Current Concepts in Congenital Portosystemic Shunts

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KEYWORDS

• Shunt • Extrahepatic • Intrahepatic • Surgical management • Embolization

KEY POINTS

- Protein C level testing may be a useful blood test to indicate liver dysfunction.
- Many methods of diagnostic imaging are available, including ultrasonography, nuclear scintigraphy, CT angiography, and MR angiography.
- Commonly performed open surgical procedures for congenital portosystemic shunt (CPSS) include the placement of ameroid constrictors or cellophane bands.
- Less invasive options for the treatment of intrahepatic and extrahepatic CPSS include laparoscopic placement of cellophane bands and interventional radiologic techniques such as coil embolization.

INTRODUCTION

This article focuses on current concepts in portosystemic shunts. A thorough review of portosystemic vascular anomalies was published in 2009.[1] Recent information on portosystemic shunts is presented.

Congenital portosystemic shunts (CPSS) are vascular anomalies that occur secondary to inappropriate closure of different portions of fetal vasculature, resulting in intrahepatic or extrahepatic CPSS. Typically, a single CPSS is present, although multiple CPSS have been reported.[2] The presence of a CPSS allows portal blood to bypass the liver and enter the systemic circulation. Operative intervention is often recommended with the goal being slow closure of the anomalous vessel to gradually accustom the liver to increased blood flow and prevent the development of portal hypertension. This goal is often accomplished through open surgical techniques, including ameroid constrictor or cellophane band placement. These surgical procedures should result in CPSS closure over approximately 2 to 5 weeks and good clinical results. More

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recently, minimally invasive methods of CPSS occlusion have been described.[3–8] Laparoscopic placement of cellophane bands has been described in dogs with extrahepatic CPSS, and endovascular occlusion has been described in dogs with either intrahepatic or extrahepatic CPSS.[3]

DIAGNOSTICS

Diagnosis and treatment of CPSS in dogs has evolved with technology. Although some aspects of the diagnostic workup and treatment of CPSS have remained similar for several years, other aspects have changed dramatically.

Clinicopathologic Findings

Laboratory testing is among the first steps recommended in the diagnostic workup of dogs suspected to have CPSS. A complete blood count (CBC), serum biochemistry profile, urinalysis, and preprandial and postprandial serum bile acids, and/or ammonia level are recommended. Recently, some veterinarians have begun testing protein C activity.

Protein C is a plasma anticoagulation protein. Along with antithrombin, protein S, and plasminogen, protein C is important for preventing thromboembolic disease. Protein C is a vitamin K–dependent serine protease enzyme that is synthesized by the liver. Once activated, protein C works to promote fibrinolysis, modulate inflammation, and inhibit apoptosis. In human patients, measurement of protein C levels has been used to assess liver function. Low protein C activity levels have been reported in people with a variety of liver diseases including inflammatory hepatopathy, cirrhosis, portal vein obstruction, and neoplastic infiltration.[9,10] Protein C activity levels may be used to assess hepatic function in a variety of liver diseases. In dogs, protein C may be useful in distinguishing CPSS from portal vein hypoplasia (also known as microvascular dysplasia).[11] Dogs with CPSS have significantly lower protein C activities than dogs with portal vein hypoplasia.[11] When protein C activity is considered with other laboratory findings, it may be useful to distinguish CPSS from portal vein hypoplasia. Further, dogs surgically treated for CPSS show postoperative improvement in protein C activity. Therefore, protein C activity levels may be a useful test, in addition to other blood tests, to aid in monitoring dogs after surgical treatment of CPSS.[11]

In humans, inflammation has been shown to be associated with hepatic encephalopathy (HE). For that reason, markers of inflammation, such as C-reactive protein, have been measured in dogs with CPSS.[12] A difference in C-reactive protein concentrations has been detected between dogs with CPSS exhibiting HE versus those not exhibiting HE and dogs without HE.[12]

Abnormalities in the CBC and serum biochemistry profile may be seen in dogs with CPSS. Changes in the CBC may include leukocytosis, microcytosis, and normocytic, normochromic, nonregenerative anemia. Leukocytosis may be present owing to increased antigenic stimulation from decreased hepatic endotoxin and bacterial clearance from the portal circulation. Anemia is seen commonly in dogs with CPSS and is associated with abnormalities in iron metabolism. The exact pathogenesis of CPSS-associated anemia has not been described. Recently, 1 study evaluated CPSS-associated anemia with abnormalities in hepcidin. Hepcidin is a hormone synthesized mainly by hepatocytes. It controls iron transport by binding and inhibiting ferroportin, an iron export protein. No evidence that dysregulated production of hepcidin was associated with anemia in dogs with CPSS was found.[13]

Changes seen on serum biochemistry profile are varied and may include decreased blood urea nitrogen, hypoalbuminemia, hypoglycemia, and hypocholesterolemia.