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VETERINARY SURGEONS

Portosystemic Shunts

Associated Terms:

Extrahepatic Portosystemic Shunt, Shunt, Intrahepatic Portosystemic Shunt, PSS, EHPSS, IHPSS, Congenital Portosystemic Shunt, Liver Shunt



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Your ACVS board-certified veterinary surgeon completed a three-year residency program, met specific training and caseload requirements, performed research and had research published. This process was supervised by ACVS Diplomates, ensuring consistency in training and adherence to high standards. After completing the residency program, the individual passed a rigorous examination. Only then did your veterinary surgeon earn the title of ACVS Diplomate.

Overview:

A portosystemic shunt (PSS) is an abnormal connection between the portal vein and systemic circulation. Blood from the abdominal organs which should be drained by the portal vein into the liver is instead shunted to the systemic circulation by the PSS. This means that a portion of the toxins and nutrients absorbed by the intestines bypasses the liver and is shunted directly into the systemic circulation. **There are two categories of shunts, extrahepatic (outside the liver) and intrahepatic (inside the liver).** While most portosystemic shunts are congenital (the dog or cat is born with the shunt), under certain circumstances portostystemic shunts may be acquired secondary to another problem with the liver.

Shunts allow portal blood to reach the systemic circulation without first passing through the liver. Normally blood exiting the intestines, spleen, and pancreas enters the portal vein, which then takes blood to the liver. The liver metabolizes and detoxifies this blood. **If a shunt is present the liver is deprived of factors that enhance liver development (hepatotrophic factors), which results in failure of the liver to reach normal size (hepatic atrophy).** A common result of hepatic atrophy is hepatic insufficiency, which then frequently results in hepatic encephalopathy (a clinical syndrome of altered central nervous system function due to failure of normal liver function).

The genetic basis of PSS in dogs is unknown, but it is considered congenital and breeds affected include:

- Miniature schnauzers
- Yorkshire terriers
- Irish wolfhounds
- Cairn terriers
- Maltese
- Australian cattle dogs
- Golden retrievers
- Old English sheepdogs
- Labrador retrievers

Single extrahepatic shunts are typically congenital and affect small and toy breeds whereas single intrahepatic shunts affect large breeds. Cats nearly always have extrahepatic shunts and the left gastric is the most common.

Acquired PSS are almost always multiple vessels, which develop in response to hepatic hypertension. They can occur in any breed or age of animal. They are a compensatory mechanism to prevent or delay liver failure. As such, they cannot be ligated without causing severe symptoms, and medical management is the only option for treatment.

Signs and Symptoms:

Animals with congenital portosystemic shunts may present for:

- small body stature
- anesthetic intolerance
- behavioral abnormalities

The signs are often episodic and may be more noticeable after eating. Signs of abnormal neurologic function include:

- ataxia (swaying as if intoxicated)
- seizures
- blindness
- head pressing

These signs are referred to as “hepatic encephalopathy.” Other signs may include:

- anorexia (loss of appetite)
- vomiting
- diarrhea
- constipation
- ptyalism (hypersalivation) – most frequently seen in cats
- polyuria/polydipsia (excessive urination/drinking)
- stranguria (difficulty urinating)
- hematuria (blood in the urine)

Diagnostics:

If your primary care veterinarian suspects that your pet has a portosystemic shunt, a full diagnostic work-up is advised. A full work-up may include:

- blood work
- urinalysis
- liver function tests (bile acids and ammonia)
- radiographs
- ultrasound (Figure 1)
- nuclear scintigraphy (a non-invasive technique involving colonic administration of a radioisotope)
- portography (an x-ray dye study that specifically highlights the portal system, (Figures 2 and 3))
- CT scan

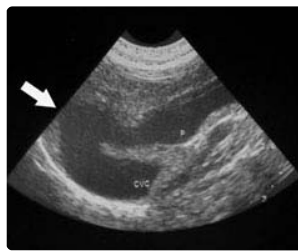


Figure 1. Ultrasound of abdomen. Arrow pointing to intrahepatic shunt

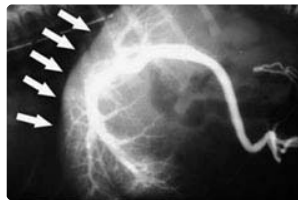


Figure 2. Normal portogram with arrows pointing to normal vasculature in liver



Figure 3. Abnormal portogram with large arrow pointing to shunt and small arrows pointing to lack of vasculature in liver

Treatment:

Medical management

Before surgery can be performed your pet may need to be stabilized medically. The goal of medical management is to improve your pet's health to a point where the risk of anesthesia and surgery is low. Medical management consists of a low protein diet and oral administration of antibiotics and lactulose. The goals are to decrease the bacterial population in the intestines and to minimize the production of toxins. Lactulose is a cathartic, which promotes the expulsion of fecal matter, as well as decreasing the bacterial load in the colon. Antibiotics help to eliminate bacteria that promote the formation of toxins. The diet should provide high-quality protein but may need to be moderately restricted in amount of protein, depending on the clinical signs for each individual animal. If seizures are a part of clinical signs, anti-seizure medication may also be used.

Surgical management

The treatment of choice for a single PSS is surgical attenuation (narrowing) or full ligation (tying off) of the abnormal shunt vessel. This full ligation may be done instantaneously using suture material or intra-venous injection of an embolus of special glue material, or delayed full ligation with an ameroid constrictor, cellophane band or an intra-venous embolic coil. This surgery is technically challenging, and your primary care veterinarian may refer you and your pet to an ACVS board-certified veterinary surgeon.

If a shunt cannot be identified at surgery, an intra-operative portogram is performed (Figures 2 and 3). When the shunt is identified, pressure in the portal vein may be measured to determine if complete ligation is possible. Excessively high portal system pressure, called portal hypertension, can result in death. Acute portal hypertension results in abdominal distension, pain, bloody diarrhea, ileus (stasis of bowel with gas build-up) and endotoxic shock (shock due to bacterial toxins).

Partial ligation is performed if there is a risk of portal hypertension (occlusion pressure is too high). (Figure 4)

Partial ligation of the shunt may be done by partially enclosing the vessel with a suture ligature until pressure rise is at its acceptable limit. About half of patients using this method will go on to scar close their shunts; but about half will maintain some shunting of blood and need a second surgery months later, when the liver has adapted to its new circulation and can withstand full ligation. This method is rarely used anymore, due to the availability of ameroid constrictors, intravenous coils and cellophane bands.

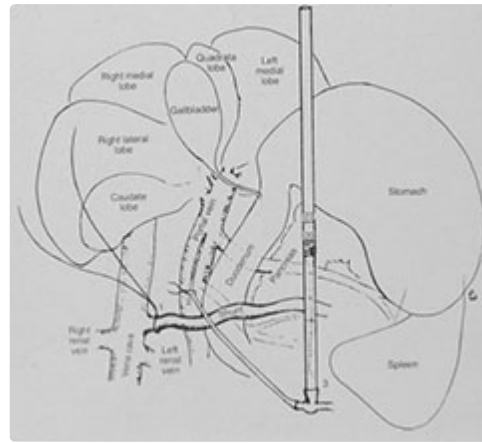


Figure 4. Manometer for measuring portal pressure



Figure 5. Ameroid constrictor bands



Figure 6. Ameroid constrictor band placed on a shunt vessel during surgery

The ameroid constrictor (Figure 5) is made of casein in a stainless steel, “C”-shaped ring. It is placed around the shunt, and the ring is closed with a small key.

Over the next few weeks, the casein swells and gradually occludes the shunt (Figure 6). This is considered a method of gradual occlusion.

The vessel may also be occluded using a special cellophane band (Figure 7). The band will incite an inflammatory response, and the vessel will slowly close down over a period of months.

Trans-venous coiling is usually used for larger, intra-hepatic shunting vessels.

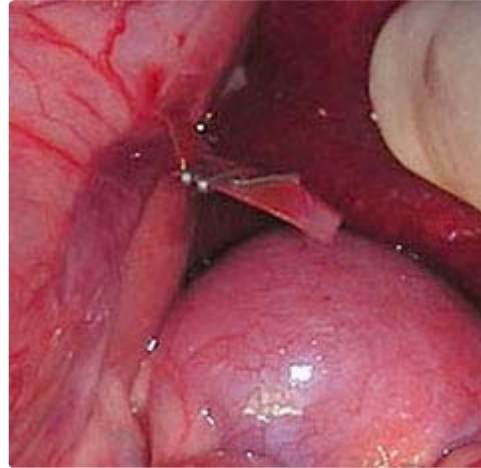


Figure 7. Cellophane band placed around a shunt vessel during surgery

Aftercare and Outcome:

Routine postoperative management includes intravenous fluids and pain medications. Lactulose and diet modification are continued, as it takes time for the liver cells to regenerate and adjust to the new circulation. These medications may be tapered depending on follow-up bile acid test results. Because serum bile acids values may or may not improve, some dogs may need long term treatment whereas others may only need some dietary restrictions or no medical restrictions. After ligation, the liver should regenerate. Failure of the procedure can occur for any of the following reasons:

- failure of shunt to close
- recanalization of the shunt (the shunt reopens)
- the presence of a second, unrecognized shunt (extremely unlikely)
- the development of multiple acquired PSS secondary to portal hypertension or fibrosis (scarring) of the liver

Complications after surgery include portal hypertension, which can lead to loss of proper blood circulation to abdominal organs and death. Animals may show signs of:

- ascites (fluid distension in the abdomen)
- vomiting
- diarrhea
- depression
- respiratory distress

Use of gradual occlusion devices has substantially reduced the chance of death by portal hypertension.

One of the most problematic complications is development of seizures that are refractory to treatment. This occurs most frequently in toy breed dogs, in the first 1-2 days after surgery. The cause of these seizures is unknown. Seizures may be managed with anti-seizure medication. In severe cases, intravenous administration of anti-seizure or anesthetic agents may be required to control seizures. Development of seizures that are poorly controlled by medication is a very poor prognosis.

The prognosis is excellent if the animal survives the immediate postoperative period and full ligation of the shunt is achieved. With partial ligation, the prognosis is not as good. In many cases, full ligation is possible in animals that were partially ligated 4-6 months previously, so follow-up bile acids tests and portal scintigraphy should be done to monitor for shunt function.

This Animal Health Topic was written by and reviewed by Diplomates of the American College of Veterinary Surgeons. Any opinions stated in this article are not necessarily the official position of the American College of Veterinary Surgeons.

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