УДК 616.12-008.46
DOI 10.17802/2306-1278-2026-15-3-200-210

КЛИНИЧЕСКИЕ ПРЕДИКТОРЫ ДОЛГОСРОЧНОЙ ВЫЖИВАЕМОСТИ ПРИ ХРОНИЧЕСКОЙ СЕРДЕЧНОЙ НЕДОСТАТОЧНОСТИ: РЕЗУЛЬТАТЫ 5-ЛЕТНЕГО ПРОСПЕКТИВНОГО КОГОРТНОГО ИССЛЕДОВАНИЯ

Н.В. Погосова[1, 2], Т.А. Тертерян[1, 2], А.А. Авагимян[3], А.А. Арутюнов[1], А.Б. Попова[1, 2], Н.М. Воробьёва[4], И.В. Осипова[5], Р.А. Жетишева[1, 2], А.К. Аушева[1, 2]

[1] Федеральное государственное бюджетное учреждение «Национальный медицинский исследовательский центр кардиологии имени академика Е.И. Чазова» Министерства здравоохранения Российской Федерации, ул. Академика Чазова, 15А, Москва, Российская Федерация, 121552; [2] Федеральное государственное автономное образовательное учреждение высшего образования «Российский университет дружбы народов имени Патриса Лумумбы», ул. Миклухо-Маклая, 6, Москва, Российская Федерация, 117198; [3] Ереванский государственный медицинский университет имени Мхитара Гераци, ул. Корюна, 2, Ереван, Республика Армения, 0025; [4] Федеральное государственное автономное образовательное учреждение высшего образования «Российский национальный исследовательский медицинский университет имени Н.И. Пирогова» Министерства здравоохранения Российской Федерации, ул. Островитянова, 1, стр. 6, Москва, Российская Федерация, 117513; [5] Федеральное государственное бюджетное образовательное учреждение высшего образования «Алтайский государственный медицинский университет» Министерства здравоохранения Российской Федерации, пр. Ленина, 40, Барнаул, Российская Федерация, 656038

Основные положения

• Хроническая сердечная недостаточность (ХСН) связана с высокими показателями смертности – они превышают таковые при онкологических заболеваниях. Традиционные ме-тоды оценки прогноза (основанные на этиологии ХСН и состоянии левого желудочка) недо-статочно точны, поэтому нужны новые предикторы неблагоприятного исхода.

• Тяжесть клинических проявлений и сопутствующие заболевания сильнее влияют на прогноз, чем исходная этиология ХСН.

• Исследование обосновывает переход от статической диагностики к динамической оценке симптоматического статуса пациентов. Акцент на коррекции сопутствующих заболе-ваний может улучшить результаты лечения ХСН в реальной клинической практике.

Актуальность	Хроническая сердечная недостаточность (ХСН) остается состоянием, связанным с высокими показателями смертности, превышающими показатели смертности при онкологической патологии. Традиционные подходы к стратификации риска, основанные на этиологии ХСН и аспектах функционирования левого желудочка, не в полной мере отражают прогноз, что диктует необходимость выявления новых клинически значимых предикторов не благоприятного прогноза.
Цель	Проведение комплексной оценки клинических симптомов, функционального класса (ФК) ХСН и сопутствующих заболеваний в отношении 5-летней смертности и выживаемости у пациентов с ХСН в условиях реальной клинической практики.
Материалы и методы	В данном проспективном когортном исследовании в феврале-мае 2018 г. были включены 150 пациентов с ХСН из амбулаторных клиник и больниц, расположенных в Москве и Барнауле. Информация о состоянии здоровья в течение 5-летнего периода наблюдения (медиана 3,29 года) была получена у 147 (98%) пациентов. Анализ выживаемости проводился с использованием метода Каплана–Мейера. Для оценки предикторов использовалась одномерная регрессия Кокса для расчета коэффициентов риска (HR) и 95% доверительных интервалов (CI).
Результаты	Общая 5-летняя выживаемость составила 59,9% (88/147). Этиология СН не показала статистически значимой связи с исходами. Гепатояремный рефлюкс ($p = 0,001$) и потеря веса > 4,5 кг в ответ на 5-дневную терапию ($p < 0,001$) были связаны с самыми высокими показателями смертности. Функциональный класс

Для корреспонденции: *Нана Вачиковна Погосова, nanapogosova@gmail.com; адрес: ул. Академика Чазова, 15А, Москва, Российская Федерация, 121552*

Corresponding author:*Nana V. Pogosova, nanapogosova@gmail.com; address: 15A, Akademika Chazova St., Moscow, Russian Federation, 121552*

	СН по NYHA продемонстрировал высокую прогностическую значимость: по сравнению с функциональным классом I риск смерти был в 7,7 раза выше у пациентов с функциональным классом III (95% ДИ 3,03–19,76) и в 19 раз выше у пациентов с функциональным классом IV (95% ДИ 4,50–80,29). Наличие сопутствующих заболеваний, включая анемию (ОР = 3,51; 95% ДИ 2,08–5,90), фибрилляцию предсердий (ОР = 2,12; 95% ДИ 1,25–3,61), хроническую обструктивную болезнь легких (ОР = 2,55; 95% ДИ 1,48–4,37) и обструктивное апноэ сна (ОР = 2,73; 95% ДИ 1,32–5,62), было связано с более высокой 5-летней смертностью. Наличие в анамнезе эндоваскулярных вмешательств выступало в качестве превентивного фактора (ОР = 0,44; 95% ДИ 0,24–0,81; p = 0,008).
Заключение	Тяжесть текущих клинических проявлений ХСН и совокупное бремя сопутствующих заболеваний оказывают большее влияние на долгосрочный прогноз, чем исходная этиология. Данные результаты подчеркивают необходимость перехода от статической диагностики к динамической оценке симптоматического статуса и активному воздействию на ключевые коморбидные заболевания для улучшения результатов лечения пациентов с ХСН в клинической практике.
Ключевые слова	Сердечная недостаточность • Исход • Анализ выживаемости • Смертность

Поступила в редакцию: *14.04.2026;* ***поступила после доработки:*** *13.05.2026;* ***принята к печати:*** *31.05.2026*

ОРИГИНАЛЬНЫЕ ИССЛЕДОВАНИЯ

CLINICAL PREDICTORS OF LONG-TERM SURVIVAL IN CHRONIC HEART FAILURE: RESULTS FROM A 5-YEAR PROSPECTIVE COHORT STUDY

N.V. Pogosova[1, 2], T.A. Terteryan[1, 2], A.A. Avagimyan[3], A.A. Arutyunov[1], A.B. Popova[1, 2], N.M. Vorobyeva[4], I.V. Osipova[5], R.A. Zhetisheva[1, 2], A.K. Ausheva[1, 2]

[1] Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, 15A, Akademika Chazova St., Moscow, Russian Federation, 121552; [2] Peoples' Friendship University of Russia named after Patrice Lumumba, 6, Miklukho-Maklaya St., Moscow, Russian Federation, 117198; [3] Yerevan State Medical University after Mkhitar Heratsi, 2, Koryun St, Yerevan, Republic of Armenia, 0025; [4] Federal State Autonomous Educational Institution of Higher Education «N.I. Pirogov Russian National Research Medical University» of the Ministry of Health of the Russian Federation, 1, bld. 6, Ostrovityanova St., Moscow, Russian Federation, 117513; [5] Altay State Medical University, 40, Lenin Ave., Barnaul, Russian Federation, 656038

Highlights

• Chronic heart failure (CHF) is associated with high mortality rates, which exceed those associated with cancer. Traditional methods for assessing the prognosis (based on the etiology of CHF and the condition of the left ventricle) are not accurate enough, so new predictors of an unfavorable outcome are needed.

• The severity of clinical manifestations and concomitant diseases has a stronger effect on the prognosis than the initial etiology of CHF.

• The study justifies the transition from static diagnosis to dynamic assessment of the symptomatic status of patients. The emphasis on the correction of concomitant diseases can improve the results of CHF treatment in real clinical practice.

Background	Chronic heart failure (HF) remains a condition associated with high mortality rates, exceeding those of oncological diseases. Traditional risk stratification approaches, based on the HF etiology and left ventricular function parameters, do not fully capture individual prognosis, necessitating the identification of new clinically significant predictors.
Aim	To conduct a comprehensive assessment of clinical symptoms, functional class (FC) of HF, and comorbid conditions in relation to 5-years mortality and survival in HF patients in real-world clinical practice settings.

Methods	In this prospective cohort study 150 consecutive patients with HF were enrolled in two outpatient clinics and two hospitals located in Moscow and Barnaul in February-May 2018. Vital status during 5-years of follow-up (median 3.29 years) was obtained in 147 (98%) patients. Survival analysis was performed using the Kaplan-Meier method. For assessment of predictors' univariate Cox regression was used to calculate hazard ratios (HR) and 95% coincidence intervals (CI).
Results	The 5-years overall survival rate was 59.9% (88/147). HF etiology showed no statistically significant association with outcomes. Hepatojugular reflux ($p = 0.001$) and weight loss > 4.5 kg in response to 5 days therapy ($p < 0.001$) were associated with the highest mortality rates. HF NYHA FC demonstrated strong prognostic significance: compared to FC I, the risk of death was 7.7 times higher for patients with FC III (95% CI 3.03–19.76) and 19 times higher for those with FC IV (95% CI 4.50–80.29). The presence of comorbidities, including anemia (HR = 3.51; 95% CI 2.08–5.90), atrial fibrillation (HR = 2.12; 95% CI 1.25–3.61), chronic obstructive pulmonary disease (HR = 2.55; 95% CI 1.48–4.37), and obstructive sleep apnea (HR = 2.73; 95% CI 1.32–5.62), were associated with higher 5-years mortality. A history of endovascular interventions acted as a protective factor (HR = 0.44; 95% CI 0.24–0.81; $p = 0.008$).
Conclusion	The severity of current clinical manifestations of HF and the cumulative burden of comorbid conditions has a greater impact on long-term prognosis than the initial etiology. These findings underscore the need to shift the focus from a static diagnosis towards dynamic assessment of symptomatic status and active targeting of key comorbidities to improve outcomes in patients with HF in clinical practice.

Keywords Heart failure • Outcome • Survival analysis • Mortality

Received: *14.04.2026;* ***received in revised form:*** *13.05.2026;* ***accepted:*** *31.05.2026*

Список сокращений

CKD – chronic kidney disease
COPD – chronic obstructive pulmonary disease
FC – functional class
HF – chronic heart failure
HR – hazard ratios
LVEF – left ventricular ejection fraction
NYHA – New York Heart Association
OSA – obstructive sleep apnea

Introduction

Chronic heart failure (HF) remains a leading cause of morbidity and mortality worldwide, with five-year survival rates that have improved only modestly despite major therapeutic advances in pharmacotherapy (angiotensin receptor–neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, sodium-glucose cotransporter-2 inhibitors) and device-based interventions (cardiac resynchronization therapy, implantable cardioverter-defibrillators) [1]. Contemporary risk stratification algorithms primarily rely on left ventricular ejection fraction (LVEF), HF etiology (ischemic versus non-ischemic), and select circulating biomarkers (natriuretic peptides) [2]. However, these static parameters incompletely capture the dynamic, multi-organ trajectory of HF progression, particularly the contributions of symptomatic status, physical signs of congestion, and the cumulative burden of comorbid conditions [3, 4].

Emerging evidence supports a paradigm shift toward a more holistic, phenotype-based approach to prognostication. Comorbidities such as anemia, chronic kidney disease (CKD), chronic obstructive pulmonary disease (COPD), and obstructive sleep apnea (OSA) are increasingly recognized not as passive epiphenomena but as active drivers of disease progression through overlapping pathophysiological cascades, including systemic inflammation, oxidative stress, neurohormonal activation, and mitochondrial dysfunction [5]. However, in clinical practice it is becoming evident that long-term outcomes of HF are significantly influenced not only by the initial cause of cardiac dysfunction but also by the severity of current clinical manifestations, concomitant and comorbid conditions [6].

Nevertheless, prospective data examining the independent prognostic value of these bedside findings, adjusted for guideline-directed medical therapy and LVEF, remain sparse, particularly in real-world cohorts outside Western Europe and North America. In this context the comparative contribution of specific clinical symptoms and the spectrum of comorbidities to mortality risk within the framework of prospective observation require further clarification, especially among population of Russian Federation.

This prospective cohort study was designed to comprehensively assess a wide range of clinical factors influencing five-year survival in patients with chronic

HF. The specific objectives were: (1) to determine the prognostic significance of individual clinical symptoms; (2) to evaluate the association between New York Heart Association (NYHA) functional class and long-term mortality; (3) to analyze the contribution of concomitant cardiovascular and non-cardiac diseases to clinical outcomes; and (4) to identify potential protective factors associated with improved survival.

Methods

This prospective cohort study was based on a registry. The study population consisted of 150 consecutive patients with chronic HF recruited from two outpatient clinics and two city hospitals in Moscow and Barnaul between February and May 2018. Patients were included either during routine outpatient visits or upon hospitalization for acute decompensation of HF. Exclusion criteria were: patient refusal to participate; severe renal, hepatic, or pulmonary insufficiency; and non-cardiovascular diseases with a potentially adverse impact on prognosis.

The study was conducted in accordance with the principles of the 1964 Helsinki Declaration and its later amendments as well as GCP guidelines. The study protocol was reviewed and approved by the Ethical Committee of the National Medical Research Center of Cardiology named after academician E. Chazov on 15.01.2018. All participants provided written informed consent.

At the enrollment visit, patients underwent a comprehensive baseline assessment, which included a detailed review of medical history, clinical examination, and evaluation of cardiovascular risk factors (smoking, excessive alcohol consumption, low physical activity, unhealthy diet, overweight/obesity, hypercholesterolemia, diabetes mellitus). Psychosocial risk factors were estimated in accordance with the current National Guidelines on Cardiovascular Prevention [6].

HF was diagnosed based on the presence of typical symptoms (e.g., breathlessness, fatigue) and signs (e.g., edema, crackles), supported by objective evidence of cardiac dysfunction (elevated NT-proBNP and/or echocardiographic abnormalities) in accordance with current Russian national HF Guidelines [7]. The HF functional class (FC) was determined using the New York Heart Association classification, depending on the degree of physical activity limitations and severity of symptoms: FC I – no chronic HF symptoms during usual physical activity, FC II – mild limitations of physical activity, FC III – severe limitations, FC IV – HF symptoms present at rest). Electrocardiographic parameters in 12 leads, transthoracic echocardiography and chest X-ray, complete blood count and biochemical blood tests (including potassium, sodium, creatinine, estimated glomerular filtration rate, urea, fasting glucose, and lipids) were evaluated. Hepatomegaly was determined by palpation and ultrasound. Anemia was diagnosed according to the WHO criteria: hemoglobin (Hb) levels < 130 g/L in men and < 120 g/L in women. Besides that, we assessed the level of adherence to previously recommended drug therapy (using the Morisky–Green-8 scale) and cognitive function (MoCA scale). Current publication focuses on the associations of clinical symptoms, HF NYHA FC and comorbidities in relation to 5-years outcomes.

To assess mortality and hospitalization rates, patients were contacted upon completion of follow-up to determine their vital status. Contact was made either directly with the patients themselves or with their relatives (spouse, children, and siblings). Vital status was recorded using a pre-designed questionnaire, which included information on vital status, as well as the date and cause of death. If neither the patient nor their relatives could be reached, information was obtained from the physician who had enrolled the patient in the study. However, since in some cases the vital status was established through communication, it was not possible to reliably ascertain the cause of death.

The statistical analyses were performed using SPSS software, version 23.0 (SPSS Inc., USA). The normality (Gaussian distribution) of quantitative and qualitative ordinal variables was not assessed; consequently, nonparametric statistical methods were employed. Missing values were not imputed. Quantitative and qualitative ordinal variables are presented as medians (25%; 75%), where the median, 25th percentile, and 75th percentile indicate the Me, Q1, and Q3, respectively; qualitative nominal variables are expressed as frequencies (%). For between-group comparisons, the Mann–Whitney U test, Pearson's chi-square test, and two-tailed Fisher's exact test were used. Survival analysis was conducted using the Kaplan–Meier method. Survival time is reported as M (95% CI), where M represents the mean and 95% CI – the 95% confidence interval. Survival curves were compared using the log-rank (Mantel–Cox) test. To evaluate the effect of psychosocial factors on mortality risk, a Cox proportional hazards model was applied, with calculation of hazard ratio (HR) and 95% CI, wherein death within 5 years of follow-up served as the dependent variable. Differences were considered statistically significant at a two-tailed p-value <0.05.

Results

The duration of prospective follow-up ranged from 0.02 years (7 days) to 5.09 years (median 3.29 years; interquartile range: 1.79 to 3.76 years). Outcomes were traced in 147 (98%) of the 150 patients enrolled in the study; contact was lost with 3 patients, and their outcomes are unknown. During the follow-up period, 59 (40.1%) patients died. Consequently, the 5-years survival rate was 59.9%.

To identify clinical factors (etiology of HF, duration, family history, symptoms and HF NYHA FC, as well as cardiovascular and concomitant diseases) associated with fatal outcomes, a comparative analysis was performed between deceased (n = 59) and survived (n = 88) patients during 5-years observation period.

The primary etiological factor of HF in patients included in the study was coronary artery disease, followed by arterial hypertension, non-rheumatic valvular heart disease, atrial fibrillation, rheumatic valvular heart disease, hypertrophic cardiomyopathy, and dilated cardiomyopathy (Figure 1). No statistically significant differences were identified regarding the etiology of HF between deceased and survived patients.

Family history of HF did not influence patients' survival. A positive family history was noted in 37.8% of deceased patients and in 32.1% of survivors (p = 0.519).

The duration of HF also had no impact on disease outcomes, the proportions of deceased and survived patients was very similar: in case of HF duration less than 3 months (5.4% vs. 6.8%; p = 1.0), 3–6 months (3.6% vs. 4.5%; p = 1.0), 6–12 months (5.4% vs. 10.2%; p = 0.368), 1–5 years (39.3% vs. 44.3%; p = 0.551), and more than 5 years (46.4% vs. 34.1%; p = 0.139).

Nearly all HF symptoms with the exception of dyspnea, acute pulmonary edema, and gallop rhythm, were detected significantly more frequently in deceased patients than among survivors (Table 1), i.e., their HF was characterized by a more severe course. Univariate regression analysis demonstrated that a number of clinical symptoms were associated with a 2.2 to 6.2-fold increase in the risk of death in HF patients during subsequent 5-years of follow-up (Table 1). The most prognostically unfavorable symptom was hepatojugular reflux and weight loss > 4.5 kg in response to therapy during 5 days. Among very important factors were also bilateral lower extremity edema and hepatomegaly. The hepatojugular reflux, jugular vein distension and weight loss > 4.5 kg in response to therapy are associated with the lowest survival rates and shortest survival time (Table 2).

The Figure 2 demonstrates adverse associations between HF NYHA FC and survival: the higher the FC, the lower the survival rate.

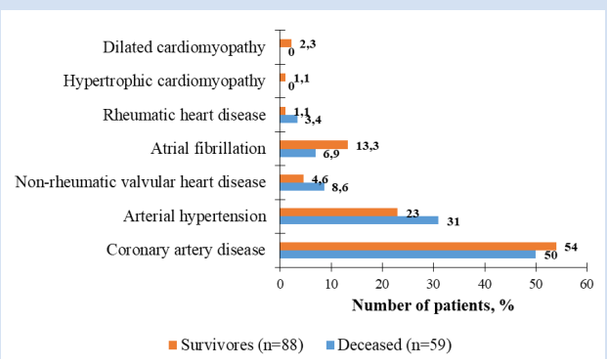

Figure 1. Etiology of HF depending on outcomes during 5-years follow-up (n = 145)

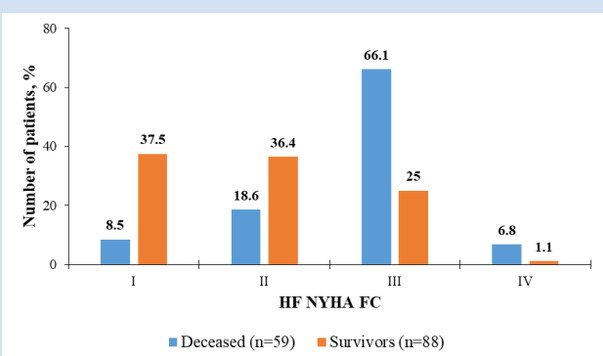

Figure 2. HF NYHA FC and outcomes during 5-years follow-up (n = 147)
Note: *FC – functional class; HF – heart failure; NYHA – New-York Heart Association.*

Table 1. Clinical symptoms of HF and their associations with risk of death during 5-years follow-up period (n = 147)

Symptoms of HF, n (%)	N	Deceased (n = 59)	Survived (n = 88)	p value	Cox regression*		
					HR	95% CI	p value
Dyspnea on usual physical exertion	146	58 (100%)	84 (95.5%)	0.152	–	–	–
Bilateral lower extremity edema	146	45 (76.3%)	35 (40.2%)	< 0.001	4.03	2.19–7.40	< 0.001
Hepatomegaly	145	36 (62.1%)	17 (19.5%)	< 0.001	4.36	2.55–7.46	< 0.001
Cardiomegaly (by chest X-ray)	140	35 (60.3%)	30 (36.6%)	0.005	2.19	1.29–3.73	0.004
Nocturnal cough	147	30 (50.8%)	25 (28.4%)	0.006	2.34	1.40–3.91	0.001
Weight loss > 4.5 kg during 5 days in response to therapy	137	29 (53.7%)	7 (8.4%)	< 0.001	6.12	3.53–10.61	< 0.001
Wet crackles in lungs	147	29 (49.2%)	14 (15.9%)	< 0.001	3.61	2.15–6.06	< 0.001
Paroxysmal nocturnal dyspnea	142	23 (42.6%)	14 (15.9%)	< 0.001	3.06	1.77–5.29	< 0.001
Pleural effusion	146	23 (39%)	8 (9.2%)	< 0.001	3.74	2.20–6.35	< 0.001
Jugular vein distension	146	11 (19%)	4 (4.5%)	0.005	3.71	1.90–7.25	< 0.001
Hepatojugular reflux	118	4 (9.5%)	0	0.015	6.19	2.18–17.60	0.001
Acute pulmonary edema	146	1 (1.7%)	1 (1.1%)	1.0	–	–	–
Gallop S3 sound	144	1 (1.7%)	1 (1.1%)	1.0	–	–	–

Note: ** dependent variable: death. HF – heart failure; HR – hazard ratio; CI – coincidence interval.*

Univariate regression analysis demonstrated that compared to patients with FC I (reference category with HR = 1.00), the 5-years mortality risk was 7.7 times higher in patients with FC III and 19 times higher in patients with FC IV (Table 3).

Univariate regression analysis, wherein HF NYHA FC was treated as a ranked (ordinal) variable, demonstrated that for each one-rank increase in FC (e.g., transition from FC II to FC III) the 5-years mortality risk increased nearly by 3 times (HR 2.94; 95% CI 2.05–4.22; p < 0.001).

Analysis of 5-years survival demonstrated that patients with HF NYHA FC III and IV exhibited significantly worse survival and shorter survival time compared to patients with FC I and II (Table 4).

As can be seen from Figure 3, the divergence of survival curves for patients with FC I–II and FC III–IV began almost immediately after the start of observation (the first patient died 7 days after enrollment in the study). Importantly, no patient with FC I–II died within the first year, whereas, in contrast, all deceased patients with FC IV died during the first year after enrollment.

Deceased patients with HF exhibited a higher prevalence of coronary artery disease, valvular heart disease, peripheral atherosclerosis, atrial fibrillation/flutter, COPD, CKD, OSA, and anemia, and a lower prevalence of a history of endovascular and surgical cardiac interventions (Table 5). None of the deceased patients had been vaccinated against influenza. Univariate regression analysis demonstrated that in HF patients with certain conditions (atrial fibrillation/flutter, anemia, valvular heart disease, peripheral

Table 3. Associations of HF NYHA FC and 5-years risk of death in HF patients according to univariate regression analysis (n = 147)

HF NYHA FC	N	HR	95% CI	p value
I	38	1.00*	–	–
II	43	2.04	0.71–5.87	0.188
III	61	7.73	3.03–19.76	< 0.001
IV	5	19.00	4.50–80.29	< 0.001

Note: ** reference category; dependent variable: death; CI – coincidence interval; FC – functional class; HF – heart failure; HR – hazard ratio; NYHA – New-York Heart Association.*

Table 4. Analysis of 5-years survival in HF patients depending on HF NYHA FC (n = 147)

HF NYHA FC	Survival, %	Survival time, years		Log-rank test	
		Mean	95% CI	Chi-square	p value
I	86.8	4.69	4.39–5.00	45.3	< 0.001
II	74.4	4.27	3.84–4.70		
III	36.1	2.23	1.82–2.65		
IV	20.0	0.95	0–2.14		

Note: *CI – coincidence interval; FC – functional class; HF – heart failure; NYHA – New-York Heart Association.*

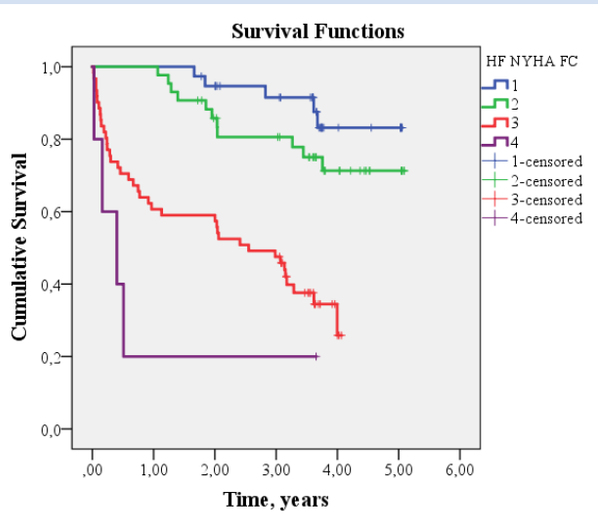

Figure 3. Five-years survival curves in HF patients depending on HF NYHA FC (n = 147)
Note: *CI – coincidence interval; FC – functional class; HF – heart failure; NYHA – New-York Heart Association.*

Table 2. Associations of HF symptoms and 5-years survival in HF patients (n = 147)

HF symptoms	N	Survival, %		Survival time, years*		Log-rank test indicator	
		Indicator (+)	Indicator (–)	Indicator (+)	Indicator (–)	Chi-square	p
Cardiomegaly	140	46.2	69.3	2.94 [2.44; 3.44]	3.96 [3.55; 4.36]	8.9	0.003
Nocturnal cough	147	45.5	68.5	2.86 [2.29; 3.43]	3.98 [3.63; 4.34]	11.1	0.001
Bilateral lower extremity edema	146	43.8	78.8	2.41 [2.04; 2.78]	4.44 [4.14; 4.75]	23.4	< 0.001
Paroxysmal nocturnal dyspnea	142	37.8	70.5	2.31 [1.79; 2.83]	4.03 [3.70; 4.36]	17.8	< 0.001
Wet crackles in lungs	147	32.6	71.2	2.12 [1.63; 2.60]	4.05 [3.72; 4.38]	26.6	< 0.001
Hepatomegaly	145	32.1	76.1	2.23 [1.74; 2.72]	4.25 [3.92; 4.57]	34.1	< 0.001
Jugular vein distension	146	26.7	64.1	1.45 [0.70; 2.19]	3.78 [3.46; 4.09]	17.0	< 0.001
Pleural effusion	146	25.8	68.7	1.93 [1.36; 2.51]	3.93 [3.61; 4.26]	27.3	< 0.001
Weight loss > 4.5 kg during 5 days in response to therapy	137	19.4	75.2	1.68 [1.19; 2.17]	4.23 [3.92; 4.54]	53.1	< 0.001
Hepatojugular reflux	118	0	66.7	1.28 [0; 2.72]	3.78 [3.43; 4.14]	15.3	< 0.001

Note: ** the results are presented as the mean value and 95% confidence interval. HF – heart failure.*

atherosclerosis, CKD, COPD, OSA) the 5-years mortality risk was 1.9 to 3.5 times higher than in individuals without these conditions (Table 5). Conversely, a history of endovascular interventions was associated with a 56% lower risk of death. It is important to note that no one in the deceased group had not received cardiac resynchronization therapy.

Analysis of 5-year survival in patients with HF showed that the lowest survival rates were observed in the presence of anemia (33.3%) and OSA (30.8%), whereas in patients with a history of endovascular interventions, the 5-year survival rate was conversely higher (72.5%) compared to patients who had not undergone such interventions (53.1%) (Table 6).

Discussion

Before discussing the main findings, the clinical profile of our cohort warrants a brief description [8]. Guideline-directed medical therapy was suboptimal: although beta-blockers and diuretics were prescribed

Table 5. Associations of cardiovascular and comorbid diseases with risk of death during 5-years follow-up in HF patients (n = 147)

Indicator, n (%)	N	Deceased (n = 59)	Survived (n = 88)	p value	Cox regression*		
					HR	95% CI	p value
Arterial hypertension	146	57 (96.6%)	76 (87.4%)	0.054	3.19	0.78–13.07	0.107
Coronary heart disease	141	56 (100%)	78 (91.8%)	**0.042**	22.02	0.15–3176.39	0.223
History of myocardial infarction	147	29 (49.2%)	49 (55.7%)	0.437	–	–	–
History of endovascular interventions	147	14 (23.7%)	37 (42%)	**0.022**	0.44	0.24–0.81	**0.008**
Type of prior endovascular intervention: Percutaneous coronary intervention Transcatheter aortic valve implantation Transcatheter mitral valve repair with MitraClip device implantation	51	 12 (85.7%) 1 (7.1%) 1 (7.1%)	 33 (89.2%) 3 (8.1%) 1 (2.7%)	0.765	–	–	–
History of cardiac surgery	147	2 (3.4%)	12 (13.6%)	**0.038**	0.27	0.07–1.11	0.069
Type of prior cardiac surgery: CABG Valve replacement CABG + valve replacement	14	 2 (100%) 0 0	 8 (66.7%) 3 (25%) 1 (8.3%)	1.0	–	–	–
Implanted devices	147	5 (8.5%)	9 (10.2%)	0.723	–	–	–
Type of implanted devices: Pacemaker ICD CRT	14	 5 (100%) 0 0	 6 (66.7%) 1 (11.1%) 2 (22.2%)	0.346	–	–	–
Heart valve disease	147	37 (62.7%)	27 (30.7%)	**< 0.001**	3.13	1.84–5.33	< 0.001
Type of valve disease: Rheumatic Non-rheumatic	63	 2 (5.6%) 34 (94.4%)	 2 (7.4%) 25 (92.6%)	0.765	–	–	–
History of stroke	147	8 (13.6%)	8 (9.1%)	0.394	–	–	–
History of transient ischemic attack	143	4 (7.1%)	6 (6.9%)	1.0	–	–	–
History of venous thromboembolic complications	144	4 (7%)	5 (5.7%)	0.740	–	–	–
Peripheral atherosclerosis	118	13 (29.5%)	10 (13.5%)	**0.034**	2.63	1.36–5.08	0.004
Atrial fibrillation/flutter	147	37 (62.7%)	37 (42%)	**0.014**	2.12	1.25–3.61	0.006
Chronic obstructive pulmonary disease	139	23 (41.8%)	16 (19%)	**0.003**	2.55	1.48–4.37	0.001
Obstructive sleep apnea	139	9 (16.4%)	4 (4.8%)	**0.022**	2.73	1.32–5.62	0.007
Anemia	141	28 (47.5%)	14 (17.1%)	**< 0.001**	3.51	2.08–5.90	< 0.001
Chronic kidney disease	146	38 (65.5%)	44 (50%)	0.064	1.90	1.10–3.28	0.021
Chronic kidney disease stage: 1 2 3a 3b 4 5	80	 0 5 (13.5%) 12 (32.4%) 10 (27%) 9 (24.3%) 1 (2.7%)	 1 (2.3%) 13 (30.2%) 14 (32.6%) 14 (32.6%) 1 (2.3%) 0	**0.030**	–	–	–
Type 2 diabetes mellitus	147	23 (39%)	26 (29.5%)	0.234	–	–	–
History of tuberculosis	147	2 (3.4%)	0	0.159	–	–	–
Acute infection in the past year	147	7 (11.9%)	5 (5.7%)	0.224	–	–	–
Influenza vaccination in the past year	147	0	7 (8%)	**0.042**	0.05	0.00–5.92	0.213

Note: ** dependent variable: death; CABG – coronary bypass grafting; CI – coincidence interval; CRT – cardiac resynchronization therapy; HR – hazard ratio; ICD – implantable cardioverter-defibrillator.*

frequently (87% and 79%, respectively), aldosterone antagonists were used in only 40% of patients and angiotensin receptor-neprilysin inhibitors in a mere 0.7%. The distribution of heart failure phenotypes according to LVEF was as follows: HF with preserved LVEF, 56.3%; HF with moderately reduced LVEF, 24.3%; and HF with reduced LVEF, 19.4%.

Against this clinical background, the results of our prospective study corroborate the concept that prognosis in heart failure is determined less by the initial etiology and more by the patient's current clinical and pathophysiological phenotype. The principal finding is that the severity of hemodynamic overload and the spectrum of comorbid pathology carry greater prognostic weight than a static diagnosis of ischemic or hypertensive heart disease, underscoring the systemic nature of the heart failure syndrome. This overarching observation is substantiated by three key lines of evidence, which we discuss in turn: the terminal significance of congestive symptoms, the synergism of extracardiac comorbidities, and the modifying role of revascularization.

1. Symptoms as manifestations of terminal pathophysiological cascades. The exceptionally high mortality rate observed in HF patients with hepatogugular reflux (0% survival) and weight loss > 4.5 kg in response to 5-days therapy (HR 6.12) requires consideration not merely as congestion, but as indicative of irreversible organ dysfunction. The symptoms of hepatogugular reflux and venous congestion serves as a clinical marker of critical venous overload and pulmonary hypertension [9]. From a pathophysiological standpoint, an elevation in central venous pressure > 15–20 mmHg leads to retrograde pressure transmission into the hepatic veins and sinusoids; this induces portal hypertension and liver fibrosis (cardiac cirrhosis). At a molecular level, venous congestion in the kidneys (rather than low cardiac output alone) is a key driver of cardiorenal syndrome, activating the production of pro-inflammatory cytokines (TNF-α, interleykin-6) within the glomerular endothelium and interstitium, leading to a progressive decline in glomerular filtration rate [10].

2. The etiology paradox and the role of comorbidities: synergism of vicious cycles. The lack of a statistically significant association between HF etiology (coronary artery disease vs. others) and outcomes can be explained by the contemporary concept of a "final common pathway". Irrespective of the primary dominant pathogenetic mechanism, late stages are dominated by non-specific remodeling processes: fibroblast activation mediated by TGF-β/Smad signaling pathway leading to accumulation of extracellular matrix, glucose uptake and insulin sensitivity problems, and cardiomyocyte dysfunction linked to impaired intracellular calcium homeostasis (dysregulation of ryanodine receptors RyR2) [11–16].

– *Anemia*: Anemia in HF is not merely a reduction in hemoglobin but rather a marker of complex interactions. We posit a key role for iron deficiency, which can exist without anemia, impairing mitochondrial cytochromes and reducing myocardial energy provision independently of oxygen delivery. Anemia exacerbates hypoxia, activating hypoxia-inducible factor (HIF-1α), leading to pathological angiogenesis, fibrosis, and further remodeling [17]. From clinical perspective the pathogenic cross-talk "anemia-COPD" should be considered as a relevant in real clinical practice [18].

– *Obstructive sleep apnea:* The contribution of obstructive sleep apnea extends beyond simple nocturnal hypoxemia. Each apneic episode induces a sharp negative intrathoracic pressure (increasing left ventricular afterload) and hypoxia, triggering a systemic ischemia-reperfusion response. This leads to a burst of reactive oxygen species generation by mitochondria and NADPH oxidase activation, exacerbating endothelial dysfunction and promoting atherogenesis [19].

– *Chronic obstructive pulmonary disease:* The link between COPD and HF is not solely explained by smoking. Systemic inflammation in COPD (elevated hs-C-reactive protein, fibrinogen, interleykin-6)

Table 6. Association of cardiovascular and comorbid diseases with 5-years survival in HF patients (n = 147)

Indicator	N	Survival rate, %		Survival time, years*		Log-rank test	
		Indicator (+)	Indicator (–)	Indicator (+)	Indicator (–)	Chi-square	p value
History of endovascular interventions	147	72.5	53.1	4.16 [3.72; 4.60]	3.19 [2.78; 3.61]	7.5	0.006
Chronic kidney disease	146	53.7	68.8	3.15 [2.69; 3.61]	4.02 [3.61; 4.43]	5.5	0.019
Atrial fibrillation / Atrial flutter	147	50.0	69.9	2.79 [2.37; 3.20]	4.04 [3.65; 4.42]	8.0	0.005
Chronic obstructive pulmonary disease	139	41.0	68.0	2.23 [1.69; 2.76]	3.94 [3.60; 4.29]	12.3	< 0.001
Peripheral atherosclerosis	118	43.5	67.4	2.25 [1.58; 2.93]	3.95 [3.60; 4.30]	8.9	0.003
Obstructive sleep apnea	139	30.8	63.5	2.08 [1.33; 2.84]	3.72 [3.38; 4.05]	8.0	0.005
Heart valve disease	147	42.2	73.5	2.47 [2.02; 2.93]	4.22 [3.89; 4.55]	19.6	< 0.001
Anemia	141	33.3	68.7	1.94 [1.43; 2.45]	4.03 [3.70; 4.36]	25.3	< 0.001

Note: ** the results are presented as the mean value and 95% confidence interval.*

potentiates myocardial stress [20]. Moreover, lung hyperinflation and pulmonary hypertension (remodeling of pulmonary arterioles) create a mechanical impediment to right ventricular ejection, which, in the setting of pre-existing left ventricular dysfunction, precipitates rapid biventricular decompensation.

3. Protective effect of revascularization. The observed protective effect of endovascular interventions (HR 0.44) is expected yet significant. Revascularization addresses the root cause of ischemia – substrate deficit [21]. This restores contractility in hibernating myocardium and reduces the risk of life-threatening arrhythmias by stabilizing the electrophysiological properties of cardiomyocyte membranes (via normalization of ion pump function). However, the extent to which this effect is attributable to the procedure itself, as opposed to patients selected for revascularization inherently possessing greater coronary reserve and fewer comorbidities (residual selection bias), remains unclear. Special attention requires extremely low usage of cardiac resynchronization therapy – only in 2 patients and no one in the deceased group.

Our findings are consistent with the results of other studies [7, 21–24] indicating that anemia, atrial fibrillation, CKD, COPD and OSA form mutually exacerbating pathophysiological "vicious cycles" with HF, potentiating disease progression.

Despite the demonstrated prognostic value of symptoms and comorbidities, this study leaves several critical questions unanswered, limiting the integration of its findings into rigid clinical algorithms. Thus, our study confirms that shifting from a static assessment of etiology to dynamic risk stratification based on physical signs and the comorbid profile allows for more precise identification of patients with the poorest prognosis. These clinical markers integrally reflect profound pathophysiological processes: systemic inflammation, mitochondrial dysfunction, and irreversible remodeling. However, to develop personalized treatment algorithms aimed at disrupting these "vicious cycles" (e.g., iron deficiency correction, CPAP therapy for OSA, nutritional support), further research incorporating biomarker data and contemporary pharmacotherapy into multifactorial models is essential.

Limitations

Several limitations should be considered when interpreting the present findings. First, although the sample size was adequate for univariate analysis and reflective of real-world clinical practice, it precluded comprehensive multivariable modeling to adjust for potential confounders and identify independent predictors. Accordingly, the possibility that some observed associations may be attributable to disease severity rather than direct causal relationships cannot be entirely excluded. Second, the lack of systematic data on guideline-directed medical therapy, including dosages of renin-angiotensin-aldosterone system inhibitors, beta-blockers, and sodium-glucose cotransporter-2 inhibitors, limits the ability to ascertain the extent to which the observed differences in mortality are related to comorbidity burden, treatment intensity, or their interaction. A detailed examination of pharmacotherapy, however, is beyond the scope of the present report and will be addressed separately. These considerations notwithstanding, the identified associations between readily ascertainable clinical signs, comorbid conditions, and long-term survival retain their relevance for clinical practice.

Conclusion

This prospective cohort study of 147 patients with chronic HF followed for a median of 3.3 years provides several clinically relevant observations. First, the severity of current clinical manifestations, particularly NYHA class III/IV, hepatojugular reflux, and weight loss > 4.5 kg in response to short-term therapy was more strongly associated with 5-year mortality than the initial HF etiology. Second, the cumulative burden of comorbidities (anemia, atrial fibrillation, COPD, OSA, and CKD) conferred a 2- to 3.5-fold increased risk of death in univariate analysis, underscoring that HF progresses as a multisystem syndrome rather than an isolated cardiac disorder. Third, a history of endovascular revascularization was associated with better survival, although this finding is susceptible to indication bias and residual confounding.

These results reinforce a fundamental shift in HF management: from static diagnostic labelling to dynamic, phenotype-based risk stratification. Bedside signs of congestion and end-organ dysfunction, often overlooked in favor of laboratory or imaging parameters carry powerful prognostic information and should be systematically incorporated into serial clinical assessments. Likewise, active screening and targeted treatment of comorbidities (e.g., iron repletion for anemia, continuous positive airway pressure for OSA, optimized COPD therapy) may interrupt the vicious cycles that accelerate HF progression.

Conflict of Interest

N.V. Pogosova declares no conflict of interest. T.A. Terteryan declares no conflict of interest. A.A. Avagimyan declares no conflict of interest. A.A. Arutyunov declares no conflict of interest. A.B. Popova declares no conflict of interest. N.M. Vorobyeva declares no conflict of interest. I.V. Osipova declares no conflict of interest. R.A. Zhetisheva declares no conflict of interest. A.K. Ausheva declares no conflict of interest.

Funding Sources

Authors declare no funding for the study.

Author Information Form

Pogosova Nana V., MD, PhD, Professor, Corresponding member of Russian Academy of Sciences, Deputy General Director for Scientific and Analytical Work and Preventive Cardiology, Head of the Laboratory of Preventive Cardiology, Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, Moscow, Russian Federation; Head of the Department of Evidence-Based Medicine, Institute of Medicine, Peoples' Friendship University of Russia named after Patrice Lumumba, Moscow, Russian Federation; **ORCID** 0000-0002-4165-804X

Terteryan Tatevik A., Cardiologist, Department of Cardiac Rehabilitation, Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, Moscow, Russian Federation; Assistant, Department of Evidence Based Medicine, Peoples' Friendship University of Russia named after Patrice Lumumba, Moscow, Russian Federation; **ORCID** 0000-0003-0702-661X

Avagimyan Ashot A., PhD, Assistant Professor, Department of Internal Diseases Propaedeutics, Yerevan State Medical University after Mkhitar Heratsi, Yerevan, Republic of Armenia; ORCID 0000-0002-5383-8355

Arutyunov Arthur A., Junior Research Fellow, Laboratory of Preventive Cardiology, Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, Moscow, Russian Federation; **ORCID** 0000-0003-1577-3623

Popova Anna B., PhD, Senior Research Fellow, Laboratory of Preventive Cardiology, Cardiologist, Department of Cardiac Rehabilitation, Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, Moscow, Russian Federation; Senior Lecturer, Department of Evidence Based Medicine, Peoples' Friendship University of Russia named after Patrice Lumumba, Moscow, Russian Federation; **ORCID** 0000-0003-4779-324X

Vorobyeva Natalya M., PhD, MD, Senior Research Fellow, Laboratory for Study of Geroprotectors and Clinical Trials, Russian Gerontology Clinical and Research Center, Federal State Autonomous Educational Institution of Higher Education "N.I. Pirogov Russian National Research Medical University" of the Ministry of Health of the Russian Federation, Moscow, Russian Federation; **ORCID** 0000-0002-6021-7864

Osipova Irina V., PhD, MD, Professor, Head of the Department of Faculty Therapy and Geriatrics, Department of Facultative Therapy and Occupational Diseases, Altay State Medical University, Barnaul, Russian Federation; **ORCID** 0000-0002-6845-6173

Zhetisheva Radima A., PhD, Research Fellow, Laboratory of Preventive Cardiology, Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, Moscow, Russian Federation; Assistant, Department of Evidence Based Medicine, Peoples' Friendship University of Russia named after Patrice Lumumba, Moscow, Russian Federation; **ORCID** 0000-0001-7211-1971

Ausheva Aza K., PhD, Leading Research Fellow, Laboratory of Preventive Cardiology; Head of the Cardiac Rehabilitation Department, Federal State Budgetary Institution "National Medical Research Center of Cardiology named after Academician E.I. Chazov" Ministry of Health of the Russian Federation, Moscow, Russian Federation; Associate Professor, Department of Evidence Based Medicine, Peoples' Friendship University of Russia named after Patrice Lumumba, Moscow, Russian Federation; **ORCID** 0000-0001-9794-7484

Author Contribution Statement

PNV – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

TTA – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

AAA – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

AAA – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

PAB – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

VNM – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

OIV – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

ZRA – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

AAK – contribution to the concept and design of the study, editing, approved final version, fully responsible for the content

СПИСОК ЛИТЕРАТУРЫ / REFERENCES

1. Liori S, Kapelios CJ, Savarese G, Filippatos G. Heart failure evidence update 2026. Heart Fail Rev. 2026 Mar 11;31(1):34. Doi: 10.1007/s10741-026-10609-3.

2. Bozkurt B, Ahmad T, Alexander K, et al; WRITING COMMITTEE MEMBERS. HF STATS 2024: Heart Failure Epidemiology and Outcomes Statistics an Updated 2024 Report from the Heart Failure Society of America. J Card Fail. 2025;31(1):66-116. Doi: 10.1016/j.cardfail.2024.07.001.

3. Madonna R, Cufaro MC, Gagliardi S, et al. Galectin-3 binding protein is upregulated in heart failure with preserved ejection fraction and associated with endothelial nitric oxide synthase deficiency. Geroscience. 2026. doi: 10.1007/s11357-026-02314-8.

4. Pogosova N.V., Boytsov S.A. Preventive Cardiology 2024: State of Problem Perspectives of Development. Kardiologiia. 2024;64(1):4-13. Doi: 10.18087/cardio.2024.1.n2636.

5. Liu JC, Cheng TH. Bridging Gaps in Heart Failure Science: Toward a More Integrated Future. Life (Basel). 2025;15(10):1613. doi: 10.3390/life15101613.

6. Boytsov S, Pogosova N, Ansheles A, et al Cardiovascular prevention 2022. Russian national guidelines. Russian Journal of Cardiology. 2023;28(5):5452. DOI: 10.15829/1560-4071-2023-5452.

7. Galyavich A, Tereshchenko S, Uskach T, et al. 2024 Clinical practice guidelines for Chronic heart failure. Russian Journal of Cardiology. 2024;29(11):6162. DOI: 10.15829/1560-4071-2024-6162.

8. Pogosova N, Terteryan T, Avagimyan A, et al. Clinical characteristics and five-year outcomes in a prospective Russian real-world heart failure cohort. Vascul Pharmacol. 2026;163:107612. doi: 10.1016/j.vph.2026.107612.

9. Pellicori P, Hunter D, Ei Khin H, Cleland J. How to diagnose and treat venous congestion in heart failure. Eur Heart J. 2024;45(15):1295-1297. doi: 10.1093/eurheartj/ehad883.

10. Ei Khin HH, Cuthbert JJ, Koratala A, et al. Imaging of Congestion in Cardio-renal Syndrome. Curr Heart Fail Rep. 2025;22(1):10. doi: 10.1007/s11897-025-00695-z.

11. Ng SM, Neubauer S, Rider OJ. Myocardial Metabolism in Heart Failure. Curr Heart Fail Rep. 2023;20(1):63-75. doi: 10.1007/s11897-023-00589-y.

12. Henry JA, Couch LS, Rider OJ. Myocardial Metabolism in Heart Failure with Preserved Ejection Fraction. J Clin Med. 2024;13(5):1195. doi: 10.3390/jcm13051195.

13. Avagimyan A, Fogacci F, Pogosova N, et al. Diabetic Cardiomyopathy: 2023 Update by the International Multidisciplinary Board of Experts. Curr Probl Cardiol. 2024;49(1 Pt A):102052. doi: 10.1016/j.cpcardiol.2023.102052.

14. Nagasaka R, Kim E, Ambrosy AP, Feinstein MJ. Targeting inflammation in heart failure: evolving insights and future directions from randomized clinical trials. Heart Fail Rev. 2025;30(6):1177-1190. doi: 10.1007/s10741-025-10538-7.

15. Huang K, Wu H, Xu X, et al. Identification of TGF-β-related genes in cardiac hypertrophy and heart failure based on single cell RNA sequencing. Aging (Albany NY). 2023;15(14):7187-7218. doi: 10.18632/aging.204901.

16. Redel-Traub G, Marx SO, Marks AR. Targeting Calcium Regulation for Heart Failure and Arrhythmia Therapeutics: A Critical Review. Circulation. 2025;152(13):957-970. doi: 10.1161/CIRCULATIONAHA.125.075150.

17. Gan T, Hu J, Liu W, et al. Causal Association Between Anemia and Cardiovascular Disease: A 2-Sample Bidirectional Mendelian Randomization Study. J Am Heart Assoc. 2023;12(12):e029689. doi: 10.1161/JAHA.123.029689.

18. Ibarra-Macia A, Garcia-Pachon E. Anemia in COPD and cardiovascular mortality. Respir Med. 2025;241:108084. doi: 10.1016/j.rmed.2025.108084.

19. Wróbel-Nowicka K, Wojciechowska C, Jacheć W, et al. The Role of Oxidative Stress and Inflammatory Parameters in Heart Failure. Medicina (Kaunas). 2024;60(5):760. doi: 10.3390/medicina60050760.

20. Magrì D, Fiori E, Agostoni P, et al; Working Group on Heart Failure of the Italian Society of Cardiology. Heart failure and chronic obstructive pulmonary disease. A combination not to be underestimated. Heart Fail Rev. 2025;30(6):1525-1538. doi: 10.1007/s10741-025-10566-3.

21. Lopaschuk GD, Karwi QG, Tian R, Wende AR, Abel ED. Cardiac Energy Metabolism in Heart Failure. Circ Res. 2021;128(10):1487-1513. doi: 10.1161/CIRCRESAHA.121.318241.

22. Loosen SH, Roderburg C, Curth O, et al. The spectrum of comorbidities at the initial diagnosis of heart failure a case control study. Sci Rep. 2022;12(1):2670. doi: 10.1038/s41598-022-06618-5.

23. Scholten M, Davidge J, Agvall B, Halling A. Comorbidities in heart failure patients that predict cardiovascular readmissions within 100 days-An observational study. PLoS One. 2024;19(1):e0296527. doi: 10.1371/journal.pone.0296527.

24. Givi M, Shafie D, Garakyaraghi M, et al. Patients Characteristics and Preliminary Outcomes of Heart Failure Registry in A Middle-Income Country: Persian Registry of Cardiovascular Disease/Heart Failure (PROVE/HF). Galen Med J. 2018;7:e1026. doi: 10.22086/gmj.v0i0.1026.

Для цитирования: *Погосова Н.В., Тертерян Т.А., Авагимян А.А., Арутюнов А.А., Попова А.Б., Воробьёва Н.М., Осипова И.В., Жетишева Р.А., Аушева А.К. Клинические предикторы долгосрочной выживаемости при хронической сердечной недостаточности: результаты 5-летнего проспективного когортного исследования. Комплексные проблемы сердечно-сосудистых заболеваний. 2026;15(3): 200-210. DOI: 10.17802/2306-1278-2026-15-3-200-210*

To cite: *Pogosova N.V. , Terteryan T.A. , Avagimyan A.A. , Arutyunov A.A. , Popova A.B. , Vorobyeva N.M. , Osipova I.V., Zhetisheva R.A. , Ausheva A.K. Clinical predictors of long-term survival in chronic heart failure: results from a 5-year prospective cohort study. Complex Issues of Cardiovascular Diseases. 2026;15(3): 200-210. DOI: 10.17802/2306-1278-2026-15-3-200-210*