Ciência Animal Brasileira
Brazilian Animal Science

DOI: 10.1590/1809-6891v26e-83346E

e-ISSN 1809-6891
Veterinary medicine | Research article

Chronic kidney disease following poisoning by ethylene glycol in a dog: a case report

Doença renal crônica após intoxicação por etilenoglicol em um cão: um relato de caso

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Received: July 08, 2025. Accepted: October 22, 2025. Published: October 29, 2025. Editor: Luiz Augusto B. Brito

Abstract: A seven-year-old, 4.65 kg, male Shih-tzu dog was referred to the veterinary hospital with a history of vomiting after eating a commercial dog treat containing ethylene glycol (EG) in its formulation. The animal showed clinical signs of kidney damage and after additional tests was diagnosed with severe grade 4 acute kidney injury. Almost one and half years later, the patient was presented to veterinary care, with a grade 4, chronic kidney injury. During this period, several tests were carried out, including a complete blood count, serum biochemistry analysis, abdominal ultrasound, and blood gas analysis. In critical condition, the animal died shortly afterward. An anatomopathological necropsy exam revealed extensive kidney injury with tubular necrosis and glomerular sclerosis, fibrosis, and mineralization, accompanied by numerous mono and dihydrates oxalate crystals. To our knowledge, this is the first report describing clinical signs and pathological findings of a dog poisoned by EG in Brazil. We aim to contribute to the understanding of the pathogenesis of EG poisoning, especially regarding chronic kidney failure.

Keywords: Canine; forensic toxicology; intoxication; kidney injury; toxicologic pathology.

Resumo: Um cão da raça Shih-tzu, macho, com sete anos e pesando 4,65 kg, foi encaminhado ao hospital veterinário com histórico de vômitos após ingerir um petisco comercial para cães contendo etilenoglicol (EG) na formulação. O animal apresentava sinais clínicos característicos de injúria renal e após exames complementares foi diagnosticado com lesão renal aguda severa de grau IV (escala de I-V). Quase um ano e meio depois, o paciente retornou ao atendimento veterinário, apresentando lesão renal crônica de grau IV. Durante esse período, foram realizados vários exames, incluindo hemograma, análise bioquímica sérica, ultrassonografia abdominal e hemogasometria. Em estado crítico, o animal evoluiu a óbito pouco tempo depois. O exame anatomopatológico da necropsia revelou lesões renais extensas com necrose tubular, esclerose glomerular, fibrose e mineralização, acompanhadas por numerosos cristais de oxalato mono e dihidratados. De acordo com o nosso conhecimento, este é o primeiro relato descrevendo os sinais clínicos e achados anatomopatológicos de um cão intoxicado por EG no Brasil. Este estudo busca contribuir para o entendimento da patogênese e diagnóstico clínico da intoxicação por EG, especialmente no que se refere à insuficiência renal crônica.

Palavras-chave: Canino; intoxicação; lesão renal; patologia toxicológica; toxicologia forense.

1. Introduccion

Ethylene glycol (EG) is a synthetic odorless liquid substance used as an antifreeze and vehicle deicing solution. It is also used in hydraulic brake fluids, inks, stamp pads, ballpoint pens, and print shops [1]. EG toxicosis has been reported in humans [2] and animals, including dogs [3], cats [4], chickens [5], calves [6] and wildlife [7]. Poisoning is characterized by severe metabolic acidosis followed by critical acute kidney injury. Clinical signs can include ataxia, vomiting, polyuria, polydipsia, hypothermia, oliguria, anuria, and dehydration [8].

Although ethanol and fomepizole can be used as antidotes since both compete with the liver enzyme alcohol dehydrogenase, mortality rates are still high in humans [9], and little data is found regarding dogs. Most poisonings in animals occur after accidental exposure to antifreeze substances and are more common in northern countries [10]. Here we describe detailed long-term clinical evolution, post-mortem, and histopathological findings of chronic kidney disease (CKD) following EG poisoning in a dog in Brazil, after a serious contamination of treats. We aim to contribute to the understanding of the pathogenesis of EG poisoning, especially regarding chronic kidney failure.

2. Case report

A seven-year-old, 4.65kg, male Shih-tzu was referred to the veterinary hospital following nine days of vomiting after eating a commercial dog treat. He had anorexia, apathy, intermittent diarrhea, increased water and urine intake, and lighter urine color. The owner denied any previous illnesses and confirmed that the animal had been vaccinated, had no access to other animals, and remained indoors. The patient was evaluated throughout approximately one and a half years, including clinical examination, imaging exams, clinical pathology, necropsy and histopathology.

During clinical examination (1st day), the patient had tachycardia (168 bpm), periodontitis, and a rigid abdomen during palpation. All remaining parameters were within the normal limits. Blood samples were collected for blood cell count (CBC), alanine aminotransferase (ALT), creatinine, phosphorus, urea, (Tables 1 and 2), and blood gas. Urine was collected via cystocentesis to perform urinalysis (Table 3). Creatinine levels reached 5.23 mg/dL (0.5 to 1.5 mg/dL), phosphorus was also increased at 6.48 mg/dL (2.6 to 6.3 mg/dL), and urea at 145 mg/dL (21.4 to 59.9 mg/dL)[11]. Urinalysis revealed low specific gravity, proteinuria (one cross), the presence of granular casts, transitional epithelial cells, and oxalate crystals. Blood gas did not reveal acidosis, but an increased CO_2, with a minor reduction of sodium (Na), calcium (Ca), and potassium (K) levels. Other parameters remained within the normal values for the species. Abdominal ultrasound revealed colitis and acute nephropathy (discrete increase in medullary echogenicity).

The diagnosis was an acute renal injury (AKI) [12] at stage four with an unidentified cause. He was then hospitalized for 8 days and treated with fluid replacement, ondansetron, omeprazole, omega-3, aluminum hydroxide, praziquantel, appetite stimulant, pyrantel, febantel, fluid therapy, and gastrointestinal diet (Table 1). Improvement was observed, but vomiting and hyporexia persisted. Nutritional supplementation was recommended after omeprazole. On the 8th day, BC was normal, except for discrete leukopenia (Table 2), whilst creatinine and phosphorus remained elevated, at 3.93 mg/dL and 3.85 mg/dL, respectively (Table 3). Urine continued to present lower density (Table 4). Home treatment was continued after hospital discharge (8th day) (Table 1).

Table 1. Treatments applied to a dog with suspected poisoning by ethylene glycol

Date	Medication/Procedure	Dosage/Frequency	Duration
1st day	Ondansetron	1 mg/kg, IV, three times a day	7 days
	Omeprazole	1 mg/kg, orally, twice a day	7 days
	Fluid therapy with ringer lactate	19 ml/h, IV	-
	Gastrointestinal pate	Twice a day	-
	Urinary output	Every 24 hours	-
2nd day	Omeprazole	1 mg/kg, orally, every 12 hours	10 days
	Ondansetron	1 mg/kg, orally, every 8 hours	5 days
	Omega-3 supplementation	1500 mg, orally, every 36 hours	18 days
	Gastrointestinal diet	In case of food rejection	7 days
3rd day	Cyproheptadine	0.5 mg/kg, orally, every 12 hours	5 days
	Aluminum hydroxide	15 mg/kg, orally, every 12 hours	5 days
	Ambulatory fluid therapy with ringer lactate	150 ml, SC	7 days
8th day	Omeprazole	1 mg/kg, orally, every 12 hours (instruct weaning after improvement in condition)	30 days
	Praziquantel 175 mg, pyrantel palmoate 504 mg, febantel 525 mg	1/2 of a tablet, orally, every 24 hours (repeat with 15 days)	3 days
	Gastrointestinal feed	-	Continuous
	Fluid therapy with ringer lactate	27 ml/h, IV	1 day
	Appetite stimulant (cobanamide 1 mg and cyproheptadine hydrochloride 4 mg)	-	-
18th day	Salute® nutritional supplement	1 sachet, orally, every 24 hours	7 days
520th day	Syrup with cyproheptadine, vitamins B1, B2, B6, C, and nicotinamide	0.1 ml/kg, orally, every 12 hours	7 days
	Endoparasiticide with fenbendazole, pyrantel pamoate, and praziquantel	½ tablet, orally, every 24 hours	3 days
543th day	Fluid therapy with 0.9% NaCl solution	14 ml/h for the first 8 hours, 11 ml/h after evaluation	-
	Ondansetron	1 mg/kg, IV, three times a day	-
	Dipyrone	25 mg/kg, IV, three times a day	-
	Tramadol	4 mg/kg, IV, three times a day	-
	Urinary output	Every 4 hours for the first 12 hours, every 6 hours thereafter	-
	Nasogastric feeding with hypercaloric pate	Every 6 hours	-
	Aluminum hydroxide 6%	30 mg/kg, orally, after feeding	-
545th day	Erythropoietin	100 UI/kg, SC, every 48 hours	
	Hylo-gel® eye drops	1 drop every 8 hours	-

IV: intravenously; SC: subcutaneously.

Table 2. Blood cell count (CBC) of a dog poisoned by ethylene glycol

	BC					
Erythrogram	**1st day**	**18th day**	**520th day**	**543th day**	**545th day**	**REFERENCE VALUES***
Red blood cells	6.34	6.85	2.67	2.05	4.04	5.5 – 8.5 x 106
Hemoglobin	13.7	15	6.0	4.3	8.7	12 – 18 g/dL
Hematocrit	42	45	18	14	27	37 – 55 %
MCV	66.2	65.7	67.4	68.3	66.8	60 – 77 fL
MCHC	32.6	33.3	33.3	30.7	32.2	32 – 36%
RDW	13.6	13.4	12.5	13.4	14.2	12 – 15 %
Total plasma protein	7	6.6	6.6	7.6	6.6	6 - 8 g/dL
Reticulocyte	-				0.2	0 – 1.5%
Platelet count						**REFERENCE VALUES***
Platelet count	440	375	446	545	224	200 - 500 x10³/uL
MPV	6.0	6.0	5.7	5.7	6	
PDW	15.4	14.9	14.5	14.6	15.5	%
Leukogram						**REFERENCE VALUES***
Leukocytes	16,000	5,300	4,200	7,500	6,900	6,000 – 17,000 x10³/uL
Myelocytes	0	0	0	0	0	0
Metamyelocytes	0	0	0	0	0	0
Band neutrophil	0	0	0	0	0	0 – 300
Segmented neutrophil	14,560	3,816	3,864	6,600	6,486	3,000 – 11,500
Eosinophil	160	530	168	0	138	150 – 1,250 mm³
Basophil	0	0	0	0	0	0
Lymphocytes	320	795	165	225	138	1,000 – 4,800 mm³
Monocytes	960	159	0	675	138	150 -1,350 mm³

*Jain, 1993 [13]. MCV: Mean Corpuscular Volume; MCHC: Mean Corpuscular Hemoglobin Concentration; MPV: Mean Platelet Volume; RDW: Red Cell Distribution Width; PDW: Platelet Distribution Width.

Table 3. Serum biochemistry analysis of a dog poisoned by ethylene glycol

BIOCHEMISTRY							
Parameters	**1st day**	**8th day**	**18th day**	**520th day**	**543th day**	**545th day**	**Reference Values***
ALT	63	-	74	91	-	-	21 – 86 UI/L
Creatinine	5.23	3.93	3.84	7.77	-	11.59	0.5 - 1.4 mg/dL**
Phosphorus	6.48	3.85	4.15	-	28.20	-	2.6 – 6 mg/dL
Urea	145	-	96	254	504	-	21.4 – 59.9 mg/dL
ALP	-	-	45	-	53	-	20 – 156 UI/dL
Lactate	-	-	-	-	-	0.55	0.5 - 2 mmol/L****
TOTAL SERUM PROTEIN AND FRACTIONS							
Total serum protein	5.9	-	6.5	-	-	-	5.4 – 7.1 g/dL
Albumin	3.4	-	3.3	-	-	-	2.6 - 3.3 g/dL
Globulins	2.5	-	3.2	-	-	-	2.7 - 4.4 g/dL
LIPIDOGRAM							
Total cholesterol	-	-	-	-	-	207	135-270 mg/dL
HDL Cholesterol	-	-	-	-	-	64	33-120 mg/dL
LDL Cholesterol	-	-	-	-	-	132	58-187 mg/dL
VLDL Cholesterol	-	-	-	-	-	11	6.5 - 16.9 mg/dL
Triglycerides	-	-	-	-	-	55	20 - 112 mg/dL

*Kaneko *et al*., 2008[11]; ** IRIS, 2023[14] (consensus CKD staging); *** IRIS, 2023[15] (consensus CKD treatment); **Silverstein, 2015[16]; ALT: alanine aminotransferase; ALP: Alkaline Phosphatase; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very low-density lipoprotein.

Table 4. Urinalysis of a dog poisoned by ethylene glycol

URINALYSIS				
	1st day	**8th day**	**18th day**	**REFERENCE VALUES***
Color	Light yellow	Light yellow	Light yellow	Citrine yellow
Odor	Sui Generis	Sui Generis	Sui generis	Sui Generis
Aspect	Cloudy	Clear	Clear	Clear
Specific gravity	1,012	1,010	1,014	1,015 – 1,045
pH	6.0	5.0	7.0	5.0 – 7.0
Proteins	+	Negative	+	<30 mg/dL
Glucose	Negative	Negative	Negative	<40 mg/dL
Ketone bodies	Negative	Negative	Negative	Negative
Urobilinogen	Normal	Normal	Normal	Normal
Bilirubin	Negative	Negative	Negative	Negative
Occult blood	+++	Negative	Negative	Negative
Nitrite	Negative	Negative	Negative	Negative
Bile salts	Negative	Negative	Negative	Negative
Sediment				
Red blood cell	40 - 50/camp	0 – 5/camp	1 - 5/camp	<5 camp
Leukocytes	0 - 3/camp	1 – 4/camp	0 - 3/camp	<5 camp
Desquamation cells	0 - 2/camp	0 – 1/camp	0 - 1/camp	Rare
Transitional cells	0 - 6/camp	0 – 3/camp	0 - 2/camp	Rare
Microbiome	Rare	Rare	Rare	Absent
Pelvic kidney cells	0/camp	0/camp	0/camp	Absent
String of mucus	Absent	Present +	Absent	Absent
Casts	Granular 0 - 2/camp	0/camp	Granular 0 - 1/camp	Absent
Crystals	Calcium oxalate +++	Absent	Absent	Absent
Sperm	Present ++	Present +++	Present +	Absent
Observation	Transition cells clusters +	Mucus +		

* Sink *et al*., 2012[17]. Sedimentoscopy was performed with a 40x objective.

A few days after the first presentation at the veterinary hospital, Brazil's National Health Surveillance Agency (Anvisa) and Ministry of Agriculture and Livestock (MAPA) revealed an outbreak of EG poisoning from different dog treat products. Two lots were diagnosed with propylene glycol contaminated with monoethylene glycol (AD5035C22 and AD4055C21) [18]. Several cases throughout the country were reported in 2022, with over 100 canine suspicious poisoning in 14 states, currently under federal investigation. In the present report, the animal had eaten a whole package of dry snacks. The patient was reevaluated after 10 days of hospitalization (18th day). During the time, the owner presented to the veterinary team, a package of dog treats from one of the brands and batches that were under investigation. The cause of AKI was clinically identified as EG poisoning.

Clinical presentation on the 18th day included persistent hyporexia, decreased defecation, and polydipsia. Blood and urine samples were again evaluated. BC revealed leukopenia and normochromic normocytic anemia. ALT and ALP (alkaline phosphatase) remained normal, while the following parameters were elevated: creatinine (3.84 mg/dL- 0.5 to 1.5 mg/dL), phosphorus (4.15 mg/dL - 2.6 to 6.3 mg/dL) and urea (96 mg/dL 21.4 to 59.9 mg/dL) [11]. Bilirubin and fractions remained within the normal limits (data not shown). Urine revealed discrete low specific gravity and the presence of cylinders' cells (Table 3). Nutritional supplementation was recommended as well as a consultation with the veterinary nephrologist; however, the patient did not return.

After almost 17 months of the first hospitalization (520th day), the owner reported that the patient had been experiencing weight loss and loss of appetite for a month. One day before the appointment, the patient began to experience vomiting and tremors. Physical examination revealed pale mucous membranes, tachycardia (160 bpm), and popliteal lymph nodes lymphadenomegaly. Body condition score was 3/9, with significant cachexia and severe periodontitis. Other parameters were within normal limits. Additional tests were requested, including CBC, ALT, creatinine, urea, phosphorus, and abdominal ultrasound. Results showed that the patient had severe normochromic normocytic anemia, with creatinine value of 7.7 mg/dL (0.5 to 1.5 mg/dL) and urea of 254 mg/dL 21.4 to 59.9 mg/dL) [14] (Table 1 and 2). Abdominal ultrasound showed both kidneys with reduced size, irregular contour, diffuse hyperechogenicity of the cortical region, and homogeneous echotexture, with slight loss of definition and alteration of the corticomedullary relationship, presence of a discrete echogenic halo in the medullary region parallel to the corticomedullary junction (medullary sign). Discreet hyperechogenic foci were noted in the topography of pelvic recesses (mineralizations) (Figure 1). In addition, signs of gastric hypersecretion/inflammatory gastropathy and prostatic hyperplasia were observed. The dog was diagnosed with stage four CKD according to IRIS, 2023 [14]. The veterinary team informed the owner of the severity of the case and the need for additional tests, including blood gas analysis and urinalysis, as well as hospitalization and a consultation with a specialized nephrologist. The owner denied the request.

Seven days after the second consultation (527th day) the patient presented clinical worsening, including inappetence, hypocolored mucosa, cachexia, anorexia, adipsia, oliguria, purulent secretion in both eyes, pain on abdominal palpation, 8% dehydration, uremic breath with visible oral ulcers and instability of the incisor teeth in the jaw. The patient was hospitalized and underwent follow-up in the nephrology sector. Blood samples were evaluated and findings included hypochromic normocytic anemia (Table 1), severe hyperphosphatemia, and uremia (Table 3). Blood gas analysis revealed metabolic acidosis without significant electrolyte changes. The recommended treatment included fluid replacement, antiemetic, phosphorus chelator, analgesia, and insertion of a nasogastric tube for feeding. In addition, a urethral probe was inserted to measure urinary output.

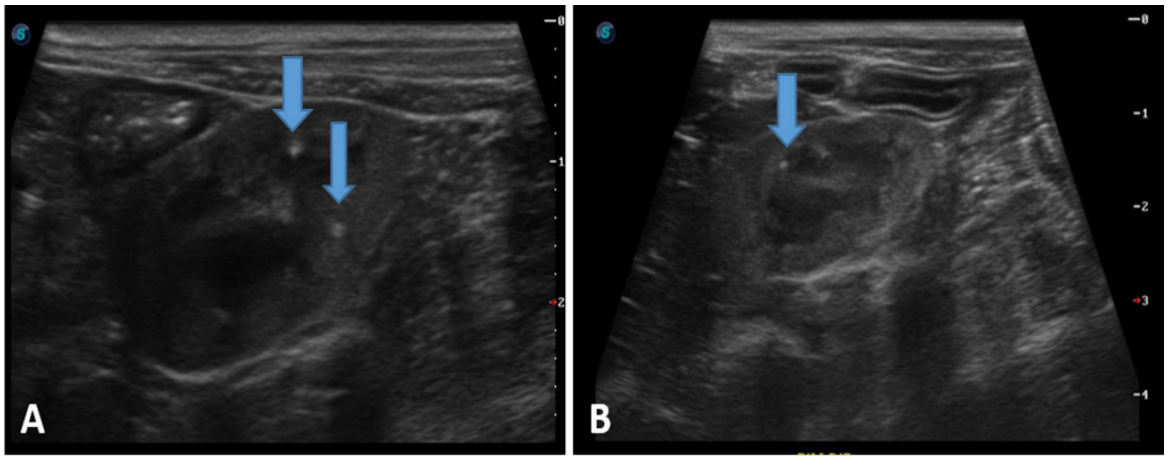

Figure 1. Abdominal ultrasound showed both kidneys with reduced size, irregular contour, diffuse hyperechogenicity of the cortical region, and homogeneous echotexture, with slight loss of definition and alteration of the corticomedullary relationship, presence of a discrete echogenic halo in the medullary region parallel to the corticomedullary junction (medullary sign). Discreet hyperechogenic foci were noted in the topography of pelvic recesses (mineralizations; arrow).

Due to severe anemia, a blood transfusion with packed red blood cells was performed. The procedure occurred without complications and on the 545th day, new tests were carried out which showed an increase in hematocrit (Table 1) and persistence of uremia (Table 2). Erythropoietin and lubricating eye drops were added to the prescription, however, on day 546 the patient died.

During the necropsy, the body condition score was 2/5. Ocular and oral mucous membranes were pale and there were ulcers in the latter. The tongue also presented ulceration in the ventral and lateral regions. When the abdominal cavity was opened, there was serosanguineous fluid, the liver was enlarged and the gastric mucosa contained multiple ulcers. Kidneys had marked hypotrophy, and the urinary bladder had an ulcerated focus on the mucosa. Lungs had diffuse congestion and discrete multifocal hemorrhages, in addition to the presence of foamy fluid in the bronchi and trachea, indicating pulmonary edema. Heart's left ventricle showed concentric hypertrophy, congestion, and hemorrhagic areas (Figura 2).

Histopathological analysis allowed us to confirm the macroscopic findings of the digestive and respiratory systems. Furthermore, foci of mineralization were observed in several organs, such as the heart, liver, stomach, intestine, pancreas, spleen, meninges, and especially the kidney. In the kidneys, it was still possible to notice moderate multifocal congestion, extensive fibroplasia, and moderate multifocal infiltrate of lymphocytes and plasma cells; as well as mineralization in collecting, proximal and distal tubules, and often basement membranes. Birefringent oxalate crystals were also found in the tubules. Furthermore, there was a desquamation of epithelial cells towards the luminal interior associated with tubular necrosis and a marked number of sclerotic glomeruli (Figure 3).

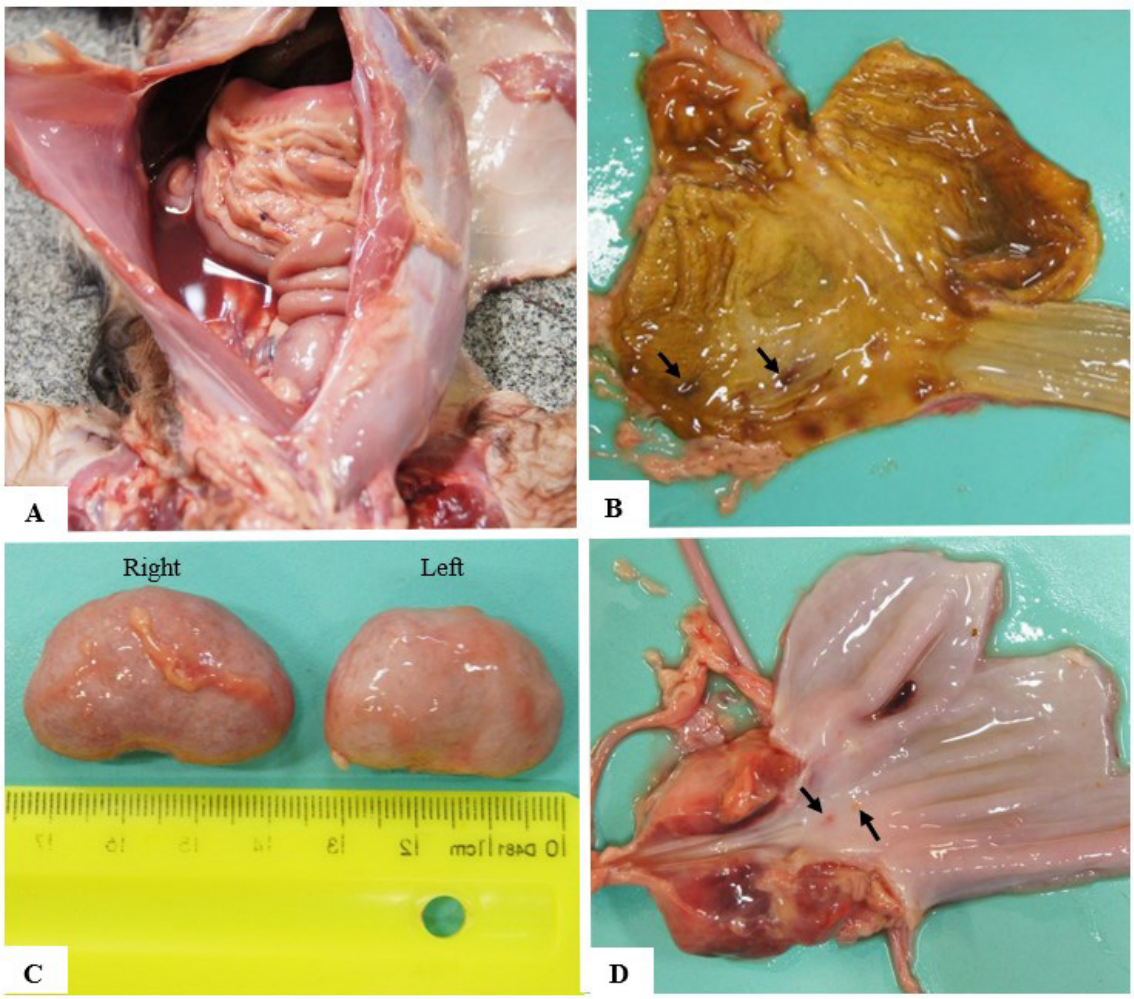

Figure 2. Macroscopic images from the necropsy of a dog with suspected ethylene glycol poisoning. During the necropsy, the body condition score was 2/5. A. Presence of serosanguineous fluid in the abdominal cavity; B. Multiple ulcers in the gastric mucosa (black arrow). C. Kidneys had marked hypotrophy and irregular capsule. D. urinary bladder had an ulcerated focus on the mucosa (black arrows).

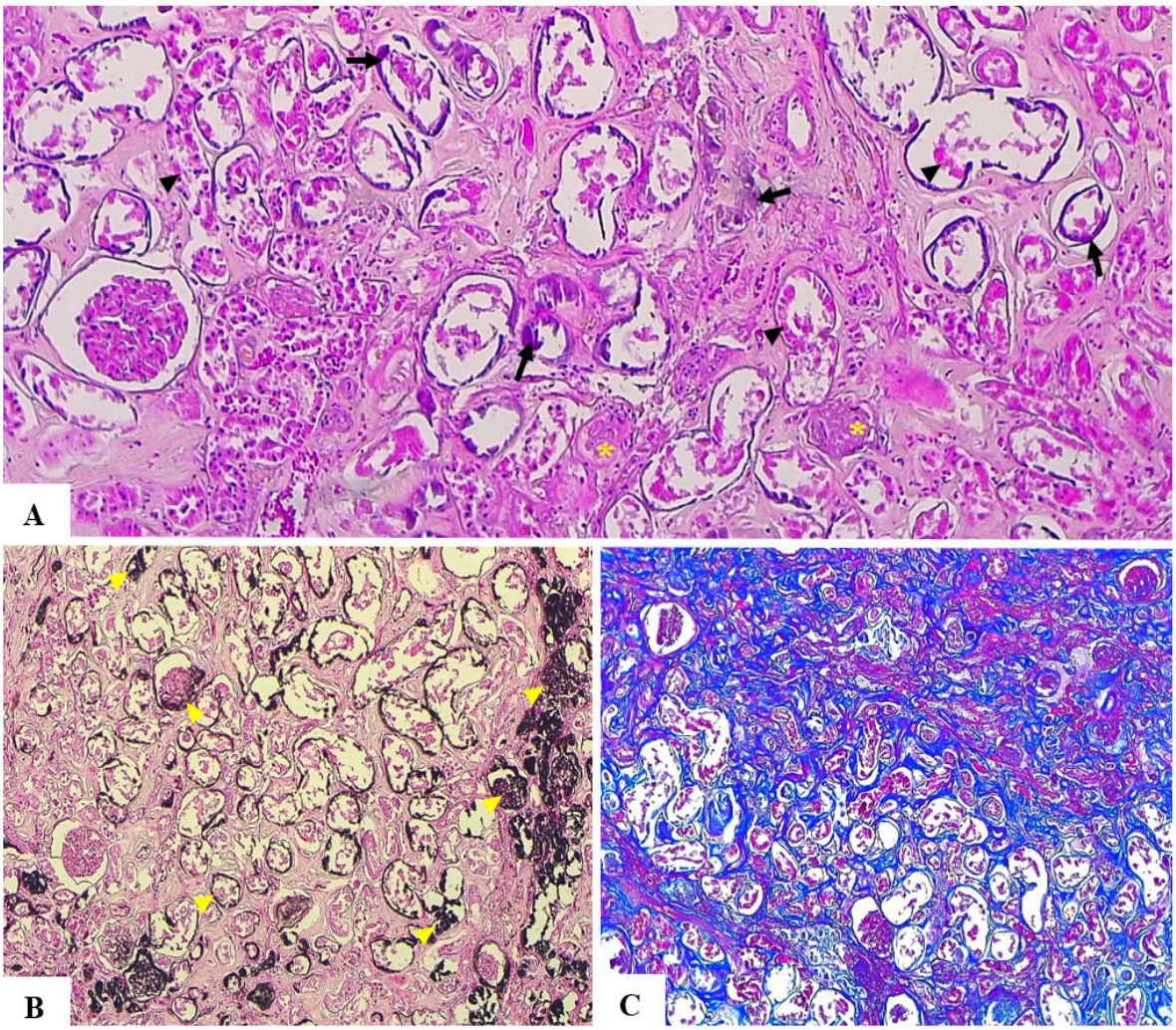

Figure 3. Photomicrograph of kidney from a dog poisoned by ethylene glycol. A) desquamation and necrosis of tubular epithelial cells (arrowhead) and sclerotic glomeruli (yellow asterisk). Birefringent oxalate crystals (arrow) in collecting, proximal and distal tubules, and basement membranes. HE, 10x. B) Oxalate crystals deposition (yellow arrowhead), which is shown in black, in proximal and distal tubules, and basement membranes. Von Kossa, 10x. C) Extensive fibroplasia in renal parenchyma (cortex renal), shown in blue. Masson's trichrome, 10x.

3. Discussion

The present work describes detailed clinical and pathological findings of chronic kidney failure following EG poisoning in a dog in Brazil, after a serious contamination of treats. Once ingested, EG goes through an extensive metabolization process that results mainly in oxalic acid, the major nephrotoxic metabolite [19]. Poisoning in dogs can cause three stages of clinical presentations. Stage I occurs between 30 min and 12 hours of the exposure, with ataxia, vomiting, polyuria, polydipsia, and hypothermia. Laboratory tests indicate metabolic acidosis, hyperosmolality, high osmolar gap, high anion gap (AG), prerenal azotemia, and calcium oxalate crystaluria. Stage II has cardiopulmonary manifestations that usually appear between 12 and 24 h, but are rarely recognized in dogs. Stage III has acute kidney injury or oligoanuric acute renal failure, with renal azotemia and hyperkalemia, that are manifested after 24 to 72 h [8]. The patient in the present study was hospitalized with clinical signs and laboratory findings compatible with stage III of the poisoning. In the present case, the most prominent findings were vomiting and azotemia.

Acute kidney injury was confirmed by a combination of exams, including high azotemia, high phosphorus, urinalysis alterations, and abdominal ultrasound. It is important to highlight the relevance of urinalysis in this diagnosis, with a remarkable presence of oxalate crystals that are typical of EG poisoning [20]. These crystals are the result of liver metabolism of oxalic acid and cause direct toxicity to kidney cell mitochondria.

Initially, the patient was diagnosed with stage four acute kidney injury [12], without probable cause. Only after the outbreak was reported by Brazilian officials, that a suspicion of EG was considered. This fact can be associated with the uniqueness of the contamination that is under investigation, with a possible replacement of propylene glycol with diethylene glycol, and the difficulty in establishing a diagnosis [8]. Several reports claim the importance of early diagnosis, highlighting decontamination and antidotal therapy with fomepizole [21]. Due to delayed diagnosis, antidotes were not prescribed in the present case and only symptomatic treatments were performed.

Despite recommendations for continuous monitoring, the patient only returned to veterinary care after 17 months. Clinical signs included tremors, loss of appetite, vomiting, pale mucous membranes, periodontitis, and cachexia, all commonly described in chronic diseases, including kidney failure [3]. Further exams confirmed severe normochromic normocytic anemia, azotemia, and ultrasound findings compatible with stage four CKD [14]. It is important to highlight the relevance of clinical monitoring since CKD is diagnosed.

The severity of anemia in the present case was remarkable, and treatment with blood transfusion was only partially successful. Anemia in patients with chronic kidney disease is multifactorial and includes decreased erythroid precursor cells, gastrointestinal bleeding, nutritional imbalance, iron deficiency, and systemic inflammation. It is believed the critical failing kidneys produce insufficient amounts of erythropoietin [12], with increased concentrations of parathormone and tumor necrosis factor-α (TNFα), that altogether induce erythroid hypoplasia [22].

An important continuous alteration was azotemia. Urea and creatinine are known biomarkers for kidney disease, increasing in cases of filtration insufficiency. However, it is important to highlight the fact that creatinine reached 11.59 mg/dL in a dog with severe cachexia, indicating this value is even more critical. Creatinine is mostly produced by muscle cells and atrophy found in cachexia can decrease blood levels [14]. This indicates that the kidney dysfunction in our patient was most likely terminal. Clinical signs worsened during the last days of monitoring, with adipsia, oliguria dehydration, and oral erosions, compatible with uremia.

Abdominal ultrasound also confirmed the progression of kidney diseases. The most common findings in dogs are increased cortical echogenicity, abnormal cortical/medullar junction, and pyelectasis [23]. Similar alterations were diagnosed in the present case, with the addition of discrete hyperechogenic foci in the topography of pelvic recesses, later diagnosed by histological analysis as mineralization areas.

Necropsy findings included significant kidney alterations with consequences to other tissues, including the tongue, stomach, and heart. Tubular necrosis and desquamation of epithelial cells, leading to the formation of cellular casts, are common alterations described in previous reports [20]. The presence of birefringent oxalate crystals is an important indication of EG poisoning [6] and corroborates with previous urinalysis results. It is also important to highlight that the degree of renal necrosis is not necessarily associated with the number of oxalate crystals present in the organ, since it is believed that kidney injury is also caused by the direct cytotoxic effect of metabolites of hippuric or glyoxylic acid. Sclerotic glomeruli are an unusual lesion found in the present case [24] and may be directly related to interstitial injury to the kidney.

Systemic reverberation of kidney disease included serosanguineous fluid [25] and tongue necrosis, common in cases of chronic renal failure due to uremia, which promotes fibrinoid necrosis of the blood vessel wall, causing thrombosis, ischemia, and consequently ulceration of the affected organ [25]. Circulatory changes, such as congestion and hemorrhagic foci in various internal organs, are also a consequence of EG poisoning [24]. As noted, oxalate crystals can precipitate in practically all tissues of the animal's body [24] and accompany the mineralization of tissues; these findings have also been observed in other cases of EG poisoning in animals and humans [26].

4. Conclusion

This is the first report of EG poisoning in a dog in Brazil and one rare case that possibly evolved into CKD. It is important to highlight that other unknown factors might be involved in the pathogenesis of CKD. Through comprehensive clinical monitoring and laboratory, ultrasound, and anatomopathological examinations, it was possible to document the progression from severe acute kidney injury to chronic kidney disease, highlighting the severity and complexity of EG poisoning in dogs. This report aims to contribute to the understanding of the pathogenesis of EG intoxication, to further explore veterinary diagnosis, and the importance of clinical pathology monitoring. We would also like to highlight the relevance of quality control regarding animal feed to avoid such critical contaminations.

Conflicts of interest statement

The authors declare no conflicts of interest.

Data availability statement

The complete dataset supporting the results of this study is available on request from the corresponding author.

Author contributions

Conceptualization: A.F.M. Botelho. Methodology: A.F.M. Botelho and D.B. Martins. Ivestigation: A.C.M. Barros and M.C. de Vicente. Formal analysis: A.F.M. Botelho, A.P.I. Santin and D.B. Martins. Writing (original draf): A.C.M. Barros, M.C. de Vicente, M. R. de souza and A.R.R. Guimaraes. Writing (review and editing): A.F.M Botelho and D.B. Martins. Supervision: A.F.M. Botelho and D.B. Martins. All authors have read and approved the fnial version of the manuscript.

Acknowledgements

We thank the following colleagues for tube contributions regarding clinical management and pathology: Laís Di Paulie Taborda Prado, Fernanda Martins Paixão, Caroline Cavalcante Rodrigues, Nelcimara Mirley de Souza Correia, Nadjanaira Barbosa Abrão, Luana de Sousa Ribeiro, Heloisa Alvim Rodrigues Fraga, Amanda Lopes Ribeiro, Washington Cleyber de Oliveira Júnior, Filipe Augusto Vieira dos Santos, Bruna Siqueira Gomes de Lima.

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