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Combined Prognostic Value of Nutritional and Inflammatory Indices for Predicting Functionally Significant Coronary Stenosis Verified by FFR: The PNI–SII–GINI Study

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Background: Systemic inflammatory and nutritional indices have been associated with coronary artery disease burden and prognosis; however, their ability to predict lesion-level functional ischemia remains uncertain. This study evaluated whether the Prognostic Nutritional Index (PNI), Systemic Immune-Inflammation Index (SII), and Global Inflammation–Nutrition Index (GINI) are associated with functionally significant coronary stenosis assessed by fractional flow reserve (FFR).

Methods: In this single-center observational study, patients with angiographically intermediate coronary lesions who underwent FFR assessment were included. PNI, SII, and GINI were calculated from routine laboratory parameters. Functionally significant stenosis was defined as FFR $\leq$ 0.80. Discriminative performance was evaluated using receiver-operating-characteristic (ROC) analysis, and associations were assessed with binary logistic regression.

Results: A total of 113 patients were analyzed, of whom 48 (42.5%) had FFR $\leq$ 0.80. Baseline clinical characteristics and inflammatory–nutritional indices were comparable between patients with FFR $\leq$ 0.80 and FFR > 0.80. ROC analysis demonstrated limited discriminative ability for all indices, with area under the curve values of 0.544 for PNI, 0.461 for SII, and 0.554 for GINI (all $p > 0.05$). In logistic regression analysis, none of the indices independently predicted FFR-defined ischemia.

Conclusion: Systemic inflammatory and nutritional indices, including PNI, SII, and GINI, did not reliably identify functionally significant coronary stenosis defined by FFR. These findings highlight the distinction between global inflammatory–nutritional status and lesion-specific coronary physiology, reinforcing the continued importance of direct physiological assessment for ischemia-guided management.

Keywords: fractional flow reserve, FFR, coronary stenosis, prognostic nutritional index, PNI, systemic immune-inflammation index, SII, global inflammation–nutrition index, GINI, coronary artery disease, biomarkers, ischemia, predictive value

Introduction

Fractional flow reserve (FFR) is widely regarded as the gold standard for identifying functionally significant coronary artery stenosis and guiding revascularization decisions in patients with coronary artery disease (CAD).[1–3] By directly quantifying the physiological impact of a coronary lesion under conditions of maximal hyperemia, FFR provides lesion-specific information that cannot be reliably inferred from angiographic severity alone. Despite its established role in clinical decision-making, the systemic biological determinants that may influence lesion-level functional ischemia remain incompletely understood.

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Beyond anatomical luminal narrowing, systemic inflammation and nutritional status are increasingly recognized as important contributors to the development, progression, and clinical expression of CAD.[4–6] Inflammatory activation accelerates atherogenesis, promotes endothelial dysfunction, and increases plaque vulnerability, while malnutrition and hypoalbuminemia impair vascular repair mechanisms and immune regulation.[5,6] Collectively, these processes may influence coronary microvascular resistance, myocardial perfusion, and ischemic burden, even in the presence of angiographically intermediate stenoses.

Importantly, systemic inflammatory and nutritional disturbances have been associated with adverse cardiovascular phenotypes extending beyond epicardial coronary disease. Studies in heart failure populations have demonstrated associations between inflammatory–nutritional derangements and thrombotic complications, including left ventricular thrombus formation, underscoring that systemic biomarkers often reflect global cardiovascular pathology rather than focal lesion physiology.[7] In addition, composite inflammatory and nutritional indices have been linked to adverse clinical outcomes across a range of cardiovascular conditions, further supporting their role as markers of overall disease activity.[8,9]

In this context, several composite inflammatory–nutritional indices have been proposed as integrative biomarkers in cardiovascular disease. The Prognostic Nutritional Index (PNI), derived from serum albumin and lymphocyte count, reflects nutritional and immunological reserve and has been associated with adverse outcomes in both coronary artery disease and heart failure populations.[8,10–12] The Systemic Immune-Inflammation Index (SII), calculated from neutrophil, platelet, and lymphocyte counts, captures the balance between inflammatory activation and immune competence and has been linked to angiographic disease burden and prognosis in CAD.[9,13,14] However, whether such systemic indices can reliably predict the functional significance of an individual coronary lesion, as assessed by FFR, remains uncertain.

More recently, composite indices integrating inflammatory and nutritional components into a single equation have been proposed to better reflect systemic biological vulnerability.[15–17] The Global Inflammation–Nutrition Index (GINI), incorporating C-reactive protein, leukocyte subtypes, platelets, albumin, and lymphocytes, represents one such approach. Nevertheless, it remains unclear whether these broader systemic signals correspond to lesion-specific ischemia verified by pressure-derived coronary physiology.

Accordingly, the present study aimed to explore the associations between PNI, SII, and GINI and functionally significant coronary stenosis defined by FFR $\leq$ 0.80 in patients with angiographically intermediate lesions. Rather than presuming additive or incremental prognostic value, this investigation was designed as an exploratory analysis to assess whether systemic inflammatory–nutritional indices provide meaningful discriminatory information for lesion-level physiological significance.

Methods

Study Design and Population

This single-center observational study included consecutive patients undergoing coronary angiography with fractional flow reserve (FFR) assessment for angiographically intermediate coronary lesions. Patients with acute coronary syndromes, prior coronary artery bypass grafting, severe valvular disease, active infection, systemic inflammatory disorders, malignancy, or incomplete laboratory data were excluded.

Fractional Flow Reserve Measurement

FFR was measured using a pressure-sensitive guidewire under conditions of maximal hyperemia induced by intracoronary or intravenous adenosine, in accordance with established methodology.[1,2] Functionally significant coronary stenosis was defined as FFR $\leq$ 0.80. Lesion selection and FFR measurements were performed at the discretion of the treating interventional cardiologist.

Laboratory Measurements and Index Calculation

Venous blood samples were obtained after overnight fasting within 24 hours prior to coronary angiography, and this sampling window was applied uniformly to all participants. Routine hematological and biochemical parameters, including leukocyte differentials, platelet count, albumin, and C-reactive protein, were recorded.

The Prognostic Nutritional Index (PNI) was calculated as:

PNI = albumin (g/L) + 5 × lymphocyte count (10^9/L).

The Systemic Immune-Inflammation Index (SII) was calculated as: SII = (neutrophil count × platelet count)/ lymphocyte count.

The Global Inflammation–Nutrition Index (GINI) was calculated as: GINI = (C-reactive protein × neutrophil count × monocyte count × platelet count)/(albumin × lymphocyte count).

Ethical Considerations

The study was approved by the Kayseri City Hospital Non-Interventional Clinical Research Ethics Committee (Approval Number: 591; Decision Date: 26 September 2025). All procedures were conducted in accordance with the ethical standards of the institutional and national research committees and with the Declaration of Helsinki and its later amendments. Written informed consent was obtained from all participants prior to enrollment.

Statistical Analysis

Continuous variables are presented as mean ± standard deviation and categorical variables as number (percentage). Group comparisons were performed according to FFR status (≤ 0.80 vs > 0.80). Receiver-operating-characteristic (ROC) curve analysis was used to evaluate the discriminative performance of each index. Binary logistic regression analysis was performed to assess associations between inflammatory–nutritional indices and FFR-defined ischemia. Due to sample size considerations, multivariable adjustment was limited to avoid model overfitting. A two-sided p value < 0.05 was considered statistically significant.

Results

Study Population

A total of 113 patients were included in the analysis, of whom 48 (42.5%) had functionally significant coronary stenosis defined as FFR ≤ 0.80, and 65 (57.5%) had FFR > 0.80.

Baseline Characteristics

Baseline demographic, clinical, and inflammatory–nutritional characteristics stratified by FFR status are summarized in Table 1. Male sex distribution was similar between groups (25.0% vs. 27.7%, p = 0.836). Patients with FFR ≤ 0.80 exhibited a numerically higher prevalence of hypertension compared with those with FFR > 0.80 (66.7% vs. 55.4%); however, this difference did not reach statistical significance (χ^2 = 1.467, p = 0.226). Other cardiovascular risk factors,

Table 1 Baseline Clinical and Laboratory Characteristics According to FFR Status

Variable	**FFR ≤ 0.80 (n = 48)**	**FFR > 0.80 (n = 65)**	**p-value**
Demographic characteristics			
Male sex, n (%)	12 (25.0)	18 (27.7)	0.836
Cardiovascular risk factors			
Hypertension, n (%)	32 (66.7)	36 (55.4)	0.226
Diabetes mellitus, n (%)	14 (29.2)	19 (29.2)	0.908
Current smoking, n (%)	11 (22.9)	14 (21.5)	0.782
Chronic kidney disease, n (%)	6 (12.5)	7 (10.8)	0.723
Cerebrovascular disease, n (%)	5 (10.4)	6 (9.2)	0.784
Inflammatory–nutritional indices			
Prognostic Nutritional Index (PNI)	43.4 ± 3.5	42.6 ± 3.6	0.428
Systemic Immune-Inflammation Index (SII)	555.6 ± 257.3	737.0 ± 731.8	0.185
Global Inflammation–Nutrition Index (GINI)	$(3.50 \pm 2.66) \times 10^{14}$	$(3.12 \pm 2.81) \times 10^{14}$	0.304

Notes: Data are presented as mean ± standard deviation or number (%), as appropriate.
Abbreviation: FFR, fractional flow reserve.

Table 2 Receiver-Operating-Characteristic (ROC) Analysis for Prediction of Functionally Significant Coronary Stenosis (FFR ≤ 0.80)

Variable	AUC	SE	95% CI	p-value
Prognostic Nutritional Index (PNI)	0.544	0.056	0.435–0.653	0.427
Systemic Immune-Inflammation Index (SII)	0.461	0.055	0.353–0.568	0.577
Global Inflammation–Nutrition Index (GINI)	0.554	0.054	0.447–0.660	0.334

Abbreviations: AUC, area under the curve; SE, standard error; CI, confidence interval; FFR, fractional flow reserve.

including diabetes mellitus, smoking status, chronic kidney disease, and cerebrovascular disease, were comparable between groups.

With respect to inflammatory–nutritional indices, PNI, SII, and GINI values did not differ significantly between patients with functionally significant and non-significant lesions (all $p > 0.05$).

Receiver-Operating-Characteristic Analysis

Receiver-operating-characteristic (ROC) curve analysis was performed to evaluate the discriminative ability of inflammatory–nutritional indices for identifying functionally significant coronary stenosis (FFR ≤ 0.80). None of the evaluated indices demonstrated statistically significant diagnostic performance (Table 2). The area under the curve (AUC) was 0.544 (95% CI: 0.435–0.653; p = 0.427) for PNI, 0.461 (95% CI: 0.353–0.568; p = 0.577) for SII, and 0.554 (95% CI: 0.447–0.660; p = 0.334) for GINI. All ROC curves approximated the diagonal reference line, and no clinically meaningful cut-off values were identified. The ROC curves are shown in Figure 1.

Binary Logistic Regression Analysis

Consistent with the ROC findings, binary logistic regression analysis demonstrated that none of the inflammatory–nutritional indices independently predicted FFR-defined ischemia (Table 3). Model calibration was acceptable (Hosmer–Lemeshow p = 0.192), but explanatory power was limited (Nagelkerke $R^2 = 0.053$). The overall classification accuracy of the model was 60.2%, indicating minimal discriminatory contribution of these indices to lesion-level physiological significance.

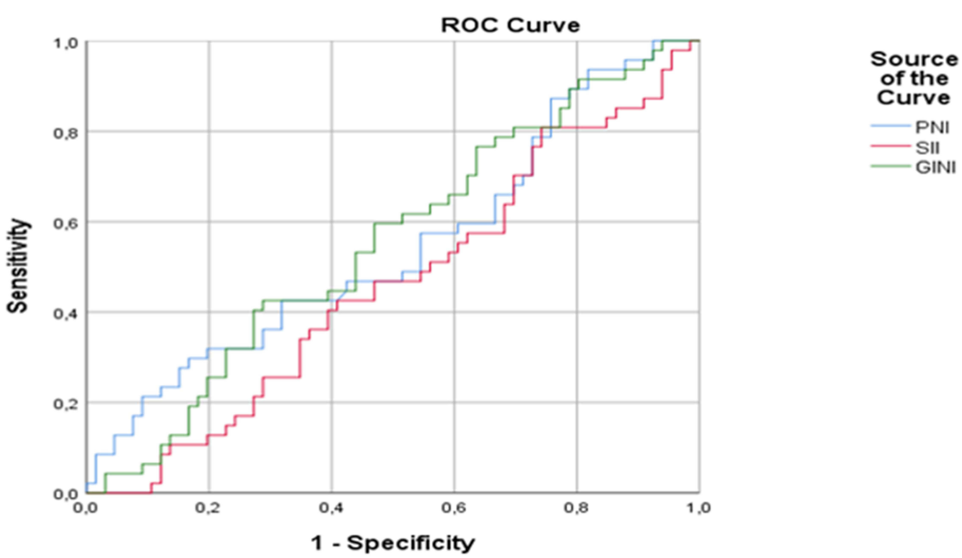

Figure 1 Receiver-operating-characteristic (ROC) curves of the Prognostic Nutritional Index (PNI), Systemic Immune-Inflammation Index (SII), and Global Inflammation–Nutrition Index (GINI) for predicting functionally significant coronary stenosis (FFR ≤ 0.80). All indices demonstrated limited discriminative ability, with areas under the curve close to the diagonal reference line.

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Table 3 Binary Logistic Regression Analysis of Inflammatory–Nutritional Indices for Predicting FFR-Defined Ischemia

Predictor	B	SE	Wald	p-value	Exp (B)	95% CI
Prognostic Nutritional Index (PNI)	0.051	0.057	0.800	0.371	1.053	0.941–1.178
Systemic Immune-Inflammation Index (SII)	−0.001	0.001	1.846	0.174	0.999	0.998–1.000
Global Inflammation–Nutrition Index (GINI)	0.000	0.000	0.419	0.518	1.000	1.000–1.000
Constant	−2.263	2.535	0.797	0.372	—	—

Notes: Model statistics: Hosmer–Lemeshow p = 0.192; Nagelkerke R^2 = 0.053; Overall classification accuracy = 60.2%. The regression model included only inflammatory–nutritional indices to assess their standalone association with FFR-defined ischemia. Multivariable adjustment was not performed due to sample size considerations.
Abbreviation: FFR, fractional flow reserve.

Discussion

In this exploratory study, we evaluated whether systemic inflammatory and nutritional indices are associated with lesion-level physiological ischemia assessed by fractional flow reserve. The principal finding is that PNI, SII, and GINI did not demonstrate meaningful discriminatory ability for identifying functionally significant coronary stenosis. ROC analyses yielded areas under the curve approximating 0.5, and none of the indices emerged as significant predictors in logistic regression analysis.

These findings contrast with prior reports linking inflammatory and nutritional indices to angiographic disease severity and adverse cardiovascular outcomes.[9,11–15,18] Such studies consistently indicate that systemic biomarkers reflect overall atherosclerotic burden and cardiovascular risk. However, our results suggest that these associations do not extend to the physiological significance of individual coronary lesions when evaluated using pressure-derived indices.

This dissociation is biologically plausible. Fractional flow reserve quantifies the focal hemodynamic impact of a specific epicardial stenosis and is determined primarily by lesion geometry, vessel size, plaque characteristics, and myocardial mass supplied.[19,20] In contrast, inflammatory–nutritional indices reflect a diffuse systemic milieu encompassing immune activation, nutritional reserve, endothelial dysfunction, and pro-thrombotic tendency. While these processes contribute to disease progression and adverse outcomes, they do not necessarily govern the flow-limiting behavior of a single coronary lesion, consistent with the known discordance between anatomical severity and physiological significance.[21–23]

Evidence from heart failure populations further supports this concept. Inflammatory and nutritional disturbances have been associated with thrombotic complications, prolonged hospitalization, and mortality, highlighting their prognostic value at the patient level rather than their ability to discriminate lesion-specific ischemia.[7,8,24] Similarly, angiographic studies in patients with systolic heart failure have demonstrated relationships between systemic risk profiles and overall, CAD burden, while lesion-level functional significance requires direct physiological assessment.[25]

Composite inflammatory–nutritional indices such as the HALP score have also been shown to predict prolonged hospitalization and adverse outcomes in patients with acute heart failure.[26] Inflammatory mechanisms have long been recognized as central drivers of atherosclerosis and overall cardiovascular risk.[27] In heart failure populations, systemic inflammatory indices have been shown to predict mortality and adverse outcomes, emphasizing their role as markers of global disease activity.[28] Metabolic and inflammatory pathways have been implicated in coronary disease progression, supporting the concept that systemic biological processes influence overall cardiovascular risk rather than lesion-specific physiology.[29] However, studies comparing anatomical and physiological assessment have demonstrated that angiographic severity does not reliably predict functional ischemia, underscoring the limitations of structural evaluation alone.[30] In contrast to these prior observations, the present study demonstrated that PNI, SII, and GINI did not exhibit meaningful discriminatory ability for identifying functionally significant coronary stenosis, with ROC curve analyses yielding values approximating 0.5 and no significant associations observed in logistic regression analysis.

The lack of association observed even with GINI suggests that increasing biomarker complexity does not overcome the fundamental biological distinction between systemic risk and focal coronary physiology. Non-cardiac influences,

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including hepatic function, subclinical infection, and hematologic variability, may further limit the specificity of such indices for lesion-level decision-making.

Several limitations should be acknowledged. The single-center design and modest sample size may limit generalizability and statistical power. Multivariable adjustment was intentionally restricted to avoid overfitting, and residual confounding cannot be excluded. Inflammatory markers may also be influenced by unmeasured clinical factors despite standardized sampling.

Nonetheless, the present study provides a clinically relevant message. While inflammatory and nutritional indices offer valuable insight into global cardiovascular risk, they do not substitute for direct physiological assessment of coronary lesions. These findings reinforce the continued central role of FFR in ischemia-guided revascularization strategies.

Conclusion

Systemic inflammatory and nutritional indices did not reliably predict functionally significant coronary stenosis defined by fractional flow reserve, underscoring that systemic biomarkers reflect global cardiovascular risk rather than lesion-specific ischemia. Future multicenter studies should evaluate integrated models combining clinical, anatomical, and physiological data; until then, direct physiological assessment with FFR remains the cornerstone of ischemia-guided management.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

Mehmet Zafer Aydın: Conceptualization, Methodology, Supervision, Writing – review & editing.
Can Baba Arın: Formal analysis, Data curation, Writing – review & editing.
Mehmet Kamil Teber: Investigation, Data curation, Writing – review & editing.
Zülfiye Kuzu: Investigation, Resources, Writing – review & editing.
Mustafa Gök: Investigation, Validation, Writing – review & editing.
Ishak Ahmed Abdi: Conceptualization, Formal analysis, Writing – original draft, Supervision.

All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest.

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