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Diagnostic Exercise
From the Latin Comparative Pathology Group and the Davis-Thompson Foundation

Uremic gastritis and calcium oxalate crystallization in a dog

Emily G. Lewis[1]*, Kayla J. Alexander[1], and Timothy W. Morgan[1].
[1]Department of Pathobiology and Population Medicine, Mississippi State College of Veterinary Medicine.

***Corresponding author:** egl141@msstate.edu
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Clinical History:

A 7-year-old female intact Welsh Corgi mix was presented to emergency services for vomiting, diarrhea, inappetence, and lethargy. Over the last 24 to 48 hours, she vomited approximately 5 to 6 times a day.

Physical exam:

8% dehydrated
Heart rate: 143 bpm
Respiratory rate: panting
Temperature: 97.1°F

Pertinent Bloodwork Findings:

CREA: unable to read (0.5-1.8 mg/dL)
BUN: >130 (7-27 mg/dL)
PHOS: >16.1 (2.5-16.1mg/dL)
Na: 139 (144-160mmol/L)
Cl: 98 (109-122 mmol/L)
iCa: 1.12 (1.16-1.40mmol/L)

**The Diagnostic Exercises are an initiative of the Latin Comparative Pathology Group (LCPG), the Latin American subdivision of The Davis-Thompson Foundation and published in cooperation with the Brazilian Journal of Veterinary Pathology. These exercises are contributed by members and non-members from any country of residence. Consider submitting an exercise! A final document containing this material with answers and a brief discussion will be posted on the CL Davis website: https://davisthompsonfoundation.org/diagnostic-exercise/*

Lactate strip:

3 mmol (<2.5mmol)

Autopsy findings:

On physical examination, the maxillary lingual mucous membranes demonstrated bilaterally symmetric, well-demarcated circular area measuring 2 x 2.5cm. The ulcerations were pale tan and marginated by a rim of dark red discoloration.

Bilaterally, both kidneys were small, pale tan, and had irregular pitted cortical surfaces (Fig. 1 and 2).

Approximately 70-90% of the serosal surface of the stomach was effaced by a sharply demarcated, dark red discoloration, which spared the region of the pylorus (Fig. 3). The wall of the stomach was markedly thickened and measured 1cm in width (Fig. 4). The mucosa was diffusely dark red to black.

The small intestines were mildly thickened and contained a small amount of black, opaque, mucoid fluid with red flecks. The cecum and colon contained a moderate amount of black, tarry material (melena).

The right and the left parathyroid glands were bilaterally enlarged.

Follow-up questions:

- *Morphologic Diagnoses:*
- *Pathogenesis of the lesions of the stomach:*
- *What are two possible causes of oxalate crystals within the renal tubules?*

ANSWERS

Morphologic diagnosis:

Kidney:

- Tubulointerstitial nephritis, lymphoplasmacytic, multifocal, chronic, and severe with marked interstitial fibrosis
- Tubular degeneration and necrosis, diffuse, severe, acute with intratubular calcium oxalate crystals, tubular proteinosis, and mineralization

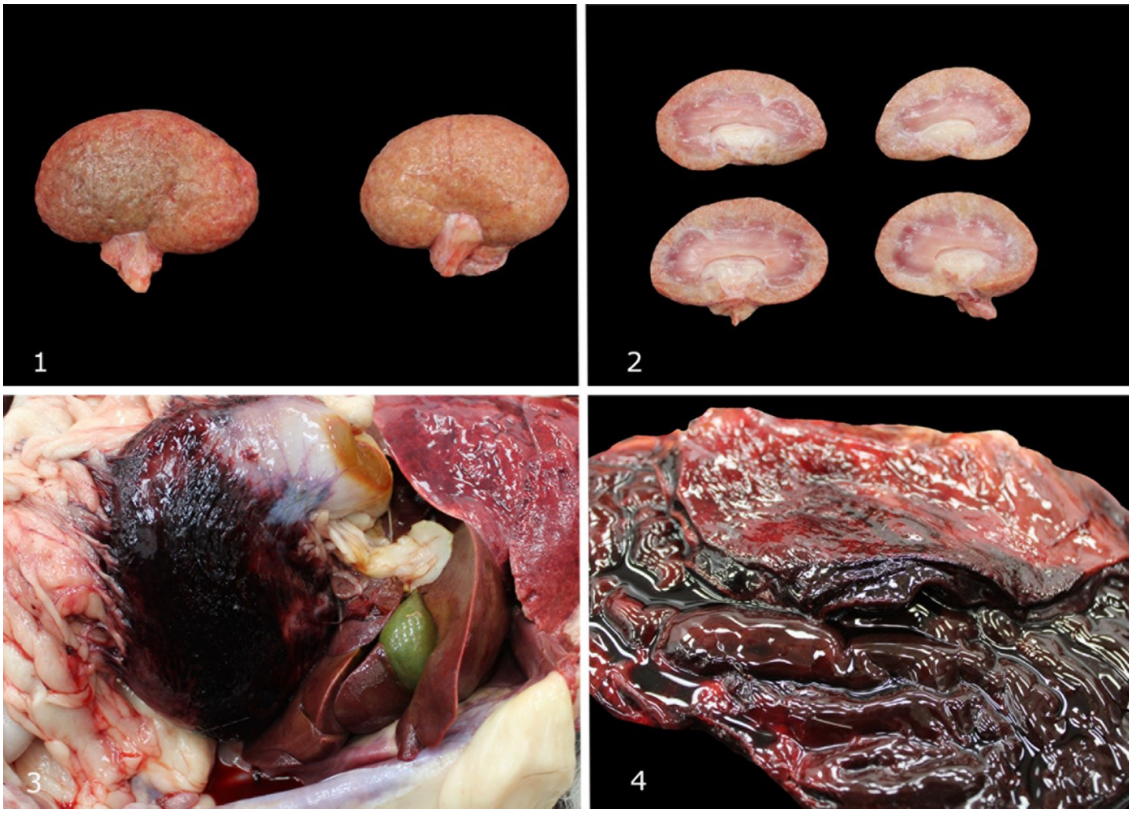

Figure 1 and 2. Left and Right Kidneys. **Figure 3.** Serosal surface of the stomach *in situ*. **Figure 4.** Opened stomach serosal and mucosal aspects

Stomach:

- Arteriolar infarction, multifocal, chronic, and severe with submucosal fibrinoid vasculitis, mineralization, and thrombosis
- Mucosal dystrophic mineralization, multifocal, chronic, and severe

Cause:

Uremic gastritis secondary to end-stage kidney disease

Suspected ethylene glycol toxicosis

Discussion:

Uremic gastritis is a unique presentation of end-stage kidney disease in dogs. Uremia is defined as the clinical syndrome of renal disease that may manifest as lethargy, inappetence, and ulcerative stomatitis and may include lesions within the gastrointestinal, respiratory, and skeletal systems due to calcium and phosphorus imbalances (2). Due to the reduced glomerular filtration rate, the kidneys are unable to excrete phosphorus from the blood into the urine. High levels of phosphorus in the blood complex with ionized calcium, causing pathological deposition of mineral within the vasculature and soft tissues, such as the gastric mucosa, which is referred to as uremic gastritis (3). The decrease in blood ionized calcium simultaneously stimulates the release of parathyroid hormone (PTH), resulting in hyperplasia of the parathyroid glands. PTH activates osteoclastic bone resorption to release more calcium to correct the deficiency.

In a study of uremic effects in cats, the second most common lesion was ulcerative or hemorrhagic gastritis, which accounted for approximately 36% of cases (1). In two of those cases, there was also vascular thrombosis and fibrinoid vascular necrosis similar to this case. Another retrospective study concluded that over half of canines with chronic kidney disease will display gastric vascular abnormalities characterized by intimal and endothelial mineralization that is not always associated with fibrinoid necrosis (7). Interestingly, the same study reported that gastric mucosal necrosis and ulceration were uncommonly

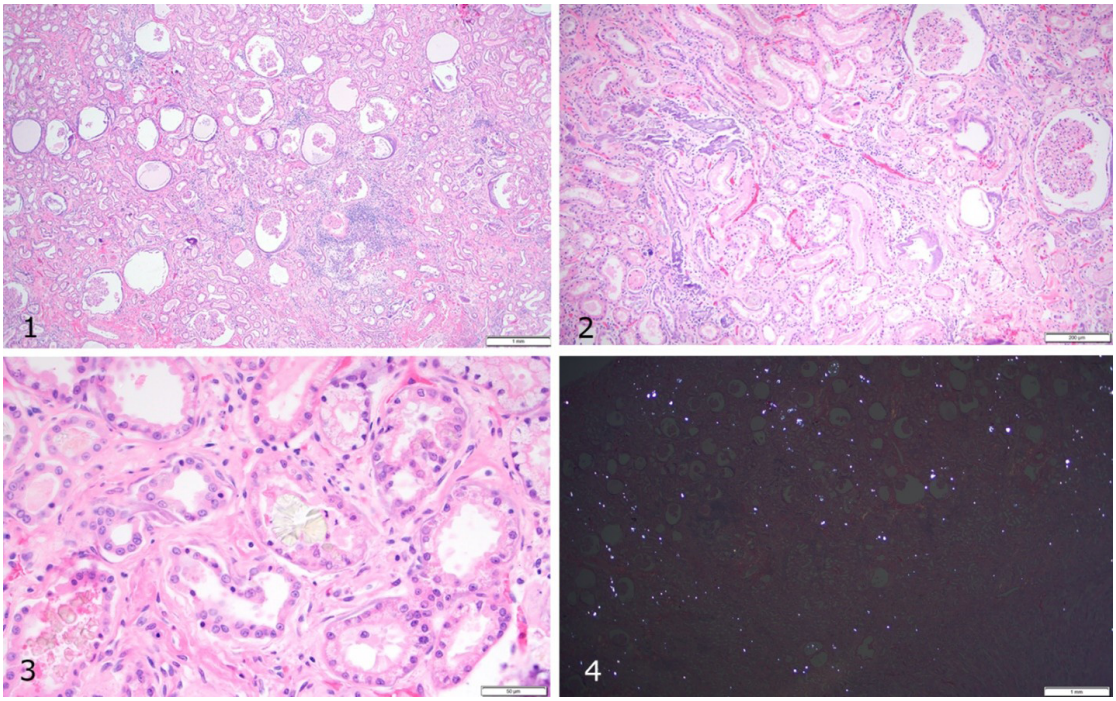

Figure 5. Renal cortex, 4x objective. The renal cortex features moderate to marked expansion of the interstitium with fibrosis and inflammation (primarily lymphoplasmacytic). Multifocally, tubules and glomerular spaces are expanded by increased clear space with occasional proteinaceous material.
Figure 6. Renal cortex, 10x objective. There is marked tubular mineralization, tubulointerstitial lymphoplasmacytic nephritis, as well as multifocal tubular degeneration and atrophy.
Figure 7. Renal cortex, 40x objective. Many of the renal tubules demonstrate vacuolar degeneration and necrosis. The renal tubules multifocally contain pale yellow crystals arranged in rosettes and sheaves.
Figure 8. Renal cortex under plane polarized light, 2x objective. Throughout the cortex, there are numerous birefringent crystals present within renal tubules.

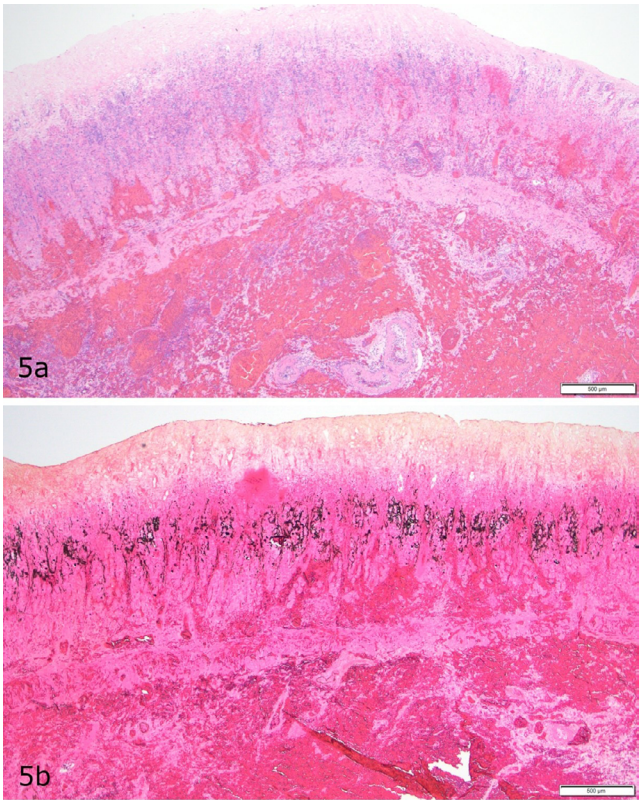

Figure 5. Stomach, 4x. The submucosa is markedly expanded by large amounts of hemorrhage mixed with eosinophilic fibrillar to amorphous material. The mucosa is diffusely hypereosinophilic and lacks cellular detail. Multifocally, the mucosa has faint basophilic stippled material which is highlighted in Figure 5a with a Von Kossa histochemical stain.

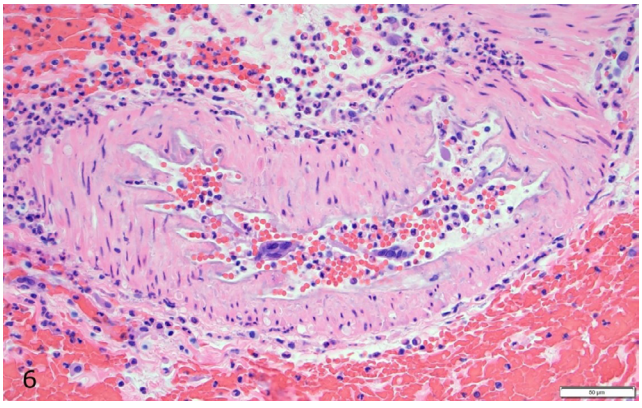

Figure 6. Higher magnification of a submucosal arteriole in Figure 5, 40x. The intimal and endothelium are mostly replaced by pale amphophilic, finely stippled material (mineral). The vessels are often surrounded and partially infiltrated by intact and degenerate neutrophils. Some of the areas of the vascular wall are hypereosinophilic and smudgy. The lumen of the arteriole contains a few multinucleated giant cells, many neutrophils, and some red blood cells with sloughed, mineralized endothelial cells.

encountered and that this information may have been inappropriately extrapolated from human literature (6, 7). In the present case, there was no grossly appreciable gastric mucosal ulceration, and transmural necrosis was likely due to vasculitis.

Another clinical factor in this case was chronic protein-losing nephropathy. Protein-losing nephropathies are associated with urinary loss of antithrombin. Antithrombin inhibits the generation of thrombin and promotes an anticoagulative state. With substantial loss of antithrombin, there is a physiologic shift into a prothrombotic state which can result in infarction of affected organs. Loss of antithrombin and fibrinoid vasculitis of the gastric vessels contributed to multifactorial vascular infarction resulting in the well-demarcated red to black discoloration of the serosal surface of stomach appreciated grossly.

In addition to the chronic components of this case, many calcium oxalate crystals were present within the renal tubules. One potential cause of acute kidney injury is ingestion of ethylene glycol, a component of antifreeze. Following ingestion, ethylene glycol is rapidly absorbed by the gastrointestinal tract and metabolized by hepatic alcohol dehydrogenase into many toxic metabolites such as glycolaldehyde, glycolic acid, and oxalate. Oxalate can then interact with calcium, forming calcium oxalate crystals within the renal tubules (4, 10). The crystals cause physical injury to the renal tubular epithelium, as well as mechanical obstruction of the tubular lumen. Premortem bloodwork and urinalysis may reveal profound metabolic acidosis and calcium oxalate monohydrate crystalluria. Confirmation testing was not performed on this case for the formation of urinary calcium oxalate crystallization include other causes of hypercalciuria such as severe, chronic kidney disease and vitamin D toxicosis due to the excess excretion of calcium (9). Few breeds are predisposed to calcium oxalate crystalluria, including miniature schnauzers, bichon frisés and shih tzus, but not Welsh Corgis (9).

Overall, this case represents a unique syndrome associated with end-stage kidney disease compounded by acute kidney injury. Clinically, this case manifested as an "acute-on--chronic" kidney injury, meaning there was an acute kidney injury to a patient with pre-existing chronic kidney disease. Chronic components of this case induced a myriad of lesions including bilateral parathyroid gland hyperplasia, and the physical manifestation of uremia due to the calcium and phosphorus imbalance with buildup of uremic toxins. The acute injury is likely attributed to ethylene glycol ingestion leading to precipitation of calcium oxalate crystals within the renal tubules, causing both mechanical epithelial damage and obstruction.

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