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# Methanol Intoxication-Associated Brain Hemorrhages: Can Early Detection Be Life-Saving? A Case Report

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Data Collection B  
Statistical Analysis C  
Data Interpretation D  
Manuscript Preparation E  
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**Patient:** **Male, 42-year-old**  
**Final Diagnosis:** **Brain hemorrhage**  
**Symptoms:** **Altered mental status**  
**Medication:** —  
**Clinical Procedure:** —  
**Specialty:** **Forensic Medicine • General and Internal Medicine • Neurology**

**Objective:** **Rare disease**





**Background:** Methanol can cause many acute complications when ingested, either intentionally or accidentally. One rare complication is cerebral hemorrhage, which can present with focal neurologic deficits, decreased consciousness, and fixed, dilated pupils. With vigilant monitoring of patients in the acute period of toxicity, rapid identification, and initiation of treatment, outcomes can potentially be improved in these patients.

**Case Report:** We present a case of a 42-year-old man who presented after ingestion of windshield wiper fluid. Initial symptoms started with fatigue and altered mental status, but he quickly developed abdominal pain and became obtunded. CT initially showed no acute hemorrhage or other pathology, but on day 2, despite receiving fomepizole, bicarbonate, and dialysis, the patient became hypotensive and showed loss of cranial nerve reflexes, and repeated CT head scans showed acute intracranial hemorrhage with mass effect.

**Conclusions:** Although the exact mechanism of intracranial hemorrhage and necrosis following methanol intoxication remains uncertain, we know beyond doubt that it can progress rapidly and lead to severe and irreversible complications, so identifying and treating it immediately is essential. In this case, methanol ingestion was known on presentation, antidote and renal replacement therapy were initiated within hours of ingestion, yet our patient still suffered fatal brain hemorrhage. Important warning signs of acute hemorrhage include loss of cranial nerve reflexes and decrease in consciousness, so these findings warrant further evaluation and prompt neuroimaging, especially in high-risk patients like the one in this report.

**Keywords:** **Intracranial Hemorrhages • Poisoning**

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## Background

Methanol is an alcohol found in commercial paint thinners, organic fluids, and windshield-cleaner products. It is similar to ethanol in smell and taste, making methanol-containing fluids a simple alcohol alternative, except that it is highly toxic and even lethal. During the year 2016 there were an estimated 106 683 deaths from unintentional methanol toxicity, and without adequate treatment its mortality has been estimated at 40% [1]. Cerebral hemorrhage is a relatively rare complication of this ingestion, occurring in the acute setting in about 13.5% of cases, though a publication based on data from the Czech Republic suggests that hemorrhagic lesions also occurred in the chronic setting months after the initial ingestion in as many as 33% of patients who survived the initial hospitalization [2]. Further, those with evidence of hemorrhage were associated with more severe clinical course as indicated by higher acidemia and lactic acidosis. Methanol poisoning more commonly causes visual disturbances and severe metabolic acidosis and, in unfortunate instances, causes intracranial hemorrhage and death. Formic acid, which is methanol's most offensive metabolite, causes irreversible neurological damage. We report a case of methanol-induced intracranial hemorrhage.

## Case Report

A 42-year-old man with past medical history of hypertension, depression, bipolar disorder, and anxiety, presented to the Emergency Department with altered mental status due to intentional ingestion of windshield wiper fluid. He was restless and according to emergency medical services, he was having of generalized abdominal pain and denied suicidal ideation. Initial vital signs documented on arrival to the Emergency Department were blood pressure 134/81 mmHg, heart rate 98 beat per minute, temperature 36.4°C, and oxygen saturating 100% on room air, in no respiratory distress. On arrival to the Emergency Department, he said he felt tired. The physical examination was unremarkable. He later developed severe generalized abdominal pain and proceeded to become obtunded, with a Glasgow Coma Score (GCS) of 3, for which he was intubated. Significant laboratory test results upon admission (hospital day 0) are shown **Table 1**.

The Poison Control Center was contacted and suggested starting fomepizole and bicarbonate drip. A computed tomography (CT) scan on admission was ordered for altered mental status, showing no acute intracranial hemorrhage, significant mass effect, or acute evolving large-vessel territory infarct. Eight hours of ultrafiltration hemodialysis was performed on day one. Based on the drop in the methanol level (**Tables 2, 3**),

**Table 1.** Significant admission laboratory test results.

Lab value	Patient value	Reference value
Alcohol percent	0.000-0.010%	<0.010%
Venous pH	6.9	7.35-7.45 mmHg
Anion gap	31	9-17 mmol/L
Serum osmolarity	518	280-290 mOsm/kg
Calculated osmolal gap	222	<10 mOsm/kg
Methanol level	0.63	<0.010 g/dL
beta-hydroxybutyrate	2.8	<0.3 mmol/L

**Table 2.** Methanol level over first 2 days.

Lab value	Day 0 at 5: 25	Day 0 at 14: 50	Day 1 at 2: 20	Reference value
Methanol level	0.630	0.339	0.135	<0.010 g/dL

**Table 3.** Anion gap over the first 2 days.

Lab value	Day 0 at 3: 24	Day 0 at 7: 07	Day 0 at 15: 24	Day 0 at 19: 27	Day 1 at 2: 20	Day 1 at 5: 37	Day 1 at 12: 17	Day 1 at 19: 05	Reference value
Anion gap	31	27	31	28	22	22	20	17	9-17 mmol/L



**Figure 1.** Non-contrast CT head of 42-year-old man about 45 hours after presenting to the emergency room with methanol ingestion showing right basal ganglia intraparenchymal hemorrhage with midline shift, indicated by the red arrow.

Poison Control suggested another session of hemodialysis for 4 more hours, to continue fomepizole every 12 hours, and to stop bicarbonate drip.

The patient's blood pressure dropped to 75/35 mmHg during dialysis, so hemodialysis was discontinued after 3.5 hours, and 1 liter of normal saline bolus was ordered and administered. Upon re-assessment about 15 minutes later, his pupils were fixed and dilated and he did not have cough or gag reflexes, with a GCS of 3. A CT scan of the head (Figure 1) was ordered immediately for acute deterioration, which showed large right basal ganglia intra-parenchymal hemorrhage with intra-ventricular extension associated with right to left midline shift. Brain death was suspected. The patient did not exhibit any brain stem reflexes, and nuclear brain blood flow did not visualize brain uptake, which ultimately confirmed brain death. The patient was declared brain dead about 46 hours after presenting at the Emergency Department.

## Discussion

Methanol is a toxic alcohol often involved in both accidental and intentional ingestions. It can be found in homemade liquor, industrial solvents, antifreeze, and windshield wiper fluid [3]. Methanol can also be found as a contaminate in products

like hand sanitizers. Standard windshield wiper fluid contains 30-50% methanol, making methanol the most likely cause for toxicity in our patient [4]. Other toxic substances like ethylene glycol may be present in smaller amounts in windshield wiper fluid. Cases of methanol ingestion can be either accidental or intentional, such as in suicide attempts. Additionally, because it causes similar euphoric and intoxicating effects as ethanol, it is sometimes also consumed recreationally as a substitute for ethanol due to its low cost and easy accessibility. According to the annual report of the American Association of Poison Control Centers, in the United States in 2020, there were over 700 reported cases of methanol poisoning, resulting in at least 11 deaths [5].

Methanol is structurally similar to and produces similar effects to ethanol, but it is processed differently in the body. As methanol is metabolized and converted to formic acid 12-15 hours after initial ingestion, its toxic effects begin. Common signs and symptoms of methanol intoxication include blurry vision, partial or "snow" blindness, hypotension, dizziness, confusion, agitation, abdominal pain, nausea, vomiting, diarrhea, seizures, and coma [3]. Significant toxicity results in high anion gap metabolic acidosis, elevated osmolar gap, and end-organ damage [3]. Without appropriate treatment, a dose of 1 g/kg of methanol or ethylene glycol can be fatal, although serious complications can be seen with much smaller quantities [6].

One of the rare potential adverse effects of methanol intoxication is cerebral hemorrhage. Reports of the incidence of methanol intoxication-induced brain hemorrhage vary but range from 13.5% [7] to 33% [2] of patients hospitalized for methanol poisoning. The mechanism of methanol-induced brain hemorrhage is not fully understood, but one hypothesis is that formic-acid-induced inhibition of cytochrome oxidase in cell mitochondria leads to necrosis, particularly in areas of the brain like the putamen with high metabolic demands [3]. These areas of necrosis may then be more vulnerable to hemorrhage. Patients with brain hemorrhage typically present acutely with decreased consciousness, unresponsiveness, and fixed, dilated pupils. The diagnosis is confirmed with a non-contrast CT of the head, where evidence of brain necrosis and hemorrhage indicates a particularly poor prognosis.

Methanol's toxic effects are a result of formic acid production when methanol is metabolized. Therefore, one approach for mitigating the damage involves limiting its metabolism by inhibiting an enzyme upstream of its production by using fomepizole, an inhibitor of alcohol dehydrogenase. Hemodialysis is also often indicated in cases of methanol poisoning with severe metabolic acidosis or evidence of organ damage, such as cerebral infarction or hemorrhage. Cerebral hemorrhage is typically treated in the intensive care unit with reversing anticoagulation, close monitoring and management of blood pressure,

and measures to decrease intracerebral pressure such as elevating the head of the bed, sedation, and antipyretics.

Here, we report a rare case of cerebral hemorrhage induced by methanol intoxication. By reporting this case, more awareness can be spread about this complication of methanol ingestion, allowing for potentially faster identification of this complication, quicker treatment, and the ability to compare outcomes with the potential to improve outcomes for future patients. Studies have suggested the potential need to minimize or avoid heparin administration during hemodialysis, to decrease the risk of hemorrhage associated with methanol poisoning [7]. However, this hypothesis is under debate. A 2016 study specifically looking at this issue found no association between brain hemorrhages and systemic anticoagulation during dialysis [2].

Patients and their physicians would benefit from the results of further investigation into the best ways to prevent and treat methanol-induced cerebral hemorrhage and increased awareness of hemorrhage as a complication of methanol ingestion. Additionally, knowledge of early, subtle signs and symptoms can significantly reduce morbidity and mortality. When providers encounter patients with decreased consciousness, especially with metabolic acidosis and increased anion gap, toxic methanol ingestion should be considered. Patients with probable or confirmed methanol intoxication should be admitted to the intensive care unit for close monitoring and early

intervention if they show signs of decompensation, particularly decreased consciousness, unresponsiveness, or fixed, dilated pupils suggestive of an intracranial bleed. Treatment with hemodialysis and fomepizole have been shown to help improve patient outcomes and should be considered by providers who encounter these patients.

## Conclusions

Although the exact mechanism of intracranial hemorrhage and necrosis following methanol intoxication remains uncertain, we know beyond doubt that it can progress rapidly and lead to severe and irreversible complications, so identifying and treating it immediately is essential. In this case, methanol ingestion was known on presentation, and antidote and renal replacement therapy were initiated within hours of ingestion, yet our patient still suffered fatal brain hemorrhage. Important warning signs of acute hemorrhage include loss of cranial nerve reflexes and decrease in consciousness, so these findings warrant further evaluation and prompt neuroimaging, especially in high-risk patients like the one in this report.

## Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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