Artykuł przeglądowy *Review*

Slow-flow vascular abnormalities of the portal vein in dogs

ANNA ŁOJSZCZYK[1], MATEUSZ WEREŻYŃSKI[2]

[1]Laboratory of Radiology and Ultrasonography, Department and Clinic of Animal Surgery, Faculty of Veterinary Medicine, University of Life Sciences in Lublin, Głęboka 30, 20-612 Lublin, Poland
[2]Radiologia Weterynaryjna TK VET, Sądowa 9/11, 50-33 Wrocław, Poland

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Łojszczyk A., Wereżyński M.

Slow-flow vascular abnormalities of the portal vein in dogs. Part I – portosystemic shunts

Summary

Vascular abnormalities are described in animals, especially portosystemic shunts (PSS), which have been documented to be greater than 0.18%-1% in dogs (14, 36). Portal vein (PV) abnormalities are one of the most important vascular problems, as they have a serious clinical impact on dogs. These include PV anomalies and PV anomalous connections (4). In the first group, the literature describes congenital absence of the PV, PV hypoplasia and primary PV hypoplasia (4). The second includes high flow (hepatic arteriovenous malformation and arterioportal fistulas) and low flow abnormalities – congenital intrahepatic and extrahepatic shunts, acquired PSS, porto-portal collaterals in cases of PV obstruction and cavo-portal collaterals in chronic increased resistance of the caudal vena cava (4). In this paper, the authors discuss low-flow venous abnormalities associated with PV pathology and PV anomalies, which first requires updating the current knowledge.

Keywords: computed tomography, ultrasound, portosystemic shunts, vascular abnormalities, portal vein

Vascular abnormalities are described in animals, especially portosystemic shunts (PSS)which have been documented in dogs at rates 0.18%-1% (15, 40). Unfortunately, there are not many publications on other possible abnormalities that could also affect dogs and cats, which are most commonly reported due to the growing availability of advanced imaging techniques in veterinary practice (23). There is also a need to systematize knowledge in this field and update it based on new publications, the number of which has increased significantly in recent times.

The standard for diagnosing vascular abnormalities is computed tomography angiography and ultrasonography. Diagnostic imaging is widely used to confirm a clinical suspicion of this diagnosis (15).

In animals, a broad spectrum of congenital and acquired anomalies has been detected in recent years (23). All anomalies of the capillary, venous, lymphatic, and arterial systems can be divided into slow-flow (capillary, venous, and lymphatic) and fast-flow (arterial and arteriovenous) disorders (4, 22, 25, 37).

Portal vein (PV) abnormalities are one of the most important in this group, as they have a serious clinical impact on dogs. These include PV anomalies and PV anomalous connections (4). In the first group, the literature describes abnormalities including main portal trunk-congenital absence of the PV, PV hypoplasia, and primary PV hypoplasia (4). The second includes abnormal vascular connections: high flow (hepatic arteriovenous malformation and arterioportal fistulas) and low flow abnormalities (congenital intrahepatic and extra hepatic shunts, acquired PSS, porto-portal collaterals in cases of PV obstruction, and cavo-portal collaterals in chronic increased resistance of the caudal vena cava) (4).

In the following paper, the authors discuss low-flow venous abnormalities associated with PV pathology and PV anomalies, which first requires updating the current knowledge. In the paper, the remaining pathologies will be discussed.

Normal portal vein

Portal flow to the liver accounts for about 80% of the blood supply to this organ and the other 20% is supplied by arterial flow). Normally, the PV is formed primarily by the confluence of the cranial and caudal mesenteric veins with the splenic (often referred to as the gastrosplenic) vein from the left side and gastroduodenal vein from the right side (19, 29, 45).

These vessels have additional tributaries:

- The cranial mesenteric vein receives blood from the jejunal veins, the ileocolic vein, and the caudal pancreaticoduodenal vein (4, 29).
- The caudal mesenteric vein receives blood from the colic veins: left, middle, and right colic veins, as well as the cranial rectal vein (4, 29).
- The gastroduodenal vein receives blood from the cranial pancreaticoduodenal vein, the right gastroepiploic vein, and the right gastric vein (4, 29).
- The splenic vein receives blood from the left gastric vein, the left gastroepiploic vein, and the pancreatic branches (4, 29).

In the liver, the PV divides into the left and right branches (19). The left branch supplies the left liver lobes and the central part: the left medial lobe, the left lateral lobe, the quadrate lobe, and the papillary process of the caudate lobe, as well as an additional central branch to the right medial liver lobe (4, 19). Sometimes, the left portal branches supply the dorsal and the right lateral liver lobe (4).

The short right branch is responsible for the right part of the liver, especially the right lateral liver lobe and the caudate process of the caudate lobe (19). In cats, there are separate right, central and left branches (19). The number of portal branches entering individual liver lobes may vary among subjects (4).

The normal PV diameter in the porta hepatis is about 0.33-1.05 cm in dogs, with a ratio to the aorta of 0.71-1.20 or 0.8-1.15 according to publications (7, 22, 33). The normal portal flow is hepatopetal, with a mean portal flow velocity of 17 ± 4 cm/s, 15 ± 3 to 18 ± 8 cm/s, according to different authors (7, 17, 21, 28).

Portosystemic shunts (PSS)

PSS develop when abnormal vascular communications between the portal and systemic vessels form during fetal life without any additional portal hypertension (19, 39). These PSS are low-flow abnormalities of the venous system and are classified into intrahepatic (IHPSS) and extrahepatic (EHPSS) depending on shunt localization (15, 39). They are typically solitary, and they are present at birth (19, 45).

These connections permit venous blood draining from the gastrointestinal tract, the pancreas, and the spleen to enter directly into the systemic circulation (7, 19). Therefore, toxins and waste products from the GI tract bypass the liver, causing general intoxication that severely affects the central nervous system (7, 19, 39). A variety of clinical signs are related to hepatic insufficiency and risk of hepatic encephalopathy if more than 70% of hepatic function is lost (4, 30, 32). In these dogs, with PSS and hepatic encephalopathy, hyperammonaemia is observed (42).

Mainly, clinical signs are related to neurological problems: other, milder symptoms include delayed growth, gastrointestinal dysfunction (30% of affected dogs) and urinary tract abnormalities as ammonium urate urolithiasis (20, 23, 30, 42).

It has been proven that dogs with EHPSS have a significantly higher incidence of ammonium urate urolithiasis when compared to dogs with IHPSS (42). In EHPSS cases, neurological signs were noted less common than in dogs with higher ammonia levels and neurological signs (significantly higher in dogs with IHPSS and without urolithiasis) (42).

In 2024, an additional, previously unknown clinical problem has been described, that dogs with IHPSS may concurrently develop discospondylitis or vertebral physitis due to dysfunction of the reticuloendothelial system, which may predispose these dogs to bacteremia or niduses of infection (38). These dogs had clinical signs as stiff gait, spinal pain and pelvic limb ataxia (38). Almost all commonly reported clinical signs are summarized in Table 1.

In some cases, PSS remain clinically unapparent until old age (39, 42).

The breeds most commonly affected by EHPSS are small breeds, such as Yorkshire Terriers, mixed-breed dogs, Miniature Schnauzers, Maltese, Pugs, and, less likely, cats (18, 24, 40). IHPSS occur in large-breed dogs such as Labrador and Golden Retrievers, German Shepherd Dogs, Irish Wolfhounds, and Doberman Pinschers (18, 40).

In 2007, the association between IHPSS type and breed, country of origin (USA vs Australia), and gen-

Tab. 1. Clinical signs in PSS (14, 17, 37)

Neurological	Gastrointestinal	Urinary	Laboratory findings
– depression – listlessness – ataxia – pacing – circling – head pressing – cortical blindness – seizures – coma – decreased motility – disorientation – aggression – episodic weakness – stiff gait – spinal pain	– vomiting – diarrhea – anorexia – ptyalism (very often in cats) – melena – poor growth or lower body condition score	– dysuria – stranguria – pollakiuria – haematuria – ammonium urate uroliths – polyuria	– microcytic normochromic nonregenerative anaemia – elevated liver enzymes activities – hypoalbuminemia – decreased blood urea nitrogen – hypocholesterolemia – hypoglycemia – prolonged coagulation – elevated – pre- and postprandial serum bile acids – hyperammonaemia

der was described (20, 23, 30). Associations between left divisional IHPSS in Irish Wolfhounds and right divisional IHPSS in Australian Cattle Dogs have been reported, but these observations are not common for all authors (20, 23, 30).

Extrahepatic shunts (EHPSS)

In EHPSS, abnormal connections between the vitelline veins form in fetal life (4, 30, 31). These vessels form the portal system and the cardinal veins, which in turn form the systemic veins (4, 24, 32). The abnormal connection is present at birth, and in cases of EHPSS, the PV is often hypoplastic (4).

There are different types of these shunts. The type I shunt is very rare (end-to-side), with segmental congenital aplasia of the PV and a direct connection between the PV and the systemic vein, most commonly the CVC (4, 52). In these dogs, the gastroduodenal vein is often enlarged and occupies the position usually taken by the PV, lateral to the hepatic artery, which is also enlarged (51). Other abnormalities, such as CVC interruption with azygos continuation, may be observed with this anomaly (10, 51).

Most often, the type of shunt is type II – a side-to-side shunt, with one or more visible abnormal connections between the PV and the systemic circulation (4). The most commonly affected vessel at the origin of the shunt is the left gastric vein (about 95%), a finding consistent with the authors' own observations (4, 44, 48). The reason for this frequency is, according to many reports, that the development of the left gastric vein, which is critical in the formation of EHPSS that communicate with the CVC at the level of the omental foramen (48).

The classification of shunts includes the name of the portal vessel from which the shunt emanates and the name of the systemic vein to which it joins (48). In most cases, the left gastric vein inserts into the splenic vein. It could also insert, according to the latest reports, into the CVC, the azygos vein, or the phrenic vein – then it is called the aberrant left gastric vein (45). According to the latest research conducted on 1082 dogs, both of these versions can account for about 66.7% and 28% of affected animals with PSS (45). In rare cases, the splenic vein could also be absent or aberrant, with insertion into the CVC, not into the PV (45).

The systemic vessel where a shunt could be inserted is the phrenic vein, the azygos vein, and the CVC (nearly 50%) (45). For this reason, the most common shunts are described: left gastrophrenic (or splenophrenic), left gastro-azygos (or splenoazygos), splenocaval, and right gastrocaval shunts (15). They are often described also as right gastrophrenic, colonocaval, and portocaval shunts (19). In cats, 92% of shunts are spleno-caval, left gastro-phrenic, and left gastro-caval (19).

There are four consistent sites where the five most common EHPSS types enter the systemic venous circulation: prehepatic CVC at the level of the epiploic foramen (splenocaval, right gastrocaval shunts), the left phrenic vein at the level of the oesophageal hiatus (left gastrophrenic shunts), the azygos vein at the level of the aortic hiatus (gastro-azygos shunts) and the CVC or iliac vein at the level of the 6th or 7th lumbar vertebra (left colocaval and left colo-iliac shunts) (42). In 2023 atypical double EHPSS has been described with atypical path within the dorsolateral esophageal wall before entering the azygous vein (ewans).

In 2020, four consistent shunt types entering the CVC at the level of the omental foramen were described (48). These shunts appear to be a result of the abnormal communication between the left gastric vein and the prehepatic CVC, the presence or absence of an abnormal communication between the splenic, the left gastric, and the PV, and the subsequent development of preferential blood flow (hepatopetal or hepatofugal) through portal vessels (48).

In 2024, a study by Weiss et al. with 1082 dogs, 43 unique canine extrahepatic PSS conformations were identified, but the five most common shunts accounted for ~85% of all cases (39, 45). In this group, there are left gastric-phrenic (27%), left gastric-azygos (19%), left gastric-caval (15%), aberrant left gastric-caval with the right gastric vein (12%), and aberrant left gastric-caval with the right gastric vein and the short gastric vein (45).

More than one shunt was confirmed in 0.1% of examined dogs, and in one-third of all cases, additional portal vessels were identified that contributed blood flow to the shunt (45). It was especially evident when the portal vessel of origin was aberrant and when a shunt was inserted into the CVC (37). The most common contributing vessel was the right gastric vein (about 29%), the splenicogastric (or splenic, 15%) vein and the pancreatic vein (45). Despite abnormal anatomical location, these vessels are enlarged compared to normal (45).

There are also a few cases of simultaneous congenital and acquired PSS in dogs (9).

The anatomy of shunts is important as it has a serious clinical impact, according to various publications. Notably, dogs with EHPSS that insert cranially into the diaphragm, especially in portoazygos or portophrenic shunts, are reported to have less severe clinical signs, a lower prevalence of neurological clinical signs, and a better postsurgical prognosis (15). The low severity of clinical signs is probably due to partial compression of the PSS by the diaphragm during respiration and gastric distension following a meal (19). Patients with azygos shunts could also have milder clinical signs probably due to the increased resistance of the azygous vein versus the vena cava due to its smaller diameter (Poiseuille's law) (24).

These factors may be explained by the inconsistent clinical symptoms in some animals and by accidental diagnoses in some patients at a later age.

Dogs with a larger PSS diameter, similar to that of the aorta (larger PSS/Ao ratio), tended to be diagnosed at an earlier age (7, 8, 24).

Clear understanding of the vascular anatomy, influences also the successful surgery, to ensure the correct

vessel is attenuated at the appropriate location and to avoid missing any contributing branches (44, 47, 48). It is also important to be sure about other possible co-existing abnormalities as PSS has been described as coexist with previously mentioned CVC interruption with azygos continuation or circumcaval ureters (43).

Intrahepatic shunts (IHPSS)

In intrahepatic portosystemic shunts (IHPSS), there is an abnormal vascular communication between the portal venous system and the systemic circulation, typically through a hepatic vein or directly into the caudal vena cava. An abnormal shunt could be a persistent fetal ductus venosus or a new connection that forms during fetal development (19). Persistent ductus venosus in fetal life diverts blood arriving at the portal sinus from the umbilical vein to the left hepatic vein in utero and should be closed in the first three weeks of life (19, 26). When the vessel does not close, it is visible as a left divisional shunt with insertion into the left hepatic or left phrenic vein (19, 29).

Two other types of IHPSS are right (both are mentioned as most common) and central-divisional shunts (19, 36).

In the right divisional shunt, the place of vessel insertion is the right lateral hepatic vein or the caudate hepatic vein (18). In the central divisional shunt, insertion is in the central hepatic vein, the dorsal right medial hepatic vein, or in the ventral aspect of the CVC (18). Left and right divisional shunts are most common (19). Very rarely, complex IHPSS are diagnosed, which represents a fusion of a few types (6, 30).

In 2019 and 2023, new classifications of IHPSS were proposed. Research by Bertolini in 2019 proposed that the first phenotype – persistent ductus venosus – should include all IHPSS shunts: left, right, and central (4). The other proposed phenotypes were aneurysmal IGPSS, one or more PSS in a single liver lobe and multiple PSS in several liver lobes (4). According to Plested et al., subclassification of the left, central, and right divisional IHPSS is most useful, taking into consideration the placement of PSS venous insertion rather than portal vasculature, as these vessels are often small or not visible in dogs with IHPSS (30). Very rarely, IHPSS with multiple insertion sites may be observed, and they should be recognized as a distinct classification type (4, 30).

In 2023, one study classified eleven types of single congenital intrahepatic PSS, distinguishing interlobar and intralobar categories, noting that not all shunt subtypes represent persistent ductus venosus (41). These shunts pass between liver lobes (in a fissure between lobes), interlobar, or through a specific lobe (intralobar) (41). In the interlobar IHPSS, the authors included patent ductus venosus, left, right, and ventral shunts (41). In the intralobar – caudate process, the right lateral, right medial, quadrate, left lateral, left medial and papillary process shunt (41). This classification, according to the authors, could be essential to therapeutic planning and the remaining venous drainage post-occlusion (30, 41). The other important issue is that PSS is the only vascular abnormality, as a few types of malformations can sometimes be observed (30). To this moment, IHPSS was reported with arterioportal malformation (APM) and with multiple acquired extrahepatic PSS (30).

Some dogs with congenital IHPSS occasionally have also multiple smaller intrahepatic, tortuous blood vessels, surrounding the primary shunt which are similar in morphology to APM (36). These intrahepatic venous collaterals are significantly associated with the presence of a focal narrowing within the shunt or draining hepatic vein (36). They form in response to a partial obstruction to flow within the shunt, and are analogous to intrahepatic collaterals seen in patients with Budd-Chiari Syndrome, hepatic vein obstructions, or previous shunt attenuations at the level of the hepatic vein (36). These collateral vessels do not impact the treatment of choice although these IVCs could impact the "completeness" of shunt attenuation ultimately achieved (36).

Portal Vein Hypertension (PH), PV Hypoplasia, Acquired PSS Shunts

In dogs with portal hypertension, portal venous flow is often markedly reduced, and values below approximately 10 cm/s are considered strongly supportive of the diagnosis, although measurements should be interpreted in the context of Doppler angle and hemodynamic conditions. This problem is very often associated with serious parenchymal and vascular liver abnormalities (1, 6, 7, 39). Extrahepatic PV may also be hypoplastic or, in very rare cases, absent (4). A smaller extrahepatic PV has been observed in dogs with extrahepatic PSS and in some dogs with idiopathic noncirrhotic PH (6, 7).

In general, PH is caused by increased resistance and blood flow in the portal circulation and is classified as prehepatic, intrahepatic, or posthepatic (33).

Prehepatic PH is due to increased resistance in the extrahepatic PV and is associated with mural or intraluminal obstruction or extraluminal compression (33).

Intrahepatic PH could be due to increased resistance in the microscopic PV vessels (presinosoidal PH), sinusoids (sinusoidal PH), or small hepatic veins (33). There is also postsinusoidal PH due to veno-occlusive disease (31). All possible causes are in Table 2. The

Tab. 2. Causes of intrahepatic portal vein hypertension (7, 8, 35)

Presinusoidal PH	Sinusoidal PH	Postsinusoidal PH
Primary hypoplasia of the PV (PHPV)	Consequence of fibrotic hepatopathy as chronic hepatitis	Veno-occlusive disease- large hepatic veins, prehepatic CVC (cranial vena cava syndrome), right atrium
Idiopathic hepatic fibrosis		
Hepatoportal fibrosis		

most common type in dogs is primary hypoplasia of the PV with PH (33). In affected dogs with primary hypoplasia of the PV, PH appears to result from intrahepatic PV hypoplasia, which may represent a severe form of microvascular dysplasia (7, 8, 39). In this disease, microscopic intrahepatic portovenous shunting is present, causing the portal blood to bypass the liver sinusoids (8). Clinical signs are similar to EHPSS but typically less pronounced (8).

The PV/Ao ration in primary portal vein hypoplasia could range between 0.24-0.67 (24).

Acquired Portosystemic Shunts (APSS)

Multiple APSS constitute approximately 20% of all PSS in dogs (1, 5). These shunts are pre-existing, rudimentary embryonic connections that are normally nonfunctional and are present between the portal and caval circulations. They are dilating and becoming functional in response to sustained prehepatic or intrahepatic PH or increased resistance in the cranial vena cava system (1, 3, 4, 6, 7, 31, 32).

The majority of multiple APSS are due to diseases causing PH, such as idiopathic noncirrhotic PH due to primary PV hypoplasia or atresia, idiopathic hepatic fibrosis and other acquired hepatopathies, arteriovenous malformations, PV thrombosis, or veno-occlusive diseases (1, 6, 7, 39). Hepatic microvascular dysplasia, reported in an increasing number of canine breeds, now represents an important differential diagnosis (7, 8).

Most often, diagnostic imaging features of APSS, besides PH and abnormal, acquired vessel formation, are ascites and pancreatic oedema (1, 6, 7). Postprandial US facilitates the detection of these changes (6, 7).

In APSS, the main branches enlarge the left-colic pudendal vein, left gastric-cardiac oesophageal branches and phrenic-PV connections, which are not perfused or minimally perfused after birth (3, 4). The shunts could be divided into large (left splenogonadal, splenophrenic shunts) and small shunts (for example, oesophageal, gastrophrenic and colic varices), which could coexist (4).

Depending on anatomical localization, varices could be subdivided into the left gastric vein, gastrophrenic, omental, gallbladder, abdominal wall, duodenal, and colic (3, 4). In patients with PH, the left gastric vein is often dilated and tortuous, and it is sometimes accompanied by paraesophageal, oesophageal, and gastric varices (3). In Bertolini's research (3), all patients with PH had varices draining directly into the CVC or through its tributaries. However, gastric varices drained either into the cranial vena cava through the oesophageal and paraesophageal veins (gastroesophageal varices) or into the CVC through the left phrenic-abdominal vein (gastrophrenic varices).

The most common observed route in dogs is the left splenogonadal shunt and splenophrenic varices (31, 32, 39), with the splenophrenic pattern shared by both APSS and congenital portosystemic shunts (CPSS) (32). In particular, a dilatation of the left gonadal vein is considered to be a very sensitive and specific indicator of APSS and PH (1).

Cavo-Portal Collaterals, Porto-Portal Collaterals (Cavernous Transformation of the Portal Vein)

A similar appearance of opening pre-existing but nonfunctional shunt vessels may be observed in cavo-portal collaterals (4, 36). They may be observed in cases of chronic obstruction of the CVC (4).

In the chronic obliteration of the PV lumen due to thrombosis or extrinsic compression, PH, portal cavernous transformation, and APSS could develop (4, 36).

In the Specchi et al. study, PV obstruction and cavernous transformation of the PV have been described as a single condition or concomitant with APSS in small animals (37). Numerous newly formed portal vessels are observed, which could exist around and in thrombus, as well as long bypassing collaterals in the porta hepatis and around the gallbladder and bile ducts (4, 36, 37). These vessels are connecting the preobstructed extrahepatic PV tract to the postobstructed tract or main intrahepatic PV branches (36, 37). These collateral vessels represented portoportal connections with presumed hepatopetal flow (37).

There are two types of these vessels:

- The short collaterals – newly formed vessels (neovascularisation) around/inside the thrombotic PV (12, 34, 37).
- Long collateral pathways – pre-existing vascular structures (pancreaticoduodenal vein, gallbladder, cystic duct, and choledochal veins and the splenic vein) that are used to bypass the site of PV obstruction. In this group, collaterals arise from the pancreaticoduodenal vein, gallbladder, cystic duct, and choledochal veins, as well as from the splenic vein (12, 37).

When portal obstruction is limited to the portal trunk, vessels forming the cavernous transformation may bypass the obstructed site, connecting the intrahepatic portal branch to maintain normal portal flow (37). In more severe PV obstruction, cavernous transformation is not sufficient to relieve PH and portosystemic collateral circulation forms (37). On CT examination, a normal PV is usually not visible (37).

Diagnostic imaging methods of diagnosis

Diagnosis of PSS is made on the basis of history and physical examination, laboratory findings, and diagnostic imaging methods, which are essential because they allow for visualisation of the aberrant vein and rule out other conditions that can cause similar signs (19, 39). In 2020, Devrient et al. reported results suggesting that dogs with clinical signs compatible with PSS and elevated fasting ammonia are likely to have PSS, whereas normal paired serum bile acids make PSS unlikely (8). It shows that it is best to combine ultrasound diagnostics with simultaneous analysis of laboratory tests to diagnose PSS (8).

Ultrasonography (US) is the most commonly used technique for detecting PSS in dogs and cats due to its

availability and low costs, but it is a highly operator-dependent technique (19). There are also some limitations of US, as the presence of gas in the gastrointestinal tract may preclude complete evaluation of some abdominal structures, and examination may be more difficult in deep-chested dogs.

On the other hand, US is the only diagnostic modality that enables Doppler sonography and allows characterization of blood flow in affected vessels, such as portal flow pattern, direction, and velocity (1).

The Doppler shift calculation and correct blood velocity measurement require an angle of insonation (the angle between the ultrasound beam and the target) of around 60° or lower; otherwise, this method has low accuracy (7, 8). The axial orientation of the PV in dogs and cats can sometimes make measurement difficult (7, 8).

In intrahepatic portosystemic shunts (IHPSS), there is an abnormal vascular communication between the portal venous system and the systemic circulation, typically through a hepatic vein or directly into the caudal vena cava. To improve the method's effectiveness, some protocols for systematic ultrasonographic evaluation of the portal system in dogs were proposed (39). Failure to locate a PSS does not rule out the possibility of its presence (7, 8, 39). In particular, EHPSS more commonly required additional examinations to confidently obtain a negative or positive diagnosis and to define shunt morphology (7, 8).

Reported sensitivities range from 47% to 95% and specificities from 57% to 100% for the ultrasonographic diagnosis of PSS, with higher accuracy reported in selected referral populations (7, 8).

Standard CTA and MR angiography primarily provide morphologic information and do not directly assess flow direction, although dynamic or perfusion-based techniques may offer indirect hemodynamic insights (14, 25, 39, 51).

These methods also require premedication or general anesthesia. It is a very useful method in surgical planning (7, 8).

CT angiography (CTA) has become the imaging technique of choice for diagnosing PSS in dogs and cats (7, 8). According to some authors, the sensitivity and specificity of CTA for the detection of PSS (96% and 89%, respectively) were significantly higher than those of abdominal US, and CTA was 5.5 times more likely to detect a shunt than abdominal US (18).

Nelson et al. recommend performing a CTA whenever available to reach a definitive diagnosis, especially if a CPSS is highly suspected and cannot be located by US, or if a complex or multiple CPSS is suspected (18, 19).

In recent years, CT examination has enabled the creation of three-dimensional models of IHPSS in dogs using vascular modeling engineering-based software, which can be utilized for preprocedural planning, depending on clinician preference (16).

Magnetic resonance angiography (MRA) has many advantages, similar to CTA (19). Seguin et al. (1999) reported sensitivity (80%) and specificity (100%) of MRA for the diagnosis of PSS, comparable to those of CTA, and differentiation of EHPSS from IHPSS was possible in the vast majority of dogs (83%) (35). However, this method is more complicated, slower and more expensive than CT (35).

In 2020, new MRI protocols using ferumoxytol in dogs, despite the gadolinium, were presented (43). This method provides vascular conspicuity comparable to that of CT in dogs with intrahepatic PSS (49).

There are numerous publications that use mesenteric portovenography as an imaging technique for detecting PSS in dogs and cats (1, 13, 19, 29, 39). It provides dynamic information about the flow through the aberrant vessel before and after occlusion and can better assess portal arborisation (30). On the other hand, this technique is invasive and time-consuming, and it requires mobile radiography or fluoroscopy. The above-mentioned factors and variations in the expected results lead to the limited use of this method in practice (1, 13, 19, 29, 39).

Similarly, although scintigraphy can detect portosystemic shunting, it cannot differentiate CPSS from APSS (39).

Taking into account all the advantages and disadvantages of the above methods, US combined with CT is most frequently used.

Diagnostic imaging findings

All possible findings indicative of extrahepatic PSS include microhepatia and reduced hepatic and PV branch visibility (22-84% of cases, with lower percentages in cats), as well as the presence of an anomalous vessel that can be observed from its origin to its termination (7, 8, 19). In Nelson's research, most shunts were the same or slightly larger in diameter than the aorta, and the cranial PV was smaller than that of the aorta, but in the literature, shunts of different sizes are described (27).

The PV diameter is reduced in the porta hepatis, and the PV/aorta ratio in dogs with EHPSS is also reduced (19). Strongly suggestive dogs and cats with a PV/Ao ratio of ≤ 0.65 have a decreased PV diameter, which means that there is a presence of an extrahepatic PSS or idiopathic noncirrhotic PH. Dogs and cats with PV/Ao and PV/CVC ratios of ≥ 0.8 and ≥ 0.75, respectively, did not have an extrahepatic PSS (7, 8). PV/Ao above 1.2 could indicate the enlargement of the PV and PH.

According to Weisse et al., based on the internal diameter of the PV at the level of the porta hepatis, in comparison to the internal diameter of the PV prior to shunt exit and based on the visibility of first, second and third order PV branches, the hepatic portal perfusion could be rated in CT (44, 45). A portal perfusion score is a 5-point scale where points are:

- Poor – no visible PV at the porta hepatis, absent first-order intrahepatic branches;
- Moderate-poor;
- Moderate – visibility of 50% internal diameter of the PV compared with the PV prior to shunt exit from

the portal system; first or second-order intrahepatic branches are visible;

- Moderate-good;
- Good – similar internal diameter of the PV compared with the PV prior to shunt exit from the portal system; second or third-order intrahepatic branches are visible.

In 2019, Amasha et al. proposed a CT protocol with a measurement of shunt fraction (percentage of total portal venous flow that is shunted) and hepatic perfusion (2). They discussed that dogs with EHPSS and higher shunt fraction values may have lower volumes of intrahepatic portal blood flow, and affected dogs may show clinical signs at earlier ages (2). They also stated that perfusion CT could be useful for distinguishing hemodynamic characteristics among different types of PSS in dogs (2).

If the place of shunt insertion is into the CVC, it is often enlarged cranially to it. The presence of a large vessel in the craniodorsal abdomen coursing along the aorta, with flow directed cranially, indicates an abnormally enlarged azygos vein and PSS azygos shunt (7).

In US, turbulence in blood flow at the CPSS insertion into the systemic venous system may be identified, as well as changes in mean portal flow velocity and portal flow variability (7, 19). Hepatofugal portal flow appears to be highly specific for portosystemic shunting in dogs (7). An increased portal flow velocity tends to occur when measured caudal to the PSS origin, and the opposite is expected if the measurement is obtained cranial to the PSS (7).

In PSS, it was also observed that an increase in hepatic arterial diameter is a regulatory mechanism in response to a reduction in portal blood flow, a phenomenon known as hepatic arterial buffer response (HABR) (7, 15). The increase in arterial perfusion in dogs with reduced portal blood flow has been previously documented (2, 50). This mechanism can buffer a 25-60% reduction in portal venous flow, minimizing the influence of afferent flow to the liver on hepatic clearance (14). The feature of increasing the hepatic artery diameter is considered helpful for assessing surgical outcomes and postoperative monitoring, as it was observed that the artery's size decreases after shunt attenuation, but at this moment a limited number of observations are available (14).

The enlargement of the aorta was also observed in some previous studies, without finding a possible cause, possibly related to an increase in systemic circulation resulting from portosystemic shunting (7).

Portal hypertension (PH) is not present in CPSS, and patients do not develop ascites unless they have profound hypoalbuminemia due to GI bleeding, hepatic insufficiency, or severe dietary protein restriction (19). These cases are very rare (1, 7, 8). In PH, the portal flow is below 10 cm/s in dogs and cats.

The other diagnostic imaging features of PSS include urolithiasis, which has a positive predictive value of 97% in suspected dogs and bilateral renomegaly (1, 7, 8, 26). In the case of renomegaly, several theories have been proposed to explain its aetiology (26). They include suspicion of homeostatic mechanisms to maintain hepatic blood flow, resulting in increased renal arterial blood flow and glomerular filtration rate, which may cause renomegaly, as well as the effect of changes in renal metabolic function or glomerulopathy as a complication of portosystemic shunting (25).

These calculi are mostly ammonium urate uroliths and are usually radiolucent on radiographic examination until they contain no additional calcium salts or struvite deposits (19). The best positive predictive value (100%) was when urolithiasis, microhepatia, and renomegaly were found together (1, 7, 8).

The latest research also reported a higher risk of GI tract ulceration due to impaired hepatic inactivation of gastrin and/or increased gastrin production stimulated by elevated serum bile acid concentrations, abnormal blood flow, hypoprostaglandinemia, poor mucosal integrity, and abnormal mucus production in dogs with hepatic disease (11). This complication negatively influences the postoperative prognosis of patients with PSS (11).

Conclusion

Ultrasound and computed tomography are used to identify and evaluate PV abnormalities in dogs, and other diagnostic methods are used less frequently. Knowledge of vessels and the morphology of additional abnormalities, as well as possible complications, is essential for prognosis and further treatment.

References

1. *Adam F. H., German A. J., McConell J. F., Trehy M. R., Whitley N., Collings A., Watson P. J., Burrow R. D.*: Clinical and clinicopathologic abnormalities in young dogs with acquired and congenital portosystemic shunts: 93 cases (2003-2008). JAVMA 2012, 241 (6), 760-765.
2. *Amasha T., Ishikawa Ch., Ishigaki K., Horikirizono H., Iizuka K., Nagumo T., Tamura K., Seki M., Edamura K., Asano K.*: Computed tomographic-derived measurements of shunt fraction and hepatic perfusion in dogs with a single extrahepatic portosystemic shunt in a clinical setting. JAVMA 2019, 255, 7, 821-827.
3. *Bertolini G.*: Acquired portal collateral circulation in the dog and cat. Vet. Radiol. Ultrasound 2010, 51 (1), 25-33.
4. *Bertolini G.*: Anomalies of the portal venous system in dogs and cats as seen on multidetector-row computed tomography: an overview and systemization proposal. Vet. Sci. 2019, 6 (10), 1-17.
5. *Boothe H. W., Howe L. M., Edwards J. F., Slater M. R.*: Multiple extrahepatic portosystemic shunts in dogs: 30 cases (1981-1993). Am. Vet. Med. Assoc. 1996, 208, 1849-1854.
6. *D'Anjou M. A., Huneault L.*: Imaging Diagnosis – complex intrahepatic portosystemic shunt in a dog. Vet. Radiol. Ultrasound 2008, 49, 51-55.
7. *D'Anjou M. A., Penninck D., Cornejo L., Pibarot P.*: Ultrasonographic diagnosis of portosystemic shunting in dogs and cats. Vet. Radiol. Ultrasound 2004, 45, 424-437.
8. *Devriendt N., Serrano G., Stock E., Paepe D., de Rooster H.*: Diagnostic value of liver function tests and ultrasonography in dogs with suspected congenital portosystemic shunts. Vet. Record 2022, e1381, 1-10.
9. *Ferrell E. A., Graham J. P., Hanel R., Randell S., Farese J. P., Castelman W. L.*: Simultaneous congenital and acquired extrahepatic portosystemic shunts in two dogs. Vet. Radiol. Ultrasound 2003, 44 (1), 38-42.
10. *Fischetti A. J., Kovak J.*: Imaging diagnosis: azygous continuation of the caudal vena cava with and without portocaval shunting. Vet. Radiol. Ultrasound 2008, 49 (6), 573-576.
11. *Frymus J., Trębacz P., Galanty M.*: Gastrointestinal ulceration in dog with portosystemic shunt. Med. Weter. 2023, 79 (6), 275-278.

12. *Gaetano de A. M., Lafortune M., Patriquin H., Franco de A., Aubin B., Paradis K.*: Cavernous transformation of the portal vein: patterns of intrahepatic and splanchnic collateral circulation detected with Doppler sonography. Am. J. Roentgenol. 1995, 165, 1151-1155.
13. *Holt D. E., Schelling C. G., Saunders H. M., Orsher R. J.*: Correlation of ultrasonographic findings with surgical, portographic, and necropsy findings in dogs and cats with portosystemic shunts: 63 cases (1987-1993). Am. Vet. Med. Assoc. 1995, 207, 1190-1193.
14. *Hong S., Kim S., Yoon J., Choi J.*: Time-resolved imaging of contrast kinetics magnetic resonance angiography for the assessment of vascular characteristics in intracranial and extracranial tumors in veterinary patients. Front Vet. Sci. 2025 May 21; 12:1579582, doi: 10.3389/fvets.2025.1579582.
15. *Humphreys W. J. E., Sompig J. C., Maddox T. W.*: Enlargement of the hepatic artery is present in dogs with a congenital extra hepatic portosystemic shunt and is independent of shunt insertion into the systemic circulation. Vet. Radiol. Ultrasound 2024, 65, 149-156.
16. *Jackson E., Markovic L. E., Perlini M.*: Comparison of two-dimensional imaging to three-dimensional modeling of intrahepatic portosystemic shunts using computed tomography angiography. Vet. Radiol. Ultrasound 2024, 65, 130-137.
17. *Kantrowitz B., Nyland T. G., Fischer A.*: Estimation of portal blood flow using duplex real-time and pulsed Doppler ultrasound. Vet. Radiol. 1989, 30, 222-226.
18. *Kim S. E., Giglio R. F., Reese D. J., Reese S. L., Bacon N. J., Ellison G. W.*: Comparison of computed tomographic angiography and ultrasonography for the detection and characterization of portosystemic shunts in dogs. Vet. Radiol. Ultrasound 2013, 54, 569-574.
19. *Konstantinidis A. O., Patsikas M. N., Papazoglou L. G., Adamama-Moraitou K. K.*: Congenital portosystemic shunts in dogs and cats: classification, pathophysiology, clinical presentation and diagnosis. Vet. Sci. 2023, 10, 160.
20. *Krotscheck U., Adin C. A., Hunt G. B., Kyles A. E., Erb H. N.*: Epidemiologic factors associated with the anatomic location of intrahepatic portosystemic shunts in dogs. Vet. Surg. 2007, 36 (1), 31-36.
21. *Lamb C. R., Mahoney P. N.*: Comparison of three methods for calculating portal blood flow velocity in dogs using duplex-Doppler ultrasonography. Vet. Radiol. Ultrasound 1994, 35, 190-194.
22. *Lowe L. H., Marchant T. C., Rivard D. C., Scherbel A. J.*: Vascular malformations: classification and terminology the radiologist need to know. Seminars in Roentgenology 2012, 47 (2), 106-117.
23. *Łojszczyk A., Kimicka A.*: Atypical Benign angioproliferatove lesion in a cat: Diagnostic Imaging pitfalls. Med. Weter. 2020, 76 (12), 723-726.
24. *MacEwan I., Thompson D.*: Intramural esophageal location of a portosystemic shunt in a dog with two congenital portosystemic shunts on CT angiography. Vet. Radiol. Ultrasound 2023, 64, E45-E49.
25. *Meyer H., Rothuizen J., Ubbink G., van den Ingh T.*: Increasing incidence of hereditary intrahepatic portosystemic shunts in Irish wolfhounds in The Netherlands (1984 to 1992). Vet. Rec. 1995, 136 (1), 13-16.
26. *Murakami M., Nishi R., Lenoir K.*: Computed tomographic measures of renomegaly vary among dogs with different types of congenital portosystemic shunts. Vet. Radiol. Ultrasound 2023, 64 (6), 1025-1032.
27. *Nelson N. C., Nelson L. L.*: Anatomy of extrahepatic portosystemic shunts in dogs as determined by computed tomography angiography. Vet. Radiol. Ultrasound 2011, 5, 498-506.
28. *Nyland T. G., Fisher P. E.*: Evaluation of experimentally induced canine hepatic cirrhosis using duplex Doppler ultrasound. Vet. Radiol. 1990, 31, 189-194.
29. *Parry A. T., White R. N.*: Comparison of computed tomographic angiography and intraoperative mesenteric portovenography for extrahepatic portosystemic shunts. J. Small Anim. Pract. 2017, 58, 49-55, doi: 10.1111/jsap.12596.
30. *Plested M. J., Zwingennberger A. L., Brockamn D. J., Hecht S., Secrest S., Cupl W. T. N., Drees R.*: Canine intrahepatic portosystemic shunt insertion into the systemic circulation is commonly through hepatic veins as assessed with CT angiography. Vet. Radiol. Ultrasound 2020, 61, 519-530.
31. *Ricciardi M.*: Splenophrenic jortosystemic shunt in dogs with and without portal hypertension: can acquired and congenital porto-caval connections coexist? Open Vet. J. 2016, 6 (3), 185-193.
32. *Ricciardi M., Martino R., Assad E. A.*: Imaging diagnosis – celiacomesenteric trunk and portal vein hypoplasia in a pit bull terier. Vet. Radiol. Ultrasound 2014, 55 (2), 190-194.
33. *Sakamoto Y., Sakai M., Watari T.*: Portal vein/aorta ratio in dogs with acquired portosystemic collaterals. J. Vet. Intern. Med. 2017, 31, 1382-1387.
34. *Seeger M., Günther R., Hinrichsen H., Both M., Helwig U., Arlt A., Stelck B., Bräsen J. H., Sipos B., Schafmayer C., Dieter B. F., Bröring C., Schreiber S., Hampe J.*: Chronic portal vein thrombosis: transcapsular hepatic collateral vessels and communicating ectopic varices. Radiology 2010, 257, 568-578.
35. *Seguin B., Tobias K. M., Gavin P. R., Tucker R. L.*: Use of magnetic resonance angiography for diagnosis of portosystemic shunts in dogs. Vet. Radiol. Ultrasound. 1999, 40, 251-258.
36. *Seller S., Weisse Ch., Fichetti A. J.*: Intrahepatic venous collaterals in dogs with congenital intrahepatic portosystemic shunts are associated with focal shunt or hepatic vein narrowing. Vet. Radiol. Ultrasound 2022, 63, 64-72.
37. *Specchi S., Per P., Ledda G., Lustgarten M., Thrall D., Bertolini G.*: Computed tomographic and ultrasonographic characteristics of cavernous transformation of the obstructed portal vein in small animals. Vet. Radiol. Ultrasound 2015, 56 (5), 511-519.
38. *Solari F. P., Case J. B., Vilaplana Grosso F. R.*: Outcome of dogs diagnosed with concurrent intrahepatic portosystemic shunts and vertebral lesions: six cases (2016-2022). J. Small Anim. Pract. 2024, 65 (7), 589-595.
39. *Szatmári V., Rothuizen J., Voorhout G.*: Standard planes for ultrasonographic examination of the portal system in dogs. J. Am. Vet. Med. Assoc. 2004, 224, 713-716.
40. *Tobias K. M., Rohrbach B. W.*: Association of breed with the diagnosis of congenital portosystemic shunts in dogs. JAVMA 2003, 223 (11), 1636-1639.
41. *Walsh N. D., Porter I. R., Miller A. V., Fischetti A. J., Chen S. H., Scrivani P. V.*: Canine intrahepatic portosystemic shunts: intelobar and intralobar classifications. Vet. Radiol. Ultrasound 2023, 64, 646-660.
42. *Walton-Clark M., Travail V., Sparks T., Eiras-Diaz A., Davenport A., Holmes A., Kent A., Prior C., Stiwell Breakspear C., Dye C., Good C., Motta C., Vallas Sanchez F., Pilati F., McLauchlan G., Ruiz G., Bras G., Scott J., Clarke K., Peak K., Goonan L., Ots M., Rossell Garcia M., Lau N., Mansbridge N., Garcia Dominguez P., Conway S., Keyte S., Chapman T., Conley T., Black V., Coates V., DiBella A.*: Retrospective analysis of the incidence and clinicopathological findings associated with ammonium urateurolithiasis in dogs with congenital portosystemic shunts: 363 cases (2010-2023). J. Small Anim. Pract. 2025, 66, 248-257.
43. *Wei B., Li L., Chen X., Guo W., Wang W., Zhao Y., Dong Y., Yao D.*: Concurrence of circumcaval ureter and congenital extrahepatic portosystemic shunt in a cat. Vet. Radiol. Ultrasound 2025, 66:e70100.
44. *Weisse Ch., Asano K., Ishigaki K., Lipscomb V., Llanos C., Zwingenberger A. L., Caroll K. A., Grosso F., Stock E., Buote N., Aly A., Murgia D., Arai S., Linden Al., Gordon J., Manassero M., Schwarz T., Wallace M., Graham J., Hardie R., Chang Y., Robbins M., Bismuth C., Karnia J., Sterman A., Saunder A., Mintinaro V., Guarnera I., McLauchlan G., Cerna P., Maurin M., Aisa J., An A.*: Anatomical classification of feline congenital extrahepatic portosystemic shunts based on CT angiography: A SVSTS and VIRIES multi-institutional study in 231 cats. Vet. Radiol. Ultrasound 2024, 65, 359-368.
45. *Weisse Ch., Fox-Alvarez W. Al., Vilaplana Grosso F. R., Asano K., Ishigaki K., Zwingenberger A. L., Caroll K. A., Scharf V. F., Lipscomb V., Wallace M. L., Aly A., Biscoe B., Davidson J. R., Arai S., Amato N. S., Ryan S. D., Woods S.*: Anatomical classification of canine congenital extra hepatic portosystemic shunts based on CT angiography: A SVST and VIRIES multi-institutional study in 1082 dogs. Vet. Radiol. Ultrasound 2024, 65, 702-712.
46. *White R. N., McDonald N. J., Burton C. A.*: Use of intraoperative mesenteric portovenography in congenital portosystemic shunt surgery. Vet. Radiol. Ultrasound 2003, 44 (50), 514-521.
47. *White R. N., Parry A. T., Shales C.*: Implications of shunt morphology for the surgical management of extrahepatic portosystemic shunts. Austr. Vet. J. 2019, 96, 11, 433-441.
48. *White R. N, Warren-Smith C., Shales C., Parry A. T.*: Classification of portosystemic shunts entering the caudal vena cava at omental foramen in dogs. JSAP 2020, 61, 659-670.
49. *Wilson S., Culp W. T. N., Wisner E. R., Cissell D. D., Finn J. P., Zwingenberger A. L.*: Ferumoxytol-enhanced magnetic resonance angiography provides comparable vascular conspicuity to CT angiography in dogs with intrahepatic portosystemic shunts. Vet. Radiol. Ultrasound 2021, 62, 463-470.
50. *Zwingenberger A. L., Schwarz T., Sauders M.*: Helical computed tomographic angiography of canine portosystemic shunts. Vet. Radiol. Ultrasound 2005, 46 (1), 27-32.
51. *Zwingenberger A. L., Shofer F. S.*: Dynamic computed tomographic quantitation of hepatic perfusion in dogs with and without portal vascular anomalies. Am. J. Vet. Res. 2007, 68, 970-974.
52. *Zwingenberger A. L., Spriet M., Hunt G. B.*: Imaging diagnosis – portal vein aplasia and interruption of the caudal vena in three dogs. Vet. Radiol. Ultrasound 2011, 52 (4), 2011, 444-447.

Corresponding author: Dr n. wet. Anna Łojszczyk, Głęboka 30, 20-612 Lublin, Poland; e-mail: