

CASE REPORT

Open Access



Suicide by ethylene glycol/brake oil poisoning—a case report

Amal Nishantha Vadysinghe^{*} , W. G. G. B. Kumarasinghe , Sarathchandra Kodikara and Navoda Wickramasinghe

Abstract

Background: Brake fluid is used for automobiles. It is a mixture of different glycol derivatives including ethylene glycol and diethylene glycol (DEG) which are metabolized into various toxic metabolites. Fatalities following brake fluid ingestion are rare in forensic practice. Here, we report a case of suicide by brake fluid ingestion complicated with severe renal failure and esophageal and gastric erosions.

Case presentation: A 52-year-old male, with a history of alcohol dependence and comorbid moderate depression, ingested a bottle of brake fluid (100ml) mixed with alcohol. He had defaulted psychiatric follow-up. He developed severe metabolic acidosis and acute renal failure which necessitated intensive care, other supportive management, and the antidote; ethyl alcohol. On the 2nd day of admission, he developed upper gastrointestinal bleeding with melena. He also had seizures and cardiovascular complications. He died 12 days after hospital admission, and the manner of death was concluded as suicidal. The autopsy revealed congested and edematous brain, flabby and pale heart without evidence of infarction, erythematous trachea, bilateral diffuse pulmonary edema, congested liver, ulcer over the lower 1/3 of the esophagus, and few ulcers in the stomach. Also, the kidneys were edematous, diffusely necrosed, and there were adrenal hemorrhages.

Conclusions: This case report highlights the severity of effects of brake fluid poisoning including the corrosive effect on gastrointestinal mucosa which is rarely encountered in clinical practice.

Keywords: Brake oil, Diethylene glycol, Ethylene glycol, Suicide

Key points

- Different methods are applied for suicide, especially firearms, agrochemicals, etc.
- Self-poisoning with brake oil is rare, and fatality is a remote possibility.
- Ethylene glycol, DEG, and its metabolites are responsible for the toxicity.
- Clinical outcomes of ethylene glycol toxicity and DEG toxicity differ from each other which can be attributed to its different pathways of metabolism.

- The presented case highlights the systemic effects and pathological changes of fatal brake oil poisoning.

Background

Brake oil is a type of hydraulic fluid used in hydraulic brake and hydraulic clutch applications in automobiles, motorcycles, light trucks, and some bicycles. Medical emergencies following its toxicity can occur in different circumstances including accidental, suicidal, or rarely homicidal ingestion of brake oil (Nahrir et al. 2012; Basnayake et al. 2019).

The mainly used types today are glycol-ether-based; DOT 3 (United States Department of Transportation), DOT 4, and DOT 5.1 (95% of the world market). In addition, mineral oil (Citröen Hydraulic Mineral Liquid

*Correspondence: amal_vadysinghe@yahoo.com
Department of Forensic Medicine, Faculty of Medicine, University of Peradeniya, Peradeniya, Sri Lanka

LHM) and silicone (DOT 5)-based fluids are also available (Nahrir et al. 2012; Basnayake et al. 2019; Schep et al. 2009). Glycol-ether-based brake oil is composed of three main components: polyglycol ethers as solvent (60–90%), polyglycols as lubricant basis (5–30%), and additives (2–5%). The main toxic agents in glycol-ether-based brake fluids are diethylene glycol (DEG) and ethylene glycol (Nahrir et al. 2012; Basnayake et al. 2019; Schep et al. 2009).

This report highlights the clinical features, autopsy features, and pathological hallmarks in a case of suicide following DEG/ethylene glycol toxicity. Such occurrences are rarely reported in scientific journals.

Case presentation

The decedent was a 52-year-old male bus conductor. He was married and had 3 children. On the day before the poisoning, he was involved in an argument with a woman at a shop near the bus stand and was assaulted by the same woman in public. The next day, he did not go to work and stayed home. His three children had the school as usual. His wife had gone to the town and came back home to find him vomiting and complaining of abdominal pain. On inquiry, he confessed to taking a bottle of brake oil (100ml) mixed with alcohol, with suicidal intention. He was first taken to the nearest hospital and was transferred to a tertiary care medical facility.

He had a past history of alcohol abuse (dependence syndrome with comorbid depression) for which he was followed up at a psychiatry unit. Although there was no history of self-harming behaviors, he had expressed suicidal ideas on previous occasions as well. However, the family did not observe any behavioral changes during this period. He had defaulted psychiatry clinic follow-up for the last 3 years. There were no other significant medical co-morbidities.

On admission, he was conscious and rational and hemodynamically stable. However, he soon became restless. He was started on the antidote, “ethanol” and other supportive measures. Later he became anuric and his laboratory test results showed acute renal failure and metabolic acidosis with a high anion gap. On day 2 of admission, he developed upper gastrointestinal bleeding with melena. He was shifted from ward to high dependency unit (HDU) and then to the intensive care unit (ICU) where he was electively intubated and started on hemodialysis. He was treated for another 11 days at the ICU for acute renal failure with high anion gap acidosis, acute pulmonary edema, acute cardiac failure, and electrolyte abnormalities. Despite the antidote and the supportive measures, the patient succumbed on the 12th day of admission to the hospital. An inquest was held and requested for a postmortem examination. Autopsy

and other laboratory investigations were conducted at Teaching Hospital Peradeniya and Department of Forensic Medicine, University of Peradeniya, Peradeniya, Sri Lanka.

The autopsy revealed the body was that of a middle-aged male with a tan complexion. He was moderately built and nourished. Peripheries were edematous, and there was evidence of medical intervention. On external examination, the body was found to be free of injuries. Histopathology was conducted at the Department of Forensic Medicine, University of Peradeniya, Sri Lanka.

On internal examination, the brain weighed 1400g, congested, and edematous with no bleeding in or around the brain matter. Microscopically, the brain tissue showed cerebral edema. Mucosal erosions were found throughout the gastrointestinal tract with well-circumscribed ulcer at the lower 3rd of the esophagus (Fig. 1).

Both kidneys were swollen with bleeding and necrosis of the renal cortex. The cortico-medullary demarcation was lost, and there were multiple areas of renal infarction. There was a 7×5×4 cm mass over a superior-medial aspect of the left kidney. This was presumed to be the left adrenal with hemorrhage (Fig. 2a). Microscopy revealed gross cortical necrosis. Thrombosed vessels were noted in relation to the areas of necrosis. Juxtamedullary and medullary areas were preserved. Calcium oxalate crystals were not found within the tubules (Fig. 2b). Both

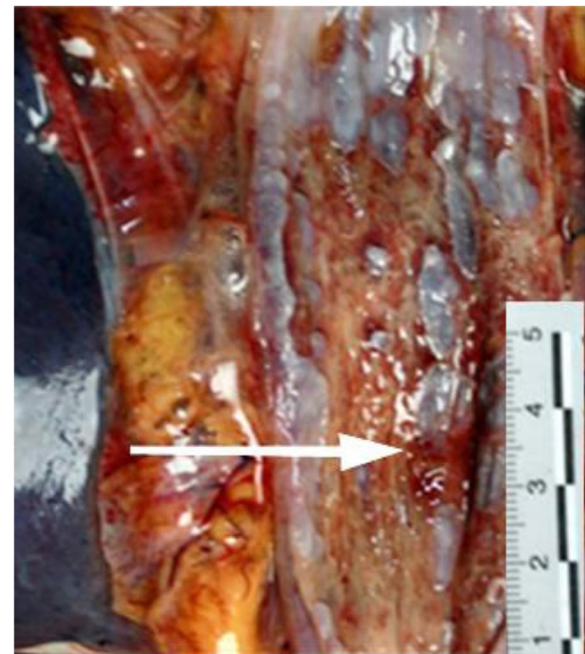
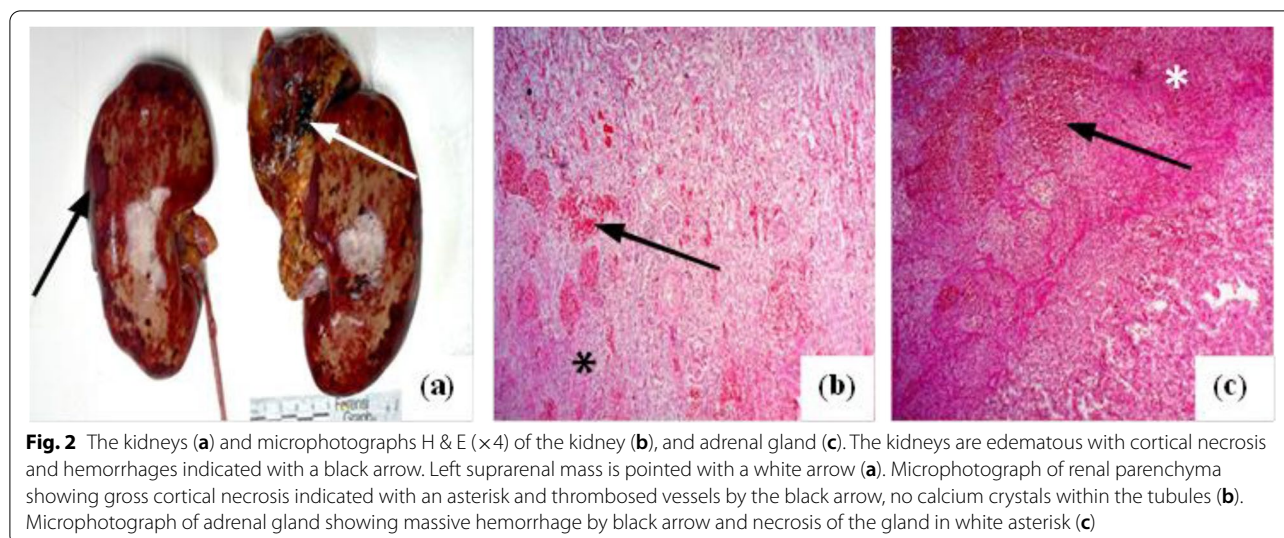


Fig. 1 Longitudinally dissected esophagus showing the mucosal surface with longitudinally placed ulcers indicated with a white arrow at the level of the lower third



adrenal glands showed massive hemorrhages with necrosis (Fig. 2c).

The right and left lungs weighed 580 and 520g, respectively. Pulmonary edema was noted in both lungs. Lungs showed pulmonary edema and hemorrhage, but there was no evidence of pneumonia. The heart was found to be normal macroscopically and microscopically. The liver was enlarged (1900g), and microscopic changes were compatible with the fatty liver. The spleen was enlarged (220g) and showed predominant autolytic changes microscopically. The pancreas was congested and edematous with few areas of hemorrhages. There were well-demarcated areas of chalky white appearance seen over the surrounding omental fat. Microscopically, the pancreas showed features of pancreatic necrosis, pancreatitis, and fat necrosis (Fig. 3).

Toxicological analysis of the blood on admission identified glycol compounds 412.4mg/L by gas chromatography and an ethanol concentration of 50mg/dl. The cause of death was concluded as multi-organ failure following brake oil ingestion. Circumstantial evidence and post-mortem findings were keeping with the manner of death as suicidal.

Discussion

Death by brake oil poisoning is an uncommon method of suicide though accidental cases are frequent. Brake oil consists of glycol ether, ethylene, or diethylene glycol and additives. Ethylene glycol or diethylene glycol and its derivatives are responsible for the toxic effects. The popular belief was that poly-ethylene glycols and diethylene glycols both break down into ethylene glycol before being degraded to its end products. Interestingly, the metabolic pathways of this monomer and dimer are not the same.

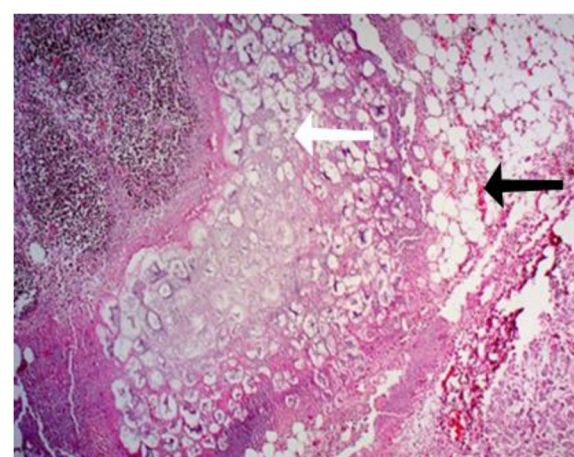


Fig. 3 Microphotograph of the pancreas H & E (x10) showing hemorrhage indicated with a black arrow, fat necrosis indicated with a white arrow, and pancreatitis

The relatively stable bond of diethylene glycol drives the metabolism through a different pathway (Friedman et al. 1962; Wanda et al. 2013).

Ethylene glycol itself is toxic, but the harmful effects mainly result from the accumulation of its more toxic metabolites. Ethyleneglycol is metabolized in the liver to glycolaldehyde, glycolate, glyoxylate, and oxalate. These metabolites inhibit many cellular functions and are responsible for most of the clinical effects (Parry and Wallach 1974; Moreau et al. 1998). The rate-limiting step of this process is the conversion of glycolic acid to glyoxylic acid. Therefore, the accumulation of glycolic acid is largely responsible for the metabolic acidosis seen with this poisoning like in our case (Moreau et al. 1998; Brent

2001). The end product-oxalate chelates with calcium ions forming the relatively insoluble calcium oxalate crystals and also results in hypocalcemia (Parry and Wallach 1974).

Severe ethylene glycol poisoning may go through three clinical stages. They are central nervous system (CNS) depression, cardiopulmonary toxicity, and renal toxicity, respectively. CNS depression is caused by the direct effect of ethylene glycol and its metabolic acids especially, glycolic acid. Seizures may occur as a result of the direct toxic effects and hypocalcemia followed by persistent coma due to encephalopathy and cerebral edema in severe poisoning (Parry and Wallach 1974; Brent 2001; Tennant et al. 2006).

Ethylene glycol-induced acidosis and hypocalcemia are known to cause myocardial depression. Calcium oxalate crystals have also been found in myocardial tissues of fatally poisoned victims in previous studies (Brent 2001) but were not seen in this case.

Accumulated calcium oxalate crystals in the tubules are mainly responsible for the renal toxicity. Previous studies have noted predominant involvement of proximal tubules with less prominent involvement of distal tubules and glomeruli. Pathological hallmark is the deposition of calcium oxalate crystals in renal tubules (Parry and Wallach 1974; Brent 2001). These deposits can also be seen in other tissues like the brain and lung in a fatal overdose. In addition, previous autopsies have also found pulmonary edema and petechial hemorrhages in the lungs, pleura, heart, and pericardium (Brent 2001).

In addition to ethylene glycol, other components in brake fluid such as diethylene glycol (DEG) may also contribute to the clinical outcome. DEG is principally metabolized in the liver via the same nicotinamide adenine dinucleotide (NAD)-dependent pathway and is excreted renally (Schep et al. 2009). In the liver, DEG is converted to 2-hydroxyethoxyacetaldehyde and 2-hydroxyethoxyacetic acid. Both DEG and 2-hydroxyethoxyacetic acid are considered to be nephrotoxic and neurotoxic. The clinical effects of DEG poisoning can also be divided into three stages which are comprised of the initial stage of gastrointestinal symptoms, the second stage of renal toxicity with high anion gap metabolic acidosis, and the final stage of various neuropathies and neurotoxicity. Other manifestations include CNS depression and hemorrhage, pancreatitis, and fatty liver (Shannon et al. 2007; Reddy et al. 2010).

Nephrotoxicity was the prominent feature in DEG poisoning according to animal poisoning studies. Renal injuries result from tubular degeneration principally involving proximal convoluted tubules. Therefore, manifests as cortical infarctions/necrosis with hemorrhage.

However, renal injury due to calcium oxalate crystal deposition is not a feature of DEG poisoning (Schep et al. 2009; Shannon et al. 2007).

In our case, clinical features are more compatible with DEG poisoning. His first symptoms were vomiting and abdominal pain. Then, he developed acute renal failure with high anion gap metabolic acidosis. He succumbed before developing late neurological sequelae. Esophageal ulceration is another presentation of brake oil poisoning which has not been reported in the literature so far. However, mixing alcohol with brake fluid must have delayed the development of clinical features. Alcohol is the antidote which competitively binds with the alcohol dehydrogenase enzyme preventing the metabolism of glycol compounds (Shannon et al. 2007). Furthermore, histology excluded calcium oxalate crystals within renal tubules and displayed massive renal cortical necrosis. Other findings included cerebral and pulmonary edema, fatty liver, pancreatitis, and adrenal hemorrhages which confirm DEG poisoning rather than ethylene glycol poisoning.

Conclusions

Although rarely encountered, brake oil poisoning by self-ingestion can manifest with fatal outcomes. Understanding the pathophysiology is important in the management of such cases. The main toxic components of brake fluid are ethylene glycol, DEG, and its metabolites. The mechanism of toxicity in DEG poisoning differs from that of ethylene glycol poisoning. Fatalities due to DEG poisoning are commonly caused by renal toxicity (renal tubular necrosis) and high anion gap metabolic acidosis. In addition, gastrointestinal manifestations such as esophageal and gastric ulceration may also contribute to the clinical outcome.

Abbreviations

DEG: Diethylene glycol; DOT: Department of Transportation; HDU: High dependency unit; ICU: Intensive care unit; CNS: Central nervous system; NAD: Nicotinamide adenine dinucleotide.

Acknowledgements

We appreciate the support given by family members of the deceased for giving the opportunity to share this information among the academic audience.

Authors' contributions

Amal Nishantha Vadysinghe, W.G.G.B. Kumarasinghe, Sarathchandra Kodikara—autopsy and writing and editing of the manuscript. Navoda Wickramasinghe—writing and finalizing the case report. The authors have read and approved the final manuscript.

Funding

None

Availability of data and materials

Not applicable

Declarations

Ethics approval and consent to participate

All procedures performed in the study were in accordance with the ethical standards of the institution and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Ethical Committee's reference number is not applicable

This is a case of the autopsy which was done for medico-legal purposes.

Therefore, written consent was obtained from the next of kin to use this case for academic purposes.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

Received: 5 August 2021 Accepted: 7 October 2021

Published online: 16 October 2021

References

- Basnayake BMDB, Wazil AWM, Nanayakkara N, Mahanama RMBSS, Premathilake PNS, Galkaduwa KKMCDK (2019) Ethylene glycol intoxication following brake fluid ingestion complicated with unilateral facial nerve palsy: a case report. *J Med Case Rep* 13:1–4. <https://doi.org/10.1186/s13256-019-2139-z>
- Brent J (2001) Current management of ethylene glycol poisoning. *Drugs* 61:979–988. <https://doi.org/10.2165/00003495-200161070-00006>
- Friedman EA, Greenberg JB, Merrill JP, Dammin GJ (1962) Consequences of ethylene glycol poisoning. Report of four cases and review of the literature. *Am J Med* 32:891–902. [https://doi.org/10.1016/0002-9343\(62\)90035-9](https://doi.org/10.1016/0002-9343(62)90035-9)
- Moreau CL, Kerns W, Tomaszewski CA, McMartin KE, Rose SR, Ford MD, Brent J (1998) Glycolate kinetics and hemodialysis clearance in ethylene glycol poisoning. META Study Group. *Clin Toxicol* 36:659–666. <https://doi.org/10.3109/15563659809162613>
- Nahrir S, Sinha S, Siddiqui KA (2012) Brake fluid toxicity feigning brain death. *BMJ Case Rep* 2012:bcr0220125926. <https://doi.org/10.1136/bcr-02-2012-5926>
- Parry MF, Wallach R (1974) Ethylene glycol poisoning. *Am J Med* 57:143–150. [https://doi.org/10.1016/0002-9343\(74\)90780-3](https://doi.org/10.1016/0002-9343(74)90780-3)
- Reddy NJ, Sudini M, Lewis LD (2010) Delayed neurological sequelae from ethylene glycol, diethylene glycol and methanol poisonings. *Clin Toxicol* 48:967–973. <https://doi.org/10.3109/15563650.2010.532803>
- Schep LJ, Slaughter RJ, Temple WA, Beasley DM (2009) Diethylene glycol poisoning. *Clin Toxicol* 47:525–535. <https://doi.org/10.1080/15563650903086444>
- Shannon MW, Barron SW, Burns MJ (2007) Chapter 32 - methanol, ethylene glycol, and other toxic alcohols. In: Haddad and Winchester's clinical management of poisoning and drug overdose (Fourth Edition). W.B. Saunders, Philadelphia, pp 605–633
- Tennant I, Crawford-Sykes A, Ward L, Thesiger C (2006) Ethylene glycol poisoning following ingestion of brake fluid. *West Indian Med J* 55:286–287. <https://doi.org/10.1590/s0043-31442006000400013>
- Wanda MH, Colin GR, Matthew AW (2013) Haschek and Rousseaux's handbook of toxicologic pathology (Third Edition). Academic Press, Cambridge

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Submit your manuscript to a SpringerOpen[®] journal and benefit from:

- Convenient online submission
- Rigorous peer review
- Open access: articles freely available online
- High visibility within the field
- Retaining the copyright to your article

Submit your next manuscript at ► [springeropen.com](https://www.springeropen.com)