Portal Vein/Aorta Ratio in Dogs with Acquired Portosystemic Collaterals

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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-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 = 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. Prehepatic PH is attributable to increased resistance in the extrahepatic portal vein (PV) and is associated with mural or intraluminal obstruction or extraluminal compression. 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. 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. These disorders include chronic hepatitis (CH). Postsinusoidal intrahepatic PH is associated with veno-occlusive disease. 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. The clinical consequences of PH include development of acquired portosystemic collaterals (APSCs) and ascites, or both.

The size of the PV increases in dogs with CH but decreases in dogs with extrahepatic congenital portosystemic shunt (EH-CPSS) because of decreased portal blood flow. Changes in PV size in dogs with these diseases have been evaluated on ultrasound by the PV/aorta (Ao) ratio. 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. It is speculated that formation of a shunt changes portal blood flow. We
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. Images obtained by MDCT also allow high-resolution multiplanar reformation and three-dimensional reconstruction. In humans, the long axes of the hepatobiliary and pancreatic duct systems have been determined by curved planar reformation (CPR) of MDCT images. This technique is used for diagnosis of pancreatic duct tumors and placement of stents in humans. 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 splenography 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 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 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 ducts.

![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.](image-url)
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 CO₂. 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. An 18-gauge over-the-needle cannulae were placed in the abdomen and the abdominal cavity was automatically maintained at 8–10 mmHg. Two cannulae were placed in the abdomen and the abdominal cavity was visualized by a laparoscope. An 18-gauge over-the-needle intravenous catheter 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. 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. 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) 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). 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](image-url) 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.)
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. In contrast, computed tomography usually provides highly detailed anatomic information and the technique is not as operator-dependent as ultrasonography. The CPR obtained from MDCT images has been used clinically to evaluate tortuous ducts and blood vessels in humans. 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]). 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. 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 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...
be caused by PH because of increased resistance in the microscopic PV tributaries. 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. 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. 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-CPS and and in healthy dogs. These results support previous reports indicating that the PV becomes enlarged with an increase in PVP. However, there was no intragroup correlation between SPP and the PV/Ao ratio in the dogs with CH, PHPV, or EH-CPS, and in the group of healthy dogs. The inclusion of groups of dogs with different liver pathologies, that is, CH, PHPV, EH-CPS, 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**

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**References**

1. Buob S, Johnston AN, Webster CRL. Portal hypertension: Pathophysiology, diagnosis, and treatment. J Vet Intern Med 2011;25:169–186.
2. Rothuizen J, Bunch SE, Charles JA, et al., eds. WSAVA Standards for Clinical and Histological Diagnosis of Canine and Feline Liver Disease, 1st ed. Philadelphia, PA: WB Saunders Elsevier; 2006.
3. Johnson SE. Portal hypertension. Part II. Pathophysiology and clinical consequences. Compend Continuing Educ Pract Vet 1987;9:917–925.
4. Respess M, O’Toole TE, Taeymans O, et al. Portal vein thrombosis in 33 dogs: 1998–2011. J Vet Intern Med 2012;26:230–237.

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5. Sztamari V, van den Ingh TS, Fenyves B, Sotonyi R. Portal hypertension in a dog due to circumscribed fibrosis of the wall of the extrahepatic portal vein. Vet Rec 2002;150:602–605.
6. James F, Knowles GW, Mansfield CS, Robertson ID. Ascites due to pre-sinusoidal portal hypertension in dogs: A retrospective analysis of 17 cases. Aust Vet J 2008;86:180–186.
7. van den Ingh TS, Rothuizen J. Hepatoporal fibrosis in three young dogs. Vet Rec 1982;110:575–577.
8. Rutgers HC, Haywood S, Kelly DF. Idiopathic hepatic fibrosis in 15 dogs. Vet Rec 1993;133:115–122.
9. Brown DL, Van Winkle T, Cecere T, et al. Congenital hepatic fibrosis in 5 dogs. Vet Pathol 2010;47:102–107.
10. Bunch SE, Johnson SE, Cullen JM. Idiopathic noncirrhotic portal hypertension in dogs: 33 cases (1982–1998). J Am Vet Med Assoc 2000;218:392–399.
11. DeMarco J, Center SA, Dykes N, et al. A syndrome resembling idiopathic noncirrhotic portal hypertension in 4 young Doberman Pinschers. J Vet Intern Med 1998;12:147–156.
12. Favier RP. Idiopathic hepatitis and cirrhosis in dogs. Vet Clin North Am Small Anim Pract 2009;39:481–512.
13. Hoffman G. Copper associated liver disease. Vet Clin North Am Small Anim Pract 2009;39:489–512.
14. Poldervaart JH, Favier RP, Penning LC, et al. Primary hepatitis in dogs: A retrospective review (2002–2006). J Vet Intern Med 2009;23:72–80.
15. Fredholm D. Multiple acquired extrahepatic portosystemic shunts secondary to veno-occlusive disease in a young German Shepherd. Can Vet J 2009;50:763–766.
16. Sakamoto Y, Sakai M, Watari T. Hepatic and plasma endothelin-1 in dogs with chronic hepatitis. J Vet Intern Med 2017;31:764–769.
17. d’Anjou MA, Penninck D, Cornejo L, Pitbarot P. Ultrasonographic diagnosis of portosystemic shunting in dogs and cats. Vet Radiol Ultrasound 2004;45:424–437.
18. Boothe HW, Howe LM, Edwards JF, Slater MR. Multiple extrahepatic shunts in dogs: 30 cases (1981–1993). J Vet Med Assoc 1996;208:1849–1854.
19. Rand JS, Best SJ, Mathews KA. Portosystemic vascular shunts in a family of American Cocker Spaniels. J Anim Hosp Assoc 1988;24:265–272.
20. d’Anjou MA. The sonographic search for portosystemic shunts. Clin Tech Small Anim Pract 2007;22:104–114.
21. Wingley RH, Kobde LJ, Park RD, Lebel JL. Ultrasonographic diagnosis of portacaval shunts in young dogs. J Am Vet Med Assoc 1987;191:421–424.
22. Holt DE, Schelling CG, Saunders HM, Orscher RJ. Correlation of ultrasonographic findings with surgical, portographic, and necropsy findings in dogs and cats with portosystemic shunts: 63 cases (1987–1993). J Am Vet Med Assoc 1995;207:1190–1193.
23. Lamb CR. Ultrasonographic diagnosis of congenital portosystemic shunts in dogs: Results of a prospective study. Vet Radiol Ultrasound 1996;37:281–288.
24. Haag K, Rössle M, Ochs A, et al. Correlation of duplex sonography findings and portal pressure in 375 patients with portal hypertension. AJR Am J Roentgenol 1999;172:631–635.
25. Bertolini G, Rolla EC, Zotti A, Caldin M. Three-dimensional multislice helical computed tomography techniques for canine extrahepatic portosystemic shunts assessment. Vet Radiol Ultrasound 2006;47:439–443.
26. King JB, Jones JC, Rossmiehl JH Jr, et al. Effect of multiplanar CT image reformating on surgeon diagnostic performance for localizing thoracolumbar disc extrusions in dogs. J Vet Sci 2009;10:225–232.
27. Kakihara D, Yoshimitsu K, Irie H, et al. Usefulness of the long-axis and short-axis reformatted images of multidetector-row CT in evaluating T-factor of the surgically resected pancreaticobiliary malignancies. Eur J Radiol 2007;63:96–104.
28. Gong JS, Xu JM. Role of curved planar reformations using multidetector spiral CT in diagnosis of pancreatic and peripancreatic diseases. World J Gastroenterol 2004;10:1943–1947.
29. Prokesch RW, Chow LC, Beaulieu CF, et al. Isosattenuating pancreatic adenocarcinoma at multi-detector row CT: Secondary signs. Radiology 2002;224:764–768.
30. Nino-Murcia M, Jeffrey RB Jr, Beaulieu CF, et al. Multidetector CT of the pancreas and bile duct system: Value of curved planar reformations. AJR Am J Roentgenol 2001;176:689–693.
31. Bang BW, Jeong S, Lee DH, et al. Curved planar reformatted images of MDCT for differentiation of biliary stent occlusion in patients with malignant biliary obstruction. AJR Am J Roentgenol 2010;194:1509–1514.
32. Hyodoh H, Katagiri Y, Sasaki T, et al. Creation of individual ideally shaped stents using multi-slice CT: In vitro results from the semi-automatic virtual stent (SAVS) designer. Eur Radiol 2005;15:1623–1628.
33. Steiner JM. Diagnostic laparoscopy. In: Steiner JM, ed. Small Animal Gastroenterology, 1st ed. Hannover, Germany: Verlagsgesellschaft mbH & Co. KG; 2008:89–93.
34. Sakamoto Y, Sakai M, Watari T. Three minimally invasive methods of measuring of portal vein pressure in healthy dogs. J Vet Med Sci 2012;74:1299–1302.
35. Brinkman EL, Biller DS, Armbrust LJ, et al. The clinical utility of the right lateral intercostal ultrasound scan technique in dogs. J Am Anim Hosp Assoc 2007;43:179–186.
36. Zwingenberger AL, Schwarz T. Dual-phase CT angiography of the normal canine portal and hepatic vasculature. Vet Radiol Ultrasound 2004;45:117–124.
37. Frank P, Mahaffey M, Egger C, Cornell KK. Helical computed tomographic portography in ten normal dogs and ten dogs with a portosystemic shunt. Vet Radiol Ultrasound 2003;44:392–400.
38. Jeong Y, Lim C, Oh S, et al. Three-dimensional CT angiography of the canine hepatic vasculature. J Vet Sci 2008;9:407–413.
39. Wehrscheutz M, Gallé G, Wehrscheutz E, et al. Thick curved planar reformation of unenhanced multislice computed tomography demonstrating urolithiasis. Urology 2009;74:528–530.
40. Takase K, Sawamyyra Y, Igarashi K, et al. Demonstration of the artery of Adamkiewicz at multi-detector row helical CT. Radiology 2002;223:39–45.
41. Fukami Y, Ebata T, Yokoyama Y, et al. Diagnostic ability of MDCT to assess right hepatic artery invasion by perihilar cholangiocarcinoma with left-sided predominance. J Hepatobiliary Pancreat Sci 2012;19:179–186.
42. van den Ingh TS, Rothuizen J, Meyer HP. Portal hypertension associated with primary hypoplasia of the portal vein in dogs. Vet Rec 1995;137:424–427.