valereferrals

Welcome... to our Summer 2021 Newsletter

Things at Vale Referrals have certainly been challenging, but we are pushing forward and already seen our build project reach key milestones. Our new dog and cat wards have opened and with our new reception due to be completed very soon. Whilst we still have a few months of build work left. Our new theatres, medicine and cardiology treatment rooms, as well as a larger prep room being created within the existing building.

This is an exciting time for the future of Vale and we're enjoying watching all the work unfold before us.

You can keep updated and see photos of our progress through our Facebook page and website.

Once the work is completed we will be hosting open day events, CPD and welcoming our referring vets to call in and see our new facilities.

Our builders have been fantastic, working so thoughtfully around us, ensuring that our clinical and patient care has not been compromised.

Our clients and patients have been at the forefront of this development and we have continued to provide a friendly and professional service.

We look forward to welcoming clients back into the practice soon.

We are delighted to welcome back to Vale Referrals, Alasdair Hotston Moore MA VetMB CertSAC CertVR CertSAS CertMedEd FRCVS Referral Surgeon Small Animal Soft Tissue Surgery.

Alasdair was a part of our team back in 2017, in 2018 he took up an exciting new position as Chief Medical Officer with Concordia Petcare, a new speciality veterinary chain in China and Hong Kong.

At the end of 2020, Alasdair returned to the UK and has rejoined our soft tissue surgery team at Vale Referrals, and as a consultant to the IVC-Evidensia Academy. Welcome back Alasdair!

To discuss a case or refer to Alasdair, call our team on 01453 547934 or email us referrals@valevets.co.uk

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We have also gained a brilliant addition to our Internal Medicine team; Vaclav Ceplecha MVDr PhD MRCVS

Internal Medicine Clinician Oncology Assistant Vaclav, has a keen interest in all aspects of small animal internal medicine, but particularly enjoys gastroenterology and hepatology cases. He undertook several small animal internal medicine externships (Purdue University, University of Melbourne), presented research at international conferences (ACVIM forum, WSAVA Congress), and authored and co-authored articles in international peer-reviewed journals. Vaclav is also a reviewer for the Journal of Veterinary Internal Medicine.

To discuss a case or refer to Vaclav, call our team on 01453 547934 or email us referrals@valevets.co.uk

Portosystemic shunts in dogs and cats

Alasdair Hotston Moore MA VetMB CertSAC CertVR CertSAS FRCVS

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).

PSS typically 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.

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, therefore, it is logical to recommend surgical intervention following a period of medical stabilisation.

PSS are seen in dogs and cats.

Anatomy and breed predisposition

Congenital (primary) shunts are almost invariably single large vessels (of comparable size to the portal vein itself). The majority are extrahepatic in position (outside of the liver parenchyma) but a minority (around 10%) 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 fetal 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 another anomalous vessel. Intrahepatic shunts are most common in large breed dogs. 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 commonest in small breed dogs, particularly terriers. Commonly identified breeds include Cairn, border, Jack Russell and Yorkshire terriers, Bichon Frise and Maltese terriers. They are also much commoner in cats than intrahepatic shunts.

Although primary shunts are a congenital disease, clinical signs most often become apparent around 4-6 months of age, and sometimes not until adult life. Occasionally signs are temporally related to feeding, although in most cases they are simply variable in extent. The onset of signs is sometimes precipitated by an external event, such as acute gastro-enteritis, scavenging or anaesthesia.

Diagnosis

Given the often vague and non-specific clinical signs, focussed diagnostic tests are required to further the diagnosis. Of these, the most useful screening test for the disease 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 200-300, micromole/litre.

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, portovenography (especially with CT) and exploratory surgery.

Although experienced ultrasonographers can find many shunts, false negatives and positives do occur. The principle is to follow the portal vein cranially within the abdomen and identify anamolous large branches, or to identify hepatofugal blood flow within the cranial part of the portal vein or turbulent flow within the abdominal caudal vena cava. CT angiography is very useful when available and has the advantage that images can be referred for interpretation.

Liver biopsy can be useful to investigate other (primary) liver diseases causing similar clinical signs and biochemical abnormalities but should not be used as a screening test in animals with a significant suspicion of PSS. In particular, it should be noted that histopathology will not distinguish PSS from MVD.

Given the limitations of non-invasive diagnosis, surgical exploration is recommended as the confirmatory test (and offers the possibility of surgical treatment) in animals where a high index of suspicion of an EH PSS exists (particularly when CT is not possible). As part of surgery, the surgeon often carries out portovenography: an X-ray study to demonstrate the shunt and localise it within the abdomen. Although portovenography is not always necessary for experienced surgeons, it does offer a "gold standard" in diagnosis and classification. However, since it is sensible to offer portovenography and surgical management at a single laparotomy, in most cases a 4-6 week period of medical management before surgery is recommended. The vast majority of animals respond well clinically to medical management (oral antibiotics, restricted protein diet and lactulose) and this reduces the risk of post operative complications (such as seizures). In the experience of the author, the incidence of anaesthetic and peri-operative complications after this system of medical management followed by elective surgery is extremely low.

Medical management

The majority of animals respond well to medical management, with improvements in signs of HE over a few days. Given this, medical management is recommended once a presumptive diagnosis has been established (on the basis of the BAST) and is always used for 4-6 weeks before surgical management is carried out.

In acute and severe cases of HE, evacuating enemata are used, together with per rectal instillation of lactulose, occasionally rectal povidone iodine (not in cats), intravenous fluid therapy and systemic antibiotics. In less severe cases and those with a chronic presentation, medical management consists of a restricted protein diet (a commercial diet intended for animals with liver dysfunction is ideal), oral antibacterials (often metronidazole or amoxycillin) and lactulose by mouth.

Animals with EH PSS in which surgical treatment is possible have a better longterm outlook than those managed by medical therapy. The general consensus is therefore that all affected animals should be offered surgery.

Although some experienced surgeons do not use portovenography, it does offer a gold standard in diagnosis and is occasionally essential to identify the shunt vessel and subsequently confirm occlusion. The most practical method of portovenography is through injection into a mesenteric vein (operative mesenteric portovenography, OMPV). (figure 1)

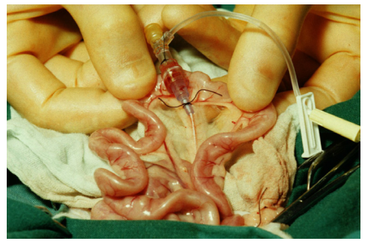

Figure 1.

The resulting image allows the presence of a shunt to be established and to be classified as extrahepatic (portocaval or portoazygous), acquired or intrahepatic. Radiographically, intrahepatic shunts are defined as those when the most caudal part of the shunt, seen on a lateral radiograph, is cranial to the thirteenth cervical vertebra on the lateral projection.

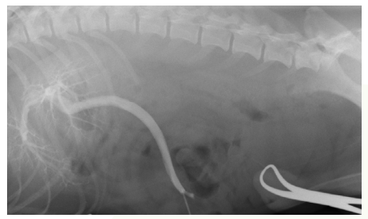

Figure 2. Portovenogram, normal dog

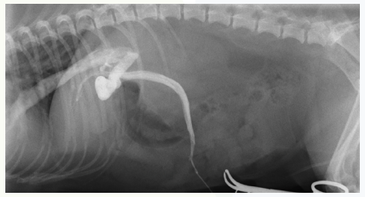

Figure 3. Portovenogram, extrahepatic shunt, dog

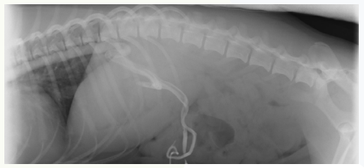

Figure 4. Extrahepatic portoazygous shunt, dog

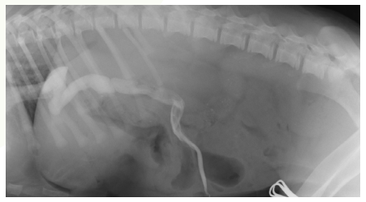

Figure 5. Intrahepatic shunt, Labrador retriever

Although ideally OMPV is performed with the benefit of fluoroscopy and image subtraction angiography, in almost all animals very satisfactory images are taken using standard CR or DR images. Taking the images in lateral recumbency is preferred by the author, although this means that the animal must be rolled during the laparotomy when the images are taken. When OMPV is performed, either the radiography is performed in theatre or the animal has to be moved during anaesthesia from theatre to the radiography room and back again, which is somewhat unsatisfactory. In either case, suitable personal protective precautions are required.

The abnormal vessel is isolated by the surgeon, using the images as a guide. Anatomically, extrahepatic portocaval shunts are typically in the mesoduodenum and enter the caudal vena cava cranial to the right renal vein and caudal to the liver. There are no other large vessels in this location (the phrenico-abdominal veins are much smaller than shunts).

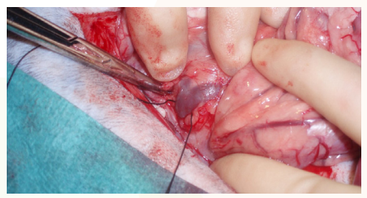

Figure 6. Test ligature placed around a typical extrahepatic portocaval shunt. The shunt is running from top to bottom of the image, with the portal vein running from left (caudally) to right (cranially).

Intrahepatic shunts are surgically much more challenging and more often are managed by interventional radiography (see specialist texts for further details).

Once the vessel has been identified, a test ligature is placed to provide temporary occlusion. The viscera are assessed for the development of portal hypertension: if this develops the shunt cannot be closed completely at once. In the absence of portal hypertension, the shunt can be ligated immediately. If portal hypertension develops (diagnosed by cyanosis of the intestine and pancreas), the shunt can be either partially ligated or closed over a few days. Options for gradual closure are placement of an ameroid constrictor, cellophane band or a hydraulic closure device. The author favours the ameroid constrictor (studies show similar outcome for all techniques).

Following surgery, significant complications can develop in the first 72 hours and patients are hospitalised during this critical period. The reported incidence of significant complications is around 5% for typical cases of extrahepatic shunts but is significantly higher for intrahepatic shunts. It should be emphasised that although anaesthetic and surgical management of extrahepatic shunts is relatively straightforward for clinical teams experienced with the disease, the outcome in other clinics may be less satisfactory.

Even after successful complete surgical occlusion, medical management is continued for at least 4 weeks until bile acid stimulation is repeated. At this time, a decline in bile acids is expected and medical management can be withdrawn in stepwise fashion.

Overview of medical versus surgical approach

Given the outcome of surgery for extrahepatic shunts, surgical investigation and management of all suspected cases is recommended and considered preferable to continued medical treatment. For the rarer cases of intrahepatic shunts, interventional radiography is preferred but the decision making process is less clear cut because of the greater mortality and failure to achieve complete ligation. In animals with either extrahepatic or intrahepatic PSS, the majority of animals managed medically alone do well in the short to medium term but there is progressive loss of liver function through life which leads to recurrence of signs or liver failure in due course.

Protein Losing Nephropathy

Eddie, a 3-year-old intact male American Cocker Spaniel, was presented for further investigation of his long-term hyporexia. Besides inappetence, Eddie experienced marked lethargy and melena a few days before presentation. On physical examination Eddie was quiet, but alert with pale mucosal membranes, a heart rate of 154, a body condition score of 3/9 and muscle condition score of 2/4 consistent with moderate muscle atrophy. Panting, gingival bleeding, several petechiae on the gums and moderate abdominal distension were noted as well.

Initial investigations included haematology, biochemistry, coagulation profile and AFAST ultrasound scan. Blood tests showed very severe thrombocytopenia (5 000/µL), non/pre-regenerative macrocytic, normochromic anaemia (PCV 11), leucocytosis with left shift and severe non-selective hypoproteinaemia (35 g/L) and hypoalbuminemia (13 g/L). Saline agglutination test and osmotic fragility test were negative. AFAST scan revealed large volume of clear fluid - ascites, which showed to be pure transudate (SG 1,008; TNCC < 0.5 x 10^3/µL, TP < 3 g/L). Clotting times and fibrinogen turned out to be within normal limits. In-house evaluation of blood film confirmed CBC results, did not reveal morphological changes of red blood cells and a blood transfusion was administered to replenish red blood cells.

After stabilisation urinalysis, chest x-rays (CXR) and abdominal ultrasound (AUS) as a part of the minimum database were obtained. CXR turned out to be unremarkable and besides ascites, AUS did not reveal any significant findings. Urinalysis showed marked proteinuria (UPC ratio 35.5), otherwise unremarkable. Eddie's blood pressure was within normal limits.

Because of high suspicion of primary immune-mediated thrombocytopenia (ITP) as a part of the multisystemic immune-mediated disease, immunosuppressive treatment (prednisolone) was commenced. Additionally, comprehensive evaluation for infectious diseases (Dirofilaria, Borrelia, Anaplasma and Ehrlichia spp.), ANA test and Coombs test were performed and showed to be negative. Meanwhile, gastroprotective (omeprazole, sucralfate) treatment, spironolactone, clopidogrel and standard therapy of suspected glomerular disease (benazepril and a diet with a reduced n-6/n-3 PUFA ratio) were initiated. Eddie responded well to the treatment, his platelet and red blood cell count returned to reference range, and corticosteroids were tapered over the course of 3 months. However, his UPC ratio remained high (8.3).

Given the suspicion for persisting glomerular disease, core needle biopsy of Eddie's kidney was performed, samples were processed under the stereomicroscope (Fig. 1-2), then transferred into Karnovsky fixative, Michel's medium and 4% buffered formalin and submitted for comprehensive renal histopathological evaluation including light microscopy with special stains, immunofluorescence and transmission electron microscopy. The results confirmed membranous glomerulonephritis (Fig. 3-6) – an immune complex-mediated glomerulonephropathy. Therefore, in addition to standard therapy of glomerular disease, treatment with mycophenolate has been instituted and Eddie is regularly monitored. One and a half year after the procedure, Eddie is doing well, he has no abnormalities in his blood tests and his last UPC ratio was 0.6.

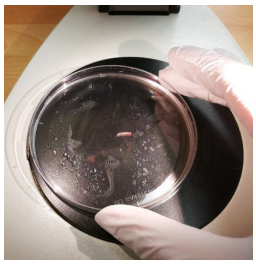

Fig 1. The renal tissue core retrieved from the needle biopsy device by using a gentle flow of sterilized normal saline solution.

Fig 2. Use a dissecting microscope to assess the composition of each biopsy core retrieved.

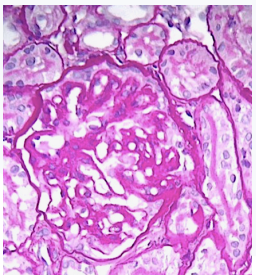

Fig 3. Glomerulus. The glomerulus is normocellular. Some capillary walls appear moderately thickened. PAS stain, 40x (Courtesy of Prof Luca Aresu)

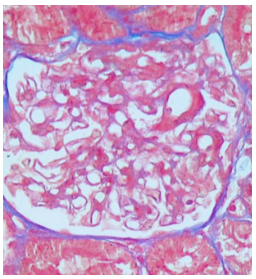

Fig 4. Glomerulus. Some capillary walls appear slightly thickened, but fuchsinophilic (red) deposits which are suggestive for immune complex deposits are not observed. Masson's Trichrome stain, 40x (Courtesy of Prof Luca Aresu)

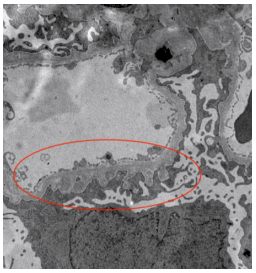

Fig 5. Capillary wall. Numerous electron dense deposits are present on the subepithelial surface of the capillary loops and remodeling of basement membrane is observed. Transmission electron microscopy (Courtesy of Prof Luca Aresu)

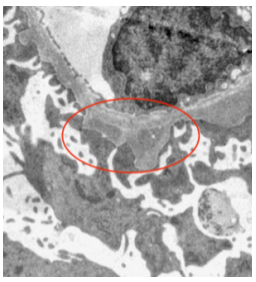

Fig 6. Capillary wall. Prominent spikes of the glomerular basement membrane are in between electron dense deposits. Transmission electron microscopy (Courtesy of Prof Luca Aresu)

To refer a case

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Please include any diagnostics reports and a case synopsis