



# Prognostic Value and Association of Platelet-to-Albumin Ratio with Coronary Artery Ectasia and Severity Classification

Yunus Emre Yavuz\*, Hasan Sari\*\*, Ahmet Taha Sahin\*\*, Serif Ahmet Kandemir\*\*\*, Muzaffer Aslan\*\*\*\*

\*Necmettin Erbakan University Meram Faculty of Medicine, Department of Cardiology, Konya, Turkey

\*\*University of Health Sciences Turkey, Konya Beyhekim Training and Research Hospital, Clinic of Cardiology, Konya, Turkey

\*\*\*Usak Training and Research Hospital, Clinic of Cardiology, Usak, Turkey

\*\*\*\*Siirt Training and Research Hospital, Clinic of Cardiology, Siirt, Turkey

## Abstract

**Aim:** Coronary artery ectasia (CAE) is characterized by abnormal dilation of coronary arteries and is associated with inflammation and thrombotic events. The platelet-to-albumin ratio (PAR) has emerged as a potential biomarker reflecting both inflammatory status and thrombotic activity. This study aimed to evaluate the relationship between PAR and the presence and severity of CAE.

**Methods:** This retrospective, cross-sectional observational study included 80 patients diagnosed with CAE who underwent coronary angiography between April 2022 and January 2024, as well as 73 age- and sex-matched control participants with normal coronary anatomy. We calculated the PAR and assessed CAE using the Markis classification, which categorizes CAE severity based on the number and distribution of ectatic segments.

**Results:** There was no significant difference between the two groups in terms of age and sex. Although the PAR was higher in the coronary ectasia group, the difference was not statistically significant. However, PAR was significantly correlated with the Marquis classification ( $p=0.014$ ).

**Conclusion:** While PAR values showed no significant difference between patients with CAE and those with normal coronary arteries, there was a notable correlation between PAR values and the Markis classification among patients with CAE, suggesting that PAR could be useful in evaluating the severity of CAE.

**Keywords:** Coronary artery ectasia, inflammation, platelet/albumin ratio

## Introduction

Coronary artery ectasia (CAE) is a distinctive form of coronary artery disease (CAD) characterized by abnormal dilation and expansion of the coronary arteries (1). It is defined as the enlargement of the coronary ectatic segment, which typically ranges from 1.5 to 2 times the diameter of the adjacent normal vessel (2). Although CAE is traditionally considered a variant of atherosclerosis, its pathophysiology remains incomplete, with a range of underlying etiologies including atherosclerosis, congenital

abnormalities, inflammatory conditions, and connective tissue disorders (3,4). Coronary artery ectasia is gaining clinical attention because of its potential to cause complications, such as myocardial infarction, coronary thrombosis, and even sudden cardiac death. Recently, various biomarkers have been used to better understand the pathophysiology of CAE and predict disease severity (5).

Platelets play a crucial role in thrombosis, as they not only increase blood coagulability but also trigger

**Corresponding Author:** Yunus Emre Yavuz, Necmettin Erbakan University Meram Faculty of Medicine, Department of Cardiology, Konya, Turkey

**E-mail:** yemre91@icloud.com **ORCID:** orcid.org/0000-0002-9901-8141

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and intensify inflammatory reactions by interacting with immune cells and producing pro-inflammatory cytokines (6). Platelets also contribute to the onset, progression, and destabilization of atherosclerotic vascular diseases (7). They play a significant role in major adverse cardiac events and peripheral arterial diseases. Low albumin levels have been shown to correlate with adverse events in various chronic inflammatory conditions and (8). Coronary artery disease the role of inflammation in the pathogenesis of CAE has been well documented in previous studies (9). The platelet-to-albumin ratio (PAR) has emerged as a novel indicator of inflammation and oxidative stress, both of which play critical roles in cardiovascular pathogenesis. PAR is calculated by dividing the platelet count by the albumin level (10). The platelet-to-albumin ratio has shown promise as a potential prognostic marker for various cardiovascular conditions; however, its role in CAE remains largely unexplored. We hypothesized that higher PAR values would be associated with CAE, as determined by the Markis classification, reflecting a greater inflammatory and thrombotic burden in patients with extensive ectatic disease.

The aim of this study is to investigate the relationship between PAR and both the presence and severity of CAE, according to the Markis classification. This study may contribute to clinical practice by offering the PAR as a simple, cost-effective, and readily available indicator for predicting CAE severity. In turn, this could provide significant clinical benefits by allowing the early identification of high-risk patients and optimization of follow-up strategies.

## Materials and Methods

### Compliance with Ethical Standards

The study was reviewed and approved by the Siirt University Ethics Committee (approval no.: 100749, date: 28.02.2024) adhering to the principles of the Helsinki Declaration.

### Study Design

This was a single-center, retrospective, cross-sectional observational study. In our investigation, we retrospectively assessed 80 patients diagnosed with coronary ectasia who underwent coronary angiography at our hospital between April 2022 and January 2024. Additionally, we evaluated 73 patients with comparable demographic profiles and normal coronary anatomies.

The inclusion criteria for this study were as follows: Patients aged  $\geq 18$  years with a confirmed diagnosis of CAE based on coronary angiography. Patients were required to have complete clinical and laboratory data available, including platelet count and serum albumin levels, measured at the time of coronary angiography.

Additionally, CAE severity was classified according to the Markis classification. The exclusion criteria for the study were defined as follows: Acute myocardial infarction, history of prior percutaneous coronary intervention, moderate to severe valvular heart disease, heart failure with reduced ejection fraction, congenital heart disease, hereditary hyperlipidemia, severe liver and kidney dysfunction, and pulmonary hypertension. The control group consisted of age- and sex-matched individuals who underwent coronary angiography during the same period and were found to have normal coronary artery structure, defined as less than 10% stenosis and no evidence of ectasia or significant CAD (Figure 1).

### Patient Evaluation

Three invasive cardiologists independently reviewed prior coronary angiograms. Coronary artery ectasia was defined as the dilation of the coronary ectatic segment, reaching 1.5-2 times the diameter of the adjacent normal vessel, while coronary artery aneurysm was characterized by dilation that is 2 times or more. Coronary angiographies were routinely conducted via the femoral route using the Judkins method, excluding cases where nitroglycerin was administered. Computerized quantitative angiography was used for coronary artery diameter measurements, considering the largest diameter in the segments as reference.

Prior to coronary angiography, we analyzed basic biochemical tests and complete blood counts from all patients, taken after overnight fasting. These included lymphocyte, leukocyte, monocyte, hemoglobin, platelet, mean platelet volume, creatinine, estimated glomerular filtration rate, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein, systemic immune-inflammation index (SII), and platelet count divided by serum albumin concentration, which yields the PAR.

### Statistical Analysis

The obtained results were evaluated using Statistical Package for the Social Sciences (SPSS) 22.0 (SPSS, Inc., Chicago, Illinois, USA) for statistical analysis. Continuous variables were recorded as mean  $\pm$  standard deviation for parametric data and as median with interquartile range (25<sup>th</sup>-75<sup>th</sup> percentiles) for non-parametric data. Categorical variables were expressed as frequencies and percentages. Data distribution was assessed using the Kolmogorov-Smirnov test. Variables showing a normal distribution among groups were compared using the Student's t-test, while those not showing a normal distribution were compared using the Mann-Whitney U test. The chi-square test was used for comparing categorical variables. Finally, correlation analysis was performed using either the Spearman or Pearson correlation test. A significance level

of  $p < 0.05$  was considered statistically significant for all analyses.

### Results

The groups were found to have similar statistical gender distributions. The mean age was determined to be 58.3 ( $\pm 10.1$ ) in the coronary ectasia group and 58.6 ( $\pm 10.6$ ) in the normal coronary group, with no significant difference observed between the groups. No differences were observed between the groups in terms of chronic diseases and smoking habits (Table 1).

There is no noticeable distinction in the echocardiographic parameters between the two groups. Although the estimated systolic pulmonary artery pressure did not reach statistical significance in the coronary ectasia group, it was found to be higher. The hemoglobin level was statistically higher in the coronary ectasia group ( $p = 0.001$ ). There was no significant difference observed between the groups in terms of cholesterol levels. Although the PAR was higher in the coronary ectasia group, the difference did not reach statistical significance (Figure 1, Table 2).

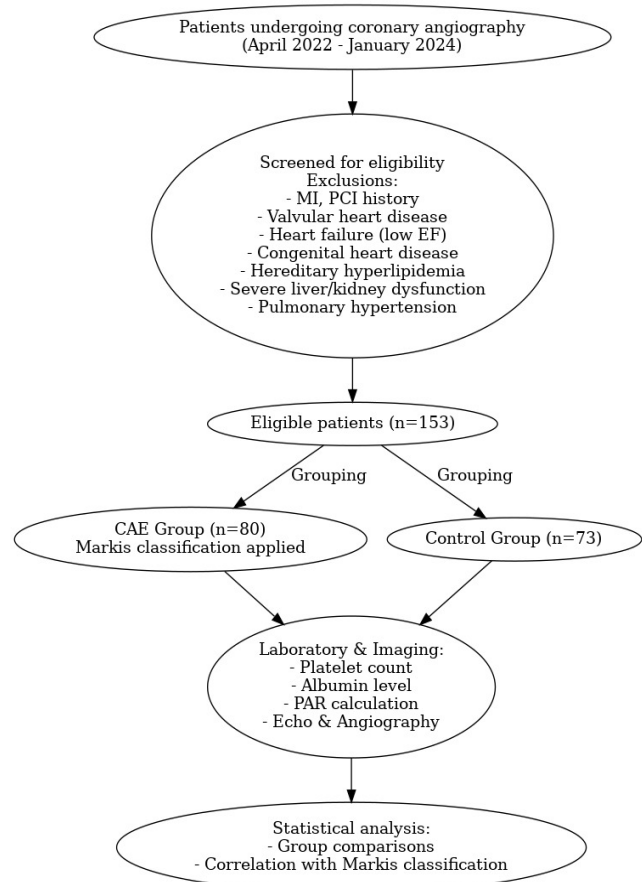
In the correlation analysis, a statistically significant correlation was found between the SII and PAR ( $p < 0.01$ ), while PAR was significantly correlated with the Markis classification ( $p < 0.014$ ) (Table 3). The logistic regression model suggests that when PAR is divided into two groups (corresponding to class 1 and 2 versus class 3 and 4), it has a significant impact on the Markis classification, whereas SII does not show a significant effect in this model (Figures 2 and 3).

### Discussion

Our findings also suggest that PAR may be a prognostic differentiator rather than a diagnostic marker. There was no statistically significant difference between the CAE group and the normal coronary artery control group in terms of mean PAR values. Although the PAR was higher in the SSI group, the p-value was not significant. This shows that PAR alone is not sufficient to detect the presence of CAE. However, it is understood that PAR may be valuable

in determining the extent and severity of the disease in patients with CAE. In other words, high PAR gives a clue to the extent of the current CAE rather than the diagnosis of CAE.

In recent years, some indices containing key blood parameters have been shown to aid in risk assessment and potential treatment strategies by providing valuable information about the inflammatory and atherosclerotic



**Figure 1.** Patients flow charts

PCI: Percutaneous coronary intervention, MI: Myocardial infarction, PAR: Platelet/albumin ratio, CAE: Coronary artery ectasia, EF: Ejection fraction

**Table 1. Demographic characteristics and distribution of comorbidities among group**

	Normal coronary (n=73)	Coronary ectasia (n=80)	p-value
Sex (women)	38 (52.1%)	37 (46.3%)	0.475*
Age	58.6 ( $\pm 10.6$ )	58.3 ( $\pm 10.1$ )	0.874+
Diabetes mellitus	11 (15.1%)	13 (16.3%)	0.841*
Hypertension	28 (38.4%)	34 (42.5%)	0.603*
Hyperlipidemia	14 (19.2%)	15 (18.8%)	0.947*
Smoking	15 (20.5%)	21 (26.3%)	0.410*
Rhythm (AF)	3 (4.1%)	4 (5%)	0.794*

\*Pearson's chi-squared test, +: Student's t-test, p-value < 0.05 indicates statistical significance, AF: Atrial fibrillation

components involved in the pathogenesis and prediction of CAE. For example, SII and the atherogenic plasma index are some of the important indices associated with CAE. Studies have shown that SII, calculated from neutrophil-to-lymphocyte ratio and platelet counts, is independently

linked to the presence and severity of isolated CAE, with higher SII values indicating a more severe inflammatory process (11,12). In our study, contrary to the literature, SII was not significant in patients with coronary ectasia. In addition, unlike PAR, there was no significant correlation

**Table 2. Distribution of echocardiographic and blood parameters between groups**

	Normal coronary (n=73)	Coronary ectasia (n=80)	p-value
LVEDD (mm)	46.4 (±3.4)	46.7 (±3.8)	0.712
LVESD (mm)	27.7 (±3.7)	28.0 (±4.2)	0.705
IVS (mm)	10.9 (±1.2)	11.1 (±1.2)	0.433
PW (mm)	10.1 (±0.9)	10.2 (±0.8)	0.751
E/A	0.9 (±0.3)	1.0 (±0.4)	0.366
sPAP (mmHg)	28.9 (21-40)	30.25 (20-47)	0.174*
Urea (mg/dL)	30.2 (±11.2)	30.9 (±9.5)	0.666
Creatinin (mg/dL)	0.99 (0.37-8.86)	0.93 (0.62-2.1)	0.342*
Sodium (mmol/L)	139.0 (133-143)	140.1 (135-149)	0.007*
Potassium (mmol/L)	30.2 (±11.2)	30.9 (±9.5)	0.732
Hemoglobin (g/dL)	13.1 (±1.8)	14.1 (±1.8)	0.001
Albumin (g/dL)	42.5 (±2,8)	41.9 (±3.6)	0.310
CRP (mg/L)	4.1 (±0-14)	5.3 (0.3-81)	0.176*
WBC (10 <sup>3</sup> /μL)	7.7 (±2.5)	7.5 (±2.0)	0.532
Neutrophil (10 <sup>3</sup> /μL)	4.9 (±2.2)	4.6 (±1.5)	0.252
Leukocytes (10 <sup>3</sup> /μL)	2.21 (±0.8)	2.24 (±0.8)	0.830
Platelet (10 <sup>3</sup> /μL)	259.9 (±78.7)	271.5 (±71.4)	0.343
Monocytes (10 <sup>3</sup> /μL)	0.44 (±0.5)	0.55 (±1.1)	0.480
LDL (mg/dL)	108.4 (40-238)	111.2 (44-268)	0.854*
HDL (mg/dL)	46.2 (±12.4)	45.5 (±11)	0.721
Total cholesterol (mg/dL)	177.8 (±37.5)	187.4 (±41)	0.156
Triglycerides (mg/dL)	176.4 (71-1104)	155.6 (51-486)	0.273*
SII	770.0 (159.1-9183)	644 (178-2409)	0.991*
PAR	6.13 (±1.84)	6.51 (±1.85)	0.201

p<0.05 indicates statistical significance, p-values with an asterisk indicate variables that were compared using the Mann-Whitney U test due to non-normal distribution, non-normally distributed variables are presented as median (minimum-maximum), other variables were compared using the Student's t-test and values are expressed as mean ± standard deviation

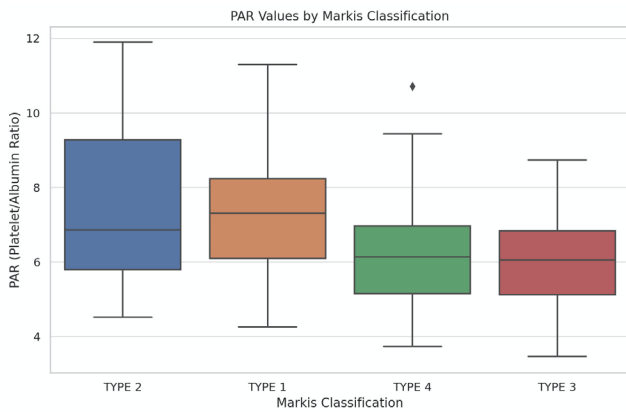
LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, IVS: Interventricular septum, PW: Posterior wall, E/A: E wave/a wave ratio, sPAP: Systolic pulmonary artery pressure, CRP: C-reactive protein, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, SII: Systemic immune-inflammation index, PAR: Platelet/albumin ratio, WBC: White blood cell

**Table 3. Correlation analysis of Platelet/Albumin ratio with Markis classification and SII**

Parameters	PAR		SII	
	r	p*	r	p*
Markis classification	-0.273	0.014	-0.002	0.987
PAR			0.446	<0.01

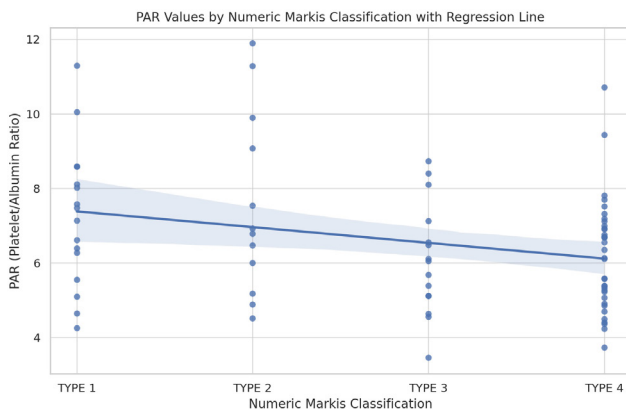
p<0.05 indicates statistical significance

PAR: Platelet/albumin ratio, SII: Systemic immune-inflammation index



**Figure 2.** Distribution of PAR values across Markis classification groups

PAR: Platelet/albumin ratio



**Figure 3.** Correlation between PAR value and Markis classification with regression line

PAR: Platelet/albumin ratio

with the Marquis classification. This may be due to the multifaceted nature of CAE. Additionally, recent evidence from Alici et al. (13) has shown that hematological ratios, such as the platelet-to-white blood cell ratio, can serve as novel prognostic biomarkers in acute coronary settings, further supporting the potential utility of simple blood-based indices like PAR in cardiovascular prognostication. Another study using platelets showed that the mean platelet volume-platelet count ratio was significantly associated with the presence and severity of CAE and that platelet count had a potential role in this disease (14). Research conducted revealed that the proportion of white blood cells to average platelet volume is significantly linked to CAE and could be a cost-efficient approach to monitor CAE (15).

Coronary artery ectasia is also associated with albumin levels in the context of inflammation and endothelial dysfunction. Studies have shown a significant relationship

between CAE and the C-reactive protein-to-albumin ratio (CAR) (16). In addition, CAE has been associated with microalbuminuria, a marker of endothelial dysfunction, suggesting a potential association with increased cardiovascular risk (17). Furthermore, the importance of CAR and CAE in predicting the phenomenon of non-reflow in patients with acute ST-segment elevation myocardial infarction has been compared, and CAR has emerged as a more reliable predictor (18). These findings highlight the complex interplay between CAE, inflammation, endothelial dysfunction, and albumin levels, emphasizing the importance of monitoring these parameters in patients with CAE. Recent research has shown a strong correlation between elevated PAR values and unfavorable outcomes in patients with certain diseases and malignancies (19,20). A recent study on patients with on ST-segment elevation acute coronary syndromes who received percutaneous coronary intervention showed that higher PAR quartiles were linked with higher rates of non-fatal myocardial infarction, ischemia-driven revascularization, and Major Adverse Cardiovascular Events (MACE). A high PAR value was found to be directly associated with an increased risk of MACE (21).

The Markis classification system is a widely accepted method for categorizing CAE based on the number and severity of ectatic segments in the coronary arteries (22). It includes four distinct types: Type I (diffuse ectasia in two or three coronary arteries), Type II (diffuse ectasia in one artery and localized ectasia in another), Type III (diffuse ectasia in one artery), and Type IV (localized or segmental ectasia in one artery). This classification scheme helps clinicians gauge the extent of CAE and can provide insights into the risk of complications such as thrombosis and coronary artery occlusion. Our analysis revealed an intriguing finding regarding the relationship between PAR and the Markis classification of coronary ectasia. This unexpected result suggests that while PAR may not be useful in distinguishing between patients with or without CAE, it could have value in predicting the severity or extent of ectasia among those with the condition. A higher PAR in individuals with lower Markis classifications (indicating fewer ectatic segments) could reflect underlying differences in platelet activation or albumin levels, which may decrease as CAE progresses.

While our study did not find a significant relationship between PAR and the presence of coronary ectasia, the correlation between PAR and the Markis classification highlights the need for further investigation into the role of PAR in predicting CAE severity. Understanding this relationship could have important clinical implications, potentially aiding in risk stratification and guiding treatment strategies for CAE patients.

### Study Limitations

Our study has limitations, including a relatively small sample size and a focus on a specific cohort, which may limit the generalizability of the results. Future studies with larger cohorts and more diverse populations could provide more profound insights into the potential role of PAR in CAE and help identify additional markers that could complement PAR in assessing ectasia severity. Despite these limitations, the study has strengths such as a well-defined patient group, rigorous statistical analysis, the use of the widely accepted Markis classification for severity assessment, and the inclusion of comparisons with established inflammatory indices, which add value to the literature.

### Conclusion

Our findings suggest that PAR could serve as a monitoring tool for identifying CAE patients at higher risk of disease progression or adverse outcomes, despite not differentiating them from healthy individuals. Its significant correlation with the Markis classification highlights its potential as a severity marker, bridging a gap in the biomarker utility for CAE. Further large-scale studies are needed to validate its role in predicting long-term clinical outcomes and guiding disease management.

### Ethics

**Ethics Committee Approval:** The study was reviewed and approved by the Siirt University Ethics Committee (approval no.: 100749, date: 28.02.2024).

**Informed Consent:** Informed consent forms were obtained from the patients and control group.

### Footnotes

#### Authorship Contributions

Concept: Y.E.Y., H.S., Design: Y.E.Y., M.A., Data Collection or Processing: M.A., H.S., Ş.A.K., Analysis or Interpretation: M.A., A.T.Ş., Literature Search: A.T.Ş., Ş.A.K., H.S., Writing: Y.E.Y., A.T.Ş.

**Conflict of Interest:** No conflicts of interest were declared by the authors.

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