

# Cranial Computed Tomography Findings in Methanol Intoxication: Two Case Reports

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**Cite this article as:** Mengi T, Ekmekci UC, Comert B, Gokmen AN. Metanol Intoksikasyonunda Kraniyal Bilgisayarlı Tomografi Bulguları: İki Olgu Sunumu. Yoğun Bakım Derg 2019; 10 (2):x-x.

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**Received:** 22.05.2019  
**Accepted:** 25.05.2019  
**Available online:** 10.06.2019

## ABSTRACT

Computed tomography (CT) may show toxic effects of methanol on central nervous system in acute methanol intoxication, which may be helpful in diagnosing and predicting prognosis. In this article we discussed two cases of acute methanol intoxication with neuroimaging findings.

**Case 1:** A 67-year-old male patient presented with loss of vision. There was a history of chronic alcoholism and alcohol consumption approximately 12 hours ago. Glasgow Coma Score (GCS) was 14. Cranial CT revealed hypodensities in putamen bilaterally. Control cranial CT of the patient with neurological progression showed diffuse hypodense areas in bilateral cerebral subcortical white matter. The time between first CT and control CT was 4 days. Modified Rankin Score was 5 while being discharged from intensive care unit.

**Case 2:** A 65-year-old male patient presented with unconsciousness. There was a history of chronic alcoholism and alcohol consumption approximately 16 hours ago. His GCS was 7. Cranial CT revealed the hypodensities in bilateral frontal lobe subcortical white matter. In the control cranial CT of the patient experiencing status epilepticus, diffuse hypodense areas were detected in bilateral putamen and bilateral cerebral subcortical white matter. The time between first CT and control CT was 1 day. The patient with neurological progression in the follow-up was diagnosed to have brain death.

In acute methanol intoxication, it is important to predict in which patients the outcome will be worse. The time of onset of neuroimaging findings, the anatomic localization of the lesions and the progression of the lesions in the control imaging may be useful in predicting the prognosis.

**Key words:** Methanol, Computed tomography, Putamen, Intensive care

## Introduction

Methanol is a clear, colorless, and very toxic liquid (1). It is available as a component of household products including paint remover or wind washer liquid (2). Intoxication may occur after the intake of methanol by accident or for suicidal purposes (3). Patients usually present with acute neurological, visual and gastrointestinal symptoms (3). Computed tomography (CT) and magnetic resonance imaging may show the toxic effects of methanol on the central nervous system and may be helpful in diagnosing and predicting prognosis (4).

In this article, we discussed two cases of acute methanol intoxication with neuroimaging findings.

## Case 1

A 67-year-old male patient was brought to the emergency department with the complaint of loss of vision that started in the morning after alcohol consumption at night. It was found out from his history that he had been drinking alcohol every day for 40 years and that he had been using the alcohol he produced at his home for 7 years and had drunk 50 cL of raki he had made approximately 12 hours ago. During his admission, his blood pressure was 139/98 mmHg, heart rate was 102 beats/minute, and respiratory rate was 28 breaths/minute. Except for neurological examination, no significant pathological finding was found in other system examinations. Limited cooperation and impaired person, place and time orientation were found in the neurological examination. Although visual acuity and visual field could not be evaluated

clearly since the patient was agitated and limited cooperative, a partial loss of vision in the left eye and the total loss of vision in the right eye were found. The Glasgow Coma Score (GCS) was 14. Blood methanol level could not be examined since the study was not conducted in our hospital. In the examination of blood gas, it was determined that pH was 6.94, PaCO<sub>2</sub> was 26.6 mmHg, PaO<sub>2</sub> was 109 mmHg, HCO<sub>3</sub> was 7 mmol/L, and lactate was 5.1 mmol/L.

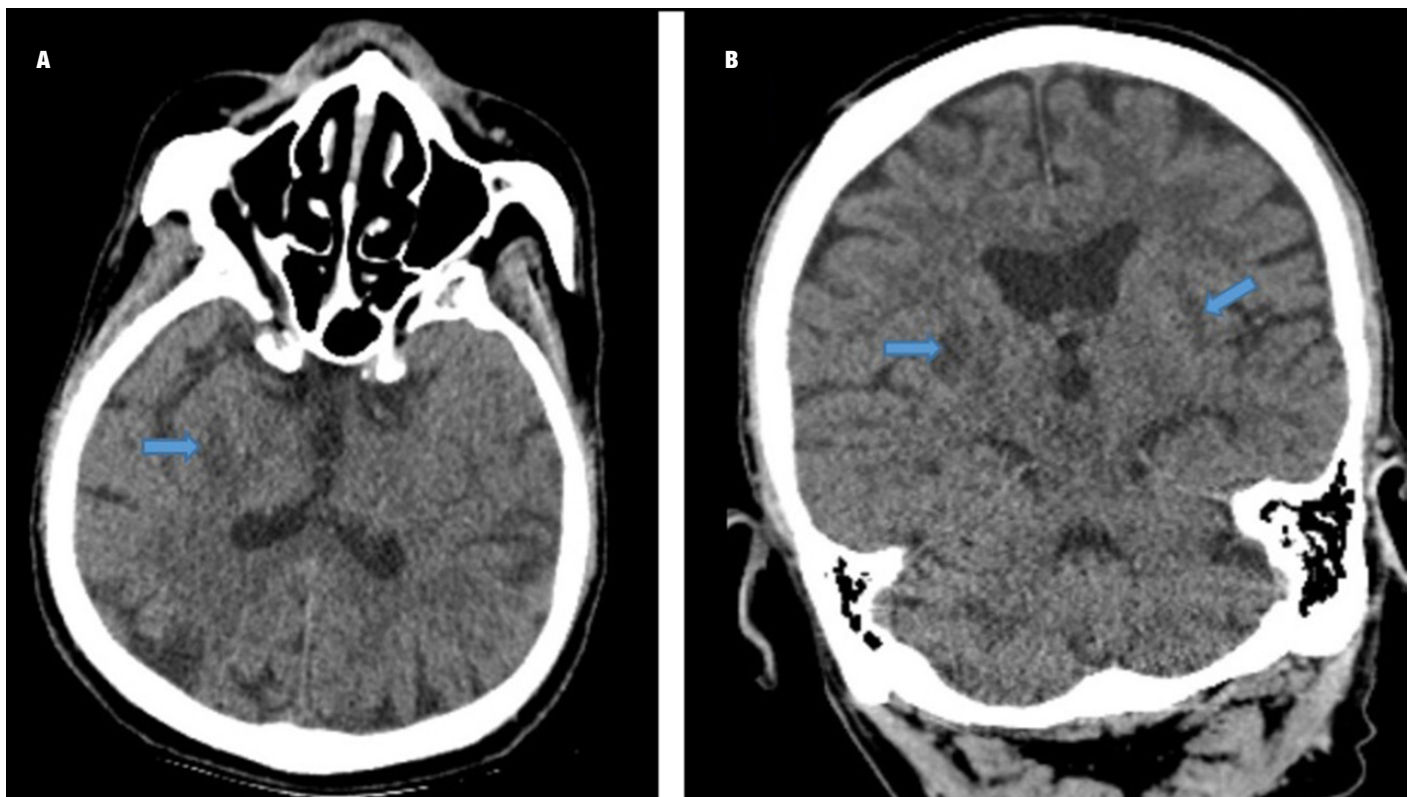
Intravenous (IV) bicarbonate treatment and hemodialysis were performed for metabolic acidosis. The patient was intubated due to a decrease in GCS (GCS=7) during his follow-ups. The patient was transferred to the Anesthesia Intensive Care Unit (ICU) with a preliminary diagnosis of methanol intoxication.

In the emergency service, an IV loading dose of ethanol (10 mL/kg 10% ethanol) was administered and maintenance IV ethanol treatment (1.5 mL/kg/hour) was continued. 0.9% NaCl was initiated parenterally for the patient with a lactate level of 3.6 mmol/L and arterial blood pressure of 86/56 mmHg. Vasopressor (noradrenaline) treatment was initiated since the mean arterial blood pressure was  $\leq$ 65 mmHg despite fluid treatment. The GCS was E1M4V<sub>E</sub>. The vasopressor dose was adjusted according to invasive blood pressure monitoring. The patient underwent another hemodialysis session in ICU follow-ups. The ethyl alcohol treatment of the patient, who had no acidosis in the control blood gas and whose anion gap was found to be within normal limits, was discontinued. In the cranial CT taken in the emergency department approximately 24 hours after alcohol consumption, there was hypodensity in the bilateral putamen and diffuse cerebral atrophy (Image 1). Cranial CT was repeated due to the lack of

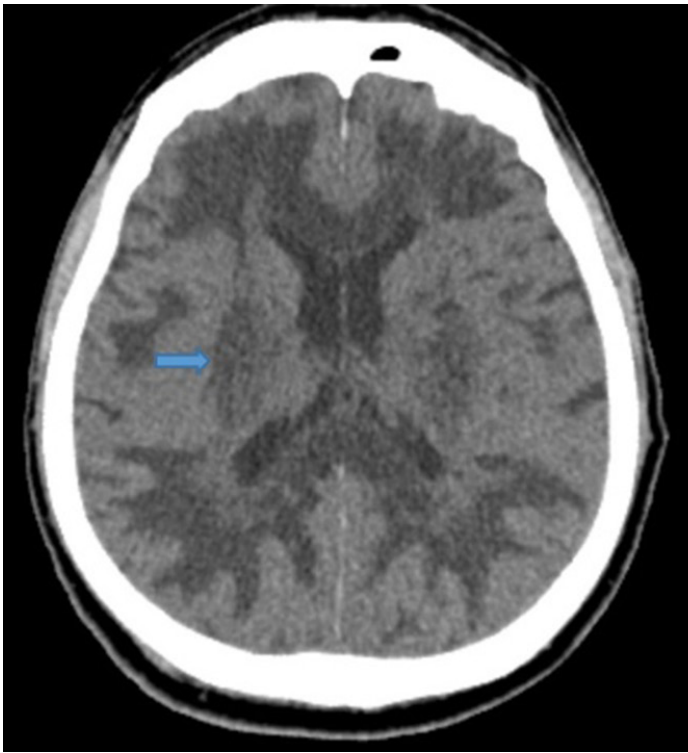
improvement in the consciousness of the patient with improved vital signs and metabolic status (the time between the first cranial CT and the control cranial CT was 4 days). It was observed that there were diffuse hypodense areas in bilateral cerebral subcortical white matter and that the decrease in density in the putaminal area became apparent (Image 2). Neuroimaging findings supported the diagnosis of methanol intoxication. Bedside percutaneous tracheostomy was performed by considering that the patient with the GCS of E1M4V<sub>E</sub> would not be able to protect the airway due to his state of consciousness. The patient was planned to be discharged with a home-type mechanical ventilator since the attempts of weaning from the mechanical ventilator failed due to hypercarbia. On the 40<sup>th</sup> day of hospitalization, the patient was transferred to the service with a home-type mechanical ventilator. The Modified Rankin Score was 5.

## Case 2

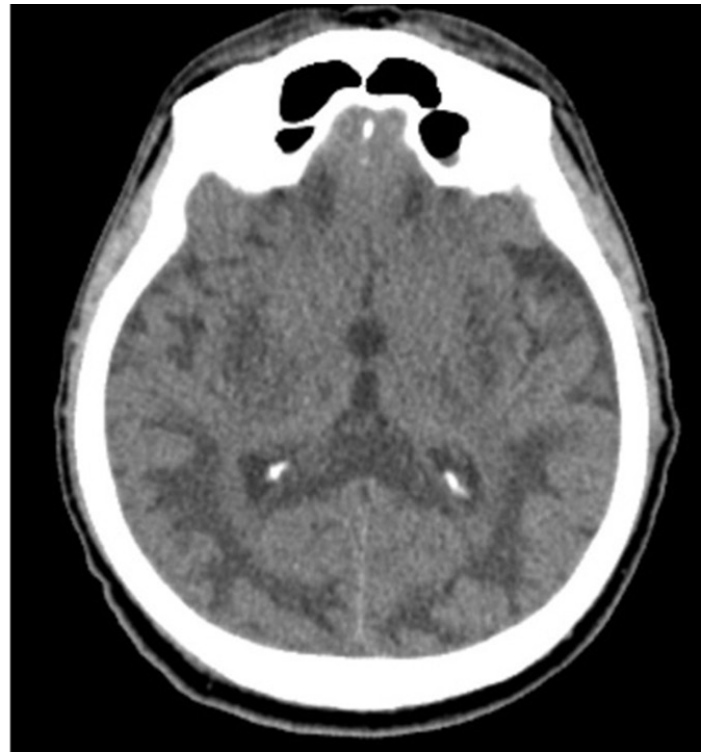
A 65-year-old male patient was brought to the emergency department with the complaint of unconsciousness that developed after a sudden onset of nausea and vomiting. It was found out that the patient with a history of chronic alcoholism had drunk illegal alcohol approximately 16 hours ago. However, how much he had drunk alcohol could not be learned. The GCS was E2M3V<sub>2</sub>. No significant pathological finding was found in the systemic examination of the comatose patient. His arterial blood pressure was 126/82 mmHg, heart rate was 82 beats/minute, and respiratory rate was 30 breaths/minute. In the examination of blood gas, it was determined that pH was 6.82, PaCO<sub>2</sub> was 21.6 mmHg, PaO<sub>2</sub> was 77.6 mmHg, HCO<sub>3</sub> was 5.3 mmol/L, and lactate was 11.5 mmol/L. IV bicarbonate infusion and



**Image 1.** Diffuse cerebral atrophy and hypodensity in the putamen in cranial CT (A) axial section and (B) coronal section (indicated by the arrow).



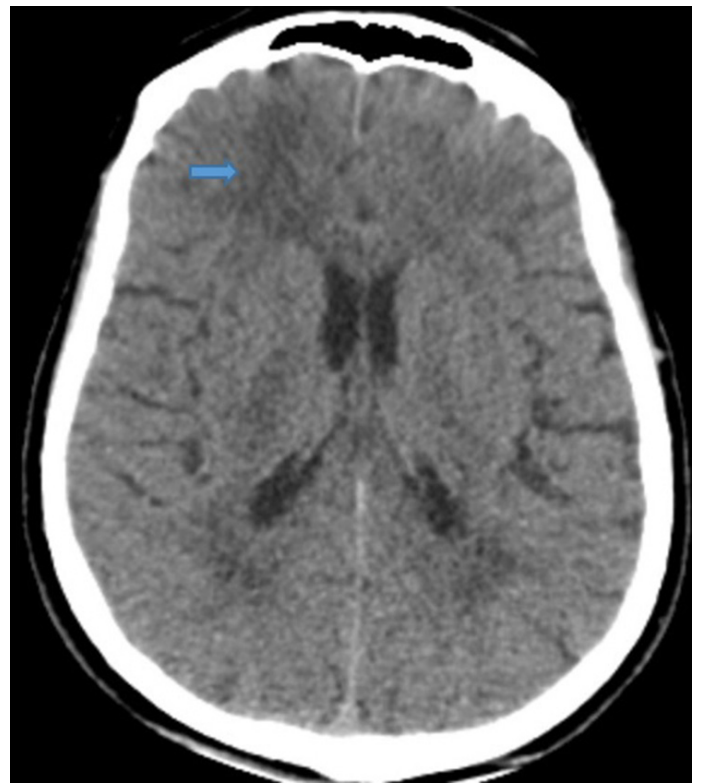
**Image 2.** Diffuse hypodensity in occipital and frontal subcortical white matter in cranial CT axial sections. Hypodensity in the putamen area became apparent (indicated by the arrow).



hemodialysis were performed for the metabolic acidosis of the intubated patient. A focal seizure lasting for seconds was observed in the right arm. In the cranial CT taken approximately 17 hours after alcohol consumption, hypodensity was detected in bilateral frontal lobe subcortical white matter (Image 3). The patient was transferred to the internal medicine ICU with a preliminary diagnosis of methanol intoxication.

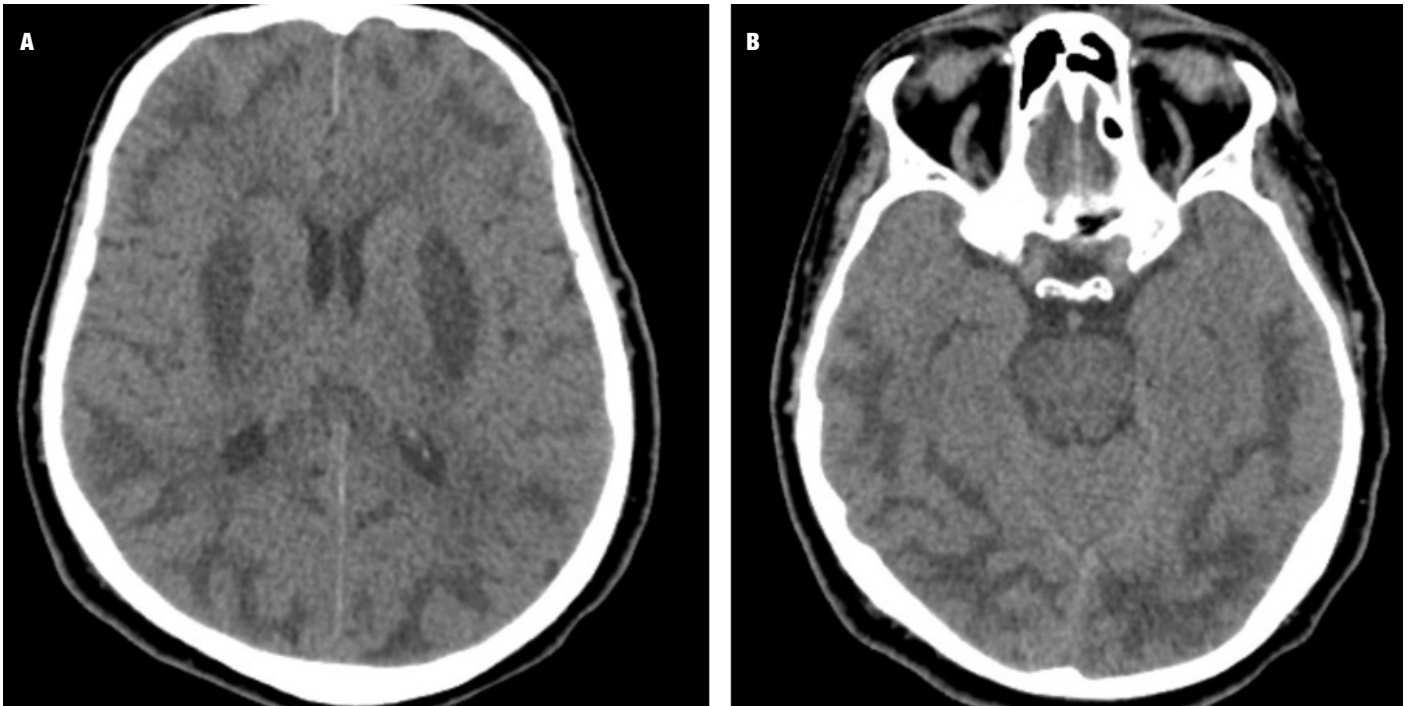
With the preliminary diagnosis of methanol intoxication, in the emergency service, an IV loading dose of ethanol (10 mL/kg 10% ethanol) was administered and maintenance IV ethanol treatment (1.5 mL/kg/hour) was continued. IV diazepam 10 mg and then valproate 30 mg/kg were administered by considering status epilepticus in the patient with a generalized tonic-clonic seizure. Neuroimaging was repeated due to the seizure (the time between the first cranial CT and control cranial CT was 1 day). In the cranial CT, diffuse hypodense areas were detected in the bilateral putamen and bilateral cerebral subcortical white matter, and the findings were considered to be progressive (Image 4). Ethyl alcohol treatment was discontinued since there was no acidosis in control blood gas examinations after the third session of hemodialysis and the anion gap was within normal limits. No seizure was observed in the follow-ups, however, the GCS was E<sub>1</sub>M<sub>3</sub>V<sub>E</sub>. Bedside percutaneous tracheostomy was performed by considering that the patient would remain connected to the mechanical ventilator due to the current neurological picture.

In the follow-up, the brain stem reflexes of the patient with progression in the GCS (E<sub>1</sub>M<sub>1</sub>V<sub>T</sub>) could not be obtained, and the apnea test was positive. Diffuse edema and hemorrhagic changes at the cerebral level were observed in the cranial CT, and no intracranial arterial and venous circulation was observed in CT



**Image 3.** Hypodensity in bilateral frontal lobe subcortical white matter in cranial CT axial sections (indicated by the arrow).

angiography. On the 26<sup>th</sup> day of hospitalization, the patient was diagnosed with brain death. Cardiac arrest developed on the 26<sup>th</sup> day of hospitalization in the patient from whom donation was not accepted by family members.



**Image 4.** Diffuse hypodense areas in the bilateral putamen, occipital and frontal subcortical white matter in cranial CT axial sections.

## Discussion

The clinical presentation of methanol intoxication may vary from patient to patient. Headache, dizziness, malaise, nausea, vomiting, and loss of vision are common findings (1). Our first case presented with a complaint of loss of vision 12 hours after alcohol consumption, and gastrointestinal symptoms appeared after 16 hours in the second case. A change in consciousness was observed in both cases within 24 hours. The latent period after methanol intake is 12-24 hours. The latent period corresponds to the period during which methyl alcohol is converted to formaldehyde and formic acid (1). The first step in methanol metabolism is oxidation to formaldehyde with alcohol dehydrogenase. In the second step, formaldehyde is oxidized to formic acid with aldehyde dehydrogenase (5). While methanol itself causes initial symptoms, methanol metabolites to persistent neurological sequelae are mostly caused by formic acid (3).

The diagnosis of methanol intoxication is based on the history and neuro-ophthalmological findings (4). In cases without a history of illegal alcohol consumption, the toxic blood methanol level (> 20 mg/dL) is diagnostic according to the gas chromatography method (4). Severe metabolic acidosis associated with the increased anion gap and osmolar gap supports diagnosis (4,6). However, both non-specific clinical features and the emergence of symptoms after the latent period cause delay in diagnosis (7).

Furthermore, another reason for the delay in diagnosis is the lack of possibility of methanol level analysis in many hospitals in Turkey.

Neuroimaging findings are helpful in diagnosing. The toxic effects of methanol on the central nervous system can be revealed by CT and magnetic resonance imaging (8). Cerebral and intraventricular hemorrhage, cerebellar necrosis, diffuse cerebral edema, bilateral

subcortical white matter necrosis or edema, optic nerve necrosis were identified as neuroimaging findings in methanol intoxication (1). The most characteristic neuroimaging finding is bilateral putaminal necrosis, and putaminal necrosis may be accompanied by hemorrhage (4). It is included in differential diagnosis since hepatolenticular degeneration, carbon monoxide poisoning, hypoxic-ischemic damage, and basal ganglion involvement in Leigh's disease can be observed (3).

In the treatment of acute methanol intoxication, fomepizole and ethanol with a higher affinity to the alcohol dehydrogenase enzyme than methanol can be used. Thus, the conversion of methanol into toxic metabolites can be prevented (9). Other therapeutic procedures include gastric lavage, correction of acidosis with sodium bicarbonate, folic acid, and hemodialysis (1). In methanol intoxication, reduction in mortality risk can be achieved if treatment is started immediately with early diagnosis. However, neurological sequelae may remain in patients despite appropriate treatment (3). Although our patients were diagnosed early and treated effectively with ethanol and hemodialysis, regression was observed in the patients' clinical picture. Neurological disability developed in the first case and brain death developed in the second case.

Prognosis is associated with the amount of methanol consumed, the degree of metabolic acidosis and the amount of formic acid deposited in the blood, the presence of coma or seizure at admission (3,10). Brain death was diagnosed in the follow-ups of our second case.

In this case, the seizure was observed during admission and status epilepticus was observed in his follow-ups. Furthermore, metabolic acidosis was more severe in the second case compared to the first case.

It is considered that neuroimaging findings may also be useful in predicting prognosis (8). The damage both in subcortical white

matter and in the putamen was claimed to be an indicator of serious methanol toxicity (11). In the study carried out by Taheri et al. (4), 42 cases of methanol intoxication were evaluated, and putaminal hemorrhage and the rate of insular subcortical necrosis were found to be higher in patients who died. In the study carried out by Sefidbakht et al. (8), from among 9 patients with methanol intoxication, two patients with the most severe radiological abnormalities died due to methanol intoxication. There was putaminal involvement in the first cranial CT of our first case, and the second case had frontal-subcortical involvement. The findings in the control cranial CT were similar in both cases, and diffuse hypodensity was present in the bilateral putamen and cerebral subcortical white matter.

The time between the first cranial CT and control cranial CT was 4 days in the first case and 1 day in the second case. More

rapid progression of neuroimaging findings in the second case, and the observation of brain death in the follow-ups of this case suggested that the progression rate of neuroimaging findings could be associated with prognosis. Cranial CT is usually normal in the first 24 hours of acute methanol inoculation (4). However, the prognosis may be more severe in cases with acute pathological findings in the first 24 hours (12). In our cases, pathological findings appeared in the first 24-hour imaging, and the prognosis was poor.

In acute methanol intoxication, it is important to predict in which patients the outcome will be worse in terms of intensive care management. The time of the onset of neuroimaging findings, the anatomic localization of the lesions and the progression of the lesions in the control imaging may be useful in predicting the prognosis.

#### AUTHOR CONTRIBUTIONS:

**Concept:** TM; **Design:** ANG; **Supervision:** BC; **Data Collection and/or Processing:** UCE; **Analysis and/or Interpretation:** BC; **Literature Search:** UCE; **Writing Manuscript:** TM; **Critical Review:** ANG.

**Informed Consent:** Written informed consent was obtained from relatives of patients or patients who participated in this study.

**Peer-review:** Externally peer-reviewed.

**Conflict of Interest:** Authors have no conflicts of interest to declare.

**Financial Disclosure:** The authors declared that this study has received no financial support.

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