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Acute Ethylene Glycol Poisoning Following Accidental Mineral Oil Ingestion With Severe Metabolic Acidosis, Acute Kidney Injury, Respiratory Failure, and Delayed Neurological Sequelae: A Case Report

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Patient: Male, 57-year-old

Final Diagnosis: Ethylene glycol poisoning • severe high anion gap metabolic acidosis • severe hyperkalemia • hypocalcemia • acute kidney injury with oliguria

Symptoms: Dizziness • vomiting • wheezing • drowsiness • ataxia • dysarthria • even coma

Clinical Procedure: —

Specialty: Critical Care Medicine • Public Health • Toxicology

Objective: Unknown etiology

Background: Ethylene glycol poisoning is uncommon but life-threatening. Early manifestations can mimic ethanol intoxication, while delayed recognition can lead to severe acid-base, renal, cardiopulmonary, and neurological complications. This report describes the case of a 57-year-old man who unintentionally ingested mineral oil containing ethylene glycol and presented with coma, a high anion gap metabolic acidosis, acute kidney injury, respiratory failure, and delayed neurological abnormalities.

Case Report: A 57-year-old Han Chinese man developed dizziness, vomiting, wheezing, drowsiness, ataxia, dysarthria, and coma within 12 hours of accidental ingestion of mineral oil containing ethylene glycol. On admission, the Glasgow Coma Scale score was 3, and he had severe metabolic acidosis (pH 7.0, bicarbonate 2.70 mmol/L, anion gap 30.0 mmol/L), hyperkalemia, hypocalcemia, acute kidney injury, and respiratory failure. The blood ethylene glycol concentration was 456.8 µg/mL, and gastric contents were positive. Treatment included intubation, mechanical ventilation, ethanol therapy, correction of acidosis and electrolyte disturbances, organ support, and early bedside blood purification followed by continuous venovenous hemodialysis. Ethylene glycol was undetectable on day 5, and he was extubated on day 14. Brain magnetic resonance imaging (MRI) on day 19 showed a recent right periventricular infarct, chronic lacunar infarcts, white-matter demyelination, and microhemorrhages. He was transferred to the general ward on day 24 and discharged on day 30. At the 1-month follow-up, serum creatinine levels had returned to normal and neurological symptoms had improved.

Conclusions: Severe ethylene glycol poisoning should be suspected in patients with coma, vomiting, high anion gap metabolic acidosis, renal dysfunction, and a history of possible toxic alcohol exposure. Early antidotal therapy, extracorporeal treatment, serial toxicological, renal, respiratory, and neurological assessments and follow-up are essential.

Keywords: Acute Kidney Injury • Hemodialysis Solutions • Neurological Rehabilitation • Case Reports • Ethylene Glycol • Poisoning

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Introduction

Ethylene glycol is a colorless, water-soluble dihydric alcohol used in antifreeze, industrial solvents, fuels, air-conditioning systems, printing fluids, and other commercial products [1]. Poison center and clinical data characterize severe poisoning as uncommon but potentially fatal. Exposure is usually oral and can be accidental, intentional, or occupational, with automotive and industrial liquids as common sources [2,3]. Although ethylene glycol has limited intrinsic toxicity, alcohol dehydrogenase (ADH) converts it to glycolaldehyde and then to glycolic, glyoxylic, and oxalic acids; its metabolites can cause high anion gap metabolic acidosis, hypocalcemia, calcium tubular injury, acute kidney injury (AKI), cardiopulmonary dysfunction, central nervous system depression, and death [4]. Early symptoms often mimic ethanol intoxication and can include nausea, vomiting, dizziness, ataxia, dysarthria, and impaired consciousness [5]. As metabolic processing advances, patients can develop profound acidosis, hyperkalemia, hypocalcemia, respiratory failure, shock, AKI, and delayed neurological complications [6]. Diagnosis is based on the synthesis of exposure history, characteristic acid-base abnormalities, biochemical findings (eg, osmolar and anion gaps, urinary calcium oxalate crystals), and toxicological confirmation when available [7].

Because confirmatory assays can be delayed, treatment should begin when the exposure pattern and metabolic abnormalities are highly suggestive. Management includes airway and circulatory support, inhibition of ADH with fomepizole or ethanol, correction of acidosis and electrolyte disturbances, and extracorporeal treatment for severe acidosis, kidney injury, or substantial toxin burden [8]. We present a case of severe accidental ethylene glycol poisoning following mineral oil ingestion,

confirmed by toxicological analysis of peripheral blood and gastric contents. The report highlights a concise stepwise clinical narrative detailing diagnosis, extracorporeal treatment guided by serial ethylene glycol monitoring, and systematic neurological follow-up during recovery.

Case Report

A 57-year-old Han Chinese man was admitted on June 1, 2025, after accidental ingestion of mineral oil later confirmed to contain ethylene glycol; the exact volume ingested was not available from the initial history. Over the subsequent 12 hours, dizziness, unsteady gait, slurred speech, and drowsiness developed, followed by repeated emesis, wheezing, and 2 hours of unresponsiveness. His history included hypertension for 30 years and cerebral infarction 3 years earlier, more than 20 years of smoking, and long-term alcohol consumption.

On admission, vital signs were as follows: temperature 36.6 °C, pulse 96 beats/minute, respiratory rate 31 breaths/minute, blood pressure 121/75 mm Hg, oxygen saturation 100% on room air, and fingerstick glucose 7.1 mmol/L. The GCS score was E1V1M1 (3/15). The pupils were equal and reactive, and painful stimuli to the orbit elicited a response. Coarse breath sounds were heard bilaterally without rales. Cardiac rhythm was regular, the abdomen was soft and non-tender, and no lower-limb edema, cutaneous abnormality, scleral jaundice, or superficial lymphadenopathy were observed. The Babinski sign was negative.

Initial arterial blood gas analysis revealed severe high anion gap metabolic acidosis and electrolyte disturbances: pH

Table 1. Emergency arterial blood gas analysis on admission.

Analyte	Admission	40 min after admission	Unit	Reference interval
pH (blood gas)	7.0	7.1	-	7.34-7.44
Partial pressure of carbon dioxide (PCO ₂)	11	17	mm Hg	35-45
Partial pressure of oxygen (PO ₂)	99	161	mm Hg	75-100
Bicarbonate (HCO ₃ ⁻)	2.7	5.3	mmol/L	22-26
Base excess (BE)	-26.5	-22.2	mmol/L	-3.0 to 3.0
Alveolar-arterial oxygen gradient (A-aDO ₂)	37	531	mm Hg	15-30
PaO ₂ /FiO ₂ ratio (P/F ratio)	471	161	mm Hg	> 400
Glucose (Glu)	7.5	22.8	mmol/L	3.3-6.1
Potassium (K ⁺)	8.3	7.7	mmol/L	3.5-5.5
Ionized calcium (Ca ²⁺)	1.0	1.03	mmol/L	1.12-1.32
Anion gap (AG)	30	33	mmol/L	8-16
Lactate (Lac)	Elevated	> 20.0	mmol/L	0.5-2.2

Table 2. Laboratory test results on emergency admission.

Analyte	Result	Unit	Reference interval
Complete blood count & infectious biomarkers			
C-reactive protein (CRP)	41.77	mg/L	0-8
Procalcitonin (PCT)	3.53	ng/mL	< 0.05
Hemoglobin (Hb)	186	g/L	130-175
Hematocrit (HCT)	54.2	%	40-50
White blood cell count (WBC)	31.68	10 ⁹ /L	3.5-9.5
Red blood cell count (RBC)	5.87	10 ¹² /L	4.3-5.8
Platelet count (PLT)	297	10 ⁹ /L	125-350
Absolute neutrophil count (ANC)	28.68	10 ⁹ /L	1.8-6.3
Neutrophil percentage (NEUT%)	90.5	%	40-75
Absolute lymphocyte count (ALC)	1.01	10 ⁹ /L	1.1-3.2
Lymphocyte percentage (LY%)	3.2	%	20-50
Coagulation			
Prothrombin time (PT)	14.3	s	10-13
Activated partial thromboplastin time (APTT)	33.3	s	23.3-32.5
International normalized ratio (INR)	1.2		0.8-1.2
Myocardial injury biomarkers			
Creatine kinase (CK)	502	U/L	50-310
Creatine kinase isoenzyme (CK-MB)	9.97	ng/mL	0-6.22
Myoglobin (MYO)	581	ng/mL	28-72
High-sensitivity cardiac troponin T (hs-cTnT)	0.0132	ng/mL	≤ 0.014
N-terminal pro-B-type natriuretic peptide (NT-proBNP)	1195	pg/mL	< 135
Biochemical markers			
Blood urea nitrogen (BUN)	12.5	mmol/L	3.1-8.0
Serum creatinine (SCr, Cr)	308.0	μmol/L	67-109
Estimated glomerular filtration rate (eGFR)	18.391	ml/min/1.73 m ²	
Lactate dehydrogenase (LDH)	287.0	U/L	120-250
Alanine transaminase (ALT)	19.7	U/L	9-50
Aspartate transaminase (AST)	21.6	U/L	15-40
Total bilirubin (T-Bil)	12.9	μmol/L	0-26
Direct bilirubin (D-Bil)	3.8	μmol/L	≤ 8
Alkaline phosphatase (ALP)	93.0	U/L	< 125
Serum albumin (Alb)	51.7	g/L	35-52
Serum uric acid (UA)	66.0	μmol/L	208-428
Inorganic phosphorus	0.67	mmol/L	0.85-1.51
Potassium (K ⁺)	7.89	mmol/L	3.5-5.5
Calcium (Ca ²⁺)	2.03	mmol/L	2.11-2.52
Carbon dioxide (CO ₂)	2.6	mmol/L	22-29

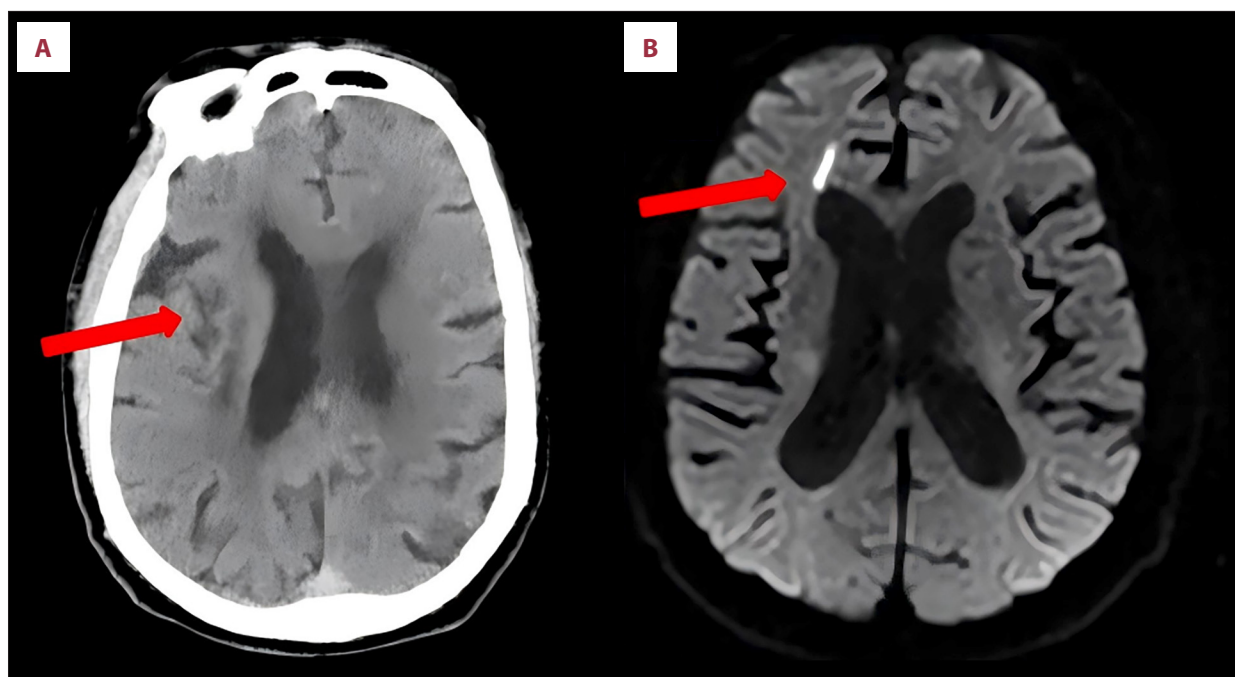


Figure 1. Neuroimaging findings during hospitalization. (A) Cranial computed tomography at admission showed multiple lacunar infarcts and diffuse white-matter hypoattenuation; the arrow marks a representative chronic lacunar infarct. (B) Axial diffusion-weighted imaging from brain magnetic resonance imaging on hospital day 19 showed a focal hyperintense lesion adjacent to the right lateral ventricle, consistent with a recent infarct (arrow), superimposed on chronic cerebrovascular abnormalities.

7.0 (reference range, 7.34-7.44), bicarbonate 2.70 mmol/L (22-26), base excess -26.5 mmol/L (-3.0 to 3.0), anion gap 30 mmol/L (8-16), and arterial carbon dioxide partial pressure 11 mm Hg (35-45), compatible with respiratory compensation. Hyperkalemia was present (potassium 8.3 mmol/L; reference range, 3.5-5.5), as was ionized hypocalcemia (1.00 mmol/L; reference range, 1.12-1.32). Serum creatinine was 308 µmol/L (67-109), estimated glomerular filtration rate was 18.391 mL/min/1.73 m², and day-1 urine output was 150 mL, consistent with acute kidney injury with oliguria; the absence of a baseline creatinine precluded formal KDIGO staging. Emergency treatment for metabolic acidosis and hyperkalemia was initiated, and the patient was admitted to the intensive care unit. Arterial blood gas results at admission and 40 minutes after admission are detailed in **Table 1**, while additional admission laboratory results and reference intervals are provided in **Table 2**.

Urinalysis revealed proteinuria, leukocyturia, and hematuria; no ketonuria was detected. Osmolar gap and urinary calcium oxalate crystal results were unavailable. Toxicological analysis identified ethylene glycol in peripheral blood (456.8 µg/mL) and gastric contents, whereas concentrations of serum ethanol and glycolic acid concentrations were unavailable. Cranial computed tomography (CT) performed at admission demonstrated multiple lacunar infarcts and white-matter demyelinating changes (**Figure 1A**). Chest and abdominal CT revealed

subpleural ground-glass opacities, pleural effusion, mild pleural thickening, and perirenal exudation.

The integrated diagnoses were confirmed as ethylene glycol poisoning, severe high anion gap metabolic acidosis, severe hyperkalemia, hypocalcemia, acute kidney injury with oliguria, probable aspiration pneumonia, and acute respiratory failure requiring airway protection and mechanical ventilation. The respiratory diagnosis was based on coma with loss of airway protection, tachypnea, vomiting with aspiration risk, and subsequent impaired oxygenation. Specifically, 40 minutes after admission, the arterial oxygen partial pressure/fraction of inspired oxygen ratio was 161 mm Hg and the alveolar-arterial oxygen gradient was 531 mm Hg (reference range, 15-30 mm Hg).

Endotracheal intubation and invasive mechanical ventilation were initiated immediately. Naloxone was administered empirically for coma, but consciousness did not improve. Ethanol was administered as a competitive ADH inhibitor due to the unavailability or delayed access to fomepizole. Sodium bicarbonate, intravenous calcium, hyperkalemia management, lung-protective ventilation, piperacillin-tazobactam for probable aspiration pneumonia, gastrointestinal prophylaxis, analgesia and sedation, B-vitamin supplementation, and other organ support were provided. Early bedside blood purification followed by continuous veno-venous hemodialysis (CVVHD)

Table 3. Serial peripheral blood ethylene glycol concentrations during emergency intensive care.

Time point of EICU admission	Day 1	Day 2	Day 3	Day 4	Day 5
Ethylene glycol concentration (µg/mL)	456.8	91.3	11.8	60.1	0.0

Table 4. Changes in renal function and urine output during hospitalization.

Time point of EICU admission	Serum creatinine (Scr, Cr)	Blood urea nitrogen (BUN)	Estimated glomerular filtration rate (eGFR)	Urine output (mL/24 h)
Day 1	308.0	12.5	18.391	150
Day 2	281.0	11.8	20.549	950
Day 3	185.0	6.3	34.061	370
Day 4	147.0	3.1	44.976	300
Day 6	176.0	5.1	36.178	205
Day 8	263.0	8.0	22.261	220
Day 10	420.0	12.9	12.640	120
Day 11	605.0	18.3	8.131	1010
Day 14	452.0	13.0	11.567	2240
Day 16	633.0	17.6	7.698	3170
Day 18	813.0	26.1	5.688	2270
Day 20	747.0	25.4	6.301	2920
Day 22	575.0	19.1	8.646	2050
Day 24	444.0	14.3	11.819	3100
Day 30	220.0	6.9	27.623	1800

was initiated for toxin removal, severe acidosis and hyperkalemia, and renal support.

By hospital day 2, consciousness and acidosis had improved, although lactate remained elevated, and extracorporeal therapy was continued. On day 3, the patient opened his eyes spontaneously but developed tremor and difficulty moving the left upper limb. The neurology consultation team recommended careful fluid management, maintenance of cerebral perfusion, and low-dose aspirin. Serial blood ethylene glycol concentrations decreased from 456.8 µg/mL on day 1 to an undetectable level on day 5 (Table 3). Extracorporeal toxin removal was discontinued after the day-5 concentration was undetectable and the metabolic abnormalities had improved.

Mechanical ventilation was discontinued, and the patient was extubated on day 14. Kidney dysfunction persisted after the initial metabolic recovery: creatinine rose to a peak of 813 µmol/L on day 18, while urine output subsequently improved during renal replacement therapy (Table 4). A brain MRI on day

19 showed a recent infarction adjacent to the right lateral ventricle, multiple chronic lacunar infarcts, white-matter demyelination, and multiple cerebral microhemorrhages (Figure 1B).

On day 24, the patient was transferred from the intensive care unit to the general ward. Following continued supportive treatment, his vital signs stabilized and hepatic and renal functions improved. Mild lethargy persisted at discharge on day 30. At the 1-month outpatient follow-up, serum creatinine level had decreased to 145 µmol/L, neurological symptoms had markedly improved, and no residual speech or limb motor deficits were identified.

Discussion

This case offers 3 practical lessons. First, severe ethylene glycol poisoning should be treated based on clinical and biochemical findings before confirmatory testing is available. Second, serial toxin and renal measurements can support treatment decisions,

but their sampling context must be reported. Third, new neurological findings after toxin clearance require follow-up and a careful assessment of causality when pre-existing cerebrovascular disease is present. The novelty and clinical significance of this case report are threefold in nature. First, it documents a rare clinical scenario of ethylene glycol poisoning associated with accidental ingestion of mineral oil. Second, it demonstrates that serial monitoring of serum toxin levels and renal function offers actionable evidence to support individualized therapeutic decision-making. Third, it underscores the critical diagnostic caution required when evaluating delayed neurological abnormalities in patients with pre-existing cerebrovascular disease. Symptoms in this population are commonly misattributed to acute stroke recurrence. This case offers a reference for differential diagnosis in similar clinical dilemmas.

The presentation was consistent with ethylene glycol metabolism and organ toxicity. ADH converts ethylene glycol to acidic metabolites; glycolate is the main mediator of high anion gap metabolic acidosis [9,10], while oxalate binding to calcium and crystal deposition contribute to hypocalcemia and tubular injury [11,12]. Severe poisoning can progress from inebriation and gastrointestinal symptoms to coma, cardiopulmonary compromise, acute kidney injury, and delayed neurological sequelae [13]. Therefore, treatment should begin when the clinical pattern and metabolic abnormalities are highly suggestive of ethylene glycol poisoning, even before definitive toxicological confirmation is available.

In this patient, the exposure history, coma, pH 7.00, bicarbonate 2.7 mmol/L, anion gap 30 mmol/L, severe hyperkalemia, ionized hypocalcemia, creatinine elevation, and oliguria formed a coherent diagnostic pattern; direct detection of ethylene glycol in blood and gastric contents provided confirmation. Ukita et al highlighted the diagnostic value of evolving anion gap acidosis and urinary findings [9]. In contrast, as osmolar gap and urinary oxalate data were unavailable in this case, their transparent reporting as missing values is essential. The respiratory diagnosis was also refined: initial room air oxygenation was preserved, but coma, aspiration risk, the need for intubation, and a post-intubation arterial oxygen partial pressure/fraction of inspired oxygen ratio of 161 mm Hg supported acute respiratory failure.

Management consisted of alcohol dehydrogenase inhibition, rapid correction of life-threatening metabolic abnormalities, organ support, and extracorporeal toxin removal, consistent with toxicology guidance [5]. Fomepizole is often preferred because of predictable dosing and fewer central nervous system effects, whereas ethanol remains an acceptable alternative when fomepizole is unavailable, though it necessitates close monitoring [14]. Deng et al similarly emphasized early recognition and the combination of definitive and supportive

treatment in a recent case and literature review [4]. In our case report, the patient received ethanol therapy combined with early blood purification and CVVHD due to coma, severe acidosis, hyperkalemia, AKI, respiratory failure, and a high ethylene glycol concentration. The day-4 concentration fluctuation in the present case cannot be mechanistically explained because the timing of sample collection relative to therapy administration was unknown; the overall decline to an undetectable concentration by day 5, together with metabolic improvement, guided treatment cessation. Although intermittent hemodialysis generally provides faster clearance, continuous therapy may be preferable in critically ill patients requiring sustained metabolic control or hemodynamic stability [15,16].

The renal course was biphasic: creatinine initially improved during extracorporeal therapy, then increased to 813 $\mu\text{mol/L}$ on day 18 prior to subsequent recovery. Taira et al described ethylene glycol-associated renal injury with pathological correlation [11]. No kidney biopsy was performed in this case; therefore, the mechanism was inferred from the exposure, oliguria, and laboratory course. Neurological follow-up was also essential. Delayed neurological syndromes following ethylene glycol poisoning have been reported to occur 5-20 days after exposure [6]. The recent right periventricular infarct identified on day 19 may have contributed to the tremor and weakness, but prior infarcts, chronic lacunes, white-matter disease, and microhemorrhages precluded attributing the lesion solely to ethylene glycol. Therefore, the present case report supports structured neurological surveillance after toxin clearance but does not establish ethylene glycol poisoning as the cause of the new infarction.

This report is limited by the unknown ingested volume; absent baseline creatinine; unavailable osmolar gap, urinary calcium oxalate, serum glycolate, and serum ethanol measurements; incomplete ethanol dosing details; and missing exact sampling times for serial ethylene glycol concentrations. No kidney biopsy was performed, and pre-existing cerebrovascular disease limited causal interpretation of the neurological findings. These limitations are stated explicitly to distinguish observed data from inference.

Conclusions

Severe ethylene glycol poisoning should be considered in patients with coma or apparent intoxication accompanied by high anion gap metabolic acidosis, renal dysfunction, hypocalcemia, and possible toxic alcohol exposure. Treatment should not be delayed pending confirmatory toxicology results when clinical suspicion is high. Treatment cessation and follow-up should be guided by serial toxin concentrations together with acid-base status, renal function, urine output, respiratory status,

and neurological examination. New neuroimaging abnormalities warrant cautious interpretation when pre-existing cerebrovascular disease is present.

Institution Where Work Was Done

Beijing Tsinghua Changgung Hospital, Beijing, PR China.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images. A copy of the written consent is available for review from the Editor-in-Chief upon request.

AI-Assisted Writing Disclosure

The authors used ChatGPT (OpenAI; accessed May 2026) for English-language editing, structural organization, and stylistic

refinement. These tools were not used to generate clinical data, interpret laboratory or imaging findings, or make clinical decisions. The authors critically reviewed and verified all AI-assisted text and take full responsibility for the accuracy, originality, and integrity of the manuscript.

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.

Abbreviations

ADH, alcohol dehydrogenase; **AKI**, acute kidney injury; **BE**, base excess; **CVVHD**, continuous venovenous hemodialysis; **EICU**, emergency intensive care unit; **EG**, ethylene glycol; **GCS**, Glasgow Coma Scale; **HCO₃⁻**, bicarbonate; **MRI**, magnetic resonance imaging; **PPI**, proton pump inhibitor.

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