International Journal of Medical Toxicology & Forensic Medicine

October 2025, Volume 15, Number 4

Case Report

Fatal Multisystem Toxicity and Vesiculobullous Eruptions Following Brake Fluid Poisoning: A Case Report

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Citation Azizikia H, Amarian M, Chitzan-Zadeh K, Sahranavard M, Sarvestani Z, Yekesadat SM Fatal Multisystem Toxicity and Vesiculobullous Eruptions Following Brake Fluid Poisoning: A Case Report. International Journal of Medical Toxicology and Forensic Medicine. 2025; 15(4):E49459.

https://doi.org/10.22037/ijmtfm.v15i4.49459

Article info:

Received: 14 June 2025

First Revision: 03 July 2025

Accepted: 03 July 2025

Published: 30 Oct 2025

Keywords:

Brake fluid, ethylene glycol, diethylene glycol, alcohol toxicity

ABSTRACT

Background: Ethylene glycol and diethylene glycol, commonly found in automotive brake fluids, are highly toxic agents. While their nephrotoxic and neurotoxic effects are well documented, delayed and atypical systemic manifestations are less frequently reported.

Case Presentation: We describe a 53-year-old male who presented with acute epigastric pain after ingesting 50 mL of brake fluid. Initial assessment and laboratory findings were unremarkable. However, he returned 36 hours later with acute kidney injury, high-anion-gap metabolic acidosis, and worsening clinical status. Over the subsequent days, he developed progressive multi-organ failure, including hypoxemia, altered consciousness, optic disc edema, intraretinal hemorrhages, and visual impairment. By day seven, he demonstrated brainstem areflexia and diffuse cerebral edema, requiring intubation. Dermatomal vesiculobullous eruptions suggestive of viral reactivation and superinfected pressure ulcers. Despite hemodialysis and supportive treatment, he died on day 43 following cardiorespiratory arrest.

Conclusion: This case highlights rare, delayed manifestations of brake fluid poisoning, including optic neuropathy, dermatomal vesiculobullous eruptions, and progressive brainstem dysfunction. Such atypical features may delay diagnosis and complicate management. Brake fluid poisoning can present with evolving multisystem complications beyond the acute phase. Extended observation and a multidisciplinary approach are crucial, even in patients initially presenting with stable vital signs.

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Introduction

Brake fluid is a hydraulic transmission fluid in automotive brake and clutch systems [1]. The primary drivers of brake fluid ingestion toxicity are ethylene glycol, DEG, and their metabolites [2]. In adults, brake fluid ingestion typically reflects suicidal intent; in children, it usually occurs accidentally during exploratory behavior [3].

Individuals exposed to the toxin may develop gastrointestinal, renal, neurological, and cardiopulmonary dysfunction [4]. An unclear or incomplete exposure history can mislead physicians due to the wide range of presentations [5]. We described a 53-year-old male who presented with acute abdominal pain following ingestion of brake fluid. Unlike most previous cases, this patient developed delayed extrarenal manifestations.

Case Presentation

A 53-year-old male first presented to the Damghan Hospital with acute epigastric pain, nausea, and vomiting following oral ingestion of 50 mL of brake fluid. Initial physical examination and laboratory studies were normal. Although the treatment team recommended continued observation, he left with personal consent.

Thirty-six hours post-ingestion, he re-presented to the Imam Hossein Hospital in Shahrood with worsened symptoms. His medical history included hypertension and hyperlipidemia. Home medications were losartan 50 mg and atorvastatin 20 mg. He was a non-smoker and non-alcoholic. His blood pressure was 171/93 mmHg, heart rate 71 beats per minute, respiratory rate 21 breaths per minute, temperature 36.7°C, and oxygen saturation 98% on room air. There was no peripheral edema. Neurological assessment revealed an awake but disoriented patient with intact muscle strength and no focal deficits. Oropharyngeal examination demonstrated a swollen, pale posterior palate. His electrocardiography demonstrated a normal sinus rhythm. The echocardiography revealed an ejection fraction of 50% with no structural abnormalities. Abdominal and pelvic ultrasonography showed grade I steatosis.

Laboratory tests on admission revealed the following results (Table 1): white blood cell count = 16.7 × 10^9/µL, hemoglobin = 14.7 g/dL, platelet count = 167 × 10^9/µL, creatinine = 2.4 mg/dL, urea = 60 mg/dL. Arterial blood gas analysis showed metabolic acidosis (pH=7.239, pCO2=31mmHg, pO2=42mmHg, HCO3=15mmol/L). Patient underwent hemodialysis

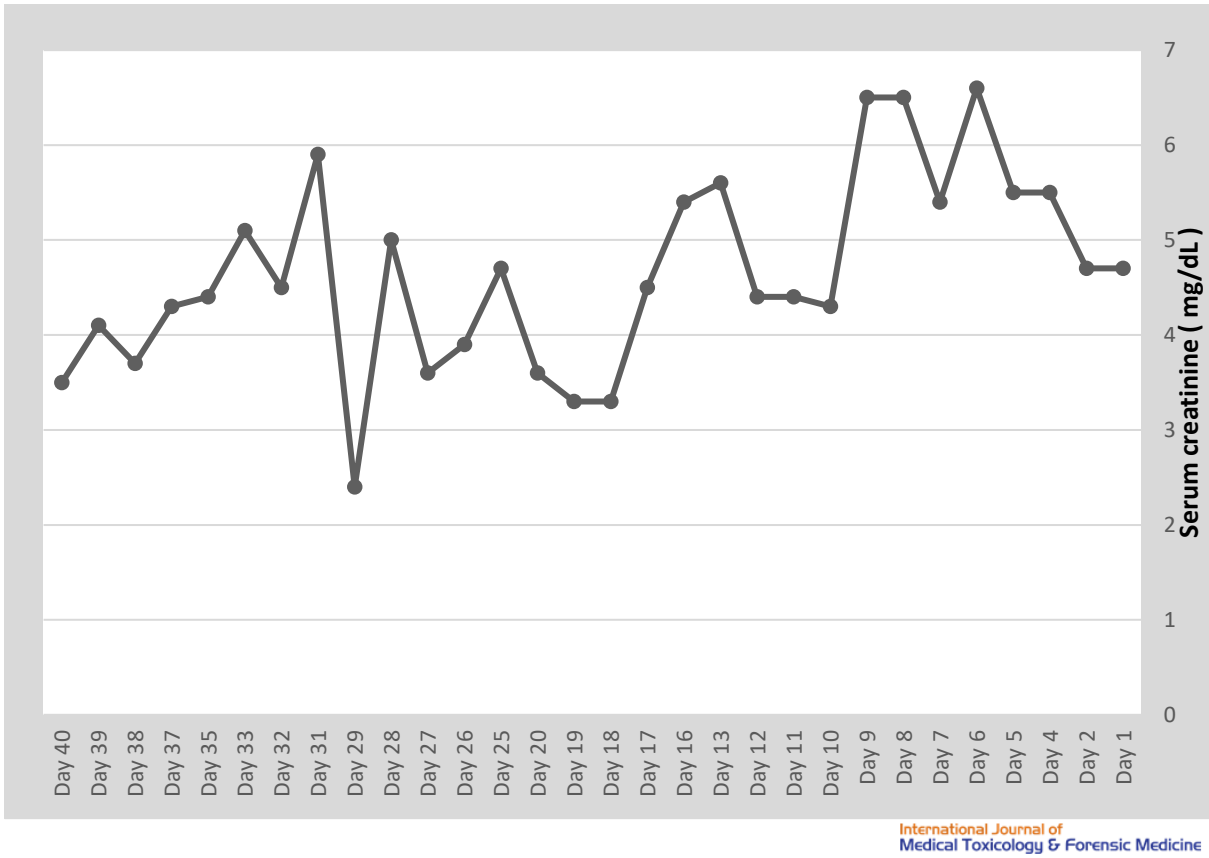

Figure 1. Serum Creatinine.

alongside supportive therapy.

Table 1. Laboratory findings.

	Day 1	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12	Day 13
WBC (mm^3)	16700	17600	-	16700	18100	18100	29800	32400	34500	26500	33000
Neutrophils (%)	97	96	-	93	98	93	96	96	94	93	95
Lymphocytes (%)	2	2	-	3	2	2	2	2	4	4	4
Platelets (mm^3)	167000	192000	-	228000	243000	236000	311000	335000	318000	259000	258000
Hb (g/dL)	14.7	10.2	-	10.4	11.2	10.2	10.6	10.3	10.8	9.6	10.6
Serum creatinine (mg/dL)	2.4	6.6	6.2	5.2	4.5	6.5	4.3	6.4	4.4	5.6	4.8
Blood urea (mg/dL)	60	87	78	62	85	154	99	193	22	192	143
Na^+ (mEq/L)	139	137	134	141	137	137	142	141	139	138	-
K^+ (mEq/L)	2.9	3.8	4.7	4	4.5	4.3	4	5.1	4.2	4.8	-
PT (sec.)	-	-	-	-	-	-	14	-	-	-	-
INR	-	-	-	-	-	-	1.3	-	-	-	-
APTT (sec.)	-	-	-	-	-	-	33	-	-	-	-
SGOT (IU/L)	373	166	148	136		72	78	42	27	21	16
SGPT (IU/L)	325	396	398	361	91	288	283	209	166	114	85
ALP (IU/L)	-	178	200	208	348	229	276	274	240	200	199
Total Bilirubin	-	1.0	-	0.9	-	-	-	-	-	-	-
CPK (IU/L)	419	119	182	122	90	-	308	985	351	202	119
Amylase (IU/L)	-	12	-	15	14	-	22	14	19	19	-
Lipase (IU/L)	-	22	30	28	20	24	18	-	49	89	75
Calcium (mg/dL)	8	8.6	8.6	8	8.7	8.4	8.3	7.5	7.4	7.4	7.5
Phosphorus (mg/dL)	-	3.4	3.8	3.5	6.3	8.9	5.5	7.7	5.7	8.3	-
pH	7.239	7.345	7.335	7.429	7.215	-	7.314	7.235	7.184	-	7.272
HCO3	15	20.4	23.8	30.5	20.1	-	22.7	18.2	22.1	-	24.8
PCO2	31	37	44.1	45.7	49.1	-	44.2	48	50.6	-	53.2
PO2	42	58.6	53.4	38.7	73.8	-	71.9	83.6	58.4	-	54.7

Abbreviations: WBC=White blood cell, HB=Hemoglobin, PT=Prothrombin time, INR=International normalized ratio, APTT=Activated partial thromboplastin time, SGOT=Serum glutamic-oxaloacetic transaminase, SGPT=Serum glutamic-pyruvic transaminase, ALP=Alkaline Phosphatase, CPK=Creatine phosphokinase.

Five days after admission, his laboratory tests showed an elevated creatinine level (Figure 1), which was consistent with hematuria. The patient also developed extrarenal signs, including reduced visual acuity, confusion, and hypoxemia (SO2=74 %). Ophthalmic assessment showed optic disc edema accompanied by intraretinal hemorrhages, absence of a relative afferent pupillary defect (negative Marcus Gunn sign), pupillary edema, and bilateral unaided visual acuity of 1/10. The chest radiograph showed a pericardial effusion (PE) at both costophrenic angles. Non-contrast chest computed tomography (CT) scan revealed dependent basal lung consolidations and a mild left-sided PE.

On day 7, the patient developed fixed, dilated pupils, a positive oculocephalic (Doll's eye) reflex, and no motor withdrawal to painful stimuli. Brainstem reflexes were absent. Non-contrast brain

CT scan at this stage demonstrated diffuse cerebral edema. As his Glasgow Coma Scale score was low (< 8), accompanied by progressive respiratory failure, endotracheal intubation was performed.

Meanwhile, a procalcitonin level of 7.5 ng/ml was reported, indicating a systemic infection. Blood, urine, influenza PCR, SARS-CoV-2 RT-PCR, and viral serologies were all negative. His nasal secretions' microbiological culture grew Escherichia coli and Pseudomonas spp., while the endotracheal aspirate yielded Klebsiella with a colony count of > 10^6 CFU/mL, sensitive to meropenem and gentamicin.

On day twenty, hemorrhage was noted at the tracheostomy. Dark blood was also aspirated from the

Table 1. Continued.

Investigation	Day 14	Day 30	Day 32	Day 33	Day 34	Day 35	Day 40	Day 41	Day 43
WBC (mm3)	30600	2800	6800	7900	9900	16700	18200	13900	23000
Neutrophils (%)	95	82	85	83	97	93	95	95	93
Lymphocytes (%)	3	12	10	10	2	3	4	3	5
Platelets (mm3)	226000	125000	106000	117000	124000	228000	108000	94000	115000
Hb (g/dL)	10.4	7.7	8.7	9	9	10.4	9	9.8	9.2
Serum creatinine (mg/dL)	4.3	4.5	3.8	4.9	3.6	5.2	4.4	3.3	4.5
Blood urea (mg/dL)	127	70	58	79	52	62	79	57	81
Na+ (mEq/L)	142	136	136	132	132	141	130	134	133
K+ (mEq/L)	4.5	3.6	3.7	4.1	3.5	4	3.9	3.4	4.2
PT (sec.)	-	-	-	-	-	-	-	-	-
INR	-	-	-	-	-	-	-	-	-
APTT (sec.)	-	-	-	-	-	-	-	-	-
SGOT (IU/L)	-	-	-	-	-	136	-	-	20
SGPT (IU/L)	-	-	-	-	-	361	-	-	16
ALP (IU/L)	-	-	-	-	-	208	-	-	224
Total Bilirubin	-	-	-	-	-	0.9	-	-	
CPK (IU/L)	-	28	17	-	15	122	-	7	7
Amylase (IU/L)	-	14	10	-	10	15	-	13	17
Lipase (IU/L)	-	24	10	-	9	28	-	7	8
Calcium (mg/dL)	-	7.7	7.9	-	8	8	-	8.1	7.6
Phosphorus (mg/dL)	-	5.4	4.1	-	3.5	3.5	-	3	4.1
pH	7.238	-	-	7.388	-	-	-	-	-
HCO3	22	-	-	27.1	-	-	-	-	-
PCO2	51	-	-	44.6	-	-	-	-	-
PO2	51.4	-	-	-	-	-	-	-	-

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nasogastric tube. Endoscopy revealed clots and mucosal lacerations in the pharynx with patchy mucosal erythema of the gastric mucosa.

On day twenty-six, he developed dermatomal vesicular eruptions on the thoracic wall. Herpes zoster was considered as a differential diagnosis, and antiviral therapy was initiated. Concurrently, infected wounds over his lumbar region prompted initiation of broad-spectrum antibiotics.

The patient experienced recurrent episodes of hypocalcemia, which were corrected with intravenous calcium gluconate. Furthermore, his hemoglobin concentration declined from 8.4 g/dL to 7.5 g/dL, prompting transfusion of packed red blood cells.

The patient had cardiorespiratory arrest on day 43 of poisoning. After 45 minutes of resuscitation, the patient was pronounced deceased.

Discussion

Ethylene glycol is one of the main components of brake fluid [6]. Its biological half-life is approximately 7–10 hours with a lethal volume of 100 ml. Although ethylene glycol itself is harmful, its toxicity derives from metabolites that persist in the body for days, disrupt cellular processes, and account for most clinical effects [2].

Ethylene glycol poisoning progresses through three distinct phases. First, neurological depression includes confusion, lethargy, hallucinations, seizures, and ataxia. In severe cases, ensuing encephalopathy and cerebral edema lead to a persistent coma. Also, damage to the basal ganglia and brainstem, neuropathy of the seventh cranial nerve, and irreversible brain injury leading to death have been reported [6].

Second, cardiopulmonary toxicity develops in 12-24 hours post-ingestion. At the terminal stage of ethylene glycol metabolism, oxalate binds free calcium ions to form insoluble calcium oxalate crystals, precipitating hypocalcemia. Concurrent ethylene glycol-induced metabolic acidosis and hypocalcemia impair myocardial contractility [7, 8].

Finally, renal toxicity stems primarily after 24-72 hours of ingestion from calcium oxalate crystal accumulation in the renal tubules. Histopathology demonstrates calcium oxalate crystals mostly in the proximal segments, with lesser involvement of the distal tubules and glomeruli. Postmortem examinations in previous cases also noted similar deposits in brain and lung tissues, accompanied by PE and petechial hemorrhages on serosal surfaces [8].

Additionally, skin contact with brake fluid can cause irritant dermatitis, blistering, and vesicles. Ocular exposure may also lead to intraocular hemorrhages, papilledema, blurred vision, and even blindness [9].

DEG, another brake-fluid constituent, is primarily metabolized in the liver, and its metabolites are known to be nephrotoxic and neurotoxic [10].

DEG poisoning typically evolves through three stages. First, patients experience gastrointestinal symptoms, including abdominal pain, nausea, and vomiting. Progression to the next stage depends on the amount of ingestion. The hallmark of the second stage is renal injury, characterized by high-anion-gap metabolic acidosis, tubular and interstitial damage, glomerulonephritis, hypokalemia, and acute or chronic kidney failure requiring hemodialysis [11]. Finally, neurological injuries emerge, ranging from central and peripheral neuropathies, in rare cases, quadriplegia [12]. Other reported complications include acute pancreatitis, inflammation-related esophageal injury, and fatty liver changes .

Because patients rarely volunteer a history of toxic alcohol intake, clinicians should base a diagnosis on any hint of exposure, together with the clinical presentation and key laboratory patterns. The key laboratory patterns are an elevated serum osmolality gap and a high-anion-gap metabolic acidosis [5].

The onset of toxic effects after ingesting toxic alcohols depends on their metabolism rate and whether ethanol was taken at the same time. Ethanol is the preferred substrate for alcohol dehydrogenase (ADH]; its presence delays the production of metabolites and can mask early manifestations of toxicity.

Delayed treatment of toxic alcohol exposure worsens patient outcomes. It has been recommended to start therapy promptly when poisoning is strongly suspected or when an unexplained high anion gap metabolic acidosis is present [5].

Brake fluid poisoning management is mainly supportive and includes airway stabilization, hemodynamic optimization, monitoring of vital signs and cardiac function, hemodialysis [13], infection prevention, administration of ethylene glycol antagonists such as fomepizole [14] or ethanol, and adjunctive supplementation with thiamine, pyridoxine, and magnesium [5].

In our case, the clinical presentation aligned more with DEG toxicity. Initial complaints included gastrointestinal symptoms, followed by acute renal failure and high-anion-gap metabolic acidosis. Finally, he developed late neurological sequelae and diffuse cerebral edema. However, esophageal ulceration has not been described in brake fluid poisoning. Endoscopy showed esophageal ulcerations with patchy mucosal erythema of the gastric mucosa. Additional findings, including PE as well as hepatic steatosis, support ethylene glycol as the primary toxic agent rather than DEG. Our patient also experienced dermatomal, vesicular eruptions on his thoracic wall, consistent with infected wounds over his lumbar region. Although the patient received intensive hemodialysis and supportive treatment, he expired at day 43 post brake fluid ingestion.

Conclusion

DEG poisoning should be considered when a patient presents with high-anion-gap metabolic acidosis alongside signs of multi-organ dysfunction. Prompt diagnosis and the early start of appropriate therapy are essential for optimal outcomes.

Conflicts of Interest

The authors report there are no competing interests to declare.

Funding

The Research Ethics Committee of Shahroud University of Medical Sciences approved this study (IR.SHMU.REC.1404.012). The present study was supported by Shahroud University of Medical Sciences and we hereby acknowledge the research deputy for grant No 14040022.

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