Uremic Gastropathy in Cats

GI signs associated with feline chronic kidney disease (CKD) are commonly attributed to uremic gastropathy; antacids and gastroprotectants are therapy mainstays. However, research on the prevalence and pathogenesis of uremic GI disease in cats is incomplete, with treatment regimens often based on findings in dogs and humans. This study evaluated uremic gastropathy in feline CKD to reevaluate medical management guidelines. Thirty-seven CKD cats and 12 nonazotemic cats were evaluated. Serum creatinine concentrations, calcium-phosphorus product (CPP), and serum gastrin concentrations were recorded. At necropsy, gross and histologic evaluation for the presence of classic uremic gastropathy lesions were performed.

Severely azotemic cats (serum creatinine >5.0 mg/dL) had significantly higher CPP compared to all other cats. No change was noted when cats receiving phosphate binders were excluded. CKD cats had significantly higher serum gastrin concentrations than controls; no change was noted when cats receiving antacid treatment were excluded. No gross or histologic evidence of gastric mucosal ulcerations, hemorrhage, edema, or vascular fibrinoid change was observed in CKD or control cats. However, gastric fibrosis and mineralization were observed more frequently in CKD cats.

Results suggest that GI signs in feline CKD may be caused by uremic toxins and centrally acting emetogens rather than gastric lesions, potentially supporting management with antiemetic and anti-nausea medications over gastroprotectants. The presence of gastric mineralization, likely from metastatic mineralization, may highlight the need for more aggressive control of hyperphosphatemia and renal secondary hyperparathyroidism. The role of hypergastrinemia also needs further study.

Commentary

There are many postulated causes for the GI signs of uremic animals—many borrowed from human medical studies—but there are few studies elucidating the actual causes of anorexia and vomiting in chronically azotemic cats. Cats resent being administered most forms of medication so insights that would reduce the number and amount of medications could be important. This study suggests that gastric protectants and H_2-blockers may not be helpful and that antiemetic and antinausea drugs might be more appropriate; however, the latter is pure conjecture because this aspect was not studied. Increased calcium-phosphorus product was associated with gastric mucosal mineralization, so early control of elevated serum phosphorus levels with diet and phosphate binders seems logical.—*David F. Senior, BVSc, DACVIM, DECVIM (Companion Animal)*

Source

Relationship among serum creatinine, serum gastrin, calcium-phophorus product, and uremic gastropathy in cats with chronic kidney disease. McLeland SM, Lunn KF, Duncan CG, et al. *JVIM* 28:827-837, 2014.

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