OLGU SUNUMU / *CASE REPORT*

Imaging Findings for Methanol Intoxication

Metanol İntoksikasyonunda Görüntüleme Bulguları

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ABSTRACT

Methanol is a highly toxic substance. Acute methanol intoxication is a rare accidental or suicidal intoxication with high morbidity and mortality rates. Because of its high toxicity, early diagnosis and management is very important in such patients. Imaging (computed tomography "CT" and magnetic resonance imaging "MR") plays an important role in diagnosis and management of these patients. With this rare suicidal case we want to emphasize the important imaging findings of methanol intoxication.

Key words: *computed tomography; magnetic resonance imaging; metanol intoxication*

ÖZET

Metanol yüksek toksisiteye sahip bir maddedir. Akut metanol zehirlenmesi yüksek morbidite ve mortaliteye sahip, kazayla ya da intihar amaçlı görülen nadir bir zehirlenmedir. Yüksek toksisitesinden dolayı bu hastalarda erken tanı ve yönetim bu hastaların prognozu için çok önemlidir. Radyolojik görüntüleme (bilgisayarlı tomografi "BT" ve manyetik rezonans görüntüleme "MR") bu hastaların tanı ve yönetiminde çok büyük öneme sahiptir. Bu nedenle, bu nadir intihar amaçlı metanol zehirlenmesi vakası ile önemli radyolojik görüntüleme bulgularına değinilmek istendi.

Anahtar kelimeler: *bilgisayarlı tomografi; manyetik rezonans görüntüleme; metanol zehirlenmesi*

Introduction

Methanol also known as methyl alcohol, carbinol or wood alcohol is a chemical with the formula CH_3OH. Methanol is the simplest alcohol, and is a light, volatile, colorless, flammable liquid with a distinctive odor very similar to that of ethanol[1]. It also occurs naturally in humans, animals and plants. Foods such as fresh fruits and vegetables, fruit juices, fermented beverages and diet soft drinks containing aspartame are the primary sources of methanol in the human body[2].

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Methanol has a high toxicity in humans with two mechanisms. First, methanol can be fatal due to its central nervous system (CNS) depressant properties in the same manner as ethanol poisoning. Second, in a process of toxication, it is metabolized to formic acid. Formic acid is toxic because it inhibits mitochondrial cytochrome C oxidase, causing the symptoms of hypoxia at the cellular level, and also causing metabolic acidosis[3].

Most methanol intoxications occur as a result of drinking beverages contaminated with methanol or from drinking methanol-containing products. In the industrial setting, inhalation of high concentrations of methanol vapor and absorption of methanol through the skin are as effective as the oral route in producing toxic effects. The initial symptoms of methanol intoxication include CNS depression, headache, dizziness, nausea, lack of coordination, and confusion. Sufficiently large doses (the median lethal dose is typically 100 ml or 1–2 mL/kg body weight of pure methanol) can cause unconsciousness and death[4].

Computed Tomography (CT) and magnetic resonance (MR) imaging are able to demonstrate toxic effects of methanol in CNS so imaging is very important for the diagnosis and prognosis of methanol intoxication. Putaminal necrosis with or without haemorrhage are most frequent reported findings[5]. Other affected areas that are reported in literature are subcortical white matter, hippocampus, optic nerve, tegmentum, cerebral gray matter and cerebellum[5,6].

Case Report

A 56 year old male who reportedly ingested a large amount of methanol for suicidal purpose, was admitted to emergency room (ER) in another center because of visual impairment and unconsciousness. His mental status deteriorated in a short period of time and

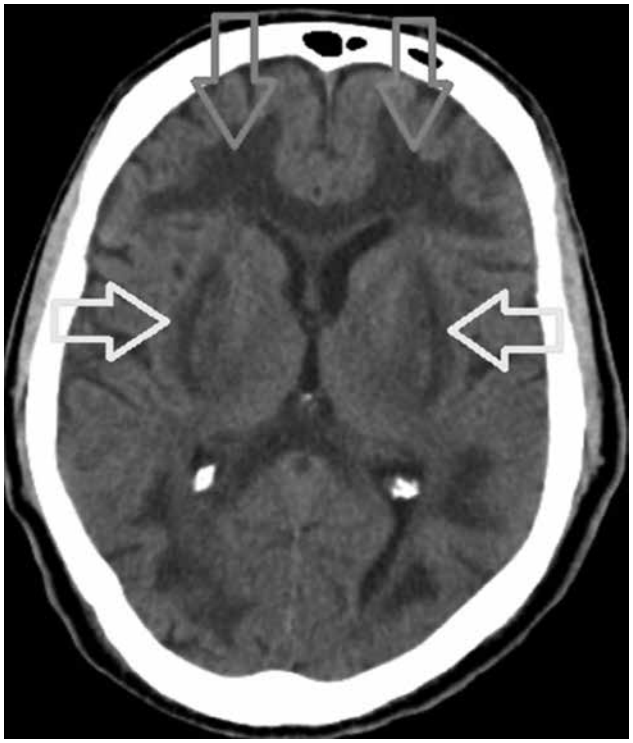

Figure 1. *In non-contrast axial carnial CT scan; there is diffuse low attenuation areas in subcortical white matter (down arrows) and both putamina with slightly high attenuation putaminal foci (left/right arrows) consistent with hemorhhage.*

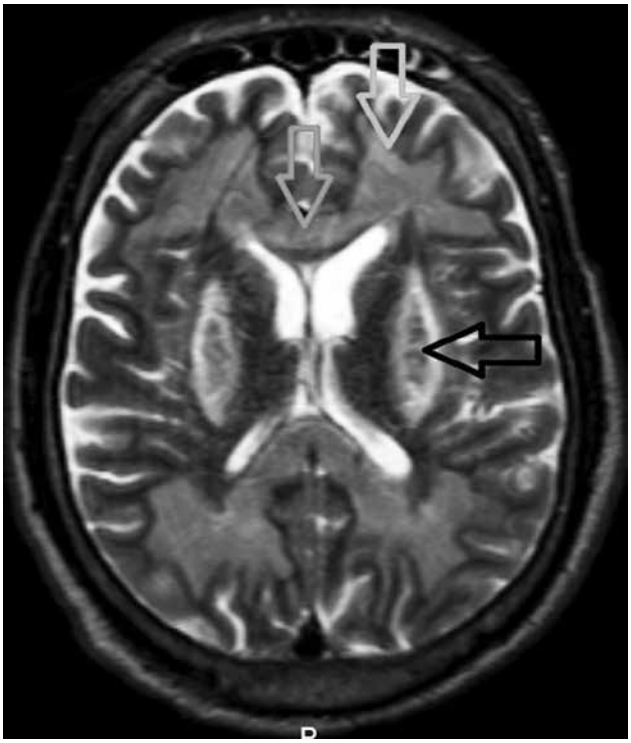

Figure 2. *In cranial MR on day 6; T2 weighted image (WI) axial image shows subcortical white matter (down arrow above), corpus callosum (down arrow below) and basal ganglia hyperintensity, low signal intensity bilateral putaminal foci (left arrow) consistent with putaminal hemorrhage.*

then he referred to our hospital. When he presented in our ER he was in a critical unconscious state with Glasgow's coma scale of 3/15. Arterial blood gas investigation showed (pH: 7.009, pCO_2:42.3, CO_3H: 9.7, BE:-19) severe systemic metabolic acidosis with high anion gap (23 mEq/L). He was intubated and taken to intensive care unit (ICU). The patient remained comatose and died 16 days of admission.

For imaging first he underwent non contrast carnial CT scan and it shows diffuse low attenuation areas in subcortical white matter and both putamina with high attenuation putaminal foci consistent with hemorrhage (Fig. 1). In cranial MR on day 6; T2 weighted image (WI) and fluid attenuated inversion recovery (FLAIR) axial images show subcortical white matter, corpus callosum and basal ganglia hyperintensity, low signal intensity bilateral putaminal foci (Fig. 2 and 3). T1WI image shows low signal intensity in subcortical white matter and basal ganglia with high signal intensity foci in both putamina (Fig. 4a). Gradient echo sequence (GRE) image shows low signal intensity foci in both putamina consistent with putaminal hemorrhage (Fig. 4b).

Discussion

Acute methanol intoxication is a rare accidental or suicidal intoxication. It has also been described as a result of fraudulent adulteration of alcoholic drinks. The clinical presentation of methanol intoxication varies greatly between patients. A latent period of 12–24 hours often follows methanol ingestion. The latent period most likely correspond to the time period in which methyl alcohol is metabolized into more toxic chemicals formaldehyde (CH_2O) and formic acid (CH_2O_2)[7,8].

Acute methanol intoxication produces severe metabolic acidosis and serious neurologic consequences. Most patients note visual disturbances, secondary to optic nerve necrosis or demyelination, as one of the

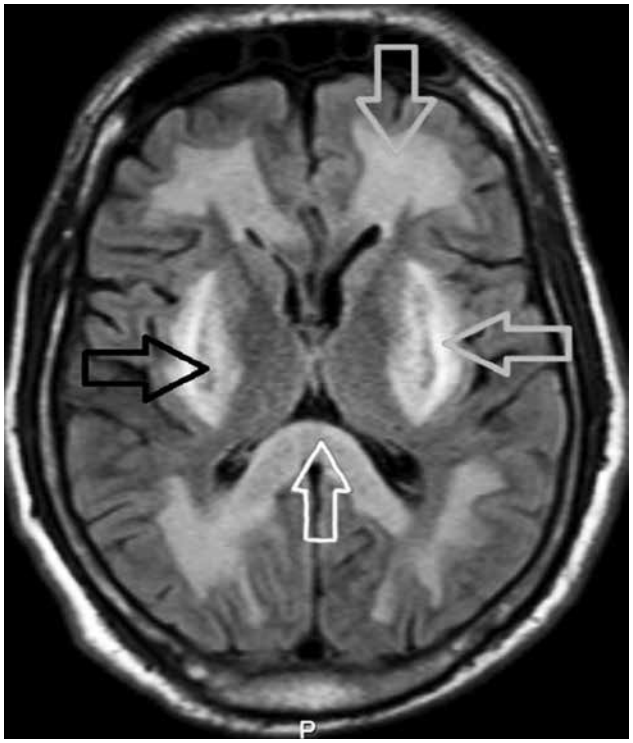

Figure 3. *In cranial MR on day 6; Fluid attenuated inversion recovery (FLAIR) axial image shows subcortical white matter (down arrow), corpus callosum (up arrow) and basal ganglia hyperintensity (left arrow), low signal intensity bilateral putaminal foci (right arrow), consistent with putaminal hemorrhage.*

first symptoms as in our case. CNS 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 based on the presence of severe metabolic acidosis with high anion and osmolar gap and high serum methanol levels. In acute methanol intoxication to prevent the conversion of methanol into toxic metabolites, ethanol is administered because of its affinity to alcohol dehydrogenase enzyme is 10–20 times greater than that of methanol[6]. Other therapeutic procedures include gastric lavage, correction of acidosis with sodium bicarbonate ($NaHCO_3$), folic acid ($C_{19}H_{19}N_7O_6$), and secondary detoxication with hemodialysis.

In imaging, the most characteristic MR findings in methanol toxicity are bilateral putaminal necrosis, which may varying degrees of hemorrhage[9]. This finding is by no means specific to methanol toxicity but is seen also in variety of conditions such as Wilson's disease, Leigh's disease, Kearns-Sayre syndrome, carbon monoxide (CO) inhalation, hypoxic-ischaemic injury, trichloroethane ($C_2H_3Cl_3$) poisoning and acute cyanide (CN-) intoxication (7). Putaminal damage is

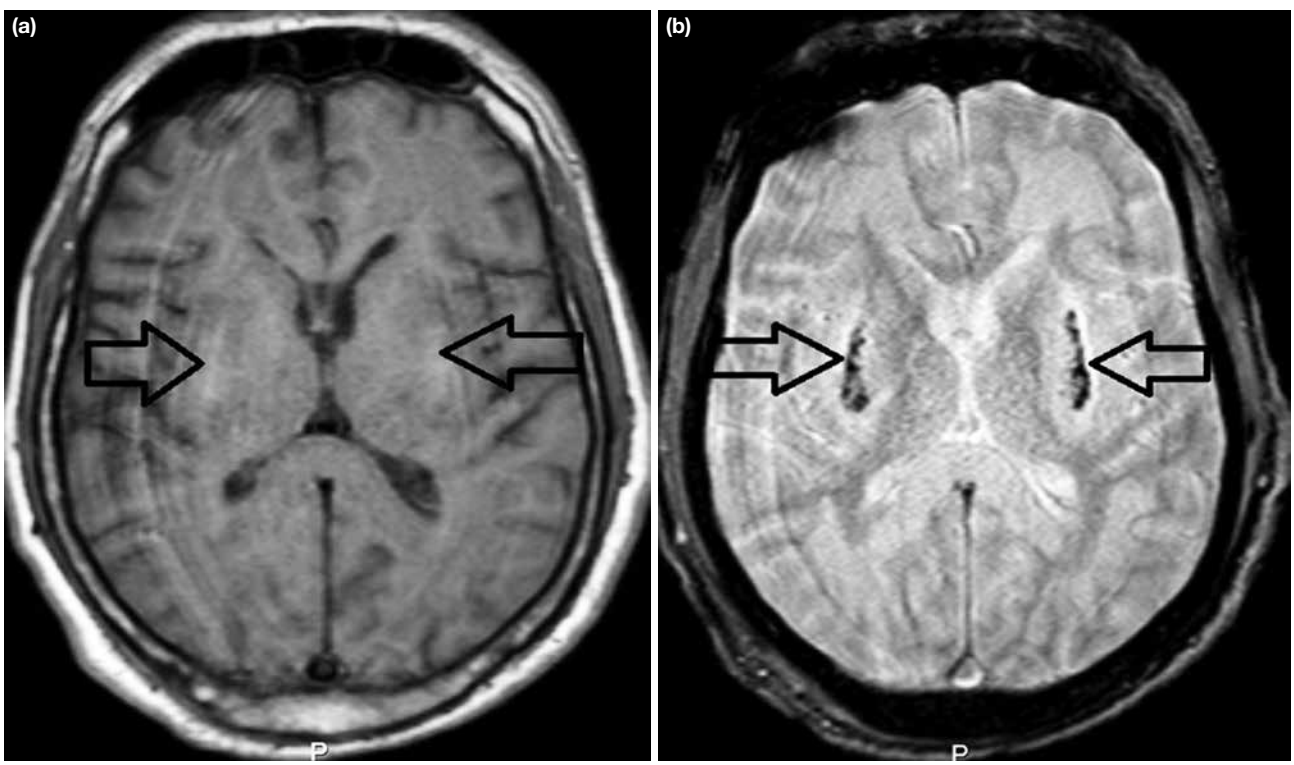

Figure 4. a, b. *In T1WI axial image shows slightly low signal intensity subcortical white matter and basal ganglia lesions with slightly high signal intensity foci in both putamina (**a**, arrows). GRE image shows low signal intensity foci in both putamina consistent with putaminal hemorrhage (**b**, arrows).*

probably result from the direct toxic effects of methanol metabolites and metabolic acidosis in the basal ganglia[10]. Cerebral and intraventricular hemorrhage, cerebellar necrosis, diffuse cerebral edema, and optic nerve necrosis all have been described in severe methanol intoxication. Optic nerve demyelination secondary to myelinoclastic effect of formic acid (CH_2O_2) has been suggested as responsible for optic nerve damage with or without axonal loss. But in our case the images from optic nerve, there were no imaging findings of optic nerve damage. It is possible that direct toxic effects of methanol metabolites also were responsible for the subcortical an putaminal lesions[6,10]. 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 perfusion, though for some authors the nature of the distribution of the lesions seems to be opposite of a vascular cause. The basis for the selective vulnerability in these regions remains unknown[10].

In conclusion, when symmetrical lesions are detected in the basal ganglia and white matter along with sudden visual disturbances, there can be a long list of differential but correct diagnosis could be reached if history of methanol contact is available. Since early diagnosis may improve the prognosis in acute phase, methanol intoxication should be considered in the differential diagnosis such lesions on MR and CT examinations.

References

1. National Institute for Occupational Safety and Health. The emergency response safety and health database: Methanol 22 August 2008.
2. Turner C, Spanel P, Smith D. A longitudinal study of methanol in the exhaled breath of 30 healthy volunteers using selected ion flow tube mass spectrometry. Physiol Measur 2006;27(7):637–48.
3. Liesivuori J, Savolainen H. Methanol and formic acid toxicity: biochemical mechanisms. Pharmacol Toxicol 1991;69(3):157–63.
4. Wikipedia.org [search:methanol toxicity]. Wikimedia foundation, Inc. [updated: 17 October 2016]. Available from: http://en.wikipedia.org/wiki/methanol_toxicity
5. Halavaara J, Valanne L, Setala K. Neuroimaging supports the clinical diagnosis of methanol poisoning. Neuroradiol 2002;44:924–8.
6. Blanco M, Casado R, Vazquez F, Pumar JM. CT and MR imaging findings in methanol intoxication. AJNR Am J Neuroradiol 2006;27:452–4.
7. Azeemuddin M, Naqi R. MRI findings in methanol intoxication: a report of three cases. J Pak Med Assoc 2012;62(10):1099–101.
8. Schneck SA. Methyl alcohol. In: Vinken PI, Bruyn GW, editors. Handbook of clinical neurology, vol 37. Amsterdam: North Holland; 1989;351–60.
9. RubinstenGn D, Escott E, Kelly JP. Methanol intoxication with putaminal and white matter necrosis: MR and CT findings. AJNR Am J Neuroradiol 1995;16:1492–4.
10. Gaul HP, Wallace CJ, Auer RN, Fong TC. MR findings in methanol intoxication. AJNR Am J Neuroradiol 1995;16:1783–6.