



ANIMAL SURGICAL CLINIC
of SEATTLE

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PORTOSYSTEMIC SHUNTS • CLIENT INFORMATION

ANATOMY

Portosystemic shunt (PSS), as a broad term, is an abnormal communication between the portal (“dirty”) and systemic (“clean”) venous circulations. The portal vein drains blood from the gastrointestinal tract to the liver. This blood contains potential toxins or other substances that are eaten and need to be metabolized by the liver. The systemic venous circulation is the blood flow from organs other than the GI tract including kidneys and muscles. In the abdomen, the caudal vena cava (CVC) is the main vein that drains “clean” blood from the back half of the body. The CVC courses through the abdomen and bypasses the liver and empties into the heart. The portal vein drains “dirty” blood from the stomach and intestines and empties into the liver. A PSS occurs when there is an abnormal connection between the portal vein and the CVC (or occasionally another “clean” vein). When this happens, some of the blood does not get filtered by the liver and subsequent clinical signs develop.

PSS are classified based on their number, etiology, and location. Single, congenital, extra-hepatic (outside of the liver) PSS are the most commonly diagnosed PSS, and typically occur in small and toy breed dogs such as Yorkshire terriers and Maltese. Single, congenital, intra-hepatic (within the liver tissue) shunts more commonly occur in large breed dogs, such as Labradors and Irish Wolfhounds. Multiple, acquired extra-hepatic PSS can develop secondary to liver disease or following occlusion of a single PSS. Other vascular abnormalities such as arteriovenous fistulas may also lead to acquired PSS. Microvascular dysplasia (MVD) is a condition in which diffuse microscopic shunting occurs within the liver tissue.

MAKING THE DIAGNOSIS

Clinical suspicion is the first step in diagnosing PSS. The most common presentation is a young dog (or cat) that is underweight, has a poor hair coat, and signs of abnormal brain function called hepatic encephalopathy (HE) that may be more severe following a meal. These signs include: head pressing, circling, staring at the wall, blindness or bumping into things, and seizures. Additional clinical findings in cats include drooling and copper colored eyes. Other symptoms that are less specific include vomiting, decreased appetite, diarrhea, difficulty urinating or increased urination. In some cases, the animal may be asymptomatic and the clinical suspicion arises based on a small liver seen on x-rays, prolonged recovery from anesthesia, or abnormal blood work.

The diagnostic workup begins with basic blood work, including a CBC and chemistry. Serum bile acids and/or ammonia levels should also be performed. When running bile acids, it is essential to obtain 2 samples: one on an empty stomach and one two hours after a meal. Elevated post-prandial (after meal) bile acids have a sensitivity and specificity near 100% for diagnosing PSS. Anecdotally, most patients with a single PSS have post-prandial bile acids >100 umol/L. Clotting profiles are often performed because the liver is responsible for making the factors that help blood clot.

Diagnostic imaging typically begins with abdominal ultrasonography. The sensitivity and specificity of ultrasound for detecting and characterizing PSS is reported to be 90%, but can be highly dependent on the experience of the ultrasonographer. Ultrasound can also be used to diagnosis bladder stones, which often accompany PSS. Computed tomography (CT, “Cat Scan”) with dye can be very useful for diagnosing and characterizing the location of PSS and may be recommended in cases in which an obvious PSS is not found on ultrasound.

When pre-operative imaging fails to detect a PSS, abdominal exploration and liver biopsy is indicated. Microvascular dysplasia (MVD) can be diagnosed based on failure to detect a macroscopic PSS and histologic results from the liver biopsy.

PRE-OPERATIVE MEDICAL MANAGEMENT

Once a PSS is diagnosed, medical therapy should be instituted. This should include an antibiotic (metronidazole, neomycin or amoxicillin) to decrease colonic production of ammonia (a toxin that is normally filtered by the liver); lactulose, which decreases transit time of intestinal contents thereby decreasing ammonia absorption; a gastro-protectant (omeprazole), particularly for intra-hepatic shunts; and a low protein diet such as Hills k/d or i/d. Homemade diets and diets with plant rather than animal protein have also been recommended. If the animal has any history of hepatic encephalopathy and/or seizures, an anti-convulsants should be instituted for at least 2 weeks prior to surgery.

SURGICAL TREATMENT

Single extra and intra-hepatic PSS should be treated by gradual occlusion in order to prevent life-threatening portal hypertension. For single, extra-hepatic PSS, this is accomplished by placing an ameroid constrictor around the shunt. This is a small ring shaped device that is carefully placed around the shunt and gradually closes down over several weeks after surgery. The intent is that this implant will not ever need to be removed. Intra-hepatic PSS can be occluded using the same technique; however these shunts may require other surgical approaches including interventional radiography techniques.

Multiple extra-hepatic shunts and MVD cannot be treated with surgery. Animals with these conditions should be managed medically for the underlying liver dysfunction.

POTENTIAL RISKS AND COMPLICATIONS

Surgery is recommended for dogs with single PSS. However, it is important to note that there are certain risks that accompany this condition and surgery. Complications that can develop immediately after surgery or within the first few days include: portal hypertension, bleeding, hypoglycemia, post-surgical seizures, sepsis, and sudden death. Portal hypertension means that the shunt closes down too fast and blood backs up into the intestines because the portal vein cannot accommodate the immediate increase in blood flow. We don't know why some dogs and cats develop seizures after shunt surgery even if they didn't have seizures beforehand. If we start to see signs of seizures we need to start treating immediately and will often induce a coma for several days and then allow the dog to recover. For this reason, most dogs will need to stay in the hospital for a few nights after surgery for observation. In some dogs, continued shunting occurs even after surgery and may or may not be associated with recurrence of clinical signs. Continued shunting can be due to either incomplete occlusion of the shunt by the ameroid constrictor or development of multiple acquired shunts. An abdominal ultrasound or CT may be performed to determine the cause of continued shunting.

POST-OPERATIVE CARE

Animals will remain on all of the pre-surgical medication and low protein diet for the first few months and will be tapered over 2-3 months. Pain medication may also be prescribed for the first week after surgery.

Blood work, including bile acids, will be repeated about 2-3 months after surgery. We are looking for the bile acids to decrease, but in some dogs they will not return to normal. Blood work may be repeated every few months for the first year after surgery.

PROGNOSIS

80% to 90% of dogs will have a good to excellent outcome following surgery for a single PSS.