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Clinical and instrumental features of cardiorenal syndrome in cats with hypertrophic cardiomyopathy

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Abstract. This research presents novel insights into the temporal dynamics of clinical and instrumental parameters pertaining to the emergence of cardiorenal syndrome in feline patients afflicted with hypertrophic cardiomyopathy. It elucidates that within pedigree felines, the progression of congestive heart failure syndrome may precipitate the subsequent evolution and exacerbation of secondary renal damage, thus significantly complicating the trajectory of the primary pathological process. This study establishes, for the first time, the prevalence of cardiorenal syndrome, affecting 51.0% of the population within the broader cohort of cats afflicted with hypertrophic cardiomyopathy ($n = 49$). Moreover, it substantiates the role of the cardiorenal continuum in felines as a predictor of a more severe course of hypertrophic cardiomyopathy. Manifesting as concentric myocardial hypertrophy in domestic felines, cardiorenal syndrome is characterized by dyspnea, hypothermia, and circulatory insufficiency. Statistically significant findings include an elevated median nocturnal respiratory

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rate of 34.0 breaths/min ($p < 0.001$) compared to clinically healthy counterparts (18.0 breaths/min) in affected felines. Additionally, felines afflicted with hypertrophic cardiomyopathy and cardiorenal syndrome exhibit a statistically significant ($p < 0.001$) elevation in median mean arterial blood pressure to 140.0 mmHg compared to clinically healthy counterparts (104.0 mmHg), sinus tachycardia at 199.0 beats/min (171.5 beats/min in healthy felines), resulting in a statistically significant ($p < 0.001$) reduction in PQ intervals by 67.0 ms (75.5 ms in healthy felines), and an increase in QT interval by 204.0 ms (165.5 ms in healthy felines). Electrocardiographic assessments reveal indications of compromised intra-atrial and intraventricular conduction in hypertrophic cardiomyopathy-afflicted felines with cardiorenal syndrome, along with an augmented amplitude of the ventricular complex. Echocardiographic evaluations confirm alterations such as pulmonary vein dilation, pronounced left atrial anteroposterior enlargement, interventricular septal and left ventricular free wall hypertrophy, decreased longitudinal contractility of the left and right ventricles, and clinically significant diastolic dysfunction.

Keywords: cardiology, echocardiography, electrocardiography, diagnostics, heart failure, nephropathy, cardiorenal continuum

Conflict of interests. The authors declare that they have no conflict of interests.

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Introduction

The incidence of poly-morbid pathologies in domestic animals is steadily increasing [1]. Among these, kidney diseases and cardiovascular disorders stand out as primary contributors to mortality in domestic felines [2–4]. Congestive heart failure is known to precipitate renal impairment, while chronic kidney disease frequently precedes the onset of cardiovascular insufficiency [5–9]. The coexistence of these comorbidities has prompted the conceptualization, within the realms of veterinary cardiology and nephrology, cardiorenal syndrome is a condition characterized by concurrent disruptions in the structural and functional integrity of the kidneys and heart. This syndrome is associated with a significantly elevated mortality rate among afflicted animals compared to the general population [10–14]. Given the heightened risk of complications, there is a pressing need for innovative diagnostic modalities aimed at early identification of renal and cardiac involvement in cardiorenal syndrome, with the ultimate goal of refining therapeutic interventions and management strategies [15–20].

Hypertrophic cardiomyopathy represents a common clinical entity encountered in feline practice, frequently culminating in chronic circulatory insufficiency and predisposing affected individuals to cardiorenal sequelae. Notably, the pathogenic mechanisms underpinning the genesis and progression of cardiorenal syndrome in cats afflicted with hypertrophic cardiomyopathy remain largely unexplored and inadequately elucidated in scientific literature.

Thus, **the primary objective of this study** is to elucidate the clinical and instrumental characteristics, alongside the underlying pathogenetic mechanisms, governing the development of cardiorenal syndrome in cats afflicted with hypertrophic cardiomyopathy.

Materials and Methods

The study cohort comprised cats diagnosed with hypertrophic cardiomyopathy complicated by cardiorenal syndrome (Group II, $n = 25$), as well as a control group of cats free from cardiorenal complications (Group I, $n = 24$). Diagnosis of hypertrophic cardiomyopathy was established through a rigorous diagnostic protocol encompassing various clinical and diagnostic modalities [2, 9]. Cardiorenal syndrome was defined by the presence of persistent azotemia, with a serum creatinine concentration equal to or exceeding $200 \mu\text{mol/L}$. A control group consisting of healthy cats ($n = 24$), matched for age and body weight, was included for comparative analysis.

Standardized clinical diagnostic procedures were employed throughout the study [3, 13]. Respiratory function was assessed in all subjects [2], while blood pressure measurements were obtained using the PetMAP graphic II device [9]. Mean arterial pressure was subsequently derived from these measurements [9, 14]. Electrocardiograms were acquired utilizing the EK1T-04 Midas apparatus [9], and echocardiographic evaluations were conducted using a Mindray DP-60 machine equipped with a P10–4E transducer [13, 17]. Echocardiographic parameters assessed included measurements of the pulmonary vein (PV) and the right branch of the pulmonary artery (RBPA) [9, 12], as well as diastolic and systolic dimensions of the interventricular septum (IVSd and IVSs), diastolic and systolic dimensions of the left ventricular free wall (LVFWd and LVFWs), and left ventricular end-diastolic and end-systolic dimensions (LVEDD and LVESD). Myocardial fractional shortening (FS) was calculated according to established methodology [16, 17].

Statistical analyses were conducted using the STATISTICA 7.0 software package [9], with the Mann — Whitney and Kruskal — Wallis tests employed for comparative analysis. Descriptive statistics, including the median (Me) and interquartile range (IQR), were computed for relevant variables.

Results and Discussion

In our study, the prevalence of cardiorenal syndrome among cats diagnosed with hypertrophic cardiomyopathy was observed in 25 out of 49 cases, representing 51.0% of the cohort. Of particular note, the median serum creatinine concentration in healthy cats was recorded at $106.5 \mu\text{mol/L}$ (interquartile range [IQR]: $93.0 \dots 136.5$), while cats in Group I exhibited a median concentration of $135.0 \mu\text{mol/L}$ (IQR: $121.0 \dots 147.0$), and those in Group II presented a notably elevated median of $290.0 \mu\text{mol/L}$ (IQR: $257.0 \dots 313.0$).

A statistically significant divergence among the groups was identified, as evidenced by high H values and a stringent level of significance ($H \geq 21.9$; $p < 0.001$) upon applying the

Kruskal — Wallis test to assess various clinical parameters, including body temperature, pulse rate, respiratory rate, sleep respiratory movements, as well as systolic, diastolic, and mean arterial blood pressure. These findings underscore the necessity for meticulous intergroup statistical comparisons, as clinical parameters across different experimental cohorts are evidently not drawn from a single homogeneous population.

Furthermore, cats afflicted with uncomplicated forms of hypertrophic cardiomyopathy displayed distinctive clinical features compared to their clinically healthy counterparts. Notable manifestations included hypothermia, tachycardia, tachypnea, heightened sleep respiratory movements, and elevated levels of systolic, diastolic, and mean arterial blood pressure (refer to Table 1).

Table 1

Clinical Parameters in Cats with Hypertrophic Cardiomyopathy Complicated by Cardiorenal Syndrome

Index	Animal groups						Kruskal – Wallis test
	Control (n = 24)		I (n = 24)		II (n = 25)		
	Me	IQR	Me	IQR	Me	IQR	
Respiration, min ⁻¹	33.0	31.0...35.0	46.0 ***	40.5...50.0	65.0*** ###	58.0...69.0	H = 59.7 p < 0.001
Temperature, °C	38.6	38.4...38.7	38.2 ***	37.9...38.3	36.9 *** ###	36.8...37.2	H = 51.6 p < 0.001
Pulse, min ⁻¹	171.5	145.5...187.0	192.5 ***	182.5... 203.5	199.0 ***	192.0...227.0	H = 26.7 p < 0.001
SBP, mm Hg	165.0	148.5...174.0	193.5 ***	176.5... 204.0	214.0 ***	162.0...231.0	H = 22.5 p < 0.001
DBP, mm Hg	75.0	69.0...83.0	105.0 ***	92.0...115.0	105.0 ***	85.0...125.0	H = 21.9 p < 0.001
MAP, mm Hg	104.0	98.0...110.5	133.0 ***	121.0... 141.5	140.0 ***	117.0...165.0	H = 23.5 p < 0.001
Sleep respiration, min ⁻¹	18.0	15.5...20.5	28.5 ***	24.5...32.0	34.0 *** ###	32.0...38.0	H = 49.1 p < 0.001

Note. Group I – cats with hypertrophic cardiomyopathy without cardiorenal complications; Group II – cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome; Me – median; IQR – interquartile range; *** – p < 0.001 – significance of the difference between Group I, Group II, and clinically healthy animals (Mann – Whitney test); ### – p < 0.001 – significance of the difference between Group I and Group II animals (Mann – Whitney test); SBP – systolic blood pressure; DBP – diastolic blood pressure; MAP – mean arterial pressure.

In cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome, compared to clinically healthy ones, a significant decrease in body temperature (by 1.05 times; p < 0.001), an increase in pulse rate (by 1.16 times; p < 0.001), respiratory rate (by 1.96 times; p < 0.001), and sleep respiratory movements (by 1.89 times; p < 0.001) were observed, along with increases in systolic (by 1.29 times; p < 0.001), diastolic (by 1.4 times; p < 0.001), and mean arterial pressure (by 1.35 times; p < 0.001). Additionally, animals with hypertrophic

cardiomyopathy complicated by cardiorenal syndrome exhibited a significant decrease in body temperature (by 1.04 times; $p < 0.001$) and an increase in respiratory rate during clinic examination (by 1.44 times; $p < 0.001$) and during sleep (by 1.19 times; $p < 0.001$) compared to cats without such complications.

Changes in electrocardiographic parameters in cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome are presented in Table 2.

Table 2

Electrocardiographic Parameters in Cats with Hypertrophic Cardiomyopathy Complicated by Cardiorenal Syndrome

Index	Animal groups						Kruskal – Wallis test
	Control (n = 24)		I (n = 24)		II (n = 25)		
	Me	IQR	Me	IQR	Me	IQR	
Pll, mV	0.100	0.075...0.150	0.200 ***	0.150...0.200	0.200 ***	0.150...0.200	$H = 18.8$ $p < 0.001$
P, msec	33.0	32.0...36.5	37.0	32.5...40.0	37.0 **	35.0...40.0	$H = 8.7$ $p < 0.05$
PQ, msec	75.5	71.0...78.0	65.5 ***	59.5...70.5	67.0 **	64.0...72.0	$H = 18.1$ $p < 0.001$
Rll, mV	0.580	0.470...0.665	0.800 ***	0.700...0.900	0.980 *** #	0.650...1.270	$H = 21.1$ $p < 0.001$
QRS, msec	40.0	38.5...43.5	38.5	35.0...45.5	44.0 *** ###	47.0...52.0	$H = 21.4$ $p < 0.001$
ST, mV	0.00	0.00...0.00	0.00	0.025...0.00	0.00	0.050...0.050	$H = 0.4$ $p < 1$
QT, msec	165.5	146.5...193.5	181.0	173.0...198.0	204.0 **	175.0...220.0	$H = 8.4$ $p < 0.05$
Tll, mV	0.100	0.100...0.150	0.100	0.100...0.175	0.150 #	0.100...0.200	$H = 5.1$ $p < 0.1$

Note: Group I – cats diagnosed with hypertrophic cardiomyopathy without cardiorenal complications; Group II – cats diagnosed with hypertrophic cardiomyopathy complicated by cardiorenal syndrome; Me – median; IQR – interquartile range; ** – $p < 0.01$; *** – $p < 0.001$ – significance of the difference between Group I, Group II, and clinically healthy animals (determined by Mann – Whitney U test); # – $p < 0.05$; ### – $p < 0.001$ – significance of the difference between Group I and Group II animals (determined by Mann – Whitney U test); P – P wave duration; PQ – P – Q interval duration; QRS – QRS complex duration; QT – QT interval duration; Pll, Rll, Tll – amplitudes of P, R, and T waves in the second standard lead of the electrocardiogram; ST – ST segment deviation from the isoelectric line.

The Kruskal – Wallis analysis revealed a significant discrepancy in the duration of the P wave among animals in distinct groups ($H = 8.7$; $p < 0.05$). Notably, cats afflicted with hypertrophic cardiomyopathy complicated by cardiorenal syndrome displayed a noteworthy augmentation in P wave duration, measuring 1.12 times higher than clinically healthy cats ($p < 0.05$). Elevated H values were also discerned for other electrocardiographic parameters, encompassing PQ and QT interval durations, QRS complex duration, and amplitudes of P and R waves in the second standard lead.

In felines afflicted with uncomplicated forms of hypertrophic cardiomyopathy, there was a discernible reduction in atrioventricular conduction time (PQ interval) by 1.15 times ($p < 0.001$), coupled with increases in P wave amplitude (by 2.0 times; $p < 0.001$) and R wave amplitude (by 1.48 times; $p < 0.001$) in the second standard lead compared to clinically healthy counterparts. Conversely, in cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome, there were significant reductions in PQ interval duration (by 1.13 times; $p < 0.001$) and increases in QRS complex duration (by 1.10 times; $p < 0.001$), QT interval duration (by 1.23 times; $p < 0.01$), and amplitudes of P (by 2.0 times; $p < 0.001$) and R (by 1.69 times; $p < 0.001$) waves in the second standard lead compared to clinically healthy cats. Additionally, cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome displayed significant increases in QRS complex duration (by 1.14 times; $p < 0.001$), R wave amplitude (by 1.23 times; $p < 0.05$), and T wave amplitude (by 1.5 times; $p < 0.05$) compared to those without such complications.

According to the Kruskal — Wallis test, statistically significant deviations were found among cats in different groups concerning several echocardiographic parameters, including left ventricular (LV) size, right ventricular end-diastolic diameter (RVED), left atrial (LA) size, LA/Ao ratio, interventricular septum thickness at end-diastole (IVSd), interventricular septum thickness at end-systole (IVSs), left ventricular posterior wall thickness at end-diastole (LVPWd), left ventricular posterior wall thickness at end-systole (LVPWs), left ventricular internal diameter at end-diastole (LVIDd), left ventricular internal diameter at end-systole (LVIDs), and fractional shortening (FS) (Table 3). Inter-group comparisons also revealed significant alterations in these parameters. Specifically, cats with uncomplicated forms of hypertrophic cardiomyopathy displayed substantial increases in LV size (by 2.39 times; $p < 0.001$), LA size (by 1.38 times; $p < 0.001$), LA/Ao ratio (by 1.42 times; $p < 0.001$), IVSd (by 1.63 times; $p < 0.001$), IVSs (by 1.36 times; $p < 0.001$), LVPWd (by 1.61 times; $p < 0.001$), LVPWs (by 1.31 times; $p < 0.001$), coupled with a notable decrease in FS (by 1.33 times; $p < 0.001$) and FS (by 1.27 times; $p < 0.001$) compared to clinically healthy cats. Conversely, cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome exhibited significant increases in LV size (by 2.5 times; $p < 0.001$), LA size (by 1.96 times; $p < 0.001$), LA/Ao ratio (by 2.0 times; $p < 0.001$), IVSd (by 1.88 times; $p < 0.001$), IVSs (by 1.54 times; $p < 0.001$), LVPWd (by 1.71 times; $p < 0.001$), LVPWs (by 1.49 times; $p < 0.001$), coupled with a significant decrease in FS (by 1.78 times; $p < 0.001$) and FS (by 1.75 times; $p < 0.001$) compared to clinically healthy cats. Additionally, cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome displayed significant increases in LA size (by 1.42 times; $p < 0.001$), LA/Ao ratio (by 1.41 times; $p < 0.001$), IVSd (by 1.15 times; $p < 0.01$), IVSs (by 1.13 times; $p < 0.01$), LVPWd (by 1.06 times; $p < 0.05$), LVPWs (by 1.14 times; $p < 0.05$), coupled with a notable decrease in FS (by 1.33 times; $p < 0.01$) and FS (by 1.38 times; $p < 0.05$) compared to cats without such complications.

Basic Echocardiographic Parameters in Cats with Hypertrophic Cardiomyopathy Complicated by Cardiorenal Syndrome

Index	Animal group						Kruskal – Wallis test
	Control (n = 24)		I (n = 24)		II (n = 25)		
	Me	IQR	Me	IQR	Me	IQR	
PV, cm	0.28	0.24...0.36	0.67 ***	0.59...0.73	0.70 ***	0.63...0.80	H = 44.2 p < 0.001
RBPV, cm	0.58	0.54...0.62	0.52	0.47...0.58	0.51	0.45...0.56	H = 8.2 p < 0.05
LA, cm	1.12	1.01...1.24	1.55 ***	1.40...2.05	2.20 *** ###	1.90...2.40	H = 50.2 p < 0.001
AO, cm	0.95	0.80...1.10	1.00	0.90...1.10	1.00	0.80...1.10	H = 0.57 p < 1
LA/AO, units	1.20	1.10...1.30	1.70 ***	1.40...2.20	2.40 *** ###	2.00...2.60	H = 46.0 p < 0.001
IVSd, cm	0.40	0.35...0.42	0.65 ***	0.59...0.72	0.75 *** ##	0.65...0.85	H = 51.4 p < 0.001
IVSs, cm	0.61	0.59...0.66	0.83 ***	0.74...0.90	0.94 *** ##	0.89...1.00	H = 46.1 p < 0.001
LVPWd, cm	0.41	0.40...0.45	0.66 ***	0.59...0.73	0.70 *** #	0.65...0.80	H = 48.7 p < 0.001
LVPWs, cm	0.67	0.61...0.72	0.88 ***	0.80...0.95	1.00 *** ##	0.90...1.10	H = 37.9 p < 0.001
LVIDd, cm	1.60	1.40...1.70	1.20 ***	1.00...1.40	0.90 *** ##	0.8...1.10	H = 35.2 p < 0.001
LVIDs, cm	0.70	0.60...0.80	0.55 ***	0.40...0.60	0.40 *** #	0.30...0.50	H = 26.5 p < 0.001
FS, %	52.50	43.50...61.50	57.00	46.00...65.50	50.00	38.00...70.00	H = 0.8 p < 1

Note: Group I – cats diagnosed with hypertrophic cardiomyopathy without cardiorenal complications; Group II – cats diagnosed with hypertrophic cardiomyopathy complicated by cardiorenal syndrome; Me – median; IQR – interquartile range; *** – $p < 0.001$ – significance of the difference between Group I, Group II, and clinically healthy animals (determined by Mann-Whitney U test); # – $p < 0.05$; ## – $p < 0.01$; ### – $p < 0.001$ – significance of the difference between Group I and Group II animals (determined by Mann – Whitney U test); PV pulmonary vein; RBPV – right pulmonary artery branch; LA – left atrium; AO – aorta; dISD and sISD, diastolic and systolic dimensions of the interventricular septum IVSd and IVSs – diastolic and systolic interventricular septum thickness; LVPWd and LVPWs – diastolic and systolic left ventricular posterior wall thickness; LVIDd and LVIDs – left ventricular internal diameter at end-diastole and end-systole; FS – fractional shortening.

Kruskal – Wallis analysis revealed significant differences in echocardiographic parameters characterizing diastolic function and longitudinal myocardial contractility in cats with cardiorenal syndrome complicating primary hypertrophic cardiomyopathy (Table 4).

Table 4

Parameters of Diastolic Function and Longitudinal Myocardial Contractility in Cats with Hypertrophic Cardiomyopathy Complicated by Cardiorenal Syndrome

Index	Animal groups						Kruskal – Wallis test
	Control (n = 24)		I (n = 24)		II (n = 25)		
	Me	IQR	Me	IQR	Me	IQR	
E, m/sec	0.70	0.60...0.80	1.10 ***	0.95...1.30	1.50 *** ##	1.10...1.70	H = 46.7 p < 0.001
A, m/sec	0.55	0.45...0.60	0.75 ***	0.65...0.80	0.60 #	0.40...0.70	H = 14.5 p < 0.001
IVRT, msec	54.50	45.0...56.50	29.50 ***	22.0...38.0	18.00 *** ###	15.00...23.00	H = 51.6 p < 0.001
TAPSE, sm	9.50	8.00...11.00	6.00 ***	5.00...6.00	4.00 *** #	4.00...5.00	H = 34.9 p < 0.001
MAPSE, sm	5.00	5.00...6.00	4.00	2.50...5.00	3.00	2.00...4.00	H = 21.1 p < 0.001

Note: Group I – feline subjects diagnosed with hypertrophic cardiomyopathy without concomitant cardiorenal complications; Group II – feline subjects diagnosed with hypertrophic cardiomyopathy complicated by cardiorenal syndrome; Me – median; IQR – interquartile range; *** – $p < 0.001$ – indicating the significance of the difference between Group I, Group II, and clinically healthy animals (determined by Mann – Whitney U test); # – $p < 0.05$; ## – $p < 0.01$; ### – $p < 0.001$ – indicating the significance of the difference between Group I and Group II subjects (determined by Mann – Whitney U test); E and A – peak velocities in early diastole and atrial contraction phase; IVRT – isovolumetric relaxation time of the left ventricle; TAPSE and MAPSE – amplitude of tricuspid and mitral annular plane systolic excursion.

In cats with uncomplicated forms of hypertrophic cardiomyopathy, compared to clinically healthy ones, statistically significant increases in E (by 1.57 times; $p < 0.001$) and A (by 1.36 times; $p < 0.001$) peaks of trans-mitral valve blood flow velocities were observed, accompanied by a reduction in IVRT (by 1.85 times; $p < 0.001$) and TAPSE (by 1.58 times; $p < 0.001$). Conversely, in cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome, compared to clinically healthy ones, significant increases in E peak (by 2.14 times; $p < 0.001$) and trans-mitral valve blood flow velocities were noted, alongside a decrease in IVRT (by 3.03 times; $p < 0.001$) and TAPSE (by 2.38 times; $p < 0.001$). Additionally, in cats with cardiorenal syndrome, compared to those without this complication, significant increases in E peak (by 1.36 times; $p < 0.01$), and reductions in A peak (by 1.27 times; $p < 0.05$) of trans-mitral valve blood flow velocities, along with decreases in IVRT (by 1.64 times; $p < 0.001$) and TAPSE (by 1.5 times; $p < 0.01$) were observed.

Cardiorenal syndrome represents a comorbid disorder of the structural and functional characteristics of the kidneys and heart, wherein damage to one of these organs triggers alternative pathogenetic mechanisms in the other [6, 15].

Significant hypothermia, tachycardia, tachypnea, and arterial hypertension were observed in animals with cardiorenal syndrome, indicating more severe cardiac dysfunction and circulatory insufficiency [17]. Notably, cats with cardiorenal syndrome exhibit more significant activation of the neurohumoral system (renin, angiotensin II, aldosterone, sympathetic nervous system), leading to the progression of cardiovascular failure, deterioration of systemic hemodynamics, decreased renal blood flow, and impaired renal excretory function. Remarkably, cats with cardiorenal syndrome displayed more significant delays in intra-atrial and intraventricular conduction, as well as increased voltage of the ventricular complex and T wave, suggesting the development of cardiac hypertrophy.

In feline subjects with hypertrophic cardiomyopathy complicated by cardiorenal syndrome, significant enlargement of the pulmonary vein and left atrium was observed, indicative of a more severe course of left ventricular heart failure and suggestive of pulmonary congestion and edema. Moreover, a substantial increase in respiratory rate during sleep in cats with cardiorenal syndrome further suggests the presence of severe pulmonary congestion, significantly correlating with other studies [2, 3, 14].

Myocardial hypertrophy was more pronounced in cats with cardiorenal syndrome. However, transverse myocardial contractility of the left ventricle was not significantly altered in affected cats. Meanwhile, longitudinal contractility of the myocardium of both the left (MAPSE) and right ventricles (TAPSE) was significantly reduced, indicating the presence of systolic dysfunction. Notably, in all cats with hypertrophic cardiomyopathy complicated by cardiorenal syndrome, pronounced diastolic dysfunction of the left ventricular myocardium was observed, as evidenced by a significant increase in E peak of the trans-mitral valve blood flow and a decrease in isovolumetric relaxation time. These findings were observed for the first time.

Thus, the presence of cardiorenal syndrome in cats with hypertrophic cardiomyopathy may serve as a potential predictor of an unfavorable disease course. Further investigation into the pathogenetic mechanisms underlying the formation of the cardiorenal continuum in animals with primary cardiac and renal pathologies is warranted. The unique insights into the formation of cardiorenal relationships in cats with hypertrophic cardiomyopathy, as described herein, hold significant implications for veterinary medicine, particularly in the timely diagnosis of this complication, facilitating the selection of optimal cardiorenal protection strategies and improving survival outcomes in affected animals.

Conclusion

The presence of cardiorenal syndrome in cats serves as a prognostic indicator for a more severe course of hypertrophic cardiomyopathy. Cardiorenal complications in cats with hypertrophic cardiomyopathy are encountered at a high frequency (51.0%) and are characterized by the presence of azotemia, signs of congestive left ventricular

heart failure, a severe disease course, hypothermia, tachypnea, increased respiratory rate during sleep, tachycardia, and a slowing of atrial and ventricular conduction. Electrocardiograms demonstrate an increase in atrial and ventricular complex voltage. Echocardiographic examination reveals significant dilatation of the pulmonary vein, enlargement of the left atrium, concentric hypertrophy of the free wall of the left ventricle and interventricular septum, preservation of normal transverse concentric myocardial function, with a simultaneous decrease in longitudinal contractility parameters of the left and right ventricular myocardium.

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Клинико-инструментальные особенности кардиоренального синдрома у больных гипертрофической кардиомиопатией кошек

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Аннотация. Приведены новые данные относительно динамики изменений клинико-инструментальных показателей при возникновении кардиоренального синдрома у больных гипертрофической кардиомиопатией кошек. Показано, что у высокопородистых кошек при развитии синдрома застойной сердечно-сосудистой недостаточности может развиваться и прогрессировать вторичное повреждение почек, что значительно осложняет течение первичного патологического процесса. Впервые установлено, что кардиоренальный синдром встречается у 51,0 % особей от общего числа больных гипертрофической кардиомиопатией кошек ($n = 49$). Доказано, что кардиоренальный континуум у кошек являются предиктором более тяжелого течения гипертрофической кардиомиопатии. Обусловленный концентрической гипертрофией сердца

кардиоренальный синдром у домашних кошек характеризуется диспноэ, гипотермией и недостаточностью функции кровообращения. У больных кошек диагностировали статистически значимое повышение медианы частоты дыхания во сне до 34,0 раз/мин ($p < 0,001$) по сравнению с клинически здоровыми животными (18,0 раз/мин). У больных гипертрофической кардиомиопатией кошек при кардиоренальном синдроме развивается статистически значимое ($p < 0,001$) повышение медианы уровня среднего артериального давления крови до 140,0 мм. рт. ст. по сравнению с клинически здоровыми животными (104,0 мм. рт. ст.), синусовая тахикардия — 199,0 раз/мин (171,5 раз/мин у здоровых кошек), что приводит к статистически значимому ($p < 0,001$) снижению продолжительности интервалов PQ 67,0 мс (75,5 мс у здоровых кошек) и повышению интервала QT 204,0 мс (165,5 мс у здоровых кошек). На электрокардиограммах больных гипертрофической кардиомиопатией кошек при кардиоренальном синдроме выявлены признаки ухудшения внутрипредсердной и внутрижелудочковой проводимости, отмечается повышение амплитуды вентрикулярного комплекса. На эхокардиограммах больных кошек верифицированы такие изменения, как дилатация максимального диаметра легочной вены, выраженное расширение переднезаднего размера левого предсердия, гипертрофия миокарда межжелудочковой перегородки и свободной стенки левого желудочка, снижение продольной контрактильности левого и правого желудочка, а также клинически значимая диастолическая дисфункция.

Ключевые слова: кардиология, эхокардиография, электрокардиография, диагностика, сердечная недостаточность, нефропатия, кардиоренальный континуум

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