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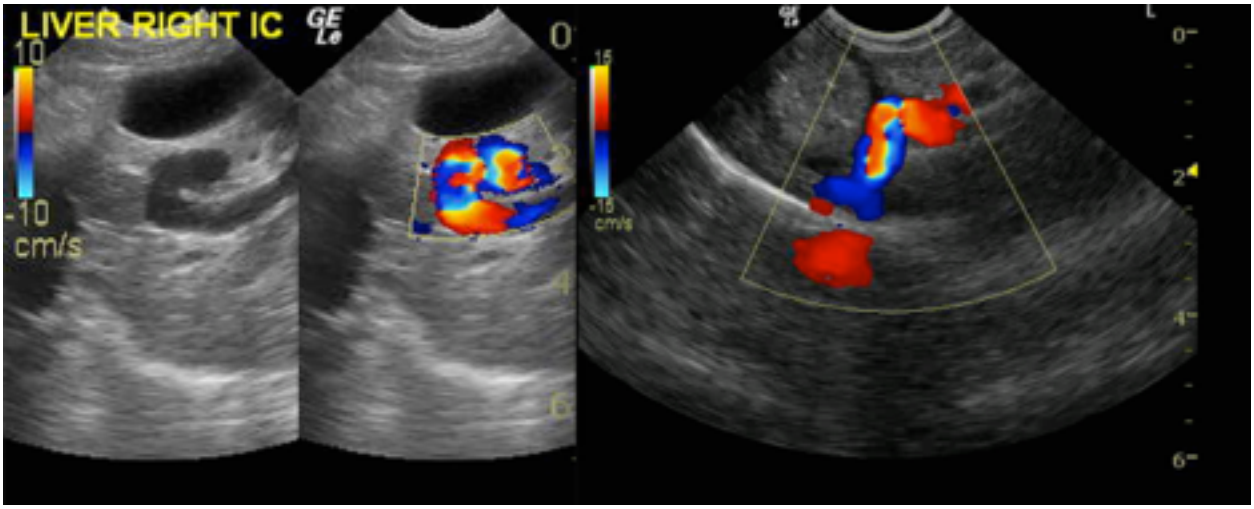
PORTOSYSTEMIC SHUNTS and MICROVASCULAR DYSPLASIA



Description: Microvascular dysplasia (MVD) is far more common in the patient population than **portosystemic shunts** (PSVA). However, both must be considered in light of increased bile acids. **Other possibilities** as a cause for elevated bile acids include but are not limited to inflammatory bowel disease, hyperlipidemia, prolonged anorexia, hyperadrenocorticism, pancreatitis, severe ileal disease or resection, and transient elevation in some breeds. **Non-vascular hepatic considerations** include diffuse hepatocellular disease, cholestatic disease, cholecystectomy, spontaneous gall bladder contraction, ursodeoxycholic acid use, and primary portal vein (PV) hypoplasia.

Breed predispositions toward **extrahepatic** shunting include miniature schnauzer, Yorkshire terrier, pug, dachshund, Cairn terrier, Shih tzu, West Highland white terrier, bichon frise, and Maltese. Extrahepatic shunts often involve a shunt from the PV or left gastric or splenic vein to the caudal vena cava. The shunt occasionally may enter the azygous vein dorsally, bypassing the VC. In cats, an extra-hepatic PSVA commonly arises from the left gastric vein. Breed predispositions toward **intrahepatic** shunting include Irish wolfhound, Australian cattle dog, golden retriever, Old English sheepdog, and Labrador retriever. These shunts most commonly involve a shunt between the portal vein and the caudal vena cava, and may coexist with portal vein hypoplasia. **Cats** with PSVA commonly have a patent ductus venosus.

Intrahepatic shunts are often difficult to access surgically as they are positioned deep within the liver parenchyma.

Clinical Signs: Dogs affected only with MVD are typically asymptomatic and their hepatic vascular abnormalities non-progressive. **Less commonly,** ascites and portal hypertension may be noted with acquired shunts.

A patient with PSVA is more often symptomatic, and clinical findings are varied. Dogs and cats with PSVA often have **small body size** compared to litter mates; **neurologic** signs such as anorexia, depression, lethargy, ataxia, head pressing, "stargazing," behavioral changes, seizures, and coma; **gastrointestinal** signs including anorexia, vomiting, and diarrhea. Drooling can be observed in cats. The **urinary tract** may also be affected, as dogs can be polyuric/polydipsic and have swollen kidneys. They may have lower urinary tract signs if urate calculi have formed. PSVA animals also have i**ncreased susceptibility to infections** due to reduced Kupffer cell function. Minor bite wounds, tick bites, subcutaneous infections, lacerations, and even vaccinations may lead to illness requiring hospitalization. Cats with PSVA may have **copper-colored irises** (36%). Dogs with **portoazygous shunts** are generally least symptomatic and often present as adults with ammonium biurate calculi or have their disorder serendipitously discovered. Generally, dogs discovered to have a PSVA later in life have been asymptomatic and often have a good response to PSVA ligation

Diagnostics: Many MVD-affected dogs are discovered serendipitously at the time of unrelated illnesses, and 15-20% are likely asymptomatic. **Clinicopathologic findings** for both PSVA and MVD may include hypoalbuminemia, hypoglycemia, hypocholesterolemia, microcytosis, and hypochromasia. Borderline non-regenerative anemia, low MCV, target cells, low blood urea nitrogen, low creatinine, hypocholesterolemia and slight hypoalbuminemia, normal to variable increases in liver enzymes (mild to modest), low to low-normal glucose concentration, and ammonium biurate crystalluria (examine a minimum of 3 urine specimens) may also be noted. **Radiographic findings** may include microhepatica in dogs, but liver size is variable in cats. Kidneys may be large in both species. **Contrast portography** yields varying patterns in patients with a PSVA. **Fasting plasma ammonium determination is more sensitive than bile acid profiles** in detecting the presence of either congenital or acquired shunting. Most dogs with portosystemic shunts have postprandial bile acid concentrations of more 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, and this can cause complications at surgery. **Protein C** is an anti-thrombotic protein which is synthesized in the liver that is used as a hepatic function test in people and is a better indicator of portal venous flow than total serum bile acids. Studies are currently underway to validate this test in dogs and cats.

Treatment: The majority of dogs affected only with MVD do not need medical treatments and have a normal life expectancy. The severity of clinical signs in symptomatic PSVA patients is highly variable and is largely modified by feeding an appropriately formulated diet. **Surgical treatment** of a PSVA is currently under much discussion, and if it is to be pursued, 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 similar surgeries, while intrahpatic shunts require availability of fluoroscopic closure evaluation. Other considerations include whether the patient should be stabilized medically before surgery is attempted, or whether full recovery is to be expected with closure of a PSVA. **Medical management** of PSVA primarily involves restriction of the dietary protein allowance (2.2 to 2.5 gm/kg body weight per day of protein, fed 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, and in the case of PSVA, surgical intervention or additional medical therapy should be considered at this point. **Lactulose** is started at a low dose (0.5 ml per 5-10 kg PO B-TID) and titrated to achieve several soft stools per day. **Metronidazole** (7.5 mg/kg PO Q24H to BID) can be used if diet and lactulose are insufficient. Alternatively, amoxicillin-clavulanate can be used to modify enteric flora if the patient is intolerant of

metronidazole. **Zinc** can also be added (1-5 mg/kg elemental zinc). Dogs with **MVD** do not require such medical management, and are expected to live a normal lifespan as these lesions are non-progressive. However, coexisting pathologies such as inflammatory bowel disease should be treated.

Conclusion: Portosystemic vascular anomalies and microvascular dysplasia are not uncommonly encountered in veterinary medicine. Medical therapy as well as surgical correction must be considered carefully in light of clinical presentation as well as shunt location. In all cases, dietary modification is the first-line treatment of choice; however, some cases of microvascular dysplasia may not require even diet change.

References:

Lamb CR, Daniel GB. Diagnostic imaging of dogs with suspected portosystemic shunting. Compend Contin Educ Pract Vet. August 2002;24(8):626-635.

Allen L, Stobie D, et al. Clinicopathologic features of dogs with hepatic microvascular dysplasia with and without portosystemic shunts: 42 cases (1991–1996). J Am Vet Med Assoc. January 1999;214(2):218-20.

Gerrizen-Bruning MJ, van den Ingh TS, Rothuizen J. Diagnostic value of fasting plasma ammonia and bile acid concentrations in the identification of portosystemic shunting in dogs. J Vet Intern Med. 2006 Jan-Feb;20(1):13-9.

Toulza O, Center S, Brooks M, et al: Evaluation of plasma protein C activity for detection of hepatobiliary disease and portosystemic shunting in dogs. J Am Vet Med Assoc. December 2006;229(11):1761-71.

Windsor RC, Olby NJ. Congenital portosystemic shunts in five mature dogs with neurological signs. J Am Anim Hosp Assoc. 2007 Nov-Dec;43(6):322-31.

Hunt GB. Effect of breed anatomy of portosystemic shunts resulting from congenital diseases in dogs and cats: A review of 242 cases. Aust Vet J. December 2004;82(12):746-9.

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