



## Case Report

## Methanol Intoxication With Bilateral Putaminal and Occipital Necrosis

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### Abstract

Methanol is a colorless fluid that smells and tastes like ethanol. It causes poisoning by accidental or intentional inhalation, ingestion or absorption through the skin. We report a 29-year-old man who was brought to the emergency room with dizziness, severe headache and blurred vision due to the ingestion of illegal liquor that contained methanol. Brain magnetic resonance imaging performed on day 38 showed necrosis of bilateral basal ganglia, especially the putamen, and bilateral occipital necrosis with hemorrhage on the left side, which was further confirmed with computed tomography. Thus, in addition to putaminal necrosis, damage to the occipital lobe could be an important aspect of methanol intoxication. (*Tzu Chi Med J* 2010;22(3):160–163)

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## 1. Introduction

Methanol is a toxic agent found mainly in varnishes, paint removers, perfumes, antifreeze, copy machine fluid and gasoline mixtures (1,2). Accidental or intentional inhalation, ingestion or absorption through the skin causes severe metabolic acidosis and clinical disturbances such as blindness, permanent neurologic dysfunction and death. Modern neuroimaging techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) can reveal many of the toxic effects of methanol in the central nervous system. Bilateral necrosis of the putamen is the most characteristic radiological feature, with or without hemorrhage (3), and brain lesions including necrosis of the subcortical white matter, cerebellar cortical lesions,

bilateral tegmental necrosis, diffuse cerebral edema and optic nerve necrosis have also been documented (1,3,4). Here, we report a case of methanol intoxication with bilateral putaminal and occipital necrosis confirmed by MRI and CT.

## 2. Case report

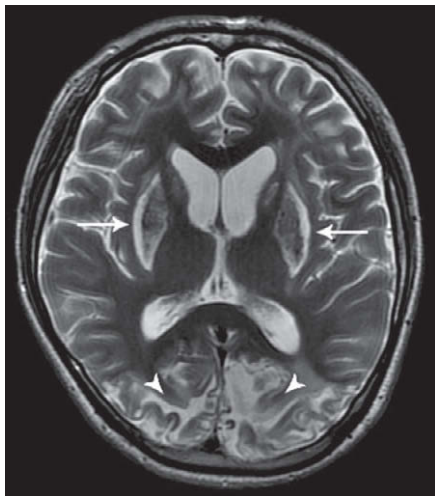
A 29-year-old man with a history of chronic alcoholism presented with drowsy consciousness in the emergency room. He had drunk half a bottle of illegal liquor the day before admission and suffered from dizziness, severe headache and blurred vision the next morning. He was admitted to a regional hospital. At presentation there, dyspnea was noted and arterial gas analysis

showed systemic metabolic acidosis (pH, 7.138;  $\text{PCO}_2$ , 7.5 mmHg;  $\text{HCO}_3$ , 2.5 mmol/L). Under the impression of alcoholic ketoacidosis, he was transferred to our emergency department for further treatment.

On admission, he was in a coma with a Glasgow Coma Scale score of E1V1M1. Arterial blood gas analysis showed systemic metabolic acidosis (pH, 6.831;  $\text{pCO}_2$ , 35.0 mmHg;  $\text{HCO}_3$ , 5.7 mmol/L) with a high anion gap (33 mmol/L). He was intubated and transferred to the intensive care unit. His blood ethanol level was less than 2.83 mg/dL. Bicarbonate replacement therapy was given and the patient underwent two courses of hemodialysis. Systemic acidosis was relieved thereafter.

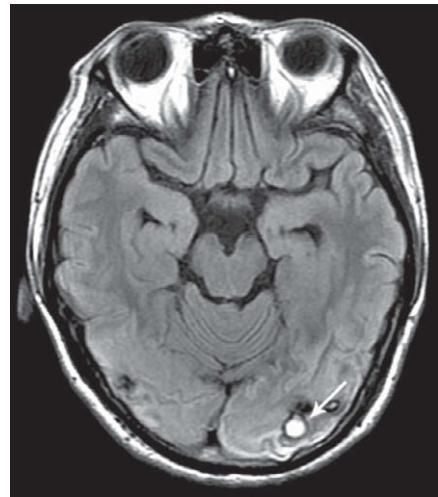
Six days later, a blood methanol of 245 mg/dL was reported. On day 18, the patient had a Glasgow Coma Scale score of E3V3M6 and was transferred to a rehabilitation ward. Bradykinesia, a mask-like face and monotonous speech were noted, but the patient showed no tremor or cogwheel rigidity. Fundoscopic examination showed mild hyperemia in bilateral discs without other abnormality. Visual evoked potentials showed bilateral visual conduction disturbance.

T2-weighted MRI on day 38 showed mixed isointense and hyperintense areas in the basal ganglia, especially the putamen, bilaterally suggesting necrosis (Fig. 1). T2-weighted MRI FLAIR showed necrosis of bilateral occipital lobes with left occipital lobe hemorrhage (Fig. 2), which was further confirmed with CT (Fig. 3) because of a lack of a corresponding axial T1-weighted MRI.



**Fig. 1** — T2-weighted magnetic resonance imaging shows the characteristic damage identified following methanol intoxication over bilateral basal ganglia, especially putaminal necrosis, as shown by mixed isointense and hypointense areas (arrows). Necrosis of bilateral occipital lobes is also noted (arrowheads).

The patient participated in a rehabilitation program including reconditioning exercises for muscle strengthening and functional task training to increase abilities to compensate for irreversible total loss of vision. As his muscle power gradually recovered, he could sit and stand up independently and walk with a walker under verbal guidance. At the end of rehabilitation, the patient could walk slowly without assistance under supervision. He was discharged on day 45 but remained dependent on others for daily routines because of blindness.



**Fig. 2** — T2-weighted magnetic resonance imaging FLAIR shows necrosis of bilateral occipital lobes with left occipital lobe hemorrhage as shown by the ovoid hyperintensity (arrow).



**Fig. 3** — Computed tomography shows necrosis of bilateral occipital lobes with left occipital lobe hemorrhage (arrow) and bilateral putaminal necrosis (arrowheads).

### 3. Discussion

Methanol is a colorless fluid that smells and tastes like ethanol (5). It is widely used as a solvent in commercial goods such as varnishes, paint removers, perfumes, antifreeze, copy machine fluid and gasoline mixtures (1,2), and thus is a source of accidental or even intentional poisoning.

Symptoms from the gastrointestinal, central nervous and ocular systems are often seen in methanol poisoning (6), and it has a characteristic 12–24 hour latent period (1). Patients develop poisoning symptoms such as nausea, vomiting, alteration of mental status and visual disturbances, from seeing spots to blindness (6). The latent period most likely corresponds to the time for methyl alcohol to metabolize into formaldehyde and formic acid, two chemicals that are more toxic than methanol (5). Respiratory failure may develop if significant metabolic acidosis develops.

Methanol poisoning is usually diagnosed based on patient history and neuro-ophthalmological symptoms. Severe metabolic acidosis with a high anion and osmolar gap and high serum methanol levels further support the diagnosis (3). Therapy mainly consists of gastric lavage, ethanol or 4-methyl pyrazole (Fomepizole) therapy, hemodialysis, alkalinization by administration of sodium bicarbonate and the use of cofactors such as folate (2,6,7). Ethanol is chosen as a therapy because it has an affinity for alcohol dehydrogenase enzyme that is 10–20 times greater than that of methanol. Fomepizole is considered because it has a higher affinity for alcohol dehydrogenase than ethanol and early treatment is believed to prevent renal injuries (3,7).

Neuroradiological manifestations of methanol poisoning have occasionally been described in the literature. Both CT and MRI give similar results but the latter yields better anatomical detail. Bilateral necrosis of the putamen with or without hemorrhage is the most characteristic radiological feature (3). Additional brain lesions including necrosis of the subcortical white matter, cerebellar cortical lesions, bilateral tegmental necrosis, diffuse cerebral edema and optic nerve necrosis have been described (1,3,4). In the present case, both MRI and CT identified the putaminal lesion and, surprisingly, bilateral occipital lobe necrosis, which to our knowledge most studies fail to emphasize (1,6). This could have contributed to the loss of vision and the lack of recovery following treatment. In this patient, hemorrhagic necrosis was noted over the left occipital lobe, and was consistent with a report that hemorrhage is a sign of poor prognosis in patients with methanol poisoning (1,8,9).

The mechanism underlying necrosis remains elusive. Putaminal necrosis has been suggested to result from increased metabolic demand (10). Alternatively, it has been postulated to result from direct toxicity from formic acid, as higher concentrations of formic

acid have been reported to accumulate in the putamen than in other areas of the brain. Another possible mechanism is a higher sensitivity of striatal neurons to the toxic metabolites of methanol (1,3). Putaminal changes revealed with MRI, however, are not specific to methanol intoxication and have been described in Wilson's disease, Leigh disease, Kearns-Sayre syndrome and striatonigral degeneration. In addition, carbon monoxide inhalation, hypoxic-ischemic injury and acute cyanide intoxication could also lead to putaminal damage, and must be considered in the differential diagnosis (4,8,11,12). In the present case, the blood methanol level was as high as 245 mg/dL, which, although not rare in methanol intoxication (8,13), could have contributed to the severe central neuronal damage that we identified. However, there appears to be no association between the blood methanol level and clinical outcome (1,14). This could be because different patients metabolize different amounts of formic acid from ingested methanol, or because subsequent therapy or ingestion of ethanol prevents methanol metabolism and results in a high methanol concentration.

Although patients with putaminal necrosis are expected to manifest extrapyramidal symptoms such as bradykinesia, mask-like face, rigidity, tremor and monotonous speech (1), our patient did not have tremor or cogwheel rigidity. This is consistent with reports that patients with methanol intoxication and basal ganglia lesions display variable extrapyramidal syndromes or are even asymptomatic (1,6). A large-scale follow-up study is needed to determine the incidence of abnormalities in patients who survive methanol poisoning.

Optic neuropathy in the form of loss of myelin in the optic nerves is another prominent neuropathological change in methanol poisoning, and loss of myelin could lead to atrophy of the optic nerves and blindness. Sharpe et al demonstrated myelin damage in the retrolaminar optic nerve in postmortem histopathological studies of cases of methanol intoxication (15). This selective myelinoclastic effect is believed to result from histotoxic anoxia by formic acid. In the present case, we found no signs of optic nerve atrophy, although Hsu et al, based on their single reported case, suggested that this could be a unique finding of subacute methanol intoxication (10). To our knowledge, with the exception of this, no imaging findings have been shown or suggested to specifically associate with either the acute, subacute or chronic stages of methanol intoxication. More thorough studies are required before this can be resolved.

In conclusion, we presented a case of methanol poisoning with a unique presentation of bilateral occipital as well as putaminal necrosis. We suggest that methanol intoxication could damage the occipital lobe and contribute, at least in part, to the development of blindness, one of the most devastating sequelae of methanol poisoning.

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