# Turning the Tide: Successful Management of Severe Ethylene Glycol Poisoning

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- ethylene glycol poisoning
- metabolic acidosis
- hemodialysis
- antidotal ethanol therapy
- kidney failure

**Ethylene glycol poisoning is a severe condition that, if left untreated, can have a lethal outcome. We illustrate an efficient treatment plan that led to a full recovery following ethylene glycol consumption of a 49-year-old patient. The clinical diagnosis was based on metabolic acidosis, an elevated osmolal gap, and a history or suspicion of ethylene glycol ingestion. The first line of treatment must focus on addressing symptoms, which includes stabilizing the cardiovascular and respiratory systems and managing seizures or arrhythmias. One of the most difficult aspects of treating ethylene glycol poisoning is controlling acidosis using sodium bicarbonate and returning the electrolyte panel to normal. Following the confirmation of the accurate diagnosis, cofactor therapy involving thiamine and pyridoxine should be employed in addition to antidotal medication. Hemodialysis is also a helpful procedure in cases of severe toxicity or renal impairment resulting of ethylene glycol poisoning.**

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## Introduction

Ethylene glycol poisoning, although not common, is a severe clinical condition [1]. Metabolic acidosis is a serious problem in emergency medicine units, and severe episodes of acidosis may pose challenges with diagnosis and treatment. A quick and accurate diagnosis, intensive supportive care, the right use of particular antidotes, and, in certain cases, hemodialysis is all necessary for a successful course of treatment [2,3]. Ethylene glycol is a major factor in metabolic acidosis, which leads to acute kidney failure. Kidney failure and death are possible in case of delayed diagnosis, but when patient receives diagnosis and therapy quickly, lethal outcomes are not common [4].

## Case report

A 49-year-old male with a history of alcohol abuse was brought early in the morning on March 5th by an ambulance to the Emergency Department (ED) of the Military Institute of Medicine. According to the ambulance chart, the last time he was seen fully conscious was the previous day in the morning. His family found him at around 4 p.m. He was initially suspected of alcohol intoxication. Due to the patient's prolonged unconsciousness, the ambulance was called at midnight. Upon arrival, the patient was found to be deeply unconscious with narrowed pupils. Suspecting opioid intoxication, 800 µg of naloxone was administered, but it gave no expected effect. The patient was then transported to the hospital.

The initial investigation conducted at the hospital revealed the following findings:

- Glasgow Coma Scale (GCS) 3;
- profound metabolic acidosis with pH of 6.5;
- lactate level was indeterminate;
- potassium serum concentration of 6.1 mmol/l;
- creatinine serum concentration: 2.2 mg/dl;
- ECG on admission revealed atrial fibrillation with a ventricular rate of 120 beats per minute without features of ischemia; on follow-up, the sinus rhythm returned.

Blood count results showed the following abnormalities, with the most important, as follows: WBC:

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51.99 x 10^9/l; MCV: 105 fl; MCHC 29.2 g/dl; PLT: 163-347 x10^9/l.

On admission, physical examination revealed a minor abrasion on the frontal region and the left cheek, along with a small amount of dried blood within the nasal cavity. No other overt post-traumatic findings were observed.

The patient was in a severe general condition, profoundly unconscious, with a GCS score of 3, blood pressure of 174/89 mmHg, heart rate of 80 beats per minute, and oxygen saturation of 98%. During auscultation, an exacerbated alveolar murmur was noted, along with multiple wheezes and crackles. There were no signs of lung congestion. Heart tones were moderately loud, with no pathological murmurs. The abdomen was soft, peristalsis was reduced, and there were no peritoneal signs.

A single tonic-clonic seizure occurred and was treated with clonazepam 1 mg i.v. Within the ED, the following medications were given: ondansetron 8 mg i.v., propofol 50 mg i.v., NaHCO3 4x20 ml, and 1000 ml 0,9% NaCl i.v.

Within the ED, the patient was intubated, cannulas were inserted into the right radial artery, and the urinary bladder was catheterized, yielding 150 ml of straw-colored urine. Regular diuresis monitoring was commenced, but no diuresis was observed thereafter.

The head CT scan was performed to rule out any possible brain injury. The result came out without any visible pathology.

After initial provision in the ED, the patient was transferred to the Clinical Cardiac Intensive Care Unit (CCICU) for further treatment.

The patient's severe acidosis required careful monitoring. The results are shown in Figure 1.

Due to a strong suspicion of toxic alcohol ingestion, the causative therapy was initiated before the toxicology results were obtained. The results came back positive, with an ethylene glycol level of 51 mg/dL. 47% ethanol was supplied via a nasogastric tube in four doses of 100 mL each, for a total of 400 mL. Following therapy, the ethylene glycol concentration was 0 mg/dl in the follow-up test.

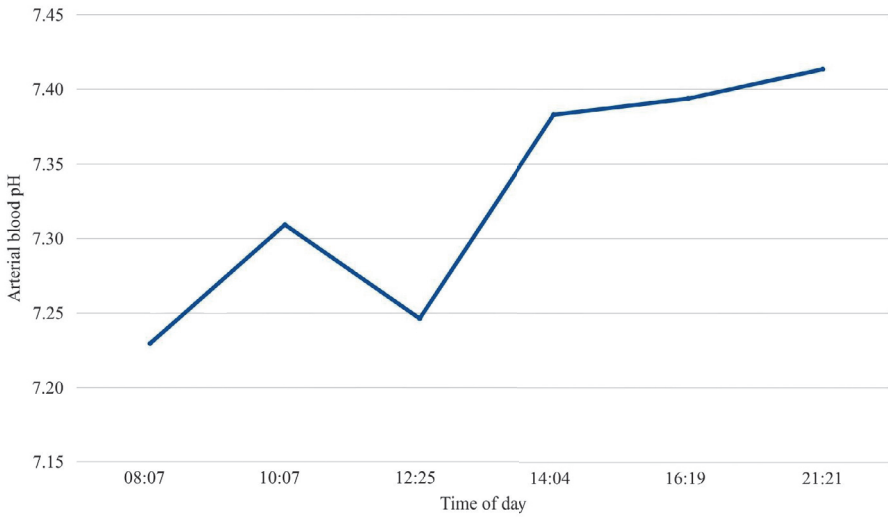


**Figure 1**
**Changes of pH on the 5th of March.**

The patient was qualified for dialysis. Even though the APTT was elevated, a low dose of enoxaparin (20 mg) was administered to prevent clotting in the extracorporeal circuit.

Intermittent hemodialysis (IHD), without ultrafiltration, was performed twice on the first day of hospitalization – once overnight and once during the day- after the patient was transported to the CCICU.

Mechanical ventilation and cardiovascular stabilization with a catecholamine infusion (norepinephrine) were administered. No pathogens were isolated in the urine culture; a blood culture was not taken.

The patient's condition gradually improved during the treatment. On March 6th, he was extubated. Torasemide was initiated to stimulate diuresis.

On March 7th, after undergoing IHD with an ultrafiltration volume (UF) of 1000 mL, he was transported to the Department of Internal Medicine, Nephrology, and Dialysis Therapy, where the diagnostic process was deepened. The patient was conscious, complying with simple commands upon basic contact, and intermittently disoriented. During the physical examination, the patient had a blood pressure of 140/70 mmHg, a heart rate of 75 beats per minute, and was breathing spontaneously through natural airways with oxygen supplementation of 6 l/min. Oxygen saturation was 95%. Furcations were audible bilaterally over the lung fields. The abdomen was tender on palpation, tense with slightly marked muscular defense, and without peritoneal signs. Attention was drawn to swelling of the trunk, and a loud systolic murmur was noted over the heart, with maximum intensity at Erb's point. Daily monitoring of renal parameters was initiated, and IHD was scheduled approximately every two days. In follow-up blood count performed on March 8th, the WBC count decreased nearly to normal values of 10.98 x 10^9/l, but the patient turned out to be anemic with a RBC of 2.96 x 10^12/l and hemoglobin of 8.9 g/dl. The treatment with iron was implemented.

Significant lung parenchymal deterioration was evident on the chest radiograph, accompanied by inflammatory opacities, suspected pleural effusion, and indications of possible congestive changes. Ventilator-associated pneumonia was suspected based on clinical and radiological findings. Empirical treatment with piperacillin - tazobactam was initiated, and oxygen therapy was continued. Antibiotic therapy was planned for 14 days. The patient required continued dialysis because of persistent

**Table 1**
**Patient's daily diuresis.**

| Date | 5th March | 6th March | 7th March | 8th March | 9th March | 10th March | 11th March | 12th March | 13th March | 14th March |
|---|---|---|---|---|---|---|---|---|---|---|
| Urine volume | 150ml | 180ml | 170ml | 100ml | 0ml | 0ml | 100ml | 0ml | 50ml | 100ml |

oliguria. Due to the lack of diuretic effect, the diuretics were discontinued on March 8th.

Before the administration of antibiotics, blood cultures were taken, revealing growth of *Actinomyces oris* with no microbial growth in the anaerobic culture. Possible contamination was suspected due to the sterility of other cultures. Infection with *Clostridium difficile* was ruled out. The patient's condition improved, but anuria persisted with the need to continue dialysis therapy. Daily diuresis is shown in Table 1. Dialysis access was replaced.

On March 14th, the urinary catheter was removed, and the patient was advised to conduct a 24-hour urine sample, but he did not comply. Diuresis-stimulating therapy was resumed, with torasemide 10 mg twice daily.

Despite antibiotic treatment, a slight cough and borderline respiratory distress syndrome (RDS) persisted. Given the positive QuantiFERON test and elevated erythrocyte sedimentation rate, it was decided to perform bronchoscopy to search for *Mycobacterium tuberculosis* complex colonies.

A bronchoscopy was performed on March 21st. A friable contact of bleeding mucosa was seen, especially in the bronchi and the middle lobe of the right lung, where a small amount of retained mucous secretion was apparent. No genetic material for *Mycobacterium tuberculosis* complex was found in the bronchoalveolar lavage.

Transthoracic echocardiography revealed a thickened aortic root wall measuring approximately 6 mm, with possible inflammatory infiltration, a tricuspid aortic valve showing small marginal lesions, and moderate regurgitation. However, these findings were not clearly visualized, prompting the decision to pursue further diagnostic evaluation. Transesophageal echocardiography was performed six days later. Aortic valve lobes were described as flaccid and segmentally creased, with visible echoes up to 4 mm long. The picture was described as consistent with infective endocarditis. Treatment with amikacin and ceftriaxone was initiated on March 25th. Given the kidney damage, gentamicin was excluded from the therapeutic regimen.

On March 25th, an otolaryngologist and stomatologist were consulted to look for the source of the infection. On clinical and radiographic examination, there were active foci of infection in teeth 14, 13, 22, 26, 28, 36, 37, 44, 45, 46, and 47. Over the next two weeks, all infected teeth were extracted.

On March 27th, according to the patient's account, urine output increased significantly. A 24-hour urine collection was resumed and the next day a diuresis of 1000 ml was obtained.

On April 3rd, due to the following results – serum potassium level of 5.7 mmol/l, serum urea concentration of 121 mg/dl, and serum creatinine concentration 10.5 mg/dl - the patient was classified for another session of IHD. Monitoring of renal parameters continued for the next seven days. Due to adequate improvement and no further indications for IHD, the patient's dialysis catheter was removed on April 10th.

After six weeks of antibiotic therapy, inflammatory parameters had decreased. Transesophageal echocardiography (TEE) was performed, showing no changes in the morphology of the previously observed abnormalities. Due to the findings in the imaging study, a thoracic surgery consultation was conducted, but the patient declined the surgical procedure. He was informed about the adverse effects of his condition, but he was released from the hospital at his request. He was given a prescription to continue his antimicrobial (amoxicillin-clavulanic acid 875 mg + 125 mg) and diuretic (torasemide 10 mg) therapy at an outpatient clinic, with a follow-up echocardiography recommended four weeks after discharge.

## Discussion

Ethylene glycol poisoning occurs worldwide and is clinically significant owing to its potential lethality and the wide range of ensuing complications. In the United States in 2022, poison control centers recorded 727 cases, including 11 deaths [2]. It is clinically significant due to the potential mortality and a vast number of health complications that may follow.

Ethylene glycol is a colorless liquid; nevertheless, the products in which ethylene is typically found are frequently colored to prevent inadvertent intake. Ethylene glycol is commonly used in brake fluids, radiator fluids, and solvents due to its ability to lower water's freezing point. It has a sweet flavor and is commonly ingested by alcohol addicts as a substitute for ethanol, occasionally for suicidal motives [1, 4, 5, 6]. Ethylene glycol, like other simple alcohols, is rapidly and completely absorbed after oral ingestion, with peak serum concentrations typically reached within an hour. In the liver, alcohol dehydrogenase metabolizes ethylene glycol to glycolaldehyde, which is subsequently oxidized to glycolic acid, glyoxylic acid, and finally, oxalic acid. While ethylene glycol produces intoxication, the accumulation of toxic metabolites is what causes the potentially deadly acidosis and kidney failure that characterize ethylene glycol poisoning [5, 7].

The early main symptoms of intoxication are typical symptoms of ethanol intoxication and are therefore not alarming to the patient's surroundings, especially if the poisoned person has a history of alcohol

dependence [5, 8]. Such a situation occurred in the case of the presented patient. There are three stages of ethylene glycol toxicity. The list of them with descriptions is shown in Table 2.

However, patients may not exhibit all of them. The absence of the distinctive ethanol odor is a useful diagnostic clue.

One research paper implies laboratory criteria that can be used for the presumptive diagnosis of ethylene glycol poisoning as follows: a history or suspicion of ingesting ethylene glycol plus any 2 of the following: arterial pH < 7.3, serum bicarbonate of < 20 mEq/L, osmolal gap > 10 mOsm/L, presence of urinary oxalate crystals or a history or suspicion of ethylene glycol ingesting within the last 1 hour [5].

In the presented case, treatment for alcohol intoxication was initiated based on strong clinical suspicion. Subsequently, laboratory results confirming the clinical diagnosis were obtained [14]. Our patient had met the criteria. There was a high probability of poisoning; the arterial pH was 6.5 at admission; the calculated osmolal gap was 289.405 mmol/l; and the serum bicarbonate level was 13.1 mmol/l; therefore, the treatment was administered.

Ethanol acts as a competitive substrate for alcohol dehydrogenase (ADH), preventing the development of toxic metabolites and allowing for renal excretion of non-toxic ethylene glycol. ADH has a higher affinity for ethanol than ethylene glycol, approximately 65-fold. Serum ethanol concentrations range from 21.7 mmol/L to 32.6 mmol/L (100–150 mg/dL) for competitive suppression of ethylene glycol metabolism, according to several sources. When the quantity of ethylene glycol is excessively high, the described dosage cannot be sufficient.

Instead of a single, arbitrary value of 100–150 mg/dL, the ideal antidotal ethanol dose is at least one-fourth to one-third of the serum ethylene glycol concentration [3]. The loading dosage is 10 mL/kg of a 10% ethanol infusion over 60 minutes to boost serum ethanol concentration to the desired level. The actual maintenance dosage needs range from 0.8 mL/kg per hour to 2 mL/kg per hour (and even more during hemodialysis), with the serum concentration of the antidote tested every 1 or 2 hours. Once serial measurements show steady ethanol serum levels, the frequency of measurements can be reduced to every 2 to 4 hours [3]. The continuation of maintenance infusions is needed until serum ethylene glycol content is undetectable (<3.2 mmol/L), the patient is symptom-free, and pH is normal. Depending on the quantity of consumption, toxicity, and usage of hemodialysis, an ethanol infusion may be necessary for two or more days [3].

The treatment dosage of ethanol can also be administered through the gastrointestinal tract, orally or through a nasogastric tube. Although ethanol treatment as an antidote to ethylene glycol poisoning is inexpensive and generally available, it does have certain adverse effects. Individually variable metabolic capabilities of ethanol, the possibility of causing inebriation and central nervous system depression, and frequent monitoring of serum concentrations are required to maintain a target level [3, 4, 5, 8].

Fomepizole (4-methylpyrazole) is the second-known antidote for ethylene glycol poisoning. Acting as a potent competitive inhibitor of ADH, it blocks the formation of toxic metabolites. Fomepizole dosing is straightforward, and clinically significant side effects are rare. The primary downside is the excessive price.

**Table 2**
**The characteristics of stages of ethylene glycol poisoning.**

| Stages | Main symptoms | Remarks |
|---|---|---|
| Neurological (0.5–12 hours) | Early symptoms may include slurred speech, ataxia, and somnolence. Gastric irritation may result in nausea and vomiting. As ethylene glycol metabolism progresses and metabolic acidosis occurs, depression in the central nervous system (CNS) becomes more severe [4, 5, 9]. | Other neurological symptoms include ataxia, myoclonus, nystagmus, cerebral edema, ophthalmoplegia, and seizures. Rarely, delayed cranial nerve neuropathy, particularly affecting the facial nerves, might occur 5–20 days after intake [10]. |
| Cardiopulmonary (12–36 hours) | The second phase is generated by the accumulation of organic acids, which are metabolites of ethylene glycol. Common symptoms include tachycardia, hypertension, metabolic acidosis with compensatory tachypnea, and Kussmaul's respiration [4, 5, 9]. Acidemia adversely affects heart contractility, leading to hypotension and myocardial impairment. Pulmonary edema can be caused by either heart failure or direct poisoning, resulting in acute respiratory distress syndrome. The majority of untreated patients die during this time [4, 5, 6]. | Severe hypocalcemia can result in hyperreflexia, muscular spasms, and QT interval prolongation. |
| Renal (24–72 hours) | Acute kidney injury caused by ethylene glycol intoxication leads to renal tubular necrosis, hemorrhagia, proteinuria, flank pain, oliguria, anuria, and kidney failure [4, 5, 6, 9]. Calcium oxalate crystals accumulate in the renal tubular lumen, causing obstruction and loss of glomerular filtration. Monohydrate crystals are building up in proximal tubular cells and can cause direct cellular damage [12]. | Kidney failure is usually reversible but may require weeks or months of hemodialysis [13]. Occasionally, bone marrow suppression can cause pancytopenia and leukocytosis [5]. |

At our hospital's ED, fomepizole was not available during the patient's admission; therefore, ethanol was implemented [3, 5, 8].

Hemodialysis is described in the protocol for the treatment of alcohol poisoning proposed by American Association of Clinical Toxicology (AACT). The criteria for implying the procedure comprise: significant metabolic acidosis (pH less than 7.25 to 7.30), kidney failure, or electrolyte imbalances that are unresponsive to conventional therapy [14].

Due to severe acidosis (pH 6.5), hyperkalemia (5.7 mmol/L), and the absence of diuresis following catheterization the patient was qualified to hemodialysis treatment, which was maintained for 40 days until the patient's kidney function improved.

In the case of kidney failure in ethylene glycol poisoning patients, dialysis is the only technique to remove both toxic acid metabolites and the parent alcohol [3, 4]. Hemodialysis also corrects the present acidemia. Traditionally, hemodialysis is performed until ethylene glycol and glycolic acid levels are undetectable and until blood pH is correct. Cofactor therapy can enhance antidotal treatment. In the management of ethylene glycol poisoning, thiamine is administered to prevent the formation of oxalic acid by converting glyoxylic acid into α-hydroxy-β-ketoadipic acid, a non-toxic metabolite [5, 7]. Two conventional regimens were described: one with 100 mg thiamine given intravenously every 6 hours until ethylene glycol is no longer detectable in serum [15], and another, with a daily single dose of 100 mg intravenously [3]. Pyridoxine prevents the development of oxalic acid by converting glyoxylic acid to glycine and hippuric acid, which are innocuous. However, the favorable impact of pyridoxine on ethylene glycol poisoning is questionable [3, 4, 5, 16]. In this situation, two dosing strategies are provided. Pyridoxine is often administered intravenously at a dosage of 50 mg every 6 hours. Taking too much pyridoxine in a short period of time might cause toxic sensory-peripheral neuropathy [17]. Therefore, treatment should not last more than 24 hours [5].

The solution of sodium bicarbonate within the ED was administered. Intravenous sodium bicarbonate is recommended to correct metabolic acidosis, eliminate kidney glycolic acid, and prevent calcium oxalate crystal formation. A bolus of 1 to 2 mEq/kg should be administered initially. Infusion rates of 150–250 mL/hour are often sufficient and are supposed to be maintained until an arterial or venous pH rises to a value of 7.35. If acidosis is treated solely with bicarbonate, organic acids will still be generated. Treatment with sodium bicarbonate may initially aggravate hypocalcemia due to calcium-protein binding. The infusion can be stopped when the pH returns to normal and no more acid generation is predicted [3, 7].

Our patient was a chronic alcohol consumer, and in this population, intestinal absorption of sodium and water was observed to be lower compared to the control group. Hence, dehydration may play an important role in the development of acute kidney injury (AKI) in our patient's situation. Another clue to suspect heavy dehydration was a WBC count of 51.99 x 10^9/l, which normalized to 10.35 x 10^9/l within 5 days after fluid treatment [20, 21].

Due to the alcohol history of the patient and the poor results in red blood cell parameters, iron therapy was implemented [22].

Oxalic acid crystals can form not only in kidney tissue, causing AKI, but also in other tissues, including the brain and heart, resulting in multi-organ failure [4, 5]. Seizures and myoclonic jerks have been linked to the harmful effects of oxalic acid crystals, as well as hypocalcemia caused by calcium oxalate crystal precipitation [19]. Initial treatment for seizures caused by ethylene glycol poisoning should be treated with benzodiazepines. In our patient's situation, 1 mg of intravenous clonazepam was provided to halt the seizures at the ED. Barbiturates are a suitable second-line treatment. Other anticonvulsants can be added as needed [5].

Ethylene glycol poisoning and its consequences, such as acidosis and hypocalcemia, are thought to be responsible for cardiotoxicity, which causes arrhythmia, hypertension, and myocardial depression [4, 5, 19]. At admission, the patient had a blood pressure of 174/89 mmHg, and an ECG revealed atrial fibrillation. Following causative ethanol therapy, cardiovascular symptoms improved throughout follow-up.

## Conclusions

Patients with ethylene glycol poisoning have to be diagnosed right away, as the prognosis is poor if left untreated. As in the presented case, management should begin with securing the patient's airway, respiration, and circulation while suppressing any further symptoms, such as seizures. The patient should be treated with bicarbonate to reduce acidemia, alcohol dehydrogenase inhibitors to suppress the formation of toxic metabolites, and extracorporeal removal methods, such as hemodialysis, to enhance the elimination of the parent alcohol and its metabolites, either after diagnosis is confirmed or based on high clinical suspicion. Since folic acid, thiamine, and pyridoxine optimize metabolic pathways for the removal of harmful acid metabolites, cofactor therapy should also be used. Our patient was promptly diagnosed, allowing for appropriate treatment and a full recovery. However, despite the discovery of a suspicious aortic valve lesion during his hospitalization, he declined thoracic surgery.

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## List of abbreviations

1. Emergency Department (ED)
2. Glasgow Coma Scale (GCS)
3. Electrocardiogram (ECG)
4. White Blood Cells (WBC)
5. Mean Corpuscular Volume (MCV)
6. Mean Corpuscular Hemoglobin Concentration (MCHC)
7. Platelets (PLT)
8. Computer tomography (CT)
9. Clinical Cardiac Intensive Care Unit (CCICU)
10. Activated Partial Thromboplastin Time (APTT)
11. Intermittent Dialysis (IHD)
12. Ultrafiltration Volume (UF)
13. Red Blood Cells (RBC)
14. Systolic Blood Pressure (SBP)
15. Respiratory Distress Syndrome (RDS)
16. Transesophageal Echocardiography (TEE)
17. Central Nervous System (CNS)
18. Alcohol Dehydrogenase (ADH)
19. American Association of Clinical Toxicology (AACT)
20. Acute Kidney Injury (AKI)