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Case Report

# Characteristics of brain magnetic resonance imaging in acute methanol intoxication: Report of 3 cases ☆

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ABSTRACT

Acute methanol intoxication is uncommon. Methanol is mildly toxic, but its metabolites are formic acid and formaldehyde, causing total metabolism, visual disturbances, and central nervous system disturbances, leading to coma and death. Magnetic resonance imaging (MRI) is very important for the diagnosis and prognosis of methanol intoxication. Putaminal necrosis with or without hemorrhage is the most frequently reported finding. Other affected areas that are reported in the literature are subcortical white matter, hippocampus, optic nerve, and cerebellum. We report 3 cases of methanol intoxication and discuss their brain lesions on MRI.



## Introduction

Methanol, also known as methyl alcohol ($CH_3OH$), is a colorless, volatile liquid that is highly flammable and soluble in water. It can enter the human body through various routes, including ingestion (most common), inhalation, skin contact, and mucous membranes [1–3]. Acute methanol poisoning is rarely caused by suicide or accidents; it is often the result of adulterated alcohol production (mixing methanol and ethanol) [4,5].

Methanol has mild toxicity, but its metabolites, formic acid and formaldehyde, are highly toxic and can cause metabolic acidosis, visual disturbances, and central nervous system damage, leading to coma and death [4,5].

The diagnosis of methanol poisoning is based on clinical symptoms and laboratory tests [5]. Brain magnetic resonance imaging (MRI) also plays an important role in diagnosis and prognosis. According to the literature, the most common and significant imaging finding in methanol poisoning is necrosis of the lentiform nucleus, predominantly putamen, with or without hemorrhage, at varying degrees. Other affected areas

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reported in the literature include the involvement of the subcortical white matter, hippocampus, optic nerves, brainstem, and cerebellum [1,2,6].

## Case report

### *Case 1*

A 52-year-old male patient was admitted to the hospital with a history of alcohol consumption 2 days prior. The initial diagnosis upon admission was alcohol poisoning and metabolic acidosis. Arterial blood gas analysis results showed a pH of 7.23, $pCO_2$ of 19.4 mmHg, pO2 of 182.5 mmHg, $HCO_3-$ of 8.1 mmol/L, and BE of −19.4 mmol/L. Methanol levels in both blood and urine were negative, 4 days after alcohol consumption.

MRI was performed 10 days after hospital admission. The imaging showed bilateral putamen necrosis with high signal intensity on T1-weighted images, heterogeneous high signal intensity on T2-weighted and FLAIR sequences, scattered hemorrhages with low signal intensity on susceptibility-weighted imaging (SWI), and high signal intensity on phase images. Additionally, there were subcortical white matter lesions in the occipital and frontal regions bilaterally, symmetric involvement of the hippocampus with low signal intensity on T1-weighted images and high signal intensity on T2-weighted and FLAIR sequences. All the lesions showed limited diffusion and mild high signal intensity on diffusion-weighted imaging (DWI) (Fig. 1).

### *Case 2*

A 52-year-old male patient was admitted to the hospital with a diagnosis of alcohol poisoning, chronic kidney disease, and metabolic acidosis. Blood gas analysis results showed a pH of 6.91, $pCO_2$ of 14 mmHg, $pO_2$ of 190.9 mmHg, $HCO_3-$ of 2.7 mmol/L, and BE of −28.7 mmol/L. The methanol level in the blood was 4.45 mg/dL.

MRI was performed 4 days after hospital admission. The imaging showed bilateral putamen with low signal intensity on T1-weighted images and high signal intensity on T2-weighted and FLAIR images, without hemorrhage. Additionally, there were subcortical white matter lesions in the bilateral parietal and frontal regions, as well as the splenium of the corpus callosum, with low signal intensity on T1-weighted images and high signal intensity on T2-weighted and FLAIR images. All the lesions showed restricted diffusion with high signal intensity on diffusion-weighted imaging (DWI) and low signal intensity on apparent diffusion coefficient (ADC) maps (Fig. 2).

### *Case 3*

A 57-year-old male patient was admitted to the hospital with a diagnosis of metabolic acidosis and coma. Arterial blood gas analysis results showed a pH below 6.9, $pCO_2$ of 33.6 mmHg, $pO_2$ of 289.1 mmHg, $HCO_3-$ of 5.0 mmol/L, and BE of −29.7 mmol/L. The methanol level in the blood on July 26, 2022, was 22.79 mg/dL.

MRI was performed 2 days after hospital admission, revealing diffuse white matter, cortical gray matter, and deep gray matter lesions in both hemispheres. The imaging also

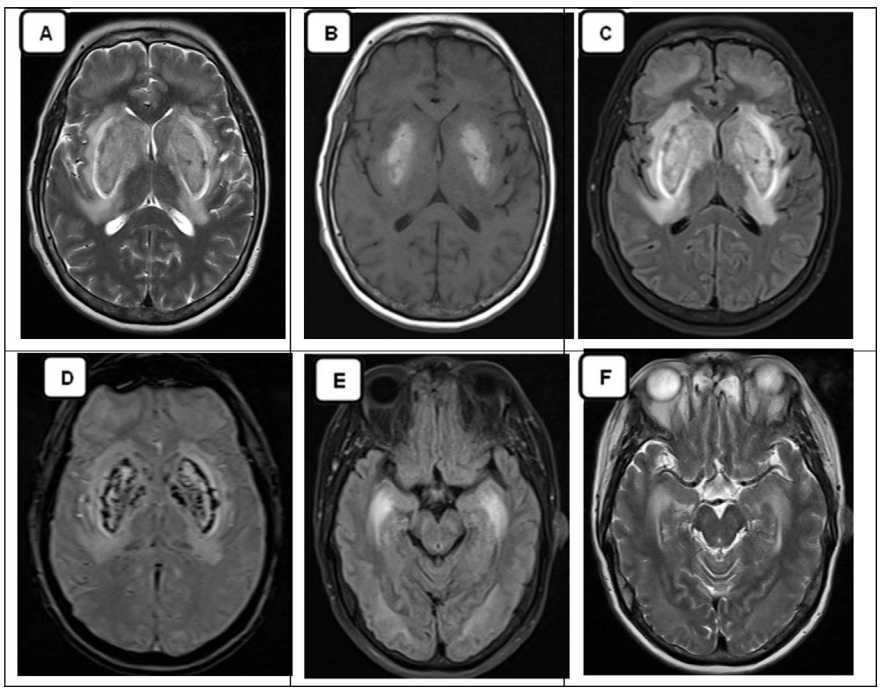


**Fig. 1 – Bilateral lentiform nucleus necrosis (predominantly putamen) (A-C), and scattered hemorrhages (D). Subcortical white matter lesions in the frontal and occipital regions; involvement of the hippocampus (E, F).**

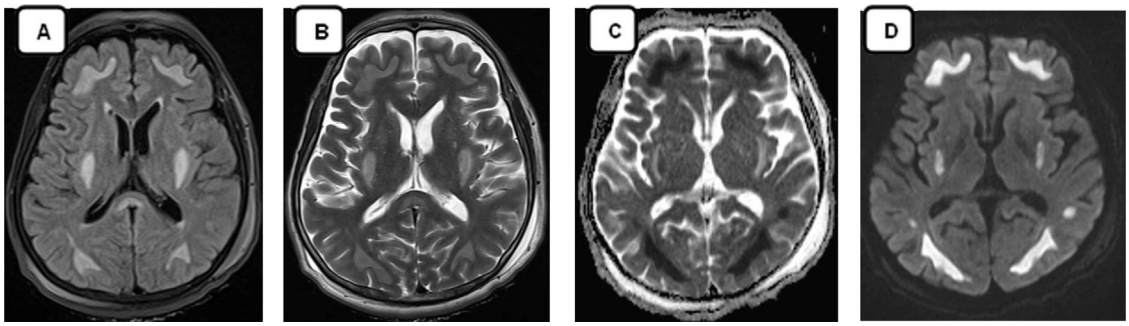


**Fig. 2 – Bilateral putamen and subcortical white matter lesions in the frontal and parietal regions, the splenium of the corpus callosum (A, B), without hemorrhage, restricted diffusion (C, D).**

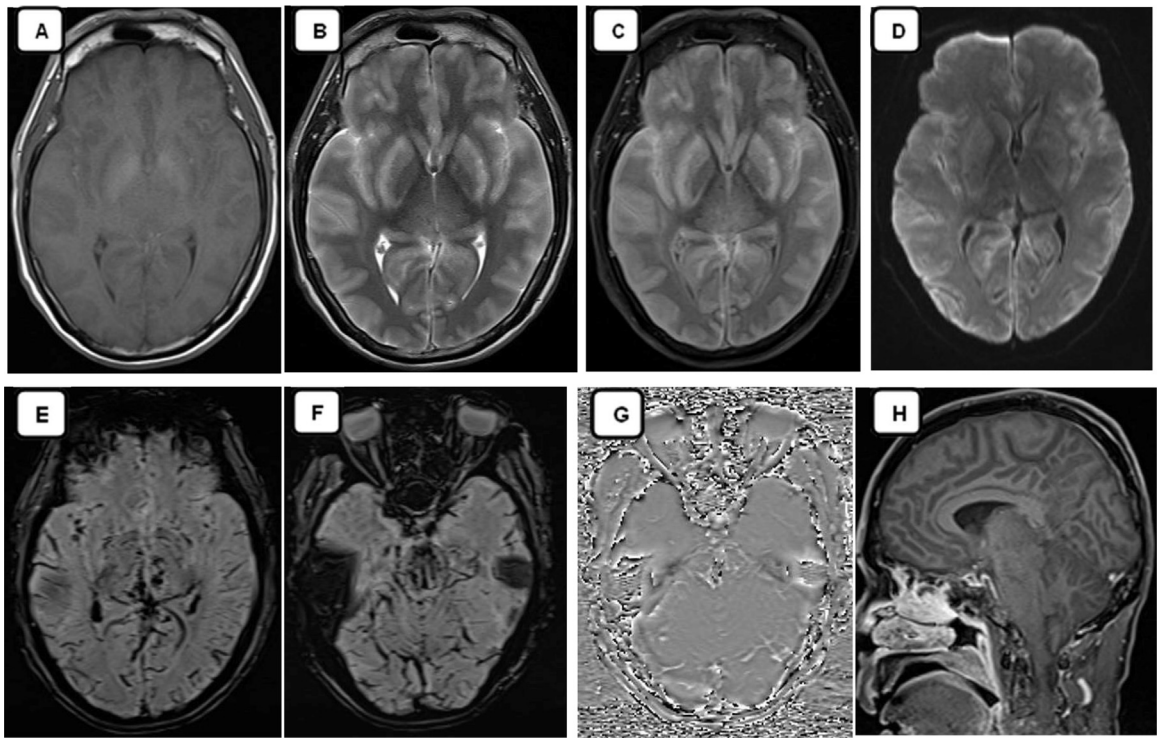


**Fig. 3 – Diffuse cerebral edema with compression of the ventricles and brainstem, bilateral lentiform nucleus necrosis (A-D). Hemorrhage in the brainstem (E-G). Tonsillar herniation (H).**

showed compression of the bilateral lateral ventricles, descending transtentorial hernia, and tonsillar hernia with accompanying hemorrhage in the brainstem (Fig. 3).

## Discussion

The sensitivity to methanol varies among individuals. The latent period typically ranges from 12 to 24 hours after methanol ingestion (this is the time when methanol is metabolized into formic acid and formaldehyde) [5].

Methanol exerts its toxicity on the human body through 2 mechanisms. First, methanol can cause death through the central nervous system depression mechanism (similar to ethanol). Second, the metabolic product of methanol, formic acid, leads to metabolic acidosis and severe neurological consequences, including visual disturbances (optic nerve necrosis or demyelination), headache, dizziness, nausea-vomiting, weakness, and discomfort. In severe cases, it can lead to seizures, coma, and death.

Methanol is primarily metabolized in the liver (>95%), with minimal unchanged excretion through the kidneys (1%-2%) and lungs (2%-3%). The elimination half-life of methanol is approximately 14-30 hours, which can be prolonged to 43-96 hours when co-ingested with ethanol. Therefore, blood methanol analysis should be performed early within 30 hours,

while urinary methanol analysis is not commonly mentioned in the research [4,5].

The diagnosis of methanol poisoning is based on severe metabolic acidosis with anion and osmolar gaps, along with a high blood methanol concentration (≥20 mg/dL if available). The American Academy of Clinical Toxicology recommends treating methanol poisoning based on the following criteria: (1) Methanol serum concentration >20 mg/dL; (2) Recent history of methanol ingestion with an osmolar gap >10 mOsm/L; (3) Clinical suspicion of methanol poisoning with at least 2 of the following parameters: arterial pH <7.3, serum $HCO_3-$ <20 mmol/L, and osmolar gap >20 mOsm/L [3,5].

MRI of the brain plays an important role in the diagnosis and prognosis of methanol poisoning. Bilateral lentiform nucleus necrosis is the most characteristic imaging finding of methanol poisoning, particularly with preferential involvement of the putamen compared to the globus pallidus, which strongly suggests the presence of methanol poisoning. Hemorrhage can occur during the acute phase and/or during the disease, explaining the presence or absence of hemorrhagic images with varying degrees [1,4]. Other possible brain lesions include subcortical white matter lesions, hippocampal damage, optic nerve involvement, cerebellar, brainstem, and thalamic lesions. Cerebral and ventricular hemorrhages, pontine necrosis, diffuse cerebral edema, and optic nerve damage have been described in severe cases of methanol poisoning [1,3,6,7]. Due to the mechanism of cytotoxic edema, DWI and ADC images are valuable. The lesions typically show restricted diffusion on DWI and decreased ADC values during the acute phase [1,4,7,8].

However, the imaging appearance of putaminal necrosis is not specific, as the putamen is selectively vulnerable to damage (possibly due to high metabolic demand, anatomical location in a watershed area, direct toxic effects on the putamen, or a combination of factors). Other conditions with putaminal lesions include Wilson's disease, primary mitochondrial disorders (Leigh disease, Kearns-Sayre syndrome), ischemic/hypoxic injury, hypoglycemia, and intoxication with other substances (cyanide neurotoxicity, carbon monoxide neurotoxicity, organophosphate neurotoxicity) [1,9].

In our first case, the MRI images correspond to other medical reports with bilateral symmetrical putamen hemorrhagic necrosis, subcortical white matter injury in the bilateral frontal and occipital region, and hippocampus injury. Takeshige et al. [10] also reported a methanol poisoning case with similar locations and characteristics of injury as this case. Methanol in blood and urine tests performed on the 4th day after alcohol usage displayed negative results, which corresponded with other evidence. This patient had a history of alcohol consumption, clinical evidence of metabolic acidosis (pH: 7.23, $HCO_3-$: 8.1 mmol/L), and typical MRI images, aiding in the diagnosis of methanol poisoning.

In the second case, the patient presented with metabolic acidosis (pH: 6.91, $HCO_3-$: 2.7 mmol/L), and the methanol blood test performed 1 day after admission showed a level of 4.45 mg/dL. The MRI images are consistent with the literature, showing bilateral putamen without hemorrhage, subcortical white matter lesions in the bilateral frontal and parietal regions, and the splenium of the corpus callosum. Keles GT et al. [11] and Gök et al. [2] also reported cases of methanol poisoning with similar findings of the bilateral putamen, subcortical white matter injury, and the splenium of the corpus callosum, similar to our case.

Additionally, in our third case, the MRI scan of the brain showed diffuse white matter and bilateral lentiform nucleus necrosis involvement, causing cerebral edema, descending transtentorial hernia, and tonsillar hernia, with brainstem hemorrhage. The brainstem hemorrhage in this case could be due to methanol poisoning or Duret hemorrhage, but Duret hemorrhage is expected to be more likely than hemorrhage due to methanol poisoning. Scientific literature also reports that in severe cases of methanol poisoning, brain injury patterns often overlap with ischemia and hypoxia injury along with bleeding. The patient's clinical picture was very severe with severe metabolic acidosis (pH <6.9, $HCO_3-$ 5.0 mmol/L) and coma; methanol blood levels were very high (22.79 mg/dL). The patient died after 5 days of hospitalization.

## Conclusion

To conclude, when symmetrically putamen and subcortical white matter lesions are observed on the magnetic resonance imaging (MRI) of the brain, methanol intoxication should be considered along with other diagnoses. The patient's history of alcohol consumption, metabolic acidosis, and methanol blood testing (if available) aids in early diagnosis, which is crucial for improving the prognosis during the acute phase.

## Patient consent

Informed consent for patient information to be published in this article was obtained.

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