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# ASSOCIATION OF NOVEL SYSTEMIC INFLAMMATION INDICES AND CORONARY ARTERY DISEASE

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***Summary.***

*The role of inflammation in coronary artery disease (CAD) pathogenesis is well-established. Emerging evidence highlights associations between novel systemic inflammation indices and CAD. Established inflammatory markers including IL-6 and CRP also demonstrate significant relationships with CAD. Furthermore, the correlation between these biomarkers and coronary artery calcification merits particular investigation, given contradictory reports in current literature.*

***Materials and methods.*** *The study enrolled 112 patients with confirmed CAD aged 45-75 years and 42 control participants without CAD (control group). We analyzed clinical parameters, laboratory measurements (systemic inflammation indices, interleukin-6, high-sensitivity C-reactive protein (hs-CRP)), and coronary artery calcium (CAC) scores quantified by multislice computed tomography. All procedures were conducted in accordance with the World Medical Association's Declaration of Helsinki (2000 amendments). Statistical analyses were performed using GraphPad Prism version 10.1.1 (GraphPad Software, Inc., La Jolla, CA, USA) and Microsoft Excel 2013. The Shapiro-Wilk test was used to assess the normality of the data distribution. For normally distributed data, the results are presented as the mean (M) ± standard deviation (SD). For non-normally distributed quantitative data, the median (Me) with the upper and lower quartiles (Q1-Q3) was used. Student's t-test was applied to the statistical significance of differences between independent groups for normally distributed data. Categorical variables were compared using the χ2 test and Fisher's exact test. Correlation analysis was performed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.*

***Results.*** *CAD patients demonstrated significantly elevated levels across five key inflammation indices compared to controls: NLR (2.1 ± 1.2 vs. 1.5 ± 0.4, p=0.0006); MLR (0.19 ± 0.08 vs. 0.13 ± 0.04, p=0.0002); with most pronounced differences in composite indices: SIRI (0.76 ± 0.69 vs. 0.33 ± 0.12, p<0.0001); SII (527.7 ± 404.2 vs. 328 ± 101, p<0.0001); AISI (136.8 vs. 75.9, p=0.0001). Only hs-CRP showed moderate positive correlation with CAC (r=0.439). No other parameters demonstrated significant correlations.*

***Conclusion.*** *Incorporating systemic inflammation markers into clinical practice improves early detection, prognostic stratification, and personalized management of CAD.*

***Keywords:*** *Coronary Artery Disease; Inflammation; Systemic Inflammation Indices; Interleukin-6; High-Senstivity C-reactive Protein; Coronary Artery Calcium.*

## Introduction

Cardiovascular diseases (CVDs) remain the leading cause of death globally. Coronary artery disease (CAD) is responsible for approximately 16% of all deaths worldwide [1]. The underlying pathology of CAD is atherosclerosis of the coronary arteries – a chronic inflammatory process characterized by significant immunological activity that primarily affects the arterial wall. Inflammation plays a fundamental role in the pathogenesis of atherosclerosis. This is evidenced by inflammatory infiltration within atherosclerotic plaques and the frequent co-localization of inflammation with lipid accumulation in the arteries [2, 3]. Given the central role of chronic inflammation in CAD pathogenesis, pro-inflammatory markers are crucial for reflecting disease activity. Previous studies have established associations between the risk of atherosclerosis and elevated levels of fibrinogen, C-reactive protein (CRP), members of the interleukin family (interleukin-1, –3, –6, –8, –10), galectin-3, tumor necrosis factor-alpha (TNF-α), and other pro-inflammatory mediators. These substances promote endothelial dysfunction, drive the formation of atherosclerotic plaques, and contribute plaque destabilization [2-5].

Contemporary literature describes novel biomarkers of systemic inflammation, including the Systemic Inflammation Index (SII), the Systemic Inflammation Response Index (SIRI), and the Aggregate Inflammation Systemic Index (AISI). These are calculated indices derived from the differential counts of leukocytes and their subtypes. Their role in cardiovascular disease is now being actively investigated globally. Elevated levels of these indices have been correlated with the severity of coronary atherosclerosis, the presence of complex lesions, and increased risk of myocardial infarction. These indices could aid in identifying high-risk patients for whom more intensive anti-inflammatory and lipid-lowering therapies may be beneficial [6-10]. Recent clinical trials targeting inflammation, including the CANTOS study, established that reducing inflammation independently lowers cardiovascular event rates [11]. The latest regulatory approval of low-dose colchicine for the secondary prevention of coronary artery disease exemplifies the successful translation of anti-inflammatory therapy into clinical practice for patients with atherosclerotic disorders [12].

Numerous studies have demonstrated that elevated levels of inflammatory biomarkers, such as high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6), are independently associated with a higher coronary artery calcium (CAC) score, a key indicator of atherosclerotic burden [13]. For instance, data from large multi-ethnic cohort studies, including the Multi-Ethnic Study of Atherosclerosis (MESA), have established that individuals with elevated baseline inflammatory markers are more likely to have prevalent CAC and to exhibit faster CAC progression over time [14]. This relationship underscores the role of chronic

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inflammation not only in the initiation of atherosclerosis but also in the progression of calcified plaque. It is important to note, however, that inflammation and CAC, while related, capture different aspects of atherosclerotic disease. Inflammation is often linked to lipid-rich, rupture-prone plaques, whereas CAC quantifies the burden of stabilized, calcified disease. This pathophysiological distinction explains why individuals with elevated inflammatory markers despite a zero CAC score remain at elevated cardiovascular risk [15]. The complementary nature of these biomarkers renders their combination synergistic, yielding a more comprehensive risk stratification than either can provide alone.

The aim of our study is to compare levels of inflammatory biomarkers (IL-6, hs-CRP) and novel systemic inflammation indices (SIRI, SII, AISI) in patients with and without coronary artery disease (CAD) and to investigate the relationship between these markers and the CAC score in patients with established CAD.

**Material and methods.** The study included 112 patients, aged 45 to 75 years, with a verified diagnosis of CAD, identified during an inpatient examination at the Republican Specialized Scientific and Practical Medical Center of Cardiology. The control group included 42 individuals without a history or clinical evidence of CAD. Key exclusion criteria for all participants comprised a history of: malignancy; cardiothoracic surgery use of corticosteroids or other hormonal therapy within the preceding month; autoimmune disorders; infective endocarditis; or any major surgery within the past year. Patients with severe chronic kidney disease or hepatic failure were also excluded. All procedures were conducted in accordance with the World Medical Association's Declaration of Helsinki (2000 amendments).

The studied parameters included clinical data, laboratory and instrumental test results. Systemic inflammation indices were calculated from complete blood count differentials as follows:

Neutrophil-to-Lymphocyte Ratio (NLR) = Absolute Neutrophil Count / Absolute Lymphocyte Count

Platelet-to-Lymphocyte Ratio (PLR) = Absolute Platelet Count / Absolute Lymphocyte Count

Monocyte-to-Lymphocyte Ratio (MLR) = Absolute Monocyte Count / Absolute Lymphocyte Count

Neutrophil-to-Monocyte Ratio (NMR) = Absolute Neutrophil Count / Absolute Monocyte Count

Systemic Inflammation Response Index (SIRI) = (Neutrophil Count × Monocyte Count) / Lymphocyte Count

Systemic Inflammation Index (SII) = (Neutrophil Count × Platelet Count) / Lymphocyte Count

Aggregate Index of Systemic Inflammation (AISI) = (Neutrophil Count × Monocyte Count × Platelet Count) / Lymphocyte Count

Plasma levels of high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6) were measured in all participants. Coronary artery calcium (CAC) scoring was assessed via multispiral computed tomography (MSCT) of the chest.

Statistical analyses were performed using GraphPad Prism version 10.1.1 (GraphPad Software, Inc., La Jolla, CA, USA) and Microsoft Excel 2013. The Shapiro-Wilk test was used to assess the normality of the data distribution. For normally distributed data, the results are presented as the mean (M) ± standard deviation (SD). For non-normally distributed quantitative data, the median (Me) with the upper and lower quartiles (Q1-Q3) was used. Student's t-test was applied to the statistical significance of differences between independent groups for normally distributed data. Categorical variables were compared using the $\chi 2$ test and Fisher's exact test. Correlation analysis was performed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered statistically significant.

## Results

The mean age of the patients with CAD was 65 years (SD 7.9; range 45-75). The cohort included 68 (60.7%) female and 44 (39.3%) male (Table 1). The patient characteristics are summarized in Table 1.

**Table 1**

| Indicator | | Patients with CAD (n=112) | Control Group (n=42) | p |
|---|---|---|---|---|
| Age, years | | 65 ± 7.9 | 51.2 ± 3.8 | 0.349 |
| Sex | Male | 44 (39.3%) | 14 (33.3%) | 0.577 |
| | Female | 68 (60.7%) | 28 (66.7%) | |

Analysis of anthropometric parameters, risk factors and clinical characteristics in both groups is presented in Table 2. Arterial hypertension, dyslipidemia, type 2 diabetes mellitus, and obesity were significantly more common in the CAD group.

The results demonstrated that patients with CAD had statistically significantly higher levels of key inflammation indices compared to the control group: NLR (2.1 ± 1.2 vs. 1.5 ± 0.4, p=0.0006); MLR (0.19 ± 0.08 vs. 0.13 ± 0.04, p=0.0002). The most pronounced differences were observed for the composite indices: SIRI (0.76 ± 0.69 vs. 0.33 ± 0.12, p<0.0001); SII (527.7 ± 404.2 vs. 328 ± 101, p<0.0001); AISI (136.8 ± 150.5 vs. 75.9 ± 40.1, p=0.0001). In contrast, no statistically significant differences were found for PLR (p=0.062) or NMR (p=0.302).

In contrast to the haematological indices, levels of classic inflammatory biomarkers did not differ significantly between groups. IL-6 levels were similar in the CAD group (median 4 pg/ml) and the control group (3.9 ± 1.7 pg/mL), with p=0.133. hs-CRP levels also showed no significant difference, with a median of 1.8 mg/L in the CAD group compared to 2.1 ± 1.9 mg/L in controls (p=0.145).

**Table 2**

| Indicator | | Patients with CAD (n=112) | Control Group (n=42) | p |
|---|---|---|---|---|
| BMI, kg/m$^2$ | | 30.8 ± 4.4 | 26.1 ± 3.2 | 0.351 |
| WC, cm | Male | 106.4 ± 8.3 | 92.3 ± 6.4 | 0.036* |
| | Female | 91.7 ± 7.3 | 79.8 ± 5.4 | 0.046* |
| Arterial hypertension | | 110 (98.2%) | 14 (33.3%) | 0.0001* |
| Dyslipidemia | | 85 (75.9%) | 6 (14.3%) | 0.0001* |
| Type 2 diabetes mellitus | | 36 (32.1%) | 2 (4.8%) | 0.0003* |
| Glucose intolerance | | 12 (10.7%) | 4 (9.5%) | 1 |
| Obesity | | 64 (57.1%) | 7 (16.7%) | 0.0001* |
| Smoking | | 18 (16.1%) | 4 (9.5%) | 0.439 |
| Angina pectoris (FC) | II FC | 48 (42.9%) | - | |
| | III FC | 64 (57.1%) | - | |
| AH grade | Normal BP | 2 (1.8%) | 28 (66.7%) | |
| | Grade 1 | 69 (61.6%) | 10 (23.8%) | |
| | Grade 2 | 21 (18.8%) | - | |
| | Grade 3 | 2 (1.8%) | - | |
| | Achieved normotension | 18 (16%) | 4 (9.5%) | |
| Systolic BP, mmHg | | 140.7 ± 8.3 | 127.2 ± 4.1 | 0.707 |
| Diastolic BP, mmHg | | 82 ± 5.5 | 78.3 ± 5.2 | 0.41 |
| Duration of AH, years | | 15.4 ± 9.2 | 6.2 ± 2.3 | 0.116 |
| Duration of CAD, years | | 9.2 ± 4.3 | - | |
| Angina episodes per week | | 1.8± 0.4 | - | |

In contrast to the hematological indices, levels of classic inflammatory biomarkers did not differ significantly between groups. Interleukin-6 (IL-6) levels were similar in the CAD group (median 4.0 pg/mL) and the control group (3.9 ± 1.7 pg/mL; p=0.133). High-sensitivity C-reactive protein (hs-CRP) levels also showed no significant difference, with a median of 1.8 mg/L in the CAD group compared to 2.1 ± 1.9 mg/L in controls (p=0.145).

The levels of inflammatory markers and indices in the study participants are presented in Table 3.

**Table 3**

| Indicator | Patients with CAD (n=112) | Control Group (n=42) | p |
|---|---|---|---|
| Neutrophil-to-Lymphocyte ratio (NLR) | 2.1 ± 1.2 | 1.5 ± 0.4 | 0.0006* |
| Platelet-to-Lymphocyte ratio (PLR) | 140.4 ± 50.9 | 124.6 ± 33 | 0.062 |
| Monocyte-to-Lymphocyte ratio (MLR) | 0.19 ± 0.08 | 0.13± 0.04 | 0.0002* |
| Neutrophil-to-Monocyte Ratio (NMR) | 11.7 ± 5.3 | 12.5 ± 6.1 | 0.302 |
| Systemic Inflammation Response Index (SIRI) | 0.76 ± 0.69 | 0.33 ± 0.12 | <0.0001* |
| Systemic Inflammation Index (SII) | 527.7 ± 404.2 | 328 ± 101 | <0.0001* |
| Aggregate Index of Systemic Inflammation (AISI) | 136.8 (87.9-242.6) | 75.9 ± 23.4 | 0.0001* |
| Interleukin-6 (IL-6), pg/mL | 4 (3-6) | 3.9 ± 1.7 | 0.133 |
| High sensitivity C-reactive protein (hs-CRP), mg/L | 1.8 (1.2-3.5) | 2.1 ± 1.9 | 0.145 |

We next evaluated the relationship between inflammatory markers and systemic inflammation indices with CAC based on MSCT. The median CAC score in patients with CAD was 10.6 (interquartile range, 0-82.3). Analysis revealed a moderate positive correlation between hs-CRP and CAC (r=0.439). No other marker investigated in our study demonstrated a correlation.

## Discussion

Our study compared the levels of novel systemic inflammation markers (SIRI, SII, AISI) in patients with and without CAD. We found that these novel inflammatory indices were significantly higher in patients with established CAD.

Our findings are consistent with the global literature. For instance, a recent study reported significantly higher SIRI, SII and AISI values in patients with coronary and peripheral artery atherosclerosis compared to those without atherosclerosis [6].

Li Q, et al. (2022) studied patients with acute coronary syndrome and a history of percutaneous intervention. The authors assessed the prognostic significance of five inflammation indices (PLR, NLR, MLR, SII, and SIRI) for major adverse cardiac events (MACE). Cox multivariate analysis demonstrated that all five indices were independent predictors of MACE, with SIRI showing the strongest predictive performance [16].

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A large retrospective study by Xia Y, et al. (2023), which included 42,875 participants over a 20-year follow-up period, showed that individuals with an SII >655.56 had higher all-cause and CVD mortality than those with an SII <335.36. Similarly, participants with an SIRI >1.43 had a higher risk of all-cause and CVD mortality than patients with an SIRI <0.68. In the general population aged over 60 years of age, elevated SII or SIRI was associated with an increased risk of all-cause mortality [8].

The interaction between these inflammatory markers and CAC is of particular interest, as the stability of calcified plaque remains a subject of debate. In our study, none of the

novel inflammation indices correlated with the CAC score, whereas the hs-CRP demonstrated a moderate positive.

Evidence from a recent large-scale investigation confirms significant correlations between hs-CRP, galectin-3, and the prevalence of both coronary and extracoronary calcium in specific vascular beds [13]. These observations are consistent with prior research indicating generally modest associations between inflammatory markers and CAC, which frequently attenuate following adjustment for cardiovascular risk factors. The process of atherosclerotic plaque calcification is driven by inflammatory cytokines, including IL-6. Support for this mechanism comes from clinical cohorts such as the MESA, which demonstrated an independent relationship between inflammatory biomarker levels (hs-CRP, IL-6) and the presence and severity of CAC [14]. Furthermore, Mendelian randomization studies substantiate a causal relationship, linking genetic predisposition to elevated IL-6 with an increased risk of CAD [17]. The association between CAC and C-CRP remains inconsistent in the scientific literature. Although several studies report a correlation, others, including large meta-analyses, have found no such association [18, 19]. The present study identified a moderate positive correlation; however, this observation requires validation in larger, prospectively designed cohorts. The confluence of a high CAC score and elevated hs-CRP serves to identify patients at the greatest risk for future cardiovascular events, thereby affirming the clinical relevance of this relationship. Evidence that therapeutic targeting of inflammation attenuates the progression of coronary artery calcification provides definitive proof of its fundamental role in the pathogenesis of this process.

**Conclusion.** Integrating systemic inflammation markers into clinical practice enhances early detection, prognostic assessment, and personalized management of coronary artery disease.

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## ЗВ'ЯЗОК МІЖ НОВИМИ ІНДЕКСАМИ СИСТЕМНОГО ЗАПАЛЕННЯ ТА ІШЕМІЧНОЮ ХВОРОБОЮ СЕРЦЯ

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### Резюме.

Роль запалення в патогенезі ішемічної хвороби серця (ІХС) добре відома. Нові дані вказують на зв'язок між новими системними показниками запалення та ІХС. Відомі маркери запалення, включаючи ІЛ-6 та С-реактивний білок, також демонструють значущий зв'язок з ІХС. Крім того, кореляція між цими біомаркерами та кальцифікацією коронарних артерій заслуговує на особливе дослідження, зважаючи на суперечливі дані в сучасній літературі.

**Матеріали та методи.** У дослідженні взяли участь 112 пацієнтів з підтвердженою ІХС віком 45-75 років та 42 учасники без ІХС (контрольна група). Ми проаналізували клінічні параметри, лабораторні показники (показники системного запалення, інтерлейкін-6, високочутливий С-реактивний білок (hs-CRP)) та показники кальцифікації коронарних артерій (САС), визначені за допомогою багатозрізової комп'ютерної томографії. Усі процедури проводилися відповідно до Гельсінської декларації Всесвітньої медичної асоціації (поправки 2000 року). Статистичний аналіз проводився за допомогою програм GraphPad Prism версії 10.1.1 (GraphPad Software, Inc., Ла-Хоя, Каліфорнія, США) та Microsoft Excel 2013. Для оцінки нормальності розподілу даних використовувався тест Шапіро-Уілка. Для нормально розподілених даних результати представлені у вигляді середнього значення (M) ± стандартне відхилення (SD). Для кількісних даних з ненормальним розподілом використовувалася медіана (Me) з верхнім і нижнім квартилями (Q1-Q3). Для оцінки статистичної значущості відмінностей між незалежними групами для нормально розподілених даних застосовувався t-критерій Стьюдента. Категоріальні змінні порівнювалися за допомогою критерію $\chi 2$ та точного критерію Фішера. Кореляційний аналіз проводився за допомогою коефіцієнта кореляції Пірсона. Значення p менше 0,05 вважалося статистично значущим.

**Результати.** Пацієнти з ІХС демонстрували значно підвищені рівні п'яти ключових показників запалення порівняно з контрольною групою: NLR (2,1 ± 1,2 проти 1,5 ± 0,4, p = 0,0006); MLR (0,19 ± 0,08 проти 0,13 ± 0,04, p=0,0002); з найбільш вираженими відмінностями в комбінованих показниках: SIRI (0,76 ± 0,69 проти 0,33 ± 0,12, p<0,0001); SII (527,7 ± 404,2 проти 328 ± 101, p<0,0001); AISI (136,8 проти 75,9, p=0,0001). Тільки hs-CRP показав помірну позитивну кореляцію з САС (r=0,439). Жодні інші параметри не продемонстрували значущих кореляцій.

**Висновок.** Включення маркерів системного запалення в клінічну практику покращує раннє виявлення, прогностичну стратифікацію та персоналізоване лікування ІХС.

**Ключові слова:** ішемічна хвороба серця; запалення; показники системного запалення; інтерлейкін-6; високочутливий С-реактивний білок; кальцій коронарних артерій.

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