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Inflammation indices in chronic stable coronary artery disease

Índices de inflamación en enfermedad arterial coronaria crónica estable

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Keywords:
inflammation, chronic stable coronary artery disease, severity, systemic immune-inflammation index.

Palabras clave:
inflamación, enfermedad arterial coronaria crónica estable, severidad, índice inmunitario-inflamatorio sistémico.

ABSTRACT

Introduction: coronary artery disease (CAD) is a chronic inflammatory disorder of multifactorial origin, with inflammation being a key pathophysiological aspect. **Objective:** to determine the relationship between inflammatory indices and the severity of chronic CAD in subjects undergoing cardiac catheterization at the Institute of Cardiovascular Disease Research of the University of Zulia. **Material and methods:** the research was descriptive, cross-sectional, and correlational, with a non-experimental design. The sample was selected through simple random sampling, with subjects over 18 years of age with chronic coronary syndrome who had inflammatory indices quantified and the SYNTAX score determined to assess the severity of CAD. **Results:** of the 73 subjects evaluated, 50.7% (n = 37) were men, the overall average age was 59.5 ± 7.7 years, 83.6% (n = 61) were hypertensive, and 68.5% (n = 50) had a previous acute coronary syndrome. A higher average of platelets (376.1 ± 85.6 × 10^3/mm^3), platelet-lymphocyte ratio (PLR) (144.9 ± 54.7), and systemic immune-inflammation index (SIII) (703.2 ± 335.9) was observed in subjects with a SYNTAX score ≥ 33. A positive correlation was found between PLR and the SYNTAX score ($r = 0.61$; $p < 0.01$) and between the SIII and the SYNTAX score ($r = 0.55$; $p < 0.01$). In the multiple linear regression analysis, the SIII was the index most independently related to the SYNTAX score ($\beta = 0.64$; $p < 0.01$). **Conclusions:** the study found that the SIII was significantly associated with a higher severity of chronic CAD, as indicated by the SYNTAX score. This association was observed independently of other inflammatory and lipid factors.

RESUMEN

Introducción: *la enfermedad arterial coronaria (EAC) es una enfermedad inflamatoria crónica con un origen multifactorial, siendo la inflamación un aspecto fisiopatológico clave.* ***Objetivo:*** *determinar la relación entre los índices inflamatorios con la severidad de la EAC crónica en sujetos sometidos a cateterismo cardiaco en el Instituto de Investigaciones de Enfermedades Cardiovasculares de La Universidad del Zulia.* ***Material y métodos:*** *la investigación fue de tipo descriptiva, transversal, correlacional con un diseño no experimental. La selección de la muestra se realizó a través de un muestreo al azar simple en sujetos mayores de 18 años con síndrome coronario crónico a los cuales se les cuantificaron índices inflamatorios, y el puntaje SYNTAX para determinar la severidad de la EAC.* ***Resultados:*** *de los 73 sujetos evaluados, 50.7% (n = 37) fueron hombres, el promedio general de edad fue 59.5 ± 7.7 años, 83.6% (n = 61) fueron hipertensos y 68.5% (n = 50) tenían síndrome coronario agudo previo. Se observó un mayor promedio de plaquetas (376.1 ± 85.6 × 10^3/mm^3), índice plaquetas linfocitos (PLR) (144.9 ± 54.7) e índice inmunitario-inflamatorio sistémico (IIIS) (703.2 ± 335.9) en los sujetos con puntaje SYNTAX ≥ 33. Se obtuvo una correlación positiva entre el PLR y el puntaje SYNTAX ($r = 0.61$; $p < 0.01$), y entre el IIIS y el puntaje SYNTAX ($r = 0.55$; $p < 0.01$). En el análisis de regresión lineal múltiple, el IIIS fue el índice más relacionado de manera independiente con el puntaje SYNTAX ($\beta = 0.64$; $p < 0.01$).* ***Conclusiones:*** *el IIIS se asoció con un mayor grado de severidad de la EAC crónica según el puntaje SYNTAX, independientemente de otros factores inflamatorios y lipídicos.*

Abbreviations:

BMI = Body Mass Index
CAD = Coronary Artery Disease
CCS = Chronic Coronary Syndrome
CRP = C-Reactive Protein
MLR = Monocyte-Lymphocyte Ratio
NLR = Neutrophil-Lymphocyte Ratio
PLR = Platelet-Lymphocyte Ratio
SIII = Systemic Immune-Inflammation Index

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INTRODUCTION

Various indices derived from cellular biomarkers inherent to the inflammatory process have been described, most of which originate from hematological values. Indices such as the neutrophil-lymphocyte ratio (NLR), the platelet-lymphocyte ratio (PLR), or the monocyte-lymphocyte ratio (MLR) have been associated with increased negative outcomes, including mortality, in various cardiovascular scenarios.[1] Recently, it has been suggested that combining these hematological values could have better predictive value than other existing tools. As a consequence, the systemic immune-inflammation index (SIII) has been created, utilizing a combination of serum levels of neutrophils, lymphocytes, and platelets in a mathematical expression.[2,3]

Assessing the severity of coronary artery disease (CAD) is crucial for the classification of cardiovascular risk and, consequently, for the selection of therapeutic strategies to be implemented.[4] Thus, a cost-effective approach to predicting which subjects are more likely to have an acute coronary event is highly desirable. Among the multiple variables used for this purpose, biomarkers or inflammatory indices associated with the atherosclerotic process could be employed in low-resource settings. Thus, the objective of this study was to determine the relationship between inflammatory indices and the severity of chronic CAD in subjects undergoing cardiac catheterization at the Institute of Cardiovascular Disease Research of the University of Zulia.

MATERIAL AND METHODS

Study design and sample selection

A descriptive, cross-sectional, and correlational study with a non-experimental design was conducted on all patients of both genders diagnosed with chronic coronary syndrome (CCS) admitted for cardiac catheterization to the Hemodynamics Service of the Institute of Cardiovascular Disease Research of the University of Zulia (IECLUZ), located in the Municipality of Maracaibo, Zulia State, during the period from October 2022 to October 2023. The sample selection for this study was performed through simple random sampling of all admitted subjects who met the following criteria: patients of both genders, over 18 years of age, diagnosed with CCS. Subjects with pathologies that could affect laboratory parameters were excluded: evidence of acute or chronic infection, autoimmune or systemic inflammatory diseases, use of glucocorticoids during the previous 2 months, active neoplasms, hematological disorders, liver failure, renal failure, thyroid disorders, trauma or surgery within the previous month, acute coronary syndrome, previous revascularization (percutaneous intervention or coronary artery bypass grafting), decompensated heart failure, clinically significant valvular heart disease; patients who were incapacitated for or opposed to participating in the study were also excluded. After all the considerations, the final sample consisted of 73 patients.

Patient assessment

Each patient selected and included in the study was informed about the study being conducted and asked for their authorization to participate. Subsequently, they were administered a questionnaire based on direct patient interviews, where data such as gender, age, and educational level were investigated and classified as primary, secondary, or university studies. Their place of residence was categorized as urban or rural.

Regarding psychobiological habits and personal history, individuals who did not engage in any degree of physical activity were considered sedentary, and those currently smoking cigarettes were classified as smokers. Ex-smokers were defined as individuals who had quit smoking for over a year prior to the interview. Family history of CAD was only recorded if it had occurred in first or second-degree relatives at a premature age. The presence of hypertension, diabetes mellitus, and dyslipidemia was recorded if documented in medical records or if the patient was taking medications for these conditions.

Subsequently, each patient's weight and height were measured using a scale and a stadiometer (health o meter brand), and the body mass index (BMI) was calculated using the formula: BMI = weight/height^2. Additionally,

Table 1: General characteristic of the sample according to sex.

	Female n (%)	Male n (%)	Total n (%)
Clinical characteristics			
Secondary education	12 (32.4)	18 (50.0)	30 (41.1)
Urban origin	34 (91.9)	34 (94.4)	68 (93.2)
Sedentarism	33 (89.2)	28 (77.8)	61 (83.6)
Current smokers	3 (8.1)	6 (16.7)	9 (12.3)
Ex-smokers	17 (45.9)	13 (36.1)	30 (41.1)
Cardiovascular disease (FH)	21 (56.8)	14 (38.9)	35 (47.9)
Hypertension	32 (86.5)	29 (80.6)	61 (83.6)
Diabetes mellitus	11 (29.7)	12 (33.3)	23 (31.5)
Dyslipidemia	12 (32.4)	17 (47.2)	29 (39.7)
Prior ACS*	20 (54.1)	30 (83.3)	50 (68.5)
Drug use			
Nitrates*	15 (40.5)	25 (69.4)	40 (54.8)
ACEi or ARB	27 (73.0)	27 (75.0)	54 (74.0)
Single antiplatelet therapy	13 (35.1)	11 (30.6)	24 (32.9)
Dual antiplatelet therapy	16 (43.2)	21 (58.3)	37 (50.7)
Statins*	13 (35.1)	27 (75.0)	40 (54.8)
Antidiabetics	9 (24.3)	9 (25.0)	18 (24.7)
SGLT2i	4 (10.8)	3 (8.3)	7 (9.6)
Age (years)‡	60.4 ± 7.1	58.7 ± 8.3	59.5 ± 7.7
Total	37 (50.7)	36 (49.3)	73 (100.0)

ACEi = Angiotensin-Converting Enzyme inhibitor. ACS = Acute Coronary Syndrome. ARB = Angiotensin Receptor Blocker. FH = Family History. SGLT2i = Sodium-GLucose coTransporter-2 inhibitors.
* χ^2 test, $p < 0.01$.
‡ Data indicate mean ± standard deviation.

abdominal circumference was measured using a calibrated measuring tape, employing anatomical landmarks for accurate determination.

Laboratory analyses

Peripheral blood samples (5 mL) were obtained prior to angiographic procedures following a fasting period of at least 8 hours. A routine complete blood cell count was performed using a Mindray BC-2600 analyzer (China). Analysis of glucose, urea, creatinine, total cholesterol, HDL, triglycerides, and C-reactive protein (CRP) was enzymatically conducted using commercial kits with the BT-3000 auto-analyzer (Biotechnica, Rome, Italy). LDL levels were determined using the Friedewald formula when serum triglyceride levels were below 400 mg/dL. Blood samples were collected in standardized EDTA tubes for cell counting, and serum measurements were performed immediately after collection.

The NLR and PLR were calculated using the formulas absolute neutrophil count/absolute lymphocyte count and absolute platelet count/absolute lymphocyte count, respectively. The systemic immune-inflammation index (SIII) was determined using the formula: absolute platelet count × (absolute neutrophil count / absolute lymphocyte count).[5]

Angiographic analysis

Coronary angiography was performed using the standard Judkins technique, with at least two projections taken for all coronary arteries. Anatomical severity was evaluated qualitatively and quantitatively by two interventional cardiologists using the SYNTAX I score, which was calculated through the virtual platform (www.syntaxscore.com). Results were divided into three categories (SYNTAX < 23, SYNTAX 23-32, and SYNTAX ≥ 33).[6]

Statistical analysis

After data collection, a tabulation sheet was designed to facilitate data entry and analysis. Results were expressed as descriptive measures of central tendency (mean), dispersion (standard deviation), as well as absolute and relative values. The χ-square test was used for evaluation between qualitative variables, the t-test was used for comparisons between quantitative variables, and Pearson's correlation coefficient was used to assess correlation between variables. Additionally, a multiple linear regression analysis was conducted with the SYNTAX score as the dependent variable, using the backward elimination method for variable selection in the model. The alpha level was set at 0.05. All analyses were performed using SPSS version 20 for Windows (Chicago, IL).

RESULTS

Out of the 73 evaluated subjects, 50.7% (n = 37) were male, with a mean age of 59.5 ± 7.7

years. According to the sociodemographic characteristics, the predominant groups were those with secondary education (41.1%; n = 30), urban residence (93.2%; n = 68), sedentary lifestyle (83.6%; n = 61), and ex-smokers (41.1%; n = 30). Regarding medical history, hypertension (83.6%; n = 61) and previous acute coronary syndrome (ACS) (68.5%; n = 50) were the most predominant, with ACS being more frequent in males (men: 83.3% vs women: 54.1%; p < 0.01). ACE inhibitors/ARBs were the most commonly used medications (74%; n = 54), followed by statins and nitrates (54.8%; n = 40), with a higher frequency of use in males for these pharmacological groups *(Table 1)*.

Table 2: Clinical and laboratory characteristics of the sample according to sex.

	Female Mean ± SD	Male Mean ± SD	Total Mean ± SD
BMI (kg/m^2)	28.9 ± 5.3	29.1 ± 7.5	29.0 ± 6.7
Abdominal circumference (cm)	101.7 ± 8.2	100.3 ± 10.1	101.0 ± 9.1
Systolic blood pressure (mmHg)	144.2 ± 19.4	139.8 ± 16.8	142.0 ± 18.2
Diastolic blood pressure (mmHg)	79.5 ± 10.0	82.2 ± 8.8	80.8 ± 9.5
Leukocytes (× 10^3/mm^3)	7.8 ± 1.9	7.5 ± 2.3	7.7 ± 2.1
Neutrophils (× 10^3/mm^3)	4.8 ± 1.4	4.6 ± 1.8	4.7 ± 1.6
Lymphocytes (× 10^3/mm^3)	3.0 ± 0.7	2.9 ± 0.8	2.9 ± 0.8
Platelets (× 10^3/mm^3)	256.6 ± 85.5	306.7 ± 95.6	281.3 ± 93.4
NLR	1.64 ± 0.46	1.63 ± 0.61	1.63 ± 0.53
PLR	91.31 ± 44.7	112.8 ± 46.5	101.9 ± 46.6
SIII	425.0 ± 206.3	515.2 ± 298.7	469.5 ± 258.3
Hemoglobin (g/dL)*	12.1 ± 1.1	13.3 ± 1.2	12.7 ± 1.3
Hematocrit (%)*	37.4 ± 4.3	40.4 ± 4.6	38.9 ± 4.7
Glycemia (mg/dL)	114.6 ± 39.3	117.7 ± 55.5	116.1 ± 47.7
Creatinine (mg/dL)*	0.9 ± 0.2	1.0 ± 0.1	1.0 ± 0.2
Urea (mg/dL)	37.4 ± 13.7	37.7 ± 9.2	37.6 ± 11.6
Total cholesterol (mg/dL)*	186.2 ± 56.3	158.4 ± 44.0	172.5 ± 52.2
LDL-C (mg/dL)*	114.5 ± 52.4	91.6 ± 40.7	103.2 ± 48.1
HDL-C (mg/dL)	45.2 ± 7.0	44.0 ± 6.0	44.6 ± 6.5
Triglycerides (mg/dL)	157.9 ± 108.3	123.5 ± 60.0	140.9 ± 89.0
VLDL-C (mg/dL)	31.1 ± 21.2	24.4 ± 12.6	27.8 ± 17.7
C reactive protein (mg/L), median [P25-P75]	5 [3-10.2]	3.4 [3-5]	4 [3-7.1]
SYNTAX score, median [P25-P75]‡	8 [1-22]	22 [11-39.3]	14 [1-35]
Total	37 (50.7)	36 (49.3)	73 (100.0)

BMI = Body Mass Index. HDL-C = High-Density Lipoprotein Cholesterol. LDL-C = Low-Density Lipoprotein Cholesterol. NLR = Neutrophils-Lymphocytes Ratio. PLR = Platelet-Lymphocytes Ratio. SD = Standard Deviation. SIII = Systemic Immune-Inflammation Index. VLDL-C = Very Low-Density Lipoprotein Cholesterol. * Student's t-test, p < 0.05. ‡ Mann Whitney's U test, p < 0.05.

Table 2 displays clinical and laboratory characteristics by sex. There were no significant differences in mean scores of inflammatory indices between sexes; however, higher levels of hemoglobin, hematocrit, creatinine, and SYNTAX scores were observed in males, while mean total cholesterol and LDL levels were higher in females.

In terms of subject distribution according to the severity of CAD, 28.8% (n = 21) had no CAD; 32.9% (n = 24) had SYNTAX score < 23; 28.8% (n = 21) had SYNTAX score ≥ 33; and 9.6% (n = 7) had SYNTAX score 23-32. When assessing inflammatory indices according to SYNTAX score, higher mean platelet counts (376.1 ± 85.6 × 10^3/mm^3), PLR (144.9 ± 54.7), and SIII (703.2 ± 335.9) were observed in subjects with SYNTAX score ≥ 33 *(Table 3)*.

Figure 1 shows the degree of correlation between inflammatory indices and the SYNTAX score, *Figure 1A* shows no correlation between Syntax score and NLR, likewise a positive correlation between PLR, and the SYNTAX score (r = 0.61; p < 0.01) *(Figure 1B)* and between the SIII and the SYNTAX score (r = 0.55; p < 0.01) *(Figure 1C)*. Finally, a multiple linear regression model was performed, where the SIII was independently most associated with the SYNTAX score (β = 0.64; p < 0.01), with the final adjustment shown in *Table 4*.

DISCUSSION

Atherosclerosis plays a pivotal role in the development and progression of CAD, associated with a low-grade inflammatory response typical of the cardio-metabolic continuum. Elevated levels of inflammatory markers such as CRP, interleukins, and tumor necrosis factor have been reported in subjects with atherosclerotic cardiovascular disease. Furthermore, various

Table 3: Inflammatory indices according to SYNTAX score.

Inflammatory indices	Normal coronary arteries (A) Mean ± SD	SYNTAX Score < 23 (B) Mean ± SD	SYNTAX Score 23-32 (C) Mean ± SD	SYNTAX Score ≥ 33 (D) Mean ± SD	p*
Neutrophils (× 10^3/mm^3)	4.8 ± 1.5	4.6 ± 1.4	4.1 ± 1.0	5.0 ± 2.1	0.60
Lymphocytes (× 10^3/mm^3)	3.1 ± 0.9	2.9 ± 0.7	3.1 ± 0.3	2.8 ± 0.8	0.43
Platelets (× 10^3/mm^3)	244.3 ± 63.6	232.4 ± 71.3	275.6 ± 39.7	376.1 ± 85.6	< 0.01
Neutrophils-lymphocytes ratio	1.56 ± 0.39	1.61 ± 0.49	1.33 ± 0.36	1.84 ± 0.70	0.12
Platelet-lymphocytes ratio	81.9 ± 27.3	85.3 ± 32.9	90.3 ± 16.5	144.9 ± 54.7	< 0.01
SIII	381.5 ± 133.6	372.5 ± 149.5	365.2 ± 102.9	703.2 ± 335.9	< 0.01
Total, n (%)	21 (28.8)	24 (32.9)	7 (9.6)	21 (28.8)	73 (100.0)

SD = Standard Deviation. SIII = Systemic Immune-Inflammation Index.
* ANOVA test.

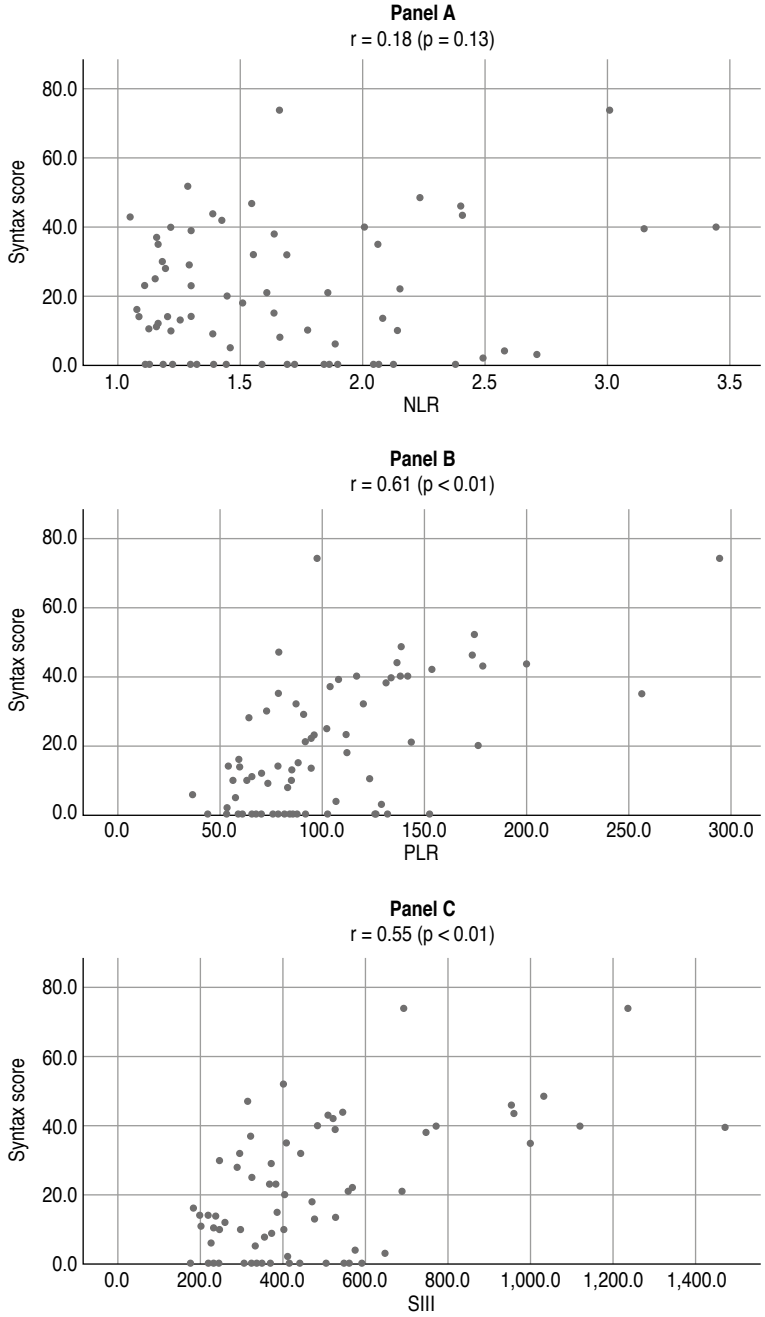
indices derived from cellular biomarkers involved in the inflammatory process and obtained from hematologic values are able to predict severity and outcomes in these patients. This report aims to determine the relationship between inflammatory indices and the severity of chronic CAD in subjects undergoing cardiac catheterization at a specialized cardiovascular institute in Maracaibo, Venezuela.

The key finding of the study is the observed association between the SIII and increased severity of CAD, independent of other risk factors, and showing far superior results than other inflammatory indices. Therefore, the combination of these easily accessible laboratory parameters, such as the three major cellular lines, could potentially serve as a tool to assess inflammatory status in patients with CCS, particularly in primary care settings and outpatient follow-up.

These findings align with those presented by Candemir et al.,[7] who, in a retrospective study of 669 subjects in Turkey, demonstrated a positive correlation between the SIII and the SYNTAX score (Rho: 0.630, p = 0.001). In their multivariate analysis, the SIII emerged as an independent predictor of high SYNTAX score (Odds Ratio: 1.004; 95% CI: 1.001-1.007; p = 0.015). Similarly, in 5,602 patients undergoing percutaneous coronary intervention, Yang et al.,[8] found that an SIII value ≥ 694.3 was independently associated with an increased risk of cardiac death (HR: 2.02; 95% CI: 1.43-2.86), non-fatal myocardial infarction (HR: 1.42; 95% CI: 1.09-1.85), non-fatal stroke (HR: 1.96; 95% CI: 1.28-2.99), and major cardiovascular events (HR: 1.65; 95% CI: 1.36-2.01). In addition, Ma & Li,[9] using multivariate logistic regression analysis of NHANES data from 2009-2018, observed that higher SIII levels could be associated with a greater incidence of CAD, particularly in men.

Remarkably, the other inflammatory index associated with CAD severity was PLR, albeit only in univariate analysis, highlighting the role of platelets in the atherosclerotic process as an essential element in prothrombotic phenomena through interactions between the endothelium and other components of the inflammatory cascade.[10] In contrast, NLR did not show a relationship with CAD severity in our study. This finding may be attributed to the chronic nature of the evaluated cardiovascular disease, with reduced plaque vulnerability and consequently lesser involvement of neutrophils in this stage of the disease's natural history. Nonetheless, these findings differ from those reported by Rodríguez et al.,[11] who analyzed

511 consecutive patients undergoing coronary angiography at the Hermanos Ameijeiras Hospital (Cuba) and found that an elevated neutrophil-to-lymphocyte ratio prior to invasive coronary angiography was associated with greater severity of coronary artery disease.



NLR = Neutrophils-Lymphocytes Ratio. PLR = Platelet-Lymphocytes Ratio. SIII = Systemic Immune-Inflammation Index.

Figure 1: Correlation between inflammatory indices and SYNTAX score.

It is imperative to highlight that this association between the previous indices, specifically the SIII, with the severity of CAD is independent of other proinflammatory factors such as CRP and lipid variables such as LDL. Therefore, it would be interesting to know the anti-inflammatory efficacy of pharmacological agents with the SIII, in particular regarding medium to long-term outcomes. Prime candidates in this regard would be monoclonal antibodies, enzyme regulators, or colchicine. Such data would allow them to standardize their determination and routine evaluation not only from the practical standpoint but also in clinical research.

Regarding population characteristics, it is essential to note that a considerable amount of the subjects were not under optimal pharmacological treatment, as no pharmacologic family had a usage rate higher than 80%, which translates to mean values of certain variables and risk factors above the suggested goal for patients with CCS. The above demonstrates the imperative need to encourage secondary prevention strategies and to emphasize the relevance of adequate treatment adherence from the patient's perspective to decrease recurrent events and, thus, higher morbimortality and disability rates. Optimal intervention in diverse cardiometabolic risk factors could make a positive impact on low-grade inflammation and, therefore, the evaluated inflammatory indices.[12-14]

Among the study limitations, the most notable was the sample size, which complicates the generalization of results to the entire population, and the cross-sectional design, which does not allow the establishment of causality. Additionally, the lack of analysis of other more specific inflammatory mediators, such as interleukins and tumor necrosis factor, due to their limited availability in our context is another constraint.

CONCLUSIONS

The SIII was associated with a higher degree of severity of chronic CAD according to the SYNTAX score, independent of other inflammatory and lipid factors in a group of Venezuelan patients. Additionally, subjects

Table 4: Linear regression model for inflammatory indices and SYNTAX score.

	Dependent variable: SYNTAX score (Log)*			
	Non-standardized β	Standard error	Standardized β	p
NLR	−13.3	3.6	−0.38	0.09
PLR	0.7	0.4	0.17	0.07
SIII	0.5	0.1	0.64	< 0.01

NLR = Neutrophils-Lymphocytes Ratio. PLR = Platelet-Lymphocytes Ratio. SIII = Systemic Immune-Inflammation Index.
* Model created using the backwards method selection, adjusted for age, nitrates usage, ACE/ARB usage, single antiplatelet therapy usage, glycemia, creatinine, LDL, triglycerides, C-reactive protein (log).

with greater CAD severity showed higher average SIII, PLR, and platelet levels, correlating directly with the SYNTAX score. Therefore, it is important to routinely assess inflammatory indices such as the SIII and PLR in patients with chronic CAD, as they may be linked to greater disease severity, allowing for the selection of subjects for more intensive anti-ischemic management. This also ensures appropriate antithrombotic treatment tailored to each patient's characteristics, highlighting the pivotal role of platelets in inflammatory processes.

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Declaration of patient consent: the authors confirm that they have complied with the relevant workplace protocols for the use of patient data. Furthermore, the authors confirm that the patient has been duly informed and has provided written informed consent for the publication of their images and other clinical information in the journal without any identifying details in order to safeguard their right to privacy. Additionally, the authors attest that no form of generative artificial intelligence was employed in the preparation of this manuscript or the creation of figures, graphs, tables, or their corresponding captions or legends.

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