

**DATE PRESENTING CLINICAL SIGNS**

3/15/23

History: Azotemia and isosthenuria diagnosed 2/22/23 after very mild SDMA elevation noted 12/22/22. Also had a dental cleaning 1/5/23 and heart murmur was worse under/after anesthesia so started Vetmedin, which caused severe vomiting and anorexia. Stopped Vetmedin after a few doses, but anorexia has remained and we are looking to see if it is just worsening chronic renal disease vs other cause.

PATIENT

Sidney Green

SPECIES

Canine

BREED

Shetland Sheepdog

SEX

Neutered Male

AGE

1/5/12

WEIGHT

21 Pounds

INTERPRETED BYEric Lindquist, DMV
DABVP, Cert. IVUSS**HOSPITAL NAME**

Airpark AH

REFERRING VET

Dr. Owens

INVOICE

21645

Current Medications: None currently.

Lab Results: Azotemia.

Date of Previous IntraPet Ultrasound: No previous.

Sedation: Not required to complete full diagnostic ultrasound.

Stat Report: Not requested.

Imaging Performed By: Rachel Brillhart, RDMS.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN**Urinary System**

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes were noted. Ureteral papillae were normal.

The **kidneys** revealed largely normal size and structure, corticomedullary definition and ratio (cortex 1/3 of medulla) were essentially maintained with some mild to moderate age-related loss of curvilinear patterns regarding the capsule and C/M junction. The cortices presented largely uniform texture with some increased echogenicity expected for his age patient. Medullary structure differed distinctly from that of the cortex. The right kidney measured 5.3 cm. Pyelectasia (0.66 cm) and echogenic debris were noted in the right kidney. Pelvic debris would suggest pyelonephritis. An anechoic cyst was noted in the caudal pole of the right kidney, measuring 1.9 cm. An anechoic cyst was noted in the cranial pole of the left kidney, measuring 0.33 cm. Pyelectasia (0.59 cm) and echogenic debris were also noted in the left kidney.

Adrenal Glands

The **adrenal glands** appeared slightly enlarged and swollen. No evidence of focal capsular expansion or invasion into the phrenic veins were noted. No overt suspicion of neoplasia was noted. This is considered likely a hyperplastic change associated with stress or adrenal endocrinopathy (PDH). If isosthenuria is persistently present and the patient morphologically suggests Cushing's disease then ACTH testing would be indicated. This is a mild change. The left adrenal gland measured 2.37 cm x 0.79 cm at the cranial pole and 0.77 cm at the caudal pole. The right adrenal gland measured 1.72 cm x 0.75 cm at the cranial pole and 0.65 cm at the caudal pole.

Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes were noted.

Liver

The **liver** revealed uniform enlargement with a vacuolar hepatopathy pattern. The gallbladder was overdistended with striating bile and suspended biliary sand.

Gastrointestinal

Gastric hypertrophy was noted with echogenic mucosal remodeling. No overt ulcers were noted, however, microulcerative disease cannot be ruled out. The small intestine and colon were unremarkable.

Pancreas

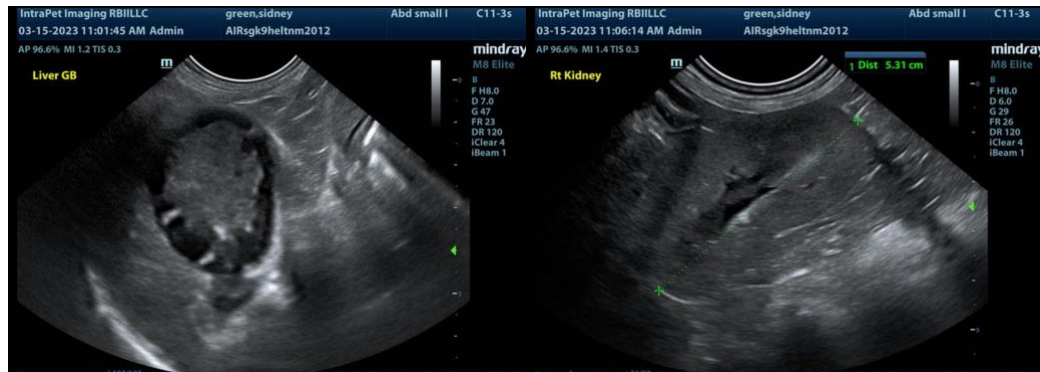
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Some mild parenchymal remodeling, however, with mild deviation from curvilinear normalcy was observed. Pancreatic duct and capsular irregularities were present consistent with age related changes. If pain upon imaging (+ Murphy sign) was present or if the patient is focally painful in subxyphoid palpation then low-grade smoldering chronic pancreatitis should be suspected.

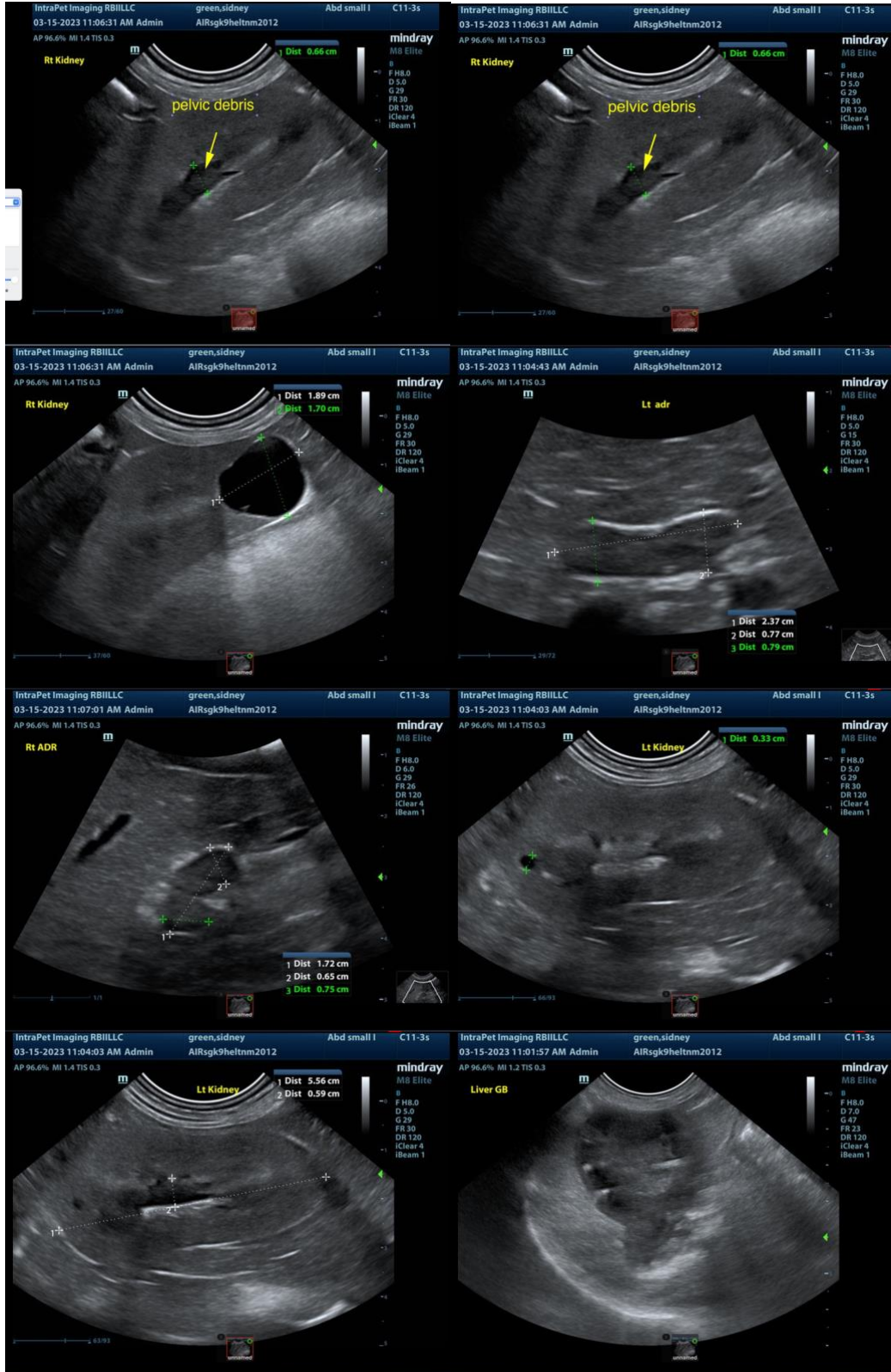
ULTRASONOGRAPHIC FINDINGS

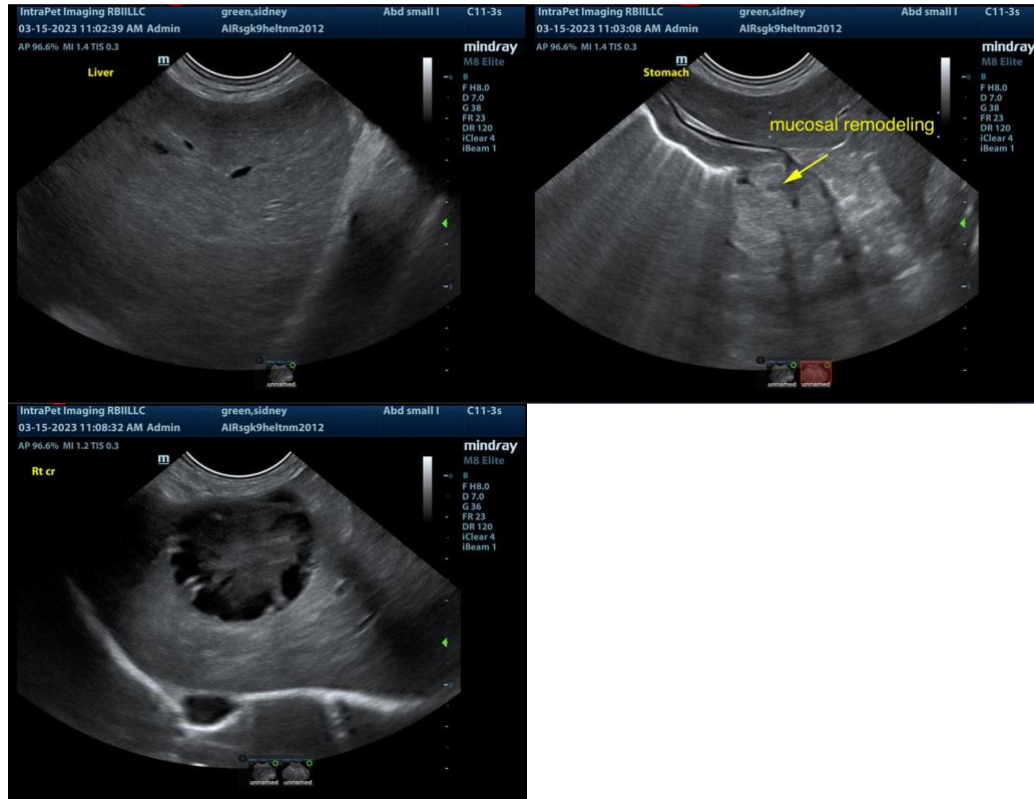
- Moderate degenerative renal changes with cortical cysts and pyelectasia
- Gallbladder mucocele- striating bile noted with kiwi type appearance
- Vacuolar hepatopathy pattern
- Chronic gastritis
- Bilateral adrenal hypertrophy
- Age-related pancreatic changes

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Urine culture and sensitivity is warranted. Medullary wash-out may be an issue, hiding inflammatory sediment. Gallbladder motility study or direct cholecystectomy and gastric biopsies would be appropriate. The kidneys do not appear end-stage. If medical management is selected, then ursodiol over 6-8 weeks could be considered, however, it's debatable on whether or not medical management will be effective. Blood pressure measurements are also warranted. Coverage for pyelonephritis/UTI is indicated. 72-hour IV fluid protocol would be ideal, and reassessment of the azotemia reported. No blood chemistry values were submitted. This is likely both a prerenal and renal effect on the azotemia given the gallbladder mucocele and gastric presentation.







The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

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Gallbladder Mucoceles

<http://www.sonopath.com/GBMucocele>
<http://www.sonopath.com/GBMucoceleSurvey>
<http://www.sonopath.com/SxBiliaryDisease>

Description: A gallbladder mucocele occurs when the gallbladder becomes overly distended with an excessive accumulation of mucus secondary to cystic mucosal hyperplasia. Previously, pathologists noted this finding at necropsy and considered it an incidental or age-related lesion. In the last decade, however, it has become evident that not all gallbladder mucoceles remain clinically silent and that they can in fact be associated with extrahepatic bile duct obstruction (EHBDO), cholecystitis, and gallbladder

wall rupture. Approximately 50% of mucocèles may result in necrosis and rupture, typically at the neck or fundic region of the gallbladder. The etiology of these mucocèles remains unknown, but researchers suspect that it is related to disordered cholestasis and/or lipid metabolism. Suggested causes include: primary or secondary gallbladder motility disorder; dyslipidemia/hypercholesterolemia; extrahepatic bile duct obstruction; and primary or secondary disorders of mucus-producing cells, such as cystic mucosal hyperplasia of the gallbladder wall. Clinical correlation is also seen with cholangitis, cholecystitis, cholelithiasis, biliary obstruction from cholelithiasis or neoplasia, and pancreatitis. Abnormal adrenal hormones may also play a causal role in altering gallbladder mucus secretion. Hypothyroid dogs are three times more likely to develop a mucocèle, and Cushinoid dogs are twenty-nine times more likely to do so. Vacuolar hepatopathy frequently co-occurs with mucocèles; practitioners are therefore advised to investigate underlying disorders associated with vacuolar hepatopathy. Bacterial infection of the gallbladder has been associated with increased mucin production in dogs; this condition may also become pathological and lead to excessive mucus accumulation. In humans, hypercortisolism is related to chronic cholecystitis and changes the biochemical composition of bile. The latter, however, was not substantiated by two studies evaluating bile composition, bacterial infection, and sludge formation during a three-month period of exogenous administration of hydrocortisone administration in dogs. There is a marked increase in the prevalence of mucocèle formation in dogs with naturally occurring hyperadrenocorticism, but a definitive correlational mechanism has yet not been ascertained.

Mucocèles are most commonly seen in middle-aged to older dogs (median age of 10 years); however, researchers have reported mucocèle development in dogs as young as 3 years old. Certain breeds—Miniature Schnauzers, Shetland Sheepdogs, Cocker Spaniels, Shih-tzus, Pugs, Bichon Frisés, Schipperkes, West Highland White Terriers, and Scottish Terriers—appear to be overrepresented among canine patients. Significant predisposition to mucocèle formation in Shelties prompted an investigation that uncovered a specific genetic mutation in the ABCB4 gene, which functions in the translocation of phosphatidylcholine across hepatocyte cell membranes. In the future, we may be able to screen young Shelties for this mutation, which would allow us to monitor gallbladder mucocèle development with ultrasound over time and thereby offer early dietary and medical management, or even surgical intervention as needed. Felines are less commonly affected.

Clinical Signs: According to several retrospective studies, the most common clinical signs include vomiting (87%), abdominal pain (87%), anorexia (78%), lethargy, icterus (57%), and fever (26%). The average duration of illness is 5 days. Focal pain upon examining the gallbladder is common and referred to as a positive Murphy sign. Patients may also be asymptomatic, despite biochemical abnormalities, even in the face of ultrasonographic evidence of a mucocèle. Patients with fully formed mucocèles are at risk of further complications, such as gallbladder rupture, peritonitis, sepsis, and related coagulopathies, and should be considered for surgical intervention.

Diagnostics: Biochemical analyses may indicate leukocytosis, with or without a left shift, and most commonly reveal elevated hepatic enzymes (SAP 100%, GGT 86%, ALT 77%, AST 60%) and bilirubin (63%). Ultrasonographic findings may include: a distended gallbladder with centrally suspended luminal content and a hypoechoic intraluminal rim; a thickened gallbladder wall; intraluminal stellate; echogenic striations (the “kiwi fruit sign”); and/or the presence of non-dependent intraluminal contents or sludge. (Note: The presence of the “kiwi fruit sign” is not essential for the diagnosis of a gall bladder mucocèle,

as many mucocele variations do not demonstrate a complete stellate pattern.) A hypoechoic ring seen around the gallbladder may indicate wall edema or early rupture. Presence of free fluid, as well as localized, echogenic hepatic parenchyma and intra-abdominal fat, are also consistent with bile leakage and peritonitis. Pain is often noted upon interrogation of the area. Dilatation of the common bile duct may also be present.

Since there exists an association between hypercholesterolemia/hyperlipidemia and gallbladder mucoceles, patients that exhibit both should be screened for hypothyroidism, adrenal hyperplasia syndromes (Cushing's disease and sex hormone dysregulation), diabetes mellitus, pancreatitis, exogenous exposure to glucocorticoids, and necroinflammatory liver disease.

Treatment: Surgical intervention is recommended due to the significant risk of peritonitis and sepsis associated with rupture, and since medical management on its own may not be effective. Candidates for surgery must be adequately stabilized prior to surgery, and a coagulation panel should be assessed beforehand. Use of ursodeoxycholic acid (Actigall) is not recommended if any sonographic aspects of wall inflammation or emergent perforation are present. Percutaneous centesis of the gallbladder is also not advised in the presence of a mucocele. Some studies that have investigated a relatively small number of dogs have found medical therapy with Actigall and SAME to be somewhat effective at a very early subclinical stage. Yet, there are no definitive guidelines for ascertaining whether certain sonographic or laboratory findings permit the differentiation of a clinically significant mucocele versus a non-clinical, stable mucocele that may become clinically significant in the future. For this reason, each patient must be assessed individually. It may be the case that very dramatic mucoceles do not cause any overt clinical signs, whereas other patients may have minor mucoceles that lead to significant clinical signs and necessitate urgent cholecystectomy to avoid the onset of bile peritonitis due to rupture. It is generally advised that dogs with clinical signs, elevated liver enzymes, elevated WBC counts, and sonographic evidence of a mucocele be treated surgically. However, if a clinically silent mucocele is found incidentally on ultrasound, careful observation and monitoring is reasonable, provided there are no underlying diseases that may promote degradation (e.g., hyperadrenocorticism). Owners should be instructed to carefully monitor their pets for the development of clinical signs that may signal progression of the disease.

Antibiotic therapy directed towards gram-negative, enteric bacteria (i.e., *E. coli*, *Klebsiella*, *Enterobacter*, etc.) and anaerobic bacteria is indicated, and will likely be necessary for several weeks following surgery. Culture and sensitivity results should guide long-term treatment. Cholecystectomy with a possible bile duct transposition is the procedure of choice. The bile duct is often flushed to remove sludge and debris to help avoid post-surgical obstruction. Cholecystotomy for removing gallbladder contents is not advised as studies have shown that mucocele concretions have reformed in several dogs treated in this manner. At the time of surgery, one should obtain liver biopsies as well as submit cultures and sensitivity of the gallbladder contents. Liver biopsies may show normal tissue, cholangiohepatitis, biliary hyperplasia, vacuolar hepatopathy, or mild to moderate portal hepatitis/fibrosis with bile duct proliferation. Ultrasound-guided cholecystocentesis is not recommended in dogs with mature mucoceles due to the risk of gallbladder rupture and seeding the abdomen with bacteria. Rupture of the gallbladder constitutes a surgical emergency and carries a worse prognosis for survival. Several investigators have noted that, upon surgical exploration of asymptomatic mucocele

patients, there is evidence of prior localized peritonitis and fibrosis, likely resulting from tears in the gallbladder's neck, which lead to minute amounts of bile leakage.

Conclusion: Possible etiological explanations for gallbladder mucoceles are quite varied, and underlying diseases must be treated. The treatment of choice is cholecystectomy, with surgical intervention especially recommended for patients with clinical signs or significantly elevated liver enzymes so as to avoid gallbladder rupture and subsequent peritonitis.

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