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Portosystemic shunts in canines – an overview

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ABSTRACT: A vascular anomaly resulting in liver bypass, portosystemic shunts are a common hepatic abnormality affecting a variety of breeds through genetic or acquired means. Their classification is based on anatomic orientation of the shunt and a collection of non-specific neurological, urinary and gastrointestinal symptoms are exhibited, requiring definitive diagnosis with the use of imaging techniques. This article aims to review relevant literature to provide an overview of portosystemic shunt presentation and nursing considerations in practice.

Aetiology and clinical signs

Within typical anatomy, circulation is directed from digestive organs to the liver via the hepatic portal vein, ensuring the products of digestion are metabolised and stored or transported accordingly. However, in the presence of a portosystemic shunt, blood is emptied directly into systemic circulation, bypassing the liver (see **Figure 1**) (Cooper, Mullineaux, Turner, & Greet, 2011). Occurrence may be congenital or acquired and categorised as either intrahepatic or extrahepatic, with further classification based on exact anatomic position (see **Table 1**) (Nelson & Nelson, 2011). Congenital shunts are prevalent among pure-bred canines, suggesting an inherent nature (Fleming, Creevy, & Promislow, 2011), with intrahepatic shunts being most common in large breeds as a result of the ductus venosus remaining patent after birth (ACVS, 2017); inheritance is suggested to be digenic in Irish Wolfhounds (van Steenbeek, 2013). Conversely, genetic acquisition of extrahepatic shunts is more complex and remains largely unestablished (University of Cambridge, 2002). Typically affecting toy breeds, extrahepatic formation of vessels that empty directly in the caudal vena cava or azygous vein are considered to be a polygenic trait displaying no sex linkage, as its prevalence is equal in both genders (UFAW, 2012). While vascular abnormalities in congenital shunts are likely to be singular, acquired shunts are often multiple and arise due to hypertension, which is secondary to numerous conditions such as chronic liver disease (Center, 2016). Both types of portosystemic shunt produce a

range of neurological, urinary and gastrointestinal symptoms (Tobias, 2009).

Reduction in portal flow deprives the liver of hepatotropic factors essential for normal morphology and function (Trotta, Cajaiba, Parra, Dagli, & Hernandez-Blazquez, 2014). This results in hepatic atrophy and subsequent encephalopathy due to the circulatory presence of toxins that have avoided filtration, such as ammonia (ACVS, 2017), with accumulation augmented by colonic bacteria as a product of nitrogenous metabolism (Liu et al., 2004; Swanson et al., 2002). Neurological signs, ranging from abnormal behaviour and ataxia to head-pressing and seizures may be exhibited (Gow et al., 2012). Accumulation of ammonia also results in nausea and vomiting alongside crystalluria, where it readily chelates with uric acid to form ammonium biurate crystals, causing stranguria, haematuria or complete blockage (Rothuizen, 2009). Additionally, hepatic dysfunction reduces urea and protein production; diminished urinary concentrating abilities result in polydipsia and polyuria, and depleted synthesis of clotting factors, which are imperative for normal haemostasis, impairs coagulation (ACVS, 2017; Kummeling, Teske, Rothuizen, & Sluijs, 2006). Conversely, further studies have shown that decreased protein C and increased factor VIII levels, common in canines with hepatic encephalopathy, contribute to hypercoagulation, suggesting haemostatic variance in the presentation of individual shunts (Kelley, Lester, DeLaforcade, & Webster, 2013; Respass et al., 2012). Although uncommon with congenital portosystemic abnormalities, ascites may occur with acquired shunts or in the presence of severe hypoalbuminaemia (Tobias, 2009). Patients

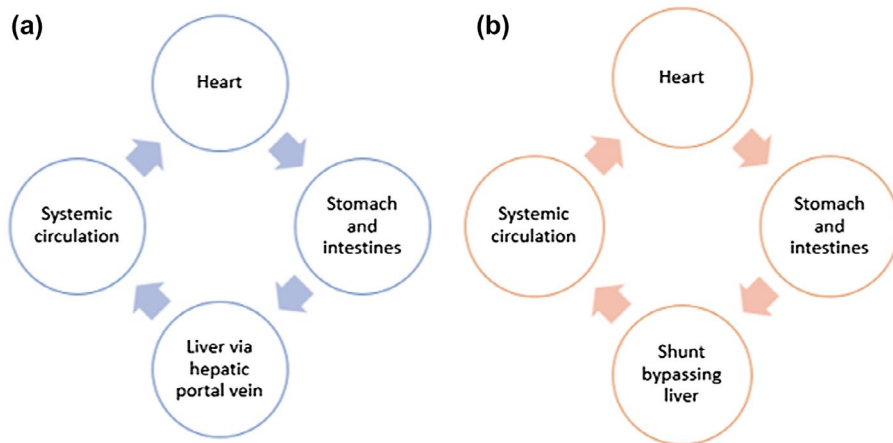


Figure 1. Comparative anatomy. (a) Normal portal flow. (b) Abnormal portal flow in the presence of a portosystemic shunt

portosystemic shunt patients attributed to its ability to be performed conscious, in contrast to other techniques which require sedation to prevent artefact traces caused by movement (King, 2016; Laitinen, Matheson, & O'Brien, 2013). However, image quality can cause difficulty identifying minor shunts; therefore, a technique producing better image resolution is indicated to form the most accurate diagnosis (King, 2016). While scintigraphy generates viable images, its limited anatomical detail proves ineffective in differentiating between intrahepatic and extrahepatic shunts, and the use of radioactive substances has negative health connotations (Morandi et al., 2005). Conversely, CT angiography uses an arguably safer

Table 1. Comparison of shunt classifications

	Congenital intrahepatic	Congenital extrahepatic	Acquired intrahepatic and extrahepatic
Anatomic orientation	Within the liver; with anomalous vessel occurring between the portal vein and vena cava	Outside of the liver; where the anomalous vessel may arise from a number of extrahepatic sites and empty into the vena cava or azygous vein	Either within or outside of the liver – dependent on the individual
Extent of abnormal vasculature	Most commonly single	Most commonly single	Often multiple
Acquisition	Genetic	Genetic	Secondary to disorders causing hypertension
Affected breeds	Large breeds, e.g. Irish wolfhound, Labrador retriever, English sheepdogs	Toy breeds, e.g. Yorkshire terrier, Cairn terrier, Maltese	No breed specificity
Treatment	Surgical with medical stabilisation – generally more challenging surgically	Surgical with medical stabilisation	Medical control only – dietary and pharmacological considerations

Source: Adapted from Tobias, 2009.

may also exhibit lethargy and poor growth, appearing significantly smaller than those of the same breed and age, attributed to impaired fat metabolism (Gaudiano, 2007). Recognition of clinical signs is the initial step in diagnosis, assisting with treatment indication.

Diagnosis

Blood profiles and urinalysis are valuable and relatively non-invasive methods of obtaining a differential diagnosis. While urine specific gravity is often low (Tobias, 2003), cytological investigation may reveal the presence of ammonium biurate crystals, appearing yellow-brown with crab-like morphology and needle-like processes (McClatchey, 2002). Haematology may exhibit abnormally small erythrocytes, with varying occurrence of defective iron binding, and a high level of leucocytes due to increased circulating bacteria (Day, 2013; Gaudiano, 2007). Additionally, biochemistry highlights hepatic dysfunction, showing reduced blood urea nitrogen and glucose. These values are non-specific; therefore, specialised liver function tests are indicated (Broome, Walsh, & Braddock, 2004). These include

testing levels of ammonia and serum bile acid, where increased pre-prandial concentrations correlate with shunt presence (Ruland, Fischer, & Hartmann, 2010). However, their individual specificity to portosystemic shunts is controversial. While Gerritzen-Bruning, Ingh, and Rothuizen (2006) suggest ammonia is of greater diagnostic benefit, Winkler, Bohling, Tillson, Wright, and Ballagas (2003) produced conflicting results, finding bile acids the most sensitive value. Nonetheless, laboratory samples are primarily a supportive measure in this instance and do not constitute a definitive diagnosis (Tobias, 2003).

Visualisation of the abnormal vessel is required for confirmation and is an imperative precursor to surgical attenuation (Nelson & Nelson, 2011). This can be achieved with a variety of imaging techniques, such as ultrasonography, computed tomography (CT) angiography and scintigraphy. Most commonly available, ultrasonography can identify portosystemic shunts and the presence of crystals, and is best utilised dorsally over the right thoracic cavity (Gaudiano, 2007). It is the safest method of diagnostic imaging for

contrast media to produce detailed anatomic images (Nelson & Nelson, 2011). Although identification issues can occur when the vessel travels transversely across the image (Zwingenberger, Schwarz, & Saunders, 2005), this non-invasive technique is a fast and reliable method of achieving accurate diagnosis of portosystemic shunts, facilitating surgical planning and guidance (Bertolini, Rolla, Zotti, & Caldin, 2006).

Treatment

Portosystemic shunts can be addressed medically and surgically, and the most appropriate treatment regime is indicated according to shunt classification (Tobias, 2003). Acquired shunts should only be managed medically, as their formation is a result of compensatory mechanisms whose prevention would cause serious adverse effects (Center, 2016). Additionally, medical control is indicated alongside surgery to achieve stabilisation and is primarily aimed at reducing circulating ammonia levels, thus preventing deleterious effects of hepatic encephalopathy (Gaudiano,

Table 2. Range of surgical techniques for shunt attenuation and subsequent considerations

Surgical technique	Description	Advantages	Disadvantages
Ameroid constrictor	A C-shaped ring of stainless steel placed around the vessel, where the inner casein absorbs surrounding fluid and swells gradually to obtain attenuation	<ul style="list-style-type: none"> Gradual occlusion allows for adaptation to portal blood flow and decreases risk of post-surgical hypertension Improvement of clinical signs 	<ul style="list-style-type: none"> May cause vessel curvature, occluding the shunt suddenly and provoking hypertension Has less application for multiple shunts
Cellophane banding	Placement of a cellophane band around the vascular anomaly, provoking inflammation and fibrosis which will gradually occlude the vessel	<ul style="list-style-type: none"> Gradual occlusion allows for adaptation to portal blood flow and decreases risk of post-surgical hypertension Improvement of clinical signs Can be fitted laparoscopically, minimising surgical wound and internal exposure 	<ul style="list-style-type: none"> Sterilisation, such as ethylene oxide, can weaken the strength of the bands May struggle to achieve full occlusion if larger than 3 mm; however, this may increase risk of portal hypertension depending on the size of the shunt
Coil embolisation	Placement of coils within the shunt, often secured with a stent; while number of coils is dependent on individual shunt size, the thrombogenic nature of the coils stimulate clotting and consequent gradual closure of the abnormal vessel	<ul style="list-style-type: none"> Minimally invasive surgery that is most useful for intrahepatic shunts, where surgical access is challenging 	<ul style="list-style-type: none"> Coil migration can occur in the absence of vena caval stent – is more common with extrahepatic shunts
Full ligation	Complete occlusion of the shunt using sutures	<ul style="list-style-type: none"> Can be achieved through a minimally invasive route, using Amplatzer vascular plugs 	<ul style="list-style-type: none"> Greater risk of portal hypertension and secondary shunting due to sudden changes in portal blood flow
Partial ligation	Partial occlusion of the shunt using sutures	<ul style="list-style-type: none"> Incomplete attenuation allows for adaptation to blood flow before complete closure, either by scar formation or secondary surgery 	<ul style="list-style-type: none"> Risk of portal hypertension and secondary shunting due to sudden changes in portal blood flow May require secondary surgery to achieve full attenuation should scar tissue not occlude it naturally Recurrence of clinical signs is common

Source: Adapted from ACVS, 2017; Frankel, Seim, MacPhail, & Monnet, 2006; Leveille, Johnson, & Birchard, 2003; Thieman Mankin, 2015; & Yoon et al., 2011.

2007). This can be achieved by moderately reducing protein intake, ensuring that of a high biological value is provided to decrease hepatic metabolism requirements (Tobias, 2009). Soy proteins are more effective in decreasing ammonia production while improving clotting ability compared to meat; however, neurologically asymptomatic canines may find protein restriction inconsequential, so care should be taken to elicit a balance, avoiding protein malnutrition (Lidbury, Cook, & Steiner, 2016). Zinc supplementation may also be required, as deficiency can exacerbate hepatic encephalopathy (Tobias, 2003). Furthermore, administration of lactulose inhibits ammonia formation and causes diarrhoea; while this can have adverse effects if used in excess, it reduces intestinal absorption and therefore hepatic workload, serving to alleviate symptoms (Salgado & Cortes, 2013). This can be administered orally or rectally in combination with an enema in severe cases (Gaudiano, 2007).

Broad-spectrum antibiotics can be used to facilitate the reduction of ammonia-producing colonic bacteria (Tobias, 2003). Metronidazole is commonly used, and while its efficacy in comparison to lactulose is controversial, diarrhoea occurrence is significantly reduced (Salgado & Cortes, 2013). Probiotics may also be advantageous in promoting positive bacterial

flora (McGee, Bakens, Wiley, Riordan, & Webster, 2011). However, surgical treatment (see Table 2) significantly improves long-term prognosis in comparison to the medical approach, and is the gold-standard treatment of congenital shunts (Greenhalgh et al., 2010). Complete or partial closures were the initial technique of choice; however, post-operation complications such as increased portal hypertension were of high risk (Broome et al., 2004). While dogs with a larger liver capacity were more likely to accept full attenuation (Doran, Barr, Hotston Moore, Knowles, & Holt, 2008), methods that supported gradual closure, such as ameroid constrictors, were developed to reduce these risks (Day, 2013). Medical stabilisation is a prerequisite of surgery and the pre-emptive use of levetiracetam, an anti-epileptic with minimal hepatic metabolism, can be useful in reducing post-operative seizing (ACVS, 2017; Fryer, Levine, Peycke, Thompson, & Cohen, 2011). However, surgery is complex and gold-standard anaesthetic monitoring is imperative (Gaudiano, 2007).

Anaesthetic and post-operative considerations

Hepatic dysfunction is detrimental to normal absorption, metabolism and elimination of anaesthetics agents, therefore careful selection is paramount (Waddell, 2012). Despite a long half-life

and hepatic metabolism (NOAH, 2017), intramuscular methadone can be used as a form of pre-medication to supply adequate analgesia and sedation that can be given incrementally, due to tolerance in those with stable liver disease and absence of toxic metabolites (Brown, Kraus, Fleming, & Reddy, 2004; Imani, Motavaf, Safari, & Alavian, 2014). Propofol induction is indicated due to rapid metabolism that also occurs extrahepatically via the lungs (Aspinall, 2011); similarly, maintenance is established with isoflurane, attributed to its minimal metabolism and subsequent lack of toxicity (Schauvliege, Seymour, Brearley, & Gasthuys, 2010). Being a largely exothermic organ, liver atrophy is detrimental to thermoregulation (Aspinall & Cappello, 2015). Exacerbated by anaesthetic agents, the patient is therefore at greater risk of hypothermia; heat-retaining techniques such as wrapping extremities in bubble wrap alongside provision of heat sources should be utilised, with regular rectal temperature readings to avoid overcompensation (Schauvliege et al., 2010).

Bearing a predisposition to hypoglycaemia due to reduced glucose metabolism (Tobias, 2009), this should be monitored incrementally with a glucometer and intravenous dextrose supplemented as necessary (Gaudiano, 2007). A further complication is the development of hypotension (Tobias,

2003); therefore, the provision of intravenous fluid therapy using a combination of colloid and crystalloid products to maintain adequate blood pressure and protein concentration is imperative (Waddell, 2012), especially as propofol can exacerbate this (Aspinall, 2011). Conversely, portal hypertension may arise during occlusion, hence the use of Doppler or direct blood pressure monitoring is indicated throughout, where although invasive, direct measurements provide greater accuracy (Cooper et al., 2011; Waddell, 2012). Additionally, clotting ability should be assessed prior to surgery via coagulation assays in order to establish the patient's haemostatic condition (Kummeling et al., 2006). This is an important consideration, ensuring that diathermy equipment and appropriate blood products are available in case of excessive haemorrhage to prevent the occurrence of hypovolaemic shock. Patients with portosystemic shunts present as a challenging anaesthetic, with hepatic dysfunction significantly altering a number of parameters which require intensive monitoring intra-operatively and upon recovery (Gaudiano, 2007).

Recovery should be monitored vigilantly during hospitalisation for at least 3–4 days post-surgery, with additional attention to signalment such as abdominal distension and the presence of blood in faeces that could indicate acute portal hypertension, requiring emergency surgery to loosen shunt ligation (ACVS, 2017). Therefore, blood pressure assessment should be continued post-operatively to ensure that developments can be addressed promptly (Thieman Mankin, 2015). Kennels should be kept warm and quiet, with a high standard of nursing care provided to assist patient well-being during recovery. Feeding should be encouraged promptly once the patient is suitably conscious to address hypoglycaemia, where greatest risk occurs immediately post-surgery (Tobias, 2009). Additionally, regular pain scores should be conducted to assess analgesia efficacy, as reduced hepatic metabolism may require dosing alterations (Waddell, 2012). Often occurring within 72 h of surgical ligation, seizures are thought to be instigated due to abrupt variations in benzodiazepines; therefore, neurological parameters, such as mentation, should be monitored closely during this time (Heidenreich, Giordano, & Kirby, 2016). These are commonly managed by intravenous propofol and phenobarbital infusion, minimising the risk of brain damage in those exhibiting status epilepticus; however, the occurrence of this is relatively uncommon, especially if medically stabilised prior to surgery

(Thieman Mankin, 2015; Tobias, 2009). Medetomidine has been suggested to possess “*neuroprotective, antihypertensive and drug sparing*” qualities that control excessive seizures, allowing reduced propofol doses and providing an effective alternative to levetiracetam (Heidenreich et al., 2016). During this period of induced unconsciousness, urinary catheterisation may be necessary to facilitate bladder relief, negating the risk of urinary scalding and providing accurate output monitoring (Welsh, 2013). Furthermore, regular turning and the use of passive-movement physiotherapy is indicated to prevent ulcer formation and joint stiffness. While surgery is often successful, possessing superior survival rates and minimal complications in comparison to medical management (Greenhalgh et al., 2010; Thieman Mankin, 2015), secondary shunts attributed to hypertension and recurrent seizures may occur; therefore, long-term medical regulation and symptom-based antiepileptic treatment is indicated (ACVS, 2017; Gaudiano, 2007).

Conclusion

Both acquired and genetic predisposing factors are attributed to portosystemic shunt formation in canines, and although the exact genetic inheritance remains unestablished for a number of breeds, this should be a consideration when assessing suitability for breeding. Accurate diagnosis is attributed to recognition of signalment, use of laboratory testing and diagnostic imaging, where experienced clinicians are often required to successfully identify this complex anatomical anomaly. This degree of hepatic deficiency presents a challenging anaesthetic, and veterinary nurses are at the forefront of providing intensive monitoring and supportive care throughout hospitalisation, executing a holistic approach to cater for patient well-being alongside clinical requirements. There is scope to advance present research and identify the exact mode of inheritance, assisting in the development of genetic screening for this condition to reduce prevalence in pure-bred canines, as although surgical management is largely successful in shunt correction and extending life expectancy, further health complications may occur in the long term.

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