

CASE REPORT

Companion or pet animals

Ethylene glycol intoxication (antifreeze poisoning) in a domestic ferret (*Mustela putorius furo*)

Sören Nieweg¹  | Sebastian Cichowski¹ | Kerstin Müller² 

¹Small Animal Practice Dr. S. Cichowski, Dissen, Germany

²Dipl. ECZM (Small Mammal Medicine and Surgery), Small Animal Clinic, Veterinary Hospital Freie Universität Berlin, School of Veterinary Medicine, Berlin, Germany

Correspondence

Sören Nieweg, Small Animal Practice Dr. S. Cichowski, Dissen, Germany.
Email: s.nieweg@icloud.com

Sören Nieweg currently works at Anicura Ahlen GmbH (specialist center for small animals).

Abstract

An 8-year-old, 1.06 kg, male castrated, domestic ferret (*Mustela putorius furo*), was presented with an antifreeze (ethylene glycol) poisoning. The relevant medical history shows a T-cell lymphoma diagnosed 8 months ago and was being treated with prednisolone. At the initial examination, an ataxic gait was noted and blood, urine and abdominal ultrasound tests were performed during the 48-h hospitalisation. During hospitalisation, clinical examinations revealed ataxia, apathy, somnolence, hypothermia, salivation, vomiting, anorexia and anuria. Blood tests and ultrasound examinations revealed the development of azotemia with reduced urine-specific gravity and loss of renal structure, with renal medulla and cortex no longer being differentiated. Treatment included ethanol infusion, metoclopramide, maropitant and furosemide. The ferret died of acute renal failure 48 h after initiation of therapy and autopsy confirmed antifreeze intoxication as the cause of death. Antifreeze poisoning in ferrets is underreported but possible. Prognosis is poor without immediate therapy.

BACKGROUND

Antifreeze poisoning has been described in small animals, but appears to be either underreported or rare in ferrets. As an analysis of the database of the Animal Poison Control Center (NAPCC) of the American Society for the Prevention of Cruelty to Animals on reported poisonings shows, in which about 0.5% of the reported cases were ethylene glycol poisonings, but only eight ferrets were represented in these 579 reported cases.¹ Ethylene glycol is a dihydric alcohol, which is often used as antifreeze and whose metabolites are highly neurotoxic and nephrotoxic.^{2–7} The sweet taste and the lack of odor lead to ingestion by humans, dogs and cats.^{5,8,9} Ethylene glycol has mainly depressive effects on the central nervous system and causes gastrointestinal irritation.¹⁰ Additionally, ethylene glycol metabolites formed by the enzyme alcohol dehydrogenase (ADH) in the liver are highly neuro- and nephrotoxic.^{2–7} Ethylene glycol is first oxidised by ADH in several steps to glyoxylic acid.^{10,11} Glyoxylic acid is primarily further metabolised into oxalic acid, but the end products also include glycine, formic acid, hippuric acid, oxalomalic acid and benzoic acid.^{10,11} Calcium is bound to oxalic acid, which leads to the formation of calcium oxalate crystals, which are deposited in the kidneys in particular and lead to crystalluria.¹⁰ The metabolites cause multisystemic organ effects, especially metabolic acidosis and acute renal failure (ARF),^{5,12,13} whereby a study on cultured kidney cells showed

that the calcium oxalate monocrystals are mainly responsible for renal failure, as they change the function and structure of the cell membrane of the kidney cells and cause dysfunction of the mitochondria, which leads to cell death.¹¹ The neurological dysfunctions are caused by metabolites of the aldehyde group, with glycoaldehyde being considered the main culprit. Glycoaldehyde affects respiration, glucose metabolism, serotonin metabolism and leads to impaired amine concentrations in the central nervous system.⁶ Common therapies for dogs and cats are ethanol or fomepizole to block ADH, as well as forced diuresis and gastric lavage to accelerate elimination of ethylene glycol, or haemodialysis or peritoneal dialysis to remove ethylene glycol and its metabolites, as well as treatment of the symptoms that occur.^{7,8,10,14} This case report describes the course of intoxication with ethylene glycol in a ferret, with the progression of clinical and laboratory changes under an established treatment protocol for dogs and cats, during which the animal died of ARF. The negative outcome in this ferret raises the question of whether ferrets, such as cats, benefit from a more suitable treatment protocol with high-dose fomepizole.^{7,15}

CASE PRESENTATION

An 8-year-old, 1.06 kg, male castrated, domestic ferret (*Mustela putorius furo*) was presented after observed ingestion

This is an open access article under the terms of the [Creative Commons Attribution](https://creativecommons.org/licenses/by/4.0/) License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.

© 2025 The Author(s). *Veterinary Record Case Reports* published by John Wiley & Sons Ltd on behalf of British Veterinary Association.

of a concentrated antifreeze solution from a puddle from the garage floor approximately 2 h prior. The exact volume was unknown. The relevant medical history shows a T-cell lymphoma diagnosed 8 months ago which was in stage 4a at the time of presentation and was being treated with prednisolone (1 mg/kg PO q24, manufacturer unknown). On presentation, the ferret showed an ataxic gait and vital signs were normal. According to the medical history, the ferret showed no clinical abnormalities prior to the intoxication. At the time of the presentation, a referral to a specialised small animal clinic was advised, which was not desired by the owner. A complete blood count (blood cell count, blood chemistry, electrolytes), a complete urinalysis (urine status, urine sediment) and an ultrasound of the abdomen were recommended, and the owner opted for a blood cell count, a blood chemistry, a urinalysis and an ultrasound of the abdomen.

INVESTIGATIONS

On clinical examination at presentation, heart rate (reference range 200–400 beats/min), respiratory rate (reference range 33–36 breaths/min), and body temperature (reference range 37.8°C–40°C) were normal,¹⁶ peripheral lymph nodes were physiologic, and the gait was ataxic. The ferret showed an independent intake of food and water. For subsequent therapy and blood sample collection for the first blood test on day 0, a venous catheter (VasoVet 0.9 × 25 mm (22G) B. Braun) was inserted into the cephalic vein under manual restraint. A blood tube with the anticoagulant ethylenediaminetetraacetic acid (EDTA) (microtube EDTA K3E, 1.3 mL, snap cap, EU, SARSTEDT AG & Co. KG) for the blood count and a tube with the anticoagulant lithium heparin (Li-Hep) (microtube lithium heparin LH, 1.3 mL, push-in cap, EU, SARSTEDT AG & Co. KG) for the blood chemistry were obtained from the venous catheter. The initial laboratory examination showed leukocytosis, thrombocytopenia and hypoglycaemia, but no azotemia. The complete results of the blood test can be found in Table 1.

The abdominal ultrasound examination at presentation under manual restraint showed a physiological image of the kidneys, as well as various multifocal round structures, which were addressed by the examining veterinarian as enlarged lymph nodes.

DIFFERENTIAL DIAGNOSIS

The known lymphoma of this ferret is a possible differential diagnosis for the renal failure and neurological signs, but less likely based on the owner's observation of the antifreeze ingestion.

TREATMENT

The ferret was hospitalised for further therapy (day 0) and showed independent food intake as well as good urine output determined subjectively via absorbent cage liners. Due to the observed intoxication with ethylene glycol, infusion therapy was administered with a crystalloid infusion solution (Ringer Lactat n.Hartm.B.Braun Ecoflasche Plus Infusionslö-

LEARNING POINTS/TAKE-HOME MESSAGES

- Ethylene glycol is found in many households and tastes sweet, so it is easy to poison curious animals and a possible intoxication should be asked about in the anamnesis.
- If intoxication is suspected or certain, a complete blood count, creatinine, urea and a urine test with urine sediment should be carried out. Attention should be paid to calcium monooxalate crystals in the urine.
- During therapy, in addition to the clinical condition (vital signs, behaviour, consciousness, gastrointestinal symptoms), the laboratory values and urine output (ideally in/out protocol) should be monitored to observe the course of therapy and to be able to escalate therapy if the clinical condition or laboratory values deteriorate.
- If intoxication is suspected, treatment should be started immediately as the toxic metabolites are formed very quickly by alcohol dehydrogenase and further laboratory tests (blood count, blood chemistry, rapid ethylene glycol test, urinalysis) should be initiated at the same time.
- The best treatment is prevention, because prognosis of ethylene glycol intoxication is poor.

sung, B. Braun) in which ethanol (Alkohol 95% Infusionslösungskonzentrat, B. Braun) was diluted to 30%. Initially, with a bolus (1.3 mL/kg IV) followed by a continuous drip infusion (10.2 mL/kg/day). This was supplemented with forced diuresis using furosemide (2 mg/kg SC q 12 h; Dimazon 50 mg/mL injection solution, Intervet) and antiemetic therapy using metoclopramide (0.4 mg/kg SC q 6 h; Emeprid 5 mg/mL injection solution, CEVA-Tiergesundheit). On the following day (day 1), the heart rate, respiratory rate and body temperature were in the physiological range and the gait pattern remained ataxic, but apathy and anorexia were new symptoms. Urine output was reduced compared to the previous day. A blood test revealed mild azotemia, leukocytosis and thrombocytopenia could no longer be detected, and hypoglycaemia was persistent. The therapy with continuous drip infusion and the administration of metoclopramide was continued. Due to the increasing creatinine, the oral administration of the prednisolone, which was given as long-term medication for the treatment of lymphoma, was discontinued. In the case of the new onset of anorexia, assisted feeding was started with a high-calorie diet mixed with water (25 g/kg/day dry matter mixed with 100 mL water; CONVALESCENCE SUPPORT INSTANT DIET, Royal Canin), with the total daily volume divided into several small portions per day. On the morning of the second day (day 2) after hospitalisation, the ferret was somnolent and hypothermic (37.2°C) with clear signs of nausea, such as salivation, smacking and vomiting. A laboratory check showed progression of azotemia, as well as a specific urine weight below the reference range and haematuria. The detectable urine volume was again reduced for the previous day. A warming mat was placed in the cage to treat the hypothermia and

TABLE 1 Haematology, blood chemistry and urinalysis from an 8-year-old, castrated male ferret after ethylene glycol intoxication.

Parameter	Reference range	Day 0 (19:00)	Day 1	Day 2 (10:36)	Day 2 (19:45)
Haematology					
Erythrocytes (T/L)	6.35–11.20	7.48	7.13	n.d.	6.45
Haematocrit (L/L)	0.37–0.55	0.396	0.38	n.d.	0.35 (L)
Haemoglobin (mmol/L)	6.83–10.55	8.32	7.94	n.d.	7.07
White blood cells (G/L)	2.0–10.0	11.42 (H)	9.56	n.d.	6.25
Neutrophils (G/L)	0.62–3.30	4.43 (H)	4.3 (H)	n.d.	2.64
Lymphocytes (G/L)	1.00–8.00	4.25	2.82	n.d.	2.47
Monocytes (G/L)	0.18–0.90	2.05 (H)	1.85 (H)	n.d.	0.81
Platelets (G/L)	270–880	212 (L)	464	n.d.	709
Blood chemistry					
Creatinine ($\mu\text{mol/L}$)	35–80	72	85 (H)	131 (H)	291 (H)
Blood urea nitrogen (mmol/L)	3.6–16.1	9.3	10.0	12.2	18.3 (H)
Blood urea nitrogen/creatinine quotient	n.d.	33	28	23	15
Symmetric dimethylarginine ($\mu\text{g/dL}$)	2.56–18.41 ²³	19 (H)	14	15	21 (H)
Anorganic phosphate (mmol/L)	1.55–2.87	n.d.	2.15	1.89	3.97 (H)
Total calcium (mmol/L)	2.00–2.95	n.d.	n.d.	n.d.	1.91 (L)
Glucose (mmol/L)	5.23–11.51	4.70 (L)	4.49 (L)	4.28 (L)	5.57
Total protein (g/L)	52–73	70	68	65	64
Albumin (g/L)	26–38	32	32	32	32
Sodium (mmol/L)	140.1–169.7	n.d.	n.d.	n.d.	153
Potassium (mmol/L)	4.6–7.6	n.d.	n.d.	n.d.	5.3
Urinalysis, cystocentesis					
Urine-specific gravity	1.034–1.070 ²⁹	n.d.	n.d.	1.020 (L)	1.015 (L)
Glucose		Negative	n.d.	+ (H)	+ (H)
Protein		+	n.d.	+	+
Blood		Negative	n.d.	++ (H)	+++ (H)

Note: Inhouse-diagnostic: IDEXX-Lasercyte (haematology) and IDEXX-Catalyst-Dx (blood chemistry). Urinalysis (Combur 9 Test 50 Stück-Roche Diagnostics, urine-specific gravity measured by handheld refractometer). Parameters in bold and marked with (L) are below the reference range. Parameters in bold and marked with (H) are above the reference range. Abbreviation: n.d., not determined.

the therapy already used with Maropitant (1 mg/kg SC q 24 h; Cerenia 10 mg/mL injection solution, Zoetis) was supplemented; the observed emesis improved under the adapted antiemetic therapy. During the evening, there was a deterioration in the general condition with a renewed worsening of somnolence and a new laboratory examination showed a significant progression of azotemia and, for the first time, hyperphosphataemia, hypocalcaemia and mild anaemia. The specific gravity of the urine had decreased further in comparison to the morning and haematuria increased. The urine output subjectively continued to decrease throughout the day and was classified as oliguria, which was confirmed by an ultrasound examination under manual restraint of the urogenital tract where the kidneys showed hyperechogenicity with no identifiable border between the renal cortex and the renal medulla (Figures 1 and 2) and striated, mineral-dense structures in the expected area of the renal medulla. The urinary bladder was only slightly filled with multifocal, crystalloid, hyperechogenic structures, which were most likely calcium oxalate monocrystals (Figure 3). The therapy was not adjusted, hyperhydration was unlikely for the treating veterinarian, as the animal did not show any significant deviations in body weight or develop peripheral edema. The owner was informed of the renewed deterioration and euthanasia was discussed.



FIGURE 1 Ultrasonographic image of the left kidney of an 8-year-old, neutered male ferret receiving intravenous drips of ethanol for ethylene glycol poisoning approximately 36 h after ingestion. The border between the renal medulla and cortex was not recognizable (marked with an asterisk).

OUTCOME AND FOLLOW-UP

The ferret was found dead shortly after the last blood test and approximately 48 h after the initial presentation. The body was transferred to pathology and the dissection revealed moderate to high-grade precipitates of crystals in the renal tubules and the vessels of the cerebrum. The precipitates were identified as oxalate crystals on the haematoxylin/eosin-stained overview preparation by polarisation optical birefringence, which are pathognomonic for ethylene glycol.⁵ Furthermore, tumour cells were detected in the lungs, lung

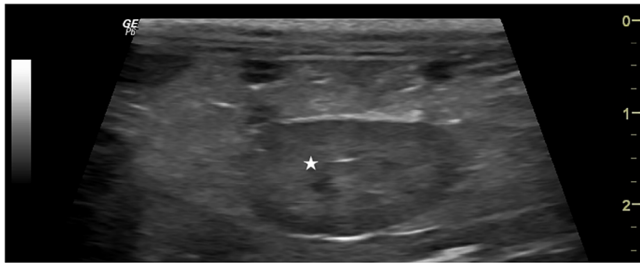


FIGURE 2 Ultrasonographic image of the right kidney of an 8-year-old, neutered male ferret receiving intravenous drips of ethanol for ethylene glycol poisoning approximately 36 h after ingestion. The border between the renal medulla and cortex was not recognizable (marked with an asterisk).

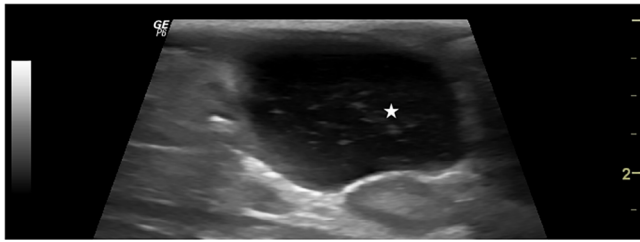


FIGURE 3 Ultrasonographic image of the urinary bladder of an 8-year-old neutered male ferret receiving intravenous ethanol drip therapy approximately 36 h after ethylene glycol poisoning. The urinary bladder was only mildly filled with multifocal, crystalloid, hyperechoic structures that were most likely calcium oxalate monohydrate crystals (marked with an asterisk).

lymph nodes, mesentery, liver, spleen and body lymph nodes, which were positive for CD3 (T-lymphocytes) and negative for CD20 (B-lymphocytes). These results were interpreted as a T-cell lymphoma. The minimally invasive therapy was performed at the owner's request. Transfer to a specialised veterinary hospital or escalation of therapy with dialysis, an in/out protocol for kidney function or fomepizole was discussed with the owner and was explicitly not desired despite the known poor prognosis, also for financial reasons.

DISCUSSION

A literature search revealed that this is only the second case report of intoxication with ethylene glycol (antifreeze) in a ferret.^{17–19} In the first case report of ethylene glycol intoxication in ferrets, the owner was also able to observe their ferrets ingesting the antifreeze, but was able to induce vomiting several minutes after ingestion and administer alcohol (ethanol) orally as recommended by their veterinarian. In the further course of the case, the owner decided against further care by his veterinarian and carried out oral alcohol therapy on his own as instructed, and all the animals survived.²⁰ Since the ferret was not presented for treatment until 2 h after ingestion and was able to consume an unknown amount of concentrated ethylene glycol, it can be assumed that a toxic dose was ingested and that the highest serum concentration had already been reached at the time of presentation and the liver had formed toxic metabolites. Since the minimum lethal dose in cats is 1.4 mL/kg^{6,7} and in dogs 6.6 mL/kg,⁹ the plasma half-life in laboratory animals is about 3 h¹⁹ and the highest serum concentration of ethylene glycol in cats is measured 1 h after

ingestion.⁷ In particular, one group of ferrets survived after direct induction of vomiting and oral alcohol treatment,²⁰ and the ferret presented here developed ARF with a fatal outcome. Clinical signs after oral ingestion of ethylene glycol in dogs, cats and humans are dose- and time-dependent and occur in three clinical stages.^{9,12,15} In stage 1, until 12 h after ingestion thirst,^{3,4} lethargy,^{3,4} vomitus,^{4,21} hypothermia,^{4,21} polydipsia and polyuria,^{3,4,8} ataxia,^{3,4,8,9} convulsions, coma and death⁹ might be present. In stage 2 (12–24 h), tachycardia and tachypnea may additionally occur.^{9,12,15} Stage 3 (24–72 h) is stage in which animals are usually admitted.⁹ Admitting symptoms are anorexia,^{9,15} hypersalivation,¹⁵ uremic halitos,¹⁵ nephralgia,⁴ polydipsia and polyuria^{3,4,8} and oliguria.^{4,8,9} Laboratory changes can be categorised into an early and late phase of ethylene glycol intoxication.¹⁰ In the early phase, an increased anion gap may be seen, and in the late phase, changes occur in response to renal injury and decreased glomerular filtration.¹⁰ Possible laboratory changes include renal azotemia,^{8,9} hyperkalaemia,^{8,9} hyperphosphataemia,⁴ hypocalcaemia,⁴ hypoglycaemia,⁴ hyperglycaemia,⁴ haematuria,⁴ proteinuria,⁴ glucosuria,⁴ decreased urine-specific gravity^{3,4} and calcium oxalate monohydrate crystalluria.^{10,15} The ferret was presented in stage 1 and progressed to stage 3 during therapy. Of the clinical symptoms, ataxia, lethargy, anorexia, hypothermia, vomitus and oliguria could be observed. Of the laboratory changes described, our ferret showed renal azotemia, hyperphosphataemia, hypocalcaemia, hypoglycaemia, proteinuria, glucosuria and haematuria (Table 1).

Symmetric dimethylarginine, which should increase with decreasing glomerular filtration rate,²² is within, or close to, the reference range despite the development of azotemia.²³ It is possible that symmetric dimethylarginine is not yet a reliable parameter for ferrets based on the current data, since larger studies and validation of this parameter are not yet available and no validated commercial test is available for ferrets.²³ Diagnosis of antifreeze poisoning is difficult without observed intake and usually based on the symptoms and laboratory alterations.^{8,9,10,15} The oral cavity and the urine can be examined with a Wood's lamp, as antifreeze products are often mixed with a fluorescent dye, and a positive fluorescence might be observed.¹⁰ Hyperechogenicity of the renal cortex and formation of a hyperechoic band in the medulla are mentioned as typical changes in ultrasound.²⁴ Ethylene glycol can also be detected by tests. An in-house test from Kacey (Kacey EG-3 Antifreeze Test) is available to detect ethylene glycol or its metabolite oxalate. The manufacturer states that this test can detect ethylene glycol from a quantity of 10 mg/dL in a time frame of 45 min–10 h after ingestion and oxalate from 0.25 mg/dL in a time frame of 9–16 h after ingestion. Laboratories offer ethylene glycol analysis from whole blood, plasma or serum. All tests can be false negative if too little ethylene glycol is ingested or too much is metabolised, 24 h after ingestion the tests are usually false negative for ethylene glycol.¹⁵ As this patient had observed ethylene glycol ingestion, no external laboratory analysis was initiated and an in-house test was not available, because such intoxications still occur, it should be considered to have these in stock. The aim of therapy is to stabilise the patient symptomatically, eliminate ethylene glycol from the bloodstream and reversibly block the enzyme ADH in the liver, which metabolises the toxic metabolites, with fomepizole (4-methyl-pyrazole) or ethanol. Ethanol has

a higher affinity for ADH than ethylene glycol and is therefore metabolised first. Fomepizole, on the other hand, is a synthetic, reversible inhibitor of ADH and does not show the typical side effects of ethanol therapy such as nausea, vomiting, apathy and somnolence.^{7,10,13} Furthermore, fomepizole has to be proven to be safe and effective in dogs and cats with a much better prognosis such as ethanol therapy.^{7,13,14} In cats, it was shown that a dose six times higher than that used in dogs is needed to achieve the same response. A single dose of Initial 125 mg/kg followed by 31.25 mg/kg doses at 12, 24 and 36 h was found to be effective and safe.⁷ But fomepizole is not available in most veterinary clinics and pharmacies in Germany to be administered in a timely manner and is associated with a high-cost factor. In addition to ADH-inhibiting therapy with ethanol or fomepizole, haemodialysis and forced diuresis are used to rapidly remove ethylene glycol and its metabolites.^{2,7,8,13,14,25–27} Haemodialysis was not an option for this patient, as no compatible haemodialysis unit was available in the local area. Peritoneal dialysis might be a cost-effective alternative,²⁸ but is not described in ferrets. Fomepizole was not available and peritoneal dialysis was not performed because of the tight budget but should be considered in future cases. Ethanol therapy should always be started if treatment with fomepizole or dialysis is not available or only available later.² In summary, it can be said that this ferret under therapy went through a progression of the stages described above until kidney failure, which was attributed to the late presentation after admission. The owner was informed about the prognosis and, in the further course, also about the development, but did not want any escalation of the therapy and decided against euthanasia until the clear clinical and laboratory deterioration on day 2. Euthanasia was to be carried out on site with the owner after the renewed deterioration on day 2, but the animal died shortly before. For future cases, consideration should be given to how, in the only other case report described, the owner was instructed over the phone to administer an oral dose of ethanol (2 mL per animal of a 45% spirit orally) when ingestion was observed.²⁰ However, it should be noted that there are no reliable dose recommendations for the oral administration of ethanol in ferrets. In dogs, however, a dose of 2.5 mL/kg of a 40% alcoholic beverage has been described, which is repeated after 3, 7, 14 and 24 h.²⁷ If the animal presents without prior ethanol intake and more than an hour has passed since the observed intake, the owners should be directly and critically advised about the possibility of haemodialysis or peritoneal dialysis in addition to the symptomatic therapy. If these measures are not desired, and if the disease progresses negatively during therapy, euthanasia should be discussed with the owner in extremely critical terms. Many dogs and cats die or are euthanased due to ARF triggered by the metabolites.^{9,10} However, prognosis of ethylene glycol intoxication is poor.^{7,13}

AUTHOR CONTRIBUTIONS

Sören Nieweg has written the article. Sebastian Cichowski collected the data. Sören Nieweg and Kerstin Müller conceived and designed the project. Sören Nieweg, Sebastian Cichowski and Kerstin Müller analysed and interpreted the data.

ACKNOWLEDGEMENTS

Open access funding enabled and organized by Projekt DEAL.

CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

FUNDING INFORMATION

No funding was provided for this case report.

ETHICS STATEMENT

This is a case report on a patient from a veterinary practice. It is not an animal study and accordingly no ethics application was submitted to the competent authority in advance.

ORCID

Sören Nieweg  <https://orcid.org/0000-0002-4527-9442>

Kerstin Müller  <https://orcid.org/0000-0003-0618-6961>

REFERENCES

1. Khan SA, Schell MM, Trammel HL, Hansen SR, Knight MW. Ethylene glycol exposures managed by the ASPCA National Animal Poison Control Center from July 1995 to December 1997. *Vet Hum Toxicol.* 1999;41:403–6.
2. Drick N, Schmidt J, Wiesner O, Kielstein J. Fomepizole, ethanol or dialysis in the case of life-threatening ethylenglycol intoxication? *Med Klin Intensivmed Notfmed.* 2021;116:698–701.
3. Grauer GF, Thrall MA, Henre BA, Grauer RM, Hamar DW. Early clinicopathologic findings in dogs ingesting ethylene glycol. *Am J Vet Res.* 1984;45:2299–303.
4. Thrall MA, Grauer GF, Mero KN. Clinicopathologic findings in dogs and cats with ethylene glycol intoxication. *J Am Vet Med Assoc.* 1984;184:37–41.
5. Amoroso L, Cocumelli C, Bruni G, Brozzi A, Tancredi F, Grifoni G, et al. Ethylene glycol toxicity: a retrospective pathological study in cats. *Vet Italiana.* 2017;53:251–54.
6. Parry MF, Wallach R. Ethylene glycol poisoning. *Am J Med.* 1974;57:143–50.
7. Connally HE, Thrall MA, Hamar DW. Safety and efficacy of high-dose fomepizole compared with ethanol as therapy for ethylene glycol intoxication in cats. *J Vet Emerg Crit Care.* 2010;20:191–206.
8. Schweighauser A, Francey T. Ethylene glycol poisoning in three dogs: Importance of early diagnosis and role of hemodialysis as a treatment option. *Arch Svizzero Med Vet.* 2016;158:109–14.
9. Grauer GF, Thrall MA. Ethylene glycol poisoning (antifreeze) in the dog and cat. *J Am Anim Hosp Assoc.* 1982;18:492–97.
10. Thrall MA, Connally HE, Grauer GF, Hamar DW. Ethylene glycol. In: KE Quesenberry, CJ Orcutt, C Mans, JL Carpenter, editors. *Small animal toxicology.* 3rd ed. Elsevier Saunders; 2013. p. 551–67.
11. McMartin K. Are calcium oxalate crystals involved in the mechanism of acute renal failure in ethylene glycol poisoning? *Clin Toxicol.* 2009;47:859–69.
12. Armstrong EJ, Engelhart DA, Jenkins AJ, Balraj EK. Homicidal ethylene glycol intoxication: a report of a case. *Am J Forensic Med Pathol.* 2006;27:151–55.
13. Tart KM, Powell LL. 4-Methylpyrazole as a treatment in naturally occurring ethylene glycol intoxication in cats. *J Vet Emerg Crit Care.* 2011;21:268–72.
14. Connally HE, Thrall MA, Forney SD, Grauer GF, Hamar DW. Safety and efficacy of 4-methylpyrazole for treatment of suspected or confirmed ethylene glycol intoxication in dogs: 107 cases (1983–1995). *J Am Vet Med Assoc.* 1996;209:1880–83.
15. Lee AJ. Chemical toxicoses. In: S Ettinger, E Feldmann, E Côte, editors. *Textbook of veterinary internal medicine expert consult.* 8th ed. Elsevier; 2017. p. 1660–70.
16. Fox JG, Bell JA. Normal clinical and biological parameters. In: JG Fox, RP Marini, editors. *Biology and diseases of the ferret.* Wiley-Blackwell; 2014. p. 157–85.
17. Clarivate. Available from: <https://www.webofscience.com>. Accessed 26 Oct 2024.
18. CABI Digital Library. Available from: <https://www.cabidigitallibrary.org>. Accessed 26 Oct 2024.
19. National Library of Medicine. PubMed. Available from: <https://pubmed.ncbi.nlm.nih.gov>. Accessed 26 Oct 2024.

20. Larsen R. Ethylene glycol toxicosis in multiple ferrets (*Mustela putorius furo*). *Exotic Pet Practice*. 1996;1:7.
21. Hopper K, Epstein SE. Falsely increased plasma lactate concentration due to ethylene glycol poisoning in 2 dogs. *J Vet Emerg Crit Care*. 2013;23:63–67.
22. Sargent HJ, Elliott J, Jepson RE. The new age of renal biomarkers: does SDMA solve all of our problems? *J Small Anim Pract*. 2021;62:71–81.
23. Pazár P, Csöndes J, Abonyi-Tóth Z, Kaba A, Molnár V, Balogh N. Reference intervals for selected blood and urinary parameters related to renal function in clinically healthy ferrets (*Mustela putorius furo*). *J Exot Pet Med*. 2024;50:15–18.
24. Graham JP. Kidneys and proximal ureters. In: F Barr, L Gaschen, editors. *BSAVA manual of canine and feline ultrasonography*. British Small Animal Veterinary Association; 2011. p. 110–23.
25. Beckett SD, Shields RP. Treatment of acute ethylene glycol (antifreeze) toxicosis in the dog. *J Am Vet Med Assoc*. 1971;158:472–76.
26. Nunamaker DM, Medway W, Berg P. Treatment of ethylene glycol poisoning in the dog. *J Am Vet Med Assoc*. 1971;159:310–14.
27. Grauer GF, Thrall MA, Henre BA, Hjelle JJ. Comparison of the effects of ethanol and 4-methylpyrazole on the pharmacokinetics and toxicity of ethylene glycol in the dog. *Toxicol Lett*. 1987;35:307–14.
28. Vale JA, Prior JG, O'Hare JP, Flanagan RJ, Feehally J. Treatment of ethylene glycol poisoning with peritoneal dialysis. *Br Med J (Clin Res Ed)*. 1982;284:557.
29. Eshar D, Wyre NR, Brown DC. Urine specific gravity values in clinically healthy young pet ferrets (*Mustela furo*). *J Small Anim Pract*. 2012;53:115–19.

How to cite this article: Nieweg S, Cichowski S, Müller K. Ethylene glycol intoxication (antifreeze poisoning) in a domestic ferret (*Mustela putorius furo*). *Vet Rec Case Rep*. 2025;13:e70126. <https://doi.org/10.1002/vrc2.70126>

MULTIPLE CHOICE QUESTION

Which urinary stones can typically be detected in ethylene glycol poisoning?

POSSIBLE ANSWERS TO MULTIPLE CHOICE QUESTION

Calcium oxalate monohydrate

Ammonium urate

Struvite

Xanthin

CORRECT ANSWER

The metabolism of ethylene glycol in the liver by alcohol dehydrogenase leads to the formation of metabolites, including oxalic acid, which precipitate in the renal tubules as calcium oxalate monohydrates and are responsible for acute renal insufficiency.