

NEUTROPHIL-TO-LYMPHOCYTE RATIO (NLR) AS A BIOMARKER IN CORONARY ARTERY ECTASIA: A CASE-CONTROL STUDY

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ОЦЕНКА НА ВРЪЗКАТА НА СЪОТНОШЕНИЕТО МЕЖДУ НЕУТРОФИЛИТЕ И ЛИМФОЦИТИТЕ И ЕКТАЗИЯТА НА КОРОНАРНИТЕ АРТЕРИИ

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Abstract.

Background and Objective: Coronary artery ectasia (CAE), a diffuse or focal dilatation of coronary arteries, remains poorly understood. While the neutrophil-to-lymphocyte ratio (NLR) is a well-established inflammatory marker in cardiovascular diseases, its diagnostic role in CAE is unclear. This study aimed to evaluate the diagnostic utility of NLR in CAE and explore its association with disease severity. **Material and Methods:** In this case-control study, 115 patients (28 CAE, 87 controls) admitted to Shahid Sadoughi Hospital (2012–2020) were analyzed. Demographic data, medical history, and complete blood count (CBC)-derived NLR were compared using SPSS and appropriate statistical tests. **Results:** The mean NLR did not differ significantly between CAE (0.79 ± 2.01) and control groups (1.46 ± 2.07) ($P = 0.830$), nor by age, sex, or ectasia extent. However, NLR was lower in three-vessel involvement (0.49 ± 1.66) versus single-vessel involvement (1.04 ± 2.32) ($P = 0.036$). **Conclusion:** NLR lacks diagnostic value for CAE but may correlate with disease burden, as evidenced by reduced NLR in multi-vessel involvement. Further research should investigate NLR's pathophysiological role and alternative inflammatory markers in CAE.

Key words:

coronary artery ectasia, neutrophil-to-lymphocyte ratio, cardiovascular diseases, inflammation, angiography

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Резюме.

Въведение и цел: Болестите на коронарните артерии са сред най-честите сърдечно-съдови заболявания. Една от тях е ектазия на коронарната артерия (CAE). Тя представлява дифузна или фокална дилатация на коронарните артерии. Съотношението на неутрофили към лимфоцити (NLR) – маркер за възпаление, е изследвано по отношение на неговата прогностична стойност при много сърдечно-съдови заболявания. Настоящото проучване има за цел да оцени връзката между NLR и ектазията на коронарната артерия. **Материал и методи:** Това проучване е от типа случай-контрола и обхваща 115 пациенти, приети в болница „Шахид Садоги“ в Язд за периода 2012-2020 г. От тях

28 пациенти са били с ектазия на коронарната артерия, а 87 са контролна група пациенти. Събрани са демографски данни, медицинска история и резултати от пълна кръвна картина (ПКК) за изчисляване на NLR. Данните са сравнени с помощта на софтуер SPSS и приложими статистически тестове. **Резултати:** Средният NLR в групата с ектазия ($0,79 \pm 2,01$) и контролната група ($1,46 \pm 2,07$) не се различава статистически (p -стойност = 0,830). Освен това няма значими разлики в NLR по пол, възраст или степен на ектазия. Средната стойност на NLR обаче е по-ниска при пациенти със засягане на три съда ($0,49 \pm 1,66$), отколкото при засягане на един съд ($1,04 \pm 2,32$) (p -стойност = 0,036). **Заклучение:** В това изследване NLR не се установява като диагностичен маркер за ектазия на коронарната артерия. Броят на участващите съдове обаче може да повлияе върху стойностите на NLR. Препоръчва се провеждането на допълнителни проучвания за изследване на патофизиологичните механизми на това заболяване и ролята на други възпалителни маркери.

Ключови думи: ектазия на коронарните артерии, съотношение неутрофили-лимфоцити, сърдечно-съдови заболявания, възпаление, ангиография

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INTRODUCTION

Coronary Artery Ectasia (CAE): Prevalence and Pathophysiology

Coronary artery ectasia (CAE) is defined as abnormal dilation of coronary arteries exceeding 1.5 times the normal vessel diameter. It is detected in 1-5% of patients undergoing coronary angiography worldwide, with regional variations – studies suggest a higher prevalence in Middle Eastern populations (3-7%) compared to Western cohorts (1-3%) [1]. While often asymptomatic, CAE can lead to angina, myocardial infarction, or sudden cardiac death due to abnormal blood flow dynamics, thrombus formation, or vasospasm [2].

The pathophysiology of CAE remains incompletely understood but involves a combination of: Endothelial dysfunction (impaired nitric oxide bioavailability, increased oxidative stress) Vascular smooth muscle degradation (elastin fragmentation, matrix metalloproteinase [MMP] activation) Chronic low-grade inflammation [3].

Atherosclerosis coexists in 50-70% of CAE cases, suggesting shared risk factors (hypertension, diabetes, and smoking). However, isolated CAE (without stenosis) implies distinct mechanisms, including: Inflammatory mediators: Elevated CRP, IL-6, and TNF- α levels are observed in CAE patients, even in non-atherosclerotic cases [3]. Autoimmune associations: CAE is linked to conditions like Kawasaki disease and systemic vasculitides [4]. Genetic predisposition: Familial cases and mutations in collagen/elastin genes are reported [5].

NLR as an Inflammatory Biomarker in Cardiovascular Disease: The neutrophil-to-lymphocyte ratio (NLR) integrates two immune pathways: Neutrophils drive oxidative stress and plaque instability. Lymphocytes regulate vascular repair and anti-inflammatory responses. Elevated NLR predicts adverse outcomes in acute cor-

onary syndromes, heart failure, and atherosclerosis [6, 7]. However, its role in CAE – a disease with unique inflammatory features – has not been explored.

Rationale and Hypothesis

Given that: CAE involves systemic inflammation beyond localized atherosclerosis. NLR reflects global inflammatory burden and is easily measurable. We hypothesized that: NLR would be higher in CAE patients vs. controls. NLR would correlate with CAE severity (e.g., number of affected vessels). This study aimed to test these hypotheses, potentially identifying NLR as a cost-effective diagnostic adjunct for CAE.

MATERIAL AND METHODS

Type and Method of Research

The study was a case-control study (retrospective).

Study Implementation, Sample Selection, and Data Collection

The study population was patients who underwent angiography at Shahid Sadoughi Hospital in Yazd during 2012-2020. Patient data were collected from the research data of Moradi and Afkhami. Of 380 reported cases of coronary artery ectasia (diffuse or focal dilation 1.5 times the diameter of the contiguous normal vessel segment without significant stenosis) identified in angiographic examinations, 28 patients with complete medical records were classified as the case group. The control group consisted of 87 patients with no dilation or stenosis on angiography and no ischemia or infarction apparent on stress tests. The two cohorts were similar in terms of demographic parameters (gender and age). The complete blood count (CBC) of the patients was obtained through archived clinical records, and neutrophil-to-lymphocyte ratio

(NLR) was calculated for both cohorts. Number of involved vessels (LAD, LCX, RCA) and extent of ectasia were recorded according to angiographic reports. Those variables such as history of hypertension (HTN), diabetes mellitus (DM), and smoking were abstracted from the patient record and documented. Markis classification was used to evaluate ectasia severity. Markis divides coronary artery ectasia into four types: Type 1: diffuse ectasia involving two or three vessels; Type 2: diffuse ectasia involving one vessel with localized ectasia in another; Type 3: diffuse ectasia involving only one vessel; and Type 4: localized or segmental ectasia in a single vessel [2]. Data was entered into SPSS software version 22. Required tables were designed, and statistical tests were done using Chi-Square, Fisher's Exact, T-Test, and ANOVA. P-value < 0.05 was taken statistically as significant.

Inclusion and Exclusion Criteria

Inclusion criteria:

- Patients referred to the cardiology departments of Shahid Sadoughi and Afshar Hospitals (2012-2020) who underwent angiography and were diagnosed with coronary artery ectasia (CAE).
- Patients with complete medical records and pure CAE (no concurrent stenosis or coronary artery disease).
- Among 380 identified CAE cases, 28 met the criteria for pure CAE and had sufficient data for analysis.

Exclusion Criteria:

- Acute Coronary Syndrome (ACS) at the time of angiography.
- CAE with coexisting coronary artery stenosis or atherosclerotic disease (CAD).
- Rheumatologic or inflammatory disorders (e.g., rheumatoid arthritis, lupus).
- History of rheumatic carditis or viral heart diseases (e.g., Kawasaki disease, myocarditis).
- Anemia (to eliminate confounding effects on cardiovascular parameters).
- Chronic Kidney Disease (CKD) (serum creatinine > 1.5 mg/dL).

Data Collection

Neutrophil-to-Lymphocyte Ratio (NLR) Calculation

NLR was derived as the ratio of absolute neutrophil count to absolute lymphocyte count obtained from:

- Complete blood count (CBC): Performed within 24 hours of angiography using [analyzer name/model] at Shahid Sadoughi Hospital laboratory.
- Formula: $NLR = \frac{\text{Neutrophils } (\times 10^3/\mu\text{L})}{\text{Lymphocytes } (\times 10^3/\mu\text{L})}$.

Variables Collected

Data were extracted from electronic medical records and angiographic reports using a standardized case report form:

1. Demographics: Age, sex.
2. Clinical History:
 - Hypertension (defined as BP \geq 140/90 mmHg or antihypertensive use).
 - Diabetes mellitus (fasting glucose \geq 126 mg/dL or hypoglycemic therapy).
 - Smoking status (current or past use).
3. Angiographic Parameters:
 - CAE Severity: Markis classification (Types I-IV).
 - Vessel Involvement: LAD, LCX, RCA (number and location).
4. Laboratory Data:
 - Absolute neutrophil and lymphocyte counts (for NLR).
 - Other relevant CBC indices (e.g., hemoglobin, platelets).

Quality Control

- Blinded Analysis: Two independent cardiologists reviewed angiograms (inter-rater agreement $\kappa = 0.85$).
- Data Verification: 10% of records were cross-checked for accuracy.

Data Analysis Method

After collection and verification, the data was transferred into SPSS version 22. Descriptive analysis was performed using percentages, means, and standard deviations. Analytical analyses were conducted using the Chi-square test for categorical variables, independent samples t-test for comparing two groups of continuous variables, and ANOVA for comparing more than two groups of continuous variables. For small cell counts, Fisher's exact test was used instead of the Chi-square test. A post-hoc power analysis was performed to assess the adequacy of the sample size. A p-value of < 0.05 was considered statistically significant in all analyses.

Innovation and Novelty of the Research

No studies assessing the correlation between NLR levels and the prevalence of CAE in Iran were found. This is the first study in Iran to examine the correlation between NLR levels and CAE.

RESULTS

Baseline Characteristics

The study population comprised **115 patients** (28 CAE cases, 87 controls) who underwent coronary angiography. Key demographic and clinical features are summarized below:

Demographics:

- **Sex distribution:**
 - Overall cohort: 68 males (59.1%), 47 females (40.9%)
 - CAE group: 17 males (60.7% \pm 9.2%), 11 females (39.3% \pm 9.2%)

Control group: 51 males (58.6% ± 5.3%), 36 females (41.4% ± 5.3%)

No significant difference ($\chi^2 = 0.04$, $p = 0.845$)

Comorbidities:

Hypertension prevalence was significantly lower in CAE patients (39.3% ± 9.2% vs. 60.9% ± 5.2%; $\chi^2 = 4.02$, $p = 0.045$)

Diabetes mellitus showed borderline significance (17.9% ± 7.2% vs. 37.9% ± 5.2%; $\chi^2 = 3.81$, $p = 0.051$)

Smoking history was comparable between groups (17.9% ± 7.2% vs. 25.3% ± 4.6%; $\chi^2 = 0.65$, $p = 0.42$)

CAE Morphological Features

Severity distribution (n = 26 assessable cases):

Grade 1: 15 cases (57.7% ± 9.7%)

Grade 2: 4 cases (15.4% ± 7.1%)

Grade 3: 1 case (3.8% ± 3.7%)

Grade 4: 6 cases (23.1% ± 8.3%)

Vessel involvement (n = 28):

Single-vessel: 8 cases (28.6% ± 8.5%)

Two-vessel: 5 cases (17.9% ± 7.2%)

Three-vessel: 15 cases (53.6% ± 9.4%)

NLR Analysis

Comparative analysis:

Mean NLR was comparable between groups (CAE: 2.01 ± 0.79 vs Controls: 2.07 ± 1.46; $t = 0.21$, $p = 0.830$)

By CAE severity:

Grade 1: 2.03 ± 0.82

Grade 2: 1.52 ± 0.36

Grade ≥ 3: 2.54 ± 0.86

No significant trend ($F = 2.31$, $p = 0.113$)

By vessel involvement:

Single-vessel: 2.32 ± 1.04

Two-vessel: 2.55 ± 0.73

Three-vessel: 1.66 ± 0.49

Significant difference ($F = 3.56$, $p = 0.036$) with post-hoc tests showing three-vessel vs single-vessel $p = 0.028$

Subgroup Analyses

Sex-based differences in CAE:

Males: 2.09 ± 0.78

Females: 1.89 ± 0.84

No significant difference ($t = 0.71$, $p = 0.244$)

Age stratification:

Younger cohort (30-59y): 1.97 ± 0.59

Older cohort (60-86y): 2.07 ± 1.03

No significant difference ($t = 0.26$, $p = 0.795$)

Table 1. Demographic and Clinical Characteristics

Variable	Total (n = 115)	Ectasia Group (n = 28)	Control Group (n = 87)	p-value
Gender				
- Male	68 (59.1%)	17 (60.7%)	51 (58.6%)	0.845
- Female	47 (40.9%)	11 (39.3%)	36 (41.4%)	
Hypertension (HTN)	64 (55.7%)	11 (39.3%)	53 (60.9%)	0.045
Diabetes Mellitus (DM)	38 (33%)	5 (17.9%)	33 (37.9%)	0.051
Smoking History	27 (23.5%)	5 (17.9%)	22 (25.3%)	0.42

Table 2. Distribution of Ectasia Severity (n = 26)

Ectasia Severity	Number of Patients	Percentage
Grade 1	15	57.7%
Grade 2	4	15.4%
Grade 3	1	3.8%
Grade 4	6	23.1%

Table 3. Distribution of Involved Vessels (n = 28)

Number of Involved Vessels	Number of Patients	Percentage
Single-vessel	8	28.6%
Two-vessel	5	17.9%
Three-vessel	15	53.6%

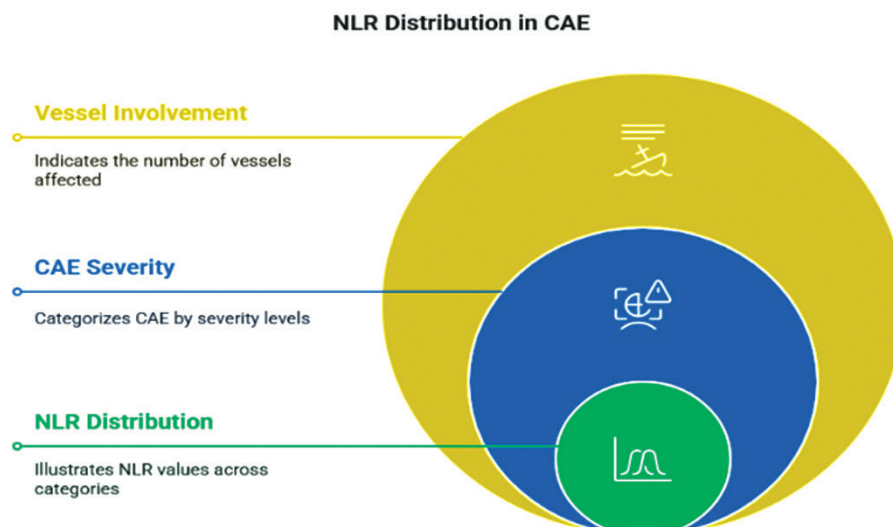


Fig. 1. NLR Distribution Patterns in CAE

***A)** Violin plot showing NLR distribution across CAE severity grades, demonstrating similar medians but wider dispersion in higher grades

B) Bar chart (mean ± SEM) of NLR by vessel involvement, highlighting significantly lower values in three-vessel disease ($p < 0.05$)*

Table 4. Mean NLR in Ectasia and Control Groups

Group	Mean NLR	Standard Deviation	P-Value
Ectasia Group	2.01	0.79	0.830
Control Group	2.07	1.46	

Table 5. Mean NLR by Ectasia Severity

Ectasia Severity	Mean NLR	Standard Deviation	P-Value
Grade 1	2.03	0.82	0.113
Grade 2	1.52	0.36	
Grade ≥ 3	2.54	0.86	

Table 6. Mean NLR by Number of Involved Vessels

Number of Involved Vessels	Mean NLR	Standard Deviation	P-Value
Single-vessel	2.32	1.04	0.036
Two-vessel	2.55	0.73	
Three-vessel	1.66	0.49	

Table 7. Mean NLR by Gender and Age

Variable	Subgroup	Mean NLR	Standard Deviation	P-Value
Gender	Male (Ectasia)	2.09	0.78	0.244
	Female (Ectasia)	1.89	0.84	
Age (Years)	30-59	1.97	0.59	0.795
	60-86	2.07	1.03	

DISCUSSION

Coronary artery diseases are one of the most common cardiovascular diseases. One such disease is coronary artery ectasia (CAE), which is diffuse or focal dilatation of a coronary artery segment to 1.5 times the diameter of the immediately adjacent normal vessel segment [8].

Gender Impact

Previous research has found CAE to be more prevalent in men [9]. In the current study, 60.7% of the patients with ectasia were male. Similarly, in Nikseresht's research, 54.6% of the patients were male [10]. In Amirzadegan's research, CAE was 2.7% in men and 1.4% in women, indicating greater prevalence among men [11]. In a Saudi Arabian research, 82% of the patients were male [12]. In general, CAE is more common in men.

Severity of Ectasia (Based on Markis Classification)

Our study results revealed that among ectasia patients, 57.7% were of type 1 ectasia, 15.4% were of type 2, 3.8% were of type 3, and 23.1% were of type 4. Of the distribution of ectasia types according to Alman-

sori, 42% was type 4, 22% was type 2, 21% was type 1, and 15% was type 3 [12].

Distribution of Involved Vessels

The results of our research on involved vessel distribution were that 53.6% of patients had three-vessel involvement, 17.9% had two-vessel involvement, and 28.6% had single-vessel involvement. In Nikseresht's study, ectasia was in one vessel in 50.9% of patients, in two vessels in 26.9%, and in three vessels in 20.4% [10]. In Amirzadegan's study, 44.4% of patients had two-vessel involvement, 7% had three-vessel involvement, and 48.6% had single-vessel involvement [11].

Role of Hematologic Indices

Certain studies have shown that hematologic indices such as total leukocyte count, neutrophil count, and monocyte count are implicated in the prognosis of CAE patients [13]. One of these parameters which is currently analyzed in CBC is the neutrophil-to-lymphocyte ratio (NLR). It has been broadly established that elevated NLR can be implicated in the development of myocardial infarction, stroke, and thromboembolism [14, 15].

Findings of Previous Studies on NLR

- A study in Turkey investigating the relationship between NLR and right ventricular dysfunction in patients with acute inferior myocardial infarction found that NLR was significantly elevated in patients with right ventricular dysfunction [16].

- In a research by Gul on the correlation between NLR and cardiac complications and mortality among acute myocardial infarction patients, it was found that patients with high NLR values had significantly increased rates of cardiac complications and mortality [17].

- In 2015 research investigating the relationship between NLR and the severity of ischemia in patients with acute myocardial infarction, the authors found that NLR was elevated in patients with grade 3 ischemia. The authors suggested that in the future, NLR would be used as a predictor and risk estimator for acute myocardial infarction patients [18].

- Soylyu's study, which aimed at investigating the relationship between reestablishment of coronary blood flow and NLR in primary angioplasty patients, found that NLR was independently elevated in patients with ischemic segments without reflow after angioplasty [19].

- A 2013 study conducted in Adana also showed that the inexpensive and easily accessible marker NLR was elevated in patients with ischemic segments without reflow after primary angioplasty [20].

- A study conducted in Hatay, Turkey, found that patients who failed with reflow and had less than 30% ST-segment resolution had greater NLR

levels compared to patients with complete or partial resolution. The study also showed that greater NLR on admission in patients with acute myocardial infarction with primary angioplasty was associated with no-reflow phenomenon and long-term prognosis [21].

NLR and CAE

Based on the results of the above studies, it can be said that there is a significant association between NLR and ischemic heart diseases. But in our research, no statistically significant difference was found between the two groups regarding the mean NLR, indicating that NLR has no effect on the formation of coronary artery ectasia.

The lack of a high statistical correlation between NLR and coronary artery ectasia (compared to ischemic heart disease) might be attributed to the two conditions having different pathophysiologies. Recent studies have shown that in ischemic heart disease, neutrophils, the initial line of defense among the white blood cells, are sequestered in the infarct region and induce the inflammatory process by releasing mediators such as myeloperoxidase, elastase, oxygen free radicals, and arachidonic acid metabolites [22]. The consequence is increased tissue damage, activation of the coagulation cascade, thrombosis, microvascular plugging, necrosis of myocytes, and enlargement of the infarct size [23]. Conversely, lymphocytes, being the controllers of the inflammatory reaction, have an effective role in limiting damage to ischemic myocardial areas [24, 25]. Thus, neutrophils, lymphocytes, and the neutrophil-to-lymphocyte ratio may determine the occurrence of ischemic heart diseases.

Limitations

Sample Size and Study Design

The study's primary limitations include its small sample size ($n = 28$ CAE patients) and single-center design, which may restrict the generalizability of findings. Notably, subgroup analyses by CAE severity were underpowered, with only 6 patients in grade ≥ 3 ectasia. The single-center recruitment could introduce selection bias, as local referral patterns and demographic factors may not reflect broader CAE populations.

Potential Confounding Factors

Comorbidities: Despite matching, the control group had significantly higher hypertension prevalence (60.9% vs. 39.3%, $*p = 0.045$), which may influence NLR independently of CAE.

Medications: Use of statins, anti-inflammatory drugs, or immunosuppressants – common in CAD patients – could modulate NLR but were not systematically adjusted for.

Temporal Variability: NLR was measured at a single timepoint, ignoring potential fluctuations due to acute infections or stress.

Recommendations for Future Research

Multi-Center Studies: Larger, prospective cohorts (e.g. $n \geq 200$ CAE patients) are needed to validate NLR's role, with stratification by severity and adjustment for confounders (e.g. medications, comorbidities).

Exploration of Additional Markers: Inflammatory biomarkers like hs-CRP, IL-6, or neutrophil extracellular traps (NETs) may better reflect CAE's pathophysiology and should be investigated alongside NLR.

Longitudinal Designs: Repeated NLR measurements pre/post CAE diagnosis could clarify its utility as a dynamic prognostic marker.

CONCLUSION

This study yielded two key findings regarding the neutrophil-to-lymphocyte ratio (NLR) in coronary artery ectasia (CAE):

Primary Finding: No significant association was observed between NLR and the presence of CAE (mean NLR 2.01 ± 0.79 in CAE vs 2.07 ± 1.46 in controls, $p=0.830$), suggesting NLR lacks diagnostic value for CAE detection.

Notable Association: Patients with three-vessel CAE demonstrated significantly lower NLR values (1.66 ± 0.49) compared to those with single-vessel involvement (2.32 ± 1.04 , $p=0.036$), indicating a potential relationship between NLR and disease extent.

Ethical Considerations

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki and was approved by the Institutional Review Board (IRB) of Shahid Sadoughi University of Medical Sciences, Yazd, Iran (Ethical code IR.IAU.KHUISF.REC.1399.222). Written informed consent was obtained from all participants prior to their inclusion in the study. Patient confidentiality was protected through anonymization of all collected data.

No conflict of interest was declared

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