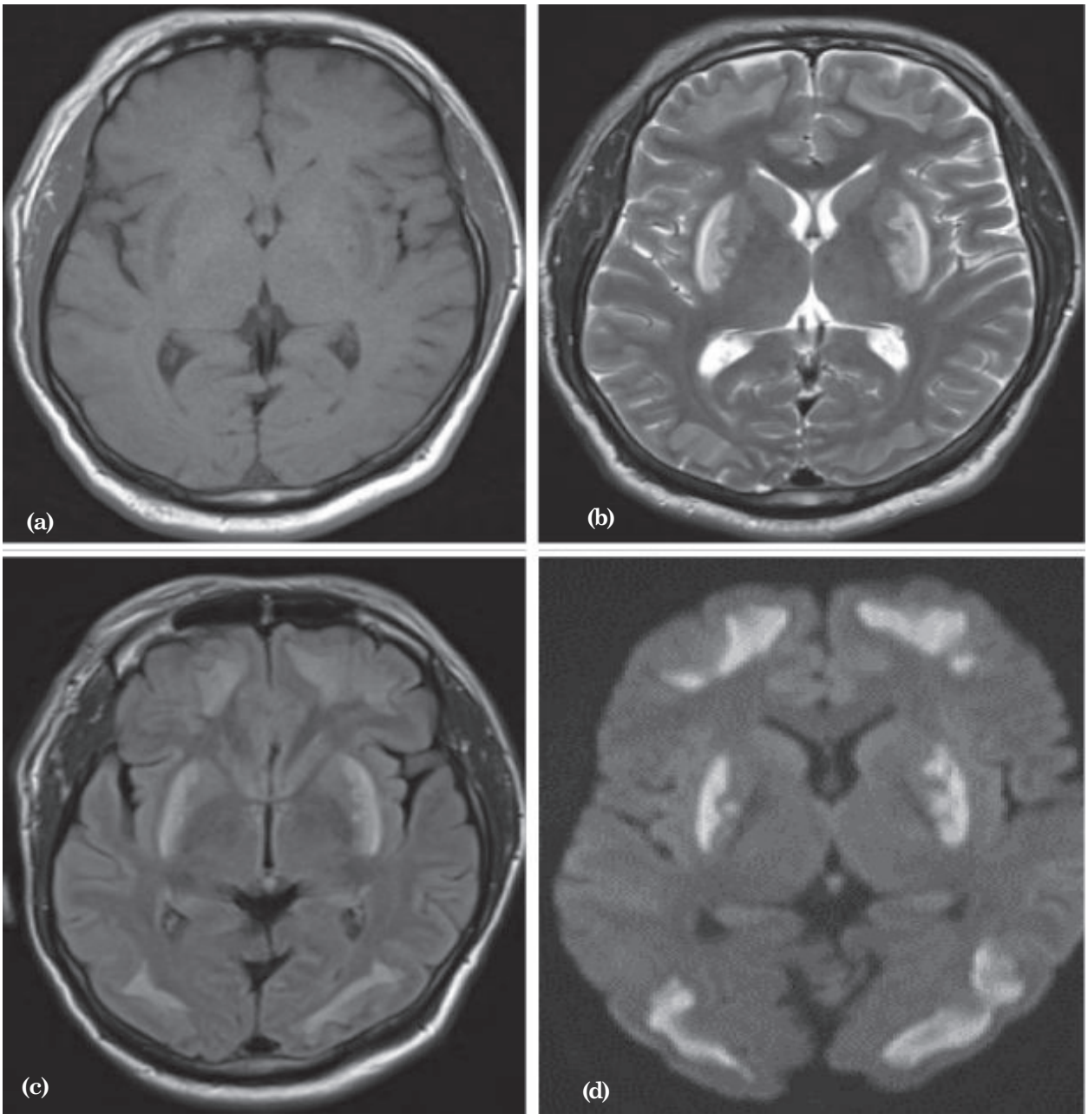

Fig 4: (a, b, c & d) - *Axial T1 hypointense, T2 & FLAIR hyper intense, DW restricted image showing putaminal non hemorrhagic necrosis and subcortical change.*

Discussion:

Acute methanol intoxication may be life threatening or can causes a permanent neurological deficit. Clinical presentation of methanol intoxication can vary greatly from patient to patient. A latent period of 12-24 hours within which methyl alcohol is metabolized in to formaldehyde and formic acid, these two chemicals are more toxic than methanol. Central nervous system symptoms in acute phase are common including vomiting, nausea, dizziness, headache, weakness, malaise. Large amount of methanol ingestion can result in seizure, stupor, coma and sometimes death. Neuroimaging helps in distinguishing methanol poisoning from other causes of acute unconsciousness in alcoholic patients such as hypoglycemic brain damage and carbon monoxide poisoning or head injury. Characteristic imaging finding of methanol poisoning are putaminal necrosis (most common) with varying degree of hemorrhage, subcortical and deep white matter lesion, cerebral and cerebellar cortical lesions, mid brain lesions, cerebral and interventricular hemorrhage, optic nerve necrosis even enhancement of necrotic lesions. Punaminal necrosis and hemorrhage probably result from the direct toxic effect of methanol metabolites and metabolic acidosis in the basal ganglia[4]. It has also been suggested that putamen is particularly at risk to various pathologic processes because of its high metabolic demand and it lies in the boundary zones of vascular perfusion[5]. The basis for the selective vulnerability in these regions remains unknown[6]. 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 matter.

Conclusion:

MRI is more informative than CT scan in Methanol intoxication. By the virtue of neuroimaging Methanol intoxication can be diagnosed earlier and we are able to detect the reversible and irreversible CNS changes, which determines the prognostic outcome of the patient and guide the clinician in patient management.

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