Portosystemic shunts in dogs and cats:
imaging portosystemic shunts in small animals – hepatic vascular anatomy, shunt morphology, radiography

Portosystemische shunts bij honden en katten:
beeldvorming van portosystemische shunts bij kleine huisdieren – hepatische vasculaire anatomie, shuntmorphologie, radiografie

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ABSTRACT

Portosystemic shunts (PSSs) are anomalous vascular communications between the portal vein or its branches and the systemic venous system. Signalment, history, clinical signs, and laboratory findings can already provide a presumptive diagnosis of a PSS. However, imaging techniques such as portography, ultrasonography (US), nuclear scintigraphy, computed tomography (CT), or magnetic resonance imaging (MRI) are required to provide a definitive diagnosis. Nuclear scintigraphy is the gold standard for detecting PSSs, but it is not useful in distinguishing the different types of shunts. Due to its high sensitivity, portography was for a long time considered the gold standard for the differentiation of PSSs, even though it is an invasive technique. However, the recent development of a standard protocol for ultrasound (US) and the routine use of Doppler modalities resulted in the same sensitivity as portography. Therefore, with the additional benefit of being fast and noninvasive, US is now more commonly performed. It may be suggested as a “new” gold standard, depending upon the experience of the radiologist. Computed tomography and MRI provide detailed anatomic information. In this third part about portosystemic shunts in dogs and cats the different types of shunts and their diagnosis using portography will be explained.

SAMENVATTING

Portosystemische shunts (PSS’s) zijn abnormale vasculaire verbindingen tussen de vena porta of zijn vertakkingen en de systemische veneuze circulatie. Via het signalement, de anamnese, de klinische symptomen en de laboratoriumbevindingen kan een waarschijnlijkheidsdiagnose gesteld worden. Beeldvormingstechnieken, zoals portografie, echografie, nucleaire scintigrafie, computertomografie (CT), of magnetische resonantie (MR), zijn echter noodzakelijk om een definitieve diagnose te bekomen. Nucleaire scintigrafie is de gouden standaard voor de detectie van PSS’s, maar is niet nuttig om het onderscheid te maken tussen de verschillende shunttypen. Omwille van de hoge sensitiviteit werd portografie lange tijd beschouwd als de gouden standaard voor de differentiatie van PSS’s, alhoewel het een invasieve techniek is. De recente ontwikkeling van een standaardprotocol voor echografie en het routinematig gebruik van dopplertechnieken resulteerden in dezelfde sensitiviteit als die bij portografie. Echografie heeft als voordeel dat het snel en niet-invasief is; tegenwoordig wordt ze meer en meer uitgevoerd. Echografie kan beschouwd worden als een “nieuwe” gouden standaard, afhankelijk van de ervaring van de radioloog. Computertomografie en MR geven gedetailleerde anatomiche informatie. In dit derde deel over portosystemische shunts bij honden en katten worden de verschillende shunttypen en hun diagnosestelling met behulp van portografie beschreven.
INTRODUCTION

Portosystemic shunts (PSSs) are macroscopic vascular connections between the portal vein (PV) system and a systemic vein such as the caudal vena cava (CVC). These abnormal communications allow the portal blood from the intestine to bypass the liver and enter the systemic circulation. PSSs can be classified as congenital or acquired, single or multiple, and intra- or extrahepatic (Birchard et al., 1989; Lamb, 1996; Lamb and Daniel, 2002; Tillson and Winkler, 2002; Santilli and Gerboni, 2003; Broome et al., 2004; Cole et al., 2005). Ferrell et al. (2003) described the rare simultaneous occurrence of congenital and acquired extrahepatic PSSs in dogs.

Congenital portosystemic shunting is considered if a single or double abnormal vessel is present without accompanying portal hypertension. Eighty percent of all PSSs in dogs are congenital PSSs (CPSSs), which are typically classified as intra- or extrahepatic, based upon their location (Boothe et al., 2004; Szatmári et al., 2004b). Intrahepatic shunts make up approximately one-third of the CPSSs (Lamb, 1996; White et al., 2003; Broome et al., 2004; Szatmári et al., 2004b).

Acquired PSSs (APSSs) are considered when multiple small extrahepatic collateral vessels occur secondary to portal hypertension (Boothe et al., 1996; Johnson, 1999; Lamb and Daniel, 2002; d’Anjou et al., 2004; Szatmári et al., 2004a). Acquired PSSs constitute approximately 20% of all PSSs in dogs (Boothe et al., 1996; d’Anjou et al., 2004). They are less commonly detected in cats. Acquired PSSs originate from normal, preexisting, nonfunctional communications between the PV and the systemic circulation. These communications open secondary to and compensate for sustained portal hypertension (Bostwick and Twedd, 1995; Boothe et al., 1996; Center, 1996b; Johnson, 1999; Lamb and Daniel, 2002; Tillson and Winkler, 2002; Ferrell et al., 2003; d’Anjou et al., 2004; Szatmári et al., 2004a).

Acquired PSSs are commonly caused by increased intrahepatic portal venous resistance due to liver fibrosis or cirrhosis, but can also appear secondary to arterioportal fistulae, PV thrombosis, PV hypoplasia (also known as idiopathic noncirrhotic portal hypertension), and PV aplasia or atresia (Johnson, 1999; Szatmári et al., 2000; Lamb and Daniel, 2002; Ferrell et al., 2003; Santilli and Gerboni, 2003; White et al., 2003; d’Anjou et al., 2004; Szatmári et al., 2004b; Zandvliet et al., 2005). Portal vein hypoplasia also occurs very commonly secondary to reduced hepatic portal flow, in association with a CPSS (White et al., 2003).

Hepatic microvascular dysplasia (HMD), unlike PSSs, involves intrahepatic microscopic shunting of blood. It may occur alone or associated with a macroscopic shunting vessel (Broome et al., 2004). The relationship between HMD and CPSSs is unclear (Johnson, 1999). Hepatic microvascular dysplasia is characterized by the presence of residual “juvenile-like” intralobular blood vessels, through which portal blood flow runs directly to the central veins, bypassing the sinusoids (Santilli and Gerboni, 2003). It is being reported in an increasing number of dog breeds and represents an important differential diagnosis of PSSs. It causes high serum bile acids concentrations without demonstrable macroscopic portosystemic shunting (Center, 1996a; Johnson, 1999; d’Anjou et al., 2004; Szatmári et al., 2004b).

Signalment, history, clinical signs, and laboratory findings can already provide a presumptive diagnosis of a PSS (Levy et al., 1995; Center, 1996b; Tillson and Winkler, 2002; Santilli and Gerboni, 2003; Broome et al., 2004). However, imaging techniques such as radiography (Wrigley et al., 1987a; Birchard et al., 1989; Santilli and Gerboni, 2003; Broome et al., 2004), ultrasonography (US) (Lamb, 1996; Lamb, 1998; Lamb and White, 1998; Szatmári et al., 2004b), nuclear scintigraphy (Daniel et al., 1990; Koblik et al., 1990a and 1990b; Cole et al., 2005; Morandi et al., 2005), computed tomography (CT) (Frank et al., 2003; Thompson et al., 2003; Zwingenberger and Schwarz, 2004; Zwingenberger et al., 2005), and magnetic resonance imaging (MRI) (Seguin et al., 1999) are required to provide a definitive diagnosis and are very helpful in localizing the shunt and assessing its morphology before surgical attenuation. These imaging techniques enable efficient and rapid surgical manipulation of the shunt, which results in less anesthetic time (Birchard et al., 1989; Johnson, 1999; Lamb and Daniel, 2002; Santilli and Gerboni, 2003).

Portography was considered the gold standard in dogs suspected of having a PSS (Johnson, 1999; Lamb and Daniel, 2002; Miller et al., 2002; Tillson and Winkler, 2002), because the interpretation of the portographic images is quite easy. It allows differentiation of CPSSs versus APSSs and it can be used to distinguish intra- from extrhepatic shunts (Szatmári et al., 2004a). However, it is time-consuming, invasive and requires radiation (Lamb and Daniel, 2002; Broome et al., 2004; Szatmári et al., 2004a). These downsides, combined with the development of more advanced and less-invasive techniques, have led to decreased use of portography.

Ultrasonography has been used for diagnostic imaging of dogs with CPSSs since the 1980s (Wrigley et al., 1987b). It has become popular because it is quick and noninvasive, and it does not require anesthesia or ionizing radiation. It was found to be nearly as sensitive a method for detecting extra- and intrahepatic PSSs as portography (Holt et al., 1995; Scrivani et al., 2001; Szatmári et al., 2004a and 2004b). Furthermore, it allows simultaneous evaluation of the abdominal organs and, when using Doppler, it can be used to assess abnormal blood flow (Szatmári et al., 2003, Szatmári et al., 2004a).

Although scintigraphy is considered the gold standard for detecting PSSs, it is not useful for distinguishing CPSSs from APSSs, or intrahepatic from extrhepatic shunts (Broome et al. 2004; Szatmári et al., 2004a; Cole et al., 2005; Morandi et al., 2005).

Computed Tomography and MRI can provide anatomic information. However, unlike US, these modalities cannot give information on the direction of blood flow in the vessels examined. Moreover, both procedures are time-consuming, use expensive equipment, and require general anesthesia (Seguin et al., 1999; Frank et al., 2003; Szatmári et al., 2004a;
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The hepatic vascular anatomy, its shunt morphology, and the diagnosis using portography are discussed in more detail.

HEPATIC VASCULAR ANATOMY AND SHUNT MORPHOLOGY

The portal system receives blood from the cranial and caudal mesenteric veins, the splenic vein and the gastroduodenal vein, which drain the gastrointestinal tract, pancreas, spleen, caudal thoracic esophagus and the greater part of the rectum (Center, 1996b; Tillson and Winkler, 2002; Broome et al., 2004).

In dogs, the intrahepatic PV consists of a right and a left branch. The left branch supplies the left (the papillary process of the caudate lobe and the left lateral and medial lobes) and central divisions of the liver (right medial and quadrate lobes). The right branch supplies the right hepatic divisions (the caudate process of the caudate lobe and the right lateral lobe) (Figure 1; Center, 1996b; Tillson and Winkler, 2002; Broome et al., 2004). The right branch reaches the liver before the left branch, which may be noted during positive-contrast portography (Center, 1996b). The PV in the cat divides into right-, central- and left divisions (Tillson and Winkler, 2002; Broome et al., 2004).

The hepatic artery originates from the celiac artery. The celiac artery lies dorsal to the PV and common bile duct. It completes an arch before terminating as the right gastric and gastroduodenal arteries. Usually, each hepatic division has its own arterial supply. Two or three arterial branches originate from the arch and supply the liver (Center, 1996a).

The hepatic veins are variable in number and location, and they enter the CVC before it crosses the diaphragm. Usually there are 6 to 8 hepatic veins. The left hepatic vein is the largest and the most consistently and most cranially located segment (Center, 1996b; Broome et al., 2004).
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Intrahepatic shunts have been classified as left-, central- or right-divisional (Figure 2(A-C); Bostwick and Twedd, 1995; Lamb et al., 1996; White et al., 1996; Lamb and White, 1998; Lamb and Daniel, 2002; Szatmári et al., 2004b). Left-divisional shunts (patent ductus venosus) are shunts originating from the left portal branch. Right-divisional shunts are long shunts originating from the right portal branch, whereas central-divisional shunts are short shunts originating from the right portal branch (Szatmári et al., 2004b). Left-divisional shunts occur most commonly (Center, 1996b; Broome et al., 2004; Tobias et al., 2004). They are caused by failure of the fetal ductus venosus to close properly after birth. The ductus connects the embryologic left umbilical vein and the cranial portion of the right vitelline vein (Bostwick and Twedd, 1995; Center, 1996b; Santilli and Gerboni, 2003; Broome et al., 2004). In puppies, the ductus venosus normally closes two to six days after birth (Lamb and White, 1998; Broome et al., 2004; Lamb and Burton, 2004; Tobias et al., 2004). The origin of central- and right-divisional intrahepatic shunts is unknown (Lamb and White, 1998; Broome et al., 2004).

Intrahepatic PSSs are diagnosed as large vessels connecting the intrahepatic portal and hepatic venous vascular beds. They usually keep a uniform diameter throughout, are larger than normal hepatic vessels, and connect the intrahepatic portal and hepatic venous vascular beds. They usually keep a uniform diameter throughout, are larger than normal hepatic vessels, and are expected to be a tributary of the gastroduodenal vein or of the PV itself. In both cases, however, the shunt originates immediately caudal to the portal bifurcation. The course of the shunt was found to be always the same: a long loop starting from the porta hepatis, along the left body wall, turning caudomedially and terminating into the CVC at the point where splenic-caval shunts terminate. The (occasionally) absent caudal shunt loop originates in the region where splenic-caval shunts are expected. This loop, however, has a caudal-to-cranial direction instead of a ventral-to-dorsal direction as in splenic-caval shunts (Szatmári et al., 2004b).

Congenital PSS’s commonly terminate into the left aspect of the CVC just cranial to the phrenicoabdominal veins, or into theazygos vein (Bostwick and Twedd, 1995; Center, 1996b; Lamb and Daniel, 2002; Broome et al., 2004; Szatmári et al., 2004b; Cole et al., 2005). Besides terminating into the abdominal portion of the CVC, CPSSs can also empty into its thoracic portion (Szatmári et al., 2004b). The shunting vessel of splenic-azygos shunts runs towards the CVC and does not terminate, but continues further dorsal to the CVC. It eventually enters the thorax (Holt et al., 1995; d’Anjou et al., 2004; Szatmári et al., 2004b). Right gastric-azygos shunts do not enter the CVC, but pass it and enter the thorax (Szatmári et al., 2004b). Occasionally, CPSSs empty into the hepatic veins, renal vein, phrenicoabdominal vein or the internal thoracic vein (Tillson and Winkler, 2002; Santilli et al., 2003).

Acquired shunts are small tortuous veins in the omentum or retroperitoneum near the left renal vein (Boothe et al., 1996; Johnson, 1999; Lamb and Daniel, 2002). They originate from the main trunk of the PV or one of its tributaries, for example the splenic vein, the left gastric vein, or the right gastric vein. The most common type of CPSS is a splenic-caval shunt (Bostwick and Twedd, 1995; Center, 1996b; Lamb and Daniel, 2002; Broome et al., 2004; d’Anjou et al., 2004; Szatmári et al., 2004b; Cole et al., 2005). Extrahepatic PSSs are diagnosed when an abnormal branch of the PV or one of its tributaries is identified caudal to the porta hepatis. PSSs that do not terminate in the CVC, but can be traced cranially and dorsally to the diaphragm, are presumed to be portaazygos shunts (Holt et al., 1995; d’Anjou et al., 2004; Szatmári et al., 2004b).

A splenic-caval shunt usually shows as a short dilated loop between the PV and the CVC. The point of origin of the shunt is very close to the point where the splenic vein enters the PV. The CPSS seems to originate from the PV itself and the splenic vein seems to enter the shunting vessel. The origin of a splenic-caval shunt is slightly cranial to the level where the celiac artery originates from the aorta (Szatmári et al., 2004b).

Fewer shunts have been described that involve the gastroduodenal and mesenteric vessels (Center, 1996b; Broome et al., 2004). Most dogs with right gastric-caval shunts have double shunts. There is a cranial shunt loop and a caudal shunt loop, which anastomose and form a common trunk before entering the systemic venous system. The cranial loop originates from the right gastric vein. The right gastric vein can be a tributary of the gastroduodenal vein or of the PV itself. In both cases, however, the shunt originates immediately caudal to the portal bifurcation. The course of the shunt was found to be always the same: a long loop starting from the porta hepatis, along the left body wall, turning caudomedially and terminating into the CVC at the point where splenic-caval shunts terminate. (occasionally) absent caudal shunt loop originates in the region where splenic-caval shunts are expected. This loop, however, has a caudal-to-cranial direction instead of a ventral-to-dorsal direction as in splenic-caval shunts (Szatmári et al., 2004b).

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They have typical patterns. In small animals, splenorenal and mesenteric collaterals are most commonly encountered, but gastrohepatic, pancreaticoduodenal, or rarely hemorrhoidal collaterals can also be found (Cole et al., 2005; Morandi et al., 2005). These last two cannot be identified on splenoportography and trans-splenic portal scintigraphy (Cole et al., 2005). The collateral vessels of APSSs only occasionally arise directly from the PV (Szatmári et al., 2004a). The morphology of APSSs that originate from the PV shows several differences compared with congenital extrahepatic splenic-caval shunts. The most important difference is that APSSs run caudally from the point of origin and it is very difficult to follow their path (Booth et al., 1996; Ferrell et al., 2003; d’Anjou et al., 2004; Szatmári et al., 2004b), whereas CPSSs go cranially from their origin and can always be followed from their origin to their termination. Furthermore, APSSs are never wider than the PV caudal to the shunting vessel, whereas a CPSS is usually wider (Szatmári et al., 2004b).

**PLAIN ABDOMINAL RADIOGRAPHY**

Abdominal survey radiographic findings are usually unremarkable but can include a small liver (microhepatica), enlarged kidneys, urinary calculi, and loss of serosal detail (Booth et al., 1989; Gonzalo-Orden et al., 2000; Steyn, 2000; Lamb and Daniel, 2002; Santilli and Gerboni, 2003). Microhepatica, a common finding in dogs with a CPSS, is a less consistent finding in cats (Levy et al., 1995; Johnson, 1999). Mild renomegaly (normal kidney size: 2.5-3.5 times the length of L2 in dogs; 2.4-3.0 times the length of L2 in cats) (Feeney and Johnston, 2002) is occasionally noted in dogs and cats with a CPSS. Ammonium urate calculi, when present, are usually invisible, unless they contain considerable amounts of magnesium and phosphate (Johnson, 1999; Steyn, 2000). Animals with PSSs frequently have reduced intraabdominal fat or ascites, resulting in a loss of serosal detail (Birchard et al., 1989; Booth et al., 1996; Johnson, 1999; Steyn, 2000; Tillson and Winkler, 2002; Ferrell et al., 2003).

**POSITIVE-CONTRAST PORTOGRAPHY**

Positive-contrast portography was considered the gold standard for imaging PSSs (Johnson, 1999; Lamb and Daniel, 2002; Miller et al., 2002; Tillson and Winkler, 2002; Frank et al., 2003; Broome et al., 2004). Water-soluble iodinated contrast agents are used. Several methods can be used: the contrast can be introduced into arteries supplying the bowel (cranial mesenteric arteriography), into a mesenteric vein (intraoperative mesenteric portography), into the pulp of the spleen (percutaneous splenoportography), or retrograde into subsequently the aygos and the CVC through the jugular vein (transvenous retrograde portography) (Schmidt and Suter, 1980; Birchard et al., 1989; Johnson, 1999; Steyn, 2000; Lamb and Daniel, 2002; Miller et al., 2002; Broome et al., 2004).

Portography confirms the presence of a PSS and it provides information on the anatomical location of the vessel, the direction of portal blood flow, and the patency of the PV and its intrahepatic branches (Levy et al., 1995; Lamb and Daniel, 2002; White et al., 2003; Broome et al., 2004).

During portography, some confusion may exist about the intra- or extrahepatic location of the PSS. For example, poor visualization of the liver due to loss of serosal detail can hamper classification of the shunt (Birchard et al., 1989). To avoid this problem, Birchard et al. (1989) evaluated the shunt location relative to the thoracolumbar spine. They found that if the caudal aspect of the PSS (where the shunt diverges from the PV) is cranial to T13, the shunt is probably intrahepatic. If this caudal aspect is caudal to T13, there is a high probability of the shunt being extrahepatic. Furthermore, if the caudal extent of the shunt does not extend caudally to the T12/T13 intervertebral disc space during full inspiration, it is likely to be intrahepatic. If any portion of the shunt is at T13 or caudally during full expiration, it is likely to be extrahepatic (Birchard et al., 1989).

Recently, Scrivani et al. (2001) found that left lateral positioning during portography resulted in the highest sensitivity for detection of PSSs. The sensitivity of mesenteric portography for detecting an abnormal portosystemic blood vessel was 85, 91 and 100% in dorsal, right lateral, and left lateral recumbency, respectively.

The most important disadvantage of operative mesenteric portography is its invasive nature (Lamb and Daniel, 2002; Frank et al., 2003; Broome et al., 2004), and the need for mobile radiographic equipment or a radiography room clean enough for a surgical procedure (Lamb and Daniel, 2002; Szatmári et al., 2003; Broome et al., 2004). Moreover, using contrast portography, the ability to outline a vessel is dependent on the flow rate, concentration and volume of the contrast agent, and the position of the patient (Scrivani et al., 2001; Cole et al., 2005).

Radiographic subtraction techniques make portography more sensitive. These techniques remove the images of the overlying intra-abdominal structures, which improves the radiographic contrast of the portal system on portal venograms. The improved quality of the subtracted portograms aids in the detection of PSSs and intrahepatic PV branches. Subtraction studies are able to reveal PSSs not detected on the initial portal venogram (Wrigley et al., 1987a; Steyn, 2000).

**Operative mesenteric portography**

This is the most commonly used and preferred technique because it allows a high-quality study of the portal system, it does not require special equipment, and it results in few complications (e.g. infection, bleeding, extravasation of contrast medium). It is performed with the patients under general anesthesia. A loop of jejunum is isolated through a ventral midline incision. A catheter is placed surgically in a jejunal vein and secured with ligatures, after which the iodinated contrast medium is injected as a bolus (1-2 ml/kg) (Holt et al., 1995; Levy et al., 1995; Center, 1996b; Johnson, 1999; Steyn, 2000; Lamb and Daniel, 2002; Tillson and Winkler, 2002; San-
tilli and Gerboni, 2003) with a maximum dose of 2-4 ml/kg (Broome et al., 2004). In the early days, the injection was recorded using a rapid film changer (Steyn, 2000; Lamb and Daniel, 2002; Santilli and Gerboni, 2003; Broome et al., 2004). Nowadays, however, videotape, CDs, and DVDs are used.

A normal portogram reveals the PV trunk and its multiple intrahepatic branches (Figure 3A). The portogram of a patient with a PSS initially shows contrast in the PV, which then moves into the CVC or azygos vein. A tortuous vessel starting from the portal system usually represents the shunt (Figure 3B; Johnson, 1999; Tillson and Winkler, 2002). Intrahepatic PV branches may or may not be opacified. Animals with portal atresia do not show any evidence of either hepatic parenchymal opacification, an intrahepatic portal vasculature, or a PV entering the liver (Johnson, 1999; White et al., 2003). In cases of portal hypoplasia, portography shows dilation of the extrahepatic PV, APSSs, an insufficient number of middle-sized intrahepatic PV branches, and sudden ending of the peripheral venous branches (Johnson, 1999; Bunch et al., 2001). Therefore, failure to outline the intrahepatic portal system may suggest higher intrahepatic vascular resistance. These patients are more likely to develop postoperative complications (Johnson, 1999).

Cranial mesenteric arteriography

With this alternative technique, contrast medium is selectively delivered to the cranial mesenteric artery via a femoral arteriotomy using fluoroscopic guidance (Schulz et al., 1993; Center, 1996b; Steyn, 2000; Lamb and Daniel, 2002). The catheter tip is placed in the origin of the cranial mesenteric artery (Center, 1996b; Steyn, 2000). Following injection of contrast medium, the contrast bolus passes through the intestinal capillary bed and opacifies the PV (Schulz et al., 1993; Steyn, 2000; Lamb and Daniel, 2002). An important disadvantage of this technique is the dilution of the contrast by the time it reaches the PV (Wrigley et al., 1987a; Schulz et al., 1993; Center, 1996b; Steyn, 2000). It is used less often than operative mesenteric portography because it requires more experience (Lamb and Daniel, 2002).

Splenoportography

Splenoportography is performed by percutaneous (or operative) injection of contrast into the splenic parenchyma. The contrast is then taken up rapidly by the sinusoids into the portal system (Schulz et al., 1993; Center, 1996b; Steyn, 2000). It is a simple and relatively easy technique to perform. In right lateral recumbency, the needle is advanced through the splenic parenchyma and the tip of the needle is positioned as close to the hilus as possible. This results in optimal venous drainage of the contrast medium (Schmidt and Suter, 1980). In right lateral recumbency, the position of the spleen is more consistent, and its inclination to be displaced when introducing the needle is less than in ventrodorsal or dorsoventral positions (Schmidt and Suter, 1980). Schulz et al. (1993) were able to position the catheter into a major splenic vein at the hilus (transsplenic portal catheterization). Complications are splenic laceration and bleeding from the puncture site, and dislodgement of the catheter (Schmidt and Suter, 1980; Schulz et al., 1993; Center, 1996b). The risk of complications decreases with experience, and when a correct technique is followed (Schmidt and Suter, 1980). The number of failures can be reduced by using fluoroscopy (Schmidt and Suter, 1980) or US (Moon, 1990).

The most important disadvantage of this technique is that shunts caudal to the splenic vein will not be recognized, thus resulting in a possible false-negative diagnosis (Schmidt and Suter, 1980; Schulz et al., 1993; Center, 1996b; Steyn, 2000; Cole et al., 2005). Moreover, the amount of contrast medium needed to opacify the PV within a short time is limited by the draining capacity of the
splenic pulp and the blood flow in the splenic vein (Schmidt and Suter, 1980).

Transvenous retrograde portography (TRP)

As for cranial mesenteric arteriography and percutaneous splenoportography, TRP provides an alternative method of identifying and characterizing PSS’s without requiring abdominal surgery (Miller et al., 2002).

The balloon-tipped catheter is inserted through the jugular vein and is directed caudally in the cranial vena cava and then in a dorsal direction into the azygos vein. The catheter is advanced as far as possible until resistance is observed. The balloon is inflated, which occludes the azygos vein. Radiographic contrast medium (1-2 mL/kg) is injected during continuous fluoroscopic evaluation. This results in retrograde filling of the intercostal and intervertebral veins. Via the intervertebral veins, the contrast flows in a retrograde fashion into the prehepatic portion of the CVC (Miller et al., 2002).

Following this injection, the catheter is withdrawn into the cranial vena cava and then advanced caudally through the right atrium into the CVC. The catheter is positioned immediately cranial to the diaphragm, after which the balloon is inflated, occluding the CVC. The second injection, performed immediately after occlusion, results in retrograde filling of the abdominal portion of the CVC and any PSS (Miller et al., 2002). A PSS could be identified and characterized with TRP in 18 of 20 dogs (Miller et al., 2002).

Once the shunt is identified, selective catheterization and injection of contrast medium allows more specific opacification of the shunt, providing more detailed anatomic information. Furthermore, the catheter can be left in place in the lumen of the shunt, thus assisting in identification of the shunt during surgery (Miller et al., 2002).

CONCLUSION

Because of the non-specificity of the clinical signs and laboratory findings, imaging techniques such as portography, ultrasonography, nuclear scintigraphy, computed tomography, and magnetic resonance imaging are required to provide a definitive diagnosis of a PSS.

Due to its high sensitivity in detecting PSSs (85, 91 and 100% in dorsal, right lateral, and left lateral recumbency, respectively) (Scrivani et al., 2001) and its simple interpretation, portography was considered the gold standard in dogs suspected of having a PSS. However, it is time-consuming, invasive and requires radiation.

Nowadays, US can be considered the “new” gold standard for the differentiation of PSSs because it is quick and noninvasive, because it does not require anesthesia or ionizing radiation, and because it has the same sensitivity as portography. This will be further explained in the next part of this review of the literature.

LITERATURE

An extended literature list can be obtained from the authors.

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