

Case Report

Fatal Ethylene Glycol Poisoning Presented with Severe Metabolic Acidosis- An Emerging Toxicological Crisis Mimicking Methanol Poisoning

Abu Shahin Mohammed Mahbubur Rahman¹, Toma Mehedi², Hosen MA³, Annie NJ³, Chowdhury FR⁴, Ghose A⁵

Abstract:

Background: Ethylene glycol poisoning is an uncommon yet potentially fatal toxicological emergency, resulting from adulterated alcohol consumption. Early recognition and rapid treatment are essential to prevent organ failure and mortality.

Case Presentation: We report a 25-year-old tribal male who presented to Rajshahi Medical College Hospital with severe respiratory distress, abdominal pain, blurred vision, and altered consciousness following ingestion of a locally brewed alcohol ("Chuani"). Initial arterial blood gas (ABG) analysis revealed profound anion-gap metabolic acidosis (pH 6.93, HCO_3^- 6.93 mmol/L, base deficit 28.2 mmol/L). The patient was treated with fomepizole, intravenous sodium bicarbonate, and folic acid, along with other supportive measures. The initial suspicion was of a case of methanol poisoning. However, serum formate levels were negative; urine microscopy demonstrated calcium oxalate crystals, confirming ethylene glycol poisoning. Despite aggressive therapy, the patient developed refractory shock and respiratory failure and subsequently died.

Conclusion: This case highlights the diagnostic challenges of adulterated alcohol poisoning in low-resource settings. Rapid metabolic evaluation, bedside urine microscopy, early antidote therapy, and community awareness regarding adulterated alcohol are crucial to reduce associated fatalities.

Keywords: Ethylene glycol, Metabolic acidosis, Methanol poisoning, RMCH

DOI: <https://doi.org/10.3329/jom.v27i2.92093>

Copyright: © 2026 Rahman ASMM. This is an open access article published under the Creative Commons Attribution-Non Commercial-No Derivatives 4.0 International License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited, is not changed in any way and it is not used for commercial purposes.

Received: 08-02-2026;

Accepted: 06-05-2026

Background:

Adulterated alcohol poisoning is a significant healthcare concern worldwide, especially in the Low- and Middle-Income Countries (LMICs) where access to safe alcohol and diagnostic facilities is limited. Methanol, ethylene glycol (EG), and isopropanol are the most common adulterated substances found in illegal liquor.¹ Adulterated alcohol poisoning might result in remarkable morbidity and mortality to its victims.² Ethylene glycol ($\text{C}_2\text{H}_6\text{O}_2$) is a colorless synthetic chemical with a sweet taste and is used as a coolant, Fog juice, antifreeze agent, and in the formulation of detergents and cosmetics.³ It has a CNS depressant effect like ethanol, including some other symptoms, such as confusion, ataxia, hallucinations, slurred speech, and coma, which is more pronounced after 6-12 hours post-consumption.⁴ The toxic dose of EG is 1-2 mL/kg of a 95% concentrated solution; though the parent compound is

relatively non-toxic, its metabolites, glycolic and oxalic acids, are highly toxic and cause metabolic acidosis, renal failure, and multi-organ dysfunction.⁵ Fomepizole and Ethyl Alcohol are antidotes that inhibit alcohol dehydrogenase and suppress the conversion of ethylene glycol to its toxic metabolites.⁶ EG poisoning has been reported globally but remains uncommon in Bangladesh and is rarely reported due to limited diagnostic capacity. This report details a fatal case of EG poisoning with severe metabolic acidosis. It highlights the importance of early suspicion and timely management.

Case report:

A 25-year-old male from the Santal tribal community was admitted to Rajshahi Medical College Hospital (RMCH) with acute respiratory distress, abdominal pain, blurred vision, and altered level of consciousness following ingestion of locally brewed alcohol. The symptoms developed after a

1. Associate Professor of Medicine, Rajshahi Medical College
2. Site trial coordinator, Methanol POCT study in RMCH
3. Clinical Research Assistant, Methanol POCT study in RMCH
4. Associate Professor, Bangladesh Medical University
5. Professor of Medicine, Chattogram Medical College

Corresponding author: Dr. Abu Shahin Mohammed Mahbubur Rahman, Associate Professor, Department of Medicine Rajshahi Medical College, +8801763248448, E-mail: drshahin76@gmail.com

religious and social celebration. On detailed history-taking, his mother reported that the patient had consumed a traditional locally produced alcoholic beverage known as *Chuani* the night before admission, around 9 PM. He was a habitual alcohol consumer but had no known history of diabetes mellitus, chronic kidney disease, liver disease, or other comorbid conditions.

Approximately eight hours after alcohol consumption, the patient developed abdominal pain and progressive respiratory distress, followed by deterioration in consciousness. He was brought to RMCH in a semiconscious state. On admission, his blood pressure was 100/60 mmHg, pulse rate 126 beats/min, respiratory rate 22 breaths/min, oxygen saturation (SpO₂) 90% on room air, temperature

99°F, and Glasgow Coma Scale (GCS) score 11/15 (Table 1). Neurological examination revealed no focal deficits, seizures, or abnormal reflexes. Oxygen supplementation (2 L/min via nasal cannula) and intravenous normal saline were initiated.

Following admission, all necessary investigations, including an Arterial blood gas (ABG), revealed profound high-anion-gap metabolic acidosis with a pH of 6.93, bicarbonate of 6.93 mmol/L, and a base deficit of 28.2 mmol/L (Table 2). Given the clinical context of adulterated alcohol ingestion and severe metabolic acidosis, methanol poisoning was initially suspected. The patient was promptly treated with a loading dose of fomepizole (780 mg), intravenous sodium bicarbonate (440 mmol), and intravenous folic acid (50 mg), along with supportive care.

Table 1: Timeline of vitals of the patient

Timeline	BP (mm Hg)	Pulse (Beats/min)	Respiratory Rate(Breaths/min)	SpO ₂	GCS (Out of 15)	Temp
On admission	100/60	126	22	90% on air	11	99.1±% F
After 6 hours of post-admission and Fomepizole therapy	60/40	138	21	96% with 4L of O ₂	11	99.1±% F
After 12 hours of post-admission	70/40	120	28	88% with 10L of O ₂	10	99.5±% F

Table 2: Arterial Blood Gas (ABG) findings and comparison over time

	Normal Value	1 st ABG (03.10.2025 at 17:30)	2 nd ABG (04.10.2025 at 10:32)
Ph	↑7.35~7.45	6.934	7.157
pCO ₂ (mmHg)	35.0~45.0	8.0	12.6
pO ₂ (mmHg)	↓70.0~100.0	165.3	65.5
Na (mmol/L)	135.0~145.0	129.3	153.1
K (mmol/L)	↓3.50~5.50	4.33	2.93
Cl (mmol/L)	90.0~110.0	95.4	109.6
Hct	↓42~52%	54.9%	28.3%
BP (mmHg)	120/80	750.90	753.10
Temperature corrected			
PH (tc)		6.93	7.16
PO ₂ (tc) (mmHg)		185.00	65.5
Calculated parameters			
HCO ₃ ⁻ (mmol/L)		6.93	4.50
BEcf (mmol/L)		-28.2	-24.23
tHb (g/L)		192.15	99.05
pO ₂ / FiO ₂ (mmHg)		787.14	311.90
pO ₂ (T)/ FiO ₂ (mmHg)		787.14	311.90
cH ⁺ (T) (mmol/L)		116.41	69.66
mOsm (mmol/L)		298.60	346.20
ctO ₂ (Vol%)		25.00	10.63

Serum formate levels, measured using UV spectrophotometry, were negative, excluding methanol poisoning. Due to ongoing clinical decline, ethylene glycol poisoning was suspected. Urine microscopy revealed albumin (+++) and calcium oxalate crystals (+), which strongly indicate ethylene glycol toxicity (Figure 1). Electrocardiography showed no abnormalities (Figure 2). The patient was urgently referred for hemodialysis; however, he rapidly progressed to refractory shock and respiratory failure. Despite maximal supportive and resuscitative measures, he died approximately 16 hours after hospital admission, before dialysis could be initiated.

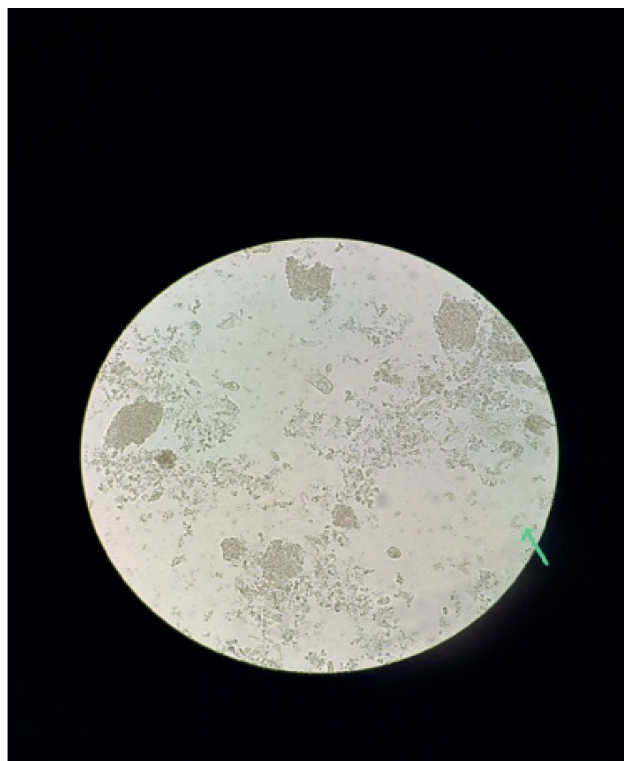


Fig.-1: Urine routine microscopy slides indicating Calcium Oxalate crystals

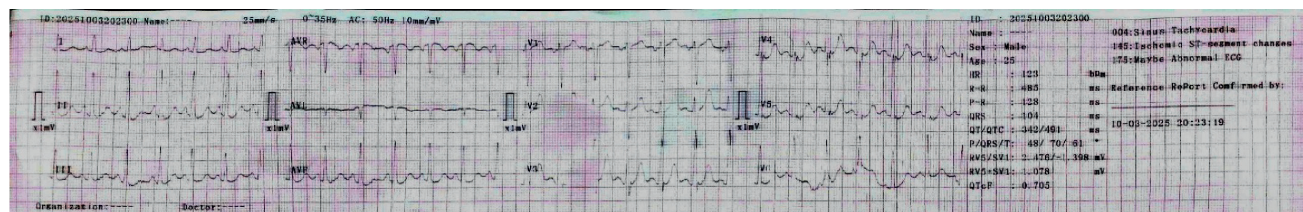


Fig.2: *Electrocardiogram showing normal cardiac activity*

This case highlights the rapid and often fatal course of EG poisoning, especially in low-resource settings where delayed presentation, diagnostic uncertainty, and limited access to hemodialysis significantly worsen outcomes. The patient presented severe high-anion-gap metabolic acidosis, respiratory distress, abdominal pain, and altered mental status, which are typically linked to adulterated alcohol ingestion.^{1,5}

cultural norms often delay disclosure of alcohol intake, complicating early diagnosis and management of alcohol poisoning from adulterated alcohol.^{8,9} In this case, the nearly 16-hour delay in presentation likely contributed to the severity of the metabolic derangement and the poor outcome.

The clinical presentation initially mimicked methanol poisoning, the most reported adulterated alcohol in Bangladesh and associated with multiple outbreaks and fatalities.^{2,13} Both methanol and ethylene glycol poisoning present with central nervous system depression, respiratory distress, visual symptoms, and severe metabolic acidosis, making early differentiation challenging without advanced laboratory testing.^{1,5} Similar diagnostic confusion has been reported in previous cases of glycol-based fluid ingestion, including fog juice poisoning.¹²

Ethylene glycol itself is relatively non-toxic; however, its metabolites, glycolic acid and oxalic acid, cause severe metabolic acidosis, renal tubular injury, and multi-organ dysfunction.⁵ Oxalic acid combines with calcium to form calcium oxalate crystals, which deposit in renal tubules and contribute to acute kidney injury and tubular necrosis.¹⁹ In this patient, the detection of calcium oxalate crystals on urine microscopy was a key diagnostic clue supporting a diagnosis of ethylene glycol poisoning, despite negative serum formate levels.

Early administration of alcohol dehydrogenase inhibitors, such as fomepizole or ethanol, is the cornerstone of management, as these agents prevent the formation of toxic metabolites.^{6,8} In the present case, fomepizole was administered promptly upon suspicion of adulterated alcohol ingestion. However, the patient had already developed profound metabolic acidosis (pH 6.93), which is a known predictor of poor prognosis in ethylene glycol poisoning despite appropriate antidotal therapy.^{20,21}

Hemodialysis is strongly indicated in severe ethylene glycol poisoning, particularly when refractory metabolic acidosis, renal failure, or hemodynamic instability is present, because it effectively removes both the parent compound and its toxic metabolites.^{6,20} Unfortunately, although dialysis was planned for this patient, rapid clinical deterioration and refractory shock prevented its initiation.

Reported mortality rates from ethylene glycol poisoning range from approximately 1% to 22%, depending on geographic region, access to early diagnosis, availability of antidotes, and access to renal replacement therapy.^{20,21} Mortality remains disproportionately high in low- and middle-income countries because of delayed presentation, limited diagnostic facilities, and a lack of timely dialysis.

This case underscores the need to maintain a high index of suspicion for ethylene glycol poisoning in all patients presenting with suspected methanol poisoning and severe metabolic acidosis. Simple bedside tests, such as urine microscopy for calcium oxalate crystals, can play a crucial diagnostic role in resource-limited settings. Strengthening toxicology services, improving access to hemodialysis, and increasing community awareness of the dangers of adulterated alcohol are essential steps toward reducing preventable deaths from adulterated alcohol poisoning.

Conclusion:

Ethylene glycol poisoning is a life-threatening yet underrecognized toxicological emergency in Bangladesh. This case highlights the urgent need for improved awareness, early diagnostic screening, including routine urine

microscopy, and timely administration of antidotes such as fomepizole or ethanol. Increasing public awareness, strengthening toxicology services, and regulating illicit alcohol production are crucial to prevent similar fatalities. Attending clinicians should be aware of the possibility of EG poisoning while receiving any case of suspected methanol poisoning and ask for a urine microscopy to differentiate between them.

Conflicts of interest:

The authors declare no conflicts of interest regarding this publication.

References:

- Gallagher N, Wood FJ. The diagnosis and management of toxic alcohol poisoning in the emergency department: a review article. *Adv J Emerg Med*. 2019;3(3):e28. doi:10.22114/ajem.v0i0.153
- Najari F, Baghbanian I. Methanol poisoning and its treatment. *Int J Med Toxicol Forensic Med*. 2020;10(1):26639. doi:10.32598/ijmtfm.v10i1.26639
- Rebbsat S, Mayer D. Ethylene glycol. In: Ullmann's Encyclopedia of Industrial Chemistry. Weinheim: Wiley-VCH; 2000. doi:10.1002/14356007.a10_101
- Scalley RD, Ferguson DR, Piccaro JC, Smart ML. Treatment of ethylene glycol poisoning. *Am Fam Physician*. 2002;66(5): 807-812.
- Jacobsen D, McMartin KE. Methanol and ethylene glycol poisonings: mechanism of toxicity, clinical course, diagnosis and treatment. *Med Toxicol*. 1986;1(5):309-334. doi:10.1007/BF03259846
- Sivilotti ML, Burns MJ, McMartin KE, Brent J; Methylpyrazole for Toxic Alcohols Study Group. Toxicokinetics of ethylene glycol during fomepizole therapy: implications for management. *Ann Emerg Med*. 2000;36(2):114-125. doi:10.1067/mem.2000.107002
- Kitchens JM. Does this patient have an alcohol problem? *JAMA*. 1994;272(22):1782-1787. doi:10.1001/jama.1994.03520220076034
- Denver PM, Balmer NJ. When patients portray their conduct as normal and healthy: an interactional challenge for thorough substance use history taking. *Soc Sci Med*. 2012;75(9):1650-1659. doi:10.1016/j.socscimed.2012.06.021
- Islam JY, Zaman MM, Bhuiyan MR, et al. Alcohol consumption among adults in Bangladesh: results from STEPS 2010. *WHO South-East Asia J Public Health*. 2017;6(1):67-74. doi:10.4103/2224-3151.206168
- Dewan G, Chowdhury FR. Alcohol use and alcohol use disorders in Bangladesh. *Asia Pac J Med Toxicol*. 2015;4(2):83-90. doi:10.22038/apjmt.2015.5091

11. Department of Narcotics Control (DNC). Annual Drug Report of Bangladesh 2021. Dhaka: Ministry of Home Affairs, Government of Bangladesh; 2021. Available from: <https://dnc.portal.gov.bd>
12. Islam FMM, Saha AK, Mannan MA, et al. Fog juice poisoning. *J Med*. 2013;14(2):195-197.
13. Islam MS, Perkins J, Ghose A. It's time to act against silent deaths from methanol poisoning. *Daily Observer*. 2025 Nov 6; p.4.
14. Baldwin F, Sran H. Delayed ethylene glycol poisoning presenting with abdominal pain and multiple cranial and peripheral neuropathies: a case report. *J Med Case Rep*. 2010;4:220. doi:10.1186/1752-1947-4-220
15. Wilson KC, Saukkonen JJ. Acute respiratory failure from abused substances. *J Intensive Care Med*. 2004;19(4):183-193. doi:10.1177/0885066604263918
16. van Leent A, Wijnia JW. Ethylene glycol intoxication: mind your gap(s)!—a case report. *J Emerg Crit Care Med*. 2014;8:13-27. doi:10.21037/jeccm-24-27
17. Oslo University Hospital. Methanol poisoning protocol – EMACC-WG. 2nd ed. MSF Norway; 2023. Available from: <https://methanolpoisoning.msf.org>
18. Mégarbane B. Treatment of patients with ethylene glycol or methanol poisoning: focus on fomepizole. *Open Access Emerg Med*. 2010;2:67-75. doi:10.2147/OAEM.S5346
19. McMartin K. Are calcium oxalate crystals involved in the mechanism of acute renal failure in ethylene glycol poisoning? *Clin Toxicol (Phila)*. 2009; 47(9):859-869. doi:10.3109/15563650903344793
20. Porter WH, Rutter PW. Ethylene glycol toxicity: the role of serum glycolic acid in hemodialysis. *J Toxicol Clin Toxicol*. 2001;39(6):607-615. doi:10.1081/CLT-100108493
21. Karlson-Stiber C, Persson H. Ethylene glycol poisoning: experiences from an epidemic in Sweden. *J Toxicol Clin Toxicol*. 1992;30(4):565-574. doi:10.3109/15563659209017942