Scientific Works. Series C. Veterinary Medicine. Vol. LXXI (1), 2025
ISSN 2065-1295; ISSN 2343-9394 (CD-ROM); ISSN 2067-3663 (Online); ISSN-L 2065-1295

EMERGENCY MANAGEMENT IN A DOG WITH ETHYLENE GLYCOL INTOXICATION: CASE REPORT

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Abstract

A 1-year-old, 20 kg, intact female mixed-breed dog was admitted to the clinic for a nephrology consultation following ethylene glycol ingestion. Upon examination, the patient displayed severe hypertension (202/154 to 205/156 mmHg, measured by oscillometric method), abdominal pain, conjunctivitis, advanced dehydration, a rectal body temperature of 38.9°C, and dry mucous membranes. Laboratory tests revealed elevated BUN 151 mg/dL, CRE 10.2 mg/dL, PHOS 9.9 mg/dL, and GLU 123 mg/dL. Urinalysis showed a borderline proteinuria (UPC 0.5-2.0), pH of 5.0, and microalbumin ≥25 mg/L. To preserve renal function, haemodialysis was indicated as extracorporeal renal replacement therapy. Over eight days, four haemodialysis sessions were performed following the placement of a central venous catheter under light sedation with oxygen therapy supplementation. These therapeutic interventions, including intensive fluid management, were crucial in improving renal function. Significant biochemical improvements were observed: BUN decreased to 66 mg/dL, CRE to 3.7 mg/dL and PHOS to 8.0 mg/dL. Additionally, Ca levels rose from 11.3 mg/dL to 13.5 mg/dL. This case emphasizes the importance of timely intervention in acute kidney injury following ethylene glycol toxicity. Continued monitoring is essential for long-term renal recovery.

Key words*: BUN, CREA, haemodialysis, canine, ethylene glycol.*

INTRODUCTION

Ethylene glycol (EG) is a dihydric alcohol (HO–CH_2–CH_2–OH) with a sweet-bitter taste. It is colourless, odourless, water-soluble, and possesses antifreeze properties. Ethylene glycol is commonly found in various products, including antifreeze, brake fluids, and industrial solvents. (Leth & Gregersen, 2005). Ethylene glycol poisoning is a medical emergency characterized by rapid progression to acute kidney injury, metabolic acidosis, and multi-organ dysfunction. This condition is well-documented in both veterinary and human toxicology literature due to its severe outcomes and the need for rapid intervention (Grauer et al., 2008; Connally & Thrall, 1999). Its sweet, palatable taste makes ethylene glycol intoxication frequent in dogs (Tarr, Hayton et al., 1985) If treatment is not initiated within a few hours (within 6-12 hours), the prognosis becomes severe, with progression from acute kidney injury to acute renal failure (Schweighauser & Francey, 2015). Following ingestion, it is rapidly absorbed from the gastrointestinal tract, with peak plasma concentrations in dogs occurring 2-3 hours post-ingestion. The compound is then primarily metabolized by the liver. The toxicity is not attributed to the primary compound itself, but rather to the formation of toxic metabolites via the enzyme alcohol dehydrogenase (ADH). Ultimately, oxalic acid binds to calcium, resulting in the formation of insoluble calcium oxalate complexes. These complexes are freely filtered by the kidneys and deposited in the renal tubules, causing acute kidney injury. To a lesser extent, deposition also occurs in the vasculature of the brain, heart, and other organs (Schweighauser & Francey, 2015). Clinical signs progress through neurologic, cardiopulmonary, and renal stages, often culminating in acute renal failure if untreated (Segev & Baneth, 2013). Ethylene glycol toxicosis manifests in three stages. The first stage (30 minutes to 12 hours post-ingestion) is characterized by central nervous system signs, including neurologic depression, ataxia,

seizures, coma, or death, likely due to aldehyde metabolites, hyperosmolarity, and metabolic acidosis. The second stage (12 to 24 hours post-ingestion) involves resolving neurologic symptoms and the onset of cardiopulmonary signs such as tachypnoea and tachycardia, with common necropsy findings including pulmonary oedema and congestion. The final stage (24 to 72 hours post-ingestion) is marked by oliguric renal failure and signs of uraemia, including anorexia and vomiting, which may develop within 12 hours of ingestion (Scherk, Koenig et al., 2013).

MATERIALS AND METHODS

A 1-year-old intact female mixed-breed dog was referred for a specialized nephrological consultation on the 22nd of June, 2024. The dog presented with a history of suspected ethylene glycol intoxication. The owner reported that the dog had access to an unknown quantity of antifreeze, approximately 6 hours before presentation. Before the incident, the dog was healthy, with no known medical issues or history of renal disease. Upon initial examination, the dog showed signs of neurologic depression, including ataxia and mild disorientation. The owner noted that the dog had become lethargic and somewhat unresponsive in the hours following ingestion. The dog was observed to have a decreased level of activity and appeared weak. A complete blood count (CBC) was performed using the Vetscan HM5 Haematology system, which evaluated white blood cells (WBC), lymphocytes, monocytes, neutrophils, eosinophils, basophils, red blood cells (RBC), haemoglobin (HGB), haematocrit (HCT), mean corpuscular volume (MCV), red blood cell distribution width (RDW), platelets (PLT), and platelet indices, including mean platelet volume (MPV) and platelet distribution width (PDW). Serum biochemistry was performed using the Vetscan VS2 Chemistry Analyzer, measuring albumin (ALB), alkaline phosphatase (ALP), alanine aminotransferase (ALT), amylase (AMY), total bilirubin (TBIL), blood urea nitrogen (BUN), calcium (CA), phosphorus (PHOS), creatinine (CRE), glucose (GLU), sodium (NA^+), potassium (K^+), total proteins (TP), and globulin (GLOB). Abdominal ultrasonography was performed using the Esaote MyLabX7 ultrasound system. Blood pressure was determined by the oscillometric method (High-Definition Oscillometry). Urinalysis was conducted using the Vetscan UA Urine Analyzer to assess leukocytes, ketones, nitrites, urobilinogen, bilirubin, glucose, protein, specific gravity, pH, blood, ascorbic acid, microalbumin, calcium, creatinine, and the protein-to-creatinine ratio. In this study, the B. Braun Dialog+ device was used to perform haemodialysis. The therapeutic approach focused primarily on haemodialysis, hydro-electrolytic rebalancing, and partial parenteral nutrition. To support rehydration and metabolic stabilization, a continuous rate infusion (CRI) of Ringer's solution was administered. Partial parenteral micronutrition was ensured through the administration of levo-microamino acids. Additionally, to aid renal function, the treatment protocol included enteric dialysis supplements, calcium-based phosphorus binders, a specialized renal diet, and nutritional support as adjuvant therapies.

RESULTS AND DISCUSSIONS

On June 22nd, 2024, the patient presented with severe metabolic acidosis, elevated serum creatinine, blood urea nitrogen, and phosphorus levels, consistent with advanced stage 5 acute kidney injury. Similar findings have been reported in acute ethylene glycol toxicity in both canine and feline patients (Bartges et al., 1998). The biochemical parameters revealed a significant elevation in creatinine (CREA 10.2 mg/dL-RR: 0.4-1.2 mg/dl), blood urea nitrogen (BUN 151 mg/dL-RR: 7-25 mg/dl), and phosphorus (PHOS 9.9 mg/dl- RR: 2.9-6.6 mg/dl), while glucose (GLU 123 mg/dl- RR: 60-110 mg/dl) was mildly elevated. The complete blood count (CBC) showed a decreased mean corpuscular haemoglobin concentration (MCHC 29.3 g/dl- RR: 31-39 g/dl) and mean corpuscular haemoglobin (MCH) within the normal range (MCH 19.1 pg - RR: 14-20 pg). Urinalysis conducted through ultrasound-guided cystocentesis, demonstrated a urine protein-to-creatinine ratio (UPC) of $\geq$0.5 to <2.0 (proteinuric), a pH of 5.0, microalbumin $\geq$25 mg/L, and creatinine at 8.8 mmol/L. At the ultrasound evaluation, bilateral renal alterations were noted, specifically characterized by the

presence of a hyperechoic cortical rim in both kidneys. Arterial blood pressure was elevated, with values ranging between 202/154 (174) mmHg and 205/156 (176) mmHg, using the oscillometric method (High Definition Oscillometry). The decision to initiate haemodialysis was based on critical biochemical abnormalities and the progression of clinical signs. Haemodialysis is considered the most effective treatment for severe ethylene glycol intoxication, as it directly removes both the parent compound and its nephrotoxic metabolites. Numerous clinical case studies and reviews have demonstrated the effectiveness of haemodialysis in treating ethylene glycol toxicosis in small animals (Hall & Grubb, 2001; Grauer et al., 2008). The procedure was performed after placing a central venous catheter under light sedation and supplemental oxygen. The procedure was performed using strict aseptic techniques, including the application of a surgical scrub, sterile gloves, and a sterile environment. Special care was taken in handling the catheter line throughout the process to ensure the highest standards of hygiene and safety. Each lumen of the catheter was scrubbed with 0.5% chlorhexidine for 2 minutes to maintain aseptic conditions.

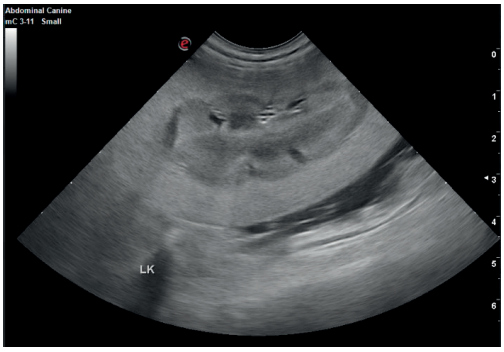

Figure 1. The presence of a cortical rim in the left kidney

Twenty-four hours after the first session of haemodialysis, the blood biochemistry results were as follows: creatinine (CREA) level was 7.4 mg/dl (reference range: 0.4-1.2 mg/dl), blood urea nitrogen (BUN) decreased to 73 mg/dl (reference range: 7-25 mg/dl), and phosphate (PHOS) showed a slight increase to 10.0 mg/dl (reference range: 2.9-6.6 mg/dl). During the haemodialysis session, the patient remained stable without any significant clinical events. After the second session of haemodialysis on the 24th of June, bloodwork results were: CREA 5.1 (RR: 0.4-1.2 mg/dl), BUN 41 (RR: 7-25 mg/dl), PHOS 8.4 (RR: 2.9-6.6 mg/dl) and GLU 118 (RR: 60-110 mg/dL). The blood biochemistry results after the 24-hour pause and after the third session of haemodialysis, were as follows: CREA 4.3 (RR: 0.4-1.2 mg/dl), BUN 43 (RR: 7-25 mg/dl), PHOS 7.6 (RR: 2.9-6.6 mg/dl), GLU 119 (RR: 60-110 mg/dL) and CA 12.6 (RR: 8.6-11.8 mg/dL). On the 28th of June, after another 24-hour pause and following the fourth session of haemodialysis, the blood biochemistry values were as follows: CREA 4.1 (RR: 0.4-1.2 mg/dl), BUN 40 (RR: 7-25 mg/dl), PHOS 9.1 (RR: 2.9-6.6 mg/dl), GLU 127 (RR: 60-110 mg/dL) and CA 13.1 (RR: 8.6-11.8 mg/dL). The latest set of tests from June 29th showed the following values: CREA 2.2 (RR: 0.4-1.2 mg/dl), BUN 32 (RR: 7-25 mg/dl), PHOS 6.8 (RR: 2.9-6.6 mg/dl), GLU 114 (RR: 60-110 mg/dL) and CA 12.5 (RR: 8.6-11.8 mg/dL). After two days of hospitalisation without any haemodialysis sessions but with fluid therapy, oral and intravenous drug therapy, the blood biochemistry values were as follows: CREA 2.0 (RR: 0.4-1.2 mg/dl), BUN 30 (RR: 7-25 mg/dl), PHOS 6.1 (RR: 2.9-6.6 mg/dl) and CA 11.5 (RR: 8.6-11.8 mg/dL).

After discontinuing haemodialysis, the patient-maintained stability with fluid therapy and medication, suggesting partial renal recovery and a favourable prognosis with continued supportive care.

Between the haemodialysis sessions, supportive therapy was implemented to optimize renal function, and systemic stabilization such as fluid therapy with Ringer solution was administered via continuous rate infusion (CRI) at a dosage of 7 mL/kg/h.

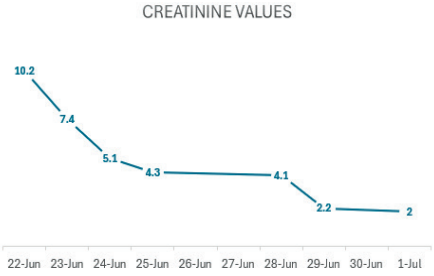

Figure 2. Creatinine values during hospitalization

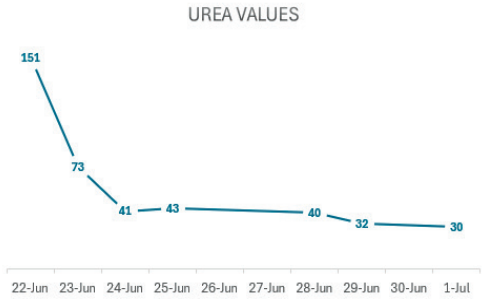

Figure 3. Urea values during hospitalization

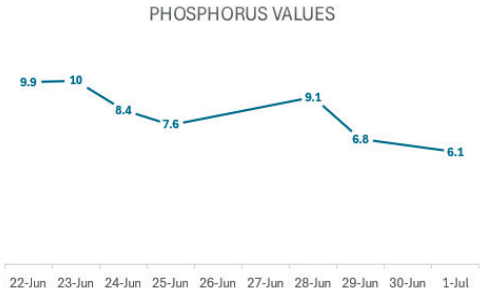

Figure 4. Phosphorus values during hospitalization

The fluid requirements were meticulously calculated based on the patient's individual needs, taking into account the degree of hydration, ongoing losses, and overall fluid balance. This approach aimed to ensure optimal rehydration, maintain electrolyte homeostasis, and support hemodynamic stability throughout the treatment. Partial parenteral micronutrition was provided using levo-microamino acids at a rate of 6 mL/kg/24 h to support metabolic demands. Additionally, enteric dialysis supplements and calcium-based phosphorus binders were incorporated to aid in toxin elimination and phosphate regulation. A specialized renal diet, and targeted nutritional supplements, were introduced as adjunctive therapy to further support kidney function and promote overall recovery. Such multimodal therapeutic strategies are consistent with current recommendations in veterinary emergency protocols (Davis et al., 2007). After eight days of hospitalization and four haemodialysis sessions, the patient was discharged with a prescribed treatment plan.

Ethylene glycol intoxication is a life-threatening condition in dogs, commonly caused by the ingestion of antifreeze. Once absorbed, ethylene glycol is rapidly metabolized into toxic compounds that induce severe metabolic acidosis and acute kidney injury. Kidney damage occurs due to calcium oxalate crystal deposition, leading to renal failure if left untreated. The sooner the patient undergoes haemodialysis, the higher the chances of recovery. Haemodialysis efficiently removes ethylene glycol and its toxic metabolites while correcting metabolic imbalances and supporting kidney function during recovery.

The clinical progression observed in this case underscores the critical importance of early intervention in ethylene glycol intoxication. According to Schweighauser and Francey (2016), initiating treatment within the first 8 hours post-ingestion significantly improves prognosis and reduces the risk of irreversible acute kidney injury (AKI). In the case presented, prompt initiation of haemodialysis contributed to the gradual decrease in creatinine and blood urea nitrogen (BUN) levels, helping stabilize renal function.

This outcome is consistent with findings reported in similar case studies, where extracorporeal therapy proved effective in removing ethylene glycol and its toxic metabolites, while correcting metabolic derangements and supporting renal recovery (Schweighauser & Francey, 2016).

In addition, the observed elevations in phosphorus and calcium may reflect renal deposition of calcium oxalate crystals - a well-documented complication of ethylene glycol toxicity. Careful monitoring and supportive interventions, including phosphate binders and fluid therapy, were essential to mitigating these secondary effects.

Although haemodialysis was highly beneficial, it is important to note that full normalization of renal parameters was not achieved within the hospitalization period. Previous reports suggest that in severe cases, persistent renal damage may occur even with timely intervention (DiBartola et al., 1985). Nonetheless, the progressive improvement in laboratory values and the patient's stable condition at discharge are promising indicators of partial renal recovery.

Overall, this case reinforces the value of haemodialysis as a life-saving therapeutic option in veterinary nephrology and highlights the necessity of early diagnosis and intervention

to prevent long-term renal dysfunction. Expanding access to extracorporeal therapies such as haemodialysis in critical care veterinary settings could significantly improve survival rates in cases of acute toxicosis (Langston, 2002).

CONCLUSIONS

This case report underscores the critical importance of early diagnosis and timely initiation of hemodialysis in ethylene glycol intoxication, even in patients presenting with advanced stage 5 acute kidney injury. The extracorporeal intervention enabled the progressive stabilization of renal function, as evidenced by the consistent decline in serum creatinine, blood urea nitrogen, and phosphorus levels over the course of therapy.

Hemodialysis proved effective not only in eliminating ethylene glycol and its nephrotoxic metabolites but also in correcting severe metabolic acidosis and preventing irreversible nephron damage associated with calcium oxalate deposition. Adjunctive therapies - including fluid resuscitation, phosphate binders, partial parenteral nutrition, and a renal-specific diet - played an essential role in restoring metabolic balance and supporting renal recovery.

Although full normalization of biochemical parameters was not achieved during hospitalization, the observed clinical improvement and sustained reduction in azotemia at discharge indicate a favorable prognosis and partial preservation of renal function. The outcome validates hemodialysis as the most effective therapeutic approach for severe ethylene glycol poisoning when initiated promptly.

Beyond its individual clinical relevance, this case emphasizes the broader implications for emergency veterinary care. Improved access to extracorporeal renal replacement therapies and heightened awareness of their efficacy could significantly enhance survival and long-term outcomes in patients with acute toxicologic nephropathies. These findings support the integration of early hemodialysis into standardized treatment algorithms for companion animals exposed to nephrotoxic agents.

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