

## NC STATE UNIVERSITY

### Case #19

### Slide #: 17-325-4

**Institution:** North Carolina State University

**Signalment:** 8-month, male intact Labrador Retriever

### History:

The dog presented to the NCSU Small Animal Internal Medicine Service for evaluation of a 4 month period of daily vomiting, frequent diarrhea, depression following meals, and lack of weight gain. Serum chemistry revealed elevations in activity of ALP (204IU/L) and ALT (76IU/L); serum ammonia concentration was elevated at 40umol/L. Complete blood count revealed a moderate, normocytic, normochromic anemia with a hematocrit of 24.6%. A CT confirmed the presence of a large arterioportal malformation centered at the gallbladder and falciform fat as well as multiple acquired extrahepatic portosystemic shunts, moderate ascites, and left-sided liver atrophy.

### Gross findings:

A large mass of vessels was seen surrounding gallbladder and infiltrating into the liver at surgery. A biopsy including the gallbladder, vessels, and adjacent hepatic parenchyma was submitted for histopathology.

### Histopathology:

Liver, gall bladder: Two sections of liver and gallbladder are examined. Surrounding the common bile duct and gall bladder is a mass-like proliferation of numerous tortuous, small to large, well-differentiated arteries and veins, which rarely anastomose, and dissect or compress the hepatic parenchyma. The endothelium of veins and arteries is occasionally hypertrophied. Arterial and arteriolar walls are diffusely moderately thickened by subintimal edema, pale basophilic, mucinous matrix, and irregular fibrosis as well as by moderate to marked smooth muscle hypertrophy of the tunica media. The internal elastic lamina is frequently fragmented or absent (confirmed by Verhoeff-Van Gieson stain). The walls of veins and venules are markedly thickened and resemble arteries (arterialization). There is expansion of the tunica intima by irregular fibrosis and the tunica media by smooth muscle hypertrophy. The lymphatics surrounding the abnormal arteries and veins, those deep to the hepatic capsule, within portal tracts, and surrounding central veins are frequently moderately to markedly ectatic. The adjacent hepatic parenchyma is compressed by the vessels, with multifocal atrophy of the hepatic lobules. Additionally, there are markedly increased numbers of bile duct profiles in portal tracts and isolated within the hepatic parenchyma; these bile ducts are frequently surrounded by concentric lamellar fibrosis ("onion-skinning"). Portal tracts also have increased numbers of small arteriolar profiles and portal vein profiles are largely absent. Centrilobular hepatocytes are frequently atrophied and diffusely, hepatocytes are mildly to moderately swollen by diaphanous vacuolar change (glycogen type). Scattered throughout the parenchyma are individual necrotic hepatocytes. There is moderate stellate cell hypertrophy; hypertrophied stellate cells frequently contain large, clear, spherical vacuoles (lipid).

### Morphologic Diagnosis:

Liver, gallbladder: Arteriovenous fistula with multifocal hepatic compression and atrophy, marked biliary hyperplasia, portal vein hypoperfusion, and moderate, diffuse glycogen type hepatic vacuolation

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### Discussion:

The diagnostic imaging, gross, and histologic findings of this biopsy sample are consistent with a diagnosis of a hepatic arteriovenous fistula. Arteriovenous (AV) fistulae can be congenital or acquired; acquired AV fistulae are documented to occur following trauma, rupture of arterial aneurysms into a vein, inflammation or necrosis of adjacent vessels, and iatrogenically (typically surgical intervention). AV fistulae have been reported in a variety of locations, including intrahepatic, pulmonary, and on the limbs. They have been reported in dogs, cats, horses, cattle, and humans.

Clinical signs of AV fistulae depend on the location; typically signs are associated with increased hydrostatic pressure within veins and subsequent increased intra-lymphatic pressure. In fistulae involving abnormal blood flow to the liver, ascites, hepatic encephalopathy and synthetic liver failure can occur, resulting in depression, seizures, vomiting, and diarrhea.

Gross findings of AV fistulae usually show one or more anastomoses of a large vein and large artery, surrounded by a tangle of medium and small arteries and venules that contribute to a mass effect. In hepatic arteriovenous fistulae, microhepatia is commonly observed due to abnormal portal blood flow. Portal hypertension may result in the development of numerous portocaval acquired shunts.

Histology of AV fistulae reveals mass-like proliferations of abnormal arteries and veins, with arterialization of veins. The internal elastic lamina is frequently fragmented, split, or duplicated; in both veins and arteries, the tunica intima is expanded by smooth muscle invasion and hypertrophy, increased deposition of elastin fibers, edema, and mucinous matrix. The tunica media of veins and arteries also exhibits smooth muscle hypertrophy. Additionally, fibrinoid necrosis may be observed. Elastin (Verhoeff-Van Gieson) and trichrome (Gomori's trichrome) stains highlight the deposition of elastin fibers and fiber fragmentation as well as the fibrosis that often accompanies the fistulae. In fistulae that affect the liver, lesions resembling portocaval shunting occur, including absence or diminution of portal veins, marked biliary and arteriolar proliferation, hepatocyte atrophy, and lymphangiectasia. Sinusoids near the fistulae are frequently dilated and congested. Liver lobes away from the fistulae may be normal with little parenchymal change.

### References

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