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Iatrogenic diethylene glycol poisoning in a herd of dairy cattle

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Case Report

Keywords: propylene glycol, kidney injury, acute renal failure, liver injury

Posted Date: November 5th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11403840/v1

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Additional Declarations: No competing interests reported.

Abstract

Diethylene glycol (DEG) poisoning is reported mainly in pets, with no previous reports in ruminants. This study describes a DEG poisoning outbreak affecting nine dairy cows in Minas Gerais, Brazil, following ingestion of contaminated drench administered during postpartum protocols. Clinical signs and biochemical analyses indicated severe renal injury. Necropsy revealed generalized petechiae, an enlarged liver with a nutmeg appearance, and pale kidneys with marked perirenal edema and multifocal petechiae. Histopathology confirmed renal tubular necrosis and degeneration. Laboratory analysis of the product, believed to contain propylene glycol, revealed that it consisted almost entirely of DEG.

Introduction

Propylene glycol ($C_3H_8O_2$) is an energy supplement commonly administered orally to postpartum dairy cows to prevent metabolic diseases such as ketosis. However, its physical properties can be similar to those of diethylene glycol (DEG) ($C_4H_{10}O_3$), a toxic industrial solvent that has been responsible for numerous poisoning incidents in both humans and animals. DEG is a notorious contaminant, frequently and illegally used as an inexpensive, sweet-tasting substitute for legitimate pharmaceutical ingredients like glycerin or propylene glycol. This issue of adulteration has caused lethal mass poisonings worldwide for nearly a century, including historical events like the 1937 Elixir Sulfanilamide disaster in the United States, which led to modern food and drug regulations. More recent outbreaks in humans have been linked to contaminated cough syrup in Panama (Sosa et al. 2014) and pediatric medication in Nigeria (Besenhofer et.al. 2011).

In veterinary medicine, DEG poisoning is most commonly reported in companion animals, particularly dogs and cats, who may be attracted to its sweet taste in products like antifreeze and brake fluids. Ingestion leads to severe multiorgan toxicity, with clinical signs appearing as early as 30 minutes and developing into acute kidney and liver damage within 72 hours (Grauer et al. 1984; Thrall et al. 1984; Claus et al., 2011; Sauder et al. 2026). The toxicity is primarily attributed to its metabolite, 2-hydroxyethoxyacetic acid (HEAA), which is directly damaging to renal tubular cells. Despite the well-documented toxicity in other species, there are currently no reports of spontaneous DEG poisoning in ruminants in scientific literature (Landry et al. 2011; Araújo et al. 2023). This article aims to fill that knowledge gap by presenting the first detailed report of an outbreak of accidental diethylene glycol poisoning in a herd of dairy cows. The incident occurred on a farm in Minas Gerais, Brazil, after the substance was mistakenly administered instead of propylene glycol as part of a postpartum drench protocol. We describe the clinical signs, biochemical profile, and pathological findings associated with toxicosis in the affected cattle.

Case description

History and Location

The outbreak occurred in a dairy farm in Ressaquinha, Minas Gerais state, Southeastern Brazil. The herd consists of approximately 130 lactating Holstein cows, with a daily milk output of around 4,500 liters.

Postpartum Protocol and Poisoning Event

Immediately following parturition, cows were transferred to a barn and subjected to routine oral fluid therapy (drench). The protocol consists of 20 liters of fluid containing mineral salts and 400 milliliters of a product labeled as propylene glycol, alongside intravenous calcium.

Clinical and Pathological Examinations

A series of veterinary visits were conducted to investigate the outbreak. Affected animals underwent thorough physical and neurological examinations. Blood samples were collected from three cows for serum biochemical panels and blood smear analysis. Two cows with severe clinical signs and a negative prognosis were euthanized for necropsy. Samples of the lung, heart, spleen, kidneys, liver, abomasum, intestine, uterus, mammary gland, brain and ocular globes, lymph nodes, intestine, and brain were collected for histopathological analysis.

Results

Epidemiology and General Clinical Signs

According to reports from the farm's veterinarian and staff, nine postpartum cows (eight primiparous and one multiparous) began showing signs of illness approximately 3 to 4 days after calving. The initial clinical sign reported was a sudden onset of blindness, with the eyes turning a bluish hue before returning to normal color, albeit with a persistent white spot, followed by a gradual recovery of vision. Despite this, the overall condition of the affected animals often worsened over time, leading to the death of five primiparous cows within about a week after initial symptoms.

Physical examination of three affected cows revealed a range of severe clinical signs pointing to multi-systemic disease. Neurological deficits were prominent, including depression, apathy, ataxia, reduced proprioception, and diminished superficial and deep sensitivity. Ocular abnormalities included near-total blindness, congested mucous membranes, engorged episcleral vessels, and cloudiness of the anterior chamber. Signs of severe gastrointestinal dysfunction were also present, including ruminal atony, metallic sounds on percussion, and scanty, dry, or foul-smelling feces.

Biochemical and Laboratory Findings

No blood parasite was found by examination of blood smears in the three cows. The serum biochemical panel, however, revealed severe kidney and liver injury (Table 1) as blood urea and creatinine levels were markedly increased in all three cows, as well as aspartate aminotransferase (AST) and gamma-glutamyl

transferase (GGT). Serum activities of alanine aminotransferase (ALT) and alkaline phosphatase (ALP) were increased in one cow, and glucose levels were increased in another cow.

Table 1
Biochemical panel of poisoned cows.

Parameter	Animals			Reference values
	A	B	C	
Urea (mg/ dL)	161.7	222.0	222.5	44.6–66.8
Creatinine (mg/ dL)	9.1	18.8	18.6	0.5–2.2
ALT (U/L)	21.9	51.2	30.0	11–40
AST (U/L)	228.5	564.5	265.8	60–125
Alkaline phosphatase (U/L)	204.6	748.6	383.6	0–488
GGT (U/L)	22.7	72.7	29.9	6.0–17.4
Glucose (mg/ dL)	41.4	63.9	148.0	40–100
Total proteins (g/ dL)	6.3	7.1	6.4	6.0–7.5
Albumin (g/ dL)	3.2	3.5	2.9	2.5–3.8
Globulins (mg/ dL)	3.1	3.6	3.4	3.07–4.20
Legend: ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transferase				

Pathological Findings

The main pathological findings were consistent among necropsied cows. Significant gross lesions included hemorrhagic ulcers in the abomasal mucosa (Fig. 1A), multifocal multifocal petechiae in the omentum, congestion in the intestinal serosa and jejunal mucosa, and significant edema in the periuterine and perirenal regions. The liver has a variegated pattern ranging from dark red to light red, similar to nutmeg appearance, and the kidneys were pale.

The histopathological examination revealed a high degree of acute renal injury. Both cow C (Fig. 1) and cow B (Fig. 2) showed several renal tubular degeneration with proteinuria, accompanied by discrete multifocal epithelial tubular regeneration. Moderate multifocal aleatory neutrophilic and histiocytic hepatitis with hepatocyte loss and marked centrilobular glycogen and lipid degeneration; moderate and diffuse lymphangiectasia in the small intestine; focally extensive and pronounced necrotizing and histiocytic metritis; and ocular aqueous flare were also encountered. Additionally, one cow demonstrated moderate multifocal fibrinopurulent bronchopneumonia with intralesional foreign body (structure resembling plant fiber). Collected mammary gland and encephalus did not present histological lesions.

The product administered to the cows, which had been purchased as propylene glycol, was analyzed for the presence of propylene glycol, ethylene glycol, and diethylene glycol using gas chromatography coupled with a flame ionization detector. This analysis revealed this product was almost entirely diethylene glycol (purity non determined). Considering clinical, biochemical and necropsy findings and analysis of the substance administered to the cows, the diagnosis was diethylene glycol poisoning.

Discussion

This report details the first documented case of diethylene glycol (DEG) poisoning in a herd of dairy cattle, which resulted in severe clinical signs and high mortality. The investigation confirmed that a product administered as propylene glycol was almost entirely DEG, leading to acute renal and hepatic failure in the affected animals.

The clinical presentation and pathological findings in these cows are consistent with DEG toxicosis reported in other species (Hesser 1986; Mathews et al. 1991; O'Brien et al. 1998; Ferrari and Giannuzzi 2005; Schep et al. 2009; Araújo et al. 2023). The severe azotemia, characterized by markedly elevated serum urea and creatinine, is a hallmark of the acute renal failure caused by DEG poisoning in humans and dogs. Likewise, the primary histopathological lesion, severe renal tubular degeneration and necrosis, is the characteristic finding in fatal DEG cases across species. The toxicity is primarily driven by the metabolism of DEG to diglycolic acid (DGA), which is directly cytotoxic to renal tubular epithelial cells (Hesser 1986; O'Brien et al. 1998; Ferrari and Giannuzzi 2005; Schep et al. 2009; Araújo et al. 2023). While ethylene glycol poisoning is famously associated with calcium oxalate monohydrate crystalluria, this finding is less consistent in DEG toxicosis (Agerholm et al. 2022; Almeida et al. 2025).

The neurological signs observed in the cattle, such as acute blindness and ataxia, are particularly significant. These findings align with severe nervous system damage reported in human poisoning cases, including a recent and tragic mass poisoning event also in Minas Gerais, Brazil, linked to contaminated beer from a local brewery (Sanchez et al. 2020; Goulart et al. 2022; Almeida et al. 2025). This highlights a similar pattern of multi-organ toxicity, affecting both the kidneys and the nervous system.

This outbreak is a stark reminder of the global public health threat posed by DEG contamination. DEG is a notorious industrial solvent frequently and illegally used as an inexpensive, sweet-tasting substitute for legitimate pharmaceutical ingredients like glycerin or propylene glycol. This issue of adulteration has caused numerous lethal mass poisonings worldwide for nearly a century, dating back to incidents as significant as the 1937 Elixir Sulfanilamide disaster in the United States, which spurred the creation of the modern Food and Drug Administration (FDA) (Wax 1995; Jarrell 2012;).

In conclusion, these cases of DEG poisoning in cattle underscores the critical importance of stringent quality control and supply chain verification for both veterinary and human products. The clinical and pathological findings were classic for DEG toxicosis, and the incident serves as a crucial alert to

veterinarians and livestock producers about the potential for fatal consequences from contaminated commercial supplements.

Declarations

Ethics approval: Não foi necessário para o relato.

Consent for publication: Todos os autores consentiram a publicação deste relato.

Availability of data and materials: Not applicable.

Competing interests: The authors declare that they have no competing interests.

Funding: This report was financed by Coordination for the Improvement of Higher Education Personnel (CAPES)- Finance Code 001.

Acknowledgments: We would like to thank the Department of Veterinary Clinical and Surgery Sciences of the School of Veterinary Medicine and the Department of Chemistry of the Institute of Exact Sciences at the Federal University of Minas Gerais (UFMG). We also gratefully acknowledge the Coordination for the Improvement of Higher Education Personnel (CAPES), Brazil, for financial support for the publication of this review - Finance code 001.

Contributions: I.B.M., M.V.G.J., G.A., C.S.O., R.M.M., E.J.F.F., and T.F.M. collected the information regarding the case, examined the cattle, performed the necropsies, and collected samples from the animals included in the study. M.V.L.M. performed histopathological analysis. B.S.B. and B.G.B. were responsible for analyzing samples for the presence of diethylene glycol. All authors contributed substantially to the content and reviewed or edited the manuscript. All authors read and approved the final manuscript.

References

1. Agerholm JS, Hansen KS, Voogd HL, Krogh AKH (2022) Ethylene glycol toxicosis in milk-fed dairy calves. Acta Vet Scand 64:7. https://doi.org/10.1186/s13028-022-00626-1
2. Almeida JS, Andrade Filho A, Nascimento ND, Paulo PHO, Cedrim Filho AJM (2025) Relato de caso: intoxicação grave por etilenoglicol e os desafios no seu diagnóstico [Case report: severe ethylene glycol poisoning and the challenges in its diagnosis]. Rev Med Minas Gerais 35:e-35402. https://dx.doi.org/10.5935/2238-3182.2025e35402
3. Araújo SA, Faria BCD, Vasconcelos JC, Cruz AF, Souza VS, Wanderley DC, Simões-e-Silva AC (2023) Renal toxicity caused by diethylene glycol: an overview. Int Urol Nephrol 55:2867-2875. https://doi.org/10.1007/s11255-023-03604-2
4. Besenhofer LM, McLaren MC, Latimer B, Bartels M, Filary MJ, Perala AW, McMartin KE (2011) Role of tissue metabolite accumulation in the renal toxicity of diethylene glycol. Toxicol Sci 123:374-383.

https://doi.org/10.1093/toxsci/kfr197

5. Bowie MD, McKenzie D (1972) Diethylene glycol poisoning in children. S Afr Med J 46:931-934.
6. Claus MA, Jandrey KE, Poppenga RH (2011) Propylene glycol intoxication in a dog. J Vet Emerg Crit Care (San Antonio) 21:679-683. https://doi.org/10.1111/j.1476-4431.2011.00688.x
7. Ferrari LA, Giannuzzi L (2005) Clinical parameters, postmortem analysis and estimation of lethal dose in victims of a massive intoxication with diethylene glycol. Forensic Sci Int 153:45-51. https://doi.org/10.1016/j.forsciint.2005.04.038
8. Goulart COL, Bordoni LS, Nascentes CC, Costa LM (2022) Analysis of diglycolic acid after mass poisoning by diethylene glycol. J Anal Toxicol 46:64-68. https://doi.org/10.1093/jat/bkaa187
9. Grauer GF, Thrall MA, Henre BA, Grauer RM, Hamar DW (1984) Early clinicopathologic findings in dogs ingesting ethylene glycol. Am J Vet Res 45:2299-2303. https://doi.org/10.2460/ajvr.1984.45.11.2299
10. Hesser L (1986) Diethylene glycol toxicity. Food Chem Toxicol 24:261-263. https://doi.org/10.1016/0278-6915(86)90248-6
11. Jarrell K (2012) Regulatory history: Elixir sulfanilamide. J GXP Compliance 16:12.
12. Landry GM, Martin S, McMartin KE (2011) Diglycolic acid is the nephrotoxic metabolite in diethylene glycol poisoning inducing necrosis in human proximal tubule cells in vitro. Toxicol Sci 124:35-44. https://doi.org/10.1093/toxsci/kfr204
13. Marraffa JM, Holland MG, Stork CM, Hoy CD, Hodgman MJ (2008) Diethylene glycol: widely used solvent presents serious poisoning potential. J Emerg Med 35:401-406. https://doi.org/10.1016/j.jemermed.2007.06.025
14. Mathews JM, Parker MK, Matthews HB (1991) Metabolism and disposition of diethylene glycol in rat and dog. Drug Metab Dispos 19:1066-1070. https://doi.org/10.1016/S0090-9556(25)09038-5
15. O'Brien KL, Selanikio JD, Hecdivert C, Placide MF, Louis M, Barr DB, Barr JR, Hospedales CJ, Lewis MJ, Schwartz B, Philen RM, St Victor S, Espindola J, Needham LL, Denerville K (1998) Epidemic of pediatric deaths from acute renal failure caused by diethylene glycol poisoning. JAMA 279:1175-1180. https://doi.org/10.1001/jama.279.15.1175
16. Sanchez ZM, Oliveira CL, Laranjeira R, Caetano R (2020) Intoxication and deaths from diethylene glycol present in Brazilian craft beers. J Stud Alcohol Drugs 81:522-523. https://doi.org/10.15288/jsad.2020.81.522
17. Sauder T, Ricard M, Loewen J, Cowan V (2026) A retrospective case series of companion animals hospitalized for ethylene glycol poisoning at a Canadian veterinary teaching hospital. Can Vet J 67:553-562.
18. Schep LJ, Slaughter RJ, Temple WA, Beasley DM (2009) Diethylene glycol poisoning. Clin Toxicol 47:525-535. https://doi.org/10.1080/15563650903086444
19. Singh J, Dutta AK, Khare S, Dubey NK, Harit AK, Jain NK, Wadhwa TC, Gupta SR, Dhariwal AC, Jain DC, Bhatia R, Sokhey J (2001) Diethylene glycol poisoning in Gurgaon, India, 1998. Bull World Health

Org 79:88-95.

20. Sosa NR, Rodriguez GM, Schier JG, Sejvar JJ (2014) Clinical, laboratory, diagnostic, and histopathologic features of diethylene glycol poisoning--Panama, 2006. Ann Emerg Med 64:38-47. https://doi.org/10.1016/j.annemergmed.2013.12.011
21. Thrall MA, Grauer GF, Mero KN (1984) Clinicopathologic findings in dogs and cats with ethylene glycol intoxication. J Am Vet Med Assoc 184:37-41. https://doi.org/10.2460/javma.1984.184.01.37
22. Wax PM (1995) Elixirs, diluents, and the passage of the 1938 Federal Food, Drug and Cosmetic Act. Ann Intern Med 122:456-461. https://doi.org/10.7326/0003-4819-122-6-199503150-00009

Figures

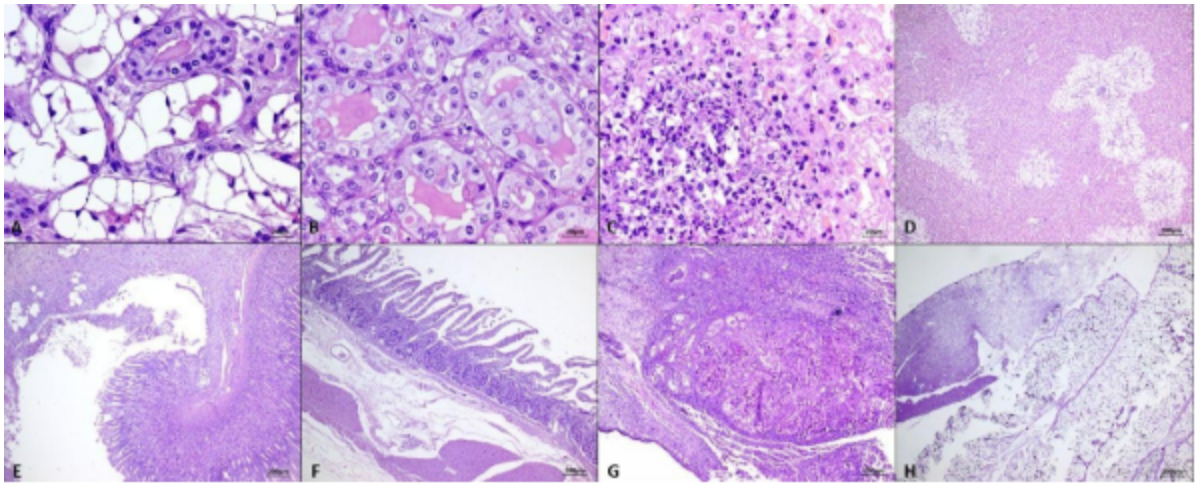


Figure 1

Cow C A) Renal tubular degeneration. B) Renal tubular proteinuria and epithelium regeneration. C) Neutrophilic and histiocytic hepatitis with loss of hepatocytes. D) Liver degeneration. E) Abomasal ulcer. F) Lymphangiectasia in small intestinal villi. G) Necrotizing metritis. H) Ocular aqueous flare. (HE).

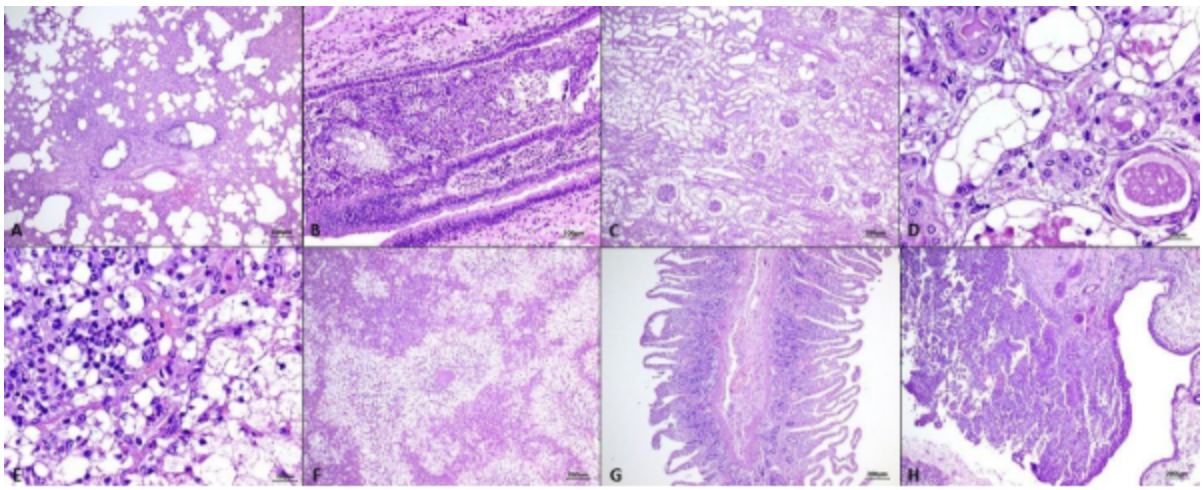


Figure 2

Cow B. A) Bronchopneumonia. B) Bronchopneumonia with intralesional plant fiber-like structure. C and D) Renal tubular degeneration and proteinuria. E) Neutrophilic and histiocytic hepatitis with hepatocyte loss and liver degeneration. F) Liver degeneration. G) Lymphangiectasia in small intestinal villi. H) Necrotizing metritis. (HE).