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Acute Ethylene Glycol Poisoning Progressing to Renal Failure and Septic Shock: A Case Report

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ABSTRACT

We present the case of a 57-year-old male patient with acute confusional state, metabolic acidemia with elevated anion gap and hyperlactatemia, suspected of ethylene glycol intoxication. Management involved ventilatory support, fluid resuscitation, intravenous bicarbonate, pyridoxine supplementation, and hemodialysis. The patient subsequently developed septic shock and died after a prolonged hospitalization.

Keywords

Ethylene glycol poisoning, Acute confusional state, High anion gap metabolic acidosis, Hyperlactatemia, Toxic alcohol ingestion.

Introduction

Ethylene glycol poisoning is a life-threatening medical emergency [1]. Its clinical presentation can mimic other causes of metabolic acidosis; therefore, early recognition is crucial for appropriate management. Treatment includes specific antidotes (fomepizole or ethanol), correction of acid-base imbalance, and hemodialysis.

Case Report

A 57-year-old male patient with a history of DM2 on treatment with metformin without specifying the dose; cholecystectomy and chronic alcoholism. He was brought to the emergency department by a family member reporting general malaise, confusion, and dyspnea, with progressive symptoms for 10 hours beginning after ingestion of "*a glass of brandy*." Upon arrival, he was stuporous, vomiting, and responded poorly to stimuli.

Vital signs on admission to the emergency room

- BP: 70/40 mmHg
- HR: 115 bpm
- RR: 32 rpm
- T: 35°C
- Glasgow: 9 (O2, V3, M4)

A dehydrated oral cavity was documented, without characteristic breath, capillary filling for 5 seconds. Hydration was initiated through peripheral and central venous accesses.

Initial arterial blood gases in the emergency department:

- pH: 7.11
- PCO_2: 13 mmHg
- HCO_3^-: 4.1 mmol/L
- Lactate: 15 mmol/L
- EB: -25.4

Initial emergency laboratories

- Glucose: 127 mg/dL
- Creatinine: 1.5 mg/dL
- Sodium: 150 mmol/L
- Potassium: 5.4 mmol/L
- Chlorine: 115 mmol/L

Intoxication with a non-ethanol alcohol, likely ethylene glycol, was

suspected due to metabolic acidemia with an elevated anion gap and a normal osmolar gap. Pyridoxine, thiamine, and bicarbonate were administered. Three hours after admission, endotracheal intubation was performed, and he was admitted to the Intensive Care Unit. Figure 1 shows the blood gas evolution during their stay in the Emergency Department.

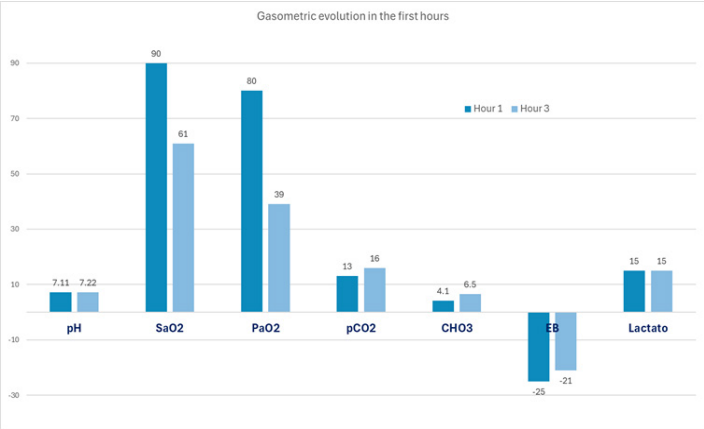

Figure 1: Arterial Blood Gas Evolution During the First Hours in the Emergency Department.

Clinical evolution in ICU

Table 1: Fluid Balance and Urine Output During ICU Stay.

Date	Entry (ml)	Outflow (ml)	Balance (ml)	Diuresis (ml)	Diuresis/kg/hour (ml/kg/h)
09/04/25	2,650	3,951	-1,301	2,020	1.8
10/04/25	8,914	3,289	5,625	1,429	0.74
11/04/25	3,203	2,460	743	334	0.17
12/04/25	2,523	2,024	499	40	0.02
13/04/25	3,702	1,679	2,023	90	0.04
14/04/25	2,999	4,299	-1,300	45	0.02
15/04/25	2,308	2,560	-252	50	0.02
16/04/25	1,646	1,785	-139	110	0.05

Table 2: Trends in Serum Creatinine and Electrolytes During ICU Stay.

Date	Hemodialysis	Creatinine (mg/dL)	Na (mmol/L)	K (mmol/L)	Cl (mmol/L)
09/04/25	No	2.6	155	7.0	116
10/04/25	Yes	3.3	159	4.7	116
11/04/25	Yes	6.1	158	4.2	114
12/04/25	No	7.3	152	3.6	107
13/04/25	Yes	10.0	150	3.6	106
14/04/25	Yes	11.2	146	4.0	103
15/04/25	No	10.3	144	5.1	104
16/04/25	Yes	11.0	143	5.3	102

It was not possible to administer the specific antidotes because they were lacking. For this reason, hemodialysis started early. Tables 1 and 2 show the water balance and the evolution of blood chemistry.

The patient died eight days after being admitted to the hospital with the following diagnoses: septic shock of pulmonary origin, pneumonia associated with mechanical ventilation due to *Klebsiella pneumoniae*, urinary tract infection due to *Acinetobacter baumannii complex*, acute kidney disease on replacement treatment with hemodialysis, and uncontrolled DM2.

Discussion

The patient presented to the emergency department ten hours after ingesting a "*glass of brandy*," likely containing ethylene glycol. On admission he was stuporous, with severe metabolic acidemia, hyperlactatemia, and an elevated anion gap. All of this is highly suggestive of ethylene glycol poisoning. Specific antidotal therapy was considered; however, antidotes were unavailable. Therefore, fluid resuscitation, intravenous bicarbonate, endotracheal intubation, and hemodialysis were initiated. Despite the measures implemented, the clinical situation deteriorated until his death.

Figure 1 shows how oxygen saturation decreased and CO2 pressure increased, which is why endotracheal intubation was performed for mechanical ventilatory management.

Table 1 shows how diuresis progressively decreased. Table 2 has a very precise correlation between the decrease in urine output indicated in Table 1 and the increase in creatinine. Although it could not be demonstrated by laboratory studies, the clinical picture is highly suggestive of intoxication by non-ethyl alcohol, most likely ethylene glycol. The patient was taken to the emergency room late, Phase 2, so there is a normal osmolar gap, as we will see in the next section.

This case highlights the importance of going in the first minutes when poisoning is suspected. Additionally, it is essential to have specific antidotes in emergency and intensive care units, as well as the need for clear protocols in the event of suspected poisoning by toxic alcohols.

Review of the Topic

Introduction

Ethylene glycol (1,2-ethanediol, or ethane-1,2-diol) is a liquid alcohol, colorless, odorless and with a characteristic sweet taste, properties that contribute to accidental poisoning. Its use is widely used in industry as a main component of antifreeze fluids and coolants, as well as solvent in the manufacture of plastics and pigments [1]. In the United States alone, between 3,000 and 4,000 cases of poisoning are reported each year, affecting the pediatric population especially frequently [2]; In 2003, 5.3% of all poisonings were due to toxic alcohol [3]. The lethal toxic dose is commonly referred to as 100 mL or 1–1.5 mL/kg, although there is considerable interindividual variability and mortality has been reported at doses as low as 30 mL [4]. The reported mortality is about 20% in cases of ethylene glycol poisoning. Although ethylene glycol itself has a low intrinsic toxicity, its metabolic biotransformation in the body results in the formation of highly toxic acidic compounds that are responsible for a severe clinical picture, characterized by metabolic acidosis, acute kidney injury and multiorgan dysfunction.

Pathophysiology of poisoning

Ethylene glycol is rapidly and completely absorbed by the gastrointestinal tract, reaching peak concentrations between 30 and 60 minutes after ingestion [4]. Contact with intact skin or inhalation does not usually produce toxic effects, although it can be absorbed through injured skin. A clear understanding of the underlying pathophysiology is essential for effective management of ethylene glycol poisoning. The damage is not caused by the original alcohol, but by its biotransformation into acidic metabolites. This principle is the pillar on which both diagnosis and treatment are built, the main objective of which is to stop this toxic metabolic conversion.

Pharmacokinetics and Hepatic Metabolism

Once ingested, ethylene glycol is rapidly and completely absorbed into the gastrointestinal tract, reaching peak plasma concentrations within minutes of exposure. Its water-soluble nature gives it a low affinity for plasma proteins and a volume of distribution like that of total body water (0.5 - 0.8 L/kg).

The elimination of ethylene glycol follows two main routes:

- **Hepatic metabolism**: Approximately 80% of ingested ethylene glycol is metabolized in the liver [5].
- **Renal excretion**: The remaining 20% is excreted unchanged through the kidneys.

The hepatic metabolic pathway is a sequential cascade of oxidation that produces the compounds responsible for systemic toxicity [5-9]:

1. Ethylene glycol is oxidized to glycoaldehyde by the enzyme Alcohol Dehydrogenase (ADH).
2. Glycoaldehyde is converted to glycolic acid by Aldehyde Dehydrogenase.
3. Glycolic acid is metabolized to glyoxylic acid.
4. Glyoxylic acid is eventually transformed into oxalic acid.

The half-life of ethylene glycol is 3 to 8.6 hours under normal conditions. However, this is significantly prolonged in the presence of ADH inhibitors, such as ethanol (up to 17-18 hours) or fomepizole (up to 20 hours), a key pharmacological intervention in early treatment when poisoning is suspected.

Mechanisms of Cellular and Systemic Toxicity

Ethylene glycol toxicity manifests itself through three main mechanisms that affect multiple organ systems [4,10,11].

1. **Metabolic acidosis with elevated anion gap**

Acidic metabolites, primarily glycolic acid, dissociate in plasma, releasing a large amount of hydrogen ions. These ions consume serum bicarbonate stores, resulting in severe metabolic acidosis with elevated anion gap (anion gap). Profound acidosis is one of the main causes of morbidity and mortality in this poisoning.

2. **Nephrotoxicity due to calcium oxalate crystals**

Acute kidney injury is the most characteristic complication. Key research, such as that of Guo and McMartin, has conclusively shown that the central mechanism is damage caused by calcium oxalate monohydrate crystals, and not by the free oxalate ion. Oxalic acid, the end product of metabolism, combines with ionized calcium in the blood and precipitates in the renal tubules, causing damage through two main pathways: 1) physical obstruction: crystals accumulate and block the tubular lumen, impeding the flow of urine, and 2) direct cytotoxic injury: tubular epithelial cells internalize calcium monohydrate oxalate crystals. This process triggers a cascade of cell damage that includes internalization of crystals in epithelial cells, inhibition of mitochondrial electron transport, blockade of the Krebs cycle, ATP depletion, and eventually acute tubular necrosis. Severe acidosis (pH < 6.5) has been shown to significantly potentiate the cytotoxicity of calcium oxalate monohydrate crystals, creating a vicious cycle of kidney damage.

3. **Neurological and cardiopulmonary toxicity**

Initially, ethylene glycol acts as an osmotically active solute, causing cerebral edema and central nervous system (CNS) depression [12]. As intoxication progresses, hypocalcemia (secondary to calcium oxalate precipitation) and severe metabolic acidosis contribute to myocardial depression, arrhythmias, and cardiopulmonary collapse.

Understanding these mechanisms of damage is essential, as they directly correlate with the observable clinical manifestations detailed below.

Clinical picture and evolution by stages

The clinical presentation of ethylene glycol poisoning is dynamic and evolves in predictable phases, the early recognition of which is crucial for effective treatment and to prevent permanent sequelae [2,13].

Stage 1: Neurological (first 12 hours)

The initial manifestations are predominantly neurological [14]. Symptoms resemble those of alcohol poisoning, including euphoria, dysarthria, ataxia, and drowsiness. However, a key finding is the absence of alcoholic breath. As intoxication progresses, more serious signs such as nystagmus, seizures, and a rapid progression to coma may appear.

Stage 2: Cardiopulmonary (12 to 24 hours)

At this stage, the accumulation of toxic metabolites causes severe metabolic acidosis, which dominates the clinical picture. Cardiovascular abnormalities may include tachycardia, bradycardia, and shock. At the respiratory level, the patient develops deep and rapid compensatory polypnea (Kussmaul breathing) to correct the acidosis. Mortality in this phase is high if aggressive treatment is not instituted.

Stage 3: Renal (after 24 hours)

This phase is characterized by the appearance of acute renal failure, a direct result of nephrotoxicity due to calcium oxalate crystals.

Clinical signs include low back pain (flank), oliguria or anuria, proteinuria, and hematuria. Although recovery of kidney function is possible, it can take weeks and often requires temporary renal replacement therapy [15].

Stage 4: Late neurological sequelae (after 1 week)

In some cases, cranial nerve neuropathies may appear as a delayed manifestation. This phenomenon, usually transient, is attributed to the deposition of calcium oxalate crystals in the cranial nerves. Given the progressive evolution of this condition, the diagnosis depends on a combination of high clinical suspicion and the use of specific laboratory tools.

Diagnostic strategies

Diagnosing ethylene glycol poisoning is challenging, especially without a clear history of ingestion. It is based on the integration of clinical suspicion, characteristic laboratory findings and, when available, toxicological confirmation [9,11].

Clinical suspicion and initial findings

Suspicion should arise in the case of a patient who presents with an altered mental status, metabolic acidosis of unknown cause, and/or unexplained acute renal failure. The combination of these findings is highly suggestive of poisoning by a toxic alcohol.

Key laboratory data

Osmolar gap (Osmolal gap): This is the first and earliest indicator. The osmolar gap is the difference between the osmolality measured in serum and the calculated osmolality. It rises immediately after ingestion due to the presence of unmetabolized ethylene glycol.

- A value > 10 mOsm/kg is considered suspicious.
- A value > 20 mOsm/kg is highly suggestive.

Limitation: This gap decreases as alcohol is metabolized. Therefore, a normal value in a late presentation does not rule out poisoning.

Anion gap (anion gap) and metabolic acidosis: As ethylene glycol is converted into its acidic metabolites (mainly glycolate), the anionic gap progressively increases. Typical findings include an arterial pH < 7.3 and serum bicarbonate < 20 mmol/L.

It is essential to understand the inverse temporal relationship between the two gaps: in the first hours after ingestion, the osmolar gap is maximum (due to unmetabolized ethylene glycol) while the anion gap is normal or minimally elevated. As metabolism progresses, the osmolar gap decreases and the anion gap increases progressively due to the accumulation of acidic metabolites. A patient who presents late may have a normal osmolar gap but severe metabolic acidosis with a very high anion gap.

Urinalysis: a) crystalluria: the search for calcium oxalate crystals is essential; b) Wood's lamp fluorescence test: This test detects fluorescein added to many antifreezes. However, its usefulness is low due to its low sensitivity and the high incidence of false positives and negatives.

Other findings: a) hypocalcemia: due to the precipitation of calcium with oxalate; b) lactate elevation: glycolate can interfere with some blood gas analyzers that use the lactate oxidase enzyme, causing a false lactate elevation; c) Increased creatinine and urea: indicative of acute kidney injury.

Toxicological confirmation

Gas chromatography is the gold standard method for directly measuring ethylene glycol levels in the blood and confirming the diagnosis. However, its availability is limited in many centers, and the processing time prevents it from being a useful tool for decision-making in the acute phase, where treatment should be initiated immediately based on clinical suspicion and indirect laboratory findings.

Diagnostic findings allow the patient's risk to be stratified and the intensity of treatment to be guided.

Risk stratification and Prognostic factors

The identification of factors with a poor prognosis at admission is essential to intensify surveillance and apply more aggressive treatment. Various studies, such as that of Grigorasi et al. [15], have identified key predictors of mortality and/or prolonged renal failure.

1) Clinical signs:
a. Coma or altered mental status on admission (e.g., low Glasgow Coma Scale).
b. Presence of seizures.
2) Laboratory Parameters:
a. Severe metabolic acidosis on admission (arterial pH < 7.1 - 7.25).
b. Markedly diminished alkaline reserve.
c. Elevated serum creatinine at initial presentation.
3) Temporal factors:
a. Delayed presentation to the hospital (more than 8 hours after ingestion).

Therapeutic management should be aggressive and multifaceted, especially in patients who have one or more of these risk factors.

Management and treatment

The treatment of ethylene glycol poisoning is based on three fundamental and simultaneous pillars: 1) life-sustaining measures, 2) inhibition of the production of toxic metabolites by antidotes, and 3) elimination of the original alcohol and its metabolites from the body [9].

General Support Measures

Hemodynamic and respiratory support: Stabilizing the patient is a priority, which may include fluid therapy, use of vasopressors and, in cases of respiratory depression or severe acidosis, intubation and mechanical ventilation.

Correction of acidosis: Intravenous sodium bicarbonate is administered to correct severe metabolic acidosis (pH target > 7.25). Not only does this improve hemodynamic status, but it can also reduce kidney toxicity.

Management of hypocalcemia: Intravenous calcium should be administered only if the patient has signs or symptoms of hypocalcemia (eg, QT prolongation on electrocardiogram, tetany). Routine administration should be avoided so as not to aggravate precipitation of calcium oxalate crystals.

Inhibition of alcohol dehydrogenase (antidotes)

Immediate blockade of the ADH enzyme is the therapeutic mainstay and the most important intervention to stop the production of toxic metabolites. The initiation of treatment with an antidote should be immediate in the event of a well-founded clinical suspicion [1,7,13].

Criteria for Initiating Antidote

Documented plasma concentration of ethylene glycol ≥ 20 mg/dL.
Documented history of toxic ingestion and an osmolar gap > 10 mOsm/kg.
Strong clinical suspicion plus at least TWO of the following: arterial pH < 7.3, bicarbonate < 20 mmol/L, osmolar gap > 10 mOsm/kg, or calcium oxalate crystalluria.

Fomepizole (4-methylpyrazole)

Fomepizole is the antidote of choice for several reasons [7]:

- High affinity: It has an affinity for ADH 8000 times greater than ethanol, making it a very potent inhibitor.
- Safety profile: Does not cause CNS depression or hypoglycemia.
- Simple dosing: Its dosing regimen is simpler and more predictable.
- Dosing regimen: loading dose of 15 mg/kg, followed by 10 mg/kg every 12 hours.

Ethanol

Ethanol is an effective alternative when fomepizole is not available.

- Mechanism: Acts as a competitive substrate for the ADH enzyme, displacing ethylene glycol.
- Objective: to maintain a serum concentration of 100-150 mg/dL for effective inhibition.
- Disadvantages: causes CNS intoxication and depression, can lead to hypoglycemia, and requires frequent monitoring of serum levels to adjust dose, complicating clinical management.

Extracorporeal purification therapy (hemodialysis)

Intermittent hemodialysis is the method of choice and the most effective for extracorporeal clearance, rapidly eliminating both ethylene glycol and its toxic metabolites (glycolate, oxalate). Its onset should not be delayed in patients with severe poisoning [5].
Indications for Hemodialysis:

- Severe metabolic acidosis (e.g., pH < 7.25) that does not respond to bicarbonate therapy.
- Acute renal failure (increased creatinine, oliguria/anuria).
- Progressive clinical deterioration despite supportive therapy and antidote.
- Very high serum ethylene glycol concentration (> 50 mg/dL).

It is crucial to understand that hemodialysis may be indicated for the sole purpose of removing the poison and its metabolites, even in the absence of established kidney failure.

Adjuvant therapies

The use of thiamine and pyridoxine has been proposed as enzymatic cofactors that could theoretically divert the metabolism of glyoxylate towards less toxic end products, such as glycine. Although its administration is safe and inexpensive, its clinical benefit has not been prospectively demonstrated in controlled studies.

Conclusion

Ethylene glycol poisoning is a life-threatening medical emergency, the severity of which lies not in the parent compound but in its toxic metabolites. The success of management depends on a high clinical suspicion in a patient with elevated anion gap and osmolar metabolic acidosis, especially if it is accompanied by neurological alterations or acute renal failure. Timely and aggressive treatment, based on inhibition of alcohol dehydrogenase with antidotes (fomepizole is the option of choice) and early indication of hemodialysis in severe cases, are interventions that save lives, prevent permanent renal failure, and significantly improve the patient's prognosis.

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