1 *Review*

Anomalies of the portal venous system in dogs and cats as seen on multidetector-row ct: an overview and systematization proposal

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8 **Abstract：This** article offers an overview of congenital and acquired vascular anomalies involving
9 the portal venous system in dogs and cats, as determined by multidetector-row computed
10 tomography angiography. Congenital absence of the portal vein, portal vein hypoplasia, portal vein
11 thrombosis and portal collaterals are described. Portal collaterals are further discussed as high- and
12 low-flow connections, and categorized in hepatic arterioportal malformation, arteriovenous fistula,
13 end-to-side and side-to-side congenital portosystemic shunts, acquired portosystemic shunts,
14 cavoportal and porto-portal collaterals. Knowledge of different portal system anomalies helps
15 understand the underlying physiopathological mechanism and is essential for surgical and
16 interventional approaches.

17 **Keywords:** Portal system; Portal vein; Portosystemic shunt; Portal hypertension; Computed
18 tomography

19

Introduction

21   The portal venous system is essential for the maintenance of the liver mass and function in
22 mammals. The portal system collects blood from major abdominal organs (i.e. gastrointestinal tract,
23 pancreas, spleen) delivering nutrients, bacteria, and toxins from the intestine to the liver. In addition,
24 the portal blood carries approximately from one-half to two-thirds of the oxygen supply to the liver
25 and specific hepatotrophic factors.[1,2] The portal blood is detoxified by the hepatocytes and then
26 delivered into the systemic circulation via the hepatic veins and caudal vena cava .[3] Gross anatomic
27 connections between the portal system and systemic venous circulation, at any level, or other portal
28 system anomalies can have serious clinical consequences. [4-8] Anomalies of the portal vein and portal
29 system in small animals have been increasingly detected in recent years with the growing availability
30 of advanced imaging techniques in veterinary practice. [9-13] A broad spectrum of congenital and
31 acquired disorders may affect the portal venous system in dogs and cats. Congenital disorders of the
32 portal system reflect a variety of embryonic or fetal disturbances, which may be isolated or combined
33 in complex vascular patterns, and may be cause of acquired vascular disorders. For example,
34 congenital arterioportal communications are associated with acquired portal collaterals that form in
35 response to the portal hypertension.[10,14] (Tab.1). Knowledge of the normal anatomy and congenital
36 and acquired patterns and their consequences is of great importance for clinical decision-making for
37 liver surgery and for planning interventional procedures such as transvenous portosystemic shunts
38 or transarterial arterioportal fistulas coil embolization . [15,16] First line imaging evaluation of the portal
39 venous system in small animals is usually performed with color Doppler ultrasonography. Invasive
40 techniques (e.g splenoportography) used to obtain more detailed morphological information, are
41 nowadays replaced by computed tomography (CT) or magnetic resonance imaging (MRI)
42 angiography.[17-21]

Tab. 1. Schematization of the anomalies of the portal vein (left) and anomalous connections involving the portal venous system. Arrows indicate possible relationships between various congenital and acquired anomalies. CAPV, congenital absence of portal vein; PVH, portal vein hypoplasia; PPVh, primary portal vein hypoplasia; SPVH, secondary portal vein hypoplasia; PVA, portal vein aneurysm; PVT, portal vein thrombosis; HAVM, hepatic arteriovenous malformation; APF, arterioportal fistula; CPSS; congenital portosystemic shunt; IHPSS, intrahepatic portosystemic shunt; EHPSS, extrahepatic portosystemic shunt; APSS, acquired portosystemic shunt.
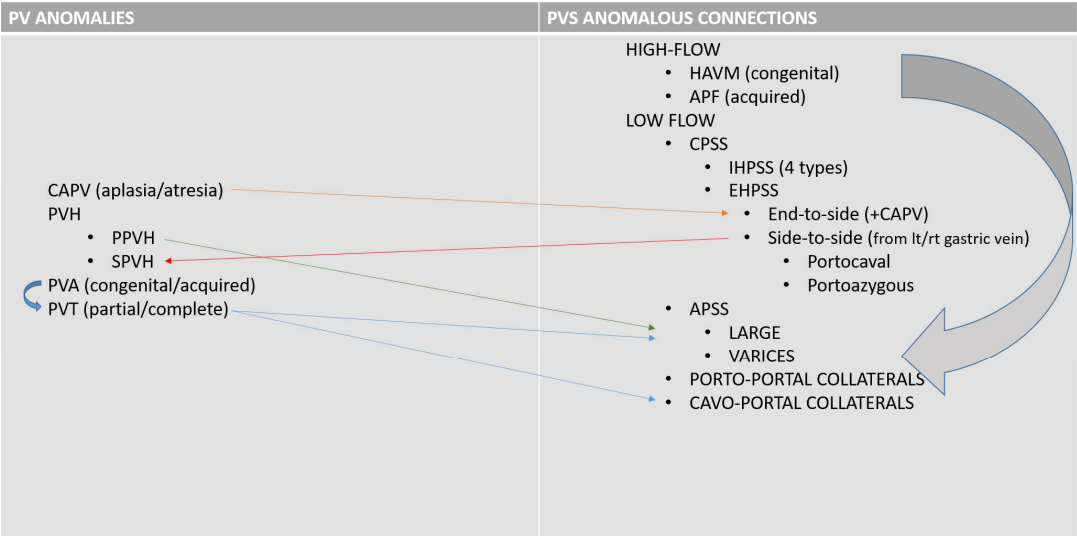
Multidetector-row computed tomography (MDCT) angiography is now widely considered to be the method of choice for the diagnosis and monitoring of portal vascular anomalies in veterinary practice. [22-24] MDCT provides excellent visualization of complex vascular anomalies and a comprehensive overview of the entire portal system and allows simultaneous evaluation of other vascular and non-vascular structures of the abdomen.

In this article, we present an overview of normal anatomy of the portal system, as seen by MDCT angiography and propose a systematization of congenital and acquired pathological conditions in dogs and cats.

Brief Anatomy Background

The portal system forms by the confluence of cranial and caudal mesenteric veins, which drain most of the intestinal tract. From the left side the splenic vein, which accompanies the corresponding artery and receives the left gastric vein, contributes to the portal vein formation. From the right side, the portal vein receives the gastroduodenal vein, right gastric, and gastroepiploic veins before entering the liver parenchyma. At the hepatic porta, the portal vein divides into right and left branches (Fig. 1).[25] At this level, the portal vein pattern is almost constant, while the number of the portal branches entering individual liver lobes can vary among subjects. [26,27] In general, the right branch is a short venous trunk, which supplies the right portion of the liver. One or two dorsolateral primary branches supply for the caudate process of the caudate lobe and one or three ventrolateral branches ran towards the right lateral lobe of the liver. The left branch of the portal vein provides several primary branches supplying the central and left portions of the liver, comprising the right medial, quadrate, left medial, and left lateral lobes. In most circumstances, the left portal branch supplies also the dorsal, right lateral liver lobe. One to three small branches for the papillary process of the caudate lobe arise directly from the left branch. The feline portal vein divides into left, central, and right portal branches providing intrahepatic subdivisions to the liver lobes. [28-30] At all sizes and subdivisions, branches of the hepatic artery, portal vein, and bile ducts pass in close proximity to one another. Interlobular branches of the hepatic artery and portal vein form hepatic sinusoids, which collect detoxified blood into central veins and finally into the vena cava system.
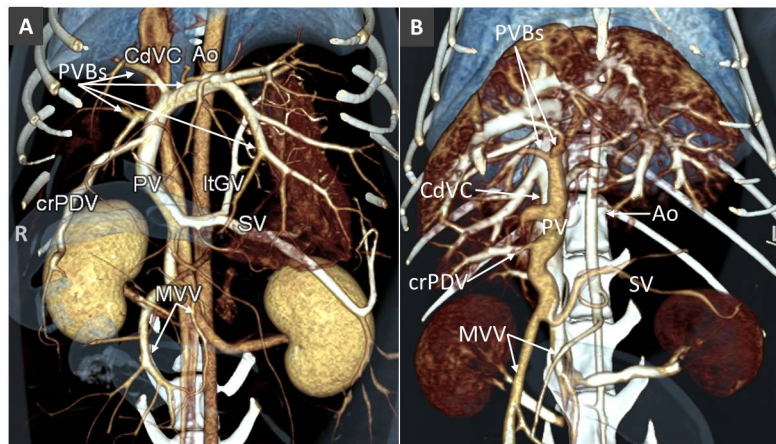
Fig. 1. Volume rendered images (ventral views) of normal portal venous system in a dog (A) and a cat (B). Ao, aorta; CdVC, caudal vena cava; PVBs, portal vein branches; PV, portal vein; ltGV, left gastric vein; crPDV cranial pancreatic duodenal vein; SV, splenic vein; MVV, mesenteric veins.

PORTAL SYSTEM VENOUS ANOMALIES (PVSA)

1. .DISORDER OF THE PORTAL VEIN SYSTEM

Congenital absence of the portal vein (aplasia and atresia)

Congenital absence of portal vein (CAPV) is a rare condition in which the portal blood bypasses the liver and a splenomesenteric shunt to the systemic circulation is present.[31,32] The CAPV is attributed to excessive involution of the periduodenal vitelline veins or failure of the vitelline veins to establish anastomosis with hepatic sinusoids, which leads to partial (of the extrahepatic portal vein) or complete (involving also the intrahepatic portal component) absence of the portal system (aplasia or agenesis). As a result, the umbilical flow and enterohepatic circulation are disturbed and the portal as well as placental venous flow is shunted systemically. There is close relationship between the vitelline veins and the complex development of the systemic veins likely explaining the occurrence of extrahepatic portosystemic shunt in case of CAPVS. [33,34] In fact, cases of CAPVS in dogs are always described in association to the portal insertion into the caudal vena cava and total diversion of portal blood into the systemic circulation (end-to-side portosystemic shunt). [35,36]

Imaging alone cannot distinguish between complete or incomplete forms of portal vein aplasia. The definitive diagnosis of complete agenesis of the portal system requires additional histological analysis of the hepatic parenchyma that demonstrates the absence of hepatic portal venules within the portal triad.[37]

In dogs, CAPV has been described in association to other developmental anomalies including situs inversus, congenital heart diseases, vena cava anomalies, and polysplenia (Fig. 2). [33,38]
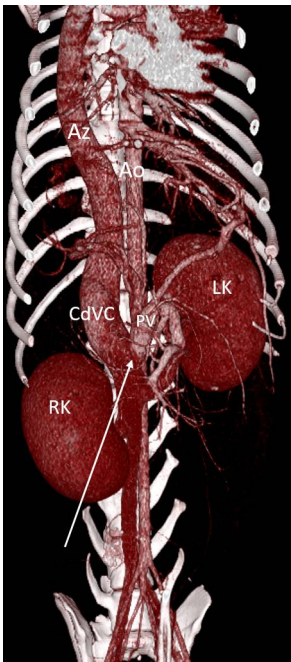
102

103   **Fig.** 2. Volume rendered image (ventral view) of a dog with extrahepatic CAPV, portal insertion in
104   the renal segment of the CdVC (arrow). There is simultanous azygous continuation of the CdVC due
105   to congenital absence of renal and prehepatic CdVC segments. Moreover, the dog showed situs
106   inversus abdominalis (note the left-sided mesenteric and portal veins, the caudal right kidney and
107   cranial left one).

108   Reduced portal blood flow in the portal vein due to flow diversion through a several types of
109   congenital extrahepatic portosystemic shunt (CPSS) may simulate the absence of the portal vein. An
110   adequate CT portal venous vascular phase may reveal a hypoplastic, hypoperfused portal vein
111   (portal vein atresia) and hepatic portal branches, which makes the patient a candidate for surgical
112   intervention of the portosystemic shunt (Fig. 3). On contrary, in case of portal vein aplasia, surgical
113   attenuation of the portosystemic connection will result in fatal acute portal hypertension.[6]
114   Histopathology data may be added to CT data for confirmation of presence or absence of the portal
115   venules within the portal triad. However, smaller or absent portal tract within the liver can be
116   detected in liver biopsy samples of both aplasia and hypoplasia portal vein conditions .[39]
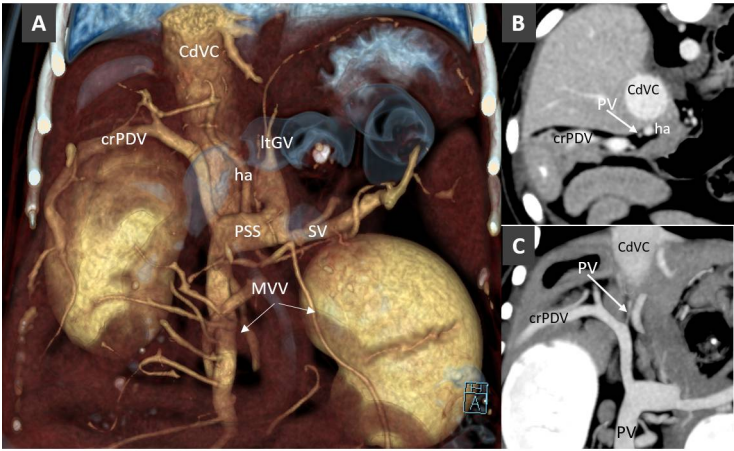

117

118   **Fig.** 3. A. Volume rendered image (ventral view) in a Mongrel dog with extrahepatic portosystemic
119   shunt (PSS). The portal vein is not visible. MVV, mesenteric veins; PSS, portosystemic shunt; SV,
120   splenic vein; ha, hepatic artery; ltGV, left gastric vein; crPDV, cranial pancreaticoduodenal vein. B.
121   Transverse view at the porta hepatis of the same dog. An hypoplastic PV is barely visible. C. Dorsal
122   thin-MIP image in the same dog, showing the thin, hypoperfuse portal vein (portal atresia).

123   *Portal vein hypoplasia*

124   Portal vein hypoplasia (PVH) refers to a disorder in which microscopic portal veins within the
125 liver are underdeveloped. PVH can be morphological (primary portal vein hypoplasia [PPVH]) or
126 functional (secondary PVH or SPVH) due to a reduction of portal blood flow toward the liver.[14,37]
127 These two conditions (morphological or functional) are not distinguishable either by radiology or
128 histopathology alone. Integration of imaging and histological information is thus mandatory to the
129 diagnosis of PVH and distinction of morphological or functional conditions. In fact, macroscopic
130 disorders causing chronic hypoperfusion of the liver, such as portosystemic shunting, arterioportal
131 fistula (APF), and portal vein thrombosis (PVT), can lead to the same hepatic histological features as
132 PPVH and should be ruled out with imaging.[40-42]

133   *Portal vein aneurysm*

134   Portal vein aneurysm (PVA) has a reported prevalence of 0.49% in dogs.[43] PVA can be congenital
135 or acquired. Proposed mechanisms for PVA are an inherent weakness of the vessel wall or incomplete
136 regression of the distal part of the vitelline vein. Larger, male dogs are more frequently affected, and
137 the boxer breed seems to be predisposed to PVA development. On MDCT images, PVA appears as
138 saccular or fusiform dilatation of the portal vein or its branches within the liver. Extrahepatic PVAs
139 are generally located at the insertion of the gastroduodenal vein into the portal vein. Intrahepatic
140 PVAs occur at bifurcations (Fig. 4). Clinical features of PVAs are related to their size and possible
141 complications, such as thrombosis, rupture of the aneurysm, and portal hypertension. Acquired
142 portal collaterals may coexist with PVA. [43,44] Aneurysm of the mesenteric veins can occur in dogs,
143 alone or in combination with PVA. Pseudoaneurysmal dilatation of portal branches is also detected
144 in cases of intrahepatic CPSS and hepatic arterioportal malformation.[45,46]
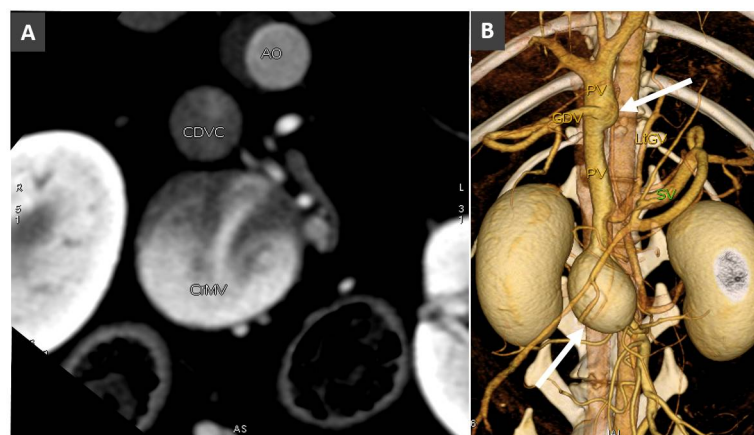

145

146 **Fig.** 4. A. Transverse view in a dog with large saccular aneurysm of the caudal mesenteric vein (CrMV)
147   at the confluence with the splenic vein (not visible here). CDVC, caudal vena cava; Ao, aorta. B.
148   Volume rendered image (ventral view) of the same dog, showing the large saccular aneurysm of the
149   cranial mesenteric vein and a fusiform aneurysmal dilatation of the prehepatic portal vein (arrows).

150   *Portal vein thrombosis*

151   Portal vein thrombosis (PVT) refers to the partial or total luminal obstruction of the portal vein,
152 with or without extension to its portal branches within the liver, the splenic vein, or the mesenteric
153 vein.[47]
154   Local causes of PVT, such as PVA and vascular invasion from hepatic or pancreatic neoplasia,
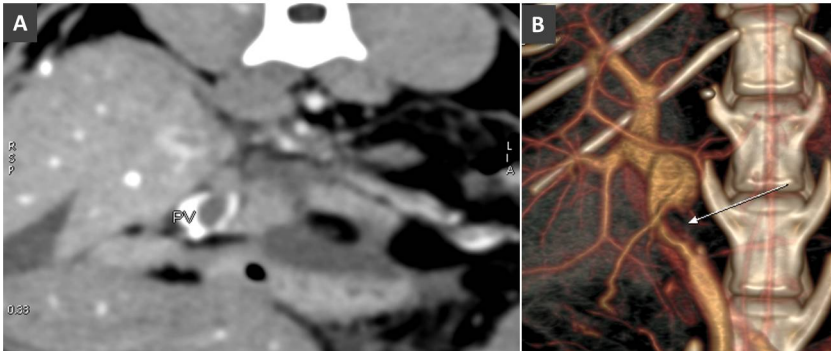155 are easily identified by MDCT. [43] (Fig. 5,6).

Fig.5. A. Transverse image of the liver at the porta hepatis in a cat with pancreatitis and PV thrombosis (arrow). B. Volume rendered image (ventral view) in the same cat. Note the large filling defect due to the thrombosis (arrow) in the prehepatic PV.
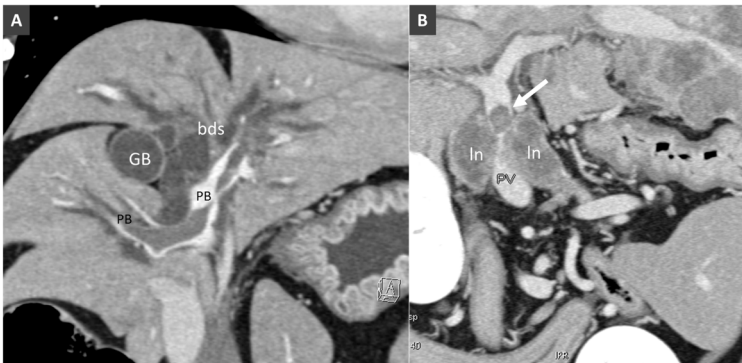

Fig. 6. a. Dorsal MPR view of the liver in a dog with suppurative hepatitis. Note the large filling defect in the portal branches (PB) within the liver. Biliary ducts (bds) are enlarged. GB, gallbladder.

PVT can lead to partial or complete obstruction of the portal lumen. Both, partial and complete PVT, may show peripheral enhancement in post-contrast series.[23,43,48] Partial and complete obstruction should be distinguished, because they can have different clinical consequences. In cases of partial occlusion, some contrast medium passes around the thrombus. In cases of complete obstruction, enhancement of the peripheral rim of the thrombus is likely due to dilatation of the vasa vasorum attempting to recanalize the vessel. [23]

Indirect signs of PVT in chronic obstruction are porto-portal collaterals (see below), arterioportal shunts, and APSS.[10,48] Because of portal vein obstruction, compensatory mechanisms are immediately activated to re-establish the portal flow to the liver, and small arterial–portal connections may form within the liver. A second mechanism is the formation of portal venous collaterals. In cases of complete extrahepatic portal vein obstruction, characterized by high pressure in the splanchnic circulation and normal pressure in the hepatic sinusoids, multiple collateral hepatopetal vessels form around the thrombotic segment (see below).

2. .ANOMALOUS VASCULAR CONNECTIONS OF THE PORTAL VEIN SYSTEM

The anomalous vascular connections of the portal system can be classified broadly as high-flow and low-flow portal connections.

High-flow anomalous portal connections

High-flow portal connections are based upon the presence of an arterial component. These conditions have been rarely described in dogs and cats and different terminology has been used interchangeably causing confusion. High-flow anomalous connections involving the portal venous system include congenital hepatic arteriovenous malformations (HAVMs) and congenital or acquired arterioportal fistulas (APFs) (Fig. 7, 8). HAVMs refers to a congenital disorder in which a plethora of

185   dysplastic arteries drain or shunt directly into arterialized portal vein branches, creating a vascular
186   nidus (arterialization of the portal system).[45,49,50] Sites of communications between the arterial and
187   portal vessels are often difficult or impossible to determine with non-selective CT angiography.[51,52]
188   Recently, HAVMs have been classified as right divisional or left divisional, and the left divisional
189   were subclassified as left medial and left lateral, depending on the site of the efferent portal vein
190   drainage (Specchi et al., 2018). Although congenital, these anomalies may be clinically silent until
191   hepatoencephalopathy and portal hypertension develop. Imaging of these patients shows direct and
192   indirect signs (ascites and acquired portal collateral circulation). HAVMs can be treated by
193   transcatheter embolization alone or in combination with surgery, but they may be difficult to treat
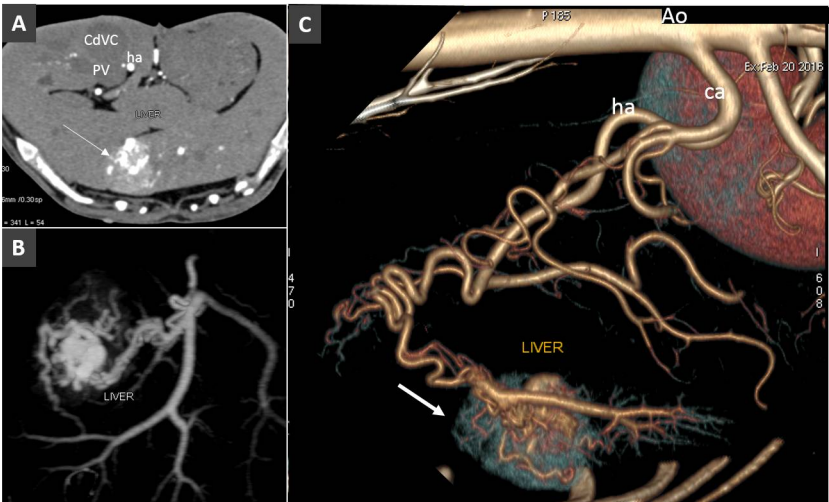194   definitively.[51]

195
196   **Fig.** 7. A. Acquired arterioportal fistula in a dog. A. Transverse view of the liver obtained during early
197   arterial phase. Note the large hypervascular hepatic lesions (arrow). B. Dorsal MIP showing the
198   arterioportal communication. C. Left lateral view of volume rendered image of the same volume. The
199   arrow indicates the focal early enhancement of the liver parenchyma due to the arterioportal
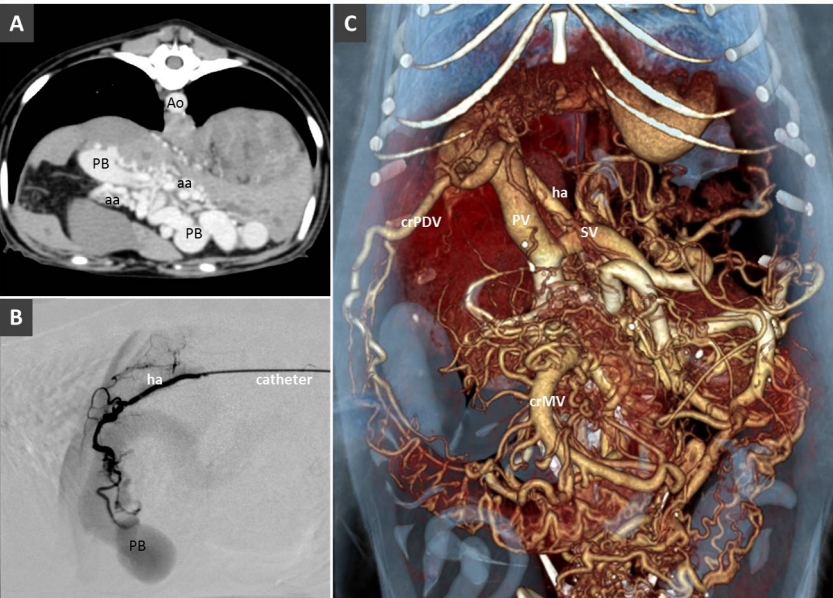200   communication.

201
202   **Fig.** 8. A. HAVM in a young dog with portal hypertension and multiple acquired portal collaterals.
203   Ao, aorta; PB, portal branches; aa, branches of the hepatic artery. B. Angiogram before selective
204   transarterial coil embolization through hepatic artery (ha) in the same patient. C. Volume rendered

205 image showing the portal venous system dilatation and multiple portal collaterals. PV, portal vein;
206 ha, hepatic artery; SV, splenic vein; crPDV, cranial pancreaticoduodenal vein.

207 A single APF is a congenital or acquired, communication between a high-pressure hepatic
208 arterial branch and a low-pressure portal branch outside or inside the liver.[53] Penetrating abdominal
209 trauma, including liver biopsy, and neoplasia are possible causes of APFs that are generally single
210 and thus easier to treat. Signs of pre-sinusoidal portal hypertension may ensue, such as ascites and
211 acquired portosystemic shunts.[14] Finally, in several hepatic pathological conditions, small or
212 microscopic acquired hepatic peripheral APFs can be identified or suspected in multiphase, thin
213 section MDCT examination of the liver. Their presence can cause areas of early parenchymal
214 opacification (hepatic perfusion disorders) enhancing in late arterial phase, rather than in portal
215 venous phase.[22,23] These APFs do not require treatment, as they usually resolve spontaneously or
216 diffuse with the progression of the underlying pathology. [54]

217 *Low-flow anomalous portal connections*

218 Low-flow anomalous portal connections are congenital and acquired portosystemic shunts,
219 porto-portal collaterals that develop in case of portal vein obstruction, and cavo-portal collaterals that
220 for chronic increased resistance at any level of the caudal vena cava.

221 **Congenital portosystemic shunt (CPSS)**

222 CPSS refers to the presence of abnormal vascular connection between portal system and a
223 systemic vein (of the caudal vena cava or azygous system), due to embryonic errors or fetal vascular
224 persistence. Based on its anatomic location, CPSS are customarily divided into two main subtypes:
225 intrahepatic and extrahepatic. *Intrahepatic portosystemic shunt (IHPSS)* mostly affects large-size dogs
226 and generally represents a patent ductus venosus (fetal vascular persistence). [55-57] Depending on the
227 side of insertion into the hepatic caudal vena cava, three phenotypes are commonly described in the
228 veterinary literature: right-sided, central, and the most common left-sided (or -divisional) IHPSS (Fig.
229 9). In humans, IHPSS are classified into four types based on the location of the shunt within the liver,
230 number of connections, and shunt characteristics.[58] With the widespread use of advanced imaging
231 modalities, complex, multiple connections between portal branches and hepatic veins, affecting one
232 or more liver lobes within the liver are increasingly described also in dogs (Fig. 10). Therefore, in the
233 author's opinion, the classification of IHPSS in dogs should be updated (Tab. 2). Complex IHPSS are
234 generally accompanied by critical clinical conditions and should be correctly identified before
235 treating.[59,60]

CUSTOM IHPSS CLASSIFICATION IN DOGS*	NEW IHPSS CLASSIFICATION IN DOGS*
1. Left divisional 2. Right divisional 3. Central	1. Persistent ductus venosus (left, right, central) 2. Aneurysmal IHPSS 3. One or more PSS in a single liver lobe 4. Multiple PSS in several liver lobes

236

237 **Tab.** 2. On the left, current classification of intrahepatic portosystemic shunt in dogs (Tisdall et al.
238 1997; Lamb and White 1998; White and Burton 2000). On the right, the new classification proposal.
239 Four different phenotypes are possible. The persistent ductus venosus is the most commonly
240 encountered IHPSS. It can be anatomically left, right or central located. Other complex IHPSS are
241 increasingly recognized with CT and may involve one or more liver lobes, as already described in
242 children (*Modified from Park et al., 1990 and van Steenbeek et al. 2012).
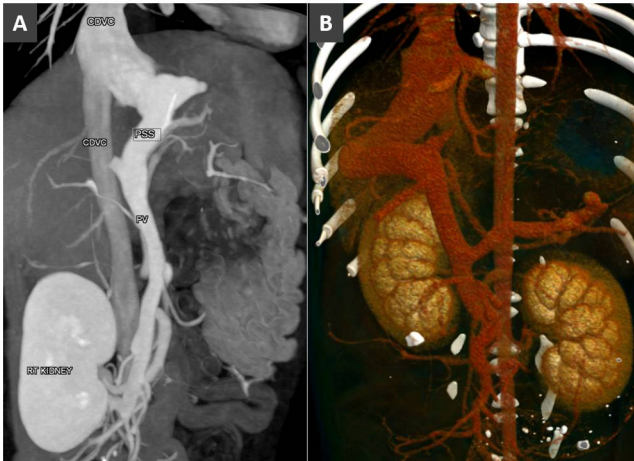
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Fig. 9. A. Dorsal MIP in a dog with left-sided intrahepatic portosystemic shunt. B. Volume rendered image (ventral view) in a dog with right-sided intrahepatic portosystemic shunt.
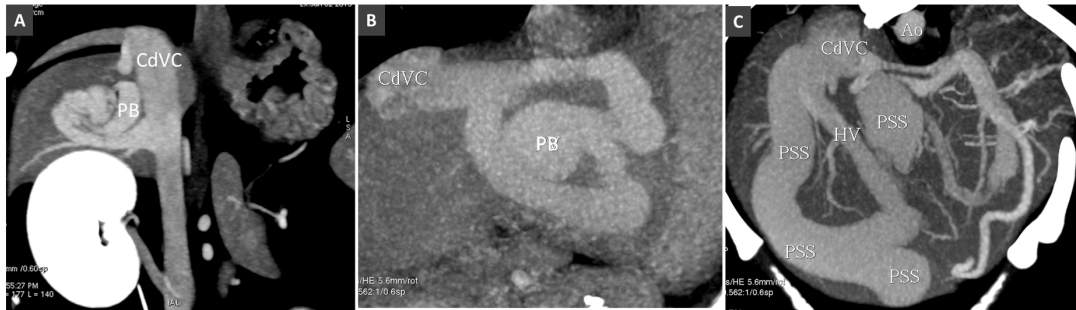

Fig. 10. A. Intrahepatic PSS in a dog involving a single hepatic lobe. PB, portal branch; CdVC, caudal vena cava. B. Intrahepatic PSS through an aneurysmal dilatation of a portal branch (PB). C.Multiple intrahepatic portosystemic connections involving several hepatic lobes.

Extrahepatic portosystemic shunts (EHPSSs) are developmental anomalies resulting from anomalous connections between the vitelline veins, which form the portal system, and the cardinal veins, forming the systemic veins. Although the genetic basis of CPSS in dogs has not been clearly established, many studies have demonstrated that this condition occurs more frequently in purebred dogs and that it is inherited in several small and toy breeds. [7,61-63]

The initial description of Abernethy originally reported in people and the following functional classification of Morgan and Superina may be adopted also in veterinary patients (Tab. 1) (Abernethy 1793; Morgan and Superina 1994; Hunt et al. 1998). [31,32] This classification system is based on whether the portal vein, often hypoplastic, is present and whether the liver is perfused with blood from the mesenteric venous system. These different types have different clinical impact, treatment options, and outcome. In the type I or end-to-side EHPSS the connection is directly between the portal vein and a systemic vein due to CAPV and there is no discernible portal flow to the liver (Fig. 2). The type II or side-to-side shunt, include a number of EHPSS between a portal tributary, most commonly emanating from left or right gastric veins, and a systemic vein (cava or azygos vein) (Fig. 11-14). These connections most commonly are emanating from left or right gastric veins. [64-66] (Tab. 3) The side-to-side is the most common type of CPSS described in veterinary patients. [10-12,64-66] Various repetitive phenotypes of side-to-side EHPSS, which reflect a common underlying embryological error, have been described in dog and cats.). Anomalous portosystemic connections can also involve the mesenteric tributaries of the portal vein system. Mesenterico-renal-caval shunt has been reported in dogs and left colic vein or cranial rectal vein to pelvic systemic vein communications (directly to the caudal vena cava or through common iliac vein or internal iliac vein) has been reported in both dogs and cats. [67,68]

273   **Tab.** 3 Most common patterns of side-to-side EHPSS reported in the veterinary literature. The black
274   dotted lines represent the normal connections between the portal vein and its tributaries.
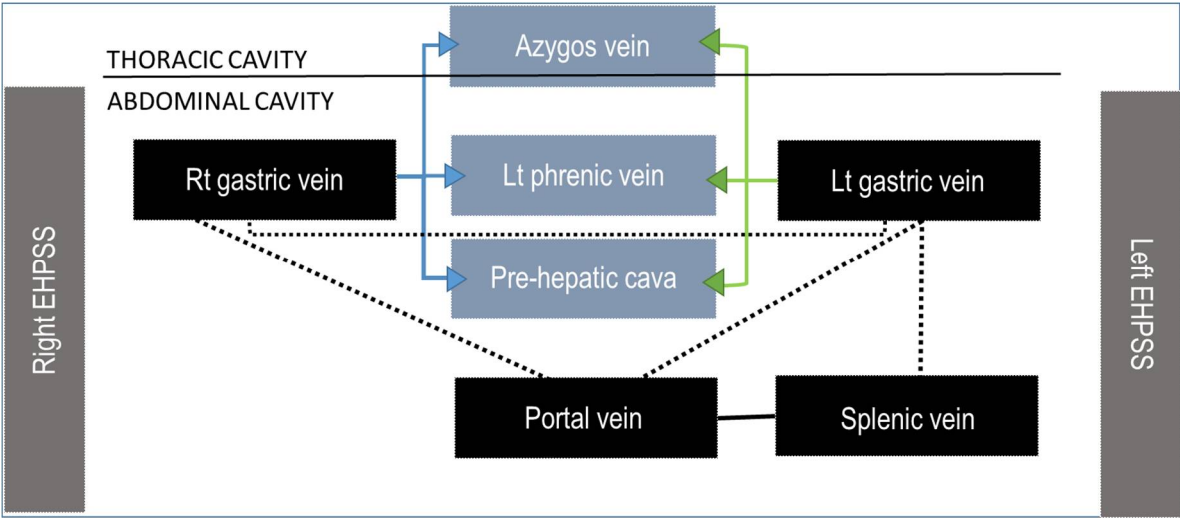
276   The left or right gastric veins can emanate shunts joining the caudal vena cava, phrenic vein or the
277   azygos vein directly or can connect with left spleno-gastric veins and then enter the systemic
278   vasculature. Caudal tributaries contributing to the portal venous system, mesenteric and colic veins
279   can connect directly to the caudal vena cava or enter the systemic circulation via renal vein or pelvic
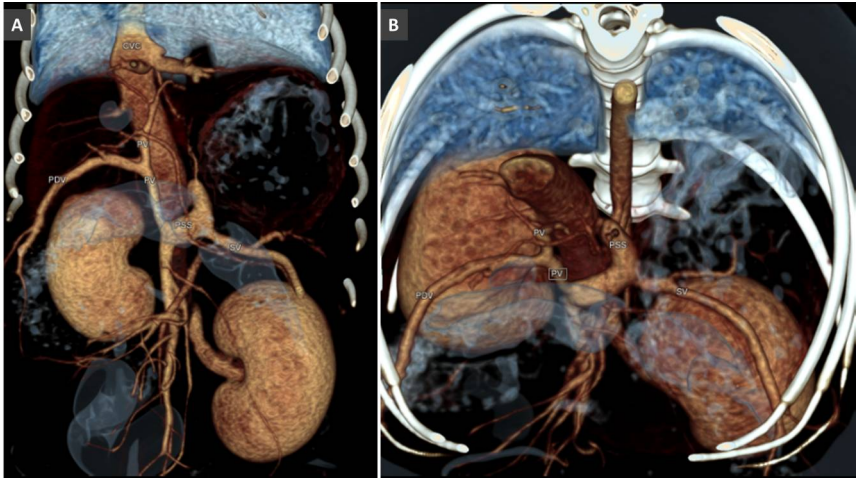280   veins.

282   **Fig.** 11. Extrahepatic PSS emanating from left gastric vein in a dog. A. Volume rendered ventral view.
283   B. Frontal-oblique view, showing the site of insertion of the PSS in the pre-hepatic segment of the
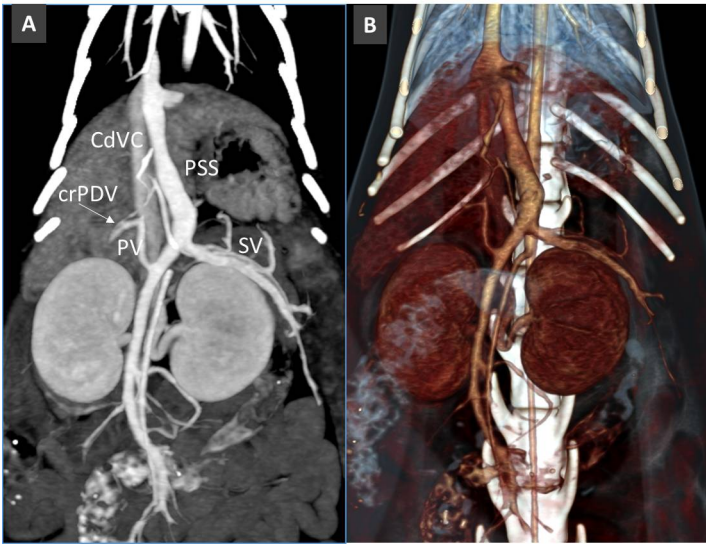284   caudal vena cava.

285

286   **Fig.** 12. Congenital PSS from left gastric vein in a cat. Note the thin, hypoplastic portal vein (PV).
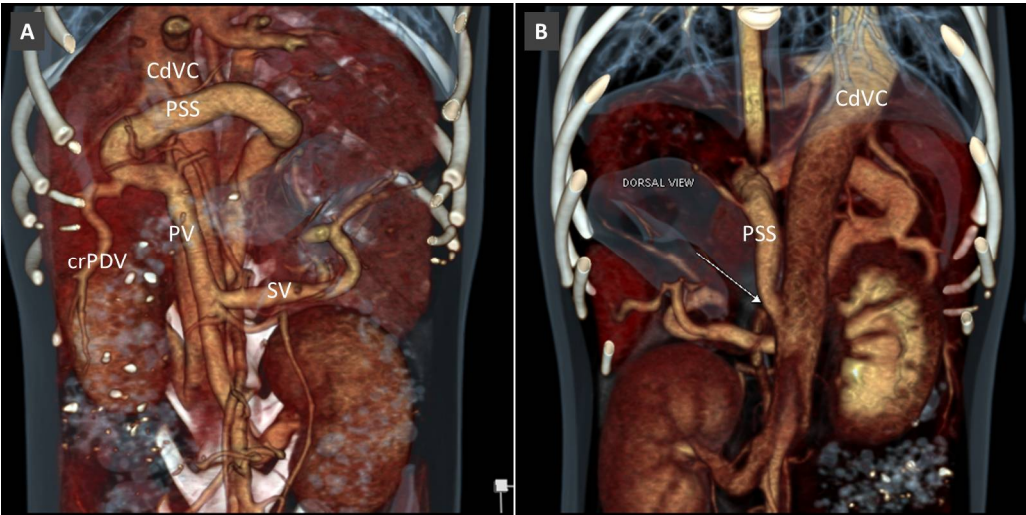

287

288   **Fig.** 13. Extrahepatic congenital PSS from right gastric vein to the pre-hepatic segment of the caudal
289   vena cava (CdVC). A. Volume rendered image, ventral view. B. Dorsal view showing the site of the
290   insertion of the PSS into the CdVC.

291   In side-to-side EHPSS, there is preservation of at least some hepatic portal flow.[69,70] Many of
292   these patients have extremely hypoplastic portal vein cranial to the shunt that is sometimes difficult
293   to visualize with conventional CT angiography. High quality, thin-slices MDCT images may help in
294   detection of a thin portal vein and portal branches. These patients can be treated using partial shunt
295   banding to allow gradual expansion of the intrahepatic portal system while avoiding irreversible
296   portal hypertension. Splenosystemic shunts with the azygous vein, the left phrenic, and left gonadal
297   veins have been reported either among acquired or congenital patterns in dogs and cats and their
298   definitive etiology is still debated.[10-12,48,71,72] The presence of other direct or indirect signs of portal
299   hypertension, such as ascites or varices, can help the interpretation of these vascular patterns.
300   However, those cases without ascites or multiple portosystemic connections are more difficult to
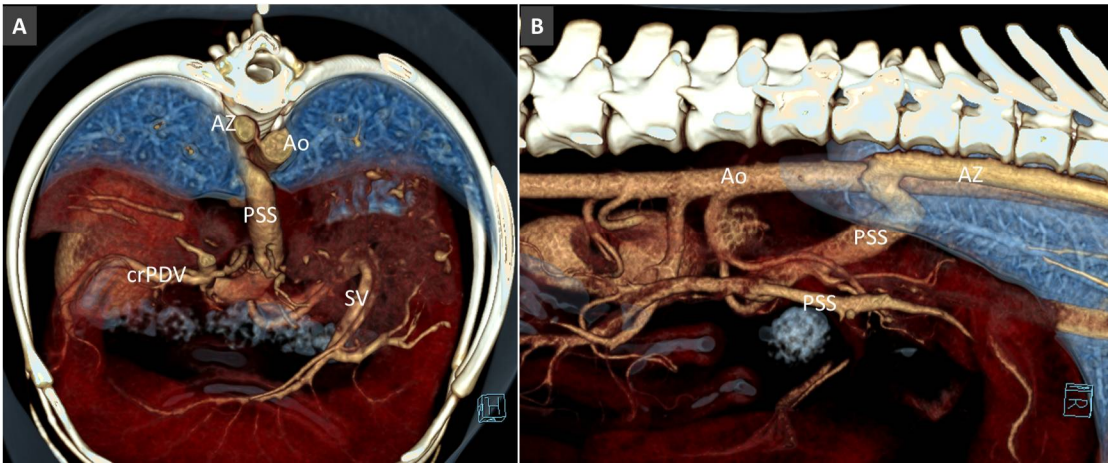301   interpret. [72]

Fig. 14. Congenital extrahepatic PSS in a dog, from left gastric vein to the right azygos vein (Az). A. Volume rendered image, frontal view. B. Right lateral view, showing the course and termination of the shunting vessel into the right azygous vein.

Acquired portosystemic shunts (APSSs)

APSSs are characterized by hepatofugal pathways that can be caused by portal hypertension (increased resistance in the portal system) or increased resistance in the cranial vena cava system. In these cases, a combination of hemodynamic, anatomic, and angiogenetic factors lead to neo-angiogenesis and the opening of pre-existing vascular connections between the portal and systemic circulations. [73] In normal mammals, no gross connection between these systems is present. However, at least three embryonic connections, with no or minimal perfusion, are present in normal animals that may enlarge in case of portal hypertension: the left colic–pudendal vein, left gastric–cardiac esophageal branches, and phrenic–portal vein connections. [4,74]

Several patterns of APSS have been reported in dogs and cats (Bertolini, 2010; Szatmari *et al.*, 2006, Ricciardi, 2016). [10,74,75] They can be divided grossly into large shunts (e.g. left splenogonadal or splenophrenic shunt) and small shunts (e.g. esophageal, gastrophrenic or colic varices). Large and small APSS often coexist in the same patient (Fig. 15, 16). Varices may be subdivided according to their anatomic location and pathways into left gastric vein, gastrophrenic, omental, gallbladder, abdominal wall, duodenal, and colic varices. [10,76]

Importantly, anomalous vascular connections between systemic and portal systems can also occur in case of chronic obstruction of the caudal vena cava (cavo-portal collaterals) allowing blood to return to the right atrium. [77] Special care should be taken to not misinterpret these vascular anomalies.
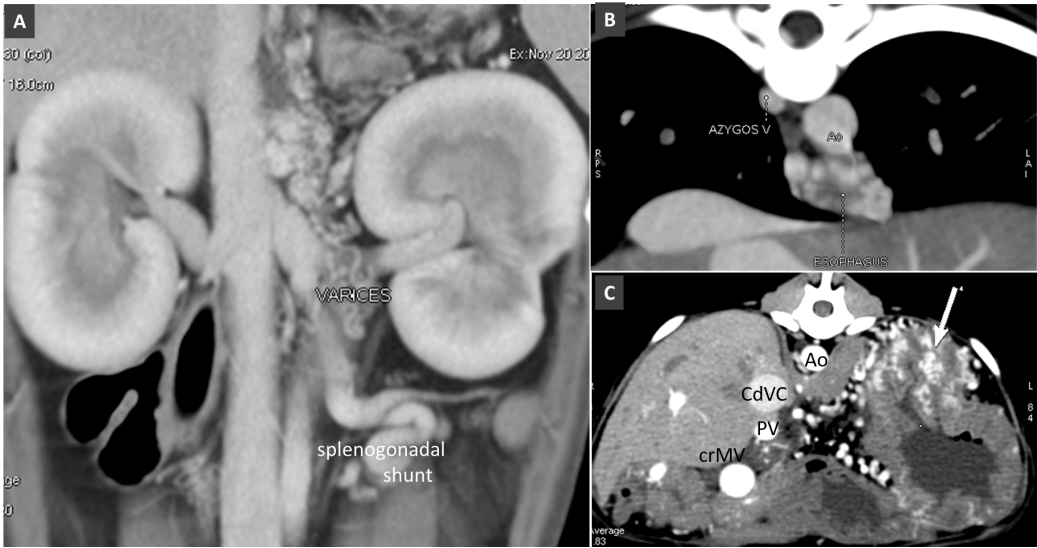
326 **Fig.** 15. A. Dorsal thin-MIP showing small varices in left retroperitoneal region (gastrophrenic varices)
327 and splenogonadal shunt in a dog with portal hypertension. B. Transverse view at the diphragmatic
328 level in a dog with portal hypertension and esophageal varices. C. Transverse view of the cranial
329 abdomen in a dog with portal hypertension due to HAVM. Arrow indicates gastric submucosal
330 varices. Ao, aorta; CdVC, caudal vena cava; PV, portal vein; crMV, cranial mesenteric vein.
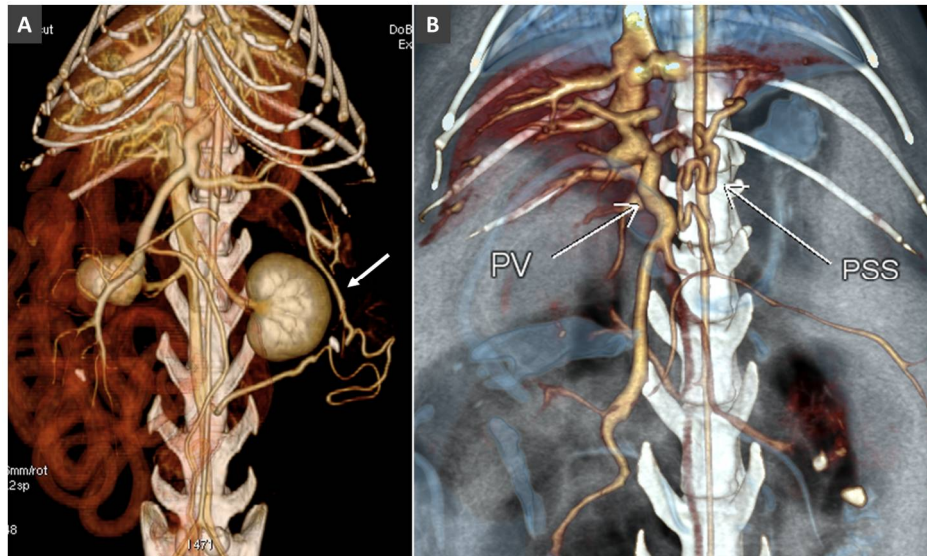
331

332 **Fig.** 16. A. Splenogonadal shunt in a cat with portal hypertension. B. Splenophrenic shunt in another
333 cat with portal hypertension.

334 **Porto-portal collaterals or cavernous transformation of the portal vein**

335 The gradual thrombosis or extrinsic compression of the portal vein stem may lead to obstruction
336 of the portal vein that will result partially absent on MDCT images. Portal vein obstruction causes
337 portal hypertension and can be accompanied by portal cavernous transformation and acquired
338 portosystemic shunts (APSSs). [10,48]
339 *Cavernous transformation of the portal vein (CTPV)* refers to the radiological appearance of porto-
340 portal collaterals around a thrombosed portal vein. [48] Two main types of porto-portal collaterals are
341 described in dogs and cats: short tortuous collaterals developing around/inside the thrombus and
342 long collaterals, forming a network to overcome the obstructed portal vein (Fig. 17). These collaterals
343 course the hepatoduodenal ligament, around the gallbladder and common biliary and cystic ducts,
344 and terminating in portal branches within the liver. When complete portal vein obstruction persists
345 for some time, portal hypertension can ensue and hepatofugal portosystemic collateral pathways will
346 develop. Portal obstruction and indirect signs of portal hypertension must be distinguished from the
347 congenital absence of the portal vein eventually associated with congenital portosystemic shunts
348 (CPSS). [33] The closure of acquired portal collaterals in patients with portal hypertension may be fatal.
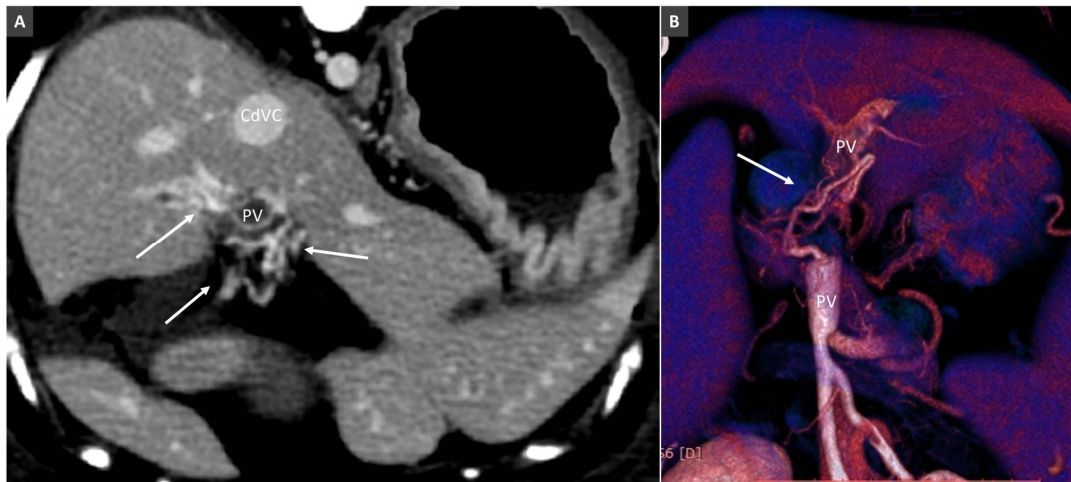
Fig. 17. Porto-portal collaterals (cavernous transformation of PV) in a dog due to PV thrombosis. A. Transverse view at the porta hepatis, showing multiple tortuous vessels surrounding the thrombosed PV (arrows). B. Volume rendered image (ventral view) in another dog with extrahepatic PV thrombosis. Arrow indicates a porto-portal collateral bypassing the site of obstruction.

Conclusion

Portal vein system anomalies are numerous and often related to each other. Correct identification of these vascular anomalies is important because they have different clinical and prognostic significance and is essential for selecting appropriate treatment options.

Conflicts of Interest statement: None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

*All collection procedures presented here were performed solely for the dog's benefit and for standard diagnostic and monitoring purposes. Previous informed written consent was obtained from all dog owners. All the procedures performed complied with the European legislation "on the protection of animals used for scientific purposes" (Directive 2010/63/EU) and with the ethical requirement of the Italian law (Decreto Legislativo 04/03/2014, n. 26).

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