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Assessment of the inflammatory profile in acute coronary syndrome patients

Avaliação do perfil inflamatório em pacientes com síndrome coronariana aguda

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Abstract

Background: The systemic immunoinflammatory index (SII) is a novel prognostic biomarker used in certain studies to analyze the prognosis of certain types of cancers and heart diseases. Its main characteristic is being a rapid, inexpensive, and simple biomarker to use. It is based on the counts of lymphocytes (L), neutrophils (N), and platelets (P). The value of SII in patients diagnosed with Acute Coronary Syndrome (ACS) - ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA) - is not well established and could contribute to the risk stratification of these individuals. **Objectives:** This study aimed to analyze the value of SII in patients diagnosed with STEMI and compare it with those of patients with NSTEMI and UA. **Methods:** Information was collected through electronic medical record analysis and laboratory tests of patients diagnosed with Acute Coronary Syndrome (ACS) between July 2022 and July 2023. **Results:** Our sample included 333 patients, of whom 163 (48.9%) were diagnosed with STEMI, 139 (41.7%) with NSTEMI, and 31 (9.3%) with U. The STEMI group exhibited higher SII values compared to the NSTEMI and UA groups (1735 × 10^3 vs. 1167 × 10^3 vs. 1069 × 10^3, p < 0.001). **Conclusions:** Our study demonstrated that patients with STEMI exhibit higher SII values, suggesting greater inflammation and severity, compared to patients diagnosed with NSTEMI/UA.

Keywords: Inflammation; Acute Coronary Syndrome; Coronary Artery Disease.

Resumo

Introdução: O systemic immunoinflammatory index (SII), ou índice imunoinflamatório sistêmico, é um novo biomarcador utilizado para analisar o prognóstico de alguns tipos de cânceres e doenças cardíacas. Sua principal característica é ser um biomarcador rápido, barato e simples de ser utilizado. Baseia-se na contagem de linfócitos (L), neutrófilos (N) e plaquetas (P). O valor do SII nos pacientes com diagnóstico de Síndrome Coronária Aguda (SCA) - infarto agudo do miocárdio agudo do miocárdio com supra ST (IAMCSST), infarto agudo do miocárdio sem supra de ST (IAMSSST) e angina instável (AI) – não está bem estabelecido e poderia contribuir na estratificação de risco na SCA. **Objetivos:** Analisar e comparar o valor do SII em pacientes com diagnóstico de SCA (IAMCSST, IAMSSST e AI). **Métodos:** As informações foram colhidas através de análise de prontuário eletrônico e exames laboratoriais dos pacientes com diagnóstico de SCA entre julho de 2022 e julho de 2023.**Resultados:** Nossa amostra incluiu 333 pacientes, dos quais 163 (48,9%) foram diagnosticados com IAMCSST, 139 (41,7%) com IAMSSST e 31 (9,3%) com AI. Na amostra geral, a média do SII foi de 1436 x 10^3 e mediana de 973 x 10^3. A comparação entre os grupos de diagnósticos de SCA, revelou que os pacientes

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com IAMCSST apresentaram um SII significativamente maior (IAMCSST: 1735 x 10^3, IAMSSST:1167 x 10^3 e AI: 1069 x 10^3, p<0,001). **Conclusões:** Nosso estudo demonstrou que pacientes com IAMCSST exibem maiores valores de SII, sugerindo maior inflamação e gravidade, em comparação aos pacientes diagnosticados com IAMSSST/AI.

Palavras-chave: Inflamação; Síndrome Coronariana Aguda; Doença da Artéria Coronariana.

Introduction

Coronary artery disease (CAD) is the leading cause of death worldwide. CAD can be identified in its chronic form, as stable angina, or in its acute form, as ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA).[1]

Despite significant therapeutic advances in recent decades, Acute Coronary Syndrome (ACS), mainly represented by myocardial infarction (MI), remains one of the most important causes of morbidity and mortality in Brazil.[1]

Atherosclerotic plaque develops from the oxidation of accumulated LDL, which deposits in the arterial walls, initiating an intense inflammatory process that eventually leads to calcification. The atheromatous plaque can rupture, releasing metabolites and necrotic products, increasing the risk of hypercoagulability and raising the expression of inflammatory markers, which can ultimately occlude the artery, leading to MI.[2]

MI is diagnosed when there is an elevation of myocardial necrosis markers (MNM), along with typical symptoms and possible suggestive changes on the electrocardiogram (ECG). In UA, there are no elevations in MNM. Cardiovascular risk factors are generally present, including smoking, hypercholesterolemia, systemic arterial hypertension, diabetes mellitus, obesity, stress, depression, and/or a family history of early cardiovascular disease, among others. The ECG results classify MI as either STEMI or NSTEMI.[3,4]

Both in STEMI and NSTEMI, the formation of atherosclerotic plaque is deeply associated with a continuous inflammatory response. In this process, various immune cells and pro-inflammatory cytokines play crucial roles[5], involving a complex interaction between innate and adaptive immunity.[6]

Differentiating between the various presentations of ACS is essential to determine immediate treatment, as most MI-related deaths occur within the first few hours of its manifestation (40 to 65% of cases in the first hour and approximately 80% within the first 24 hours).[7-10]

Due to this high morbidity and mortality in the early hours of hospitalization, rapid and efficient patient management is necessary. For this, easily measurable tests and indicators would be useful for risk stratification, in addition to considering the "classic" risk factors. One of the most cost-effective and efficient ways to assess inflammatory processes in CAD is through the leukocyte count and its subtypes (neutrophils, lymphocytes, and monocytes) in a complete blood count.[11] In addition to assessing systemic inflammation, the leukocyte count would be an independent predictor of mortality in MI.[12] This highlights its relevance as a significant prognostic marker.

A new inflammatory indicator, the systemic immunoinflammatory index (SII), has been studied to assess the degree of inflammation and analyze the prognosis of patients with neoplasms (bladder, non-small cell lung, gastric, pancreatic), certain inflammatory, and cardiovascular diseases,[13] In these studies, a high SII was associated with a worse prognosis in the presence of colorectal and stomach cancer, being a comparatively better risk marker than indicators such as neutrophil-to-lymphocyte ratio (NLR); platelet-to-lymphocyte ratio (PLR); monocyte-to-lymphocyte ratio (MLR), and isolated CRP. Moreover, in studies involving cardiovascular diseases, a high SII in patients with CAD has been associated with an increased risk of developing cardiac death, non-fatal MI, or non-fatal stroke.[5]

The SII is calculated using the formula: (N x P) / L (where N, P, and L are the counts of neutrophils, platelets, and lymphocytes, respectively). This index thus considers three important pathways of the immune response: inflammation, represented by neutrophilia; thrombosis risk, by platelets; and the body's stress response, reflected by a low lymphocyte level when immunodeficient.[14] In addition, the SII is a low-cost and easy-to-use clinical method, requiring only cell counts from a complete blood count.[15]

The SII reflects a significant association between the host's inflammatory and immunological status and the risk of

cardiovascular diseases. Therefore, patients with elevated SII could receive recommendations to adopt a healthier lifestyle to reduce inflammation: smoking cessation, weight control, emotional regulation, modification of sedentary behavior, regular exercise, improved sleep, and reduced intake of saturated fats and refined carbohydrates in the diet.[16]

The SII has been positively associated with the risk of total stroke and has been studied in various contexts related to cardiovascular diseases.[12] In the diagnosis of acute massive pulmonary embolism, it has proven to be superior to other inflammation-related indices[17] It was also significantly associated with a worse prognosis in the postoperative period of off-pump coronary artery bypass surgery[18] and was independently related to in-hospital mortality in patients with infective endocarditis.[19]

Moreover, it has been linked to adverse clinical outcomes in elderly patients (65-85 years old) with ACS[20] and was an independent predictor of adverse events in patients with CAD, including those with stable angina, STEMI, and NSTEMI.[21]

Thus, the SII could be a highly efficient inflammatory biomarker for cardiovascular risk stratification.

Considering the high residual risk presented by patients, especially those with severe conditions, the SII would be an important tool for identifying those at greater risk of future complications.

No studies are comparing the SII in patients with STEMI, NSTEMI, and UA. Therefore, this index is not well-established in the literature for this specific group of patients

The main objective of our study was to analyze the SII of patients diagnosed with ACS, comparing the values in STEMI, NSTEMI, and UA for better cardiovascular risk stratification. Our secondary objective was to explore its relationship with other comorbidities and systemic inflammation indices: NLR, MLR, and PLR.

Methods

After approval by the local ethics committee (05/30/2022), patients diagnosed with ACS between July 2022 and July 2023 were retrospectively evaluated. A total of 333 eligible patients diagnosed with UA, NSTEMI, and STEMI—based on their electrocardiographic, clinical, and laboratory characteristics—were included in the study.[11,12]

Patients who were using immunosuppressive or chronic anti-inflammatory medication, had congestive heart failure (ejection fraction < 40%), severe valvular disease, systemic inflammatory or autoimmune disease, trauma, recent major surgery, hematological diseases, severe hepatic or renal insufficiency, abnormal liver enzymes (alanine aminotransferase > 120 U/L), or a glomerular filtration rate <30 mL/min/1.73 m^2 were excluded.

From the sample of 333 eligible patients, clinical and demographic characteristics were collected, such as age, gender, and the presence of cardiovascular risk factors (arterial hypertension, hypercholesterolemia, smoking, previous MI, and diabetes mellitus). Laboratory parameters, including leukocyte, neutrophil, monocyte, platelet, and lymphocyte counts, as well as NLR, PLR, MLR, and SII, were determined upon admission to the emergency unit.

The SII was calculated as (platelets x neutrophils/lymphocytes), as previously described and studied. The NLR was calculated as (neutrophils/lymphocytes), the PLR as (platelets/lymphocytes), and the MLR as (monocytes/lymphocytes).

Statistical Analysis

All study data were analyzed using SPSS software (IBM SPSS Statistics for Windows, Version 21.0. Armonk, NY, USA, IBM Corp.). The Kolmogorov-Smirnov test was conducted to confirm whether the variables followed a normal distribution. Continuous variables were presented as median and interquartile range (25th-75th percentile). Categorical variables were presented as frequencies and percentages. The Kruskal-Wallis H test was used to compare three independent groups for variables that did not follow a normal distribution. The Dunn-Bonferroni post hoc test was used for pairwise comparisons. Categorical variables were analyzed using the appropriate chi-square test. The correlation between variables was assessed using Spearman's Rank correlation test. The Receiver Operating Characteristic (ROC) curve analysis was used to determine the predictive role of the variables.

Results

Our sample included 333 patients (222 males - 66.7%), of whom 163 (48.9%) were diagnosed with STEMI, 139 (41.7%) with NSTEMI, and 31

(9.3%) with UA. For the statistical analysis of SII and comorbidities, patients with NSTEMI and UA were combined into a single group due to their more similar clinical presentations and because CAD guidelines have considered their grouping.

The comparison of the two groups (UA + NSTEMI; STEMI) regarding comorbidities can be seen in Table 1. When comparing the personal history of comorbidities, the characteristics: male gender ($p = 0.016$); hypertension ($p = 0.001$); hypercholesterolemia ($p < 0.001$), diabetes mellitus ($p = 0.003$), and smoking ($p = 0.03$) were considered statistically significant. Thus, it was found that within the original sample, the diagnosis of STEMI was more associated with male gender and smoking, and paradoxically a lower presence of hypertension, hypercholesterolemia, and diabetes mellitus compared to the NSTEMI + UA group.

Table 1 – Baseline characteristics of the study population

	All (333)	*STEMI (163)*	*Unstable Angina + NSTEMI (170)*	*P*
Male, n (%)	**222 (66,7)**	**119 (73)**	**103 (60,6)**	**0,016**
Acute coronary syndrome, n (%)	61 (18,3)	25 (15,3)	36 (21,2)	0,169
Hypertension, n (%)	**213 (64)**	**90 (55,2)**	**123 (72,4)**	**0,001**
Diabetes mellitus, n (%)	**99 (29,7)**	**36 (22,1)**	**63 (37,1)**	**0,003**
Dyslipidemia, n (%)	**95 (28,5)**	**32 (19,6)**	**63 (37,1)**	**<0,001**
Smoking, n (%)	**172 (51,7)**	**94 (57,7)**	**78 (45,9)**	**0,031**

The comparison of the two groups regarding results from the complete blood count can be seen in Table 2. In this comparison, the values of: Leukocytes ($p < 0.001$); Neutrophils ($p < 0.001$); Platelets ($p = 0.026$); Lymphocytes ($p < 0.001$); Total Neutrophils ($p < 0.001$); Total Monocytes ($p < 0.001$); Total Lymphocytes ($p = 0.002$); NLR ($p < 0.001$); PLR ($p = 0.028$); MLR ($p < 0.001$); and SII ($p < 0.001$) were considered statistically distinct. Thus, it can be observed that within the original sample, the diagnosis of STEMI was more associated with higher values of: Leukocytes, Total Neutrophils, Total Monocytes, NLR, PLR, MLR, and SII (Figure 1); and lower values of: Platelets and Total Lymphocytes compared to the NSTEMI + UA group, reflecting higher levels of inflammation in patients with STEMI.

Table 2 – Laboratory Parameters

	All (333)	*STEMI (163)*	*Unstable Angina + NSTEMI (170)*	*P*
SII, rate (median)	***1436 x 10^3 (973 x 10^3)***	***1735x10^3 (1256 x 10^3)***	***1149x10^3 (764x10^3)***	***<0,001***
WBC count, rate (median)	***11112 (10300)***	***12581 (12200)***	***9704 (9000)***	***<0,001***
Neutrophils count, rate (median)	***8355 (7534)***	***9909 (9183)***	***6865 (6278)***	***<0,001***
Lymphocytes count, rate (median)	***1883(1755)***	***1734 (1547)***	***2025 (1936)***	***0,002***
Monocytes count, rate (median)	***775(655)***	***921 (763)***	***634 (589)***	***<0,001***
Platelet Count, rate (median)	***239x10^3 (229x10^3)***	***231x10^3 (224x10^3)***	***247x10^3(233x10^3)***	***0,026***
NLR, rate (median)	***6 (4)***	***7,6 (5,7)***	***4,6 (3,3)***	***<0,001***
PLR, rate (median)	***160 (127)***	***166 (136)***	***155 (118)***	***0,028***
MLR, rate (median)	***0,48 (0,36)***	***0,6 (0,5)***	***0,4 (0,3)***	***<0,001***

In the sample of 333 patients, the mean SII was 1436 x 10^3 and the median was 973 x 10^3. Table 3 shows the correlation of the SII values with the potential comorbidities of the patients. Only dyslipidemia statistically affected the SII values. Surprisingly, was associated with lower SII values: 1209 x 10^3 versus 1527 x 10^3 ($p = 0.044$).

When analyzing each group individually according to the diagnosis, UA + NSTEMI represented 51.05% of the sample with 170 patients. The mean SII was 1149 x 10^3 and the median was 764 x 10^3. Table 4 shows the correlation of the SII values with the potential comorbidities of patients with Unstable Angina + NSTEMI. No comorbidity significantly altered the SII in this group.

In the STEMI group, which accounted for 48.95% of the sample with a total of 163 patients, the mean SII was 1735 x 10^3 and the median was 1256 x 10^3. Table 5 shows the correlation of the SII values with potential comorbidities in patients with STEMI. Only smoking significantly affected the SII values. Non-smoking patients with IAMCSST paradoxically had higher SII values: 2075 x 10^3 versus 1486 x 10^3 ($p = 0.041$).

Regarding SII values, the comparison among the three diagnostic groups revealed that patients with IAMCSST had a significantly higher SII compared to both NSTEMI and UA, respectively (1735 x 10^3 versus 1167 x 10^3 versus 1069 x 10^3, $p < 0.001$). Additionally, in the comparison between the Unstable Angina + NSTEMI group and the IAMCSST group, the IAMCSST group also had statistically significantly higher values: 1735 x 10^3 versus 1149 x 10^3, $p < 0.001$. Higher SII levels indicate more intense inflammation, confirming greater severity.

Figure 1: Graphical comparison of the two giagnostic groups (UA+NSTEMI; STEMI)

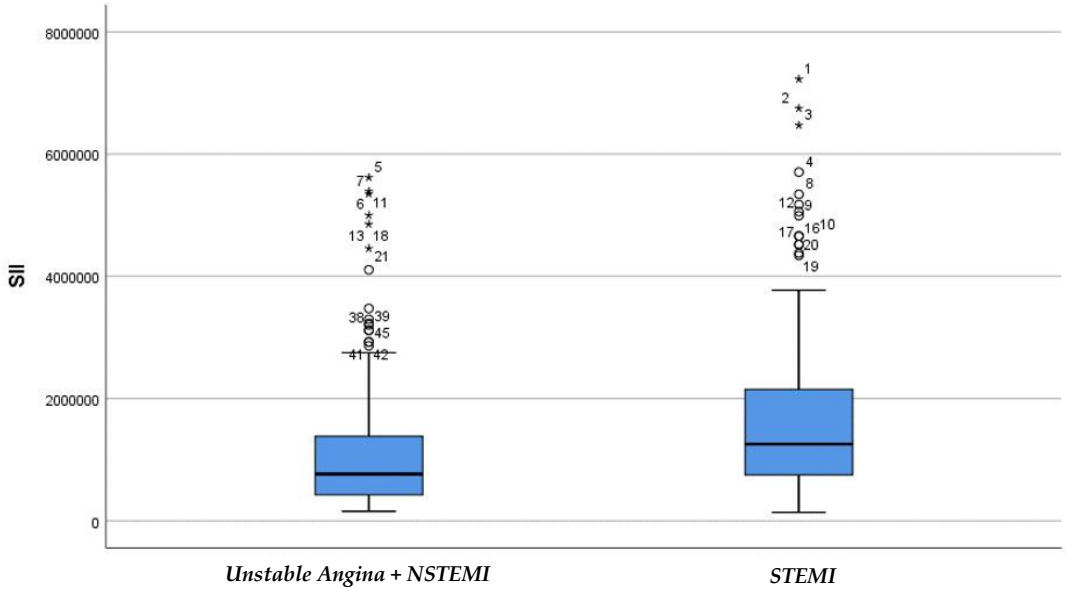

Table 3 – Correlation of the sample's SII values with the patients' possible comorbidities.

VARIABLE		SII	P
Gender	F	1278995	0,109
	M	1515407	
Acute coronary syndrome	No	1479846	0,591
	Yes	1243784	
Hypertension	No	1399613	0,633
	Yes	1457443	
Smoking	No	1646843	0,052
	Yes	1239809	
Dyslipidemia	No	1527368	**0,044**
	Yes	1209213	
Diabetes Mellitus	No	1446567	0,457
	Yes	1413053	

Table 4 – Correlation of SII values with Unstable Angina + NSTEMI with possible comorbidities of patients.

VARIABLE		SII	P
Gender	F	1189106	0,900
	M	1124172	
Acute coronary syndrome	No	1199798	0,903
	Yes	963525	
Hypertension	No	970409	0,147
	Yes	1218297	
Smoking	No	1325527	0,066
	Yes	942453	
Dyslipidemia	No	1212437	0,919
	Yes	1043319	
Diabetes Mellitus	No	1163328	0,981
	Yes	1126726	

Table 5 – Correlation of SII values with STEMI with possible patient comorbidities.

VARIABLE		SII	VALOR P
Gender	F	1.415.871	**0,080**
	M	1.854.040	
Acute coronary syndrome	No	1.751.776	0,965
	Yes	1.647.357	
Hypertension	No	1.675.950	0,515
	Yes	1.784.275	
Smoking	No	2.075.265	**0,041**
	Yes	1.486.550	

Continua

Continuação

Dyslipidemia	No	1.784.602	0,148
	Yes	1.535.818	
Diabetes Mellitus	No	1.685.201	0,810
	Yes	1.914.124	

Discussion

In our study, comparing patient groups with ACS, those diagnosed with STEMI had higher SII scores, reflecting more pronounced inflammation and, likely, greater severity.

It is known that patients with CAD exhibit increased inflammatory markers such as leukocytes and high-sensitivity C-reactive protein (hs-CRP), which are associated with a higher cardiovascular risk[22], impairment of cardiac muscle perfusion[23,24], atherosclerotic plaque instability[25], and mortality[26]. This underscores the importance of monitoring and controlling inflammatory biomarkers as part of clinical evaluation.

Biomarkers derived primarily from three cell lineages (neutrophils, lymphocytes, and platelets) are being investigated and presented in the literature as relevant prognostic markers. They stand out for being economically accessible and easy to calculate. The most commonly used are: PLR (Platelet-to-Lymphocyte Ratio) and NLR (Neutrophil-to-Lymphocyte Ratio)

Both NLR and PLR have been strong independent predictors of major cardiovascular events (MACE) in patients with STEMI[27]. PLR has been reported as an effective predictor for severe atherosclerosis[6], while elevated NLR has been associated with worse clinical outcomes, both in patients with ACS and those with stable CAD undergoing Percutaneous Coronary Intervention. NLR is also associated with the severity and complexity of CAD, as represented by the SYNTAX score[28].

When comparing SII with other composite biomarkers, such as NLR and PLR, SII shows superior prognostic value, primarily due to its unique advantages[12]. It considers three important aspects of the immune response: inflammation, represented by neutrophilia; the risk of thrombosis, reflected by platelet levels; and the immune response to body stress, indicated by lymphopenia[14].

In our sample, despite lower platelet concentrations in the STEMI group, SII was elevated. This result likely reflects the greater acute inflammation and severity in this group at the acute event stage. Thus, it is somewhat speculative to relate the larger extent of STEMI to increased platelet consumption rather than the actual state of blood coagulation at this acute moment. It is important to note that the hemogram value used in the study refers to the initial sample collected upon patient admission. However, this result warrants further investigation in larger studies.

Another insight into the acute inflammatory state of ACS is that RCP is less stable and reliable compared to SII, as hsCRP quantification is susceptible to various factors such as dehydration, malnutrition, and fluid overload. Furthermore, regarding the prediction of CAD occurrence, SII demonstrated better predictive power compared to hsCRP, as well as compared to NLR and PLR[12].

In our study, the STEMI group exhibited higher SII values compared to the NSTEMI and unstable angina groups (1735×10^3 vs. 1167×10^3 vs. 1069×10^3, $p < 0.001$). Additionally, in STEMI patients, other inflammatory biomarkers were elevated, such as leukocyte counts, total neutrophils, total monocytes, NLR, PLR, and MLR, but with lower values of platelets and total lymphocytes. These findings suggest greater severity and an inflammatory state related to the pathogenesis of STEMI.

When comparing the STEMI group with the NSTEMI + UA group, STEMI was more associated with male gender ($p = 0.016$) and smoking ($p = 0.03$) but had lower rates of hypertension ($p = 0.001$), hypercholesterolemia ($p < 0.001$), and diabetes mellitus ($p = 0.003$).

Non-smoking STEMI patients had higher SII values (2075×10^3 vs. 1486×10^3, $p = 0.041$). This association is likely more related to STEMI itself rather than to smoking status prior to hospitalization. Once again, the clinical

presentation is likely to better define these laboratory parameters due to its greater relevance.

Moreover, we also found paradoxical relationships, such as Dyslipidemia being associated with lower SII values: 1209 × 10^3 vs. 1527 × 10^3 ($p = 0.044$).

This finding could be explained by the effect of hypolipidemic treatment, often performed with statins prior to hospitalization. The lower SII values observed would indicate less systemic inflammation due to these anti-inflammatory effects[29,30].

The anti-inflammatory effects of statin therapy are well demonstrated and include reductions in cardiovascular outcomes[31], as shown in the JUPITER study, for example[29]. Zhang *et al*. published a meta-analysis examining CRP concentrations using various types and doses of statins, demonstrating benefits, including long-term benefits[32]. Another study, the Pravastatin Inflammation/CRP Evaluation (PRINCE), showed that 40 mg/day of pravastatin significantly reduces plasma levels of CRP, independent of any changes in LDL-C levels[33]. The HOPE-3 (Heart Outcomes Prevention Evaluation-3) study in intermediate-risk patients without cardiovascular disease showed that rosuvastatin use in this population reduces hs-CRP concentration and adverse cardiovascular events, regardless of CRP and lipid levels at the start of the study[34].

Although statins are the most studied, other lipid-lowering agents have also been analyzed, demonstrating satisfactory effects. Morrone *et al*. reported greater reductions in CRP with a combination therapy of ezetimibe and statins, compared to monotherapy with statins[35]. The bempedoic acid (BA), evaluated in the CLEAR OUTCOMES study, reduced LDL-C and hs-CRP concentrations by approximately 20 to 22% in patients considered intolerant to statins[36]. Lomitapide and mipomersen also demonstrated potential anti-inflammatory effects in the presence of familial hypercholesterolemia[37]. Eicosapentaenoic acid (EPA), an omega-3 used to treat severe hypertriglyceridemia, is thought to have anti-inflammatory properties by inhibiting IL-1β and IL-6.[38]

These data from the literature support our findings of lower SII values, even in patients with dyslipidemia, as these patients were using lipid-lowering agents, mostly statins

The anti-inflammatory effects of lipid-lowering agents, particularly statins, have been studied in the context of seeking measures to prevent and attenuate the systemic inflammatory response in the atherosclerosis process. This concern arises mainly because a significant proportion of patients continue to experience adverse cardiac events despite optimized LDL-C levels or improvements in dyslipidemia, remaining at increased risk of cardiac events, primarily due to persistent inflammation. Therefore, it is believed that the significant impact of inflammation on atherogenesis suggests the existence of an additional, neglected mechanism that could be a pharmacological target[31].

Nevertheless, attenuating the inflammatory process is crucial for preventing cardiovascular events, especially in individuals at higher cardiovascular risk. Cardiovascular risk stratification through inflammatory biomarkers becomes relevant and could offer clinical benefits, as it allows for the early identification of patients who are more severe from a clinical standpoint

In this sense, understanding the SII would enable early detection of the intensity of the inflammatory process, aiding in the stratification and identification of individuals with greater severity in ACS. However, it is considered that evaluating the SII requires more in-depth, randomized, and well-planned research in larger populations.

Possible Study Limitations

The limitations of this study include its retrospective and single-center design. Additionally, the data were collected over only one year using hospital records, which may have introduced selection bias and the exclusion of patients with missing information, as well as limiting sociodemographic diversity.

The individual impact of specific medications used by patients, such as lipid-lowering agents, could not be analyzed, despite their impact on inflammation.

Some blood parameters, such as uric acid and high-sensitivity C-reactive protein (hs-CRP), could not be evaluated due to a lack of data and unavailability at our institution.

Platelet, neutrophil, and lymphocyte counts were performed only once, upon admission, without follow-up during hospitalization.

Conclusions

In patients with ACS, the SII showed higher values in the group with STEMI, suggesting greater inflammation compared to patients with NSTEMI/UA. Other inflammatory cell parameters also followed this elevation.

Larger, multicenter, randomized, and prospective studies could provide further insights into the use of this index in stratification and, consequently, in the prevention of morbidity and mortality in patients with ACS.

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