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Research article/ Научная статья

## Features of the course of hepatocardial syndrome in cats with hypertrophic cardiomyopathy

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**Abstract.** The issues of changes in clinical, laboratory and instrumental parameters in cats with hepatocardial syndrome formed against the background of hypertrophic cardiomyopathy were studied. It is known that in high-bred cats with congestive heart failure, secondary hepatopathy can develop and progress. It was shown that hepatocardial syndrome occurs in 33.7 % of cats, out of the total number of patients with hypertrophic cardiomyopathy (n = 83). It has been established that hepatocardial complications in cats are a risk factor for a more severe course of hypertrophic cardiomyopathy. Hepatocardial syndrome in cats with hypertrophic cardiomyopathy is characterized by severe hypothermia, circulatory and respiratory failure. In sick animals, an increase in frequency of breathing during sleep was recorded (33.3±9.3 versus 17.9±1.8 times/min; p < 0.001). Domestic cats with hepatocardial syndrome had a decrease in mean arterial blood pressure (100.2±19.3 versus 107.2±19.1 mm Hg; p < 0.05), sinus tachycardia (200.3±19.6 versus 177.8 times/min; p < 0.001), which leads to a significant decrease in PQ intervals (57.9±9.9 versus 64.9±9.9 ms; p < 0.001) and QT intervals (168.9±17.2 versus 157.5±18.6 ms; p < 0.001). Sick cats had a significant increase in the time of refilling of capillaries with blood, slowdown in intraventricular conduction, increase in voltage of ventricular and atrial complex on electrocardiograms, expansion of pulmonary vein, significant dilatation of left atrium, extreme concentric hypertrophy of left ventricle, increase in transverse contractility of myocardium of left ventricle and decrease in longitudinal contractility myocardium of left and right ventricles, cardiomyocyte cytolysis syndrome, cholestasis, and hypoalbuminemia.

**Keywords:** cardiology, hepatology, diagnostics, heart failure, hepatopathy

**Conflicts of interest.** The authors declare no conflicts of interest.

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## Introduction

Cardiomyopathy and hepatopathy are extremely common in highly bred animals and represent a potentially lethal pathology [1–5]. In clinical practice, situations often develop for the simultaneous course of heart and liver diseases [2, 6]. In this case, it is called hepatocardial syndrome, which is a supra-nosological problem of the formation of multimorbidity [7, 8]. Hepatocardial syndrome can occur as a complication of both primary hepatic [6] and cardiac pathology [2, 7]. Metabolic diseases, for example, in highly productive cows, can initiate the development of hepatocardial syndrome [8]. It is believed that systemic inflammation, oxidative stress, endogenous intoxication, immune and autoimmune processes may be a reason for hepatocardial complications in animals [9–12]. Hepatocardial syndrome in cats with primary myocardial pathology caused by hypertrophic cardiomyopathy was not described in scholarly literature.

**The aim of our study** was to give empirically and theoretically a clinical and pathogenetic characterization of the course of hepatocardial syndrome in cats with hypertrophic cardiomyopathy.

## Materials and methods of research

When analyzing the literature data on the evaluation of indicators in highly pedigreed cats, it was found that the ratio of clinically significant difference in group mean values to the standard deviation should be at least 0.9 [11]. Then, at a statistical significance level of 0.05 and a power of 0.80, the minimum sample size should be at least 20 in both experimental and control groups. The study included cats with hypertrophic cardiomyopathy complicated by hepatocardial syndrome ( $n = 28$ ) and those free from hepatocardial complications ( $n = 55$ ). Healthy cats ( $n = 20$ ) of the same age and body weight were used as a control group. Clinical research methods were carried out according to the standard method [6, 11, 12]. The indicators of respiratory function were assessed [2]. High-resolution tonometry was carried out on a PetMAP graphic II [13]. Mean arterial pressure was determined according to the generally accepted method [6]. Electrocardiographic diagnostics was performed on EK1T-04 Midas [2]. Echocardiographic research methods were performed using a Mindray DP-60 scanner with a P10–4E probe [7]. Biochemical studies of blood sera were conducted on a Stat Fax 1904 Plus using standard biochemical kits [4, 8]. The concentration of cardiac troponin in blood serum was determined on the analyzer Architect i2000 by chemiluminescent

immunoassay on microparticles [2, 8]. The severity of congestive left ventricular circulatory failure was assessed by the size of pulmonary vein (PV) and the right branch of pulmonary artery (RBPA) [10]. The characteristic of heart remodeling was described according to a unified method [13, 14]. Both transverse and longitudinal contractility of the left and right chambers of heart were assessed [15, 16]. Mathematical processing was carried out using STATISTICA 7.0 software [17]. The Mann — Whitney test was applied, the standard deviation (SD) and 95 % confidence interval (95 % confidence interval — 95 % CI) were calculated [18, 19].

### Research results and discussion

The experiment included 83 cats with hypertrophic cardiomyopathy. The prevalence of hepatocardial syndrome in cats with hypertrophic cardiomyopathy was 33.7 %. Clinical indicators of different groups of animals are given in Table 1.

Table 1

**Clinical parameters in cats with feline hypertrophic cardiomyopathy complicated by hepatocardial syndrome**

Index	Healthy cats (n = 20)		Cats with hypertrophic cardiomyopathy			
			Free from hepatocardial syndrome (n=55)		Hepatocardial syndrome (n=28)	
	M±SD	95 % CI	M±SD	95 % CI	M±SD	95 % CI
Temperature, °C	38.5±0.3	38.3...38.6	38.1±0.8	37.9...38.3	37.8±0.9**	37.4...38.1
Pulse, min <sup>-1</sup>	171.4±12.4	165...177	187.2±17.8***	182...191	200.3±19.6*** ##	192...207
Respiratory rate, min <sup>-1</sup>	32.2±3.0	30.8...33.6	40.9±14.4*	37.0...44.8	54.5±20.0*** ##	46.8...62.3
Sleeping respiratory rate, min <sup>-1</sup>	17.9±1.8	16.9... 18.7	25.7±7.8***	23.6...27.7	33.3±9.3*** ###	29.6...36.9
CRT, s	1.3±0.4	1.1...1.4	1.8±0.7**	1.6...2,0	2.1±0.7***	1.8...2.4
SBP, mmHg.	160.0±8.6	156...164	154.9±24.9	148...161	147.2±28.9	136...158
DBP, mmHg.	84.1±10.9	79...89	82.9±17.6	78...87	76.8±25.4	67...87
MAP, mmHg.	109.3±9.3	105...114	107.2±19.1	102...112	100.2±9.3*	90...110

Note: CRT – capillary refill time; SBP – systolic blood pressure; DBP – diastolic blood pressure; MAP – mean arterial pressure; \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001 – significance of the difference between the indices of the experimental groups of animals and clinically healthy ones (Mann – Whitney test); #p < 0.05, ##p < 0.01, ###p < 0.001 – significance of the difference between the indices of experimental groups of animals (Mann – Whitney test).

According to the data in Table 1, compared with healthy animals, cats with hypertrophic cardiomyopathy without complications in the form of hepatocardial syndrome had a significant increase in: pulse rate (1.09 times; p < 0.001), respiratory rate at the clinic appointment (1.27 times; p < 0.05) and during sleep (1.43 times, p < 0.001), time of refilling of capillaries with blood (TRCB; 1.38 times; p < 0.01). Compared with intact animals, cats with hypertrophic cardiomyopathy and hepatocardial syndrome had the following features: significant decrease in rectal body temperature (0.7 °C; p < 0.01) and

mean arterial pressure (MAP; by 9.1 mm Hg;  $p < 0.05$ ), significant increase in heart rate (1.17 times;  $p < 0.001$ ), respiratory rate during examination in the clinic (1.7 times;  $p < 0.001$ ) and during sleep (1.86 times;  $p < 0.001$ ), TRCB (1.62 times;  $p < 0.001$ ). In sick cats with hepatocardial syndrome, a significant increase in heart rate, breathing during sleep and at the clinic, TRCB was revealed compared with animals free of such complications.

Table 2 shows the results of electrocardiographic studies in cats with hepatocardial complications against the background of primary pathology in the form of hypertrophic cardiomyopathy.

Table 2

### Electrocardiographic parameters in cats with hypertrophic cardiomyopathy complicated by hepatocardial syndrome

Index	Healthy cats (n = 20)		Cats with hypertrophic cardiomyopathy			
			Free from hepatocardial syndrome (n = 55)		Hepatocardial syndrome (n = 28)	
	M±SD	95 % CI	M±SD	95 % CI	M±SD	95 % CI
P, ms	35.8±3.4	34.1...37.4	34.3±3.5	33.4...35.3	33.0±3.5**	31.7...34.4
PQ, ms	64.9±9.9	60.2...69.5	58.8±11.8*	55.6...62.0	57,9±9.9**	54,0...61.7
QRS, ms	38.1±4.7	35.9...40.3	44.2±9.6**	41.7...46.8	49.3±7.7*** #	46.3...52.2
QT, ms	157.5±18.6	149...166	163.4±16.7	159...168	168.9±17.2*	162...176
PII, mV	0.13±0.05	0.10...0.15	0.12±0.05	0.11...0.14	0.17±0.06* ##	0.14...0.19
RII, mV	0.41±0.17	0.33...0.49	0.59±0.33*	0.49...0.68	0.68±0.27***	0.58...0.78
SII, mV	0.03±0.04	0.01...0.05	0.05±0.06	0.03...0.06	0.06±0.08	0.03...0.09
ST, mV	0.01±0.02	-0.01...0.02	-0.01±0.05	-0.02...0.01	-0.01±0.07	-0.04...0.02
T, mV	0.15±0.06	0.12...0.18	0.19±0.08	0.17...0.21	0.16±0.07	0.13...0.18

Note: P is the width of the atrial complex, PQ is the atrioventricular conduction parameter, QRS is the width of the ventricular complex, QT is the indicator of the electrical systole of the ventricles of the heart, PII, RII, SII, TII is the voltage of the P, R, S, T waves in the II standard electrocardiographic lead; ST – deviation of the ST segment from the zero line; \* $p < 0.05$ , \*\* $p < 0.01$ , \*\*\* $p < 0.001$  – significance of the difference between the indices of the experimental groups of animals and clinically healthy ones (Mann – Whitney test); # $p < 0.05$ , ## $p < 0.01$ , ### $p < 0.001$  – significance of the difference between the indices of experimental groups of animals (Mann – Whitney test).

Compared to healthy animals, electrocardiograms of cats with hypertrophic cardiomyopathy (Table 2) without complications in the form of hepatocardial syndrome had a significant decrease in the duration of the PQ interval, an increase in the duration of the QRS complex, R wave voltage. Compared with healthy animals, electrocardiograms of cats with hypertrophic cardiomyopathy and hepatocardial syndrome revealed a significant decrease in the duration of atrial complex (P wave) and atrioventricular conduction time (PQ interval), a significant increase in the time of electrical systole of heart ventricles (QT interval), atrial voltage (PII) and ventricular complex (RII). Hepatocardial complications led to more significant changes in QRS and PII in electrocardiograms of cats with hypertrophic cardiomyopathy.

The results of studies on changes in the main echocardiographic parameters are given in Table 3.

**Echocardiographic parameters in cats with feline hypertrophic cardiomyopathy complicated by hepatocardial syndrome**

Index	Healthy cats (n = 20)		Cats with hypertrophic cardiomyopathy			
			Free from hepatocardial syndrome (n=55)		Hepatocardial syndrome (n=28)	
	M±SD	95 % CI	M±SD	95 % CI	M±SD	95 % CI
PV, sm	3.3±0.9	2.9...3.7	5.6±2.1***	4.8...6.3	8.0±2.1*** ###	7.2...8.8
RBPA, sm	4.2±1.1	3.7...4.7	4.9±1.0	4.6...5.2	5.5±0.6** #	5.2...5.7
Ao, sm	0.91±0.11	0.85...0.96	0.98±0.11	0.98...1.01	0.99±0.13	0.94...1.04
LA, sm	0.94±0.42	0.74...1.13	1.64±0.49***	1.51...1.77	1.95±0.53*** ###	1.75...2.16
IVSd, sm	0.39±0.07	0,36...0,43	0.71±0.12***	0.68...0.74	0.81±0.11*** ###	0.76...0.85
IVSs, sm	0.69±0.08	0.64...0.72	1.14±0.21***	1,08...1,19	1.30±0.19*** ###	1.23...1.37
FWLVd, sm	0.37±0.06	0.34...0.40	0.71±0.10***	0.69...0.74	0.75±0.06*** ###	0.73...0.77
FWLVs, sm	0.65±0.07	0.61...0.69	1.19±0.19***	1.14...1.24	1.37±0.19*** ###	1.29...1.44
EDS, sm	1.49±0.12	1.44...1.54	1.44±0.16	1.40...1.49	1.50±0.18	1.43...1.57
ESS, sm	0.73±0.08	0.68...0.76	0.59±0.13***	0.55...0.64	0.56±0.09***	0.53...0.60
FS,%	51.3±5.9	48.5...54.0	58.6±11.0**	55.6...61.6	61.7±8.1*** ##	58.5...64.8
TAPSE, sm	9.5±1.0	9.0...9.9	7.8±1.5***	7.4...8.2	6.9±1.1*** ###	6.5...7.4
MAPSE (fwlv), sm	5.4±1.2	4.8...5.9	4.4±1.2**	4.1...4.8	3,8±0,8*** ###	3,5...4.1
MAPSE (ivs), sm	5.3±0.9	4.8...5.7	4.3±0.9**	4.1...4.6	3.7±0.9*** ###	3.3...4.0

Note: PV – pulmonary vein; RBPA – right branch of the pulmonary artery; LA – left atrium; Ao – aorta; IVSd, IVSs – interventricular septum in diastole and systole; FWLVd, FWLVs – free wall of the left ventricle in diastole and systole; EDS, ESS – end-diastolic and end-systolic size of the chamber of the left ventricle; FS – fraction shortening; TAPSE, MAPSE (fwlv) and MAPSE (ivs) – amplitude of systolic movement of the fibrous ring of the tricuspid, mitral valve in the projection of the free wall of the left ventricle and interventricular septum; \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001 – significance of the difference between the indices of the experimental groups of animals and clinically healthy ones (Mann – Whitney test); #p < 0.05, ##p < 0.01, ###p < 0.001 – significance of the difference between the indices of experimental groups of animals (Mann – Whitney test).

Compared with a group of clinically healthy animals, echocardiograms of cats with hypertrophic cardiomyopathy without hepatocardiac complications (Table 3) showed a significant increase in PV, LA, IVSd, IVSs, FWLVd, FWLVs, FS, a significant decrease in ESS, TAPSE, MAPSE(fwlv) and MAPSE(ivs). In cats with hepatocardial syndrome, which arose as a complication of hypertrophic cardiomyopathy, compared with healthy animals, there was a significant increase in PV, RBPA, LA, IVSd, IVSs, FWLVd, FWLVs, FS, a significant decrease in ESS, TAPSE, MAPSE(fwlv) and MAPSE(ivs). It should be noted that the fact of development of hepatocardial complications in sick cats induced more significant venous congestion in pulmonary vein system (significant increase in PV), dilatation of the left atrium (significant increase in LA), concentric hypertrophy

of the left ventricle (significant increase in IVSd, IVSs, FWLVd, FWLVs), increased transverse contractility of the myocardium of the left ventricle (significant increase in FS), decreased longitudinal contractility of myocardium of the left (MAPSE (fwlv) and MAPSE(ivs)) and right ventricle (TAPSE).

Further studies established the biochemical parameters of blood serum of cats with hypertrophic cardiomyopathy depending on the presence of hepatocardial syndrome (Table 4).

Table 4

**Biochemical parameters of blood serum in cats with feline hypertrophic cardiomyopathy complicated by hepatocardial syndrome**

Index	Healthy cats n = 20		Cats with hypertrophic cardiomyopathy			
			Free from hepatocardial syndrome (n=55)		Hepatocardial syndrome (n=28)	
	M±SD	95 % CI	M±SD	95 % CI	M±SD	95 % CI
Total protein, g/l	68.4±6.6	65.3...71.5	69.7±6.7	65.3...71.5	71.0±5.1	69.0...72.9
Albumins, g/l	31.2±3.2	29.7...32.7	30.5±3.8	29.4...31.5	28.2±3.9** ##	26.6...29.7
ALT, U/l	50.6±12.0	44.9...56.2	59.2±22.2*	53.2...65.2	122.4±20.6*** ###	114.4...130.4
AST, U/l	29.7±7.6	26.1...33.2	68.4±41.5***	57.3...79.7	79.4±35.8*** ##	65.4...93.2
ALP, U/l	34.5±11.3	29.2...39.8	42.2±15.2*	38.0...46.5	49.6±24.5* #	40.1...59.2
Troponin, ng/ml	0.03±0.03	0.01...0.03	0.21±0.11***	0.17...0.23	0.25±0.10*** ###	0.22...0.29
Urea, mmol/l	6.8±1.7	6.0...7.6	9.6±4.7*	8.3...10.9	12.5±4.5*** ###	10.8...14,3
Creatinine, μmol/l	91.9±17.8	83.6...100.3	145.8±56.5***	130.5...161.1	189.2±80.3*** ###	158.1...220.4

Note: \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001 – significance of the difference between the indices of the experimental groups of animals and clinically healthy ones (Mann – Whitney test), #p < 0.05, ##p < 0.01, ###p < 0.001 – significance of the difference between the indices of experimental groups of animals (Mann – Whitney test).

Compared with the healthy animals, the blood serum of cats with hypertrophic cardiomyopathy without hepatocardial syndrome (Table 4) showed a significant increase in activity of ALT, AST and ALP, concentrations of troponin, urea and creatinine. Cats with hepatocardial syndrome, which arose as a complication of hypertrophic cardiomyopathy, had a significant increase in serum ALT, AST, ALP, serum concentrations of troponin, urea and creatinine, and a decrease in serum albumin concentrations compared with healthy animals. It should be noted that the presence of hepatocardial syndrome in sick cats was characterized by more significant biochemical changes.

### Conclusion

Hepatocardial syndrome in cats, which arose as a complication of hypertrophic cardiomyopathy, is characterized by a severe course, hypothermia, severe tachypnea, tachycardia, increased capillary refill time, slowed intraventricular conduction, increased voltage of atrial and ventricular complex on electrocardiograms, pulmonary vein dil-

atation, significant dilatation of the left atrium, extreme concentric hypertrophy of the left ventricle, increased transverse contractility of the left ventricular myocardium and decreased longitudinal contractility of the left and right ventricular myocardium, cardiomyocyte cytolysis syndrome, cholestasis, hypoalbuminemia. Hepatocardial complications in cats are a marker of more severe hypertrophic cardiomyopathy.

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## Особенности течения гепатокардиального синдрома у больных гипертрофической кардиомиопатией кошек

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**Аннотация.** Рассматриваются вопросы изменения клинических, лабораторных и инструментальных параметров у кошек при развитии гепатокардиального синдрома, который сформировался на фоне гипертрофической кардиомиопатии. Известно, что у высокопородистых кошек при развитии застойной сердечной недостаточности может развиваться и прогрессировать вторичная гепатопатия. Показано, что гепатокардиальный синдром встречается у 33,7 % кошек от общего числа больных гипертрофической кардиомиопатией ( $n = 83$ ). Установлено, что гепатокардиальные осложнения у кошек являются фактором риска более тяжелого течения гипертрофической кардиомиопатии. Гепатокардиальный синдром у больных гипертрофической кардиомиопатией кошек характеризуется тяжелой гипотермией, недостаточностью кровообращения и респираторной системы. У больных животных регистрировали повышение частоты дыхания во сне ( $33,3 \pm 9,3$  против  $17,9 \pm 1,8$  раз/мин;  $p < 0,001$ ). У домашних кошек при гепатокардиальном синдроме развивается снижение среднего



артериального давления крови ( $100,2 \pm 19,3$  против  $107,2 \pm 19,1$  мм рт. ст;  $p < 0,05$ ), синусовая тахикардия ( $200,3 \pm 19,6$  против  $187,2 \pm 17,8$  раз/мин;  $p < 0,001$ ), что приводит к достоверному снижению интервалов PQ ( $57,9 \pm 9,9$  против  $64,9 \pm 9,9$  мс;  $p < 0,001$ ) и QT ( $168,9 \pm 17,2$  против  $157,5 \pm 18,6$  мс;  $p < 0,001$ ). У больных кошек установлено существенное повышение времени обратного наполнения капилляров кровью, замедление внутривентрикулярной проводимости, повышение вольтажа желудочкового и предсердного комплекса на электрокардиограммах, расширение легочной вены, значительная дилатация левого предсердия, экстремальная концентрическая гипертрофия левого желудочка, повышение поперечной сократимости миокарда левого желудочка и снижение продольной контрактильности миокарда левого и правого желудочка, синдром цитолиза кардиомиоцитов, холестаза, а также гипоальбуминемия.

**Ключевые слова:** кардиология, гепатология, диагностика, сердечная недостаточность, гепатопатия

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