South American Publishing

SAP Primary Care. 2025; 1:46
doi: 10.62486/pc202546

CASE REPORT

Ethylene glycol poisoning as a cause of severe high–anion gap metabolic acidosis: a case report and therapeutic approach in intensive care

Intoxicación por etilenglicol como causa de acidosis metabólica severa con brecha aniónica elevada: reporte de un caso y abordaje terapéutico en cuidados intensivos

Maria Rene Sendoya Alvarez[1], Agar Melvy Paredes Carpio[1], Rommer Alex Ortega Martinez[2], Edson Américo Antequera Rocha[1]

[1]Hospital Obrero N.° 2 de la Caja Nacional de Salud. Cochabamba, Bolivia.
[2]Universidad Privada Abierta Latinoamericana. Bolivia

Cite as
Sendoya Alvarez MR, Paredes Carpiob AM, Ortega Martinez RA, Antequera Rocha EA. Ethylene glycol poisoning as a cause of severe high–anion gap metabolic acidosis: a case report and therapeutic approach in intensive care. SAP Primary Care. 2025; 1:46. https://doi.org/10.62486/pc202546

Publication History
Submitted: 14-06-2025
Revised: 11-08-2025
Accepted: 02-10-2025
Published: 03-10-2025

Corresponding Author
Rommer Alex Ortega Martinez

ABSTRACT

Severe high-anion gap metabolic acidosis is a medical emergency in the Intensive Care Unit and is associated with high morbidity and mortality. Ethylene glycol poisoning is an uncommon but potentially fatal cause, often diagnosed late and challenging to manage, particularly in settings with limited access to specific antidotes. We report the case of an 18-year-old female patient with a history of autism spectrum disorder and moderate intellectual disability who was admitted to the Intensive Care Unit 24 hours after accidental ingestion of ethylene glycol. The patient developed toxic-metabolic encephalopathy and severe high–anion gap metabolic acidosis (pH 6,99; HCO_3^- 2-3 mEq/L), accompanied by critical hyperkalemia and acute kidney injury (KDIGO stage 3). In the absence of specific antidotes, advanced life support and urgent hemodialysis through serial sessions were initiated, resulting in rapid correction of metabolic disturbances, full recovery of renal function, and a favorable clinical outcome. This case highlights the importance of systematic acid–base assessment and early hemodialysis as key strategies to improve outcomes in ethylene glycol poisoning.

Keywords: Metabolic Acidosis; Ethylene Glycol; Poisoning; Hemodialysis; Intensive Care Units.

RESUMEN

La acidosis metabólica severa con brecha aniónica elevada constituye una urgencia médica en la Unidad de Terapia Intensiva y se asocia a elevada morbimortalidad. Entre sus causas infrecuentes, pero potencialmente letales se encuentra la intoxicación por etilenglicol, cuyo diagnóstico suele ser tardío y cuyo manejo representa un desafío clínico, particularmente en contextos con acceso limitado a antídotos específicos. Se presenta el caso de una paciente femenina de 18 años, con antecedentes de trastorno del espectro autista y discapacidad intelectual moderada, que ingresó a la Unidad de Terapia Intensiva 24 horas después de la ingesta accidental de etilenglicol. La paciente desarrolló encefalopatía tóxico-metabólica y acidosis metabólica severa con brecha aniónica elevada (pH 6,99; HCO_3^- 2-3 mEq/L), asociada a hiperpotasemia crítica e injuria renal aguda estadio KDIGO 3. Ante la ausencia de antídotos específicos, se instauró soporte vital avanzado y hemodiálisis urgente mediante sesiones seriadas, logrando la corrección del medio interno, recuperación completa de la función renal y una evolución clínica favorable. Este caso resalta la importancia del análisis sistemático del equilibrio ácido-base y de la instauración precoz de hemodiálisis como estrategias determinantes para modificar el pronóstico en pacientes con intoxicación por etilenglicol.

Palabras clave: Acidosis Metabólica; Etilenglicol; Intoxicación; Hemodiálisis; Unidades de Cuidados Intensivos.

INTRODUCTION

Metabolic acidosis is among the most common and clinically relevant acid–base disorders in critical care medicine and is associated with a significant increase in morbidity and mortality when it reflects the accumulation of unmeasured acids or the failure of compensatory mechanisms. It is defined by a decrease in arterial pH (< 7,35), serum bicarbonate concentration (< 22 mEq/L), and base excess (<-2 mEq/L), parameters whose interpretation must be contextualized according to the patient's altitude, ventilatory status, and hemodynamic condition.[1,2]

In the intensive care unit (ICU), metabolic acidosis is often the epiphenomenon of complex pathophysiological processes such as tissue hypoperfusion, sepsis, acute renal failure, or exogenous poisoning, where early identification of the underlying mechanism is crucial for guiding timely and targeted therapeutic decisions.[3] From a pathophysiological point of view, it can be caused by bicarbonate loss, increased acid load, or decreased renal excretion of hydrogen ions; in this context, calculating the anion gap is the most useful clinical tool for differential diagnosis.[3,4]

Ethylene glycol poisoning, described since the mid-20th century, has become established as a classic, albeit rare, cause of severe metabolic acidosis with a high anion gap in emergency departments and intensive care units.[4,5] Worldwide, toxic alcohols account for approximately 1–2 % of poisoning consultations, with ethylene glycol responsible for a significant proportion of severe cases, especially in regions with limited access to specific antidotes such as fomepizole. Mortality can reach between 20 % and 50 % in the absence of timely treatment but is significantly reduced with early initiation of hemodialysis.[5,6,7]

In Latin America, available reports are scarce and are probably overlooked; published series describe late presentations, profound metabolic acidosis, and established acute kidney injury, which lead to a more sluggish clinical course than European or North American series.[8,9,10] In Bolivia, published evidence is minimal and limited to isolated reports, and the irregular availability of antidotes makes extracorporeal purification the main therapeutic pillar in these cases.

After ingestion, ethylene glycol is metabolized by alcohol dehydrogenase to glycolaldehyde, glycolic acid, and oxalic acid, which are the metabolites responsible for progressive neurological compromise, severe metabolic acidosis, and characteristic acute kidney injury associated with the precipitation of calcium oxalate crystals.[4,5,6,7] Clinically, the disease usually progresses in the neurological, cardiopulmonary, and renal phases, the overlap of which can make initial diagnosis difficult.

In this context, we present the case of a young patient with severe metabolic acidosis with a high anion gap secondary to ethylene glycol poisoning whose management required advanced life support and urgent extracorporeal purification, highlighting the diagnostic challenges and clinical implications relevant to the practice of the intensivist.

CASE REPORT

An 18-year-old female patient with a history of autism spectrum disorder and moderate intellectual disability was admitted to the Intensive Care Unit of the Caja Nacional de Salud's Hospital Obrero No. 2, 24 hours after accidentally ingesting ethylene glycol (brake fluid), as confirmed by the responsible family member through identification of the container.

Upon admission, the patient presented with toxic metabolic encephalopathy characterized by agitation, asthenia, and progressive drowsiness, with a compromised general condition. The initial evaluation revealed severe metabolic acidosis with an elevated anion gap (pH: 6,99; bicarbonate concentration: 2–3 mEq/L; base excess: -29.3), which was associated with critical hyperkalemia (K^+: 6.0 mEq/L) and acute kidney injury stage KDIGO 3. Clinical assessment was limited by the underlying neurological conditions, which reinforced the importance of the history of exposure and acid–base analysis for diagnosis.

Given the neurological deterioration and hemodynamic and ventilatory instability, advanced life support was initiated with orotracheal intubation, invasive mechanical ventilation, and vasopressor support with norepinephrine. Given the impossibility of accessing specific antidotes and the severity of the metabolic disorder, it was decided to initiate emergency hemodialysis through a femoral venous catheter and perform three serial sessions aimed at clearing toxic metabolites and correcting the internal environment. As part of the adjunctive management, B-complex vitamins (thiamine and pyridoxine) were administered.

During her stay in the ICU, the patient developed ventilator-associated pneumonia, with *Escherichia coli* identified by tracheal secretion culture, leading to targeted antibiotic therapy with an adequate clinical response. Upper gastrointestinal endoscopy was performed to rule out caustic lesions associated with ingestion, which revealed no evidence of structural involvement of the upper digestive tract (figure 1).

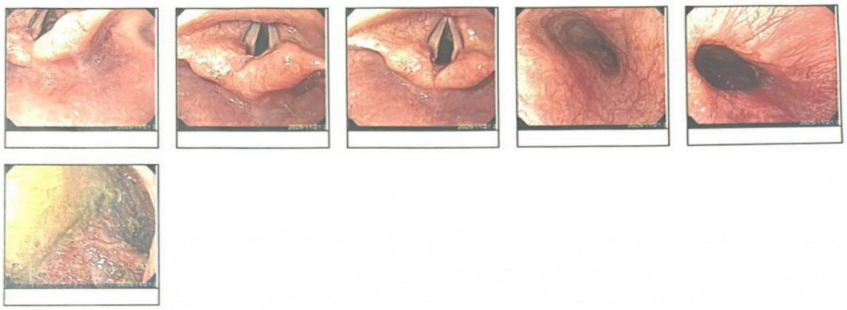

Figure 1. Upper gastrointestinal endoscopy; showed a pale pink oropharynx with no evidence of lesions, an esophagus with preserved distensibility and pale pink mucosa, an irregular squamocolumnar- n junction at 36 cm from the upper dental arch (UDA), diaphragmatic pinching at 37 cm from the UDA, and a stomach with abundant solid food content

After five days of multidisciplinary management, the patient showed marked metabolic and neurological improvement, with progressive normalization of acid–base balance and complete recovery of renal function (estimated GFR 128 mL/min). Given the patient's hemodynamic stability, adequate ventilatory mechanics, and effective cough reflex, successful scheduled extubation was performed, and conventional oxygen therapy was continued.

Finally, the patient was transferred to the internal medicine ward with a favorable outcome and no organic sequelae attributable to the acute event, with low severity scores at ICU discharge (APACHE II 5; SOFA 2).

DISCUSSION

Ethylene glycol poisoning is among the classic, albeit rare, causes of severe metabolic acidosis with a high anion gap in critically ill patients and represents a real diagnostic and therapeutic challenge when medical history is limited or when the history of exposure is not initially evident. In this context, systematic analysis of acid–base balance and early identification of characteristic biochemical patterns are essential to avoid potentially lethal therapeutic delays.[4,5]

From a pathophysiological point of view, clinical severity is not determined by ethylene glycol per se but by its acidic metabolites, mainly glycolic acid and oxalic acid, which are responsible for profound metabolic acidosis, neurological compromise, and acute kidney injury. Several clinical series agree that the degree of acidosis (pH < 7.1 and bicarbonate concentration < 5 mEq/L) is closely correlated with increased mortality and the need for urgent extracorporeal purification, regardless of the amount ingested.[6,7,8]

In international series from Europe and North America, standard management includes the early use of specific antidotes such as fomepizole or ethanol, with the aim of inhibiting alcohol dehydrogenase activity and preventing the formation of toxic metabolites. In these scenarios, hemodialysis is reserved for patients with severe acidosis, neurological compromise, acute renal failure, or high serum ethylene glycol concentrations.[7,8,9] However, recent clinical guidelines and systematic reviews report that, even in the presence of antidotes, hemodialysis remains the most effective method for eliminating toxic metabolites and rapidly correcting the internal environment.[9,10,11]

However, multiple Latin American reports describe a different reality, characterized by limited access to specific antidotes and frequently delayed diagnosis, which leads to greater dependence on hemodialysis as the initial therapeutic pillar.[10,11,12] In this context, Carrasco-Nieva and Sánchez-Villegas reported a case of ethylene glycol poisoning in Mexico that was managed in the emergency room, where delayed diagnosis was associated with profound metabolic acidosis and established renal injury, necessitating urgent hemodialysis as a life-saving measure.[12]

Similarly, Esper et al. described a series of cases with rapid clinical evolution in the absence of alcohol dehydrogenase inhibitors, highlighting that early initiation of extracorporeal purification was decisive for survival, even in scenarios of extreme acidosis.[13] Other Latin American authors have reported that the severity of the initial metabolic compromise is a more robust predictor of mortality than the dose ingested or the exact time of exposure.[14]

Our case presents several elements of clinical interest. First, 24 hours after ingestion, the patient was already in an advanced stage of intoxication, with severe metabolic collapse (pH 6.99; bicarbonate concentration 2–3 mEq/L) and acute kidney injury stage KDIGO 3. These findings are consistent with the literature, where delayed treatment initiation is associated with greater clinical severity and worse prognosis.[6,7,8,9] In addition, underlying neurological conditions limited the initial clinical evaluation, underscoring the importance of acid–base analysis and the exposure history provided by the family member to guide the diagnosis.

From a therapeutic standpoint, the inability to access specific antidotes led to a strategy focused on advanced life support and urgent hemodialysis as a definitive treatment. This approach is widely supported by evidence indicating that hemodialysis is the most effective intervention for the elimination of ethylene glycol and its metabolites, as well as for the rapid correction of metabolic acidosis and associated electrolyte disturbances.[9,10,11,15] In our case, three serial sessions allowed for progressive normalization of the internal environment, complete recovery of renal function, and resolution of neurological compromise, without complications related to the procedure.

The favorable outcome observed contrasts with the high mortality described in patients with extreme metabolic acidosis when treatment is delayed or incomplete. This outcome reinforces the concept that, even in scenarios with therapeutic limitations, a timely, protocolized, and multidisciplinary approach can significantly modify the prognosis.[13,14,15] Likewise, the exclusion of caustic lesions by endoscopy and the adequate management of intercurrent infectious complications contributed to the patient's full recovery.

Finally, this case provides relevant clinical evidence in a context where national literature is scarce, highlighting the need to maintain a high index of suspicion for severe metabolic acidosis with a high anion gap, particularly in young and vulnerable patients. The results of this study confirm that early hemodialysis is an effective, safe, and life-saving strategy when specific antidotes are not available, establishing itself as the central therapeutic approach in intensive care units in countries with limited resources.[12,13,14,15]

FUNDING

The authors did not receive funding for the conduct of this research.

CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest.

AUTHORSHIP CONTRIBUTIONS

Conceptualization: Maria Rene Sendoya Alvarez, Agar Melvy Paredes Carpio, Rommer Alex Ortega Martinez, Edson Américo Antequera Rocha.

Formal analysis: Maria Rene Sendoya Alvarez, Agar Melvy Paredes Carpio, Rommer Alex Ortega Martinez, Edson Américo Antequera Rocha.

Research: Maria Rene Sendoya Alvarez, Agar Melvy Paredes Carpio, Rommer Alex Ortega Martinez, Edson Américo Antequera Rocha.

Project management: Maria Rene Sendoya Alvarez, Agar Melvy Paredes Carpio, Rommer Alex Ortega Martinez, Edson Américo Antequera Rocha.

Writing – original draft: Maria Rene Sendoya Alvarez, Agar Melvy Paredes Carpio, Rommer Alex Ortega Martinez, Edson Américo Antequera Rocha.

Writing – proofreading and editing: Maria Rene Sendoya Alvarez, Agar Melvy Paredes Carpio, Rommer Alex Ortega Martinez, Edson Américo Antequera Rocha.

REFERENCES

1. Hopkins E, Sanvictores T, Sharma S. Physiology, Acid Base Balance. [Updated 2022 Sep 12]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK507807/

2. Sánchez-Díaz JS, Martínez-Rodríguez EA, Méndez-Rubio LP, Peniche-Moguel K. Equilibrio ácido-base. Puesta al día. Teoría de Henderson-Hasselbalch. Med Int Mex. 2016;32(6):640–660. Disponible en: https://medicinainterna.org.mx/article/equilibrio-acido-base-puesta-al-dia-teoria-de-henderson-hasselbalch/

3. Hidalgo-Acosta IV, Mena-Miranda VR, Fernández de la Paz B, Heredero-Valdés M, Ruiz-Baldrich WA. Acidosis metabólica: un reto para los intensivistas. Rev Cubana Pediatr. 2005;77(2). Disponible en: http://scielo.sld.cu/scielo.php?script=sci_arttext&pid=S0034-75312005000200008

4. Sepúlveda AR, Selamé E, Roessler E, Tagle R, Valdivieso A. Intoxicación por etilenglicol: fisiopatología y enfrentamiento clínico. Rev Med Chil. 2019;147(12):1572–1578. doi:10.4067/S0034-98872019001201572

5. Gallagher N, Edwards FJ. The Diagnosis and Management of Toxic Alcohol Poisoning in the Emergency Department: A Review Article. Adv J Emerg Med. 2019 May 22;3(3):e28. doi: 10.22114/ajem.v0i0.153.

6. Sasanami M, Yamada T, Obara T, Nakao A, Naito H. Oral ethanol treatment for ethylene glycol intoxication. Cureus. 2020;12(11):e12268. doi:10.7759/cureus.12268

7. Stašinskis R, Stašinska K, Mukāns M, Graudiņš A, Liguts V, Lejnieks A. Changes in ionized calcium in ethylene glycol poisoning. Proc (Bayl Univ Med Cent). 2022 Apr 27;35(4):460-465. doi: 10.1080/08998280.2022.2062550.

8. Carrasco-Nieva M, Sánchez-Villegas MC. Intoxicación por etilenglicol: reporte de caso, un reto en el tratamiento en la sala de urgencias. Arch Med Urg Mex. 2024;16(2):140–148. Disponible en: https://www.medigraphic.com/cgi-bin/new/resumen.cgi?IDARTICULO=117761

9. Kraut JA, Madias NE. Metabolic acidosis: pathophysiology, diagnosis and management. Nat Rev Nephrol. 2010 May;6(5):274-85. doi: 10.1038/nrneph.2010.33.

10. Esper RC, González D, Quintana L, Rea E, Bravo L. Intoxicación por etilenglicol: reporte de un caso y revisión de la literatura. Acta Médica. 2006;4(4):243–248. Disponible en: https://www.medigraphic.com/cgi-bin/new/resumen.cgi?IDARTICULO=11128

11. Barceloux DG, Bond GR, Krenzelok EP, Cooper H, Vale JA; American Academy of Clinical Toxicology Ad Hoc Committee on the Treatment Guidelines for Methanol Poisoning. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. J Toxicol Clin Toxicol. 2002;40(4):415-46. doi: 10.1081/clt-120006745.

12. Laverde-Sabogal CE, Valencia-Cárdenas AR, Vega-Sandoval CA. Una causa inusual de acidosis metabólica severa: ayuno prequirúrgico. Rev Colomb Anestesiol. 2014;42(4):312–316. Disponible en: http://www.scielo.org.co/scielo.php?script=sci_arttext&pid=S0120-33472014000400010

13. Brent J. Fomepizole for ethylene glycol and methanol poisoning. N Engl J Med. 2009;360(21):2216–2223. doi:10.1056/NEJMct0806112

14. Kruse JA. Methanol and ethylene glycol intoxication. Crit Care Clin. 2012;28(4):661–711. doi:10.1016/j.ccc.2012.07.002

15. Duraković Z, Gasparović V, Plavsić F. Otrovanje etilen glikolom lijeceno hemodijalizom. Arh Hig Rada Toksikol. 1990 Jun;41(2):201-7. Disponible en: https://pubmed.ncbi.nlm.nih.gov/2248555/