

Updates on Extrahepatic Portosystemic shunts
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Portosystemic shunts are vascular anomalies that permit the diversion of portal blood flow to the systemic circulation without perfusion of the hepatic parenchyma. Portosystemic shunt may be congenital or acquired secondary to portal hypertension. Congenital shunts are the result of developmental abnormalities in the cardinal and vitelline veins of the developing embryo that allow functional communications between these two systems. When these abnormal communications persist after birth, a portosystemic shunt is present.

The prevalence of canine congenital portosystemic shunt has been reported at 0.18%; the prevalence in mixed breed dogs is 0.05%. Breeds documented to be at increased risk of extrahepatic shunts include Havanese, Yorkshire terriers, Maltese, Dandie Dinmont Terriers, and Pugs. The relative risk for portosystemic shunt in Yorkshire terriers is 20 times greater than for all breeds combined. Medium and large-breed dogs, including Irish wolfhounds, golden retrievers and Australian cattle dogs are predisposed to intrahepatic shunting. Approximately 80% of all canine portosystemic shunt are congenital with greater than 60% being extrahepatic.

The clinical signs of a portosystemic shunt are often vague; the most common clinical signs include neurologic abnormalities (dogs) and ptyalism (cats). Gastrointestinal signs (vomiting and diarrhea) and signs of urinary tract dysfunction (polyuria, pollakiuria, and stranguria) are commonly seen. Greater than 95% of animals diagnosed with portosystemic shunt present with clinical signs attributed to the shunt. Asymptomatic patients are generally identified by abnormalities evident upon presurgical blood work at the time of elective neutering or delayed recovery from anesthesia. Hematological abnormalities may include microcytosis with normochromia, mild nonregenerative anemia, target cell formation, or poikilocytosis. Serum biochemical abnormalities supportive of a portosystemic shunt include abnormal liver function tests (decreased albumin, BUN, cholesterol, and glucose) and mild elevations in hepatocellular leakage enzymes. Ammonium biurate urolithiasis is frequently documented. Elevated paired bile acid measurement in a young dog with microhepatica and clinical signs consistent with a portosystemic shunt is highly sensitive.

A definitive, preoperative diagnosis of a portosystemic shunt may be made with contrast radiography, ultrasonography, scintigraphy, contrast enhanced computed tomography, or magnetic resonance angiography. Given their lack of invasiveness and relative availability, ultrasonography and scintigraphy are presently the most widely utilized diagnostic tests for portosystemic shunt. The reported sensitivity of abdominal ultrasound for the diagnosis of a portosystemic shunt is 80.5 to 95%. A previous study reported the overall accuracy of ultrasound for the diagnosis of a portosystemic shunt was 95% (d'Anjou et al., 2004). The same study reported a positive predictive value of 100% for dogs with concurrent microhepatica, renomegaly, and cystic calculi. The presence of turbulent flow within the caudal vena cava had a positive predictive value of 91% for portocaval shunting. The value of ultrasound for portosystemic shunt diagnosis is

extremely user dependent. Transcolonic portal scintigraphy (using technetium pertechnetate) is both a sensitive and specific test for portosystemic shunting. While uncommon, the calculated shunt fraction can be falsely lowered with poor colonic absorption (may occur with concurrent lactulose administration), inadequate colonic preparation, rectal deposition of technetium resulting in a false negative result. Recently, trans-splenic portal scintigraphy (TSPS) has been described in dogs. TSPS offers the advantage of decreased radiation exposure, improved image detail (via a higher count density), and offer the ability to differentiate between single congenital and multiple acquired shunts.

Following the documentation of an extrahepatic portosystemic shunt, surgical treatment options for shunt attenuation include suture ligation, ameroid constrictor (AC) placement, and cellophane banding (CB). Thirty two to 60% of dogs and cats undergoing portosystemic shunt attenuation can tolerate a complete occlusion. If complete occlusion results in an inappropriate rise in portal pressure (or alteration in CVP, MAP or HR) the patient is considered a candidate for AC placement or CB. AC and CB provide shunt attenuation by extraluminal compression, thrombus formation, and perivascular inflammation and fibrosis. Complete closure of the shunting vessel is anticipated in approximately 5 to 8 weeks. Recent studies report excellent clinical outcomes (defined as clinically normal patients not requiring medial management) in up to 80% of surgical cases. A 7.1% (Mehl et al., 2005) and 5.5% (Hunt et al., 2004) perioperative mortality was associated with the use of AC and CB respectively; these mortality rates are significantly lower than those previously published. With the use of AC, persistent shunt (as determined with transcolonic scintigraphy) is reported in up to 25% of postoperative cases. The use of AC in cats has also been reviewed (Kyles et al., 2002 and Havig et al., 2002). Despite a high perioperative morbidity (including central blindness and seizures), the perioperative mortality was low. Unfortunately, the long term outcome differed greatly in these retrospective papers. It should be noted that CB should be avoided in cats due a limited inflammatory response and resultant increased risk of incomplete shunt occlusion.

A primary prognostic factor for long term outcome is the degree of shunt attenuation. Numerous retrospective studies have demonstrated that patients with incomplete portosystemic shunt occlusion have a high risk of recurrence of clinical signs (up to 50%). Preoperative bile acids, preoperative neurologic status, shunt fraction, and age do not correlate with long term outcome. Recently, Mehl et al. identified the following predictive factors for persistent postoperative shunting: 1) low preoperative albumin concentration, 2) high portal pressure with temporary intraoperative occlusion, and 3) high portal pressure difference (difference between baseline pressure and the portal pressure following temporary intraoperative occlusion). Factors that were significantly associated with postoperative mortality included preoperative leukocytosis and the presence of postoperative complications (including seizures and ascites). The reported incidence of seizures following extrahepatic shunt attenuation is 0-18% in dogs and 0-25% in cats; the reported incidence of portal hypertension is 2-4%.

Currently, the Veterinary Specialty Hospital utilizes both ultrasound and transcolonic portal scintigraphy as diagnostic modalities for extrahepatic portosystemic shunt identification. Contrast enhanced spiral computed tomography and percutaneous splenoportography are used in select cases. Presently, both AC and CB are utilized at the Veterinary Specialty Hospital for shunt attenuation. Postoperative evaluation includes paired bile acids approximately 6-8 weeks following surgery. Significant elevations in post attenuation bile acids warrant scintigraphy to help determine if liver pathology, persistent shunt flow or acquired multiple extrahepatic shunts are present. A high shunt fraction six weeks postoperative may warrant diagnostics with minimally invasive diagnostic modalities or exploratory surgery with mesenteric portography.