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ORIGINAL PAPERS

ESTABLISHED INFLAMMATORY MARKERS AND CARDIOVASCULAR DISEASE WITH INCREASED INCIDENCE

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Abstract. In cardiovascular diseases with increased incidents such as acute coronary syndrome or heart failure, studying the associated or unrelated evolution of certain biochemical parameters is very important in the prognostic, diagnostic, predictive picture or in the development of modern therapies. The aim was to present some markers that are already established and used in inflammation. Two major categories of heart disease are presented, such as acute coronary syndrome and heart failure. For these, established, inexpensive, and commonly used markers such as ESR, CRP, or fibrinogen were selected, both in the specialized literature and through statistical determinations. Galectin-3 is presented, for which there are studies on its proinflammatory role in the atherogenic process and involvement in cardiac fibrosis.

Keywords: Biomarkers, Inflammation, Cardiovascular diseases.

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Introduction

Cardiovascular diseases are conditions with a very high incidence globally, the leading cause of morbidity and mortality (Blanda et al., 2020), constituting an important target for research, prevention, diagnosis, and prognosis methods, and for the development of modern effective therapies. Assessing cardiovascular risk in healthy individuals or patients remains a difficult problem (Hogas et al., 2017), as is the case with vascular risk factors, which include insulin resistance and hypertension, dyslipidemia (Rossi et al., 2021). The most prevalent and challenging medical conditions of cardiovascular diseases, remanins heart failure (HF) (De Boer et. al., 2009).

Chronic inflammation is an important healthcare problem; it can lead to fibrosis with loss of tissue architecture and subsequent organ failure. Tissue fibrosis represents the last common pathway of chronic tissue injury. (Klyosov et al., 2008, Lupu et al., 2023). However, chronic inflammation, damaged tissues, and organ failure are major causes of morbidity and mortality (El Nahas et al., 2005, Klyosov et al., 2008).

1. Inflammatory Biomarkers

In atherothrombosis, an important cause of cardiovascular mortality, inflammation plays a very important role. The process is described by Willerson et al., 2004. Monocytes are bound by leukocytes at the site of the developing vascular lesion at the beginning of the process. This occurs in response to oxidized low-density cholesterol (LDL-C), injury, or infection. These monocytes become macrophages as they ingest chemically modified lipids and lipoproteins, and fatty deposits begin to form. At the site of plaque detachment, there are macrophage cells, the dominant type of atherosclerotic inflammatory cells; all these inflammatory cells contribute to the formation of atherosclerotic plaque.

A number of different biomarkers were studied recently with the aim to help in prognostics and diagnostics of inflammation, metabolic and cardiovascular disease, in cancer (Buliga-Finis et al., 2025).

1.1. Galectin-3

An animal lectin that binds beta-galactoside, according to Lupu et al., 2022, Galectin-3 can be found intracellularly and extracellularly. Galectin-3 is a lectin that has different roles under normal and pathophysiological conditions. It exhibits great flexibility as a specific regulator of inflammation, as is the case in many biological systems. Many studies have shown that it can play an important role as a prognostic or diagnostic biomarker in various conditions in which inflammation is majorly involved, such as cardiovascular, renal, and hepatic diseases, cancer, autoimmune diseases, etc.

Galectin-3 as a biomarker is correlated with changes in the normal values of various specific markers of inflammation; for example, it is closely correlated with markers of oxidative stress. This confirms its participation in various pathogenic processes.

Extracellularly, it is predominantly found in the cytoplasm, but also in the nucleus (Bänfer et al., 2020). It is a regulator of various biological systems, including inflammation (Henderson et al., 2009), acting in different stages, from acute to chronic inflammation and tissue fibrogenesis. Galectins are expressed in many

inflammatory cells and can generate pro-inflammatory or anti-inflammatory responses, depending on the inflammatory environment (Brzački et al., 2025).

Galectin-3 has important role in physiological and pathological processes. It is a multifunctional protein linked to pathogenesis in different conditions. Various studies revealed the role of Galectin-3 in different conditions like cardiovascular diseases, atherosclerosis, metabolic diseases, renal and hepatic fibrosis, cancer (Lupu et al., 2021; Buliga-Finis et al., 2025).

This has been the basis for studies showing that Gal-3 inhibition could reduce inflammation and fibrosis, indicating it as a target for therapeutic development (Brzački et al., 2025).

It contributes to chronic inflammation and inflammatory bowel disease and liver disorders such as non-alcoholic fatty liver disease and liver fibrosis, according to a study by Brzački et al., 2025 on the potential biomarker of circulating and tissue Galectin-3 in gastrointestinal inflammation.

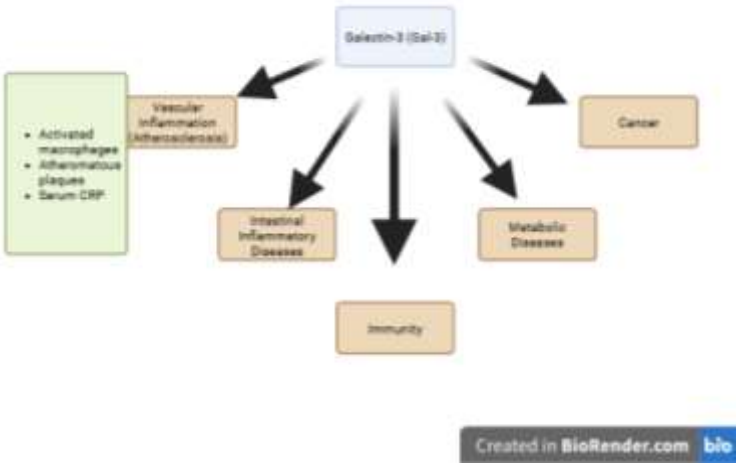

Figure 1. Galectin-3 and inflamatory linked diseases.

Galectins may play a role in tumor biology according to Liu et al., in 2005, in immunity or inflammation, and are therefore also studied in the context of cardiovascular disease development. They can modulate immune and inflammatory responses in cancer (Liu et al., 2005).

Galectin-3 (ng/mL) correlates with INR ($r = 0.490$, $p = 0.024 < \alpha = 0.05$). Correlation is positive, low to moderate, statistically significant. From the Pearson correlation profile analysis, a direct correlation is observed between Gal-3 and INR, CK-MB with K and TGO. This indicates a potential diagnostic or physiological adaptive remodeling index of the functional dynamics of the heart in patients with heart failure (Lupu et al., 2023).

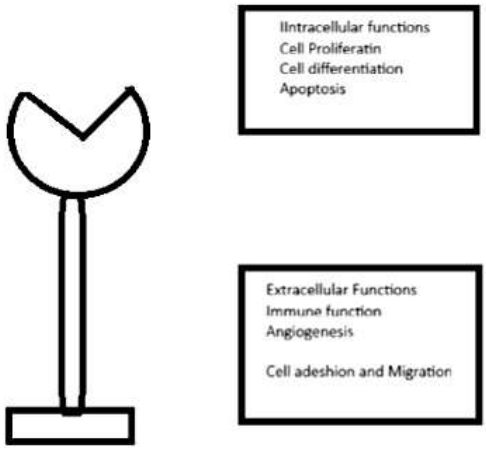

Figure 2. Galectin-3. Structure and main functions. (Adapted after Brzački V, Jovanović A, Rančić A, Tešić-Rajković S, Petrović G, Nagorni I, Stojanović M, Stanković E, Momčilović S. Circulating and Tissue Galectin-3 in Gastrointestinal Inflammation: Clinical Significance and Biomarker Potential. Cells. 2025)

1.2. Erythrocyte sedimentation rate (ESR)

Epidemiological and clinical studies have shown correlations between cardiovascular risk and inflammatory marker values. This has led to research aimed at gaining a deeper understanding of inflammation in atherogenesis and thrombosis, and thus to the development of modern therapies for treatment (Willerson et al., 2004). It is important to understand the mechanisms responsible for the initiation and progression of cardiac fibrosis (Kong et al., 2014).

Inngelsson et al., in 2005 studied ESR as a simple, established, inexpensive marker for inflammation in the further development of HF. Many studies have been conducted over the years on the importance of inflammation in HF, and their correlation and predictive value have been demonstrated. They conducted a prospective community study on a group of 2,314 middle-aged men without HF, MI, or valvular disease. The study results highlighted the significant predictive value of HF independent of established risk factors, i.e., hypertension, diabetes, obesity, and serum cholesterol.

1.3. C-Reactive Protein

According to Willerson et al., 2004, inflammation plays a role in the atherosclerotic process, whether in its early or advanced stages. The CRP value provides a method for determining this risk. These authors have shown that inflammation plays an essential role in cardiovascular disease, and elevated CRP levels are associated with increased risk.

1.4. Fibrinogen

It is a biomarker, an acute phase reactant, with elevated levels in inflammation or infection, but also in trauma, being a factor in coagulation.

In cardiovascular diseases, its values are a biomarker that can be associated with increased cardiovascular risk. It reflects the acute inflammatory response and may be elevated in acute coronary syndromes, also correlated with a higher risk of coronary events. It may also be elevated in HF, but the inflammation is chronic, not acute.

In a study published by Shi et al., 2010, fibrinogen levels measured in a cohort of 136 patients with acute coronary syndromes, 142 patients with stable coronary heart disease, and 82 healthy control participants were higher in patients with acute coronary syndrome than in the other two groups. High CRP levels were also a predictor of poor long-term prognosis (Shi et al., 2010).

2. Materials and methods

The review study used a search and analysis of literature to highlight the importance of inflammatory biomarkers in two of most important cardiovascular diseases. Established markers of inflammation in general were selected from the literature, then correlations were analyzed for a cohort of 45 patients, grouped into a group with acute coronary syndrome and a group of patients with heart failure, but also with secondary metabolic disorders, liver and kidney damage.

It reveals the link between inflammation in human diseases and the role of inflammation biomarkers in prognosis and diagnosis.

After correlations of inflammatory markers in groups of cardiovascular diseases, in further studies it could be useful to study and correlate with Gal-3.

Important markers such as troponin and CK-MB, which have different significance in ACS (SCA) compared to HF (ICC), were also presented.

3. Results and discussion

The study conducted on a group of patients with cardiovascular conditions such as acute coronary syndrome and heart failure revealed the following data for specific biochemical parameters such as troponin and Creatine kinase-MB.

Group Statistics

	Diagnostic	N	Mean	Std. Deviation	Std. Error Mean
Troponina T	ICC	32	43,9484	45,09375	7,97152
	SCA	12	425,1083	628,68443	181,48556

Troponin T levels were significantly higher in patients with acute coronary syndrome (ACS) compared to those with chronic heart failure (CHF).

The mean troponin was 425.1 ng/L in the ACS group and 43.9 ng/L in the CHF group, with a large variation in values in the acute setting (SD = 628.68 vs. 45.09).

These results reflect the acute, ischemic, and necrotic nature of myocardial damage in ACS, in contrast to the moderate and stable increases observed in CHF.

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Troponina T	Equal variances assumed	61,682	,000	-3,475	42	,001	-381,15990	109,69603	-602,53545	-159,78434
	Equal variances not assumed			-2,098	11,042	,060	-381,15990	181,66055	-780,8452	18,48473

Troponin T values were compared between patients with chronic heart failure (CHF) and those with acute coronary syndrome (ACS) using the t-test for independent samples.

The Levene test indicated unequal variances between groups ($p < 0.001$), so the results were interpreted for unequal variances.

The mean troponin T was significantly higher in the ACS group (425.1 ng/L) compared with the HF group (43.9 ng/L), with a mean difference of 381.2 ng/L.

Although the difference did not reach strict statistical significance ($t(11.042) = 2.098$; $p = 0.060$), the clinical variation is notable and reflects acute myocyte damage in an ischemic context.

Group Statistics

	Diagnostic	N	Mean	Std. Deviation	Std. Error Mean
CK-MB	ICC	32	35,91028	35,080620	6,201436
	SCA	12	66,31250	59,598956	17,204737

In the comparative analysis of serum CK-MB enzyme levels, mean values were higher in patients with acute coronary syndrome (66.31 ± 59.59 U/L) compared to those with chronic heart failure (35.91 ± 35.08 U/L).

Although there is a tendency for CK-MB to increase in ACS, the high dispersion of data indicates significant variability depending on the severity of myocardial ischemia and the time of sampling.

These results confirm the role of CK-MB as a useful enzymatic marker in the diagnosis of myocardial infarction, with significantly increased values in acute episodes compared to chronic compensatory cardiac conditions.

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
CK-MB	Equal variances assumed	2,232	,143	-2,095	42	,042	-30,402219	14,514702	-59,694073	-1,110365
	Equal variances not assumed			-1,662	13,960	,119	-30,402219	18,288269	-69,637099	8,832661

In the comparative analysis, the mean values of CK-MB isoenzyme were significantly higher in patients diagnosed with acute coronary syndrome (66.31 ± 59.59 U/L) than in those with chronic heart failure (35.91 ± 35.08 U/L).
The t-test for independent samples showed a statistically significant difference between the two groups (t = 2.095; p = 0.042), confirming the involvement of CK-MB as a marker of acute myocardial necrosis.
The results support the diagnostic value of CK-MB in the evaluation of patients with suspected acute coronary syndrome, compared to the nonspecific increases observed in chronic heart failure.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
ESR	,100	44	,200*	,919	44	,004

*. This is a lower bound of the true significance.
a. Lilliefors Significance Correction

Normality tests applied to ESR values (n=44) showed an abnormal distribution of data. Although the Kolmogorov–Smirnov test did not indicate a significant difference (p=0.200), the Shapiro–Wilk test, which is more sensitive for small

samples, showed a significant deviation from normality (p=0.004). Therefore, ESR shows an asymmetric distribution, which is why nonparametric tests were used in subsequent statistical analyses.

Ranks

	diagn_num	N	Mean Rank	Sum of Ranks
ESR	,00	12	27,29	327,50
	1,00	32	20,70	662,50
	Total	44		

ESR values were compared between patients with heart failure and those with ACS using the nonparametric Mann–Whitney U test.

The mean ESR rank was higher in the ACS group (27.29) than in the HF group (20.70), but the difference was not statistically significant ($p > 0.05$).

The result suggests that ESR levels do not differ significantly between patients with heart failure and those with ACS in the sample analyzed.

Test Statistics[a]

	ESR
Mann-Whitney U	134,500
Wilcoxon W	662,500
Z	-1,516
Asymp. Sig. (2-tailed)	,130
Exact Sig. [2*(1-tailed Sig.)]	,131[b]

a. Grouping Variable: diagn_num
b. Not corrected for ties.

Erythrocyte sedimentation rate (ESR) values were compared between patients with heart failure and patients with ACS using the nonparametric Mann–Whitney U test, due to the abnormal distribution of data.

The mean ESR rank was higher in the ACS group (27.29) compared to the HF group (20.70), without a statistically significant difference ($U = 134.5$; $Z = -1.516$; $p = 0.130$).

These results indicate a comparable level of inflammation between patients with ACS and those with heart failure in the study group.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
Fibrinogen	,086	44	,200*	,984	44	,776

*. This is a lower bound of the true significance.
a. Lilliefors Significance Correction

The Kolmogorov–Smirnov (p = 0.200) and Shapiro–Wilk (p = 0.776) normality tests indicated a normal distribution of fibrinogen values (n = 44). Therefore, parametric tests were used in subsequent comparative analyses.

Group Statistics

	diagn_num	N	Mean	Std. Deviation	Std. Error Mean
Fibrinogen	,00	12	480,92	79,454	22,936
	1,00	32	413,78	92,348	16,325

Fibrinogen values were compared between patients with heart failure and patients with ACS using the t-test for independent samples, as the distribution of the variable was normal.

The mean fibrinogen was higher in patients with ACS (480.9 ± 79.4 mg/dL) compared to those with HF (413.8 ± 92.3 mg/dL).

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Fibrinogen	Equal variances assumed	,639	,429	2,225	42	,032	67,135	30,178	6,234	128,037
	Equal variances not assumed			2,385	22,884	,026	67,135	28,153	8,880	125,391

Fibrinogen values were compared between patients with heart failure and patients with ACS using the t-test for independent samples, as the distribution of the variable was normal.

The Levene test confirmed the equality of variances (p = 0.429).

These results suggest more intense inflammatory activation in the group without heart failure, possibly in the context of other comorbidities.

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
PCR	,265	44	,000	,603	44	,000

a. Lilliefors Significance Correction

The Kolmogorov–Smirnov ($p < 0.001$) and Shapiro–Wilk ($p < 0.001$) tests showed an abnormal distribution of C-reactive protein (CRP) values. For this reason, nonparametric tests were used in subsequent comparative analyses.

Ranks

	diagn_num	N	Mean Rank	Sum of Ranks
PCR	,00	12	22,08	265,00
	1,00	32	22,66	725,00
	Total	44		

C-reactive protein (CRP) values were compared between patients with heart failure and patients with ACS using the nonparametric Mann–Whitney U test, given the abnormal distribution of data.

The mean CRP ranks were similar between the two groups (without HF = 22.08; with HF = 22.66), with no statistically significant difference (U = 187.0; Z = –0.132; p = 0.895).

These results suggest a comparable level of systemic inflammation between patients with heart failure and those with ACS in the study cohort.

Test Statistics[a]

	PCR
Mann-Whitney U	187,000
Wilcoxon W	265,000
Z	-,132
Asymp. Sig. (2-tailed)	,895
Exact Sig. [2*(1-tailed Sig.)]	,907[b]

a. Grouping Variable: diagn_num
b. Not corrected for ties.

C-reactive protein (CRP) values were compared between patients with heart failure and patients with ACS using the nonparametric Mann–Whitney U test, given the abnormal distribution of data.

The mean ranks were almost identical between the two groups (without HF = 22.08; with HF = 22.66), and the difference was not statistically significant (U = 187.0; Z = –0.132; p = 0.895).

These results indicate similar systemic inflammatory activation in patients with heart failure and those with ACS.

Varsta * diagn_num Crosstabulation

			diagn_num		
			,00	1,00	Total
Varsta	40-49	Count	0	3	3
		% within Varsta	0,0%	100,0%	100,0%
		% within diagn_num	0,0%	9,4%	6,8%
	50-59	Count	4	4	8
		% within Varsta	50,0%	50,0%	100,0%
		% within diagn_num	33,3%	12,5%	18,2%
	60-69	Count	5	11	16
		% within Varsta	31,3%	68,8%	100,0%
		% within diagn_num	41,7%	34,4%	36,4%
	70-79	Count	3	7	10
		% within Varsta	30,0%	70,0%	100,0%
		% within diagn_num	25,0%	21,9%	22,7%
	80-89	Count	0	7	7
		% within Varsta	0,0%	100,0%	100,0%
		% within diagn_num	0,0%	21,9%	15,9%
Total		Count	12	32	44
		% within Varsta	27,3%	72,7%	100,0%
		% within diagn_num	100,0%	100,0%	100,0%

The frequency of heart failure was analyzed according to age groups (40–49, 50–59, 60–69, 70–79, 80–89 years) using the Chi-square test.

The results showed a progressive increase in the proportion of patients with HF with age, from 50% in the 50–59 age group to 100% in those aged ≥80 years.

The Chi-square test confirmed a significant association between age and the presence of HF, suggesting that advancing age is a determining factor in the progression and staging of heart failure.

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	5,998[a]	4	,199
Likelihood Ratio	8,381	4	,079
Linear-by-Linear Association	1,228	1	,268
N of Valid Cases	44		

a. 6 cells (60,0%) have expected count less than 5. The minimum expected count is ,82.

The distribution of heart failure according to age was analyzed using the Chi-square test.

Although the proportion of patients with HF increased progressively with age (50% in the 50–59 age group, 68.8% in the 60–69 age group, and 100% in the ≥80 age group), the differences were not statistically significant ($\chi^2(4) = 5.998$; $p = 0.199$).

However, the Likelihood Ratio test indicated a trend toward significance ($p = 0.079$), suggesting that age may influence IC, but the current sample is insufficient for statistical confirmation.

Conclusions

1. The data indicate that the CVD approach is related to the approach to inflammation in acute or chronic cardiovascular diseases.
2. The study results confirm the data in the literature and have significant values in the groups studied.
3. Troponin T values were significantly higher in patients with acute coronary syndrome (ACS) compared to those with chronic heart failure (CHF).
4. The levels of the biochemical parameters analyzed can be used in the assessment and stratification of risk groups. The markers indicate a comparable inflammatory level between the group with acute coronary syndrome and the group with heart failure in the study cohort.

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