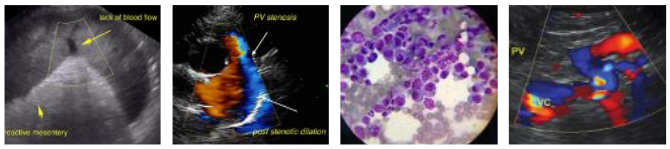

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1-800-838-4268 info@sonopath.com

PATIENT

Coco Filomeno

SPECIES

Canine

BREED

Dachshund

SEX

Male

AGE

7

WEIGHT

15

INTERPRETED BY

Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Jenn

HOSPITAL NAME

Rockaway AH

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Dr. Maniar

INVOICE

21569

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PRESENTING CLINICAL SIGNS

History: increased ALT and GGT, excessive teeth grinding at home, otherwise NSF

Abnormal PE/Chem/CBC/UA Results: ALT 160 GGT 12

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 pelvic urethra was imaged 2.0 cm beyond the cystourethral junction.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The right kidney measured 5.04 cm. The left kidney measured 5.04 cm.

Adrenal Glands

Both **adrenal glands** were visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 1.83 cm x 0.54 cm at the caudal pole and 0.64 cm at the cranial pole. The right adrenal gland measured 1.83 cm x 0.68 cm at the caudal pole and 0.97 cm at the cranial 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.

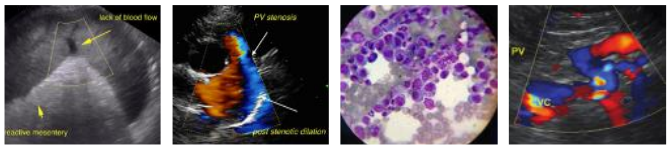
Liver

The **liver** images submitted revealed subjectively normal liver size, contour, and structure. Parenchymal echogenicity was naturally coarse and hypoechoic to the spleen. Vascular and biliary tracts were of normal volume with no evidence of congestion. The gallbladder presented acceptably thin walls with primarily anechoic content. The cystic and common bile ducts were normal. No pathological hepatic lymphadenopathy was evident. No overt structural evidence of inflammatory, infiltrative or regenerative pathology was evident.

Gastrointestinal

Some retention of ingesta was noted in the **stomach**, postprandial type presentation. The small intestine and colon were unremarkable.

Pancreas



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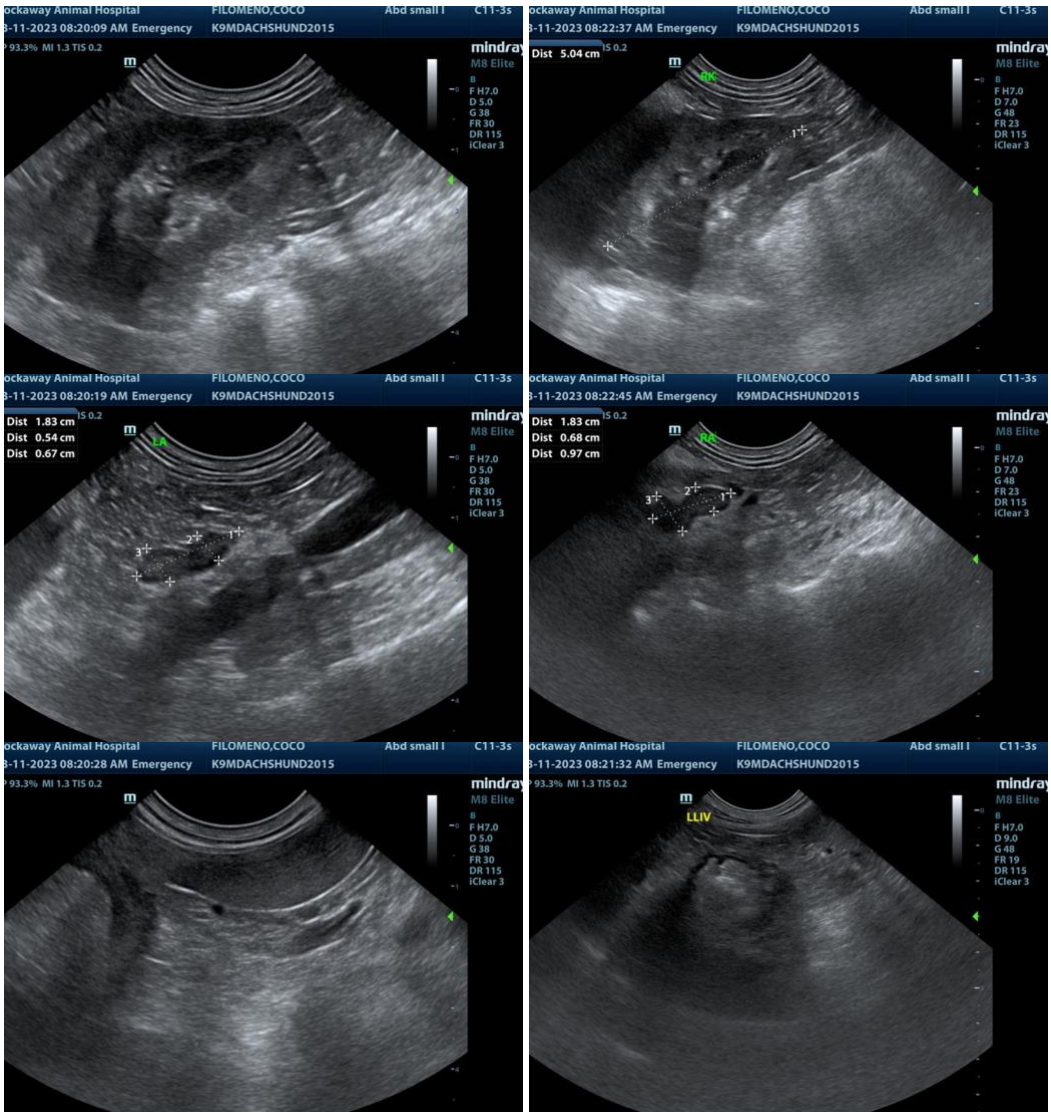
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

ULTRASONOGRAPHIC FINDINGS

- Retention of ingesta, postprandial presentation- no overt foreign matter

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

FNA of the liver is indicated for further definition, yet structurally the liver appears unremarkable. This is likely a reactive hepatopathy.



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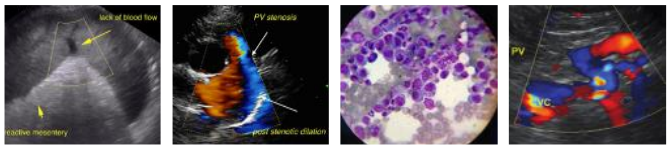

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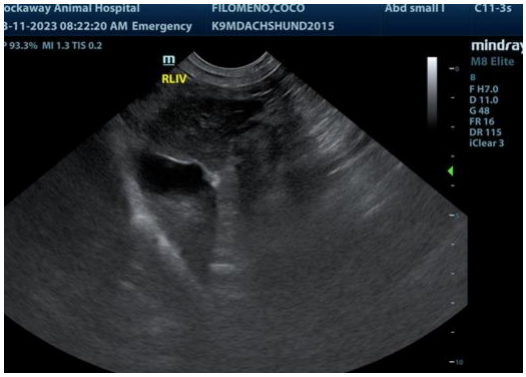
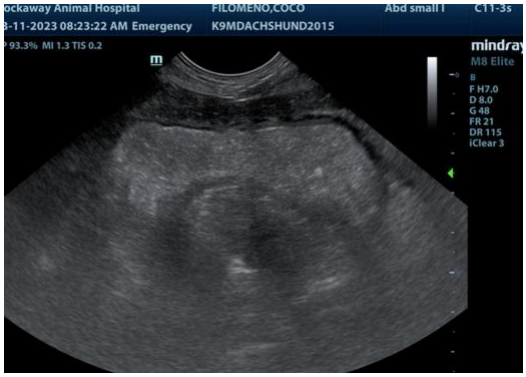




The information and recommendations provided are based on the images presented by the referring veterinarian. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

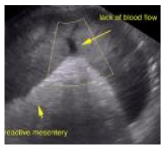
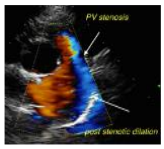
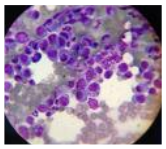
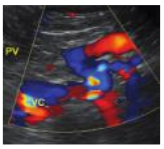
Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

Eric Lindquist, DMV, DABVP, Cert. IVUSS, CEO of SonoPath.com
info@SonoPath.com

Causes of Liver Enzyme Elevation in Dogs and Cats

http://www.sonopath.com/LiverEnzymes

Analyte	Dogs	Cats
Serum Alkaline Phosphatase (SAP) SAP is a specific brush border enzyme in the biliary tree, but is also present in other organs, such as the kidneys, intestines, bones, and placenta. An increase in SAP is caused by cholestasis and is more specific for clinical liver dysfunction in cats than in dogs. An elevation in SAP in dogs may occur in low-grade benign states, such as vacuolar hepatopathy, or can be the result of a significant disease process. Rapid	• Primary hepatic cholestasis ❖ vacuolar hepatopathy ❖ nodular hyperplasia ❖ neoplasia ❖ endocrinopathy ▪ hyperadrenocorticism ▪ diabetes mellitus ▪ hypothyroidism ❖ drugs (enzyme induction) ❖ hormonal influence ❖ chronic hepatitis ❖ infectious diseases ❖ inflammation ❖ toxicity ❖ hyperlipidemia ❖ reactive hepatopathy incited by other systemic diseases	• Primary hepatic cholestasis ❖ hepatic lipidosis ❖ inflammatory cholangitis ▪ neutrophilic ▪ chronic neutrophilic/ lymphoplasmacytic ❖ non-inflammatory parenchymal disease ❖ endocrinopathy ▪ diabetes mellitus ▪ hyperthyroidism ▪ hyperadrenocorticism ❖ neoplasia ❖ infectious ▪ bacterial ▪ FIP ▪ *Toxoplasma* ▪ *Bartonella*

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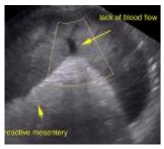
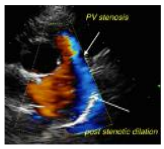
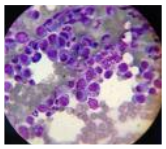
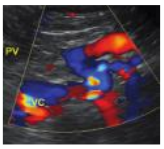
increases on serial blood work evaluations should prompt the clinician to initiate a more detailed workup. **Diagnostic Workup**: Signalment: • history • clinical signs • blood work • abdominal ultrasound • pre- and post-bile acids • low-dose dexamethasone suppression test and/or ACTH stimulation test (to assess both cortisol and other reproductive hormones) • fasting triglyceride levels (measure if the serum is lipemic to assess for hyperlipidemia) • serum and urine glucose levels (and/or fructosamine, as necessary) • assess for primary bone disease	▪ pancreatitis ▪ gastrointestinal ❖ passive congestion ▪ right-sided cardiac disease ▪ intra-thoracic disease causing elevated pressure in the caudal vena cava • Posthepatic cholestasis ❖ GB disease (sludge, mucocele, cholelithiasis) ❖ CBD obstructions ❖ pancreatitis ❖ neoplasia • Other ❖ primary bone disease	▪ parasitic ❖ toxicity ❖ amyloidosis ❖ reactive hepatopathy ❖ drugs • Posthepatic biliary disease ❖ GB disease ▪ cholangitis ▪ cholelithiasis ❖ CBD obstructions ❖ pancreatitis ❖ neoplasia
Serum Gamma-Glutamyl Transpeptidase (GGT) GGT is a liver-specific enzyme that is indicative of biliary disease and cholestasis, and typically rises in conjunction with SAP. A main exception occurs in cases of hepatic lipidosis in which the GGT is within normal limits while the SAP is disproportionately elevated.	• Same as SAP	• Same as SAP

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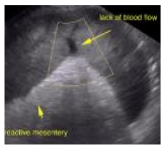
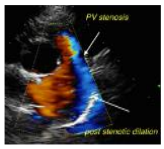
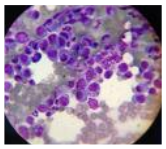
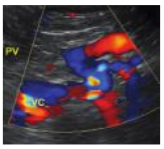
Serum Alanine Transferase (ALT) Serum ALT is a liver-specific enzyme derived from the cytoplasm of hepatocytes. Thus, an elevation reflects a leakage secondary to damage of the hepatocyte membrane. The degree of elevation does not always correspond to the degree of disease. Cytotoxic injury results in an increase within 24-48 hours, and will peak by day 5 and normalize over 2-3 weeks, provided the inciting cause resolves.	• Acute hepatitis ❖ infectious ❖ toxic • Chronic hepatitis • Reactive hepatopathy • Infectious diseases ❖ bacterial ❖ viral parasitic ❖ fungal • Cirrhosis • Pancreatitis • Neoplasia ❖ lymphoma ❖ adenocarcinoma ❖ mast cell ❖ other neoplasms • Endocrinopathy ❖ hyperadrenocorticism ❖ diabetes mellitus • Hypoxia • Toxins • Copper toxicity • Drugs • Trauma • Hemolysis • Portosystemic shunting (congenital and acquired) • Portal vein hypoplasia	• Inflammatory disease ❖ neutrophilic cholangitis ❖ chronic neutrophilic cholangitis/ lymphoplasmacytic cholangitis • Reactive hepatopathy • Pancreatitis • Neoplasia ❖ lymphoma ❖ adenocarcinoma ❖ other neoplasms • Infectious diseases ❖ bacterial ❖ FIP ❖ *Toxoplasma* ❖ *Bartonella* ❖ parasitic • Endocrinopathy ❖ hyperthyroidism • Hepatic lipidosis • Hypoxia • Toxins • Drugs • Trauma • Hemolysis • Portosystemic shunting (congenital and acquired)
Aspartate Transferase (AST) Serum AST is a non-specific liver enzyme that can also increase with intramuscular injections. If serum AST levels are much higher than those of serum ALT, then a muscular source should be investigated. AST originates in the mitochondria of the hepatocyte; its presence therefore reflects more serious damage to the cell.	• Hepatic parenchymal disease (see list for ALT) • Muscle disease • Hemolysis	• Hepatic parenchymal disease (see list for ALT) • Muscle disease • Hemolysis
Total Serum Bilirubin Bilirubin is an excretory waste product produced in the liver,	• Prehepatic ❖ hemolysis ▪ infectious ▪ immune-mediated ▪ toxic ▪ paraneoplastic	• Prehepatic: ❖ hemolysis ▪ infectious, especially *Mycoplasma* spp. ▪ immune-mediated ▪ toxic

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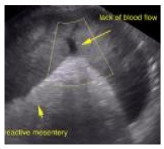
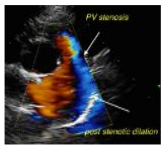
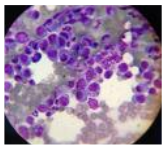
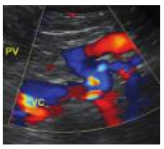
excreted into the common bile duct (CBD), and stored in the gallbladder (GB). Bile passes from the GB following a meal into the CBD via the cystic duct (CD). From the CBD, the bile passes into the duodenum through the duodenal papilla. Bile is conjugated by bacteria to become urobilirubin, which assists with fat digestion. It is partially absorbed in the ileum and therefore reenters the portal circulation. Unabsorbed bilirubin is excreted in the feces as stercobiliurubin. To evaluate for prehepatic causes of icterus, assess CBC for anemia. To evaluate for intrahepatic causes, assess liver enzymes and abdominal ultrasound for evidence of hepatic disease, and consider sampling the hepatic parenchyma. To evaluate for posthepatic causes, assess liver enzymes for evidence of hepatic disease and perform an ultrasound of the liver, paying particular attention to the biliary tree, CBD, and pancreas.	• Hepatic (parenchymal liver disease) ❖ cholangiohepatitis/cholangitis ❖ infectious disease ❖ chronic hepatitis ❖ neoplasia ▪ lymphoma ▪ adenocarcinoma ▪ adenoma ▪ mast cell disease ▪ miscellaneous neoplasms ❖ cirrhosis ❖ hypoxia ❖ toxicity ❖ drugs ❖ portosystemic shunting (congenital and acquired) ❖ portal vein hypoplasia • Posthepatic ❖ biliary obstruction (partial or complete) ▪ GB mucocele ▪ CBD plug ▪ cholelith in GB or BD ▪ neoplasia ▪ pancreatitis or neoplasia ▪ duodenal disease ▪ obstruction of the duodenal papilla/CBD	▪ paraneoplastic • Hepatic (parenchymal liver disease) ❖ cholangitis ▪ acute neutrophilic ▪ chronic neutrophilic/ lymphoplasmacytic ❖ infectious diseases ▪ bacterial ▪ FIP ▪ *Toxoplasma* ▪ *Bartonella* ▪ parasitic ❖ neoplasia ▪ lymphoma ▪ adenocarcinoma ▪ biliary cystadenoma ▪ adenocarcinoma ▪ miscellaneous neoplasia ❖ cirrhosis ❖ hypoxia ❖ toxicity ❖ drugs ❖ portosystemic shunting (congenital and acquired) ❖ portal vein hypoplasia. • Posthepatic ❖ biliary obstruction (partial or complete) ▪ GB mucocele ▪ CBD plug ▪ cholelith in GB or BD ▪ neoplasia ▪ pancreatitis or neoplasia ▪ duodenal disease ▪ obstruction of the duodenal papilla/CBD
Serum Bile Acids (SBAs) SBAs circulate within the enterohepatic system and are produced in the liver; they are reabsorbed in the ileum. Alterations in any step of this	• Hepatic (parenchymal liver disease) ❖ cholangiohepatitis/cholangitis ❖ infectious diseases ❖ chronic hepatitis ❖ neoplasia ▪ lymphoma ▪ adenocarcinoma ▪ adenoma ▪ mast cell disease	• Hepatic (parenchymal liver disease) ❖ cholangitis ▪ acute neutrophilic ▪ chronic neutrophilic/ lymphoplasmacytic ❖ infectious diseases ▪ bacterial ▪ FIP ▪ *Toxoplasma*

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enterohepatic cycle may cause SBA elevations. In other words, SBAs are not hepatic-specific. An SBA test is clinically redundant in the face of hyperbilirubinemia.	▪ miscellaneous neoplasms ❖ cirrhosis ❖ hypoxia ❖ toxicity ❖ portosystemic shunting (congenital and acquired) ❖ portal vein hypoplasia • Posthepatic ❖ biliary obstruction (partial or complete) ▪ GB mucocele ▪ CBD plug ▪ cholelith in GB or BD ▪ neoplasia ▪ pancreatitis or neoplasia ▪ duodenal disease ▪ obstruction of the duodenal papilla/CBD • Nonhepatic disease ❖ resection or disease of the ileum ❖ spontaneous GB contraction ❖ variability in gastric emptying ❖ idiopathic breed variation (e.g. Maltese) ❖ inadequate fat and amino acid content in the test meal ❖ drug therapy (ursodiol)	▪ *Bartonella* ▪ parasitic ❖ neoplasia ▪ lymphoma ▪ adenocarcinoma ▪ biliary cystadenoma ▪ adenocarcinoma ▪ miscellaneous neoplasia ❖ cirrhosis ❖ hypoxia ❖ toxicity ❖ drugs ❖ portosystemic shunting (congenital and acquired) ❖ portal vein hypoplasia • Posthepatic ❖ biliary obstruction (partial or complete) ▪ GB mucocele ▪ CBD plug ▪ cholelith in GB or BD ▪ neoplasia ▪ pancreatitis or neoplasia ▪ duodenal disease ▪ obstruction of the duodenal papilla/CBD • Nonhepatic disease ❖ resection or disease of the ileum ❖ spontaneous GB contraction ❖ variability in gastric emptying ❖ inadequate fat and amino acid content in the test meal ❖ drug therapy (ursodiol)