Portosystemic shunts in cats and dogs: signs and diagnosis

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THE liver has many essential functions within the body, including nutrient metabolism, removal of toxins and ammonia, protein synthesis (including coagulation factors), bile acid synthesis, vitamin storage, immunoregulation and bilirubin excretion.

To achieve this, blood flow to the liver is vast, averaging 30ml/min/kg to 45ml/min/kg (Witte, 1976). The majority of this (80 per cent of the blood flow and 50 per cent of the oxygen requirements) is supplied by the portal vein and the remainder by the hepatic artery.

Portosystemic shunts (PSS) are vascular anomalies that connect the portal circulation to the systemic circulation, bypassing some or all of the hepatic tissue.

They can form as a result of a congenital abnormality, either as persistence after birth of the ductus venosus, or because of anomalous, functional communications between the foetal hepatic vessels (vitelline system) and non-hepatic abdominal vessels (cardinal system, which later develops into the caudal vena cava and azygos vein).The former abnormality leads to an intrahepatic shunt and the latter to an extrahepatic shunt.

Alternatively, shunts may be acquired, usually secondary to portal hypertension. This article will focus on congenital PSS (Table 1).

Diagnosis

Presenting complaints include:

• Poor growth/weight gain.

• Drug (such as anaesthetic) intolerance due to reduced liver metabolism.

• In cats, copper-coloured irises (Figure 1) are reported. Controversy exists, however, as to whether this is truly a disease association.

• Neurological abnormalities, including behavioural changes, lethargy, ataxia, circling, head pressing, blindness or seizures. These abnormalities may have gone unnoticed by owners, either because the changes are very subtle, or because they believe this behaviour to be normal for their

new puppy/ kitten. Hepatic encephalopathy is seen not uncommonly in animals with PSS, and the cause is likely to be multifactorial.

Theories include increases in blood ammonia (with or without other synergistic toxins), alterations in monoamine or amino acid neurotransmitters, or increased cerebral concentration of an endogenous benzodiazepine-like substance. Experimentally, none of these factors alone consistently initiates encephalopathic coma.

• Increased incidence of urinary tract infection due to decreased urea production and increased ammonia excretion (which may also cause the development of urinary calculi). Polyuria and polydipsia (PUPD) may also be evident.

• Gastrointestinal signs, including vomiting.

• Hypersalivation, particularly in cats (Figure 2).

Cryptorchidism is recognised in some dogs with PSS.

Occasionally, other anomalies are found, such as heart murmurs. It is important to evaluate whether this is a flow murmur secondary to the shunt or represents a cardiac anomaly.

On routine serum biochemistry and haematology, changes are often subtle and may be difficult to interpret in a young, growing animal.

Abnormalities may include microcytosis, with or without anaemia (Figure 3), and variable leukocytosis. Microcytosis may be underestimated on postal samples due to red cells swelling in transit. Low cholesterol, low glucose and low blood urea nitrogen (BUN) may be seen and, in dogs, low albumin and low total protein may also be evident.

Elevated liver enzymes are uncommon because the hepatocytes themselves are usually either undamaged or are affected gradually, as the process is an atrophic one. However, mild-to-moderate increases in liver enzymes are seen in some patients without additional hepatic pathology.

Routine urinalysis may demonstrate features consistent with cystitis (such as haematuria, pyuria or proteinuria) and PUPD (low specific gravity of urine). Ammonium biurate crystals may be detected (Figure 4), but the absence of the distinctive “thorn apple” crystals may be a false negative and so multiple samples may be required before they are detected.

Additional tests are usually required to assess liver function in cases with PSS. Plasma ammonia can be assessed, but the sample should be transported on ice and analysed without delay since in-house analysers can be unreliable.

Elevated ammonia typically demonstrates abnormal hepatic function, but a normal level does not rule out PSS and is seen in seven per cent to 21 per cent of dogs with PSS, particularly after fasting or with effective medical treatment (Center, 1990). Postprandial ammonia can be evaluated, but measurement of bile acids is more commonly performed.

Bile acids measurement requires no special sample handling, and their elevation in animals with PSS is the result of bile acids shunting away from the liver to the systemic circulation, avoiding re-uptake by the hepatocytes. Usually, bile acids are measured preprandially and then two hours after a fatty meal. In animals with PSS or poor liver function, postprandial bile acids will be markedly elevated compared to normal animals. Occasionally, preprandial bile acid levels are significantly elevated and, therefore, postprandial testing is not required.

Finally, because the liver is responsible for the synthesis of many clotting factors, the coagulation profile should be assessed where surgery is considered.

Often, this is normal. However, given the limitations of conventional coagulation testing, it is still possible bleeding will be seen.

Imaging

Definitive diagnosis of a PSS requires visualisation, either directly at surgery or indirectly using a variety of diagnostic imaging modalities.

At the authors' hospital, ultrasonographic examination is the modality of choice, and changes include microhepatica, decreased number of hepatic and portal veins in the parenchyma, detection of the anomalous vessel and abnormal flow using Doppler (Figure 5 to 7).

Mesenteric portovenography can also be useful and involves catheterisation of a jejunal vein and injection of a water-soluble, radio-opaque, contrast agent observed under fluoroscopy (Figures 8 and 9). The disadvantage of this is the requirement for a laparotomy and the risk of thrombus formation in the catheterised vein. The authors reserve this technique for cases in which intraoperative location of the shunting vessel is challenging. Other techniques described, but less commonly used, include scintigraphy, computed tomography or magnetic resonance angiography.

Non-specific changes can also be seen, and include microhepatica, enlarged kidneys and urinary calculi. Although urate stones are radiolucent, secondary infection may lead to struvite deposition, which is radiopaque.

Medical management

The aims of medical management are to correct fluid, electrolyte and glucose imbalances, and to prevent hepatic encephalopathy.

Occasionally, animals will present in hepatic encephalopathic crisis. Seizures should be treated initially with intravenous (0.5mg/kg) or rectal diazepam (1.0mg/kg to 2mg/kg), followed by a loading dose of a longer acting drug. The authors have tended to use phenobarbitone in dogs, 12mg/kg to 18mg/kg IV followed by standard oral doses of 2mg to 3mg/kg/day, and levetiracetam in cats, 20mg/kg q8h. However, given the potential for hepatotoxicity, one could consider the use of levetiracetam in both species if justified for use under the cascade.

Lactulose enemas (preferably as a 10 to 15-minute retention enema; three parts lactulose [20ml/kg in dogs] to seven parts water) rapidly decrease colonic bacteria, preventing further absorption of toxic substrates.

Intravenous antibiotics, such as clavulanic acid potentiated amoxicillin 20mg/kg IV q8h, will decrease the number of ammonia-producing bacteria in the gastrointestinal tract, reducing absorption.

Some clinicians use metronidazole, but the hepatic metabolism of this drug should be considered and neurological side effects may be mistaken for encephalopathy. For these reasons, when metronidazole is desired, a lower dose should be used – for example, less than 7.5mg/kg. Electrolytes and glucose should be assessed and corrected using intravenous fluids spiked with potassium or dextrose as required, and changes in serum concentrations monitored hourly.

Importantly, no oral food should be provided while animals are severely encephalopathic to minimise substrate absorption, which could worsen clinical signs. Thiamine has also been recommended.

Management

Once animals are stabilised, the mainstay medical management comprises:

• **Diet.** The main aim is to provide moderate protein restriction to reduce ammonia production. Many commercially available liver diets also pro vide L-carnitine, antioxidants, restricted copper and increased zinc. Normal fat metabolism should be maintained and the high energy available from fat avoids excessive protein catabolism. Occasionally, animals will be reluctant to eat these preparations, and a diet of rice or potato with high quality, highly digestible protein (such as cottage cheese, soya or white chicken meat) may suffice. Some controversy exists about protein restriction in young patients, given their need for protein for growth. The authors recommend that patients are provided with a diet with sufficient protein for growth (mon– itor serum albumin and increase the protein content of the diet if this is decreasing), but not enough to cause encephalopathic signs. A good-quality protein source can be added to a liver diet, or a higher protein diet can be selected if necessary.

• **Lactulose.** This works by decreasing colonic pH, increasing entrapment of ammonium,

increasing faecal nitrogen excretion, inhibiting protein and amino acid metabolism, and decreasing intestinal transit time. The dosage is empirical, but should be titrated until two or three soft, but formed, stools per day are passed.

• **Antibiotics.** Once animals are no longer encephalopathic, oral antibiotics may be provided and the authors prefer ampicillin or amoxicillin. Occasionally, lower dose metronidazole may be used.

• **Regular, routine parasite control.**

• **Gastroprotectants.** Gastrointestinal haemorrhage may be seen in patients with PSS. This is the equivalent of a high-protein meal, making encephalopathy more likely. Gastroprotectants are not used routinely and are reserved for patients with documented gastrointestinal bleeding, or a history and investigation that are highly suggestive.

Medical management alone may be possible and up to one third of dogs will do well. Ultimately, once the animal is stabi– lised medically, the authors prefer to pursue surgical correction where possible, and anecdotally believe that the low postoperative incidence of seizures seen at their hospital is due to good preoperative medical management.

Without surgery, animals may live for up to seven years and survival has been correlated to age at presentation and serum BUN concentration.

Ultimately, more than half of dogs managed medically will be euthanised within 10 months because of neurological deterioration or progressive hepatic fibrosis (Watson, 1998). However, long-term survival studies for animals with surgically corrected PSS are not available.

Summary

PSS are congenital or acquired vascular abnormalities that connect the portal and systemic circulation. Haematological and serum biochemical changes may be suggestive of PSS, but definitive diagnosis is achieved via imaging or surgical evaluation. Medical management should be initiated in the first instance, the mainstay of which is an appropriate diet, lactulose and antibiotics.

Surgical management and postoperative care will be discussed in a later article.

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