Case Report

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Methyl Alcohol Intoxication with Extensive Involvement of the Central Nervous System Shown By Magnetic Resonance Imaging

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Abstract

The focus of this study was to present methyl alcohol intoxication by citing clinical and laboratory, as well as radiological findings. A case of a 47-year-old man with a 20-year history of alcohol use disorder, brought to the emergency department in a coma. He had headache and blurred vision symptoms that started after drinking cologne and spirits. He had a Glasgow Coma Scale score of four. Increased anion gap metabolic acidosis was detected. Diffusion weighted imaging showed diffuse restriction regions in the subcortical white matter, cerebellar hemispheres, brain stem, bilateral optic nerve and putamen. He treated with hemodialyze, sodium bicarbonate, dextrose and ethanol infusion. However, the patient died on the seventh day. Bilateral putaminal necrosis is the most characteristic radiological finding, but white matter necrosis rarely reported in the literature. As in our case, such extensive involvement of the brain may be correlated with mortality in the acute period.

Keywords: Methyl alcohol; Methanol; Intoxication; Magnetic resonance imaging.

Introduction

A Acute methanol poisoning is frequently seen in suicidal or accidental. Drinking cologne and spirits to get drunk is not a usual practice, however, it can be seen in rare cases of alcohol use disorder when ethyl alcohol is not available.

The aim of this report is to emphasize the recognition of methanol intoxication in respect to clinical and radiological features, and the importance of early diagnosis and timely treatment. In addition, it is thought that the diffuse involvement of the Central Nervous System (CNS) in neuroradiological imaging may be correlated with the risk of death in the acute phase.

Case Report

A 47-year-old male was intubated due to respiratory arrest, referred to the emergency department on admission. According to the medical history taken from his family, he had a history of alcohol abuse for 20 years. Thirty hours before admission he had ingested intentionally an unknown amount of methanol to get drunk. It was learned that he had headache and blurred vision symptoms started 24 hours after ingestion, gradually became unconscious. Although the patient did not receive medication for sedation, he had a Glasgow Coma Scale (GCS) score of 4/15 (E1V1M2).

Biochemical investigation revealed wide anion gap metabolic acidosis. Methyl alcohol levels were not measured due to technical inability. The laboratory findings are shown in Tables 1 and 2.

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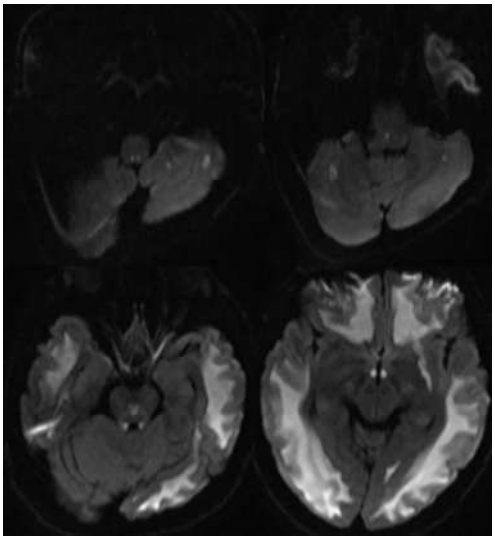
Table 1: Investigated laboratory findings related to acute methanol intoxication

Detected hematological and biochemical abnormalities	
Serum osmolality	286.4 μmol/kg
Blood ethanol level	Below 0.1 g/L
Arterial blood gas	
pH	6.7
pO_2	406.9 mm Hg
pCO_2	20.0 mm Hg
HCO_3	3.0 μmol/L
Base deficit	-38.2 μmol/L

Table 2: Other laboratory findings examined in blood

Detected hematological and biochemical abnormalities	
Glucose	44 mg/dl
Urea	60 mg/dl
Creatinine	2.0 mg/dl
Aspartate Aminotransferase (AST)	73 u/L
Gamma glutamyl transferase (GGT)	80 u/L
Lactate dehydrogenase (LDH)	436 u/L
Amylase	667 u/L
Potassium	2.9 μmol/L
White blood cell (WBC)	18.5 10^9/L
Hematocrit (HCT)	55%
Mean corpuscular volume (MCV)	102.3 fl
International normalized ratio (INR)	1.3
Prothrombin time (PT)	15.5 s
Activated partial thromboplastin time (aPTT)	66.6 s

Brain Computed Tomography (CT) and Diffusion weighted imaging (DWI) and Apparent Diffusion Coefficient (ADC) map were performed on admission. CT showed bilateral putaminal low attenuation, DWI demonstrated bilaterally putaminal restricted diffusion, ADC maps demonstrated low values suggesting necrosis in the same areas. Conventional Magnetic Resonance Imaging (MRI) was performed while DWI and CT were repeated on the third day of exposure and showed new lesions (CT performed after MRI) (Figs. 1A-E).



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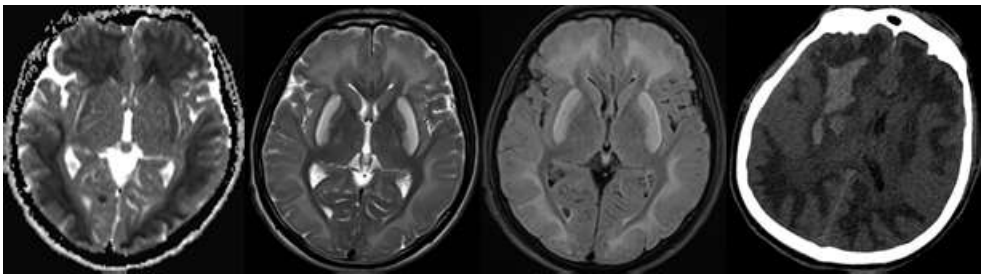

B C D E

Fig. 1: Methanol-induced brain lesions (A) – DWI, (B) – ADC maps demonstrated restricted diffusion in the cerebellum, brain stem, putamen, subcortical white matter; (C) – T2-weighted, (D) – T2 flair MRI showed hyperintensity in the same regions and (E) – CT also showed right putaminal and frontal hemorrhage.

Hemodialyze, intravenous (i.v.) sodium bicarbonate and dextrose infusions, 40% ethanol from the nasogastric until i.v. form was provided, followed by i.v. ethanol infusion was performed. Blood ethyl alcohol levels were monitored frequently. Fornepizol could not be given because it was not found in the country. The patient did not respond to treatment and died on post-admission day seven.

Discussion

A 47-year-old male patient who was brought to the emergency department in a coma, who had a history of alcohol use disorder, who had been drinking cologne and spirits to get drunk, with diffuse subcortical white matter, brain stem, optic nerve, cerebellum, basal ganglia involvement on brain imaging, whose follow-up resulted in death, is presented.

The main feature of methanol intoxication is the latent period between ingestion and onset of symptoms. Symptoms and signs are nausea, vomiting, impaired visual function, ranging from blurred vision to complete blindness, change of consciousness, ranging from confusion to coma, respiratory arrest and death.[1]

The CNS effects of acute methyl alcohol intoxication are caused by its toxic metabolite, formic acid. Formic acid inhibits cytochrome oxidase, a mitochondrial enzyme that is functionary for oxidative phosphorylation; this inhibition leads to anoxia, edema and ultimately cell death.[2]

Especially if medical history is insufficient, diagnosis of methanol poisoning is difficult. Therefore neuroimaging findings and clinical manifestations of methanol intoxication should be better understood and recognized. It was aimed to recognize methyl alcohol intoxication by presenting patient's clinical features, blood biochemical examinations and radiological findings, and interrogates the history of alcohol use disorder and to suggest methanol intoxication in the differential diagnosis. In addition, it was aimed to emphasize the importance of the early treatment of this intoxication which could result in death.

MRI and CT as neuroradiological imaging techniques are able to show toxic effects of methanol intoxication at the CNS but the neuroimaging finding can be normal in acute phase.[3] Putaminal necrosis is the most common and characteristic,[4] but not a specific radiological finding of methanol intoxication. Subcortical white and grey matter necrosis and edema, cortical cerebellar and optic nerve lesions, intracerebral hemorrhage, tegmental necrosis have all been described as well as other brain lesions, in previous reports.[5,3]

Here, a case with a very diffuse involvement of CNS, which is rarely detected, is presented. Conventional MRI and DWI showed diffuse subcortical white matter lesions accompanied by bilateral basal ganglia, brainstem, optic nerve and cerebellum. There was no case that showed the involvement of all these areas together in the literature. This is a unique case that showed all MRI finding seen in the methanol poising.

Target areas of methanol poisoning are basal ganglia especially putamen. It is not precisely known why especially putamen is affected—possibly because of its high metabolic demand, its cerebral microvascular anatomy or direct toxic effects of methanol metabolites.[4] Probably it is multifactorial.

Also, methanol intoxication has a characteristic necrosis structure in relation to parenchymal involvement and it is remarkable that subcortical arcuate fibers are preserved. The difference in the involvement of gray and white matter is due to the fact that the vascular networks where these

regions provide blood and venous drainage are different. Although there are rich vascular networks for arterial circulation of gray matter and subcortical arcuate fibers, arterial circulation of deeper white matter is provided by longer and larger caliber vessels that reach through the cortex without branching. Thus, while diffuse cerebral edema disrupted arterial and venous drainage of the deep white matter, subcortical arcuate fibers continue to be perfused due to rich anastomosis vascular networks. In addition, penetrating vessels that deliver the blood supply to the deep white matter are more susceptible to hypoxia-induced vasospasm.[6]

Treatment of acidosis and removal of toxic substances are intended. Therapeutic procedures are gastric lavage when the patient arrives early, ethanol or fomepizole, sodium bicarbonate, folinic acid and hemodialysis. Fomepizole and folinic acid could not be given to the patient due to technical insufficiency, but all other treatment options were applied quickly. Early diagnosis and timely treatment can be life-saving. While one-third of untreated cases result in death, sequelae such as blindness, dementia and parkinsonism.[2] Severe metabolic acidosis, coma at the presentation, brain edema, infarction and increased pCO_2 are associated with poor prognosis. There is no statistically significant association between prognosis and radiological abnormalities.[7,5] However, comatose patients showed more diffuse involvement of CNS.[5]

Considering the poor prognosis and comatose status of this presented case it is avowable that it was a serious intoxication in this case and it can be suggested that increasing degree of poisoning damage started from the optic nerve and the central gray matter and may cause spread to areas other than peripheral gray matter like subcortical white matter, cerebellum and brainstem. MRI and especially DWI should be performed as soon as possible to understand the degree of methanol intoxication. This case was found to be worth presenting due to the detection of such diffuse CNS lesions. There may be a correlation between the clinical outcome and the diffusiveness of radiological abnormalities. It may be suggested that the more extensive CNS involvement is indicative of poor prognosis and worse clinical outcomes. In addition, it may also be correlated with the degree of intoxication and the risk of death in the acute phase. This case is also important due to the progression of MRI findings on the 2nd and 4th days of methanol exposure.

Some of the previous studies have used diffusion weighted imaging in methanol poisoning.[5] In this case, DWI demonstrated bilaterally putaminal hyperintensity, ADC maps demonstrated low values corresponding to the lesions at DWI with restricted diffusion at the presentation. This case developed new lesions in the subcortical white matter, brainstem, optic nerve, cerebellum and CT demonstrated right basal ganglia hemorrhage on the third day after admission.

Conclusion

Especially in unconscious patients, both clinicians and radiologists should be familiar with the symptoms and recognize the finding of methanol poisoning and begin treatment early.

Prognosis studies involving a large number of patients associated with methanol intoxication may reveal the relationship between the degree of mortality and the extent of brain parenchyma involved in neuroimaging.

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