Portosystemic shunts in dogs and cats

Portosystemic shunts ("liver shunts", PSS) are an important and relatively frequent disease of dogs and cats. Anatomically, a PSS is one or more venous connections between the portal vein and the systemic venous system, typically the caudal vena cava or azygous vein. Most PSS are macroscopic venous structures but in some breeds abnormalities of the microcirculation within the liver results in functional shunting without macroscopic vascular abnormalities (microvascular dysplasia, MVD).

Clinical signs are often somewhat non-specific (unthriftiness, vomiting, anorexia) and also typically include either neurological abnormalities (hepatic encephalopathy) or urinary obstruction (urate urolithiasis).

Shunts can be classified as congenital or acquired. Acquired shunts are a feature of chronic liver disease and secondary hypertension, and are of little surgical interest. Affected animals are treated for the effects of hepatic encephalopathy and for the underlying liver disease if possible.

Animals with congenital shunts can be managed with medical treatments and dietary manipulation to reduce the signs of hepatic encephalopathy and in the majority there is a good or excellent short to medium term improvement in clinical signs. However, if surgical occlusion of the shunt is possible, the long term outcome is improved compared to those managed medically alone. For most animals, it is therefore logical to recommend surgical intervention following a period of medical stabilisation. PSS are seen in dogs and cats. In dogs, breed dispositions to both congenital macroscopic PSS and MVD are reported.

Key words: Dogs, cats, surgery, liver, portosystemic

Introduction

Portosystemic shunts ("liver shunts", PSS) are an important and relatively frequent disease of dogs and cats. The first cases were reported in the 1970s and they have been diagnosed with increasing frequency since the 1980s. Anatomically, a PSS is one or more venous connections between the portal vein and the systemic venous system, typically the caudal vena cava or azygous vein. Pathological effects result not from the haemodynamic consequences of this, but because this arrangement allows substances absorbed from the intestine to enter the systemic circulation without passing through the “metabolic filter” of the microcirculation within the liver. Most PSS are macroscopic venous structures, but in some breeds abnormalities of the microcirculation within the liver results in functional shunting without macroscopic vascular abnormalities (microvascular dysplasia, MVD).

In animals with portosystemic shunting, various chemicals that would usually be eliminated from the portal circulation on first pass through the liver enter the systemic circulation and cause intoxication. An example of this is ammonia: this is a product of bacterial metabolism within the large intestine that is absorbed into the portal circulation. In animals with normal liver function and circulation, very little ammonia is detectable in the systemic blood but in animals with PSS (or severe parenchymal disease) it is present in detectable amounts and causes neurological signs (hepatic encephalopathy, HE) or the formation of urate uroliths.

Shunts can be classified as congenital or acquired. Acquired shunts are a feature of chronic liver disease and secondary hypertension and are of little surgical interest. Affected animals are treated for the effects of hepatic encephalopathy and for the underlying liver disease if possible. These are not the focus of this article.

Animals with congenital shunts can be managed with medical treatments and dietary manipulation to reduce the signs of hepatic encephalopathy. In the majority there is a good or excellent short to medium-term improvement in clinical signs. However, if surgical occlusion of the shunt is possible, the long term outcome is improved compared to those managed medically alone. For most animals it is therefore logical to recommend surgical intervention following a period of medical stabilisation.
PSS are seen in dogs and cats. In dogs, breed dispositions to both congenital macroscopic PSS and to MVD have been reported.

**Anatomy and breed predisposition.**

**Portosystemic shunts** are congenital or acquired.

- **Acquired (or secondary) shunts** consist of multiple small venous anastomoses that develop within the abdomen. In people, oesophageal varices are common but these are not a feature in dogs and cats. Much more prominent in these species are vessels that develop in the base of the mesentery in the region of the right kidney, between the portal vein and caudal vena cava. These are visible during surgical exploration and can be demonstrated radiographically. Acquired shunts occur in both species and in any breed.

- **Congenital (primary) shunts** are almost invariably single large vessels (of comparable size to the portal vein itself). The majority are extrahepatic (outside of the liver parenchyma) but some are intrahepatic (completely or partly within the parenchyma).

- **Intrahepatic shunts** connect intrahepatic branches of the portal vein to lobar hepatic veins. In some cases they represent persistence of the foetal ductus venosus (“patent ductus venosus”, PDV) and this is described in the Irish Wolfhound and other giant breeds. However most intrahepatic shunts are not PDV but are other anomalous vessels. Intrahepatic shunts are most common in large breed dogs, including the Irish Wolfhound, Labrador Retriever and Border Collie. They occur occasionally in cats.

- **Extrahepatic shunts** connect the portal vein to either the abdominal caudal vena cava or (less commonly) the azygous vein. Portocaval shunts can occur anywhere in the abdomen but the majority are in the cranial abdomen and arise from the gastroduodenal or splenic tributaries of the portal vein. Typically they are within the mesoduodenum or the lesser omentum. Similarly, portoazygous shunts also pass within the mesoduodenum.

Extrahepatic shunts are approximately 10x more common than intrahepatic shunts. They are most common in small breed dogs, particularly terriers. Commonly identified breeds include Cairn, Border, Jack Russell and Yorkshire Terriers, Bichon Frise and Maltese Terriers. Similarly, in cats, extrahepatic shunts are commoner than intrahepatic shunts.

**Microvascular dysplasia** is recognised in the same breeds as the other shunts but is particularly seen in small breeds. It is less common than either extra- or intrahepatic shunts.

**Pathophysiology**

Blood from the portal vein in normal animals is processed through the liver, but in animals with shunts, portal blood bypasses the metabolic effects of the liver parenchyma and enters the systemic circulation directly (Fig 1). This can have multiple metabolic effects. For example, byproducts of bacterial metabolism within the large intestine enter the systemic circulation in much higher concentrations in animals with PSS. These chemicals include ammonia, mercaptans, short chain fatty acids and aromatic amino acids. These chemicals can act directly as false neurotransmitters or have secondary effects resulting in clinical signs of neurological dysfunction (including coma, seizures, central blindness). Other less specific signs include lethargy, failure to thrive, vomiting and diarrhoea. Affected animals often have urate crystals in the urine and occasionally gross urinary stones (urate) develop.

**Diagnosis**

Given the often vague and non-specific clinical signs, focused diagnostic tests are required to further the diagnosis. Of these, the most useful screening test is the bile acid stimulation test (BAST). Following an overnight fast, serum is drawn before and 2 hours after feeding a fatty meal. Most affected animals have post prandial bile acids of over 100, and often over 200-300 µmol/L, although lower bile acid results do not rule out a PSS. Measuring blood ammonia is impractical and prone to error (dry chemistry in-house testing is inaccurate). Routine haematology and biochemistry tests can also reveal a number of non-specific indicators, such as hypoglycaemia. Note that jaundice is not a finding with congenital PSS.

A strongly positive result to the BAST indicates that a PSS is an important differential diagnosis but does not allow the clinician to distinguish between severe liver disease, primary or secondary PSS or MVD. Follow up tests include ultrasonography, liver biopsy, scintigraphy,