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Portal Vein/Aorta Ratio in Dogs with Acquired Portosystemic Collaterals

Y. Sakamoto, M. Sakai, and T. Watari

Background: The portal vein (PV) diameter increases in humans with portal hypertension (PH). However, there is no evidence of PV enlargement in dogs with PH.

Objectives: To measure the PV-to-aorta (PV/Ao) ratio in dogs with PH (chronic hepatitis [CH], primary hypoplasia of the PV [PHPV]), in dogs with extrahepatic congenital portosystemic shunt (EH-CPSS), and in healthy dogs, and to evaluate the relationship between PV/Ao ratio and splenic pulp pressure (SPP).

Animals: Twenty-five dogs with acquired portosystemic collaterals (APSCs; 15 with CH, 10 with PHPV), 32 dogs with EH-CPSS, and 20 healthy dogs.

Methods: Retrospective study. The PV/Ao ratio was calculated with images obtained by computed tomography. SPP was measured at the time of liver biopsy in 45 dogs.

Results: Median PV/Ao ratio was similar between dogs with CH (1.35, range 1.05–2.01) and healthy dogs (0.95, 0.80–1.15), but differed significantly between the CH group and both the PHPV (0.40, 0.24–0.67) and EH-CPSS groups (0.30, 0.11–0.64) ($P < .001$). The PV/Ao ratio was significantly lower in the PHPV group than in healthy dogs ($P < .05$). It also correlated positively with SPP ($r_s = 0.71$; $P < .001$). However, there was no intragroup correlation between SPP and the PV/Ao ratio in any group.

Conclusions and Clinical Importance: The PV/Ao ratio can be evaluated in dogs with APSCs on computed tomography. Further studies are needed to examine the relationship between SPP and the PV/Ao ratio in larger groups of dogs with PH and to determine its clinical relevance.

Key words: Canine; Multidetector computed tomography; Portal hypertension; Splenic pulp pressure.

Portal hypertension (PH) is caused by increased resistance and blood flow in the portal circulation and is classified as prehepatic, intrahepatic, or posthepatic.[1–3] Prehepatic PH is attributable to increased resistance in the extrahepatic portal vein (PV) and is associated with mural or intraluminal obstruction or extraluminal compression.[4,5] Intrahepatic PH is caused by increased resistance in the microscopic PV tributaries, sinusoids, or small hepatic veins and is classified histologically as presinusoidal, sinusoidal, or postsinusoidal. Presinusoidal PH occurs because of increased resistance in the terminal intrahepatic PV tributaries. The most common causes in dogs are primary hypoplasia of the portal vein (PHPV), idiopathic hepatic fibrosis, and hepatoportal fibrosis.[6–11] Sinusoidal intrahepatic PH is usually a consequence of fibrotic hepatopathy. In these disorders, sinusoidal endothelial cells lose their fenestrae and acquire a collagenous basement membrane that increases intrahepatic venous resistance.[12–14] These disorders include chronic hepatitis (CH). Postsinusoidal intrahepatic PH is associated with veno-occlusive disease.[15] Posthepatic PH is associated with obstruction of the larger hepatic veins, the posthepatic caudal vena cava, or the right atrium. Most PH in dogs is intrahepatic and associated with CH and PHPV.[1,3,16] The clinical consequences of PH include development of acquired portosystemic collaterals (APSCs) and ascites, or both.[1–3]

The size of the PV increases in dogs with CH[17–19] but decreases in dogs with extrahepatic congenital portosystemic shunt (EH-CPSS) because of decreased portal blood flow.[20–23] Changes in PV size in dogs with these diseases have been evaluated on ultrasound by the PV/aorta (Ao) ratio.[17,20] The PV diameter increases in humans with PH and there is a significant but weak correlation between portal vein pressure (PVP) and PV size in 364 patients with PH.[24] It is speculated that formation of a shunt changes portal blood flow. We

Abbreviations:

Ao	aorta
APSCs	acquired portosystemic collaterals
CH	chronic hepatitis
CPR	curved planar reformation
EH-CPSS	extrahepatic congenital portosystemic shunt
MDCT	multidetector computed tomography
PH	portal hypertension
PHPV	primary hypoplasia of the portal vein
PV	portal vein
PVP	portal vein pressure
SPP	splenic pulp pressure

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hypothesized that changes in PV size might reflect PVP and that these changes could allow PH to be diagnosed in a minimally invasive manner. However, there is little information concerning changes in the size of the PV in dogs with APSCs. Moreover, there has been no report on the relationship between the PV/Ao ratio and splenic pulp pressure (SPP) as PVP in dogs.

Recent reports in dogs have alluded to the popularity of multidetector computed tomography (MDCT), which can rapidly provide a considerable amount of information.[25,26] Images obtained by MDCT also allow high-resolution multiplanar reformation and three-dimensional reconstruction.[25,26] In humans, the long axes of the hepatobiliary and pancreatic duct systems have been determined by curved planar reformation (CPR) of MDCT images.[27–31] This technique is used for diagnosis of pancreatic duct tumors and placement of stents in humans.[27–32] CPR of MDCT images might also be able to measure the sizes of the PV and Ao correctly and calculate the PV/Ao ratio in dogs with PH. The aims of this study were to measure the PV/Ao ratio in dogs with PH by CPR of MDCT images and to evaluate the relationship between the PV/Ao ratio and SPP.

Materials and Methods

Dogs

The study included 25 client-owned dogs with APSCs that visited the Nihon University Animal Medical Center between October 2005 and June 2012 and had a diagnosis of liver disease with histologic evidence of CH or PHPV. APSCs were suspected in 24 dogs on laparoscopic splenoportography[33] and in 1 dog on portography performed during laparotomy. Dogs with APSCs confirmed by portal angiography were allocated to a PH group. Liver biopsy specimens were examined histopathologically by an American College of Veterinary Pathologists board-certified pathologist, and CH and PHPV were diagnosed according to the criteria developed by the World Small Animal Veterinary Association Liver Standardization Group. The controls comprised 20 laboratory-bred Beagle dogs and 32 dogs with EH-CPSS. Ethical approval for use of the Beagle dogs in this study was granted by the College of Bioresource Sciences, Nihon University, and the study protocol was conducted in accordance with our institution's Guide for Animal Experimentation. For convenience purposes, the group sizes in this study were small.

Measurement of PV/Ao Ratio

MDCT was performed in all 3 groups of animals. The dogs were premedicated with a combination of midazolam (0.1 mg/kg administered intravenously [IV]) and butorphanol (0.2 mg/kg IV). General anesthesia was induced by propofol (4 mg/kg IV) and maintained with isoflurane and oxygen. The dogs were then placed in the sternal recumbent position. The area between the cranial aspect of the diaphragm and the ala of the ilium was visualized by a 16-slice MDCT scanner[a] under the following conditions: rotation time, 0.5 seconds; slice thickness, 1–2 mm; reconstruction interval, 0.5–1 mm; table speed, 16–32 mm/rotation; helical pitch, 16.0; X-ray tube potential, 120 kV; and X-ray tube current, 150 mA. The data obtained were analyzed by the image analysis software[b] supplied with the workstation. The cross-sectional areas of the PV and Ao were calculated by CPR (Fig 1). The CPR tool can extract blood vessels and other tubular structures, such as pancreatic

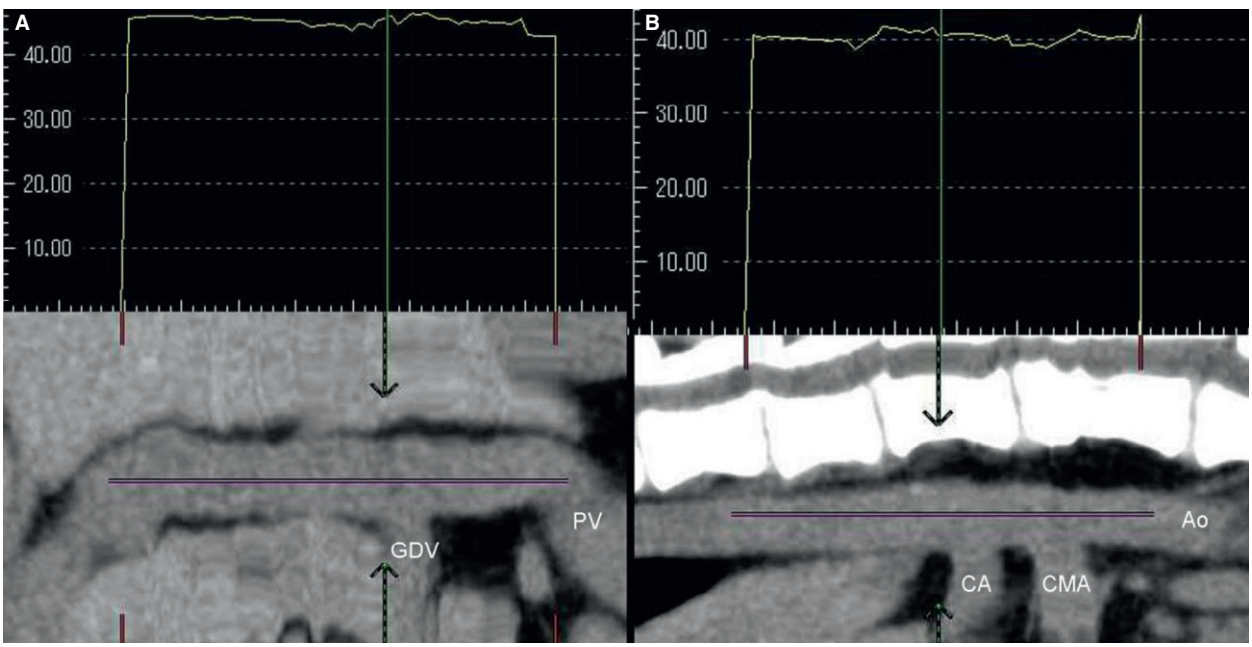

Fig 1. Curved planar reformation (CPR) images obtained on multidetector computed tomography (MDCT) from a healthy dog. CPR images of the PV **(A)** and Ao **(B)** and graphs of these areas. This tool allows quantitative analysis of vascular anatomy without using contrast medium and provides cross-sectional areas of true orthogonal sections of the PV and Ao at selected anatomic points in unfolded two-dimensional views. The mean values for the PV and Ao areas were determined at 3 cranial points (0, 5, 10 mm) at the levels of the GDV and the CA. These points comprised points at 5 mm and 10 mm from the 0 mm point on the cranial side of the GDV to the head side and points at 5 mm and 10 mm from the 0 mm point on the cranial side of the CA to the head side, respectively. Ao, aorta; CA, celiac artery; CMA, celiac mesenteric artery; GDV, gastroduodenal vein; PV, portal vein.

ducts or bile ducts, on two-dimensional or three-dimensional images, and measure their diameter and area. After manually selecting a blood vessel, the CPR image is displayed (Fig 1). After confirming the site to be measured, the central line is placed at the center of the orthogonal cross-sectional image of the site, the outline of the selected blood vessel is manually corrected and reflected on the image, and the diameter and cross-sectional area of the target vessel can be measured (Fig 2). The vascular images of the Ao were straightforward to obtain and easily measured. However, the vascular images of the PV tended to meander and it was unclear whether this was related to the high PVP in dogs with APSCs. Therefore, measurements were obtained at 3 discrete points to minimize these fluctuations. The mean values for the areas of the PV and Ao were determined at 3 cranial points (0, 5, 10 mm) at the levels of the gastroduodenal vein and celiac artery. These points comprised points 5 mm and 10 mm from the 0 mm point on the cranial side of the gastroduodenal vein to the head side and points at 5 mm and 10 mm from the 0 mm point on the cranial side of the celiac artery to the head side, respectively. The ratio of the mean luminal area of the PV to the Ao was then calculated. One veterinarian (MS) interpreted the computed tomography images and another veterinarian (YS) measured the PV/Ao ratio. Both veterinarians were blinded to group allocation.

Measurement of Splenic Pulp Pressure

The dogs were placed in the right lateral recumbent position and the abdominal cavity was insufflated with CO_2.[c] Intra-abdominal pressure was automatically maintained at 8–10 mmHg. Two cannulae were placed in the abdomen and the abdominal cavity was visualized by a laparoscope.[d] An 18-gauge over-the-needle intravenous catheter[e] was inserted percutaneously into the spleen parallel to the long axis. After the pneumoperitoneum was stopped and all gas was removed from the abdominal cavity, the splenic pulp pressure (SPP) was measured via the catheter as described elsewhere.[16,34] SPP was measured at the time of liver biopsy in 45 dogs (15 dogs with CH, 8 dogs with PHPV, 16 dogs with EH-CPSS, and 6 healthy dogs).

Statistical Analysis

All data were analyzed by GraphPad Prism for Mac OS X version 5.0b software.[f] All variables measured were tested for normal distribution in each group by the Kolmogorov-Smirnov and Shapiro-Wilk normality tests. The data were not normally distributed and are reported as the median and range. The PV/Ao ratio was compared between the groups by the Kruskal-Wallis test with Dunn's multiple comparison post test. Correlations between the SPP and PV/Ao ratio were evaluated by Spearman's correlation coefficients. Differences were considered to be statistically significant at $P < .05$.

Results

The 20 Beagle dogs (14 males, 6 females) used as controls were confirmed to be healthy by clinical examination, a complete blood cell count, serum biochemistry, radiography, and abdominal ultrasonography; their median age and body weight were 3.7 (2.3–5.8) years and 9.8 (8.5–13.0) kg, respectively. The 32 client-owned dogs with EH-CPSS (confirmed by computed tomography angiography as described elsewhere[23]) comprised 14 breeds (Table 1). Twenty-five dogs with APSCs were histologically differentiated into a group with CH (n = 15) and a group with PHPV (n = 10) according to the criteria developed by the World Small Animal Veterinary Association Liver Standardization Group (Table 1).[2]

Initially, we speculated that the size of the PV in a dog with APSCs might be increased. However, given that the size of the PV was small in dogs with PHPV and large in dogs with CH, we divided the APSCs into 2 groups of CH and PHPV after completing the experiment. We confirmed macroscopically that the cross-sectional contours of the Ao and PV were accurately traced. The median PV/Ao ratio was 1.35 (1.05–2.01) in dogs with CH, 0.40 (0.24–0.67) in dogs with PHPV, 0.95 (0.80–1.15) in healthy dogs, and 0.30 (0.11–0.64) in dogs with EH-CPSS. There was no significant difference in PV/Ao ratio between the group of dogs with APSCs and the healthy group. However, there was a significant difference in PV/Ao ratio between the dogs with EH-CPSS and the healthy dogs ($P < .001$, Fig 3). The PV/Ao ratio in dogs with CH was significantly higher than that in dogs with EH-CPSS ($P < .001$), but there was no significant difference in PV/Ao ratio between dogs with CH and healthy dogs. The PV/Ao ratio in dogs with PHPV was significantly lower than that in healthy dogs ($P < .05$) and dogs with CH ($P < .001$). There was

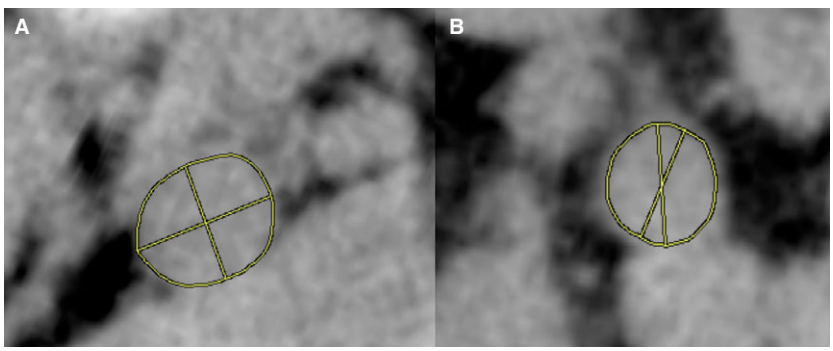

Fig 2. CPR images obtained on MDCT from a healthy dog. Orthogonal cross-sectional images of the PV **(A)** and Ao **(B)** are shown. After confirming the site to be measured, the central line is placed at the center of the orthogonal cross-sectional image of the site, the outline of the blood vessel is manually corrected and reflected on the image, and the cross-sectional area of the target vessel is measured. The yellow lines indicate the outline of the blood vessel. Ao, aorta; PV, portal vein.

Table 1. Patient signalments.

Group	CH (n = 15)	PHPV (n = 10)	Control	
			EH-CPSS (n = 32)	Healthy (n = 20)
Breed	American Cocker Spaniel (6) English Cocker Spaniel (2) Labrador Retriever (2) Miniature Dachshund (2) Jack Russell Terrier (1) Miniature Schnauzer (1) West Highland White Terrier (1)	Maltese (2) Miniature Dachshund (2) American Cocker Spaniel (1) Chihuahua (1) Papillon (1) Siberian Husky (1) Toy Poodle (1) Welsh Corgi (1)	Miniature Schnauzer (9) Papillon (4) Yorkshire Terrier (4) Pug (3) Chihuahua (2) Toy Poodle (2) Italian Greyhound (1) Jack Russell Terrier (1) Maltese (1) Miniature Dachshund (1) Mix breed (1) Norfolk Terrier (1) Shiba (1) Shih Tzu (1)	Beagle (20)
Age[a]	6.4 years (2.2–11.3 years)	2.5 years (0.6–14.3 years)	1.7 years (0.3–7.9 years)	3.7 years (2.3–5.8 years)
Sex[a]	4M, 4MC, 1F, 6FS	1M, 1MC, 5F, 3FS	14M, 5MC, 7F, 6FS	14M, 6F
BW[a]	8.5 kg (5.5–27.1 kg)	3.6 kg (1.1–16.2 kg)	4.0 kg (1.0–10.7 kg)	9.8 kg (8.5–13.0 kg)

Numbers in parentheses indicate number of dogs of that breed. CH, chronic hepatitis; PHPV, primary hypoplasia of the portal vein; EH-CPSS, extrahepatic congenital portosystemic shunt. M, male; MC, male-castrated; F, female; FS, female-spayed. BW, body weight.
[a]Median (range).

no significant difference in PV/Ao ratio between the dogs with PHPV and those with EH-CPSS. The SPP was measured in dogs with CH (n = 15), PHPV (n = 8), and EH-CPSS (n = 16) and in healthy dogs (n = 6). There was a positive correlation between the SPP and the PV/Ao ratio in 45 dogs ($r_s = 0.71$, $P < .001$, Fig 4). Moreover, there was a significant positive correlation between the SPP and the PV/Ao ratio in 37 of the dogs when the dogs with PHPV were omitted ($r_s = 0.86$, $P < .001$). However, there was no intragroup correlation between SPP and the PV/Ao ratio in the dogs with CH, PHPV, or EH-CPSS, or in the group of healthy dogs.

Discussion

The PV and Ao are not easily visualized by ultrasonography because of interference from factors such as gas and feces, or both, in the gastrointestinal tract and the need for a highly experienced veterinarian.[20,35] In contrast, computed tomography usually provides highly detailed anatomic information and the technique is not as operator-dependent as ultrasonography.[36–38] The CPR obtained from MDCT images has been used clinically to evaluate tortuous ducts and blood vessels in humans.[39–41] In the present study, the PV/Ao ratio determined in healthy dogs by MDCT was in agreement with the range determined on ultrasonography in 24 dogs without portosystemic shunt (PV/Ao ratio 0.89 [0.71–1.20]).[17] Further, the PV/Ao ratio was reported to be significantly lower in dogs with EH-CPSS than in healthy dogs when the ratio was also determined ultrasonographically.[17,20] The reduction in size of the PV in dogs with EH-CPSS might be caused by portal blood directly entering the systemic venous system via the shunt vessel before flowing into the liver and by

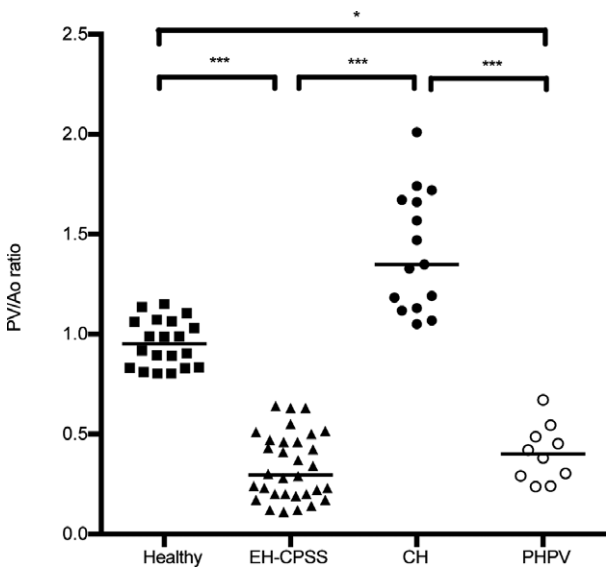

Fig 3. The ratio of the portal vein (PV) to aorta (Ao) in dogs with CH (n = 15), PHPV (n = 10), or EH-CPSS (n = 32) and in healthy dogs (n = 20). Symbols: ■ healthy dogs; ▲ EH-CPSS; ● CH; ○ PHPV. The lines indicate the median values. The PV/Ao ratio did not differ between dogs with CH and healthy dogs, but was significantly reduced in dogs with PHPV when compared with healthy dogs and dogs with CH as well as in those with EH-CPSS when compared with healthy dogs and dogs with CH. *$P < .05$, ***$P < .001$. CH, chronic hepatitis; EH-CPSS, extrahepatic congenital portosystemic shunt; PHPV, primary hypoplasia of the portal vein.

congenital hypoplasia of the main PV. We believe that these findings support the reliability of the PV/Ao ratio determined by CPR on MDCT.

Enlargement of the PV in dogs with CH identified angiographically and ultrasonographically is thought to

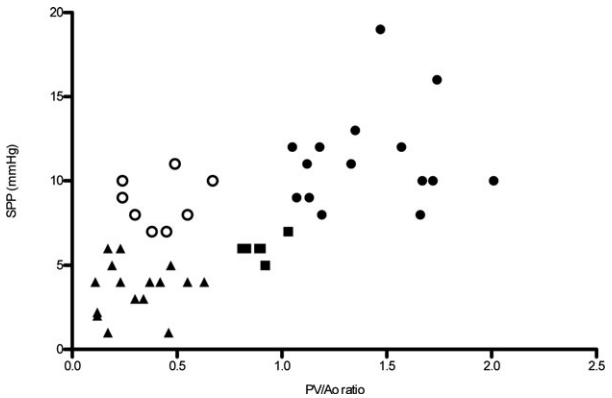

Fig 4. PV/Ao ratio versus SPP in dogs with CH (n = 15), PHPV (n = 8), or EH-CPSS (n = 16) and in healthy dogs (n = 6). The PV/Ao ratio is significantly and positively correlated with SPP in dogs with CH, those with PHPV, and those with EH-CPSS, and in healthy dogs. However, there was no intragroup correlation between SPP and the PV/Ao ratio in the dogs with CH, PHPV, or EH-CPSS, or in the group of healthy dogs. Symbols: ■ healthy dogs; ▲ EH-CPSS; ● CH; ○ PHPV. CH, chronic hepatitis; EH-CPSS, extrahepatic congenital portosystemic shunt; PV/Ao, portal vein/aorta; PHPV, primary hypoplasia of the portal vein; SPP, splenic pulp pressure.

be caused by PH because of increased resistance in the microscopic PV tributaries.[17–19] Although the size of the PV in our dogs with CH tended to be larger than that in the healthy dogs, the difference was not statistically significant. Because all dogs with CH in our study had APSCs, portal flow was diverted into the systemic circulation via the APSCs and the size of the PV might have been decreased when compared with that of the PV before development of APSCs. Additional studies are needed to determine whether the size of the PV could increase in dogs with CH before development of APSCs.

The present study found a decreased PV/Ao ratio in dogs with PHPV, in which hypoplasia of the extrahepatic PV is obvious at the time of surgery.[42] A reduction in the size of the PV has also been identified by ultrasonography in dogs with PHPV including noncirrhotic portal hypertension as classified by the World Small Animal Veterinary Association Liver Standardization Group.[2,17] Therefore, the PV in dogs with PHPV can be affected by congenital hypoplasia of not only the intrahepatic PV tributaries but also the extrahepatic PV, and not increase because portal flow is diverted into the systemic circulation via APSCs. Further investigation is required to define the pathogenesis of this anomaly.

There was a positive correlation between SPP and the PV/Ao ratio in the dogs with CH, PHPV, or EH-CPSS and in the healthy dogs. These results support previous reports indicating that the PV becomes enlarged with an increase in PVP.[17–19] However, there was no intragroup correlation between SPP and the PV/Ao ratio in the dogs with CH, PHPV, or EH-CPSS, or in the group of healthy dogs. The inclusion of groups of dogs with different liver pathologies, that is, CH, PHPV, EH-CPSS, and healthy dogs might have acted as a confounder with regard to whether the positive correlation reported here is valid or not.

Our study has several limitations. First, the PV in dogs with APSCs might be smaller than that before development of APSCs in dogs with CH. Second, the size of the PV in dogs with PHPV before development of APSCs could not be evaluated. Third, given the small size of the study groups, the possibility of type II error cannot be excluded.

In conclusion, it is possible to measure the PV/Ao ratio on MDCT images by CPR. However, further studies are needed to examine the relationship between SPP and the PV/Ao ratio in larger groups of dogs with PH to determine its clinical relevance. Moreover, we believe that it is necessary to take periodic measurements of the PV/Ao ratio and SPP in dogs with CH and in those with PHPV before and after development of APSCs, and both before and after treatment.

Footnotes

[a] Aquilion 16, Toshiba, Tokyo, Japan
[b] Virtual Place, AZE Ltd., Tokyo, Japan
[c] Endoflator, Karl Storz GmbH & Co. KG, Tuttlingen, Germany
[d] Hopkins II, 5 mm, 0°C, Karl Storz GmbH & Co. KG, Tuttlingen, Germany
[e] SR-FF1832; length, 6.4 cm; Terumo, Tokyo, Japan
[f] GraphPad Software Inc., La Jolla, CA

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Conflict of Interest Declaration: Authors declare no conflict of interest.

Off-label Antimicrobial Declaration: Authors declare no off-label use of antimicrobials.

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