

Computed tomography-derived liver volume is not detectably lower in dogs with histopathologic findings compatible with primary hypoplasia of the portal vein

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Objective

To compare CT-derived liver volume and noncontrast attenuation variables between dogs with histopathologic findings compatible with primary hypoplasia of the portal vein (PHPV) and dogs without liver disease.

Methods

Medical records were retrospectively reviewed for dogs with abdominal CT and liver histopathology compatible with PHPV. Controls were 121 dogs without liver disease from a published CT reference cohort and were not matched by breed, age, sex, or body weight. Liver volume was evaluated with allometric regression of log-transformed liver volume on log-transformed body weight and group. A body weight-restricted sensitivity analysis was performed.

Results

26 dogs with PHPV-compatible histopathology and 121 controls were analyzed. Affected dogs were substantially lighter than controls (median body weight, 2.13 vs 38.0 kg). In the primary allometric model, the adjusted geometric mean ratio for liver volume in affected dogs versus controls was 0.887 (95% CI, 0.751 to 1.047; $P = .156$). The body weight-restricted sensitivity analysis was similar (0.923; 95% CI, 0.769 to 1.107; $P = .377$). Noncontrast hepatic attenuation did not differ significantly between groups, and qualitative CT abnormalities were uncommon.

Conclusions

After allometric adjustment, CT-derived liver volume was not detectably lower in dogs with PHPV-compatible histopathology. This finding should be interpreted cautiously because groups differed substantially in body weight and closely body weight-matched controls were unavailable.

Clinical Relevance

These findings do not support using absence of CT-detectable microhepatia to exclude PHPV. Computed tomography volumetry should be interpreted with body size, vascular imaging, clinical findings, and histopathology, especially when controls are not closely matched by body weight.

Keywords: computed tomography, dog, hepatic attenuation, portal vein hypoplasia, primary hypoplasia of the portal vein

PPrimary hypoplasia of the portal vein (PHPV) is a congenital microscopic portal vascular disorder characterized histopathologically by small or absent portal venules within portal tracts.^{1,2} Similar histopathologic changes can occur secondary to reduced portal blood flow caused by macroscopic vascular disorders; therefore, PHPV cannot be diagnosed by histopathology alone without considering imaging

and clinical findings.¹ Conditions such as congenital portosystemic shunts, arterioportal fistulae, and portal vein thrombosis or obstruction can produce chronic portal hypoperfusion and may mimic PHPV histologically.^{1,3-9} As a result, diagnosis of PHPV requires an integrated approach that confirms compatible histopathologic findings while excluding macroscopic vascular abnormalities that could account for secondary portal hypoperfusion.^{1,2,10}

Computed tomography is well suited for this diagnostic role because it allows cross-sectional evaluation of the hepatic parenchyma and portal venous system. Computed tomography angiography

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has been widely used to identify and characterize congenital portosystemic shunts in dogs and can also help detect arterioportal fistulae and portal vein thrombosis.³⁻⁹ In dogs suspected of PHPV, CT can therefore provide important information by excluding macroscopic vascular causes of portal hypoperfusion. In addition, CT permits evaluation of liver size, hepatic contour, parenchymal attenuation, contrast enhancement patterns, and secondary findings such as acquired portosystemic shunts, peritoneal effusion, and lymphadenomegaly. Despite this potential clinical value, CT features of dogs with histopathologic findings compatible with PHPV have not been well characterized.

Liver size in dogs with PHPV remains incompletely defined. Previous imaging studies and case reports¹¹⁻¹⁵ have described variable subjective findings, with microhepatia reported in some dogs with PHPV or related portal vascular disorders, whereas other reports did not identify reduced liver size. These discrepancies may reflect differences in imaging modality, patient body size and conformation, observer subjectivity, and disease severity. Radiography and ultrasonography can be useful clinically, but assessment of liver size with these modalities is often subjective and may be limited by superimposition, operator dependence, or lack of standardized quantitative thresholds.

Computed tomography hepatic volumetry provides a more objective method for quantifying liver volume. In veterinary medicine, CT-derived liver volume has been evaluated in dogs without liver disease and in dogs with congenital portosystemic shunts.¹⁶⁻²¹ Reference data for CT-based liver volume in dogs without hepatic disease have also been reported.^{16,17,20} These studies support the use of CT volumetry as a reproducible quantitative approach for evaluating canine liver size. However, CT-derived liver volume has not been specifically compared between dogs with histopathologic findings compatible with PHPV and dogs without liver disease.

In addition to liver volume, noncontrast hepatic parenchymal attenuation may provide information about diffuse hepatic composition or parenchymal change. Computed tomography attenuation has been evaluated in dogs with other hepatic or metabolic disorders, including cirrhosis and hepatic steatosis.^{22,23} However, whether hepatic attenuation differs between dogs with PHPV and dogs without liver disease is unknown. Similarly, the frequency of ancillary CT findings such as hepatic parenchymal heterogeneity, nodules or mass-like lesions, acquired portosystemic shunts, peritoneal effusion, and hepatic lymphadenomegaly has not been established in dogs with PHPV.

The purpose of this study was to compare CT-derived liver volume, with explicit consideration of body weight, and noncontrast hepatic attenuation variables between dogs with histopathologic findings compatible with PHPV and dogs without liver disease. Secondary objectives were to describe qualitative CT findings in dogs with PHPV-compatible histopathology and to evaluate the influence of body size on CT-derived liver volume by use of allometric regression.

We hypothesized that CT-derived liver volume would be lower in dogs with PHPV-compatible histopathology after body weight adjustment and that noncontrast hepatic attenuation variables would differ between groups.

Methods

Study design

This retrospective comparative cross-sectional study was designed to compare CT-derived liver volume and noncontrast attenuation variables between dogs with histopathologic findings compatible with PHPV and dogs without liver disease. The primary outcome was body weight-adjusted CT-derived liver volume from the allometric regression model. Secondary quantitative outcomes were liver volume-to-body weight ratio, noncontrast hepatic parenchymal attenuation, splenic parenchymal attenuation, and hepatic-to-splenic attenuation ratio. The hepatic-to-splenic attenuation ratio was considered exploratory because splenic attenuation could be influenced by pre- and perianesthetic drugs. Qualitative CT findings in dogs with PHPV-compatible histopathology were recorded descriptively as secondary observations.

Because the control group was derived from a previously published CT reference cohort and was not matched to the PHPV group by breed, age, sex, or body weight, exploratory allometric analyses were planned to evaluate the influence of body weight on CT-derived liver volume. No a priori sample size calculation was performed because this study used all eligible dogs that met the final case definition during the study period.

Ethics statement

This retrospective study used deidentified clinical imaging and medical record data generated during routine patient care, and no animals underwent additional procedures for research purposes. Therefore, IACUC approval was not required.

PHPV case selection

Medical records from a referral veterinary hospital were retrospectively reviewed to identify dogs with available abdominal CT examinations and liver histopathology reports describing PHPV or PHPV-like hepatic vascular abnormalities between February 12, 2019, and November 4, 2024. Dogs were eligible for inclusion in the PHPV group when the original diagnostic histopathology report included an explicit diagnosis of PHPV or a diagnostic interpretation that the microscopic findings were suspicious for or compatible with PHPV, consistent with published descriptions of PHPV and PHPV-like microscopic portal vascular abnormalities.^{1,2}

Liver tissue was obtained by laparoscopic liver biopsy. At least 2 biopsy specimens were collected from separate liver lobes in each dog and submitted for diagnostic histopathologic examination. Histopathologic classification was based on the original diagnostic reports rather than centralized blinded rereview by a single study pathologist.

Histopathologic features abstracted from the original diagnostic reports included hypoplasia or narrowing of portal or interlobular veins, hypoplasia or narrowing of central veins, hepatic lobular or hepatocellular atrophy, arteriolar proliferation, venous arterIALIZATION, copper staining results when available, and concurrent hepatic lesions. Arteriolar proliferation was recorded but was not required as an isolated inclusion criterion because the case definition was based on the reporting pathologist's integrated diagnostic interpretation, in conjunction with published descriptions of PHPV, rather than on any single microscopic feature.^{1,2}

Dogs were excluded when CT image quality or patient size precluded reliable hepatic volumetry. Dogs were also excluded when the histopathology report did not meet the prespecified PHPV case definition, including reports with only equivocal, minimal, or nonspecific vascular changes or an alternative primary diagnosis. To reduce the risk of including dogs with secondary portal hypoperfusion caused by macroscopic or clinically relevant vascular disorders, multiphasic CT studies were reviewed for congenital portosystemic shunts, portal vein thrombosis, portal venous obstruction, and macroscopic arterioportal or arteriovenous fistulae. Dogs were excluded when any of these abnormalities were identified. Dogs were also excluded when the histopathology report described venous arterIALIZATION or raised concern for arteriovenous shunting.

Control group

The comparison group consisted of 121 dogs without liver disease from a previously published CT reference cohort.²⁰ That cohort was originally assembled to define reference data for CT-derived liver volume in dogs without hepatic disease and included dogs with no clinical history, clinical signs, clinicopathologic abnormalities, or CT findings suggestive of hepatobiliary disease. The control cohort was reused in the present study as a previously assembled reference population rather than as a contemporaneously enrolled, breed-, sex-, age-, or body weight-matched control group. Control data

used in the present analysis included body weight, CT-derived liver volume, noncontrast hepatic attenuation measurements, and splenic attenuation measurements when available.

Key demographic characteristics of the PHPV and control groups are summarized in **Table 1**.

CT image acquisition

All CT examinations in the PHPV group were performed with the same multidetector CT scanner (Revolution ACT; GE Healthcare Japan Inc). Dogs were positioned in sternal recumbency under general anesthesia or sedation. The CT protocol included helical acquisition with the following parameters: 120 kVp, 250 to 300 mA, pitch of 1, slice thickness of 1.25 mm, and matrix size of 512 X 512. Computed tomography image quality, including respiratory motion, was reviewed before measurement, and dogs were excluded when image quality precluded reliable hepatic volumetry. Images were acquired before and after IV administration of iodinated contrast medium (iohexol at 300 mg I/mL; Omnipaque; GE Healthcare) at a dose of 2 mL/kg and an injection rate of 3 mL/s with a power injector. Postcontrast imaging included arterial, portal venous, and delayed phase acquisitions. Portal venous phase images were acquired approximately 40 to 60 seconds after contrast administration. Images were reconstructed with a standard soft-tissue algorithm and reviewed in DICOM format with image-viewing software (Horos 64-bit, version 3.3.6; Horos Project).

Control CT examinations were acquired as part of the previously published reference cohort with a 64-slice multidetector CT scanner (LightSpeed VCT; GE Medical Systems Inc). In that cohort, abdominal CT examinations were performed between June 2, 2020, and July 25, 2022, and acquisition parameters included helical acquisition at 120 kVp, 280 mA, pitch of 1, and slice thickness of 1.25 to 3.75 mm. Only noncontrast images were used for CT hepatic volumetry and attenuation analysis in the present comparison. Scanner-specific calibration and quality-assurance records at the time of each retrospective scan were not available for all dogs; therefore,

Table 1—Demographic characteristics of 26 dogs with histopathologic findings compatible with primary hypoplasia of the portal vein (PHPV) and 121 control dogs without liver disease from a retrospective study comparing CT-derived liver volume and noncontrast hepatic attenuation.

Variable	Dogs with PHPV-compatible histopathology (n = 26)	Dogs without liver disease (n = 121)
Age at CT (y)	2.17 (0.71–4.96; 0.33–12.42)	7.0 (4.0–9.22; 0.56–15.0)
Body weight (kg)	2.13 (1.81–3.27; 1.32–24.0)	38.0 (27.0–43.4; 2.2–104.3)
Sex/neuter status	5 intact males; 2 castrated males; 14 intact females; 5 spayed females	19 intact males; 70 castrated males; 2 intact females; 30 spayed females
Predominant breeds	Toy Poodle, 8 (30.8%); Chihuahua, 5 (19.2%); Pomeranian, 3 (11.5%); Maltese, 2 (7.7%); mixed-breed dog, 2 (7.7%); other breeds, 6 (23.1%)	Mixed-breed dog, 24 (19.8%); German Shepherd Dog, 16 (13.2%); Golden Retriever, 11 (9.1%); Labrador Retriever, 7 (5.8%); Goldendoodle, 6 (5.0%); other breeds, 57 (47.1%)
Cohort source	Present PHPV study cohort	Previously published CT reference cohort

Dogs with PHPV-compatible histopathology underwent CT between February 12, 2019, and November 4, 2024. Control dogs were from a previously published CT reference cohort that reviewed abdominal CT examinations performed between June 2, 2020, and July 25, 2022. Values are reported as median (IQR; range) or number (%) unless otherwise indicated. The control group was reused as the comparator population rather than as a contemporaneously enrolled, breed-, age-, sex-, or body weight-matched control group.

scanner-related effects were considered when interpreting attenuation measurements.

Qualitative CT assessment

Computed tomography studies from dogs with PHPV-compatible histopathology were reviewed for hepatic and abdominal findings. Qualitative variables included precontrast hepatic parenchymal heterogeneity, postcontrast hepatic parenchymal heterogeneity, nodules or mass-like hepatic lesions, acquired portosystemic shunts, peritoneal effusion, and hepatic lymphadenomegaly.

Hepatic parenchymal heterogeneity was recorded when nonuniform hepatic attenuation or enhancement was visible relative to adjacent hepatic parenchyma. Nodules or mass-like lesions were recorded when discrete focal hepatic lesions with recognizable margins were identified. Acquired portosystemic shunts were defined as tortuous collateral vessels connecting the portal and systemic venous circulations in the absence of a congenital portosystemic shunt. Peritoneal effusion was recorded when free abdominal fluid was visible. Hepatic lymphadenomegaly was recorded when hepatic, portal, or regional abdominal lymph nodes were considered enlarged on CT. Qualitative CT findings were recorded descriptively only in the PHPV group.

CT hepatic volumetry

Computed tomography hepatic volumetry was performed on noncontrast transverse images with image-viewing software (Horos 64-bit, version 3.3.6; Horos Project). Manual segmentation was performed by a trained investigator who was not blinded to group status under the supervision of a board-certified veterinary radiologist. The liver was segmented from the cranial margin at the diaphragm to the most caudal hepatic margin. Intrahepatic portal and hepatic venous branches contained within the hepatic contour, including visible main intrahepatic branches, were included in the segmented liver volume, whereas the gallbladder, visible interlobar fissures, and vessels outside the hepatic contour were excluded (**Figure 1**).

If the liver was encompassed by 20 or fewer transverse slices, all slices were segmented. If more than 20 slices encompassed the liver, slices were sampled at evenly spaced intervals so that at least 20 slices were included in the volumetric analysis, as previously described.¹⁶ Hepatic volume was calculated with the following formula: liver volume = Σ (each slice area X slice thickness) X (total number of hepatic slices/number of slices used for calculation).

Liver volume was normalized to body weight by dividing total CT-derived liver volume by body weight and was reported as the liver volume-to-body weight ratio in cubic centimeters per kilogram. Control volumetric data were derived from the previously published cohort of dogs without liver disease by use of the same segmentation protocol.²⁰

CT attenuation measurements

Attenuation measurements and qualitative CT assessments were performed by a trained investigator

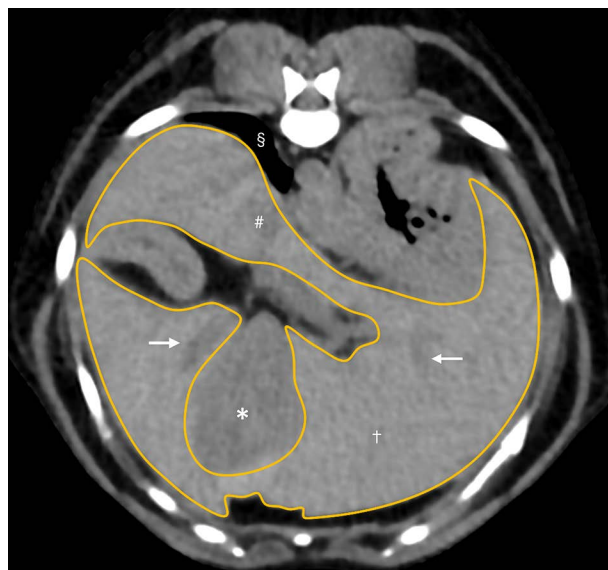


Figure 1—Representative transverse noncontrast CT image from a retrospective study comparing CT-derived liver volume and noncontrast hepatic attenuation in 26 dogs with histopathologic findings compatible with primary hypoplasia of the portal vein and 121 control dogs without liver disease from a previously published reference cohort. Dogs with primary hypoplasia of the portal vein-compatible histopathology underwent CT between February 12, 2019, and November 4, 2024; control dogs were from a reference cohort that reviewed abdominal CT examinations performed between June 2, 2020, and July 25, 2022. The image is displayed in a soft-tissue window (window width, 350 HU; window level, 40 HU) with a 1.25-mm slice thickness. Manual liver segmentation was performed by tracing the hepatic contour as a region of interest (orange outline). Hepatic vessels within the hepatic contour were included in the segmentation (white arrows), whereas the gallbladder, visible interlobar fissures, and hepatic vessels outside the hepatic contour were excluded. Liver parenchyma (†), gallbladder (*), caudal vena cava (#), and pulmonary parenchyma (§) are labeled.

under the supervision of the board-certified veterinary radiologist. Noncontrast hepatic parenchymal attenuation was measured by placing 6 circular or oval regions of interest (ROIs) within the hepatic parenchyma. Four ROIs were placed at the level of or caudal to the left hepatic vein to sample the dorsal right, ventral right, dorsal left, and ventral left portions of the liver. Two additional ROIs were placed cranial to the right kidney, including 1 ROI in the caudate process and 1 ROI in the caudoventral mid-to-left liver. The ROIs were drawn as large as possible while avoiding visible vessels, bile ducts, visible artifacts, focal lesions, and organ margins. Mean hepatic parenchymal attenuation was calculated as the unweighted arithmetic mean of the available ROI-level mean HU values. The whole-liver volumetric segmentation was not used to calculate attenuation because it included intrahepatic vessels and peripheral partial-volume voxels, whereas the attenuation measurement was intended to represent hepatic parenchyma.

Splenic parenchymal attenuation was measured on noncontrast images by placing 3 ROIs in the

dorsal extremity, central body, and ventral extremity of the spleen while avoiding vessels and visible lesions. Mean splenic parenchymal attenuation was calculated as the unweighted arithmetic mean of the 3 splenic ROI-level mean HU values. The hepatic-to-splenic attenuation ratio was calculated for each dog as mean hepatic parenchymal attenuation divided by mean splenic parenchymal attenuation.

Splenic attenuation was measured concurrently so that a spleen-referenced attenuation index could be evaluated in parallel with absolute hepatic attenuation values. In dogs without spleen measurements, splenic parenchymal attenuation and the hepatic-to-splenic attenuation ratio were treated as missing, whereas hepatic parenchymal attenuation was retained.

Statistical analysis

Descriptive statistics were calculated for all variables. Continuous variables were summarized as median, IQR, and range because several variables were not normally distributed and because body weight varied widely among dogs. Categorical variables were summarized as counts and percentages.

Mean hepatic parenchymal attenuation was calculated as the unweighted arithmetic mean of available noncontrast hepatic ROI-level mean HU values. Mean splenic parenchymal attenuation was calculated as the unweighted arithmetic mean of available splenic ROI-level mean HU values. The hepatic-to-splenic attenuation ratio was calculated as mean hepatic parenchymal attenuation divided by mean splenic parenchymal attenuation and was considered an exploratory secondary variable. Dogs without available splenic attenuation measurements were excluded from analyses of splenic attenuation and hepatic-to-splenic attenuation ratio but were retained in analyses of liver volume and hepatic attenuation.

Unadjusted group comparisons between dogs with PHPV-compatible histopathology and dogs without liver disease were performed with Wilcoxon rank-sum tests for liver volume-to-body weight ratio, noncontrast hepatic parenchymal attenuation, splenic parenchymal attenuation, and hepatic-to-splenic attenuation ratio.

Because the PHPV and control groups differed substantially in body weight distribution, allometric regression was performed. Computed tomography-derived liver volume and body weight were natural log transformed ($\log[\text{LV}]$ and $\log[\text{BW}]$). A linear regression model was first fitted with $\log(\text{LV})$ as the dependent variable and $\log(\text{BW})$, group, and a group-by-log body weight interaction as independent variables. When the interaction term was not significant, the final model included $\log(\text{BW})$ and group without an interaction term. The final model was expressed as $\log(\text{LV}) = \beta_0 + \beta_1 \times \log(\text{BW}) + \beta_2 \times \text{group}$, where group was coded as 0 for control dogs and 1 for dogs with histopathologic findings compatible with PHPV. The exponent β_1 described the allometric relationship between body weight and liver volume, and exponent β_2 was interpreted as the adjusted geometric mean ratio for affected dogs relative to control dogs at the same body weight.

To address the limited overlap in body weight between groups, a body weight-restricted sensitivity analysis was performed after restricting the dataset to dogs within the body weight range observed in the affected group. A second sensitivity analysis repeated the allometric model after restricting the PHPV group to dogs with documented arteriolar proliferation while retaining the full control cohort. These sensitivity analyses were considered exploratory.

Values of $P < .05$ were considered significant. Because attenuation comparisons and sensitivity analyses were secondary or exploratory, P values were not adjusted for multiplicity. Statistical analyses were performed with R Statistical Software, version 4.4.1 (The R Foundation).

Results

Case selection and clinical characteristics

Thirty-four dogs with liver histopathology reports describing PHPV or PHPV-like hepatic vascular abnormalities and available abdominal CT examinations were initially reviewed. One dog was excluded because hepatic volumetry could not be performed owing to small body size. Five dogs were excluded because the histopathology report did not meet the final prespecified PHPV case definition, and 2 dogs were excluded because venous arterialization or possible arteriovenous shunting was described histopathologically. The final PHPV cohort included 26 dogs.

The PHPV cohort consisted predominantly of toy- and small-breed dogs. Breeds represented were Toy Poodle ($n = 8$), Chihuahua (5), Pomeranian (3), Maltese (2), mixed-breed dog (2), and 1 each of Bernese Mountain Dog, Boston Terrier, French Bulldog, Miniature Dachshund, Papillon, and Yorkshire Terrier. The cohort included 5 intact males, 2 castrated males, 14 intact females, and 5 spayed females. Median age at CT was 2.17 years (IQR, 0.71 to 4.96 years; range, 0.33 to 12.42 years), and median body weight was 2.13 kg (IQR, 1.81 to 3.27 kg; range, 1.32 to 24.0 kg).

The control group consisted of 121 dogs without liver disease from a previously published CT reference cohort. The control cohort was not matched to the PHPV cohort by breed, age, sex, or body weight. Median body weight was 38.0 kg (IQR, 27.0 to 43.4 kg; range, 2.2 to 104.3 kg) in the control group.

CT hepatic volumetry

In dogs with PHPV-compatible histopathology, median CT-derived liver volume was 58.8 cm³ (IQR, 51.5 to 78.6 cm³; range, 36.8 to 616.2 cm³), and median liver volume-to-body weight ratio was 27.4 cm³/kg (IQR, 24.6 to 29.3 cm³/kg; range, 19.0 to 34.3 cm³/kg). In dogs without liver disease, median CT-derived liver volume was 774.8 cm³ (IQR, 597.3 to 979.9 cm³; range, 66.2 to 2,174.6 cm³), and median liver volume-to-body weight ratio was 22.0 cm³/kg (IQR, 19.1 to 25.3 cm³/kg; range, 11.1 to 43.7 cm³/kg). The unadjusted liver volume-to-body weight ratio was higher in dogs with PHPV-compatible histopathology than in control dogs ($P < .001$; **Table 2**).

Table 2—Quantitative CT variables and allometric analyses in the same dogs as in Table 1.

Variable	Dogs with PHPV-compatible histopathology	Dogs without liver disease	Effect estimate	P value
No. of dogs	26	121	—	—
CT-derived liver volume (cm ³)	58.8 (51.5–78.6; 36.8–616.2)	774.8 (597.3–979.9; 66.2–2,174.6)	—	—
Liver volume-to-body weight ratio (cm ³ /kg)	27.4 (24.6–29.3; 19.0–34.3)	22.0 (19.1–25.3; 11.1–43.7)	—	< .001
Noncontrast hepatic parenchymal attenuation (HU)	60.6 (56.0–64.0; 48.9–74.6)	58.8 (55.0–63.0; 44.0–74.0)	—	.501
Splenic parenchymal attenuation (HU)	61.0 (59.7–64.1; 56.0–69.8)	65.0 (61.0–67.5; 45.2–75.6)*	—	.010
Hepatic-to-splenic attenuation ratio	0.96 (0.92–1.01; 0.81–1.20)	0.93 (0.84–0.99; 0.70–1.34)*	—	.024
Group-by-log body weight interaction in allometric model	—	—	—	.140
Body weight-adjusted CT-derived liver volume, PHPV vs control	—	—	Adjusted geometric mean ratio, 0.887; 95% CI, 0.751–1.047	.156
Body weight-restricted sensitivity analysis within the PHPV body weight range	26	23	Adjusted geometric mean ratio, 0.923; 95% CI, 0.769–1.107	.377
Sensitivity analysis restricted to PHPV dogs with documented arteriolar proliferation	23	121	Adjusted geometric mean ratio, 0.886; 95% CI, 0.749–1.049	.158

Values for quantitative CT variables are reported as median (IQR; range). *P* values for unadjusted quantitative group comparisons were calculated with Wilcoxon rank-sum tests. The allometric model used log-transformed CT-derived liver volume as the dependent variable and log-transformed body weight and group as independent variables. Effect estimates for allometric models are exponentiated coefficients and are reported as adjusted geometric mean ratios for dogs with primary hypoplasia of the portal vein (PHPV)-compatible histopathology relative to control dogs. Values less than 1 indicate lower body weight-adjusted CT-derived liver volume in dogs with PHPV-compatible histopathology. The hepatic-to-splenic attenuation ratio was considered exploratory.

— = Not applicable.

*Splenic parenchymal attenuation and hepatic-to-splenic attenuation ratio were available for 119 control dogs because splenic measurements were unavailable in 2 dogs due to previous splenectomy.

In the primary allometric model using natural logarithms, log-transformed CT-derived liver volume was modeled as a function of log-transformed body weight and group. The final model was $\log(LV) = 3.5367 + 0.8741 \times \log(BW) - 0.1199 \times \text{group}$, where group was coded as 0 for control dogs and 1 for dogs with histopathologic findings compatible with PHPV (**Figure 2**). On the original scale, the corresponding equations were $LV = 34.35 \times BW^{0.874}$ for control dogs and $LV = 30.47 \times BW^{0.874}$, where LV is CT-derived liver volume and BW is body weight for affected dogs. The adjusted geometric mean ratio for affected dogs relative to control dogs was 0.887 (95% CI, 0.751 to 1.047; *P* = .156). No clear group-by-log body weight interaction was detected (*P* = .140); therefore, the final primary model used a common allometric slope for both groups. Because body weight distributions differed substantially between groups, a body weight-restricted sensitivity analysis was performed by restricting the dataset to dogs within the body weight range observed in the affected group. This analysis included 23 control dogs and 26 affected dogs and yielded an adjusted geometric mean ratio of 0.923 (95% CI, 0.769 to 1.107; *P* = .377), which was consistent with the primary analysis. In a second sensitivity analysis restricted to dogs with PHPV-compatible histopathology and documented arteriolar proliferation, the PHPV subgroup included 23 dogs, and the adjusted geometric mean ratio was 0.886 (95% CI, 0.749 to 1.049; *P* = .158).

CT attenuation

Median noncontrast hepatic parenchymal attenuation did not differ significantly between dogs with PHPV-compatible histopathology and control dogs (60.6 HU [IQR, 56.0 to 64.0 HU; range, 48.9 to 74.6 HU] vs 58.8 HU [IQR, 55.0 to 63.0 HU; range, 44.0 to 74.0 HU]; *P* = .501). Median splenic parenchymal attenuation was lower in dogs with PHPV-compatible histopathology than in control dogs (61.0 HU [IQR, 59.7 to 64.1 HU; range, 56.0 to 69.8 HU] vs 65.0 HU [IQR, 61.0 to 67.5 HU; range, 45.2 to 75.6 HU]; *P* = .010). The hepatic-to-splenic attenuation ratio was treated as an exploratory secondary variable and was higher in dogs with PHPV-compatible histopathology than in control dogs (0.96 [IQR, 0.92 to 1.01; range, 0.81 to 1.20] vs 0.93 [IQR, 0.84 to 0.99; range, 0.70 to 1.34]; *P* = .024). Splenic parenchymal attenuation and hepatic-to-splenic attenuation ratio were available for 119 control dogs because splenic measurements were unavailable in 2 dogs due to previous splenectomy.

Qualitative CT findings

Qualitative CT abnormalities were uncommon in dogs with PHPV. Precontrast hepatic parenchymal heterogeneity was not identified in any dog. Postcontrast hepatic parenchymal heterogeneity was identified in 1 of 26 dogs (**Table 3**). Nodules or mass-like hepatic lesions, acquired portosystemic shunts, and peritoneal effusion were not identified in any dog. Hepatic lymphadenomegaly was identified in 1 of 26 dogs.

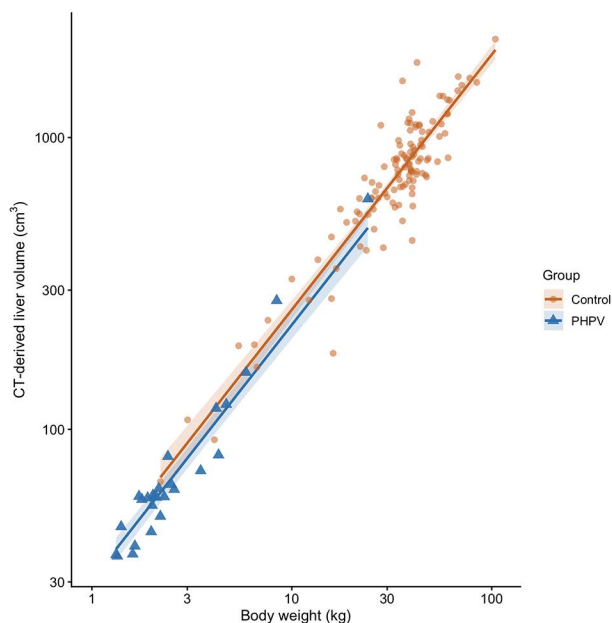


Figure 2—Relationship between body weight and CT-derived liver volume in dogs with histopathologic findings compatible with primary hypoplasia of the portal vein (PHPV) and dogs without liver disease. Each point represents 1 dog. Both axes are displayed on logarithmic scales. Lines represent fitted values from the final allometric regression model, $\log(LV) = 3.5367 + 0.8741 \times \log(BW) - 0.1199 \times \text{group}$, where LV is CT-derived liver volume, BW is body weight, and group was coded as 0 for control dogs and 1 for dogs with PHPV-compatible histopathology. On the original scale, the fitted equations were $LV = 34.35 \times BW^{0.874}$ for control dogs and $LV = 30.47 \times BW^{0.874}$ for affected dogs. Shaded bands indicate 95% CIs for fitted mean values and are shown only across the observed body weight range within each group. The plot demonstrates the marked difference in body weight distribution between groups and supports interpretation of CT-derived liver volume in relation to body size rather than by liver volume-to-body weight ratio alone.

Table 3—Qualitative CT findings in the 26 dogs with histopathologic findings compatible with primary hypoplasia of the portal vein (PHPV) described in Table 1.

Qualitative CT finding	Dogs with PHPV-compatible histopathology (n = 26)
Precontrast hepatic parenchymal heterogeneity	0/26 (0%)
Postcontrast hepatic parenchymal heterogeneity	1/26 (3.8%)
Nodules or mass-like hepatic lesions	0/26 (0%)
Acquired portosystemic shunts	0/26 (0%)
Peritoneal effusion	0/26 (0%)
Hepatic lymphadenomegaly	1/26 (3.8%)

Qualitative findings were recorded descriptively only in dogs with PHPV-compatible histopathology. Data are reported as number/total number (%).

Discussion

In this retrospective cohort, CT-derived liver volume was not detectably lower in dogs with histopathologic findings compatible with PHPV after allometric adjustment for body weight. The estimated adjusted liver volume in affected dogs was approximately 11% lower than that in control dogs, but the 95% CI included no difference, and a body weight-restricted sensitivity analysis produced a similar result. When liver volume was normalized by body weight alone, dogs with PHPV-compatible histopathology had a higher liver volume-to-body weight ratio than control dogs; however, this unadjusted result should not be interpreted as evidence that PHPV is associated with hepatomegaly. The PHPV cohort consisted predominantly of toy- and small-breed dogs, whereas the control cohort included many medium- and large-breed dogs. Therefore, the higher unadjusted ratio in the PHPV group was likely driven at least in part by between-group differences in body weight distribution rather than by a disease-specific increase in liver volume.

Allometric analysis provided a more cautious interpretation of the volumetric findings. The fitted allometric exponent was 0.874, indicating a strong relationship between body weight and CT-derived liver volume and supporting a body weight-adjusted interpretation. The group-by-log body weight interaction was not significant, and the body weight-restricted sensitivity analysis yielded an adjusted geometric mean ratio similar to that of the primary analysis. Nevertheless, the comparison still depended substantially on the fitted allometric relationship because the available control cohort contained few dogs in the body weight range represented by most affected dogs. These findings suggest that simple liver volume-to-body weight ratios may be misleading when applied across dogs with markedly different body sizes and that allometric adjustment is important when CT hepatic volumetry is used in heterogeneous canine populations. Because the adjusted geometric mean ratios were less than 1 but the CIs included 1, the findings do not demonstrate a clear reduction in body weight-adjusted liver volume and should not be interpreted as definitive evidence of normal liver volume.

These results help reconcile previous variable descriptions of liver size in dogs with PHPV or related microscopic portal vascular disorders. Microhepatia has been described subjectively in some dogs with portal vascular disorders, whereas other studies have not consistently identified reduced liver size.^{11,13,15} Differences among studies may reflect variation in imaging modality, observer subjectivity, patient body size, breed conformation, and disease severity. Computed tomography hepatic volumetry provides a more objective method for assessing liver size than subjective radiographic or ultrasonographic evaluation and has been used in dogs without liver disease and in dogs with congenital portosystemic shunts.^{16–21} The present study extends this approach to dogs with histopathologic findings compatible with PHPV but indicates that body size imbalance

can substantially affect interpretation. A body size-matched control cohort, particularly one enriched for healthy toy- and small-breed dogs, would be needed to determine whether PHPV is associated with a consistent volumetric phenotype.

The attenuation findings were modest and should be interpreted cautiously. Absolute noncontrast hepatic parenchymal attenuation did not differ significantly between dogs with PHPV-compatible histopathology and control dogs, suggesting that diffuse hepatic hypoattenuation or hyperattenuation was not a consistent CT feature of this cohort. Splenic attenuation was lower, and the exploratory hepatic-to-splenic attenuation ratio was higher in dogs with PHPV-compatible histopathology; however, these findings should not be interpreted as primary evidence of a disease-related hepatic attenuation difference. Because hepatic attenuation itself was not significantly different, the higher hepatic-to-splenic ratio may have been influenced primarily by lower splenic attenuation rather than by a primary hepatic parenchymal difference. In addition, CT attenuation values can be affected by scanner platform, acquisition and reconstruction parameters, object size, patient positioning, ROI placement,²⁴⁻²⁷ and pre- or perianesthetic effects on the spleen.²⁸ Therefore, neither absolute hepatic attenuation nor the hepatic-to-splenic attenuation ratio should be considered a standalone CT marker for PHPV. These variables should be interpreted only in combination with vascular imaging, clinical findings, and histopathologic assessment.

Qualitative CT abnormalities were uncommon in this cohort. Acquired portosystemic shunts, peritoneal effusion, and nodules or mass-like hepatic lesions were not identified in any dog, and post-contrast hepatic heterogeneity and hepatic lymphadenomegaly were each identified in only 1 dog. These findings suggest that dogs in this cohort did not have CT evidence of advanced portal hypertension or marked secondary hepatic remodeling. This is clinically relevant because PHPV and secondary portal hypoperfusion can overlap histologically, and macroscopic vascular disorders such as congenital portosystemic shunts, arterioportal fistulae, portal vein thrombosis, and portal venous obstruction can produce histopathologic changes that resemble PHPV.^{1,3-9} Computed tomography remains valuable in dogs suspected of having PHPV because it can help exclude these macroscopic vascular abnormalities, even when volumetric and attenuation metrics are not independently diagnostic.

The principal limitation of this study was the marked difference in body weight distribution between affected and control dogs. Most affected dogs were small, whereas the previously published reference cohort included many medium- and large-breed dogs. Therefore, the comparison depended substantially on allometric adjustment for body weight. Although the group-by-log body weight interaction was not significant and the body weight-restricted sensitivity analysis produced results consistent with the primary model, the available control

cohort contained few dogs in the body weight range represented by most affected dogs. Formal body weight matching was therefore not feasible without creating a very small and potentially unstable control sample. Consequently, these findings should not be interpreted as definitive evidence that liver volume is normal in dogs with histopathologic findings compatible with PHPV. Rather, CT-derived liver volume was not detectably lower in this cohort after body weight adjustment. A second important limitation was the case definition for PHPV. Histopathologic classification was based on original diagnostic reports rather than centralized blinded rereview by a single veterinary pathologist. Reports were generated by multiple pathologists and laboratories, and the diagnosis of PHPV or PHPV-compatible change may have been influenced by differences in reporting style and diagnostic threshold. Arteriolar proliferation was not required for inclusion because the final case definition was based on the reporting pathologist's integrated interpretation rather than on any single microscopic feature. However, this approach may have introduced diagnostic misclassification. To reduce this risk, dogs with equivocal, minimal, or nonspecific vascular changes that did not meet the prespecified case definition were excluded, and a sensitivity analysis was performed in the subgroup with documented arteriolar proliferation. Dogs with venous arterialization or histopathologic concern for arteriovenous shunting were also excluded to reduce the likelihood of including dogs with secondary portal hypoperfusion. Nevertheless, PHPV cannot be diagnosed by histopathology alone without integration of imaging and clinical findings, and some degree of diagnostic uncertainty remains inherent in retrospective studies of this condition.^{1,2}

Additional limitations should be considered. Liver biopsy specimens were obtained clinically, and the severity and distribution of microscopic portal venous lesions were not quantitatively graded for correlation with CT findings. Portal pressure measurements and long-term clinical outcomes were not available, so CT variables could not be related to hemodynamic severity or prognosis. Computed tomography attenuation comparisons may also have been influenced by differences in scanner platform, calibration status, acquisition conditions, reconstruction settings, scanner age, object size, patient positioning, and artifacts between cohorts. Although a consistent ROI placement scheme was used, original ROI areas or voxel counts were not available for voxel-weighted recalculation, and retrospective attenuation measurements cannot fully eliminate technical variability. Pre- and perianesthetic drug protocols were not standardized and may have affected splenic size or attenuation, thereby limiting interpretation of splenic attenuation and hepatic-to-splenic attenuation ratios. Respiratory motion was assessed as part of image-quality review, but respiratory control was not prospectively standardized or separately graded. Finally, the number of dogs with PHPV remained small, especially for sensitivity analyses, and the study may have been underpowered to

detect modest differences in body weight-adjusted liver volume.

This study did not compare postcontrast hepatic attenuation or enhancement patterns between affected and control dogs because the control cohort did not have a standardized timed postcontrast acquisition protocol comparable with that used in affected dogs. Future studies with matched controls and standardized multiphasic CT protocols would be needed to determine whether PHPV is associated with altered hepatic enhancement.

In conclusion, CT-derived liver volume was not detectably lower in dogs with histopathologic findings compatible with PHPV after allometric adjustment for body weight, despite a higher unadjusted liver volume-to-body weight ratio. This finding should be interpreted cautiously because the affected and control groups differed substantially in body weight and closely body weight-matched controls were not available. Noncontrast hepatic attenuation also was not detectably different between groups. The exploratory hepatic-to-splenic attenuation ratio was higher in affected dogs, but this finding may have been influenced by lower splenic attenuation and nonstandardized anesthetic or sedative protocols. Computed tomography volumetry and attenuation indices should therefore be interpreted in conjunction with body size, CT assessment of macroscopic vascular abnormalities, clinical findings, and histopathologic interpretation in dogs suspected of having PHPV.

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