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# Canine and feline abdominal arterioportal communications can be classified based on branching patterns in computed tomographic angiography

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**Introduction**

In people, vascular malformations are subdivided into two categories: slow-flow and fast-flow malformations. Slow-flow malformations represent anomalous connections between capillary, venous and lymphatic components. High-flow vascular malformations contain arterial components in combination with other vascular structures (Mulliken et al. 1982). Arteriovenous fistulae (AVF) and arteriovenous malformations (AMVs) are both high-flow anomalies. AVFs have a direct connection between the artery and vein without any intervening network (Lowe et al. 2012) and are usually acquired lesions secondary to trauma (Tidwel et al. 1997), surgical interventions (Aiken et al. 1993) and neoplasia (Lewis et al. 1002, Ninomiya et al. 1995). Alternatively, AVMs have a dense network of abnormal vessels representing the arteriovenous communication, called a “nidus”. Arterioportal vascular anomalies are communications between the splanchnic arteries and the portal system that represent a rare cause of presinusoidal portal hypertension in small animals.

Computed tomography angiography or selective fluoroscopic angiography (Chanoit et al. 2007) allow direct visualization of the canine hepatic and related vasculature, such as arterioportal communications. The advantages of CT angiography compared to fluoroscopic angiography include the ability to perform a minimally invasive peripheral injection of contrast medium for the evaluation of the whole abdominal vasculature in a short period of time (Zwingenberger et al 2005).

There is little information concerning the imaging findings of arterioportal communications in small animals (Zwingenberger et al 2005, Chanoit et al. 2007) and no classification is provided for radiologists and surgeons. The aims of this study are to describe the computed tomographic imaging findings of arterioportal communications in small animals and to propose a classification based on the computed tomographic anatomy.

**Materials and Methods**

This is a retrospective descriptive multicentric study. Medical electronic report databases of twelve veterinary hospitals (Animal Medical Center, the Clinica Veterinaria dell’Orologio, North Carolina State Veterinary Hospital, the Policlinico Veterinario Roma Sud, Royal Veterinary Collage Royal (Dick) School of Veterinary Studies of the University of Edinburgh, Hope Advanced Veterinary Hospital, Faculty of Veterinary Science of Chulalongkorn University, Clinica Veterinaria Santa Fara, Ospedale Veterinario Pingry, Alphavet, École Nationale Vétérinaire d’Alfort) were reviewed to identify dogs with arterioportal communications diagnosed between 2007 and 2017. Dogs were included if abdominal computed tomography angiography including pre- and post-contrast images was available for review. Images were analyzed by two board-certified veterinary radiologists (S.S and F.R.) and an imaging intern (S.M.) through a two-step approach.

The collected data included contrast bolus direction when arterial and portal phases were available (performed through the evaluation of the attenuation in the cranial and caudal abdominal aorta and in the vessels of the cranial and caudal aspect of the portal system), localization of the arterioportal communication (as intra or extra-hepatic), identification of the afferent artery (or arteries) and efferent vein(s), change in diameter of the aorta caudal to the afferent artery, subjective enlargement of the afferent artery, aneurysmatic dilatation of the portal branches (defined as subjectively saccular or fusiform dilatation of the portal system), segmental or diffuse microhepatica, acquired portal collateral circulation, presence of indirect imaging findings of portal hypertension (ascites, pancreatic edema, gastric wall edema and gallbladder wall edema), biliary abnormalities (intra or extra-hepatic biliary ducts dilatation and/or biliary lithiasis), and/or presence of other concomitant abdominal vascular abnormalities.

In order to propose a computed tomographic classification for the anatomical appearance of arterioportal communications, images were reviewed multiple times until observers reached a consensus on the classification criteria. When available, the presence of portal hypertension through direct invasive catheterization of the portal vein was recorded.

**Results**

Thirty-five patients were included (12 from Animal Medical Center, 6 from the Clinica Veterinaria dell’Orologio, 4 from North Caroline State University Veterinary Hospital, 3 from the Policlinico Veterinario Roma Sud, 2 from the Royal (Dick) School of Veterinary Studies, 2 from Royal Veterinary College, 1 from Hope Advanced Veterinary Hospital, 1 from the Faculty of Veterinary Science of Chulalongkorn University, 1 from Clinica Veterinaria Santa Fara, 1 from Ospedale Veterinario Pingry, 1 from Alphavet, 1 from the École Nationale Vétérinaire d’Alfort).

There were 32 dogs (4 intact females, 11 spayed females, 9 intact males and 8 neutered males) and 3 cats (2 spayed females and 1 male neutered). Mean age was 30 months (range from 3 to 108 months). Breeds included were 17 mixed breed dogs, 5 Labrador Retrievers, 3 Welsh Corgie, 2 Poodle, 2 Weimaraner, 1 Beagle, 1 Golden Retriever and 1 Great Dane. The 3 cats were all domestic shorthair.

A computed tomography arterial phase was available in 20/35 patients. There were 31 intrahepatic and 4 extrahepatic arterioportal communications. In the intrahepatic arterioportal communications, a clear identification of the afferent vessel was possible in only 12/31 cases while the efferent vessels were identified in all patients. Hepatofugal direction of the contrast bolus in the portal system was observed in 25/35 patients demonstrated by the presence of contrast medium in the cranial mesenteric vein during the arterial phase without contrast in the jejunal vein or caudal mesenteric vein. In addition, in these patients, there was equal distribution of the contrast medium bolus in the aorta and cranial mesenteric vein and/or its tributaries during the arterial phase.

Other imaging findings included subjective enlargement of the afferent artery (22/35), aneurysmatic dilatation of the portal branches (20/35), decreased diameter of the abdominal aorta caudal to the afferent artery (18/35), and segmental (9/35) or diffuse (14/35) microhepatica.

Multiple patterns of acquired portal collateral circulation were observed in 33/35 patients and gallbladder varices were observed in 11/35 patients. The two patients with no acquired collateral portal circulation had concomitant congenital intra-hepatic shunts.

Indirect imaging features of portal hypertension were pancreatic edema in 20/35, ascites in 19/35 and gastric wall edema 18/35. Invasive evaluation of portal pressure was available in 12 patients with hepatofugal bolus dynamic in the portal system and confirmed portal hypertension in all of them.

Biliary abnormalities were observed in 6/35 patients. Two patients had intra-hepatic biliary lithiasis, one patient had subjective dilatation of the gallbladder, one patient had gallbladder lithiasis and subjective dilatation of the gallbladder, one patient had dilatation of the intra-hepatic biliary ducts in the quadrate lobe and one patient had subjective distention of the common bile duct.

Other concomitant vascular abnormalities that were identified were a congenital left divisional intra-hepatic porto-systemic shunt in 2/35 patients. In patients with concomitant intra-hepatic shunt contrast medium was visible in the intra and post-hepatic tract of the caudal vena cava during the arterial phase. Indirect imaging features of portal hypertension were absent in these patients.

Due to the inconsistent visualization of the afferent vessel, we classified the intrahepatic arterioportal communications only based on the efferent vessel in left (20/31) and right (11/31) divisions. The left divisional were sub-classified according to the corresponding portal branch in left medial (if the quadrate or right medial were involved [16/20]) and left lateral (if the left lateral or left medial were involved [4/20]). Right divisional communications always involved the right lateral portal vein (11/19) and in 2 cases concomitant dilation of the caudate portal branch was observed. For this reason, a sub-classification for the right divisional was not possible. In case of intra-hepatic left divisional AVMs and medial conformation, enlargement of the portal branches of both right medial and quadrate lobe were observed in 6/16 patients. These branches were in continuity with the gallbladder varices. Only animals with left divisional and medial intrahepatic arterioportal communications had gallbladder varices.

In the 4/20 cases of intra-hepatic left divisional and lateral conformation, only one case showed clear involvement of the portal vessel of the left medial hepatic lobe with normal conformation of the portal vessel of the left lateral hepatic lobe. In the other three cases of intra-hepatic left divisional and lateral conformation, the arterioportal malformation involved the portal vessels of both left lateral and medial hepatic lobes.

See Table 1 for schematic classification of single versus double efferent veins in left and right divisional arterioportal communication.

In 20 cases, a single intra-hepatic portal branch was involved, including 11 left divisional and 9 right divisional (Table 1).

In three patients with extra-hepatic arterioportal communications the afferent artery was represented by the caudal mesenteric artery or one of its branches. The efferent veins were the cranial mesenteric vein or the left colic vein. One dog had two extrahepatic arterioportal communications, a more cranial arterioportal communication with the afferent artery consistent with the distal portion of the cranial mesenteric artery and the efferent vein was identified as the cranial mesenteric vein. A segmental saccular aneurysmatic dilatation of the caudal portion of the cranial mesenteric vein was also observed. The afferent artery of the caudal arteriovenous communication was the caudal mesenteric artery and the efferent vessel was the left colic vein. One dog with extra-hepatic arterioportal communication showed also multiple healed pelvic fractures.

**Discussion/Conclusions**

Computed tomographic features of arterioportal communications in small animals are reported in this study and a classification based on the anatomical conformation is proposed. In this cohort of dogs, intra-hepatic arterioportal communications were more common than the extra-hepatic fistulas. Computed tomographic visualization of the afferent and efferent vessels was variable. In patients with intra-hepatic arterioportal communications, the branch of the hepatic artery responsible of the arterioportal communication was inconstantly visualized. In patients with extra-hepatic arterioportal communication there was no “nidus” and the artery responsible for the arterioportal communication was always identified. In the authors’ opinion, there are two different factors influencing the ability to determine the afferent artery: the more complex anatomical conformation of the hepatic artery compared to the cranial and caudal mesenteric arteries and the type of arterioportal communication. In particular, intra-hepatic arterioportal communications always showed a nidus, with presence of a multitude of adjacent and tortuous vessels making the identification of the afferent artery difficult. In contrary, the extra-hepatic arterio-portal communication did not show any nidus with an easier identification of the direct communication between the afferent artery and efferent vein. For these reasons, the term arterioportal malformations is more appropriate when referring to intra-hepatic arterioportal communications while arterioportal fistulae is a more suitable term for extra-hepatic arterioportal communication in these cases. However, even if there was no evidence of a previous trauma or surgery, it was not possible to finally confirm the congenital nature of the intra-hepatic communication in our cases. Interestingly, one of the 4 dogs with extra-hepatic fistula, presented healed pelvic fractures suggesting a possible acquired origin.

All patients with intra-hepatic right divisional arterioportal malformation, showed involvement of the portal vein of the right lateral hepatic lobe. Concomitant involvement of the portal vein of the caudate lobe was observed in two patients. This is a useful information for surgeons approaching this group of patients because vascular occlusion of the main right portal branch may allow complete closure of the arterioportal malformation.

In patients with arterial phase available, we detected the same bolus dynamic in the aorta and portal circulation (portal vein and cranial mesenteric vein). This finding demonstrates that in the arterial phase the bolus direction in the aorta and portal vein/cranial mesenteric vein has similar dynamics suggesting a hepatofugal direction of the blood flow in the cranial mesenteric vein. Furthermore, the presence of severe portal hypertension was confirmed through invasive catheterization of the portal vein in twelve patients. The intrahepatic arterioportal malformations have hepatofugal portal blood flow unless a concurrent intrahepatic portosystemic shunt is present (CW personal communication).

As previously reported, we commonly observed an increased size of the afferent artery (Zwingenberger et al. 2005). This is due to low resistance to blood flow through the arterioportal communication compared to the blood flow resistance in arteries, capillaries and hepatic sinusoids with secondary increased blood volume through the afferent vessel over time. There is also “siphoning” of blood from the nearby normal vessels into the arterioportal communication contributing to the enlargement of the afferent artery and with secondary decreased oxygenation of the adjacent organs, also called “blood steal phenomenon” (Kube et al. 2004, Westworth DR 2006, Zwingenberger et al 2005, Argawala et al. 2000, Hosgood et al. 1989, Landers et al. 1978). The “blood steal phenomenon” causes decreased blood flow in the arteries adjacent to the arterioportal communication explaining the abrupt decreased diameter of the aorta and cranial mesenteric artery observed in this study and as previously reported by Zwingenberger et al.

Aneurysmatic dilatation of the portal vein and its branches was a common finding in patients with intra-hepatic arterioportal malformations. Aneurysmatic dilatation of the cranial mesenteric vein was also observed in a patient with extra-hepatic arterioportal fistula. Aneurysmatic dilatation of the portal vein and of its intra-hepatic branches has been previously reported alone (Bertolini et al. 2012) or associated to other concomitant vascular diseases (Bertolini et al. 2010, d’Anjou et al. 2008, Zwingenberger et al. 2005, Kumar et al. 2005). As reported in humans, aneurysmatic dilatation of the portal vein and its branches has likely resulted from chronic portal hypertension and turbulent arterial flow with secondary weakening of the wall with progressive thickening of the intima and replacement by fibrous tissues (Zafer et al. 2007, Gallego et al 2002, Lopez-Machado et al 1998).

Ascites was a frequent finding. Factors that contribute to ascites formation are portal hypertension and hypoproteinemia. Portal pressure was invasively evaluated in 12/35 patients confirming the presence of portal hypertension, and indirect computed tomographic imaging findings of portal hypertension such as pancreatic and gastric wall edema or acquired porto-systemic shunts were observed in all patients with intra-hepatic malformation except the two dogs with concomitant intra-hepatic portosystemic shunts. Imaging findings of “blood steal phenomenon” in the cranial mesenteric artery were observed in patients with intra-hepatic arterioportal malformations. We did not retrospectively evaluate the total proteins and albumin levels in these patients. However, we presume that the association of arterial “blood steal phenomenon” and portal hypertension may have caused venous congestion of the gastrointestinal tract with secondary malabsorption in the small bowel and possible hypoproteinemia as previously reported (Zwingenberger et al. 2005).

Presence of microhepatica was a frequent finding. We presume microhepatica is related to both “blood steal phenomenon” and portal hypertension. With “blood steal phenomenon” the arterial blood bypasses the arterial capillaries with decreased amount of oxygen to the hepatocytes (Zwingenberger 2005).

Arterioportal malformation with concomitant congenital intrahepatic portosystemic shunting was detected in two patients with no ascites and no portal collateral circulation. We believe that the absence of ascites in these patients is due to the arterial blood entering the caudal vena cava directly, bypassing the portal system, with lack of portal hypertension signs compared to patients with arterioportal malformation only.

Multiple extra-hepatic arterioportal fistulae were observed in one patient. The presence of multiple concomitant vascular anomalies is related to the common embryological origin of these vessels, and developmental abnormalities may affect multiple vessels as previously reported (Bertolini et al. 2014). One patient with extra-hepatic arterioportal fistula had multiple healed pelvic fractures. It is possible that in this patient the fistula is acquired post-traumatic as reported in humans (Tidwel et al. 1997).

Variable patterns of portal collateral circulation have been observed in our cohort of dogs reflecting the previously proposed classification (Bertolini 2010). We observed gallbladder varices only in patients with arterioportal malformations with an intrahepatic left divisional and medial conformation. Interestingly, also in a previous study concerning acquired portal collateral circulation due to different causes, gallbladder varices were observed only in a patient with an intra-hepatic arterioportal malformation (Bertolini 2010). The arterial perfusion of the gallbladder has been previously described as originating from the right medial branch of the left hepatic artery (Miller Anatomy of the dog) or directly from the left hepatic artery (Ursic). The right medial branch of the hepatic artery is also responsible for the perfusion of the right medial lobe, dorsal portion of the quadrate lobe and part of the left medial lobe. Little information is available concerning the venous drainage of the gallbladder wall in small animals. In humans, the gallbladder wall drainage consists in multiple cystic/cholecystic veins that drain mainly within the portal system to subsequently join the middle or right hepatic veins (AJR 169 August 1997). We observed enlargement of the portal branches of the right medial and quadrate lobes in patients with gallbladder varices and intrahepatic left divisional and medial arterioportal malformation. Our hypothesis is that gallbladder varices in these patients do not represent collateral portal circulation but enlargement of the venous and arterial capillary bed due to direct involvement of these vessels in the arterioportal malformation nidus with secondary increased blood flow. The enlargement of the portal branches of the right medial and quadrate lobe suggests that these branches are responsible for the gallbladder wall venous drainage in these patients.

Gallbladder, intra- and extra-hepatic biliary tract distension and gallbladder lithiasis were observed. Biliary ischemia due to arterioportal shunt (Wu JS et al. 2006) is recognized in human as cause of biliary dilatation, biliary cyst or strictures. Our hypothesis is that these conditions may have predisposed to biliary tract distention and gallbladder lithiasis. In dogs with portosystemic shunts, arteriolar proliferation and biliary hyperplasia have been reported and could also play a role in the development of biliary abnormalities (Parker 2008).

The main limitations of the study were the different computed tomographic angiography protocols, the absence of arterial phase and the invasive evaluation of portal pressure in only a fraction of patients. Furthermore, computed tomography doesn’t offer the advantages of complete dynamic as provided by digital fluoroscopy, therefore the representation of the abnormal vascular anatomy might be incomplete.

The consensus on the classification of the arterioportal communication was the following: patients with intra-hepatic arterioportal malformations could be classified based on the efferent portal vein (or veins) as left or right lateral divisional and in case of left lateral divisional, they could be subclassified as medial versus lateral. Patients with extra-hepatic arterioportal communications could be classified based on the name of the afferent and efferent vessel only.

There are still no clear guidelines in the treatment of arterioportal malformations, but different possibilities have been proposed such as embolization of the afferent vessel or liver lobectomy (Chanoit et al. 2007). Future promising treatments such as outflow vein embolization are recently proposed also in veterinary medicine and these techniques would benefit from an outflow portal vein classification scheme (Weiss book). In order to facilitate the radiologist-surgeon communication, to help the pre-surgical planning and to support the treatment choice, we decided to emphasize the name of the afferent and efferent vessel (in case of extra-hepatic arterioportal malformation) or the lobe/lobes in which the efferent portal vessel (and number of efferent vessels) was located (in case of intra-hepatic arterioportal malformation).

In conclusion, we reported the computed tomographic features of arterioportal malformations in small animals and we proposed an anatomical classification based on CT angiography that will allow veterinary radiologists to have a more systematic approach helping the radiologist-surgeon communication.

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