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DOPPLER ULTRASONOGRAPHIC ASSESSMENT OF RENAL AND HEPATIC HEMODYNAMICS IN DOGS WITH CONGESTIVE HEART FAILURE DURING STANDARD THERAPY

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Congestive heart failure in dogs is one of the relevant problems of modern veterinary cardiology, as it is accompanied not only by impaired intracardiac hemodynamics but also by secondary changes in target organs, primarily the kidneys and liver. In clinical practice, the main focus is often placed on cardiac assessment, whereas early hemodynamic disturbances of renal and hepatic perfusion may remain insufficiently detected, especially in the absence of pronounced changes in blood biochemical parameters. This complicates the timely diagnosis of cardiorenal and cardiohepatic disorders and may affect the choice of therapeutic strategy and prognosis.

This study presents the results of a comprehensive assessment of echocardiographic, renal Doppler, and hepatic Doppler ultrasonographic parameters in dogs with congestive heart failure. The study included 40 dogs aged 6 to 12 years, which were divided into two groups: an experimental group of 20 dogs with ACVIM stages C and D heart failure and a control group of 20 clinically healthy animals. In dogs with congestive heart failure, before treatment initiation, a decrease in ejection fraction and fractional shortening, as well as an increase in the LA/Ao ratio, was observed, indicating impaired myocardial systolic function, dilation of the left-sided cardiac chambers, and hemodynamic overload. Renal Doppler ultrasonography revealed increased resistive index and pulsatility index values in both kidneys, indicating increased vascular resistance and impaired renal perfusion associated with heart failure. Assessment of hepatic hemodynamics showed that portal vein blood flow velocity did not differ significantly from the control group, whereas the hepatic vein S/D ratio was significantly lower and the hepatic arterial resistive index was higher in dogs with CHF. These findings indicated impaired hepatic venous outflow and increased vascular resistance within the hepatic arterial circulation. After standard therapy, which included pimobendan, furosemide, and amlodipine, gradual improvement in echocardiographic parameters, a decrease in renal RI and PI values, and an increase in the hepatic vein S/D ratio were observed, especially at week 4 of treatment. The obtained results confirm the feasibility of using renal and hepatic Doppler ultrasonography as additional non-invasive methods for monitoring dogs with congestive heart failure, early detection of cardiorenal and cardiohepatic hemodynamic disorders, and assessment of treatment efficacy.

Keywords: dogs, congestive heart failure, echocardiography, Doppler

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ultrasonography, renal perfusion, hepatic hemodynamics, resistive index, pulsatility index, hepatic vein S/D ratio, cardiorenal syndrome, cardiohepatic disorders.

ДОПЛЕРОГРАФІЧНА ОЦІНКА НИРКОВОЇ ТА ПЕЧІНКОВОЇ ГЕМОДИНАМІКИ У СОБАК ІЗ ЗАСТІЙНОЮ СЕРЦЕВОЮ НЕДОСТАТНІСТЮ НА ФОНІ СТАНДАРТНОЇ ТЕРАПІЇ

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Застійна серцева недостатність у собак є однією з актуальних проблем сучасної ветеринарної кардіології, оскільки супроводжується не лише порушенням внутрішньосерцевої гемодинаміки, але й вторинними змінами з боку органів-мішеней, насамперед нирок і печінки. У клінічній практиці основну увагу часто приділяють оцінці серця, тоді як ранні гемодинамічні порушення ниркової та печінкової перфузії можуть залишатися недостатньо виявленими, особливо за відсутності виражених змін біохімічних показників крові. Це ускладнює своєчасну діагностику кардіоренальних і кардіогепатичних порушень та може впливати на вибір терапевтичної тактики й прогноз.

У роботі наведено результати комплексної оцінки ехокардіографічних, ниркових і печінкових доплерографічних показників у собак із застійною серцевою недостатністю. Дослідження проводили на 40 собаках віком від 6 до 12 років, яких було розподілено на дві групи: дослідну групу з 20 собак із серцевою недостатністю категорій C та D за класифікацією ACVIM і контрольну групу з 20 клінічно здорових тварин. У собак із застійною серцевою недостатністю до початку лікування встановлено зниження фракції викиду та фракційного вкорочення, а також збільшення співвідношення LA/Ao, що свідчило про порушення систолічної функції міокарда, дилатацію лівих відділів серця та гемодинамічне перевантаження. Доплерографічне дослідження нирок показало підвищення індексу резистентності та пульсаційного індексу в обох нирках, що вказувало на зростання судинного опору та порушення ниркової перфузії на фоні серцевої недостатності. Під час оцінки печінкової гемодинаміки встановлено, що швидкість кровотоку у ворітній вені істотно не відрізнялася від показників контрольної групи, тоді як співвідношення S/D печінкової вени було достовірно нижчим, а печінковий артеріальний індекс резистентності — вищим у собак із ХСН. Це свідчило про порушення печінкового венозного відтоку та підвищення судинного опору в печінковому артеріальному руслі. Після проведення стандартної терапії, що включала пімобендан, фуросемід та амлодипін, спостерігалося поступове покращення ехокардіографічних показників, зниження ниркових RI та PI, а також підвищення співвідношення S/D печінкової вени, особливо на 4-й

тиждень лікування. Отримані результати підтверджують доцільність використання ниркової та печінкової доплерографії як додаткових неінвазивних методів моніторингу собак із застійною серцевою недостатністю, раннього виявлення кардіоренальних і кардіогепатичних гемодинамічних порушень та контролю ефективності терапії.

Ключові слова: собаки, застійна серцева недостатність, ехокардіографія, доплерографія, ниркова перфузія, печінкова гемодинаміка, індекс резистентності, пульсаційний індекс, S/D печінкової вени, кардіоренальний синдром, кардіогепатичні порушення.

Introduction. Congestive heart failure (CHF) is one of the most common cardiovascular disorders in dogs and represents a major cause of morbidity and mortality in small animal practice (Keene et al., 2019; Kittleson & Côté, 2021). The progression of CHF is associated not only with impaired intracardiac hemodynamics but also with systemic neurohumoral activation, venous congestion, reduced tissue perfusion, and dysfunction of target organs, particularly the kidneys and liver (Orvalho & Cowgill, 2017; Zamoshnikov, 2024). These pathophysiological mechanisms may significantly affect the clinical status, prognosis, and quality of life of affected animals.

In veterinary cardiology, increasing attention has recently been directed toward the cardiorenal syndrome, which describes the bidirectional relationship between cardiac and renal dysfunction (Orvalho & Cowgill, 2017; Todorov & Zamoshnikov, 2024). Chronic heart disease may lead to decreased renal perfusion, activation of the renin–angiotensin–aldosterone system, increased vascular resistance, and progressive renal impairment even before overt biochemical abnormalities become evident (Szczepankiewicz et al., 2021; Agut et al., 2020). Moreover, chronic kidney disease itself may contribute to cardiovascular remodeling and progression of heart failure (Zamoshnikov, 2024). Doppler ultrasonography has therefore become an important non-invasive diagnostic method for evaluating renal blood flow and renal vascular resistance in dogs with cardiovascular disorders (Morita et al., 2025).

In addition to renal alterations, hepatic circulation may also be significantly affected in dogs with CHF due to increased central venous pressure and systemic venous congestion (Esin & Güzel, 2025). The liver is particularly sensitive to hemodynamic disturbances because hepatic veins directly communicate with the caudal vena cava and right atrium (Kim et al., 2017). Increased right-sided cardiac pressure may impair hepatic venous drainage and alter hepatic blood flow patterns, potentially resulting in cardiohepatic syndrome and congestive hepatopathy (Silva et al., 2022).

Previous studies demonstrated that Doppler ultrasonographic evaluation of hepatic veins may reflect the severity of tricuspid regurgitation and right-sided cardiac overload in dogs (Kim et al., 2017). In healthy dogs, hepatic venous Doppler patterns correlate with structural and functional variables of the right ventricle (Silva et al., 2020). Furthermore, hepatic venous Doppler abnormalities have been reported in dogs with pulmonary hypertension, suggesting that hepatic venous hemodynamics may provide additional information regarding systemic venous congestion and right ventricular dysfunction (Silva et al., 2022). Recent investigations have also shown that ultrasonographic assessment of the caudal vena cava and venous circulation may be useful for evaluating volume overload and venous hypertension in dogs with CHF (Chou et al., 2021; Cambournac et al., 2026).

Echocardiography remains the primary diagnostic tool for the assessment of cardiac structure and function in dogs with CHF (Hoque et al., 2019). In dogs with

myxomatous mitral valve disease, echocardiographic parameters are essential for Standard therapy, including pimobendan, diuretics, angiotensin-converting enzyme inhibitors, and antihypertensive drugs, may improve survival, quality of life, and clinical stabilization in dogs with CHF, although progression of secondary cardiorenal and cardiohepatic alterations may still occur despite treatment (Boswood et al., 2016; DeFrancesco et al., 2017; Donati et al., 2022). Nevertheless, despite improvements in cardiac function, secondary hemodynamic alterations in target organs may persist and require additional monitoring methods.

Although the use of Doppler ultrasonography in veterinary cardiology is increasing, information regarding combined renal and hepatic Doppler changes in dogs with CHF remains limited. Most previous studies focused either on cardiac evaluation or isolated assessment of renal or hepatic circulation. Data regarding the dynamics of renal and hepatic hemodynamic parameters during standard therapy for CHF are still insufficient. Therefore, further investigation of Doppler ultrasonographic markers may improve early detection of organ perfusion abnormalities and provide additional tools for monitoring treatment efficacy in dogs with congestive heart failure.

The aim of this study was to evaluate echocardiographic, renal Doppler, and hepatic Doppler ultrasonographic changes in dogs with congestive heart failure during standard therapy.

The scientific novelty of this study consists in the integrated evaluation of echocardiographic, renal, and hepatic Doppler ultrasonographic parameters in dogs with congestive heart failure during standard therapy. The study provides a comprehensive assessment of cardiorenal and cardiohepatic hemodynamic interactions and demonstrates the dynamics of renal and hepatic blood flow changes during treatment. Particular attention was paid to hepatic venous and arterial Doppler indices, which remain insufficiently investigated in veterinary cardiology.

Materials and research methods. The study included 40 dogs aged 6 to 12 years. The animals were divided into two groups: an experimental group consisting of 20 dogs diagnosed with congestive heart failure (CHF) and a control group consisting of 20 clinically healthy dogs. Dogs in the experimental group were classified as stage C or D according to the American College of Veterinary Internal Medicine (ACVIM) classification system. The most common clinical signs observed in dogs with CHF included exercise intolerance, dyspnea, coughing, fatigue, and respiratory distress.

The CHF group consisted of 12 males and 8 females. The breed distribution included small-, medium-, and large-breed dogs: Miniature Yorkshire Terriers (n = 5), Chihuahuas (n = 4), French Bulldogs (n = 3), Pugs (n = 2), Toy Terriers (n = 2), Pekingese (n = 2), Beagle (n = 1), and Golden Retriever (n = 1). The control group consisted of clinically healthy dogs presented to the clinic for routine vaccination, preventive antiparasitic treatment, or general health examination. This group also included 12 males and 8 females aged 6 to 12 years. The breed distribution in the control group included Miniature Yorkshire Terriers (n = 6), French Bulldogs (n = 6), Toy Terriers (n = 3), Golden Retrievers (n = 2), Pug (n = 1), Chihuahua (n = 1), and Beagle (n = 1).

All dogs with CHF received standard medical therapy aimed at improving hemodynamic stability and reducing the clinical manifestations of heart failure. The treatment protocol included pimobendan at a total daily dose of 0.5 mg/kg divided into two administrations every 12 hours, furosemide at a dose of 2 mg/kg administered according to the clinical status of the patient, and amlodipine at a dose of 0.1 mg/kg administered orally once daily. The therapeutic regimen remained unchanged throughout the observation period in order to ensure consistent evaluation of echocardiographic, Doppler ultrasonographic, and biochemical parameters during treatment. Amlodipine

was included in the therapeutic protocol as an adjunctive vasodilatory and antihypertensive agent aimed at reducing systemic vascular resistance and afterload. Its use was considered clinically justified in dogs with congestive heart failure because increased vascular tone and systemic hemodynamic load may aggravate cardiac workload and contribute to impaired renal and hepatic perfusion. The drug was administered at a low dose under clinical monitoring to support hemodynamic stabilization during the observation period.

Electrocardiographic examination was performed in all dogs using a recording speed of 50 mm/s and a standard calibration of 1 mV = 10 mm. ECG recordings were obtained in automatic mode without the use of filters. Dogs that tolerated restraint well were positioned in right lateral recumbency. In animals unable to maintain lateral recumbency because of dyspnea, anxiety, or poor general condition, ECG recordings were obtained in standing or sternal positions. Electrocardiograms were evaluated in all standard leads with assessment of heart rate, cardiac rhythm, conduction abnormalities, and arrhythmias.

Echocardiographic examination was performed using an Esaote MyLab 50 ultrasound system equipped with a phased-array cardiac transducer operating at 4.0–7.5 MHz. Examinations were performed without sedation or general anesthesia to avoid pharmacological effects on cardiac function and hemodynamic parameters. Dogs were examined in both right and left lateral recumbency. Cardiac evaluation was performed from standard parasternal and apical views using long-axis and short-axis projections.

Two-dimensional echocardiography, M-mode, color Doppler, and spectral Doppler were used to determine the morphofunctional status of the heart. Two-dimensional and M-mode echocardiography were used to assess the size of the left atrium and left ventricle, interventricular septal thickness, left ventricular free wall thickness, and systolic and diastolic function. Color Doppler was used to detect regurgitation through the mitral, tricuspid, aortic, and pulmonary valves. Spectral Doppler was used for quantitative assessment of blood flow velocity characteristics, particularly in the valve regions and pulmonary artery.

In M-mode, the right ventricular diastolic diameter, interventricular septal thickness in diastole and systole, left ventricular diastolic and systolic diameters, and posterior wall thickness of the left ventricle in diastole and systole were determined. Left ventricular systolic function was assessed using ejection fraction and fractional shortening. The diameters of the left atrium and aortic root were measured from the right parasternal short-axis view, followed by calculation of the LA/Ao ratio. Echocardiographic examination in the control group was performed once, whereas in dogs with congestive heart failure it was performed four times: before the start of treatment and after 1, 2, and 4 weeks of therapy. All echocardiographic measurements were performed by one investigator, which reduced the risk of interobserver variability.

Doppler ultrasonographic examination of the abdominal organs was performed using a microconvex transducer with a frequency of 5–8 MHz. As with echocardiography, abdominal ultrasonography in the control group was performed once, while in dogs with congestive heart failure it was performed at four time points: on day 0 before treatment and after 1, 2, and 4 weeks of therapy.

To assess renal hemodynamics, dogs were placed in lateral recumbency. The interlobar arteries in the renal parenchyma were identified using color Doppler. After vessel visualization, spectral Doppler ultrasonography was performed with an insonation angle of approximately 45° relative to the longitudinal axis of the vessel. The resistive index and pulsatility index were evaluated. The resistive index was calculated automatically based on peak systolic and end-diastolic blood flow velocities. The

pulsatility index was determined from three consecutive waveform cycles. Each measurement was repeated three times, and the mean value was used for further analysis.

To assess hepatic hemodynamics, the portal vein diameter, peak portal vein blood flow velocity, systolic-to-diastolic ratio of hepatic vein blood flow, and hepatic artery resistive index were determined. The portal vein was visualized from the right intercostal approach in the region of the 9th–11th intercostal spaces with the dog in dorsal recumbency. It was identified by its characteristic hyperechoic wall and anatomical location caudal to the right liver lobe and to the left of the gallbladder. The hepatic artery resistive index was determined in the region of its entry into the liver, near the caudal vena cava. The hepatic vein S/D ratio was assessed in the right liver lobe, dorsal to the caudal vena cava and ventral to the gallbladder. This approach allowed a comprehensive assessment of both the arterial and venous components of hepatic circulation.

For laboratory examination, blood samples were collected from the cephalic vein into serum-separating gel tubes. The samples were centrifuged at 3000 rpm for 5 minutes to obtain serum. Biochemical parameters characterizing the functional status of the liver, kidneys, and myocardium were determined in serum. The investigated parameters included alanine aminotransferase, alkaline phosphatase, gamma-glutamyltransferase, total bilirubin, urea, and creatinine. In addition, symmetric dimethylarginine was measured as a marker of early renal dysfunction, and cardiac troponin I was measured as a marker of myocardial injury. SDMA was determined using Baywellbio® test systems, and cardiac troponin I was measured using Vcheck® according to the manufacturers' instructions. In the control group, biochemical testing was performed once, whereas in dogs with congestive heart failure it was performed on day 0 and after 1, 2, and 4 weeks of treatment.

To ensure homogeneity of the study population and reduce the influence of concomitant diseases on the results of Doppler assessment, exclusion criteria were applied. Dogs with severe primary liver or kidney diseases, neoplasia, systemic infectious processes, endocrine disorders, marked dehydration, and animals that had received drugs capable of substantially affecting renal or hepatic hemodynamics before the start of the study were excluded. This approach made it possible to more objectively evaluate the influence of congestive heart failure and standard therapy on hepatic and renal blood flow parameters.

Statistical analysis was performed using standard statistical software. Data are presented as mean ± standard deviation (M ± SD). The normality of data distribution was assessed using the Shapiro–Wilk test. Differences between the control group and dogs with congestive heart failure at baseline were evaluated using the independent samples Student's t-test for normally distributed variables or the Mann–Whitney U test for non-normally distributed data. Repeated measurements obtained during the treatment period (day 0, week 1, week 2, and week 4) were analyzed using repeated-measures analysis of variance (ANOVA) followed by post hoc pairwise comparisons with Bonferroni correction. In cases of non-normal data distribution, the Friedman test followed by Dunn's post hoc test was applied. Differences were considered statistically significant at $p < 0.05$.

Research results. Analysis of echocardiographic parameters revealed marked differences between clinically healthy dogs and animals with congestive heart failure (CHF). At baseline, dogs with CHF had significantly lower ejection fraction (EF) compared with the control group, indicating reduced myocardial systolic function before the initiation of therapy.

During treatment, EF values progressively increased in the CHF group at weeks 1, 2, and 4. These changes were statistically significant compared with baseline values,

indicating improvement in left ventricular contractile function under standard therapy. The most pronounced increase in EF was recorded at week 4; however, the value remained significantly lower than that of the control group.

According to Table 1, dogs with CHF showed significant baseline changes in LVDd, IVSs, LVDs, PWs, EF, FS, and LA/Ao compared with clinically healthy animals. During treatment, statistically significant improvement was observed mainly in LVDd, LVDs, EF, FS, LA/Ao, and selected systolic wall parameters. The most consistent positive dynamics were recorded for EF, FS, and LA/Ao, supporting partial improvement of systolic function and reduction of left-sided cardiac overload during standard therapy.

Table 1

Echocardiographic parameters in dogs of the control group and dogs with congestive heart failure

Parameter	Control	CHF, day 0	CHF, week 1	CHF, week 2	CHF, week 4
RVDd, mm	9.50±1.88	8.72±4.23	9.14±4.50	9.80±4.71	11.42±5.13
IVSd, mm	9.64±2.01	11.24±4.14	10.15±3.95	9.36±3.91	9.14±4.12
LVDd, mm	24.52±3.36	45.02± 13.21*	40.14± 12.20*	37.92± 12.90*	31.44± 14.07*†
PWd, mm	8.01±1.10	8.92±2.75	9.44±3.04	9.15±3.33	8.82±2.77
IVSs, mm	11.52±3.02	15.01±4.24*	12.12±5.22	11.53±2.81†	11.56±2.23†
LVDs, mm	12.82±4.17	30.16± 10.85*	27.26± 9.85*	24.32± 11.34*	20.05± 9.02*†
PWs, mm	12.77±3.55	17.22±5.44*	13.12±5.35†	11.56±4.24†	10.72±3.04†
EF, %	73.56±6.62	46.12± 9.35*	55.20± 8.36*†	56.23± 9.21*†	61.02± 5.26*†
FS, %	39.05±3.21	23.17± 5.04*	26.17± 5.92*	29.14± 4.22*†	33.33± 5.05*†
LA/Ao	0.94±0.01	1.72±0.52*	1.49±0.40*	1.42±0.40*†	1.05±0.40†

*Note. Data are presented as mean ± standard deviation. * — statistically significant difference compared with the control group within the same row ($p < 0.05$). † — statistically significant difference compared with CHF, day 0 within the same row ($p < 0.05$). The control group consisted of clinically healthy dogs that did not receive treatment. Dogs in the CHF group received standard therapy for 4 weeks.*

The fractional shortening value was also significantly lower in dogs with CHF than in the control group on day 0 of the study, further confirming impaired left ventricular systolic function. Following the initiation of therapy, FS values progressively increased over the observation period. A mild improvement was already evident at week 1, whereas more substantial and statistically significant increases were recorded at weeks 2 and 4 compared with pretreatment values.

Analysis of the LA/Ao ratio demonstrated marked left atrial enlargement in dogs with CHF compared with clinically healthy animals at baseline. These findings reflected volume overload of the left atrium and hemodynamically significant cardiac remodeling associated with congestive heart failure. During treatment, the LA/Ao ratio progressively decreased, suggesting reduction of left-sided cardiac volume overload. Although only a moderate decrease was noted at week 1, more pronounced improvement was observed at weeks 2 and 4 of therapy.

Overall, the obtained findings demonstrated that dogs with CHF exhibited

echocardiographic signs of cardiac remodeling before treatment, including dilation of the left ventricle and left atrium together with reduced myocardial systolic function. Standard therapy was associated with progressive improvement of the main echocardiographic parameters, particularly increased EF and FS values and decreased LA/Ao ratio. The greatest positive changes were recorded at the end of the fourth week of treatment.

According to renal Doppler ultrasonographic findings, dogs with CHF had increased renal vascular resistance at baseline compared with clinically healthy dogs. Both resistive index (RI) and pulsatility index (PI) values of the left and right kidneys exceeded those of the control group. These changes suggested impaired renal perfusion and increased vascular resistance associated with CHF.

During treatment, RI and PI values gradually decreased in both kidneys, although the pattern of improvement differed between the left and right kidneys. In the left kidney, RI showed a significant reduction already at week 1, with further improvement observed at weeks 2 and 4. The PI of the left kidney demonstrated a similar tendency, with mild improvement during the first week followed by more substantial and statistically significant reductions at subsequent examinations.

In the right kidney, RI values showed only minor improvement at week 1, whereas statistically significant reductions were detected at weeks 2 and 4 of therapy. A comparable pattern was observed for right kidney PI values: changes during the first week were limited, while more evident decreases were recorded during the later stages of treatment.

Table 2

Renal Doppler ultrasonographic parameters in dogs of the control group and dogs with congestive heart failure

Parameter	Control	CHF, day 0	CHF, week 1	CHF, week 2	CHF, week 4
Left renal RI	0.62 ± 0.03	0.71 ± 0.02*	0.69 ± 0.02*†	0.66 ± 0.02*†	0.63 ± 0.02†
Left renal PI	1.11 ± 0.31	1.42 ± 0.12*	1.34 ± 0.12*†	1.25 ± 0.13†	1.16 ± 0.03†
Right renal RI	0.62 ± 0.03	0.71 ± 0.03*	0.70 ± 0.03*	0.67 ± 0.03*†	0.63 ± 0.03†
Right renal PI	1.13 ± 0.12	1.36 ± 0.13*	1.32 ± 0.05*	1.24 ± 0.11*†	1.15 ± 0.05†

*Note. Data are presented as mean ± standard deviation. RI — resistive index; PI — pulsatility index. * — statistically significant difference compared with the control group within the same row ($p < 0.05$). † — statistically significant difference compared with CHF, day 0 within the same row ($p < 0.05$). The control group consisted of clinically healthy dogs that did not receive treatment. Dogs in the CHF group received standard therapy for 4 weeks.*

By the end of the fourth week, most renal Doppler parameters approached the values observed in the control group, indicating partial restoration of renal hemodynamics during standard CHF therapy. These findings support the usefulness of RI and PI as additional non-invasive indicators for monitoring renal perfusion and vascular resistance in dogs with congestive heart failure.

According to hepatic Doppler ultrasonographic findings, portal vein blood flow velocity in dogs with CHF on day 0 did not differ significantly from the values recorded in the control group. Throughout the 4-week treatment period, this parameter remained relatively stable, and no statistically significant changes were detected compared with baseline values. These findings suggest relative preservation of portal circulation despite

the presence of CHF.

The portal vein diameter in dogs with CHF was slightly greater at baseline than in clinically healthy animals, although this difference was not statistically significant. A significant increase in portal vein diameter was observed at weeks 1 and 2 compared with baseline. By week 4, the value decreased compared with weeks 1 and 2 and no longer differed significantly from baseline, although it remained higher than the control value. These fluctuations may reflect adaptive changes in portal hemodynamics and compensatory regulation of hepatic blood flow during therapy.

The most prominent alterations were detected in the hepatic vein systolic-to-diastolic (S/D) ratio. In dogs with CHF on day 0, the S/D ratio was significantly lower than in the control group, indicating impaired hepatic venous drainage and increased venous pressure associated with heart failure. During treatment, a gradual increase in the S/D ratio was recorded. Mild improvement was observed at weeks 1 and 2, whereas a more pronounced and statistically significant increase was evident by week 4. These changes may reflect partial restoration of hepatic venous hemodynamics during standard CHF therapy.

The hepatic arterial resistive index was significantly higher in dogs with CHF at baseline compared with clinically healthy animals. At week 1, hepatic arterial RI increased further and differed significantly from baseline, whereas at weeks 2 and 4 the values remained higher than those of the control group but did not show a significant decrease compared with day 0. These findings suggest that disturbances in hepatic arterial blood flow may persist despite clinical improvement.

Table 3

Results of liver ultrasonographic examination

Parameter	Control	CHF, day 0	CHF, week 1	CHF, week 2	CHF, week 4
Portal vein blood flow velocity, cm/s	17.10 ± 1.23	18.40 ± 5.21	18.64 ± 4.19	18.92 ± 6.20	18.34 ± 4.23
Portal vein diameter, cm	0.46 ± 0.03	0.48 ± 0.04	0.53 ± 0.02*†	0.52 ± 0.03*†	0.50 ± 0.02*
Hepatic vein S/D ratio	1.62 ± 0.12	1.21 ± 0.32*	1.29 ± 0.23*	1.35 ± 0.14*	1.49 ± 0.22*†
Hepatic arterial RI	0.67 ± 0.03	0.75 ± 0.04*	0.79 ± 0.05*†	0.78 ± 0.07*	0.77 ± 0.08*

*Note. Data are presented as mean ± standard deviation. RI — resistive index; S/D — systolic-to-diastolic ratio of hepatic vein blood flow. * — statistically significant difference compared with the control group within the same row ($p < 0.05$). † — statistically significant difference compared with CHF, day 0 within the same row ($p < 0.05$). The control group consisted of clinically healthy dogs that did not receive treatment. Dogs in the CHF group received standard therapy for 4 weeks.*

Overall, hepatic Doppler ultrasonography demonstrated that the hepatic vein S/D ratio and hepatic arterial RI were the most informative indicators of hepatic hemodynamic alterations in dogs with CHF. Reduced S/D values before treatment reflected impaired venous outflow and venous congestion, whereas elevated hepatic arterial RI indicated increased vascular resistance within the hepatic arterial bed. During standard therapy, more pronounced improvement was observed in the venous component of hepatic circulation, while arterial resistance remained elevated throughout the observation period.

Table 4 summarizes serum biochemical parameters in the control group and in dogs with CHF. In clinically healthy animals, serum concentrations of cTn-I, urea,

creatinine, SDMA, ALT, ALP, GGT, and total bilirubin were within physiological reference ranges.

Table 4

Serum biochemical parameters in the control group and CHF group

Parameter	Control	CHF, day 0	CHF, week 1	CHF, week 2	CHF, week 4
cTn-I, ng/mL	0.08 ± 0.05	0.21 ± 0.15*	0.19 ± 0.13*	0.16 ± 0.12*	0.14 ± 0.02*
Urea, mg/dL	26.17 ± 12.20	49.80 ± 2.70*	38.32 ± 14.12*†	35.20 ± 10.24*†	33.42 ± 9.82*†
Creatinine, μmol/L	67.55 ± 15.24	112.25 ± 22.21*	105.16 ± 19.32*	97.61 ± 22.30*†	93.55 ± 18.71*†
SDMA, nmol/L	15.60 ± 2.41	38.21 ± 3.56*	34.22 ± 4.20*†	28.22 ± 2.33*†	22.24 ± 2.33*†
ALT, U/L	40.01 ± 6.33	48.23 ± 7.23*	36.20 ± 6.23†	34.23 ± 6.32*†	32.12 ± 4.21*†
ALP, U/L	118.22 ± 25.21	260.68 ± 26.44*	178.70 ± 34.20*†	197.32 ± 35.72*†	200.20 ± 25.32*†
GGT, U/L	5.33 ± 1.40	12.94 ± 21.73	9.81 ± 17.68	12.10 ± 27.49	12.72 ± 26.94
TBIL, mg/dL	0.21 ± 0.05	0.34 ± 0.51	0.28 ± 0.44	0.56 ± 1.35	0.54 ± 1.38

*Note. Data are presented as mean ± standard deviation. cTn-I — cardiac troponin I; SDMA — symmetric dimethylarginine; ALT — alanine aminotransferase; ALP — alkaline phosphatase; GGT — gamma-glutamyl transpeptidase; TBIL — total bilirubin. * — statistically significant difference compared with the control group within the same row ($p < 0.05$). † — statistically significant difference compared with CHF, day 0 within the same row ($p < 0.05$). The control group consisted of clinically healthy dogs that did not receive treatment. Dogs in the CHF group received standard therapy for 4 weeks.*

At baseline, dogs with CHF showed significantly higher serum cTn-I concentrations compared with the control group, indicating increased myocardial stress and possible myocardial injury associated with heart failure. Following initiation of therapy, cTn-I values gradually declined, with the greatest numerical reduction observed by week 4; however, this decrease was not statistically significant compared with baseline according to the table annotations.

Renal biochemical parameters in dogs with CHF also differed from those of clinically healthy animals. On day 0, serum urea, creatinine, and SDMA concentrations were elevated relative to the control group, suggesting impaired renal perfusion and reduced renal functional status associated with CHF. Over the course of treatment, these parameters progressively decreased. The most evident improvement was recorded for SDMA and urea concentrations, whereas creatinine values declined more gradually, with clearer changes observed at weeks 2 and 4.

Activities of ALT and ALP were higher in dogs with CHF at baseline than in clinically healthy dogs. During therapy, ALT activity progressively decreased, which may be associated with improvement of systemic circulation and reduction of hepatic congestion. ALP activity also declined after treatment initiation, although its values remained above those of the control group throughout the observation period. These findings may reflect persistent alterations in hepatic perfusion and mild manifestations of congestive hepatopathy.

Serum GGT and total bilirubin concentrations did not differ significantly between groups and remained relatively stable during the study period. This suggests the absence

of marked cholestatic disturbances or severe impairment of biliary function in the examined animals.

Overall, the biochemical findings demonstrated that dogs with CHF exhibited alterations primarily associated with myocardial stress, renal dysfunction, and hepatic circulatory disturbances. The gradual reduction in cTn-I, urea, creatinine, SDMA, ALT, and ALP during standard therapy may reflect partial stabilization of systemic hemodynamics, reduced venous congestion, and improvement in the functional status of target organs.

Discussion. The findings of the present study indicate that congestive heart failure (CHF) in dogs is associated with systemic hemodynamic consequences that extend beyond the heart. Along with echocardiographic evidence of impaired systolic function and left-sided cardiac remodeling, dogs with CHF showed Doppler ultrasonographic signs of altered renal and hepatic circulation. This supports the concept that advanced heart failure should be assessed as a multisystem disorder involving target organs rather than as an isolated cardiac condition.

Echocardiographic changes observed in dogs with CHF were consistent with reduced myocardial contractility, increased cardiac loading conditions, and progression of congestive remodeling. The decrease in ejection fraction and fractional shortening, together with an increased LA/Ao ratio, reflected impaired left ventricular systolic performance and left atrial volume overload. According to the ACVIM consensus guidelines, echocardiographic assessment remains essential for diagnosis, staging, and therapeutic monitoring in dogs with myxomatous mitral valve disease and CHF (Keene et al., 2019).

The improvement in EF, FS, and LA/Ao during therapy suggests partial stabilization of intracardiac hemodynamics. This effect can be explained by the combined action of standard CHF treatment, particularly pimobendan, which provides positive inotropic and vasodilatory effects, and diuretic therapy aimed at reducing congestion (Keene et al., 2019; Kittleson & Côté, 2021). Amlodipine was included as an adjunctive antihypertensive and afterload-reducing agent. Its use was clinically justified because increased systemic vascular resistance may aggravate cardiac workload and contribute to impaired renal and hepatic perfusion. Therefore, the drug was used to support systemic hemodynamic stabilization during the observation period.

Renal Doppler findings are particularly important because changes in renal perfusion may develop before severe biochemical abnormalities become evident. Increased renal RI and PI values at baseline reflected elevated renal vascular resistance and impaired renal blood flow in dogs with CHF. These alterations are consistent with the pathophysiology of cardiorenal syndrome, in which reduced effective cardiac output, activation of the renin-angiotensin-aldosterone system, sympathetic stimulation, sodium and water retention, and venous congestion interact and further compromise renal function (Orvalho & Cowgill, 2017; Szczepankiewicz et al., 2021).

The reduction of renal RI and PI during treatment suggests partial improvement of renal hemodynamics. However, Doppler indices should not be interpreted as direct equivalents of renal function. They reflect vascular resistance and perfusion characteristics, whereas renal function is influenced by glomerular filtration, tubular activity, hydration status, neurohumoral activation, and therapeutic interventions. For this reason, renal Doppler ultrasonography should complement, rather than replace, biochemical evaluation. The diagnostic value of RI as a sensitive marker of early renal involvement in dogs with myxomatous mitral valve disease has also been emphasized by Szczepankiewicz et al. (2021).

The observed decrease in renal Doppler indices is in agreement with the study by

Esin and Güzel, who reported alterations in renal and hepatic Doppler parameters in dogs with ACVIM stage C and D heart failure and partial improvement of several indices during standard therapy (Esin & Güzel, 2025). In the present study, the dynamics of RI and PI differed slightly between the left and right kidneys. Such variation may be related to individual vascular responses, local renal perfusion differences, respiratory motion, or technical aspects of Doppler measurement. Nevertheless, the general trend supports the usefulness of renal Doppler ultrasonography for monitoring cardiorenal involvement in dogs with CHF.

Hepatic Doppler ultrasonography added an important dimension to the assessment of target-organ involvement. In contrast to renal indices, portal vein blood flow velocity remained relatively stable, suggesting that portal circulation was comparatively preserved during the study period. This agrees with observations by Esin and Güzel, who found that hepatic Doppler changes in dogs with CHF were more pronounced in venous and arterial parameters than in portal flow velocity (Esin & Güzel, 2025). Therefore, portal vein velocity alone may be insufficient for detecting cardiohepatic hemodynamic disturbances.

The hepatic vein systolic-to-diastolic (S/D) ratio appeared to be one of the most informative hepatic Doppler parameters. Its reduction before treatment suggests impaired hepatic venous drainage and increased central venous pressure. This interpretation is physiologically reasonable because hepatic veins communicate directly with the caudal vena cava and right atrium and are therefore sensitive to changes in right-sided cardiac pressure. Kim et al. showed that hepatic venous Doppler waveforms change progressively in dogs with tricuspid regurgitation, ranging from normal systolic predominance to systolic blunting or reversed systolic flow in advanced disease (Kim et al., 2017).

The increase in hepatic vein S/D ratio after treatment indicates partial improvement of hepatic venous outflow and reduction of congestion. Similar improvement of hepatic Doppler parameters during standard CHF therapy was described by Esin and Güzel (2025). These findings suggest that hepatic venous Doppler assessment may provide useful information about systemic venous congestion and response to therapy, especially when interpreted together with echocardiographic findings.

In contrast, hepatic arterial RI remained elevated during the observation period. This may indicate that hepatic arterial perfusion does not recover as rapidly as hepatic venous drainage. Persistent elevation of hepatic arterial resistance may be associated with ongoing neurohumoral activation, altered vascular tone, redistribution of hepatic blood flow, or an insufficient duration of therapy for complete vascular adaptation. These findings suggest that cardiohepatic changes in dogs with CHF may involve both venous congestion and arterial perfusion disturbances.

The relationship between hepatic venous flow and right-sided cardiac hemodynamics has also been demonstrated in previous studies. Silva et al. reported that hepatic vein Doppler spectra in healthy dogs correlate with selected structural and functional variables of the right ventricle, supporting the dependence of hepatic venous blood flow on right-sided cardiac function (Silva et al., 2020). In dogs with pulmonary hypertension, hepatic venous Doppler abnormalities have also been associated with right-sided cardiac overload (Silva et al., 2022). These data support the use of hepatic Doppler ultrasonography as an additional tool for evaluating venous congestion and cardiohepatic interactions in dogs with CHF.

Biochemical findings further supported the involvement of target organs. Increased cTn-I values reflected myocardial stress or injury, whereas changes in urea, creatinine, and SDMA were consistent with impaired renal perfusion and functional renal involvement. Alterations in ALT and ALP may be associated with hepatic congestion or changes in hepatic perfusion. At the same time, the absence of marked changes in GGT

and total bilirubin suggests that severe cholestatic or biliary dysfunction was not a dominant feature in the studied dogs.

The scientific novelty of this study consists in the integrated evaluation of echocardiographic, renal, and hepatic Doppler ultrasonographic parameters in dogs with CHF during standard therapy. Unlike studies focused mainly on cardiac function or isolated assessment of renal or hepatic circulation, this work evaluated cardiorenal and cardiohepatic hemodynamic interactions within the same clinical model. Particular attention was given to hepatic venous and arterial Doppler indices, which remain insufficiently investigated in veterinary cardiology.

Overall, the results support the inclusion of renal and hepatic Doppler ultrasonography in the comprehensive assessment of dogs with CHF. Echocardiography remains the basis for cardiac evaluation, but Doppler assessment of target organs may provide additional information about systemic hemodynamic consequences of heart failure. This approach may help detect early cardiorenal and cardiohepatic disturbances, monitor therapeutic response, and support individualized clinical decision-making.

The main limitations of this study were the relatively small number of animals and the 4-week observation period. Future investigations should include larger populations, longer follow-up, and correlation of Doppler-derived parameters with clinical outcomes and biomarkers of organ dysfunction. Such studies may help determine the prognostic value of renal and hepatic Doppler indices in dogs with congestive heart failure.

Conclusions

1. In dogs with congestive heart failure, marked changes in echocardiographic parameters were established, manifested by a decrease in ejection fraction and fractional shortening, as well as an increase in the LA/Ao ratio compared with clinically healthy animals. These findings indicate impaired myocardial systolic function, dilation of the left-sided cardiac chambers, and the presence of hemodynamically significant changes characteristic of CHF.

2. Against the background of standard therapy, dogs with CHF showed positive dynamics in the main echocardiographic parameters. Ejection fraction gradually increased during 4 weeks of treatment, while fractional shortening demonstrated more pronounced improvement from the second week of therapy. A decrease in the LA/Ao ratio indicated a gradual reduction in hemodynamic load on the left atrium.

3. Doppler ultrasonographic examination of the kidneys showed that, before treatment, the values of the resistive index and pulsatility index in both kidneys were higher in dogs with congestive heart failure than in clinically healthy dogs. This indicates increased vascular resistance in the renal blood flow and impaired renal perfusion associated with heart failure.

4. During treatment, renal RI and PI gradually decreased, indicating an improvement in renal hemodynamics. The most pronounced changes were observed at weeks 2 and 4 of therapy, when most Doppler ultrasonographic parameters approached the values of the control group.

5. According to the results of hepatic Doppler ultrasonography, portal vein blood flow velocity in dogs with CHF did not differ substantially from the values of the control group and remained relatively stable throughout the observation period. This may indicate preservation of portal blood flow in the studied animals despite the presence of heart failure.

6. The most informative indicators of hepatic hemodynamics were the hepatic vein S/D ratio and the hepatic arterial resistive index. In dogs with CHF before treatment, the hepatic vein S/D ratio was significantly lower, whereas the hepatic arterial RI was

significantly higher compared with the control group. These changes indicate impaired venous outflow from the liver and increased vascular resistance in the hepatic arterial bed.

7. Against the background of standard therapy, a gradual increase in the hepatic vein S/D ratio was observed, most pronounced at week 4 of treatment. This may indicate partial improvement in hepatic venous blood flow and a reduction in congestive changes. At the same time, hepatic arterial RI remained elevated, which may suggest more prolonged persistence of impaired hepatic arterial perfusion.

8. The obtained results confirm the close relationship between impaired cardiac function and changes in blood flow in target organs in dogs with CHF. Increased renal RI and PI, decreased hepatic vein S/D ratio, and increased hepatic arterial RI may be considered additional non-invasive markers of cardiorenal and cardiohepatic hemodynamic disorders.

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