Ethylene glycol toxicity in two calves

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ABSTRACT

Burdur Mehmet Akif University Veterinary Faculty Animal Hospital was brought from livestock to 3 calves aged between 15 and 20 days with complaints of weakness, dehydration, inability to get up and nervous findings. Two of them failed the treatment and were brought to the pathology department after being euthanized. Both animals were macroscopically dehydrated, cachectic, and had hair loss in some parts of the body. There was 1-2 liters of white light-colored transparent fluid in the abdominal cavity. Many organs and serous membranes in the abdominal cavity were hyperemic. Histopathological examination revealed bleeding, edema and degenerative changes in the heart, lungs and kidneys. Calcium oxalate crystals were found in many tubuler lumens, especially the proximal convoluted tubules in the kidneys. In the light of clinical, macroscopic, and histopathological findings, the case was diagnosed with ethylene glycol toxicity. Ethylene glycol toxicity is often seen in cats and dogs living near industrial or auto car repair shops, and in animals that accidentally drink antifreeze waste. This case report is the first case of ethylene glycol toxicity reported in livestock calves in Türkiye.

Ethylene glycol (EG) is an automotive product and can be toxic when exposed to human and animals. EG usually causes irreversible kidney damage. However, it may also affect many systems (5). EG is often drunk accidentally since it is sweet (7). Affected animals are mostly cats and dogs, but ruminants may also rarely be affected rarely (1, 8). Clinical signs of affected animals include dyspnea, hypersalivation, ataxia, paraparesis, depression, and death (3). Toxicity is related to the in vivo conversion of the main compound into toxic metabolites. It does not have a direct toxic effect. Metabolites such as oxalic acid and calcium chelates may lead to the formation of calcium oxalate complexes to become difficult-to-soluble. The accumulation of these metobolites into renal tubules causes acute tubuler damage and renal failure (10). The crystals are light yellow in color and have rosettes or prismatic formations (11).

In this case, renal oxalosis was encountered due to ethylene glycol poisoning. Renal oxalosis, which is characterized by the appearance of oxalate crystals in the kidneys, can also be seen when the autosomal recessive gene is dominant genetically, the mothers consume oxalate-containing plants during pregnancy, and the patient directly consumes oxalate-containing plants. In this case, the onset of the disease in 10 animals at a time eliminates the first cause. Since it is known that the mothers do not leave the farm and are fed with roughage and concentrated feed during pregnancy, they are eliminated for the second reason. The fact that the leak in the machine used to feed the calves on the farm was noticed and when it was repaired, no other cases were seen, also indicates that the diagnosis was correct (6).

Three of 13 calves, aged between 15 and 20 days, were brought to Burdur Mehmet Akif University

Veterinary Faculty Animal Hospital for treatment with complaints of weakness, dehydration, inability to get up and nervous symptoms. Two animals were sent to the Pathology Department for autopsy after they died during the treatment. At autopsy, approximately 2 liters of yellowish-white transparent fluid was found in the abdominal cavity of one of the calves (No: 2). The kidneys were swollen and pale in appearance. The lung was swollen, dark red in color, and contained areas of petechiae scattered over the lobes. Petechial hemorrhages were observed on the epicardium and endocardium, especially in the m papillaris and areas under the valves. In the other calf (No: 1), no significant macroscopic finding was observed except severe hyperemia in the serosa of the organs in the abdominal cavity and pulmonary edema. Samples taken from lesioned tissues were fixed in 10% buffered formaldehyde and embedded in paraffin blocks after routine tissue follow-up. Sections of 5 µm thickness were taken from the blocks and stained with routine hematoxylin & eosin. Histopathological examination revealed subpleural hemorrhage and alveolar edema in both calves (Figure 1A). One of the calves (No: 1) had subepicardial hemorrhages in the heart sections (Figure 1B). Epithelial desquamation, edema in the propria and mononuclear cell infiltrations were detected in the intestines of both calves. In one of the calves (No: 2), the Virchow-Robin spaces were enlarged due to edema in the brain (Figure 1C). Calcium oxalate crystal deposits were found in both calves with marked enlargement of Bowman's spaces (Figure 1D) and in the lumens of many proximal convolutes and distal tubules.

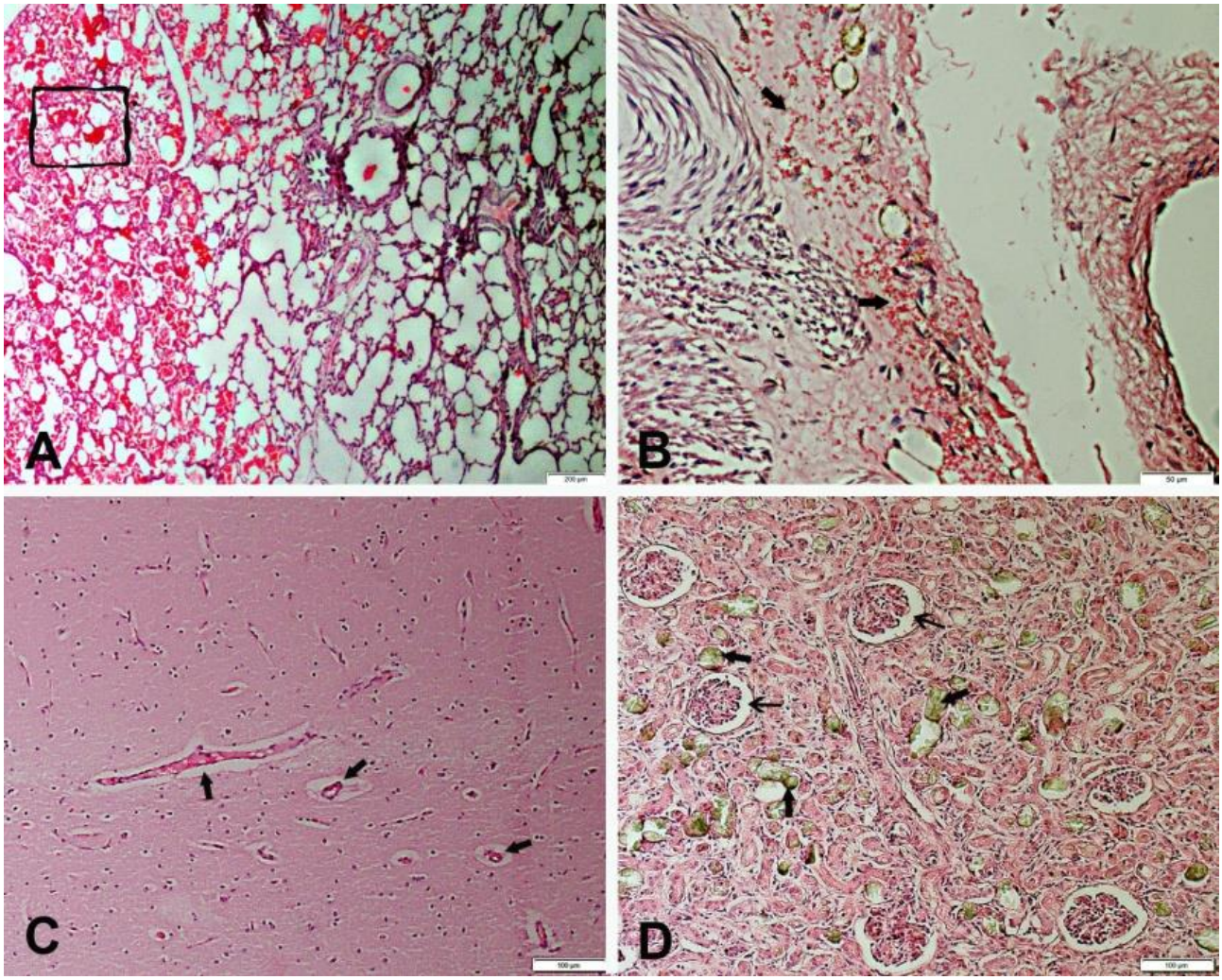

Figure 1. A. Calf, lung, subpleural hemorrhage, (rectangular), HE X40, Bar=200 µm. **B.** Heart, subepicardial hemorrhages (arrows), HE X100, Bar=100 µm. **C.** Brain, enlargement of Virchow-Robin spaces due to edema (arrows) HE X40, Bar= 200 µm. **D.** Kidney, calcium oxalate crystals in many tubuler lumens (thick arrows), Bowman spaces enlargement (thin arrows), HE X200, Bar= 50 µm.

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Although EG toxicity is rarely reported in farm animals, it is seen frequently in cats and dogs that drink EG due to its sweetness. On the other hand, poisonal exposure in calves may be rare and it is usually by accident or food contamination. EG has no use in food preparation units other than its accidental intake. However, it is a matter of curiosity how food contamination can occur. Today, automatic machines are used to feed calves on modern farms. EG is used to maintain the temperature of the food in these machines. In this case report, EG leakage from an automatic machine used in calf feeding is mixed with calf food. The cause of the calcium-oxalate crystals seen in histopathological examinations was not understood at first, but it was revealed as a result of deep anemnesis investigation.

Previous studies report that laboratory results such as serum urea, creatinine, phosphate, total protein, and the amount of white blood cells increase but hemoglobin decrases (11). The laboratory findings of this study showed that despite increasing serum creatinine and lactate levels, hemogram levels were surprisingly normal.

At the necropsy, the marked lesions were gastrointestinal hyperemia and pulmonary edema. Petechial or echymotic hemorrhages were observed in the supcapsular surface of the kidneys, and the heart was enlarged (2). The macroscopic lesions of this study were similar to the previous reports. In addition, petechial hemorrhages were found near the valves.

The renal toxicity of ethylene glycol is generally described as renal tubuler damage caused by the accumulation of calcium oxalate crystals (4, 10). Additionally, focal interstitial lymphocytic infiltration and mild degenerative-necrotic tubuler changes may be seen in some cases (1). In this case, characteristic renal lesions such as Bowman's spaces and dilatation and calcium oxalate deposits, especially in the distal tubules, and tubular epithelial eruptions in some areas were consistent with the findings of previous reports.

Glycolic acid, which is one of the ethylene glycol metabolites, may result in coma due to encephalopathy or brain edema (9, 12). In this case, enlargement of Virchow-Robin spaces in the neuropile confirms that there was brain edema due to glycolic acid but no damage to neurons.

In this case, clinical, macroscopic, and microscopic findings of ethylene glycol poisoning due to food contamination in fattening calves were reported. This extremely unusual case demonstrated how important the synthesis of anemnesis and pathological findings is in diagnosis.

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Conflict of Interest

The authors declared that there is no conflict of interest.

Author Contributions

ZÖ, ÖÖ and RY conceived and planned the experiments. İYA, GO and MA carried out the experiments. ZÖ, ÖÖ and RY planned and carried out the simulations. İYA, GO and MA contributed to sample preparation. ZÖ, ÖÖ and RY contributed to the interpretation of the results. ZÖ took the lead in writing the manuscript. All authors provided critical feedback and helped shape the research, analysis and manuscript.

Ethical Statement

This study does not present any ethical concerns.

References

1. **Barigye R, Mostrom M, Dyer NW, et al** (2008): *Ethylene glycol toxicosis in adult beef cattle fed contaminated feeds.* Can Vet J, **49,** 1018-1020.
2. **Berman LB, Schreiner GE, Feys J** (1957): *The nephrotoxic lesion of ethylene glycol.* Ann Intern Med, **46,** 611-619.
3. **Crowell WA, Whitlock RH, Stout RC, et al** (1979): *Ethylene glycol toxicosis in cattle.* Cornell Vet, **69,** 272-279.
4. **Dalefield R** (2004): Ethylene glycol. In: Plumlee KH (ed.): Clinical Veterinary Toxicology, 1st ed. Mosby, St. Louis, 150-173.
5. **Doty RL, Dziewit JA, Marshall DA** (2006): *Antifreeze ingestion by dogs and rats: influence of stimulus concentration.* Can Vet J, **47,** 363-365.
6. **Gülbahar MY, Kaya A, Gölen İ** (2002): *Renal oxalosis in a calf.* Turk J Vet Anim Sci, **26,** 1197-1200.
7. **Keller N, Goddard A** (2005): *First report of suspected ethylene glycol poisoning in 2 dogs in South Africa.* J S Afr Vet Assoc, **76,** 116–119.
8. **LaKind JS, McKenna EA, Hubner RP, et al** (1999): *A review of the comparative mammalian toxicity of ethylene glycol and propylene glycol.* Crit Rev Toxicol, **29,** 331–365.
9. **Mair W** (1983): *Cerebral computed tomography of ethylene glycol intoxication.* Neuroradiol J, **24,** 175-177.
10. **Porter WH** (2012): *Ethylene glycol poisoning: Quintessential clinical toxicology; analytical conundrum.* Clin Chim Acta, **413,** 365–377.
11. **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.
12. **Walder AD, Tyler CKG** (1994): *Ethylene glycol antifreeze poisoning: three case reports and a review of treatment.* J Anaesth, **49,** 964-967.