ANIMAL PATHOLOGY, MORPHOLOGY, PHYSIOLOGY, PHARMACOLOGY AND TOXICOLOGY

ПАТОЛОГИЯ ЖИВОТНЫХ, МОРФОЛОГИЯ, ФИЗИОЛОГИЯ, ФАРМАКОЛОГИЯ И ТОКСИКОЛОГИЯ

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Biochemical Parameters of Blood Serum in Cats with Nephrocardial Syndrome

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Abstract

Introduction. Nephrocardial syndrome (NCS) is a complex of interrelated disorders, which includes chronic kidney disease (CKD) resulting in development of secondary myocardial dysfunction and structural cardiac remodeling. CKD is one of the most common pathologies in older cats. Although cases of nephrocardial syndrome in cats have been described in the literature, its precise pathophysiological mechanisms are still poorly understood. Particularly insufficient are the scientific data regarding the role of systemic inflammation and protein and energy metabolism disorders related to NCS pathogenesis and progression. The aim of the study is to investigate the biochemical parameters of blood serum in cats with nephrocardial syndrome for identifying the specific patterns of disorders and developing the improved diagnostic and therapeutic techniques.

Materials and Methods. A retrospective analysis of 82 medical records of cats admitted to the Moscow veterinary clinics in the period of 2021 — 2025 was conducted. Two experimental groups of animals were formed (37 cats with NCS; 23 cats with CKD without cardiac complications) and one control group (22 healthy cats). Research methods included serum biochemistry (total protein, albumin/globulin fractions, C-reactive protein, azotemia and ketonemia markers), as well as instrumental methods (echocardiography, ECG).

Results. In cats with NCS, pronounced dysproteinemia was revealed: hypoalbuminemia (28.1±0.5 g/l versus 34.2±0.6 g/l in the control group), hyperglobulinemia (42.6±1.4 g/l), a significant decrease in the albumin to globulin ratio (0.69±0.03 versus 1.02±0.07 in the control group). The level of C-reactive protein in animals with NCS exceeded the values of both healthy cats (164.1±5.6 g/l versus 82.9±1.4 g/l) and the cats from the group with CKD without cardiac complications (128.5±3.2 g/l), which confirms the presence of systemic inflammation. Azotemia parameters (urea, creatinine, SDMA) did not significantly differ in groups with CKD and NCS, but were higher, compared to the control group.

Discussion and Conclusion. The increased ketogenesis was detected in cats with both pathologies, which is likely due to uremic intoxication and catabolic processes. NCS in cats is characterized by pronounced systemic inflammation, impaired protein metabolism, and metabolic shifts. The obtained results emphasize the importance of integrating inflammatory and metabolic markers interpretation into the diagnostics of NCS, as well as the need for further research to establish the cause-and-effect relationships within the nephrocardial continuum.

Keywords: nephrocardial syndrome, diagnostics, biochemical parameters, secondary cardiomyopathy, pathogenesis, cats, chronic kidney disease

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Оригинальное эмпирическое исследование

Биохимические параметры сыворотки крови у кошек с нефрокардиальным синдромом

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Аннотация

Введение. Нефрокардиальный синдром (НКС) — это комплекс взаимосвязанных нарушений, при котором хроническая болезнь почек (ХБП) приводит к развитию вторичной миокардиальной дисфункции и структурному ремоделированию сердца. ХБП является одним из наиболее распространенных патологических состояний у кошек старшего возраста, и хотя случаи развития у кошек нефрокардиального синдрома описаны в литературе, его точные патофизиологические механизмы остаются не до конца изученными. В частности, недостаточно научных данных, касающихся роли системного воспаления, нарушений белкового и энергетического обмена в патогенезе и прогрессировании НКС. Целью настоящего исследования явилось комплексное изучение биохимических параметров сыворотки крови у кошек с нефрокардиальным синдромом для выявления специфических паттернов нарушений и разработки усовершенствованных методов диагностики и терапии.

Материалы и методы. В ходе ретроспективного анализа изучены 82 истории болезни кошек, поступивших на лечение в московские ветеринарные клиники за период с 2021 по 2025 гг. Было сформировано две опытные группы животных (37 кошек с НКС; 23 кошки с ХБП без кардиальных осложнений) и одна контрольная (22 здоровые кошки). Методы исследования включали биохимический анализ сыворотки крови (общий белок, фракции альбуминов/глобулинов, С-реактивный белок, маркеры азотемии и кетонемии), а также инструментальные методы (эхокардиография, ЭКГ).

Результаты исследования. У кошек с НКС выявлена выраженная диспротеинемия: гипоальбуминемия (28,1±0,5 г/л против 34,2±0,6 г/л в контроле), гиперглобулинемия (42,6±1,4 г/л), значительное снижение альбумин-глобулинового коэффициента (0,69±0,03 против 1,02±0,07 в контроле). Уровень С-реактивного белка у животных с НКС превышал показатели как здоровых кошек (164,1±5,6 г/л против 82,9±1,4 г/л), так и группы с ХБП без кардиальных осложнений (128,5±3,2 г/л), что подтверждает наличие системного воспаления. Параметры азотемии (мочевина, креатинин, СДМА) не имели значимых различий между группами с ХБП и НКС, но были повышены относительно контроля.

Обсуждение и заключение. Обнаружен усиленный кетогенез у кошек с обеими патологиями, вероятно, связанный с уремической интоксикацией и катаболическими процессами. НКС у кошек характеризуется выраженным системным воспалением, нарушением белкового обмена и метаболическими сдвигами. Полученные результаты подчёркивают важность интеграции оценки воспалительных и метаболических маркеров в диагностику НКС, а также необходимость дальнейших исследований для установления причинно-следственных связей в рамках нефрокардиального континуума.

Ключевые слова: нефрокардиальный синдром, диагностика, биохимические параметры, вторичная кардиомиопатия, патогенез, кошки, хроническая почечная недостаточность

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Introduction. Chronic kidney disease (CKD) is a widespread, progressive disorder in older cats that significantly increases the risk of their death [1]. Often, CKD becomes a background for developing comorbidities including hypertension, liver damage, hyperparathyroidism, electrolyte disturbances and anaemia [2, 3]. Due to advances in veterinary nephrology, many cats now live longer, however, renal dysfunctions can lead to development of secondary cardiac damages [4–7]. This condition is known as nephrocardial syndrome (NCS) — a combination of CKD and secondary cardiomyopathy [8, 9]. Many studies ascertain the crucial role of systemic inflammation in progression of both renal and cardiac failures in animals [10–12]. Cardiomyopathies and valvular heart diseases are often associated with elevated inflammatory markers (C-reactive protein, fibrinogen, cytokines), as well as with changes in blood values such as neutrophilia and elevated ESR [13–15]. Similar changes are observed in cats with CKD.

Another important factor is neurohormonal activation (involving Renin-Angiotensin-Aldosterone System, Sympathetic Nervous System): high level of angiotensin II increases oxidative stress, inflammation and cell death, which accelerates kidney and cardiac damage [9]. Furthermore, pharmacotherapy itself can contribute to the development of nephrocardial and cardiorenal syndromes [16]. For example, loop diuretics (furosemide, torasemide) can cause hypovolemia and impair renal blood flow, while simultaneously activating the Renin-Angiotensin-Aldosterone System, which aggravates damage to renal and cardiac tissues [17, 18].

Cases of cardiac damage caused by renal dysfunctions in cats described in the literature do not provide a complete understanding of the mechanism of syndrome development. In particular, the role of systemic inflammation and protein-energy metabolism disorders in the pathogenesis of NCS remains unclear. Identification of specific biochemical markers can reveal the key mechanisms of syndrome development. The aim of the study is to investigate the profile of biochemical parameters (protein metabolism, markers of inflammation, azotemia and ketonemia) in the blood serum of cats with nephrocardial syndrome to identify specific patterns of disorders that would become a basis for development of a comprehensive approach to early diagnostics and efficient treatment of this comorbidity.

Materials and Methods. Data for the retrospective analysis were collected from 82 medical records of cats admitted to Moscow veterinary clinics (Vetlife, Epiona, and Zooacademy) in the period from 2021 to 2025. Two experimental groups (37 cats with NCS; 23 cats with CKD without cardiac complications) and one control group (22 healthy cats) of animals were formed.

Exclusion criteria for all groups were: acute kidney injury; prerenal or postrenal azotemia; infectious, toxic or obstructive nephropathies (including pyelonephritis); acute inflammatory processes; pathologies of the liver and pancreas, oncological diseases; FeLV/FIV virus infection carrier state; platelet aggregation that might distort haematological analysis.

The main experimental group (NCS, n=37) consisted of cats that met the following diagnostic criteria: CKD stages III–IV according to the IRIS classification (mean value 3.4±0.1); proven cardiomyopathy manifested in concentric left ventricular hypertrophy and/or left atrial dilation; average age – 132.9±4.9 months.

The second experimental group (CKD without cardiac complications, n=23) consisted of animals meeting the following inclusion criteria: CKD stages III–IV according to IRIS classification (mean value 3.4±0.1) and absence of structural cardiac changes as follows from the results of echocardiography. The average age – 131.4±7.3 months.

The control group (n=22) included healthy cats (average age 126.1±6.5 months), which were selected based on the following criteria: absence of clinical deviations; normal laboratory parameters; negative tests for FeLV/FIV. These animals underwent routine preoperative examination or prophylactic medical examination.

Laboratory tests were used to form groups and verify the diagnosis. Haematological analysis was performed using a HEMAX 53 VET automated analyzer (B&E Biotechnology Co., Ltd, China) [1, 18]. The leukocyte count was determined in smears in compliance with the Romanovsky-Giemsa method. Serum biochemical parameters (urea, creatinine, total protein, and albumin) were determined using a CS-600B analyzer (Dirui Industrial Co., Ltd., China) [17]. Instrumental methods such as echocardiography and electrocardiography were also used [9].

Statistical analysis at the first research stage included Shapiro-Wilk test of normality (Shapiro-Wilk test). At the second stage, groups were compared using Student's t-test (for parametric data). Data were presented as M±m (mean value ± standard error). Statistical significance level was determined using the Mann-Whitney test and was considered significant at $p<0.05$.

Results. Examination of blood serum biochemical parameters reflecting protein metabolism is crucial for understanding the pathogenesis of feline nephrocardial syndrome (Table 1).

Analysis of protein metabolism and inflammatory markers in cats with nephrocardial syndrome is of particular practical importance for understanding the pathogenetic mechanisms underlying comorbid nephrocardial pathology. Regarding the parameters of the main protein fractions in sick animals, it is important to note the following: the concentration of total protein in the blood serum was significantly elevated in NCS compared to the CKD group. Concurrently, the serum albumin concentration in animals with both CKD and NCS was significantly reduced compared to the control group. Moreover, the intergroup significance for this biochemical parameter was quite high (NCS relative to CKD). Regarding the serum globulin, it is necessary to emphasize the presence of more pronounced hyperglobulinemia in animals with NCS compared to CKD. The A/G ratio in animals with CKD, compared to the healthy group, was not statistically different. However, this parameter significantly decreased in NCS group compared to both CKD and the control groups.

Analysis of globulin fractions showed that in cats with NCS, compared to cats with CKD, the concentration of α1-, α2-, and β-globulins in serum was significantly increased, whereas, γ-globulin concentration was sharply decreased.

Table 1

Biochemical parameters of blood serum in cats of three groups (n=82) showing protein metabolism

Parameter	Control (healthy cats, n=22)	Experimental groups (sick cats)		Statistical significance (Mann-Whitney test)		
		CKD (n=23)	NCS (n=37)	p1	p2	p3
Total protein, g/l	69.9±1.6	66.6±2.1	70.7±1.4	≤0,1	≤1	≤0,05
Albumins, g/l	34.2±0.6	31.6±0.7	28.1±0.5	≤0.05	≤0.001	≤0.001
Globulins, g/l	35.7±1.7	34.9±2.1	42.6±1.4	≤0.5	≤0.01	≤0.01
A/G ratio, units	1.02±0.07	0.97±0.06	0.69±0.03	≤1	≤0.001	≤0.001
Alpha-1 globulins, g/l	7.0±0.3	8.7±0.2	11.8±0.4	≤0.001	≤0.001	≤0.001
Alpha-2 globulins, g/l	7.1±0.2	8.6±0.2	10.6±0.2	≤0.001	≤0.001	≤0.001
Beta globulins, g/l	8.6±0.5	14.9±0.7	17.6±0.4	≤0.001	≤0.001	≤0.01
Gamma globulins, g/l	13.5±1.4	6.8±1.3	8.1±0.9	≤0.001	≤0.01	≤0.5
C-reactive protein, g/l	82.9±1.4	128.5±3.2	164.1±5.6	≤0.001	≤0.001	≤0.001

Note: CKD — chronic kidney disease;
NCS — nephrocardial syndrome;
p1 — between healthy cats and cats with chronic kidney disease;
p2 — between healthy cats and cats with NCS;
p3 — between cats with chronic kidney disease and NCS.

Similar changes, albeit to a lesser extent, were also observed in the CKD group compared to the control group. Such an inflammatory marker as C-reactive protein, was also measured in the serum of cats with NCS. This biochemical parameter was found to be elevated in cats with CKD (compared to control group). However, the greatest increase of C-reactive protein concentration in serum was observed in cats with NCS (compared to cats from control and CKD groups).

Thus, cats with NCS exhibit pronounced dysproteinemia, manifested in hypoalbuminemia, hyperalphaglobulinemia (especially of the α1 fraction), and significant increase of β-globulin concentration in serum. Therefore, a pronounced inflammatory component has been established in the pathogenesis and progression of NCS in cats. This statement is also confirmed by a sharp increase of C-reactive protein concentration in serum. The development of a pronounced systemic inflammatory response to renal and cardiac failures has been determined in other studies too. The degree of inflammatory response correlates with the severity of many chronic diseases. The obtained data indicate the presence of more pronounced systemic inflammation in domestic cats with NCS, presence of significant protein metabolism disorder and highly probable synergism of renal and cardiac damage. This requires a separate, detailed analysis to understand the pathogenetic mechanisms.

Data on the assessment of azotemia levels in sick cats are presented in Table 2.

Table 2

Assessment of azotemia parameters in cats of three groups (n=82)

Parameter	Control group (healthy cats, n=22)	Experimental groups (sick cats)		Statistical significance (Mann-Whitney test)		
		CKD (n=23)	NCS (n=37)	p1	p2	p3
Urea, mmol/l	6.8±0.3	28.9±1.1	29.9±0.6	≤0.001	≤0.001	≤1
Creatinine, μmol/l	104.0±8.4	356.6±11.0	390.4±13.1	≤0.001	≤0.001	≤0.1
Urea/ creatinine, units	81.8±12.3	83.6±4.8	79.9±3.2	≤0.1	≤0.1	≤0.5
SDMA, μg/dl	12.2±0.7	34.0±2.0	36.2±1.0	≤0.001	≤0.001	≤0.5

Note: CKD — chronic kidney disease;
NCS — nephrocardial syndrome;
SDMA — symmetric dimethylarginine;
p1 — between healthy cats and cats with chronic kidney disease;
p2 — between healthy cats and cats with NCS;
p3 — between cats with chronic kidney disease and NCS.

Urea, a biochemical parameter determining azotemia level, was significantly elevated in cats with both CKD and NCS (compared to the cats from control group). No intergroup differences in serum urea concentration were found between cats with CKD and NCS. Creatinine, the end product of creatine metabolism, is considered the most important biochemical parameter of azotemia level in small animals. A significant increase in serum creatinine levels was observed in cats with CKD and NCS (compared to cats from control group). Whereas, no differences were found between the CKD and NCS groups. The "urea/creatinine" ratio showed no statistically significant differences between the three groups of animals. SDMA, a marker of renal dysfunction, was statistically significantly elevated in animals with CKD and NCS (compared to animals from the control group). At the same time, this biochemical parameter did not differ between the experimental groups with pathologies (NCS vs. CKD).

When assessing metabolic changes in cats with CKD and NCS, it is important to consider the concentration of ketone bodies—intermediate products of lipid, fat, and carbohydrate metabolism. Significant ketonemia was found in cats with CKD (compared to cats from control group) and in cats with NCS (compared to cats from controls group) (Fig. 1). However, no intergroup differences were found in cats with pathologies (NCS vs. CKD).

Thus, the values of serum biochemical parameters in cats with CKD and NCS indicate that cardiac component does not have additional effect on azotemia levels. Significant metabolic disorders were identified: increased ketogenesis in both CKD and NCS conditions. Ketogenesis in cats with CKD was also noted in another study [17]. In cats with NCS, we observed a systemic inflammatory syndrome, manifested in dysproteinaemia. Serum SDMA concentration in cats with NCS confirms renal dysfunction but does not reflect involvement of cardiac pathophysiological mechanisms. Metabolic disorders (ketogenesis) may be associated with uremic intoxication, energy metabolism disorders, and underlying catabolic state.

Analysis of blood serum parameters such as total protein and its fractions, albumin to globulin ratio and protein-catabolic markers is particularly important for assessing severity of NCS in cats. These parameters clearly reflect impaired liver synthesis function, protein loss through damaged renal filters, the systemic inflammatory response, and metabolic changes associated with the development of nephrocardial continuum.

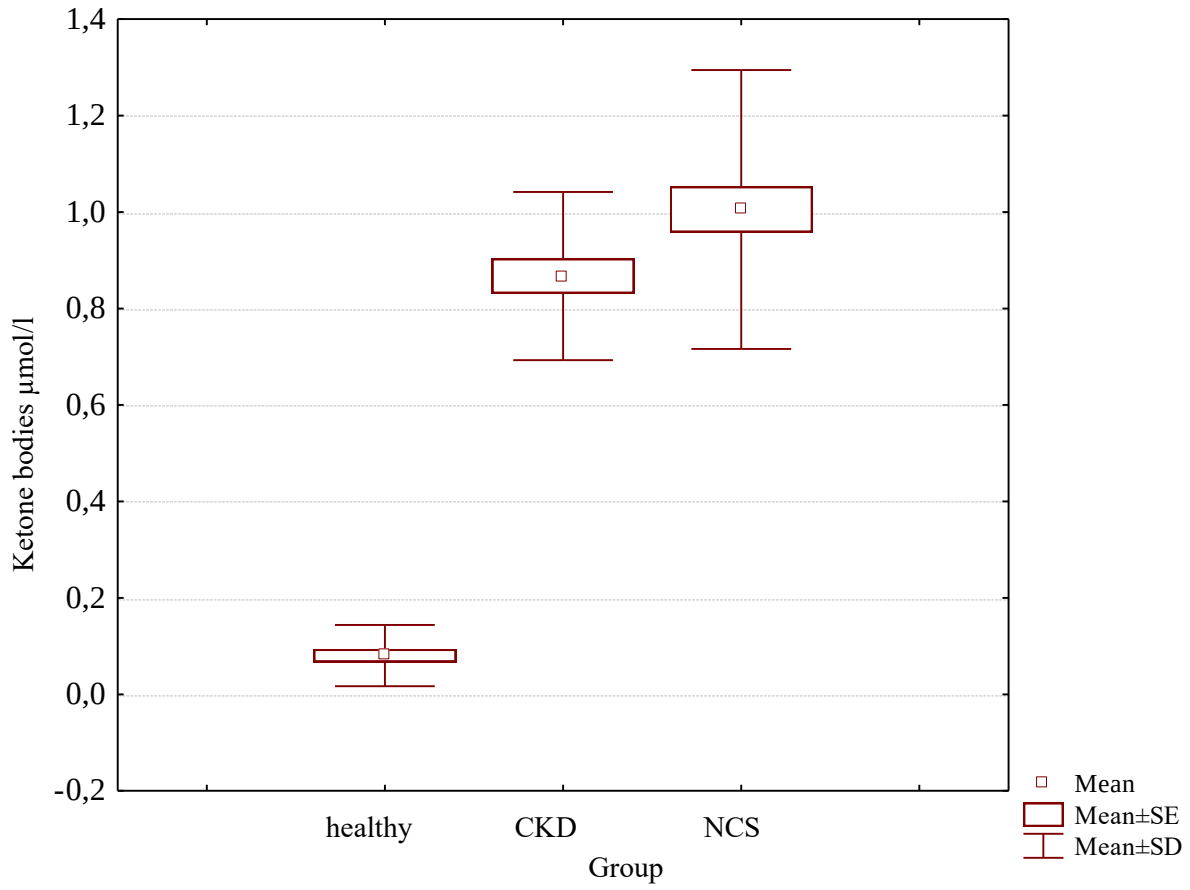

Fig. 1. Ketonemia in cats with nephrocardial syndrome

Discussion and Conclusion. The study revealed the main differences in biochemical parameters in cats with chronic kidney disease and nephrocardial syndrome, emphasizing the role of systemic inflammation and metabolic disorders. NCS in cats is pathogenetically characterized by a pronounced inflammatory component, manifested in dysproteinaemia (hypoalbuminemia, hyperalpha- and beta-globulinemia), as well as a sharp increase in serum C-reactive protein concentration. Azotemia parameters (urea, creatinine, SDMA) are significantly elevated in both pathologies but do not differ between the groups of cats with CKD and NCS. This suggests that the cardiac component of nephrocardial syndrome does not aggravate azotemia but increases the inflammatory response. Moderate ketonemia was verified in sick cats, which is related to the uremic intoxication and high catabolic state.

The results of the study emphasize the need for an integrated assessment of inflammatory and metabolic markers in animals suffering from nephrocardial syndrome, as well as require further research to determine cause-and-effect relationships in the nephrocardial continuum. This will help to improve diagnostic methods and therapeutic strategies aimed at controlling inflammation and metabolic imbalance.

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ED Sotnikova: developing methodology and editing the manuscript.
EA Notina: writing the manuscript, preparing a review, making translation.
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