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CASE REPORT

Antifreeze Poisoning Complicated by Severe Metabolic Acidosis, Toxic Encephalopathy, and Multiple Organ Dysfunction Syndrome: A Case Report

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Abstract: A 20-year-old female patient ingested approximately 1200 mL of antifreeze and was found comatose five hours later, after which she was transported to the hospital. On admission, she was in deep coma with respiratory failure and severe metabolic acidosis (pH 6.82). Laboratory findings revealed a high anion gap, elevated lactate levels, and acute kidney injury. Chest computed tomography (CT) showed aspiration pneumonia. Based on clinical history and examination, a diagnosis of ethylene glycol poisoning complicated by toxic encephalopathy and multiple organ dysfunction syndrome (MODS) was established. The patient received gastric lavage, ethanol antidote therapy, combined hemoperfusion and continuous renal replacement therapy (CRRT), as well as multi-organ supportive treatment. Mechanical ventilation was withdrawn on day 4, she was transferred to a general ward on day 6, and discharged on day 28 after recovery of renal function. This case highlights that early recognition of ethylene glycol poisoning, together with combined detoxification, blood purification, and organ support therapy, is essential for improving patient outcomes.

Keywords: antifreeze poisoning, ethylene glycol, toxic encephalopathy, metabolic acidosis, multiple organ dysfunction syndrome, sepsis

Introduction

The main chemical component of antifreeze is ethylene glycol (EG), a colorless, transparent, viscous liquid with low volatility, whose chemical structure is similar to that of ethanol.[1] Ethylene glycol is readily soluble in water and has a slightly sweet taste. It is often dyed pink or blue-green, resembling beverages in appearance, which contributes to occasional accidental ingestion.[2,3] If acute ethylene glycol poisoning is not promptly diagnosed and treated, after entering the human body, it is absorbed into the bloodstream through the stomach and intestines and metabolized by hepatic alcohol dehydrogenase into glycolaldehyde, which is further metabolized into glyoxal, glycolic acid, and oxalic acid. Oxalate subsequently binds with calcium to form calcium oxalate, which readily obstructs renal tubules, thereby resulting in severe metabolic acidosis, acute renal failure, central nervous system dysfunction, and even death4. The core value of this case lies in the management of a critically ill patient with massive ethylene glycol poisoning complicated by severe metabolic acidosis, toxic encephalopathy, and multiple organ dysfunction. Under resource-limited conditions where toxin concentration monitoring was unavailable and fomepizole could not be administered, an innovatively optimized and integrated treatment strategy was implemented and resulted in a favorable prognosis, providing a reproducible practical reference for clinical management.

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Case Presentation

The patient, a 20-year-old female college student, had no prior history of significant illness, no smoking or alcohol habits, and denied any psychiatric history. Due to academic stress, she ingested approximately 1200 mL of "antifreeze" (as reported by the family based on the antifreeze container found at the scene and the presence of vomitus). Five hours later, she was found by her family unconscious and unresponsive, with pink frothy sputum at the mouth, urinary and fecal incontinence, the patient was admitted to the emergency department of our hospital following prehospital emergency care. The family denied any history of drug or food allergies and had no history of long-term medication use. On admission, she was comatose with a Glasgow Coma Scale (GCS) score of E1V1M1. Both pupils were equal and round, 3.0 mm in diameter, with sluggish light reflexes. Her body temperature was 34.5°C, pulse 115 beats/min, respiratory rate 28 breaths/min (irregular, deep and labored breathing), fingertip oxygen saturation 89% (on oxygen via reservoir mask, FiO_2 100%), and blood pressure 135/90 mmHg. Auscultation revealed coarse breath sounds in both lungs with scattered moist rales; the abdomen was flat and soft with hypoactive bowel sounds. The heart rhythm was regular, and no murmurs were heard over any cardiac valve areas. Abdominal examination was limited due to poor cooperation; tenderness and rebound tenderness could not be clearly assessed. The liver and spleen were not palpable below the costal margin. No edema was observed in the lower limbs. Physiological reflexes were present, pathological reflexes were absent, and intermittent convulsions were noted. The extremities were cold and clammy, with visible vomitus around the mouth and face.

To monitor disease progression and treatment efficacy, serial arterial blood gas analyses were performed, with results shown in Table 1. Additional laboratory findings obtained on admission are presented in Table 2. Complete blood count showed significantly elevated white blood cell and neutrophil counts, along with a markedly increased procalcitonin level. Among the cardiac injury markers, creatine kinase-MB was markedly elevated. Blood ammonia and blood urea nitrogen levels were increased, and serum creatinine rose to 431 μmol/L on day 2 after admission. Arterial blood gas analysis demonstrated severe high–anion gap metabolic acidosis (pH 6.82), with markedly elevated lactate levels and substantially reduced bicarbonate and base excess. Coagulation parameters were mildly abnormal, while blood glucose and D-dimer levels were markedly elevated. Urinalysis revealed positive occult blood and protein, increased microalbumin and red blood cells in the sediment, and the presence of numerous needle-shaped calcium oxalate crystals. Imaging findings: Chest CT on admission showed bilateral lower-lobe infiltrates suggestive of aspiration pneumonia (Figure 1A). On day 18, repeat CT demonstrated progression of pulmonary lesions with increased bilateral lower-lobe infiltrates (Figure 1B) and a small amount of pleural effusion (Figure 2A), along with bilateral pleural effusions, partial atelectasis of both lower lobes, and interstitial pulmonary edema (Figure 2B).

Table 1 Dynamic Changes in Arterial Blood Gas

Parameter	February 28 (After Admission to the Department)	February 28 (Re-examination after 4 Hours)	March 1st	March 2nd	March 3rd	March 5th
PH	6.82	7.21	7.45	7.35	7.34	7.39
PO_2 (mmHg)	184.18	199.69	126.35	169.3	134.64	182.69
PCO_2 (mmHg)	10.91	35.53	45.86	51.06	46.01	44.75
SaO_2 (%)	99.35	99.95	99.58	100	99.68	100
Lac (mmol/L)	9.36	9.43	8.06	2.32	1.57	1.17
HCO_3^- (mmol/L)	1.8	14	31.2	27.4	24.30	26.6
BE (mmol/L)	−31.47	−12.83	6.37	1.15	−1.57	1.44
AG (mmol/L)	37.3	28.3	17	6	7.9	4.6

Abbreviations: PH, potential of hydrogen; PO_2, partial pressure of oxygen; PCO_2, partial pressure of carbon dioxide; SaO_2, saturation of arterial oxygen; Lac, lactate; HCO_3^-, bicarbonate Ion; BE, base excess; AG, anion gap.

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Table 2 Laboratory Findings After Admission

Test Category	Parameter	Value (Unit)	Reference Range
Complete blood count	White Blood Cell (WBC)	56.25×10^9/L	$3.5\sim9.5\times10^9$/L
	Red Blood Cell (RBC)	4.89×10^{12}/L	$3.8\sim5.1\times10^{12}$/L (Female)
	Hemoglobin (Hb)	152g/L	115~150g/L (Female)
	Platelet (PLT)	365×10^9/L	$100\sim300\times10^9$/L
	Neutrophil Count (ANC)	46.35×10^9/L	$1.8\sim6.3\times10^9$/L
	C-Reactive Protein (CRP)	9.29mg/L	0~10mg/L
	Procalcitonin (PCT)	39.69μg/L	0~0.15μg/L
Cardiac injury markers	Creatine Kinase-MB (CK-MB)	1219IU/L	0~25IU/L
	Cardiac Troponin I (cTnI)	<0.010μg/L	0~0.04μg/L
	N-terminal Pro-B-type Natriuretic Peptide (NT-proBNP)	67ng/L	0~300ng/L
Liver and renal function	Alanine Aminotransferase (ALT)	12U/L	7~40U/L
	Aspartate Aminotransferase (AST)	29U/L	13~35U/L
	Total Bilirubin (TBIL)	4.3μmol/L	3.4~17.1μmol/L
	Direct Bilirubin (DBIL)	1.4μmol/L	0~6.8μmol/L
	Blood Ammonia (NH_3)	206μmol/L	18~72μmol/L
	Creatinine (Cr)	83μmol/L	44~97μmol/L (Female)
	Blood Urea Nitrogen (BUN)	8.32mmol/L	3.2~7.1mmol/L
Coagulation and Others	Prothrombin Time (PT)	17.4sec	11~13.7sec
	Activated Partial Thromboplastin Time (APTT)	43.7sec	25~37sec
	Blood Glucose (GLU)	25mmol/L	3.9~6.1mmol/L
	D-Dimer	2980μg/L	0~500μg/L
	Serum Calcium (Ca)	1.98mmol/L	2.11~2.52mmol/L
Urinalysis	Occult Blood	3+	Negative
	Urine Protein	3+	Negative
	Microalbumin	>150mg/L	<30mg/L
	Sediment Red Blood Cell	452.1cells/μL	0~17cells/μL
	Special Finding	A large number of needle-shaped crystals (calcium oxalate crystals)	No crystals

Detoxification therapy: The patient had a confirmed history of antifreeze ingestion, whose main chemical component is ethylene glycol; therefore, the detoxification plan was formulated as follows: ①gastric lavage with clean water; ②Ethanol was continuously administered via gastrointestinal infusion at a rate of 30 mL/h, with no supplementation through other routes. Owing to limitations in medical resources, serial monitoring of serum ethanol concentrations was not performed; therefore, the infusion regimen was dynamically adjusted based on changes in the patient's level of consciousness, arterial blood gas parameters, and recovery of renal function to ensure effective antidotal therapy while minimizing the risk of overdosage. Due to these constraints, toxin concentration measurements and intravenous administration of fomepizole were not available. On day 2, arterial blood gas analysis showed that pH had normalized;

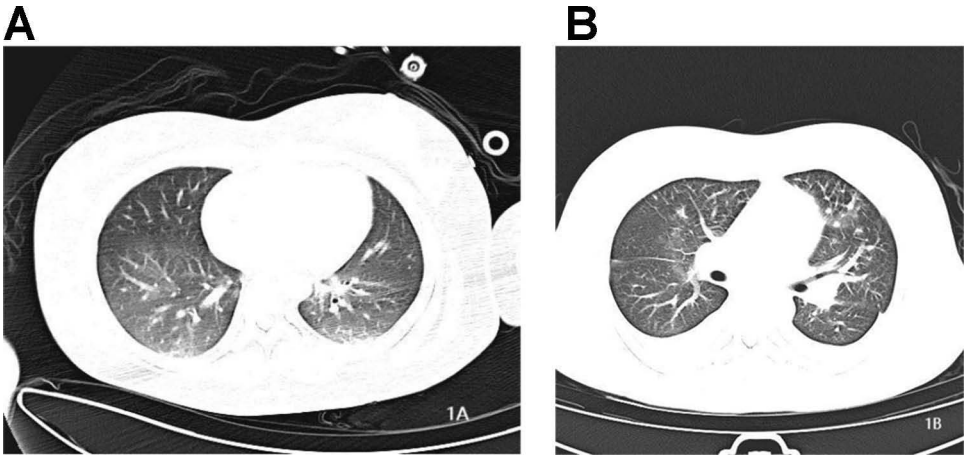

Figure 1 (**A**) Chest CT (lung window) on hospital day 2 shows bilateral lower-lung exudation. (**B**) On day 18, bilateral lower-lung exudation is increased.

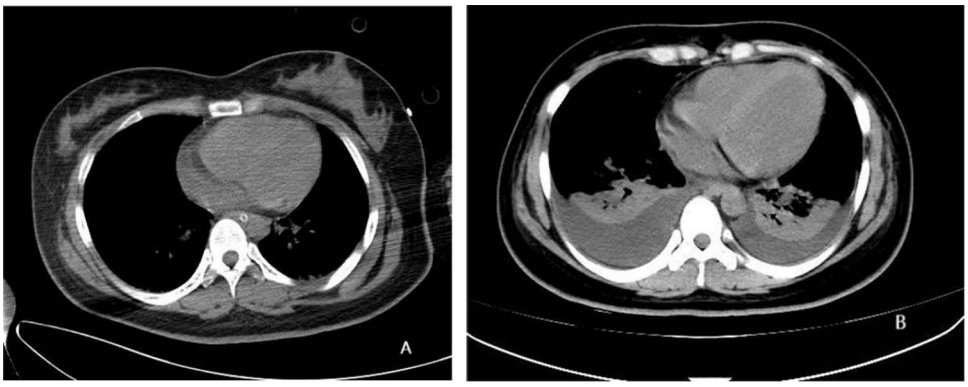

Figure 2 (**A**) Chest CT (mediastinal window) on hospital day 2 reveals a small amount of bilateral pleural effusion. (**B**) On day 18, bilateral pleural effusion has increased, accompanied by interstitial pulmonary edema in both lungs.

however, given the role of CRRT in correcting acid–base disturbances, ethanol therapy was continued to achieve effective inhibition of ethylene glycol metabolism, ethanol infusion was continued for detoxification until day 4, when treatment was discontinued; ③hemoperfusion with HA330 cartridges was performed twice at 12-hour intervals; and ④gastrointestinal catharsis with 250 mL of mannitol administered via gastric tube three times daily.

Correction of metabolic disorders: ①Fluid resuscitation: The patient's blood pressure was normal (135/90 mmHg), but the extremities were cold and clammy, with a lactate (Lac) level of 9.36 mmol/L and an anion gap (AG) of 37.3 mmol/L, indicating inadequate tissue perfusion. Immediate volume resuscitation was performed using 500 mL of compound sodium chloride injection combined with 500 mL of dextran administered intravenously. ②Alkali supplementation: 250 mL of 5% sodium bicarbonate injection ($NaHCO_3$) was infused intravenously. ③Blood purification: After admission, the patient was treated with continuous renal replacement therapy (CRRT) in the continuous veno-venous hemodiafiltration (CVVHDF) mode, with a blood flow rate of 150 mL/min. Both the replacement fluid and dialysate were administered at 1500 mL/h. Systemic anticoagulation with heparin was initiated with an intravenous loading dose of 1000 U, followed by a continuous intravenous infusion at 5–10 U/kg/h, maintaining the activated partial thromboplastin time at 70–85 s. ④At admission, the serum calcium level was 1.98 mmol/L (below the laboratory reference range of 2.11–2.52 mmol/L). Intravenous calcium supplementation was administered to alleviate convulsive symptoms and to prevent myocardial injury. By the second day of hospitalization, the patient's anion gap (AG) normalized (15.0 mmol/L), and by the third day, the lactate (Lac) decreased to normal (2.32 mmol/L).

Organ protection and management of complications: (1) Acute kidney injury: Upon admission, urinalysis revealed positive occult blood, and needle-like crystalline deposits were visibly present within the urinary catheter. Serum creatinine

was 83.0 μmol/L and blood urea nitrogen was 8.32 mmol/L; Considering the potential nephrotoxicity of antifreeze,[1] continuous renal replacement therapy (CRRT) and urinary alkalization with 5% $NaHCO_3$ were initiated on the day of admission; On day 6 of hospitalization, at the time of transfer, the patient's 24-hour urine output was less than 50 mL, with serum creatinine at 431 μmol/L and blood urea nitrogen at 11.72 mmol/L, indicating persistent renal failure; intermittent hemodialysis was continued; By day 21, the patient's 24-hour urine output exceeded 500 mL, with both serum creatinine and blood urea nitrogen showing a downward trend, and hemodialysis was temporarily discontinued; By day 28, serum creatinine had decreased to 138 μmol/L, and blood urea nitrogen had returned to normal levels. (2) Toxic encephalopathy: The patient presented with impaired consciousness, with a Glasgow Coma Scale (GCS) score of E1V1M1. Both pupils were equal and round, with a diameter of 3.0 mm, and sluggish pupillary light reflexes; Respiration was irregular (deep and labored breathing), and the peripheral oxygen saturation (SpO_2) was 89%; Anion gap (AG) and osmolar gap (OG) were elevated; Generalized convulsions were observed, Midazolam combined with calcium gluconate was administered via continuous intravenous infusion to control the convulsions. Invasive mechanical ventilation was initiated to improve oxygenation and maintain SpO_2 above 95%.On day 2 of hospitalization, the patient's AG returned to the normal range, the Richmond Agitation-Sedation Scale (RASS) score was −3, and under volume-controlled ventilation, the respiratory rate was 20 breaths/min (with 8 spontaneous breaths), and the P/F ratio was 315.88 mmHg; By day 4, the patient regained full consciousness with a GCS score of E3VTM5; both pupils were equal and round (3.0 mm in diameter) with brisk light reflexes, and no limb convulsions were observed; The osmolar gap had decreased to the normal range (6.73 mOsm/L). The spontaneous breathing trial was positive, with a P/F ratio of 384.69 mmHg and SpO_2 of 95%, allowing successful weaning from mechanical ventilation and extubation. (3) Aspiration pneumonia: ①Airway protection: The patient had impaired consciousness and was unable to clear airway secretions effectively. Auscultation revealed scattered moist rales in both lungs. Endotracheal intubation and bronchoalveolar lavage via fiberoptic bronchoscopy were performed. Under the bronchoscope, a small amount of pinkish, thin fluid was observed in the main airway, and mild mucosal edema was noted in both main bronchi. ②Respiratory support: Invasive mechanical ventilation was administered (assist/control mode, tidal volume 0.42 L/min, PEEP 5 cmH_2O, FiO_2 40%), along with effective sputum drainage (intermittent suctioning and postural drainage). On day 4 of the course, the patient was successfully weaned from the ventilator and extubated, followed by high-flow oxygen therapy (FiO_2 35%), maintaining a peripheral oxygen saturation (SpO_2) above 95%. ③Pharmacological therapy: On admission, the patient's white blood cell count, neutrophil count, and neutrophil percentage were all elevated. Based on clinical history, bacterial infection was considered the primary cause. According to *The Sanford Guide to Antimicrobial Therapy (2024 Edition)*, piperacillin–tazobactam (4.5 g every 8 hours) was administered to cover Gram-negative (G−) pathogens. Intravenous ambroxol hydrochloride (90 mg once daily) and nebulized acetylcysteine (0.3 g) plus budesonide (1 mg every 12 hours) were also given. On day 4, sputum culture revealed *Staphylococcus aureus* infection; intravenous linezolid (600 mg every 12 hours) was added based on antimicrobial susceptibility results; On day 12, laboratory tests showed a white blood cell count of 10.76×10^9/L, neutrophil count of 8.28×10^9/L, C-reactive protein (CRP) level of 10.37 mg/L, and procalcitonin (PCT) level of 1.17 μg/L, indicating overall improvement in inflammatory markers. However, the patient continued to experience exertional dyspnea, high oxygen dependency (high-flow oxygen at 35 L/min), and SpO_2 of 91%, suggesting incomplete control of pulmonary infection, as piperacillin–tazobactam had been used for 12 days, a step-down antibiotic regimen was implemented: piperacillin–tazobactam 4.5 g every 12 hours plus linezolid 600 mg every 12 hours. Concurrently, intravenous ulinastatin (10 WU once daily) was administered for anti-inflammatory therapy. On day 18, follow-up chest CT revealed increased bilateral pleural effusion (Figure 2B), and bilateral closed thoracic drainage was performed. On day 28, the white blood cell count was 9.47×10^9/L, neutrophil count 6.18×10^9/L, CRP 6.85 mg/L, and PCT 1.17 μg/L. The patient received nasal cannula oxygen (SpO_2 95%) and antibiotic therapy was discontinued.

Through the aforementioned treatment, the patient's metabolic acidosis was promptly corrected, and both pulmonary infection and renal injury were effectively controlled. On the fourth day of hospitalization, the patient was successfully weaned from mechanical ventilation and extubated. On the sixth day, the patient was transferred to the Department of Infectious Diseases of our hospital for further treatment. At the follow-up on day 11, the patient had an Activities of Daily Living (ADL) score of 55, experienced shortness of breath after activity, received high-flow oxygen therapy (FiO_2 35%), showed a peripheral oxygen saturation of 91%, had a 24-hour urine output of approximately 100 mL, and underwent intermittent hemodialysis. At the follow-up on day 28, the patient's urine output had increased to approximately 1500 mL,

renal function showed marked improvement, and dialysis treatment had been discontinued. On day 33, the patient's condition had improved, and they were discharged from the hospital.

Discussion

The most authoritative reference on the treatment of ethylene glycol poisoning is the *Practice Guidelines on the Treatment of Ethylene Glycol Poisoning*,[4] issued by the American Academy of Clinical Toxicology (AACT). The guideline primarily recommends a comprehensive treatment approach, including early administration of ethanol as an antidote, extracorporeal blood purification to remove the toxin, correction of acid–base imbalance, and symptomatic supportive care.

Ethanol Detoxification

The main component of antifreeze is typically ethylene glycol. After oral ingestion, ethylene glycol reaches its peak plasma concentration approximately 2–4 hours post-ingestion. Its toxicity primarily arises from hepatic metabolism catalyzed by alcohol dehydrogenase (ADH), through which ethylene glycol is gradually oxidized into toxic metabolites such as oxalic acid, resulting in renal injury and metabolic acidosis.[5] At this stage, reducing the concentrations of ethylene glycol and its metabolites (glycolic acid and oxalic acid) in the body is of critical importance. Ethanol competitively binds to the active site of ADH, thereby preventing ethylene glycol from being further metabolized into toxic products, allowing the less toxic parent compound to be excreted unchanged via the kidneys;[6] Although fomepizole is currently recommended as the first-line antidote, ethanol remains widely used for the treatment of ethylene glycol poisoning when fomepizole is unavailable because of its low cost and ease of use. Early and adequate administration of ethanol as an antidote can effectively block the metabolism of ethylene glycol and prevent the formation of its toxic metabolites. Since ethanol itself exerts a depressant effect on the central nervous system, it may interfere with the patient's level of consciousness and judgment; therefore, the infusion rate of ethanol should be carefully adjusted and controlled according to continuous monitoring of its serum concentration.

Combined Hemoperfusion and Hemodialysis for "Dual Clearance" of Toxins and Metabolites

In this case, an innovative "dual-elimination" strategy combining HA330 hemoperfusion with CRRT (CVVHDF mode) was applied, which laid a critical foundation for the reversal of the patient's condition. Hemoperfusion, using the HA330 cartridge with neutral macroporous adsorption resin, enables nonspecific adsorption of ethylene glycol and its metabolites (glycolic acid and oxalic acid), with particular effectiveness in removing middle-molecular-weight metabolites and those with higher protein-binding affinity8, thereby rapidly reducing circulating toxin concentrations and interrupting the toxic cascade9. CRRT, through diffusion and convection mechanisms, efficiently eliminates ethylene glycol and acidic metabolites, achieving clearance rates far exceeding those of natural renal excretion, thereby reducing circulating free oxalate before calcium oxalate crystal deposition occurs and preventing irreversible tissue injury; at the same time, it corrects metabolic acidosis, improves tissue perfusion, and substitutes for impaired renal function, thereby simultaneously addressing the three core problems of toxin accumulation, acid–base imbalance, and organ injury, and acting synergistically and complementarily with hemoperfusion. This combined modality provides comprehensive coverage for toxins with different physicochemical properties and significantly enhances treatment efficiency, and is particularly suitable for resource-limited settings where toxin concentration monitoring is unavailable and fomepizole cannot be used, thus offering a reproducible therapeutic pathway for critically ill patients with severe ethylene glycol poisoning.

Rapid Correction of Metabolic Acidosis

Metabolic acidosis is a severe complication of antifreeze poisoning. In this case, arterial blood gas analysis indicated severe anion gap (AG)-increased metabolic acidosis. The pathogenesis is associated with the accumulation of acidic metabolites derived from ethylene glycol metabolism and increased lactate production due to tissue hypoxia.[7] Treatment included volume resuscitation, sodium bicarbonate therapy, and continuous renal replacement therapy (CRRT), which

effectively corrected the acidosis and normalized the patient's lactate and anion gap levels. Upon admission, the patient had normal blood pressure (135/90 mmHg) but tachycardia (115 beats/min), and arterial blood gas results indicated severe lactic acidosis, providing clear evidence of tissue hypoperfusion. Therefore, we performed volume resuscitation with a rapid infusion of 500 mL normal saline within 10 minutes. The patient's heart rate and blood pressure responded, and bedside ultrasonography showed an inferior vena cava (IVC) diameter of 12 mm with an IVC collapsibility index of 30%, indicating effective resuscitation. Volume resuscitation corrects metabolic acidosis by replenishing effective circulating volume, improving tissue perfusion and oxygen delivery, reducing the production of acidic metabolites, and promoting the clearance of acidic substances; In addition, rapid fluid infusion can transiently mitigate acidosis by diluting the plasma concentration of H^+ ions and acidic metabolites. Administration of sodium bicarbonate ($NaHCO_3$) primarily corrects metabolic acidosis by supplying exogenous bicarbonate ions (HCO_3^-) that directly neutralize excessive acids within the body.[8] The patient weighed 65 kg, and the initial arterial blood gas analysis showed an actual HCO_3^- level of 1.8 mmol/L. To avoid over-alkalinization during correction, we set the target HCO_3^- level at 13 mmol/L, based on the formula, 291 mmol of $NaHCO_3$ (approximately 485 mL of 5% $NaHCO_3$ solution) was required for correction, Following the half-dose rule, we initially administered 250 mL of 5% $NaHCO_3$ intravenously. Two hours later, repeat blood gas analysis showed a pH of 7.21 and actual HCO_3^- of 15 mmol/L, after which $NaHCO_3$ infusion was discontinued, Acid–base balance was subsequently maintained through blood purification therapy.[9]

Protection and Improvement of Organ Function

Toxic encephalopathy, acute renal failure, and aspiration pneumonia are common complications of antifreeze poisoning. Toxic encephalopathy is primarily associated with the toxic effects of ethylene glycol metabolites, disturbances in acid–base balance, osmotic imbalance, and neuronal injury.[10] Glycolic acid, a metabolite of ethylene glycol, can be further metabolized into glyoxylic acid and oxalic acid, among which glycolic acid is the principal contributor to high–anion gap metabolic acidosis. Oxalic acid can induce increased neuromuscular excitability and cerebral vascular smooth muscle contraction through the formation of calcium oxalate crystals, with the main clinical manifestations including convulsions, muscle twitching, somnolence, and coma.[11] In contrast, elevated lactate levels observed in ethylene glycol poisoning are primarily related to inadequate tissue perfusion and the inhibitory effects of toxic metabolites on cellular metabolism, rather than being a direct metabolic product of ethylene glycol itself. In addition, metabolic acidosis and an elevated osmolar gap (OG) can further inhibit neuronal function, causing cerebral vasodilation and brain edema. Therefore, the pathogenesis of ethylene glycol–induced toxic encephalopathy lies in the synergistic toxicity of its metabolites, and early recognition with timely interruption of the metabolic cascade is crucial to saving life and preserving neurological function.

Both ethylene glycol and its metabolites are small, water-soluble molecules, and their clearance through hemodialysis is significantly higher than that achieved by renal excretion; Before calcium oxalate crystals are deposited, hemodialysis effectively reduces circulating free oxalate, prevents its deposition in organs such as the kidneys and myocardium, and contributes to the correction of acidosis and maintenance of internal homeostasis. The American Academy of Clinical Toxicology (AACT) and the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT) have clearly recommended hemodialysis as the preferred blood purification method for ethylene glycol poisoning.[9] In this case, the patient was treated promptly with continuous renal replacement therapy (CRRT) and urine alkalinization after admission. Although renal impairment persisted for a prolonged period, the patient ultimately recovered after treatment. Therefore, CRRT plays an important therapeutic role in treating ethylene glycol–induced renal injury. The patient had a confirmed history of accidental antifreeze aspiration, and chest CT on admission revealed bilateral pulmonary infiltrates, According to the *Guidelines for Aspiration Pneumonia*,[12] tracheal intubation and invasive mechanical ventilation were performed upon admission to correct hypoxemia, Bronchoscopic alveolar lavage combined with effective sputum drainage (intermittent suctioning and postural drainage) was conducted to maintain airway patency, Infection was managed rationally based on pathogen culture and antibiotic susceptibility results, and local nebulization therapy was applied for expectoration and anti-inflammatory purposes. The patient was successfully weaned from mechanical ventilation on the fourth day of hospitalization.

Conclusion

This case provided valuable experience and lessons that hold significant summarizing value. Its value is mainly reflected in three aspects. First, we propose a stepwise detoxification and elimination strategy consisting of continuous gastrointestinal ethanol infusion (with full-course monitoring of serum ethanol concentrations) combined with HA330 hemoperfusion and CRRT, which avoids reliance on high-end equipment and scarce antidotes and is more applicable to primary or resource-limited medical settings. Second, a targeted acid–base correction strategy of "half-dose bicarbonate supplementation plus CRRT maintenance" was adopted to reduce the risk of over-alkalinization, reflecting an individualized treatment approach. Third, an integrated "detoxification–elimination–organ protection" model was established, with refined management strategies tailored to multiple complications, enabling the simultaneous reversal of toxic encephalopathy, acute kidney injury, and aspiration pneumonia, thereby enriching the therapeutic paradigm for critically ill poisoned patients. The successful rescue in this case not only fills a practical gap in the comprehensive management of massive ethylene glycol poisoning but also validates the feasibility of optimized treatment strategies under resource-limited conditions, demonstrating important scientific significance and practical value for improving the care of critically ill poisoned patients and promoting techniques applicable in primary healthcare settings.

Data Sharing Statement

No datasets were generated or analysed during the current study.

Ethics Approval and Consent to Participate

The present case report was approved by the Ethics Committee of the First Clinical Medical College of China Three Gorges University (NO.2025-345-01). Written informed consent was obtained from the patient and the patient's family for the publication of this clinical case report.

Consent for Publication

Written informed consent was obtained from the patient and the patient's family for publication of this case report and any accompanying images.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare no conflicts of interest in this work.

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