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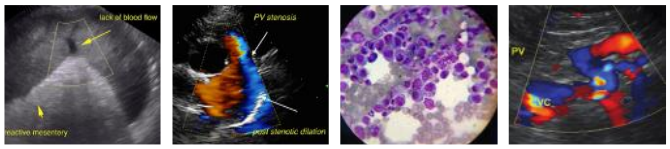

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1-800-838-4268 info@sonopath.com

PATIENT
Riggs Mills

SPECIES
Canine

BREED
Chihuahua

SEX
MN

AGE
10mo

WEIGHT
3.3kg

INTERPRETED BY
R. McKenzie Daniel, DVM, DABVP (Canine and Feline)

IMAGING PERFORMED BY
Dr. Belan

HOSPITAL NAME
Legacy Vet Clinic

REFERRING VET
Dr. Jajouei

INVOICE
12617ag

DATE
01/06/2023

PRESENTING CLINICAL SIGNS

Suspect PSS or MHVD

Abnormal PE/Chem/CBC/UA Results: Elevated ALT -mild and mild elevation of bile acids pre prandial 2.6 range 0-14 and post prandial 54 range 0-29

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The urinary bladder, trigone, cystourethral junction, and visible pelvic urethra exhibited normal thickness and tone. Anechoic urine was present in the lumen with no uroliths or sediment. The ureteral papillae were normal. The ureters were not visible which is normal. No evidence of inflammatory or neoplastic changes were noted.

Normal size and margination were present in the kidneys. A normal 1:3 cortex / medulla ratio and normal corticomedullary definition were maintained. The echogenicity of the cortex was similar to or slightly less than normal liver parenchyma while the medulla echogenicity was hypoechoic to the cortex with no evidence of pelvic dilation. No overt evidence of renomegaly or calculi. The left kidney measured 3.4 cm in length. The right kidney measured 3.5 cm in length.

The area of the aortic trifurcation was free of pathology.

Adrenal Glands

The left adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The left adrenal gland measured 0.27 cm width at the caudal pole and 0.29 cm width at the cranial pole. The right adrenal gland was uniform in size and contour with a uniformly hypoechoic parenchyma. The right adrenal gland measured 0.28 cm width at the caudal pole and 0.34 cm width at the cranial pole.

Spleen

The spleen exhibited a finely textured and homogenous parenchyma which was hyperechoic to the liver and renal cortical parenchyma. The capsule was smooth and regular without apparent expansion. The splenic vasculature at the hilus was normal in volume with no evidence of congestion or thrombosis. Acute to chronic inflammatory, neoplastic, or benign parenchyma changes were not noted.

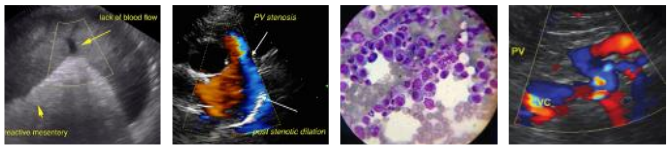
Liver/Gallbladder

The liver was normal in size exhibiting subjective normal to adequate hepatoportal vascular volume. Normal hepatic parenchyma echogenicity with mild to moderate coarse echotexture was present. The visualized portal vein exhibited subjective potential for mild reduced volume yet subjective normal cranial branching. No overt evidence of anomalous vessel associated with the portal vein. Concurrent normal caudal vena cava volume with subjective normal laminal cranial flow was present.

The gallbladder was non-distended in size with thin walls and primarily anechoic luminal content. The cystic and common bile ducts were normal.

Gastrointestinal

The stomach presented wall thickening secondary to echogenic mucosa hypertrophy. Intact wall layering was maintained and distinct. Mild gastric distension with primarily anechoic fluid and luminal gas was present.



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The small intestine presented intact wall layering with 1:3 muscularis/mucosa ratio. The lumen of the small intestine was empty with no signs of ileus, obstruction or foreign material.

Normal visible colon wall layers were present with apparent formed feces in lumen.

Pancreas

The parenchyma of the left limb, body and right limb of the pancreas presented isoechoic to the adjacent omental fat. A normal curvilinear capsule contour of the pancreas was present. The visible pancreatic duct was normal. No signs of active inflammation or neoplastic disease was evident.

Free Abdomen

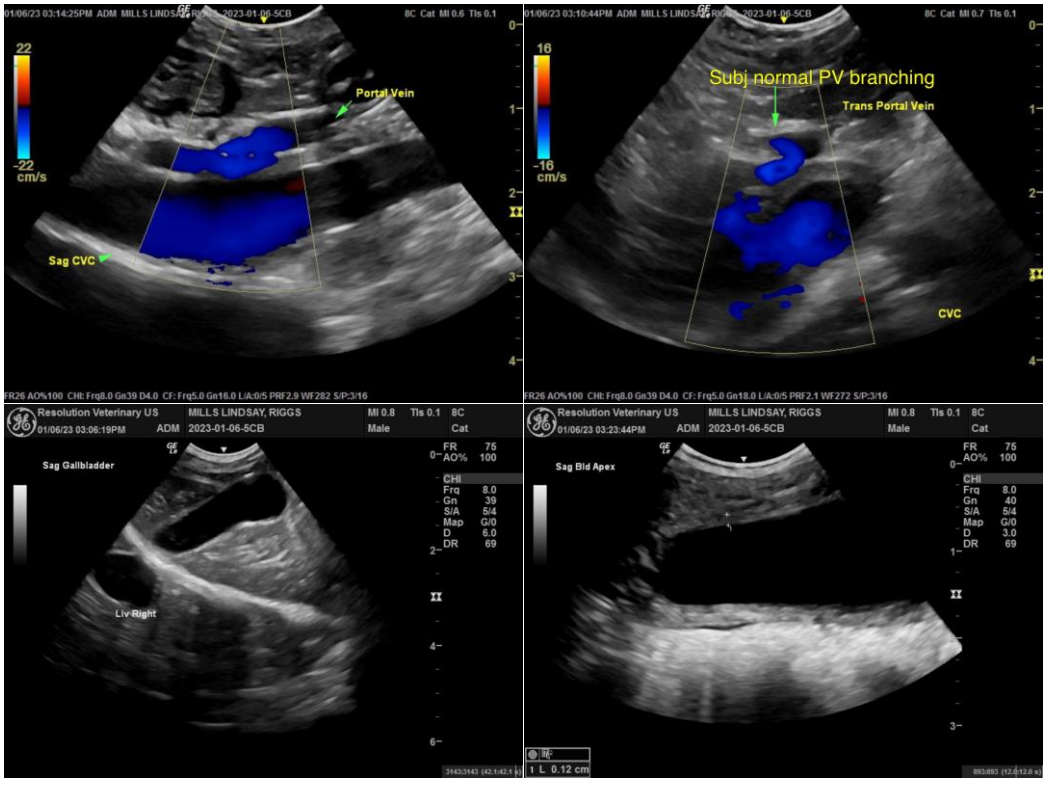
No omental masses, overt lymphadenopathy or peritoneal effusion was present.

ULTRASONOGRAPHIC FINDINGS

- Normal hepatic vascular volume-potential low-grade inflammatory hepatopathy or portal hypoplasia/microvascular dysplasia

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

No overt or obvious evidence of intrahepatic or extrahepatic shunting was present. Assuming normal clotting status and using a 25g needle, a hepatic FNA for screening cytology could be considered to assess possible inflammatory cell type. Core surgical biopsy may be necessary for further definition as to whether portal hypoplasia/microvascular dysplasia vs primary hepatic disease is present. A clinical trial of some or all of the following could be considered if clinically indicated. The patient is overtly negative for macroscopic portosystemic shunting yet if clinical signs suggestive of a shunt are present, gold standard CT with contrast may be considered for definitive assessment.



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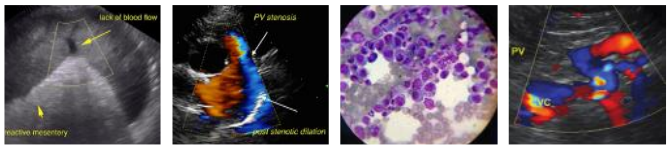

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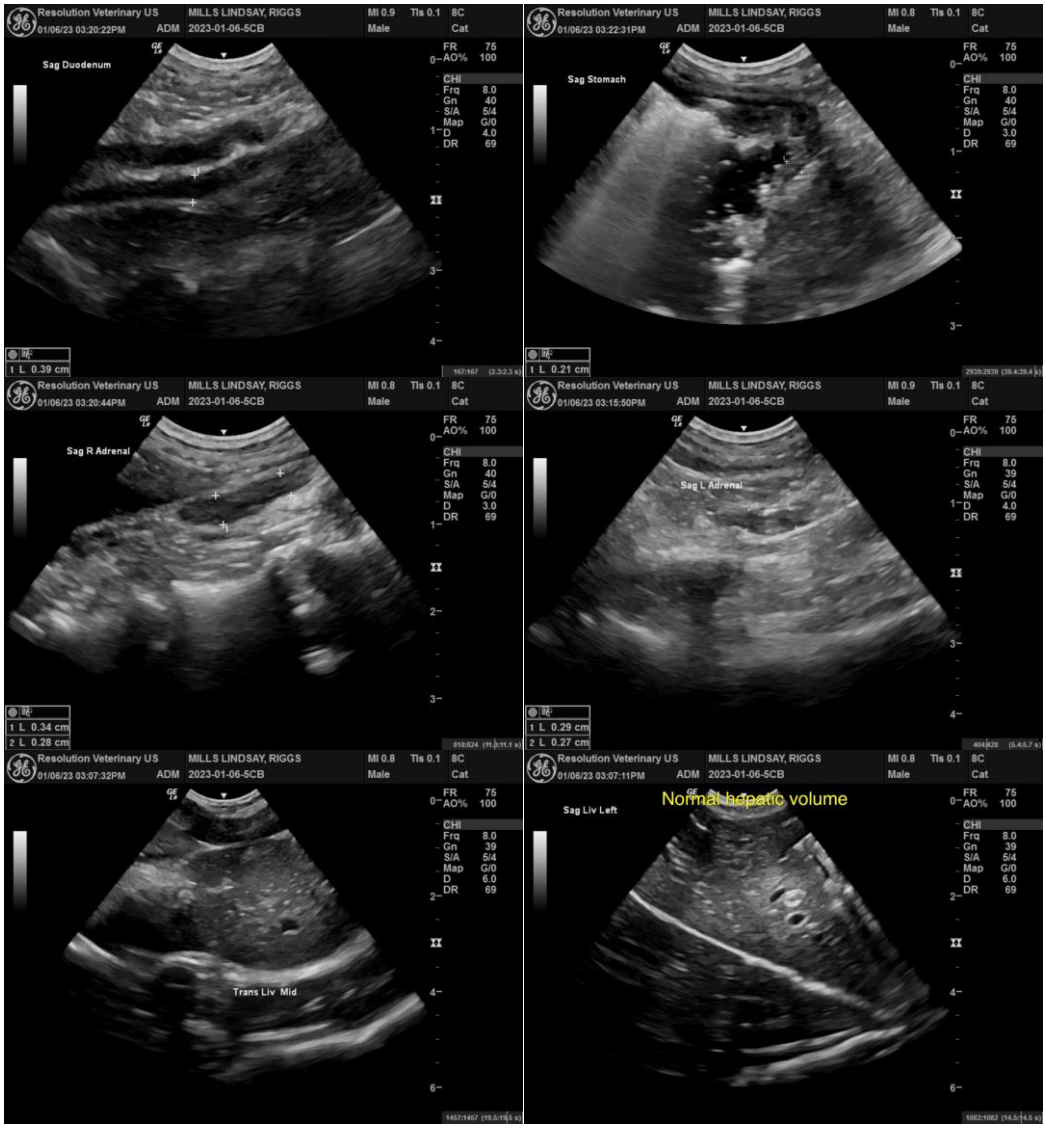


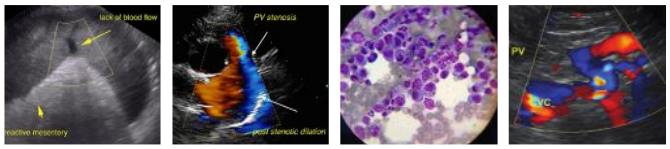
The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

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.

R. McKenzie Daniel, DVM, DABVP (Canine/Feline Practice)
mac.daniel@sonopath.com

Bile Acid Elevations and Hepatic Vascular Disorders:
Portosystemic Shunts and Portal Vein Hypoplasia (Microvascular Dysplasia)

http://www.sonopath.com/BAShunts



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Non-Shunt Pathologies and Elevated Bile Acid Levels

Description: Bile acids are conjugated with cholesterol in the liver; they then enter the biliary tree and are stored in the gallbladder. Under the stimulation of cholecystokinin, the gallbladder contracts and bile acids are released from the cystic duct into the common bile duct; they then pass through the sphincter of Oddi to reach the duodenum. Bile acids are absorbed primarily in the ileum (95%), and then reenter the portal system and move into the liver. This enterohepatic circulation cycle can occur 2-5 times within the space of a single meal. When bile flow is obstructed and the bile secretory pressure reaches 30 cm H_20, bile acids accumulate in the blood. Obstruction can occur due to calculi, the accumulation of acids (also known as "bile sludge") in the common bile duct, or extrahepatic obstruction, such as pancreatitis. Unconjugated bile acids are cytotoxic and result in inflammation, intestinal necrosis, poor permeability, bacterial translocation, sepsis, endotoxemia, poor micelle formation, and a deficiency of fat-soluble vitamins.

Causes of Bile Acid Elevation:

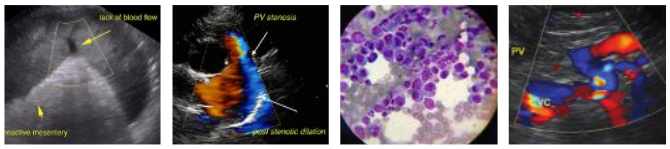
1. Nonhepatic Causes
Inflammatory bowel disease or intestinal dysbiosis
Delayed gastric emptying
Spontaneous gallbladder contraction
Hypertriglyceridemia or lipemia
Ursodeoxycholic acid treatment
Severe disease or resection of the ileum (site of bile acid reabsorption)
Cholecystectomy
Prolonged anorexia
Hyperadrenocorticism
Pancreatitis
Transient elevation, which occurs most commonly in Irish wolfhound puppies

2. Hepatic Causes
Diffuse hepatocellular disease
Cholestatic disease
Primary portal vein hypoplasia or microvascular dysplasia

Hepatic Vascular Diseases

Description: Hepatic vascular diseases can be divided into congenital and acquired forms. Congenital disorders include: portosystemic shunting (PSS) or portosystemic vascular anomalies (PSVA), both intrahepatic (IHPSS) and extrahepatic (EHPSS); microhepatic PSS, also called portal vein hypoplasia (PVH) (previously known as microvascular dysplasia [MVD]) without portal hypertension; portal vein atresia; and hepatic arteriovenous (AV) malformations. Acquired forms include: acquired shunting secondary to portal hypertension due to primary hepatic disease; fibrosis/cirrhosis; and non-cirrhotic portal hypertension. Although PSVA can result in elevated liver enzymes and bile acids, other possible causes for elevated bile acids include, but are not limited to: diffuse hepatocellular disease; cholestatic disease; cholecystectomy; spontaneous gallbladder contraction; ursodeoxycholic acid use; inflammatory bowel disease; hyperlipidemia; prolonged anorexia; hyperadrenocorticism; pancreatitis; severe ileal disease or resection; delayed gastric emptying; prolonged or rapid intestinal transit time; small intestinal bacterial overgrowth; and breed-associated increases, as observed in the Maltese breed, for example, in the absence of primary hepatic disease. Given the long list of differentials, the assessment for PSVA often depends on the clinical presentation, such as signalment, clinical signs, and specific laboratory findings, which may suggest PSVA. Ultrasound and additional diagnostics are imperative in the diagnostic process.

The following canine breeds—typically small breed dogs—are predisposed to congenital extrahepatic



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shunting: Miniature Schnauzer, Yorkshire Terrier, Pug, Dachshund, Cairn Terrier, Shih Tzu, West Highland White Terrier, Bichon Frisé, Havanese, Dandie Dinmonts, and Maltese. Extrahepatic shunts often involve a shunt from the portal vein (PV), left gastric, or splenic vein, to the caudal vena cava. The shunt may occasionally enter the azygous vein dorsally, bypassing the vena cava (VC). The following breeds—typically large breed dogs—are predisposed to intrahepatic shunting: Irish Wolfhound, Australian Cattle Dog, Australian Shepherd, Golden Retriever, Old English Sheepdog, and Labrador Retriever. Intrahepatic shunting in the latter breeds most commonly presents as a shunt between the PV and the caudal vena cava, and may coexist with PVH. Yorkshire Terriers and Cairn Terriers are predisposed to PVH.

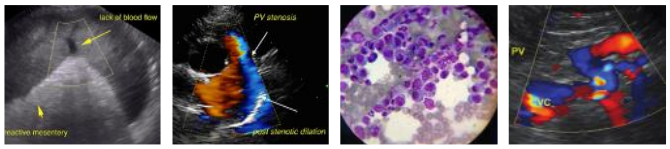
PVSA are not seen as commonly in cats compared to dogs. In cats, extrahepatic PSVA usually arise from the left gastric vein; they also often have a patent ductus venosus. The following feline breeds are predisposed to PVSA: domestic shorthair, Persian, Siamese, Himalayan, and Burman.

Clinical Signs: Dogs affected with PVH uniquely are typically asymptomatic and their hepatic vascular abnormalities are non-progressive; however, patients with severe PVH may sometimes display clinical signs similar to those with PSVA.

A patient with PSVA is often more symptomatic; clinical findings vary. Dogs and cats with PSVA often have smaller bodies compared to their litter mates, and may exhibit anorexia, vomiting, diarrhea, depression, lethargy, ataxia, head pressing, “stargazing,” behavioral changes, seizures, and/or coma. Drooling is common in cats, but can be seen in dogs as well. Renomegaly is common in patients with PSVA, and polyuria and polydipsia (PU/PD) can occur due to low BUN in the face of hepatic insufficiency. Signs of lower urinary tract disease manifest if urate calculi have formed. Animals with PSVA also have an increased susceptibility to infections due to reduced Kupffer cell function. Minor bite wounds, tick bites, subcutaneous infections, lacerations, and even vaccinations may cause illness that can require hospitalization. Cats with PSVA may have copper-colored irises (36%). Dogs with portoazygous shunts are generally the least symptomatic and frequently present with ammonium biurate calculi as adults; their disorder is often discovered serendipitously. Generally, asymptomatic dogs (15-20%) whose PSVA is only detected later in life usually respond well to PSVA ligation. Acquired shunting may occur later in life secondary to chronic hepatic disease and can result in portal hypertension and ascites.

Diagnostics: Clinicopathologic findings for both PSVA and PVH may include mild hypoalbuminemia, hypoglycemia, hypocholesterolemia, microcytosis (low MCV), and hypochromasia. One may also note the following: borderline, non-regenerative anemia; target cells; low BUN; low creatinine; normal to variable increases in liver enzymes (mild to modest); and ammonium biurate crystalluria (a minimum of 3 urine specimens should be examined). Radiographic findings may include microhepatica in dogs; however, liver size is variable in cats, and kidneys may be large in both species. Contrast portography yields varying patterns in patients with PSVA. Fasting plasma ammonium determination is more sensitive than bile acid profiles when gauging the presence of either congenital or acquired shunting; however, ammonium levels must be measured immediately upon collecting blood in a lithium heparin tube. The ammonium tolerance test or baseline ammonium level measurement is not practical if in-house testing is not available. Most dogs with PSVA have postprandial bile acid concentrations greater than 100 nmol/L, but values do not correlate with the severity of the disease. Dogs with PSVA have lower clotting factor activity than healthy dogs; this can cause complications during surgery. Protein C is an anti-thrombotic protein that is synthesized in the liver; it is used as a hepatic function test in people and is a better indicator of portal venous flow than total serum bile acids. In combination with serum bile acids, it can help differentiate PSVA from PVH, as dogs with PVH will have more normal protein C levels than those with PSVA. Markedly low levels of protein C suggest that a patient is likely a poor candidate for surgical ligation and also help identify dogs with hepatic failure.

Treatment: The majority of dogs affected with PVH alone do not require medical treatment and have a normal life expectancy. The severity of clinical signs in symptomatic PSVA patients is highly variable and can be regulated in large part by an appropriately formulated low-protein diet. Surgical treatment for



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PSVA is the subject of much debate; however, a recent study confirmed that long-term survivability was improved by surgical correction. Medical management remains a reasonable alternative. If surgery is to be pursued, it should be considered in light of comorbidities that influence hepatic integrity. Extrahepatic shunts are more accessible and therefore more amenable to ameroid ring constriction or shunt ligation, while intrahepatic shunts are often difficult to access surgically, as they are positioned deep within the liver parenchyma but may be closed with coil embolization under fluoroscopic guidance. Other considerations include whether the patient should be stabilized medically before surgery is attempted or if full recovery is to be expected once the PSVA is closed. The most common and severe complications of surgical ligation include portal hypertension and ascites, which is why slow attenuation via ameroid ring placement is often preferred, as well as the development of seizures/status epilepticus. Seizure development cannot always be predicted and is more common in small breed dogs, especially Maltese, and in cats.

The medical management of PSVA primarily involves restricting dietary protein (2.2-2.5 g/kg/day of protein, administered in small, frequent meals). Protein sources such as dairy, soy, and egg are enriched in branched-chain amino acids, which bypass liver metabolism and help reduce blood ammonia levels. Unsuccessful medical management is determined by recurrent hepatic encephalopathy or persistent ammonium biurate crystalluria. In both cases, if the animal has PSVA, one should consider surgical intervention or additional medical therapy. Lactulose should be started at a low dose (0.25 ml-1 ml/kg BID-TID) and titrated to achieve several soft stools per day. It acidifies the pH in the colon, which reduces urease activity and reduces urease-producing bacteria. Antibiotics, such as metronidazole (7.5 mg/kg PO BID) and neomycin (22 mg/kg PO BID), are utilized to modify enteric flora and reduce toxin production from urease-producing bacteria. Dogs with unresponsive hepatic encephalopathy are also managed with retention enemas (5-10 ml/kg with 20% lactulose), which rapidly acidify colonic contents.

Conclusion: PSVA and PVH are not uncommon in veterinary medicine. Medical therapy as well as surgical correction must be considered carefully in light of clinical presentation and shunt location. In all cases, dietary modification is the first-line treatment of choice; however, mild cases of PVH may not even require diet change.

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