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Portosystemic Vascular Shunts

Portosystemic vascular shunts are anomalous (abnormal) blood vessels that shunt blood away from the liver. This diversion of blood leads to liver failure and the collection of toxins in the blood which frequently causes behavioral changes, seizures, or coma. Portosystemic shunts are divided into congenital (from birth) or acquired shunts. Congenital shunts originate from the portal vein draining the intestines and empty into the caudal vena cava (draining directly into the heart). This effectively allows all of the blood draining from the intestines and containing products of digestion to bypass the liver. Without the liver to process the blood from the intestines, some toxins (i.e. ammonia) are sent directly to the heart for circulation within the body.

Most congenital shunts are extrahepatic (outside the liver) and singular (see Figure 2). These are easily found and are surgically correctable. If multiple extrahepatic shunts or an intrahepatic shunt (within the liver) are present, surgical correction may not be possible (see Figure 3). Congenital, single, extrahepatic shunts are generally found in small breeds of dogs and most breeds of cats. Acquired shunts are usually secondary to severe liver disease in older animals but are occasionally present in young animals secondary to hepatic microvascular dysplasia.

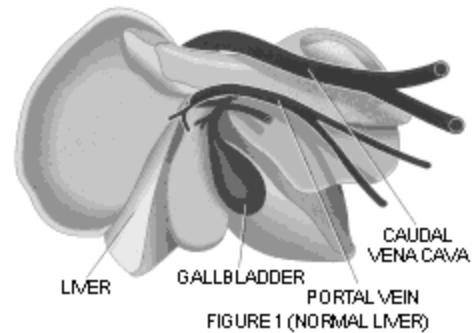


FIGURE 1 (NORMAL LIVER)



FIGURE 2 (SINGULAR SHUNT)



FIGURE 3 (MULTIPLE SHUNTS)

Clinical Signs

There are a variety of clinical signs associated with portosystemic shunts. These signs are related to the increased level of toxins circulating throughout the body. Most affected animals are less than one year of age but animals may exhibit signs of the disease at any time. These patients may be thin, lethargic, stunted, or in generally poor condition.

Neurologic disorders are noted in the majority of cases and can involve seizures, personality changes, wall walking or head pressing, disorientation, visual deficits, stupor, or coma.

Symptoms of portosystemic shunts are exacerbated by high protein diets and may worsen after eating even an average meal. Gastrointestinal signs can include anorexia (food avoidance) and/or vomiting. Because the liver cannot adequately metabolize the products of digestion, certain types of cystic calculi (bladder stones) are frequently a concurrent finding.

Diagnosis

A tentative diagnosis of a portosystemic shunt is made based on the history, clinical presentation, and laboratory results. Any or all of the following laboratory abnormalities may be seen:

- Elevated white blood cell count
- Mild to moderate anemia
- Elevated liver enzymes
- Low blood urea nitrogen (BUN)
- Low cholesterol
- Elevated bile acids
- Elevated blood ammonia

While there is no one laboratory test that conclusively diagnoses this problem, the bile acid and blood ammonia levels are excellent indicators.

Abdominal radiographs (x-rays) often reveal a small liver. However, the definitive diagnosis is made with the use of a transcolonic nuclear scan. This involves placing a minute amount of a radioactive material into the colon and tracing the absorption of that material. The normal route of absorption is from the colon to the liver and then to the systemic circulation. In animals with shunts, the flow is from the colon directly to the systemic circulation. Vascular contrast studies and ultrasound have also been used to diagnose portosystemic shunts but are not as definitive.

Treatment

The management of portosystemic shunts consists of both medical and surgical modalities. Medical treatment of shunts will often decrease the severity of the clinical signs but should in most cases be considered as an adjunct to surgical correction of the shunt.

Medical therapy consists of:

- A low protein diet
- Antibiotics to decrease gastrointestinal bacteria that metabolize protein into ammonia
- Lactulose (an ammonia binder)

In a neurological emergency, careful fluid and electrolyte therapy combined with intravenous or oral antibiotics will usually result in rapid stabilization. Medicated enemas to lower nitrogen (ammonia) absorption may also be indicated along with careful intravenous dextrose administration.

Surgical Intervention

The goal of surgical intervention for both intrahepatic and extrahepatic shunts is to identify and completely close (ligate) the shunting vessel(s). This procedure restores the normal flow of portal blood to the liver. Sometimes the liver is unable to handle the increased portal flow. In these cases, the shunting vessel is attenuated (partially ligated) to divert as much blood into the portal circulation as possible. The degree of attenuation is determined by carefully measuring the portal venous pressure during surgery. This allows as much blood to flow to the liver as it can tolerate without causing permanent damage.

Surgical intervention for patients with multiple extrahepatic shunts is different. In these cases, the blood is shunting around the liver through many blood vessels due to portal hypertension (high portal blood pressure) secondary to liver disease. In order to force blood back into the portal circulation, the systemic venous pressure must be raised. This is accomplished by attenuating (partially closing) the caudal vena cava until the caval pressure exceeds the portal pressure. This procedure must also be used occasionally with intrahepatic shunts if they cannot be identified within the liver tissue.

Postoperative Care

Animals are monitored extremely carefully during the postoperative period. Generally, the first 48 hours is the most critical time. On occasion the liver is unable to handle the increased portal blood flow. If signs of this occur, the ligature must be removed immediately. Animals with attenuated shunts may develop

blood clots that occlude the shunt. If this occurs, the ligature must be released. Older animals may develop seizures that need to be controlled during this period. These seizure episodes generally last for 48-72 hours and are controlled with intravenous anticonvulsant medication. Generally, older patients are placed on prophylactic oral anticonvulsant therapy for at least 48 hours prior to surgery. This protocol has dramatically decreased the risk of post-operative seizures.

Once these animals are sufficiently recovered to go home from the hospital, they should be on a low protein diet, antibiotics, and lactulose. Over the next several weeks as the liver function improves, the lactulose and antibiotics will be discontinued. A normal diet may be slowly introduced as well. In most cases, the prognosis for a normal, active life is excellent.

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