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Radiology Findings in a Case of Acute Methanol Intoxication Complicated with Intracranial Haemorrhage

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ABSTRACT

Objective: Methanol intoxication is rare and it can be fatal. This case report aimed to describe the radiological findings in acute methanol intoxication complicated with intracranial haemorrhage in a young patient. A 32-year-old man applied to emergency department with a major complaint of nausea, vomiting, double vision, and confusion. Glasgow coma score (GCS) was 7 at the initial referral. Laboratory tests showed high anion gap metabolic acidosis. Acute methanol intoxication was diagnosed by aid of the patient anamnesis. Diffusion-weighted MRI (DW-MRI) showed similar diffusion restriction lesions in the putamen and cingulate gyrus of both cerebral hemispheres. The patient had a cardiac arrest during follow-up in the emergency department. He was converted to sinus rhythm with the interventions, intubated, and taken to the intensive care unit. GCS reduced to 3 during follow-up at the 4th day. Cerebral intraparenchymal haemorrhage at the level of left basal ganglia was observed in the brain computed tomography (CT). He was operated however remained comatose after the operation and died at the 8th day of admission. Possibility of methanol intoxication should be included in the differential diagnosis in patients with altered consciousness, vision disturbances and a high anion gap admitted to the emergency service. DW-MRI may be helpful in diagnosis by detecting symmetrical diffusion restrictions at the basal ganglia. Additionally radiologic methods like CT can be used to determine complications such as bleeding that may occur during the follow-up of the cases.

Keywords: Methanol Intoxication, Putamen, Diffusion Restriction, MRI, Cerebral Haemorrhage.

İntrakraniyal Kanama ile Komplike olan Bir Akut Metanol İntoksikasyonu Vakasında Radyolojik Bulgular

ÖZ

Amaç: Metanol intoksikasyonu nadir görülen bir antidedir ve ölümcül olabilir. Bu olgu sunumunda, intrakraniyal kanama ile komplike olan metanol intoksikasyonlu genç hastada radyolojik bulguların tanımlanması amaçlanmıştır. Otuz iki yaşındaki bir erkek hasta bulantı, kusma, çift görme ve bilinç bulanıklığı ana şikayetleri ile acil servise başvurdu. İlk başvuruda Glasgow koma skoru (GKS) 7 idi. Laboratuvar testlerinde yüksek anyon açıklı metabolik asidoz görüldü. Hastanın anamnezi ile birlikte akut metanol intoksikasyonu tanısı konuldu. Difüzyon ağırlıklı MRG (DA-MRG) incelemesinde, her iki putamen ve singulat girusta benzer difüzyon kısıtlılığı görüldü. Hastanın acil servisteki takibi sırasında kardiyak arrest gelişti. Yapılan müdahalelerle sinüs ritmine döndürülüp, entübe edilerek yoğun bakıma alındı. Takibinin 4. gününde GKS 3'e düştü. Beyin bilgisayarlı tomografisinde (BT) sol bazal ganglion seviyesinde intraparenkimal hematoma izlendi. Hasta opere edildi ancak ameliyattan sonra da komada kaldı ve yatışının 8. gününde kaybedildi. Acil servise bilinç değişikliği, görme bozukluğu ile başvuran ve yüksek anyon açığı olan hastalarda metanol intoksikasyonu olasılığı ayırıcı tanıda yer almalıdır. DA-MRG'de, bazal ganglionlarda simetrik difüzyon kısıtlamaları görülmesi ayırıcı tanıda yardımcı olabilir. Ayrıca olguların takibinde oluşabilecek kanama gibi komplikasyonları belirlemek için BT gibi radyolojik yöntemler kullanılabilir.

Anahtar Kelimeler: Metanol Intoksikasyonu, Putamen, Difüzyon Kısıtlılığı, MRG, Beyin Kanaması.

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INTRODUCTION

Methanol is a quite poisonous fluid and has similar physical properties to ethanol. (Camurcuoglu et al 2022). Methanol intoxication often emerge as a result of counterfeiting drinking alcohol, mistakenly use of industrial products. (Sahar M et al 2016). Taken into to body methanol is converted to formic acid and lactic acid, because of these metabolites formed emerge to serious metabolic acidosis. Methanol intoxication causes various clinical symptoms by affecting important structures related to the central nervous system. (Hyun Jim Kim et al, 2017). Radiologic imagings are able to indicate detrimental effects of methanol in CNS therefore cross sectional imaging is very significant for the management of methanol intoxication. In these patients, putaminal necrosis, subcortical white matter, gray matter, optic nerve and cerebellum involvements are often presented. (Raisa F et al 2021; Gök M et al, 2017). In this case report, the radiologic findings of acute methanol intoxication and its complication in a young man were described.

CASE PRESENTATION

A 32 year-old man admitted to emergency department with a major complaints of nausea, vomiting, double vision, and confusion. The patient did not have a chronic disease and no history of drug use. He had a history of acute wild spirit intake 24 hours prior to the admission. In the physical examination, the patient's Glasgow Coma Scale (GCS) was 7. At first an arterial blood gas analysis showed a high anion gap metabolic acidosis (ph 6.98, HCO₃ 5.3, CO₂ 19.8, pO₂ 129). Residual blood methanol level could not be measured. The patient was treated with Na₂CO₃, ethanol and hemodialysis. Non-contrast brain CT that as the initial imaging modality performed approximately 24 hours after the methanol ingestion was normal (Figure 1).

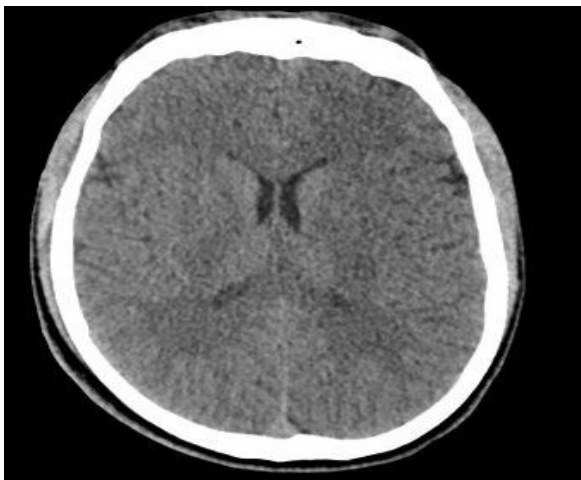


Figure 1: There was no distinguishable abnormality found in the axial plane non-contrast brain CT image at the level of basal ganglia.

Diffusion-weighted MRI (DW-MRI) examination performed immediately after non-contrast brain CT. DW-MRI showed symmetrical well defined marked diffusion restriction in bilateral putamen which appears hyperintense in DW images and suppressed in ADC map. In addition, two focal focus showing diffusion restriction in the bilateral cingulate gyri were observed (Figure 2).

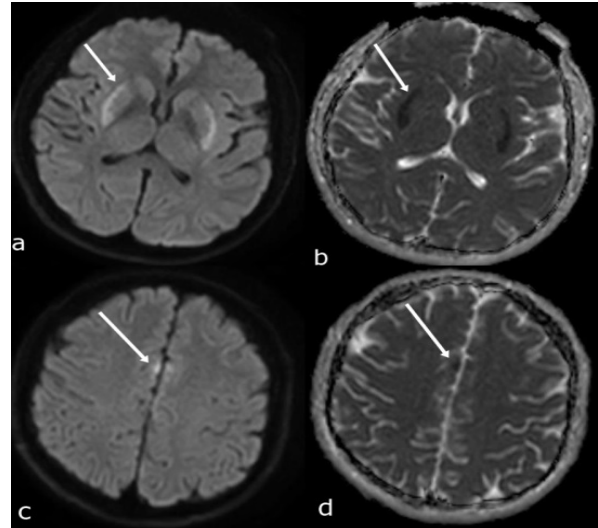


Figure 2: MRI shows marked diffusion restriction (arrows) which appears hyperintense in DW image and suppressed in ADC at the both putamens (a,b) and cingulate gyri (c,d).

Cardiac arrest became during follow-up in the emergency department cardiopulmonary resuscitation was performed. Ventricular fibrillation current was observed on the monitor. He was defibrillated with 200 joules. The patient returned to sinus rhythm after 15 minutes. He was intubated and taken to intensive care unit (ICU). During the follow-up GCS reduced to 3 on the 4th day in the ICU. Parenchymal hemorrhage was observed in the brain CT at the 4 level of left basal ganglia (Figure 3). The patient was operated due to intracranial high pressure. After operation, the patient remained comatose and died 8th day of admission.

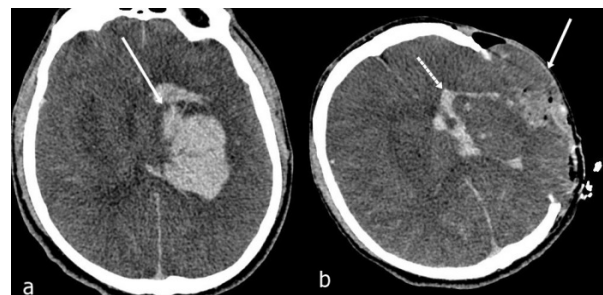


Figure 3: The preoperative CT shows the paraneural hemorrhage (arrow) the level of left basal ganglia (a). External brain herniation (dashed arrow) is seen after craniectomy in postoperative CT image (b).

DISCUSSION

Acute methanol intoxication due to oral ingestion of methanol may be as a result of an accident or suicide attempt (Sahar M et al 2016). Methanol is found in numerous industrial materials used in daily life in plenty of countries. (Hyun Jim Kim et al, 2017; Gök M et al, 2017). As less as 4 ml of methanol lead to blindness, almost 15 ml-30 ml of methanol and blood levels of 1500 mg/l are adequate to lead to death. (Raisa F et al 2021). Clinical signs emerge within 12-24 hours. In this time, happen for convert of methanol in the liver to its more harmful products and the simultaneous emerge of acidosis. Methanol is metabolized to toxic components resulting in varied clinical symptoms (Rumitha Kayastha et al, 2022). These symptoms range from nonspecific findings such as headache and dizziness to death. (Camurcuoglu et al 2022; Sahar M et al 2016; Hyun Jim Kim et al, 2017; Raisa F et al 2021; Gök M et al, 2017; Mehdi Mesri et al, 2021; Rumitha Kayastha et al, 2022; Nirdeh Jain et al 2013; Zeinali M et al, 2021; Dipoce J et al, 2012).

Methanol intoxication typical of influence to the putamen, optic nerves and retina bilaterally. In addition may be influence the other basal ganglia nuclei, cerebellum and subcortical white matter. The damage to basal ganglia may be derived from direct effect of toxic metabolite of methanol, formic acid, and the neural cells. Also, basal ganglia are more susceptible to acidosis in proportion to the rest of the brain. High metabolic demand of putamen or its microvascular structure may be cause more vulnerable to the toxic effects of formic acid. The myelinoclastic influence of the formic acid is supposed to be liable for optic nerve lesions or axonal damage. Complications such as permanent blindness due to optic nerve damage, parkinsonism due to basal ganglia damage, cerebral hemorrhage due to dialysis or heparin use, transverse myelitis and polineuropathy due to metabolic acidosis are possible to develop (Camurcuoglu et al 2022, Raisa F et al 2021, Mehdi Mesri et al, 2021, Nirdeh Jain et al 2013, Zeinali M et al, 2021). Methanol intoxication can be uncommonly affect of cerebral and cerebellar hemispheres. Intraventricular hemorrhage, diffuse cerebral edema and optic nerve necrosis may be lead to. There are rare cases of subarachnoid hemorrhage also reported. (Raisa F et al 2021). Diffusion restriction in methanol intoxication occurs due to necrosis. (Matthew P. Quinn et al 2020). There are many neurodegenerative, genetic, toxic, metabolic and genetic diseases that affect the basal ganglia symmetrically. Diseases such as hypoglycemic brain injury, carbon monoxide poisoning, hypoxic-ischemic encephalopathy, hyperammonemia, Wilson, Leigh, which causes restricted diffusion in the basal ganglia, should be considered in the differential diagnosis. But, diffusion MRI examination, showed symmetrical diffusion restriction in the dorsolateral putamen suggests methanol toxicity. (Matthew P.

Quinn et al 2020). Radiological (MRI) findings in related to methanol intoxication are typical and contain putaminal necrosis (Non-hemorrhagic or hemorrhagic), white matter (subcortical and deep), cortical (cerebral and cerebellar) and midbrain lesions, hemorrhage (Intraaxial-ventricular), enhancement of necrotic lesions. (Nirdeh Jain et al 2013). Unlike the cases in the literature, there was cingulate gyrus involvement found in DW-MRI in this case.

In treatment, intravenous ethanol is administered to decrease the formation of methanol metabolites. For balance of metabolic acidosis preferred to intravenous sodium bicarbonate, gastric lavage and hemodialysis. Fomepizole and folinic acid are supportive methods (Camurcuoglu et al 2022, Raisa F et al 2021, Mehdi Mesri et al, 2021, Zeinali M et al, 2021). In this patient, Na₂CO₃ treatment was started in the emergency room. Haemodialysis and general supportive treatment were applied in the intensive care unit. However, the patient died due to the development of intracerebral hemorrhage in the follow-up.

CONCLUSION

Possibility of methanol intoxication should be included in the differential diagnosis in patients with altered consciousness, vision disturbances and a high anion gap admitted to the emergency service. DW-MRI may be helpful in diagnosis by detecting symmetrical diffusion restrictions at the putamen. Additionally radiologic methods like CT can be used to determine complications such as bleeding that may occur during the follow-up of the cases.

Conflict of Interest

The author declare no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

Author Contributions

Plan, design: AA, EB; **Material, methods and data collection:** AA, EB, BÇ; **Data analysis and comments:** AA, EB, BÇ; **Writing and corrections:** AA, EB

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