



Update on Portosystemic shunts

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Introduction: Portosystemic shunting (PSS) describes any abnormal vessel that bypasses the normal portal circulation to the liver. These vessels can be congenital or acquired. They may be intrahepatic, extrahepatic, single, multiple or microvascular.

Signalment: Any breeds may have PSS. Small breed dogs, esp. Yorkshire terriers, poodles, Shih Tzu, Peekinese, Schnauzers and Pugs are prone to congenital, single extrahepatic shunting. Large breed dogs such as Labradors, Wolfhounds, Golden Retrievers and Sheepdogs are more prone to congenital intrahepatic shunting. Cats may also have PSS. Himalayan and Persian breeds are predisposed. Most animals show clinical signs by 6 months to 2 years of age, but animals may also present later in life.

Clinical presentation: Animals with PSS present with variable clinical signs. Signs are most often associated with one of 4 different categories: hepatic encephalopathy, decreased hepatic metabolism, gastrointestinal or urinary tract signs.

Hepatic encephalopathy (HE): Despite intensive research, the neurochemical basis of the disorder has not been defined. Four current theories include 1. Ammonia acting as a neurotoxin 2. alteration of monoamine (serotonin and tryptophan) neurotransmission as a result of altered plasma amino acid metabolism 3. an imbalance between excitatory and inhibitory neurotransmission. 4. an increase in an endogenous benzodiazepine like substance.

Animals with HE may show many different neurological behaviors associated with diffuse cerebral signs. Seizures, altered mentation, blindness, behavior changes, circling, and head pressing are common.

Decreased hepatic metabolism: is seen most notably in animals that have prolonged recovery from anesthesia. Drugs that require hepatic metabolism will have an increased half-life.

Gastrointestinal: nausea, vomiting, polyphagia, pica and diarrhea are common. Cats often have ptyalism as the only presenting sign.

Urinary: Due to increased ammonia excretion, ammonium biurate crystals or uroliths are common. Some animals may show only urinary signs of straining, hematuria, pollakiuria, or PU/PD.

Diagnosis: Animals suspected of PSS should have a minimum data base consisting of a complete blood count (CBC), biochemical profile, urinalysis (UA) and bile acids (BA). A CBC may be normal or show anemia and microcytosis. Leukocytosis is often present. A biochemical profile will often show decreased hepatic function with low cholesterol, BUN, protein (albumin and globulin) and glucose. Liver enzymes are often normal, but

may be elevated. Marked elevations of AST, ALT, ALP or GGT are not common with PSS. Urinalysis will often show ammonium biurate crystals. Fasting and post prandial bile acids are usually elevated. Post prandial BA elevation is usually marked and can be as high as 4-500.

If a shunt is suspected based on the minimum database, the next step is portal scintigraphy. Technetium 99 scintigraphy via enema administration is a very sensitive and specific test for PSS. However a few false positives and negatives have been reported and the study does not give much anatomic information.

Abdominal ultrasound is useful in some cases to localize the shunting vessel. It has been more useful for localizing intrahepatic shunts. The importance of abdominal ultrasound is more for preoperative evaluation of the kidneys and bladder to determine if stones are present. Pure ammonium biurate uroliths are radiolucent and are often missed on survey radiographs.

Portography: this is the most definitive way to get anatomic information about the shunt. With experience it is rarely needed. I perform portograms on less than 10% of animals with portosystemic shunts. It can be helpful if a shunt cannot be found at surgery.

Treatment: Surgical attenuation of the shunting vessel is the treatment of choice for all single congenital shunts. Prior to anesthesia and surgery, medical management is useful. A low protein diet with most of the calories from carbohydrates and fat is ideal. Hill's K/D diet or Purina NF fulfill dietary requirements. Lactulose (0.25-0.5ml/kg BID-TID) is useful for acidifying the colon and trapping ammonia. Lactulose also shortens intestinal transit time, alters colonic flora and has an antiendotoxin effect. Antibiotics (amoxicillin, metronidazole and neomycin) are useful for decreasing urease producing bacteria in the colon. I generally use amoxicillin due to potential for neurologic side effects of metronidazole and potential nephrotoxic side effects of neomycin. Animals with severe hepatic encephalopathy may need stabilization with IV fluids, anticonvulsants, and enemas.

Surgical management of PSS has changed substantially in the last few years. The advent of the ameroid constrictor has enabled gradual occlusion of PSS. The constrictor is made up of a stainless steel ring filled with a ring of hydroscopic casein material that absorbs abdominal fluid causing occlusion. There is generally a rapid occlusion over the first 2 weeks with gradual occlusion for the following 6 weeks. The constrictors come in multiple different sizes. The rings are most useful for single extrahepatic shunts.

Intrahepatic shunts require a completely different technique than extrahepatic shunts. Intrahepatic shunts are often large and cause portal hypertension with complete occlusion. A new technique has been created that allows complete attenuation of the intrahepatic shunt. A venograft is harvested from the jugular vein and used to create an extrahepatic shunt between the portal vein and vena cava. The intrahepatic shunt is localized and completely attenuated. The venograft helps prevent portal hypertension. An ameroid constrictor (which usually can't be placed around intrahepatic shunts) is then placed around the venograft to allow gradual occlusion.

Prognosis: Prognosis for single extrahepatic shunts is excellent. 90% of dogs and cats will show improvement of clinical signs and can return to a normal lifestyle. Animals with intrahepatic shunts have a higher incidence of perioperative mortality.

Approximately 60-70% of dogs with intrahepatic shunts will improve. Complications include: post operative seizures, ascities, bleeding, anesthetic death, and creation of multiple extrahepatic shunts.