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Systemic inflammatory–nutritional status and coronary collateral development in chronic total occlusion: The role of the CALLY index

Kronik tam tıkanıklıkta sistemik enflamatuvar– beslenme durumu ve koroner kollateral gelişimi: CALLY indeksinin rolü

Muhammet Raşit TANIRCAN[1]
Adem AKTAN[2]
Raif KILIÇ[2]
Tuncay GÜZEL[3]
Mehmet KIŞ[4]
Ali EVSEN[5]
Mehmet Sait COŞKUN[6]
Mehmet Zülküf KARAHAN[2]

[1]Department of Cardiology, Mardin Training and Research Hospital, Mardin, Türkiye
[2] Department of Cardiology, Mardin Artuklu University Faculty of Medicine, Mardin, Türkiye
[3]Department of Cardiology, Health Sciences University, Gazi Yaşargil Training and Research Hospital, Diyarbakır, Türkiye
[4]Department of Cardiology, Dokuz Eylül University Faculty of Medicine, İzmir, Türkiye
[5]Department of Cardiology, Dağkapı State Hospital, Diyarbakır, Türkiye
[6]Department of Cardiology, Ergani State Hospital, Diyarbakır, Türkiye

CALLY Index and CTO Collaterals
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Corresponding author
Muhammet Raşit TANIRCAN
ORCID ID: 0000-0002-7480-9983
drtanircan21@hotmail.com

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ABSTRACT

BACKGROUND

In patients with chronic total occlusion (CTO), collateral development varies despite similar angiographic disease burden. The C-reactive protein–albumin–lymphocyte (CALLY) index may reflect systemic inflammatory, nutritional, and immune status, but its association with collateral development in CTO is not well defined. This study aimed to evaluate the relationship between the CALLY index and angiographic collateral status in patients with CTO.

METHODS

We retrospectively evaluated 306 consecutive patients diagnosed with CTO by coronary angiography at a single center between January 2023 and December 2025. Angiographic collateralization was graded according to the Rentrop classification; grades 0–1 were defined as poor collateral development and grades 2–3 as good collateral development. Baseline clinical features, laboratory measurements, and angiographic findings were analyzed according to collateral status. Logistic regression models were used to test the independent relationship between the CALLY index and good collateral development, while receiver operating characteristic (ROC) curve analysis was performed to assess its discriminatory performance.

RESULTS

Patients with poor collateral development had a more pronounced inflammatory profile, accompanied by lower albumin levels, lymphocyte counts, and CALLY index values. The median CALLY index value was lower among patients with poor collateralization compared to those with good collateralization [2.70 (1.30–7.33) vs. 4.56 (2.42–9.27), $p < 0.001$]. After adjustment for potential confounders, each 1-unit increase in the CALLY index was independently associated with higher odds of good collateral development (odds ratio: 1.03, 95% confidence interval: 1.01–1.05, $p = 0.005$). ROC curve analysis showed that the CALLY index had limited but statistically significant discriminatory ability for identifying good collateral development (area under the curve: 0.621, $p < 0.001$).

CONCLUSION

Higher CALLY index values were independently associated with better coronary collateral development in patients with CTO. Conversely, lower CALLY index values may reflect an unfavorable inflammatory and nutritional profile related to impaired collateralization.

KEYWORDS

CALLY index, chronic total occlusion, coronary artery disease, coronary collateral circulation, inflammation

ÖZ

AMAÇ

Kronik total oklüzyon (KTO) hastalarında, benzer anjiyografik hastalık yüküne rağmen kollateral gelişimi değişkenlik göstermektedir. C-reaktif protein-albümin-lenfosit (CALLY) indeksi sistemik inflamatuar, beslenme ve bağışıklık durumunu yansıtabilir; ancak KTO'da kollateral gelişimi ile olan ilişkisi tam olarak tanımlanmamıştır. Bu çalışma, KTO hastalarında CALLY indeksi ile anjiyografik kollateral durumu arasındaki ilişkiyi değerlendirmeyi amaçlamıştır.

GEREÇ YÖNTEM

Ocak 2023 ile Aralık 2025 tarihleri arasında tek bir merkezde koroner anjiyografi ile KTO tanısı alan ardışık 306 hasta geriye dönük olarak değerlendirildi. Anjiyografik kollateralizasyon Rentrop sınıflamasına göre derecelendirildi; 0–1. dereceler zayıf kollateral gelişimi, 2–3. dereceler ise iyi kollateral gelişimi olarak tanımlandı. Başlangıç klinik özellikleri, laboratuvar ölçümleri ve anjiyografik bulgular kollateral durumuna göre analiz edildi. CALLY indeksi ile iyi kollateral gelişimi arasındaki bağımsız ilişkiyi test etmek için lojistik regresyon modelleri kullanılırken, ayırt edici performansını değerlendirmek amacıyla alıcı işletim karakteristiği (ROC) eğrisi analizi yapıldı.

BULGULAR

Zayıf kollateral gelişimi olan hastalarda daha belirgin bir inflamatuar profil izlenirken, bu duruma daha düşük albümin düzeyleri, lenfosit sayıları ve CALLY indeksi değerleri eşlik etmekteydi. İyi kollateralizasyonu olanlara kıyasla, zayıf kollateralizasyonu olan hastalarda medyan CALLY indeksi değeri daha düşüktü [2,70 (1,30–7,33) ve 4,56 (2,42–9,27); $p < 0,001$]. Olası karıştırıcı faktörlere göre yapılan düzeltmenin ardından, CALLY indeksindeki her 1 birimlik artış, iyi kollateral gelişimi olasılığının artmasıyla bağımsız olarak ilişkili bulundu (olasılık oranı: 1,03; %95 güven aralığı: 1,01–1,05; $p = 0,005$). ROC eğrisi analizi, CALLY indeksinin iyi kollateral gelişimini belirlemede sınırlı ancak istatistiksel olarak anlamlı bir ayırt etme yeteneğine sahip olduğunu gösterdi (eğri altındaki alan: 0,621; $p < 0,001$).

SONUÇ

KTO hastalarında daha yüksek CALLY indeksi değerleri, daha iyi koroner kollateral gelişimi ile bağımsız olarak ilişkili bulunmuştur. Buna karşılık, daha düşük CALLY indeksi değerleri, bozulmuş kollateralizasyonla ilişkili olumsuz bir inflamatuar ve beslenme profilini yansıtabilir.

ANAHTAR KELİMELER

CALLY indeksi, , inflamasyon, kronik total oklüzyon, koroner arter hastalığı, koroner kollateral dolaşım

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Coronary artery disease (CAD) is a progressive vascular disorder in which atherosclerosis, systemic inflammation, and cardiometabolic risk factors interact over time. Inflammation has a central role in the initiation and progression of atherosclerotic disease (1). In addition, the increasing burden of cardiovascular risk factors, aging, chronic kidney disease, and inflammation-related biological mechanisms continues to contribute to CAD-related morbidity and mortality (2,3).

Chronic total occlusion (CTO) represents an advanced form of CAD and is generally defined as complete interruption of antegrade coronary flow for at least 3 months (4). Despite improvements in percutaneous coronary intervention techniques, CTO remains clinically important because it is associated with ischemic symptoms, impaired myocardial perfusion, and procedural complexity (5,6).

Coronary collateral circulation is an adaptive vascular response that may develop in the presence of severe coronary stenosis or occlusion (7,8). When sufficiently developed, collateral vessels can support distal myocardial perfusion and may reduce the extent of ischemic injury (9,10). However, the degree of collateralization varies markedly among patients with similar angiographic disease severity. This suggests that collateral development is influenced not only by coronary anatomy but also by systemic biological conditions.

Inflammatory activity, nutritional reserve, and immune status may all contribute to vascular remodeling and collateral vessel formation. C-reactive protein (CRP) is a routinely available marker of systemic inflammation and has been associated with coronary inflammation and plaque vulnerability (11). Serum albumin and lymphocyte count may provide additional information about nutritional status, inflammatory burden, and immune–inflammatory balance in cardiovascular disease (12–14). Previous studies have shown that inflammation, oxidative stress, and impaired nutritional status are related to adverse cardiovascular outcomes (15,16).

The CRP–albumin–lymphocyte (CALLY) index is a composite marker calculated from CRP, serum albumin, and lymphocyte count. It has been studied mainly in malignancies and, more recently, in clinical settings characterized by systemic inflammation and cardiovascular risk (17–19). However, whether the CALLY index is associated with coronary collateral development in patients with CTO has not been fully clarified. Therefore, this study aimed to evaluate the relationship between the CALLY index and angiographic collateral status in patients with CTO.

Materials and Methods

Study design and patient selection

This single-center retrospective study included patients who underwent coronary angiographic evaluation at Mardin Training and Research Hospital between January 2023 and December 2025. Patients with chronic total occlusion in at least one major epicardial coronary artery were screened. After applying the eligibility criteria, 306 patients were included in the final analysis. Of these, 144 patients had poor collateral development and 162 had good collateral development. Demographic data, clinical characteristics, laboratory parameters, and angiographic findings were obtained from electronic medical records.

Eligibility criteria

Patients aged 18 years or older were eligible if they had CTO in at least one major epicardial coronary artery and available CRP, albumin, and lymphocyte measurements within 24 h of admission. Patients with acute coronary syndrome within the previous 30 days, active infection, malignancy, chronic inflammatory or autoimmune disease, severe hepatic dysfunction, advanced renal impairment defined as estimated glomerular filtration rate of <30 mL/min/1.73 m2, or missing data were excluded.

Angiographic assessment of collateral flow

Coronary angiography was performed through radial or femoral access using the standard Judkins technique. All angiograms were independently evaluated by two experienced interventional cardiologists. In cases of disagreement, the final decision was reached by consensus. Coronary collateral flow was assessed according to the Rentrop classification. Rentrop grades 0–1 were considered poor collateral development, whereas grades 2–3 were considered good collateral development.and were aware that all data could be used anonymously for scientific purposes only.

Calculation of the CALLY index

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The CALLY index was calculated using CRP, serum albumin, and lymphocyte count as follows:
CALLY = Serum albumin (g/dL) × absolute lymphocyte count (103/μL) / CRP (mg/L) The CALLY index was entered into logistic regression models as a continuous variable and the odds ratios (ORs) were reported per 1-unit increase. The neutrophil-to-lymphocyte ratio (NLR) was calculated by dividing the neutrophil count by the lymphocyte count, and the platelet-to-lymphocyte ratio (PLR) was calculated by dividing the platelet count by the lymphocyte count. Only laboratory values obtained within the first 24 h of admission were used.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was evaluated before group comparisons. Continuous variables were presented as mean ± standard deviation or median with interquartile range according to the data distribution. Categorical variables were expressed as numbers and percentages.
The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Normally distributed continuous variables were compared using the independent-samples t-test, whereas non-normally distributed variables were compared using the Mann–Whitney U test. Categorical variables were compared using the Pearson chi-square test or Fisher exact test as appropriate.

Logistic regression analysis was used to identify factors associated with good collateral development. Good collateral development was coded as 1, whereas poor collateral development was coded as 0. Variables with a p-value of <0.10 in univariable analysis, together with clinically relevant covariates, were considered for inclusion in the multivariable logistic regression model. The CALLY index was analyzed as a continuous variable, and the corresponding ORs were expressed per 1-unit increase. Results were reported as ORs with 95% confidence intervals (CIs).

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the ability of the CALLY index to identify good collateral development. The optimalcut-off value was determined using the Youden index. A two-sided value of $p < 0.05$ was considered statistically significant.

Ethics approval

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the local ethics committee (Protocol No: 2026/04-14; Approval Date: 24.04.2026). Because of the retrospective design, the requirement for written informed consent was waived.

Results

Among the 306 patients analyzed, 144 had poor collateral development and 162 had good collateral development. Most baseline demographic and clinical variables did not differ significantly according to collateral status. Smoking was numerically more frequent in the group with poor collateral development, but this difference was not statistically significant (39.6% vs. 30.2%, p = 0.087). The prevalence of heart failure and multivessel coronary disease was similar between the groups (p = 0.215 and p = 0.288, respectively). Left ventricular ejection fraction tended to be higher in patients with good collateralization, although the difference remained below the level of statistical significance (47.16 ± 9.84% vs. 44.99 ± 10.38%, p = 0.061).

The target CTO vessel distribution was comparable between the groups (p = 0.341), with the right coronary artery being the most commonly involved vessel in both groups. Lesion-related parameters, including J-CTO score, lesion length of >20 mm, blunt entry, calcification, and tortuosity, were also comparable between patients with poor and good collateralization (Table 1).

Laboratory comparisons showed a more pronounced inflammatory profile among patients with poor collateral development. CRP values were higher in patients with poor collateralization than in those with good collateralization (2.60 [1.41–4.24] vs. 1.89 [1.20–2.98] mg/L, p = 0.001). Conversely, patients with good collateralization had higher albumin levels (4.02 ± 0.80 vs. 3.51 ± 0.70 g/dL, p < 0.001) and lymphocyte counts (2.31 ± 0.98 vs. 2.06 ± 0.78 × 103/μL, p = 0.014). Neutrophil count was greater in the group with poor collateral development (7.08 ± 3.57 vs. 6.31 ± 2.92 × 103/μL, p = 0.039), whereas the difference in white blood cell count showed only borderline significance (p = 0.065).

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The CALLY index was higher among patients with good collateral development compared to those with poor collateral development (4.56 [2.42–9.27] vs. 2.70 [1.30–7.33], p < 0.001). In contrast, both NLR and PLR were increased in the group with poor collateral development (p = 0.042 and p = 0.049, respectively). Hemoglobin, platelet count, renal function parameters, glucose, and lipid measurements were similar between the two groups (Table 2).

In logistic regression analysis, the CALLY index value was evaluated as a continuous variable. In univariable analysis, each 1-unit increase in the CALLY index was associated with higher odds of good collateral development (OR: 1.03, 95% CI: 1.01–1.05, p = 0.004). After adjustment for age, sex, smoking status, and left ventricular ejection fraction, the CALLY index remained independently associated with good collateral development (OR: 1.03, 95% CI: 1.01–1.05, p = 0.005). Left ventricular ejection fraction also showed an independent association with good collateral status (OR: 1.02, 95% CI: 1.00–1.04, p = 0.046). Age, sex, and smoking did not retain statistical significance in the adjusted model (Table 3).

ROC curve analysis indicated that the discriminatory ability of the CALLY index for identifying good collateral development was significant but modest. The area under the curve was 0.621 (95% CI: 0.558–0.684, p < 0.001). Using the Youden index, the optimal threshold was found to be approximately 3.0, with sensitivity of 67% and specificity of 53% (Figure 1). These results indicate that the CALLY index may provide additional clinical information regarding collateral status, although its discriminatory performance was limited.

Table 1. Baseline clinical and angiographic characteristics.

Variable	Poor collateral development (Rentrop 0–1) n= 144	Good collateral development (Rentrop 2–3) n= 162	p
Age (years)	61.88 ± 10.23	63.66 ± 9.14	0.108
Female sex	37 (25.7)	49 (30.2)	0.377
Hypertension	65 (45.1)	62 (38.3)	0.224
Diabetes mellitus	53 (36.8)	48 (29.6)	0.183
Current smoking	57 (39.6)	49 (30.2)	0.087
Family history of CAD	23 (16)	18 (11.1)	0.213
Chronic kidney disease	7 (4.9)	10 (6.2)	0.617
Previous MI	10 (6.9)	15 (9.3)	0.461
Previous PCI	12 (8.3)	18 (11.1)	0.415
Previous CABG	7 (4.9)	4 (2.5)	0.262
Heart failure	76 (52.8)	74 (45.7)	0.215
LVEF (%)	44.99 ± 10.38	47.16 ± 9.84	0.061
Multivessel disease	95 (66)	116 (71.6)	0.288
Target CTO vessel			0.341
LAD	36 (25.0)	52 (32.1)	
LCx	40 (27.8)	37 (22.8)	

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RCA	68 (47.2)	73 (45.1)	
J-CTO score			0.256
<2	87 (60.4)	108 (66.7)	
≥2	57 (39.6)	54 (33.3)	
Lesion length (>20 mm)	79 (54.9)	74 (45.7)	0.109
Blunt stump	28 (19.4)	44 (27.2)	0.112
Calcification	46 (31.9)	42 (25.9)	0.246
Tortuosity	47 (32.6)	52 (32.1)	0.920

Continuous variables are presented as mean ± standard deviation; categorical variables are presented as number (%). CAD; Coronary artery disease, MI; myocardial infarction, PCI; percutaneous coronary intervention, CABG; coronary artery bypass grafting, LVEF; left ventricular ejection fraction, CTO; chronic total occlusion, LAD; left anterior descending artery, LCx; left circumflex artery, RCA; right coronary artery, J-CTO score; Japanese Multicenter CTO Registry score.

Table 2. Laboratory parameters and calculated indices.

Variable	Poor collateral development (Rentrop 0–1)	Good collateral development (Rentrop 2–3)	p
CRP (mg/L)	2.60 (1.41–4.24)	1.89 (1.20–2.98)	0.001
Albumin (g/dL)	3.51 ± 0.70	4.02 ± 0.80	<0.001
Lymphocyte count (×10³/µL)	2.06 ± 0.78	2.31 ± 0.98	0.014
White blood cell count (×10³/µL)	10.15 ± 3.45	9.44 ± 3.27	0.065
Neutrophil count (×10³/µL)	7.08 ± 3.57	6.31 ± 2.92	0.039
Hemoglobin (g/dL)	13.86 ± 1.88	13.52 ± 1.71	0.094
Platelet count (×10³/µL)	253.38 ± 75.80	246.54 ± 65.67	0.398
Creatinine (mg/dL)	1.01 ± 0.48	1.03 ± 0.46	0.696
eGFR (mL/min/1.73 m²)	83.79 ± 25.82	80.37 ± 23.47	0.225
Fasting glucose (mg/dL)	132 (107–153)	117 (92–212)	0.097
Total cholesterol (mg/dL)	181.09 ± 45.30	175.00 ± 43.23	0.230
LDL-C (mg/dL)	108.76 ± 35.83	103.32 ± 35.69	0.185
HDL-C (mg/dL)	38.93 ± 8.06	38.11 ± 7.19	0.348
Triglycerides (mg/dL)	136 (98–183)	151 (92–210)	0.389
CALLY index	2.70 (1.30–7.33)	4.56 (2.42–9.27)	<0.001
NLR	2.87 (2.11–5.10)	2.70 (1.77–4.14)	0.042
PLR	130 (90–180)	120 (80–160)	0.049

Continuous variables are presented as mean ± standard deviation or median (interquartile range) according to data distribution. CRP; C-reactive protein, eGFR; estimated glomerular filtration rate, LDL-C; low-density lipoprotein cholesterol, HDL-C; high-density lipoprotein cholesterol, CALLY; C-reactive protein–albumin–lymphocyte, NLR; neutrophil-to-lymphocyte ratio, PLR; platelet-to-lymphocyte ratio.

Table 3. Logistic regression analysis for good collateral development.

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Variable	Univariable OR (95% CI)	p	Multivariable OR (95% CI)	p
Age	1.01 (0.99–1.04)	0.109	1.01 (0.99–1.04)	0.193
Female sex	1.25 (0.75–2.07)	0.377	1.17 (0.68–2.03)	0.555
Diabetes mellitus	0.72 (0.44–1.16)	0.183		
Hypertension	0.75 (0.47–1.18)	0.224		
Smoking	0.66 (0.41–1.06)	0.087	0.72 (0.43–1.20)	0.217
eGFR	0.99 (0.98–1.00)	0.225		
LVEF	1.02 (0.99–1.04)	0.062	1.02 (1.00–1.04)	**0.046**
Multivessel disease	1.30 (0.80–2.11)	0.288		
J-CTO score	0.76 (0.47–1.21)	0.257		
CALLY index per 1-unit increase	1.03 (1.01–1.05)	0.004	1.03 (1.01–1.05)	**0.005**

Variables with values of $p < 0.10$ in univariable analysis, together with age and sex as clinically relevant covariates, were included in the multivariable logistic regression model. The dependent variable was good collateral development, defined as Rentrop grades 2–3. OR: Odds ratio; CI; confidence interval, eGFR; estimated glomerular filtration rate, LVEF; left ventricular ejection fraction, J-CTO score; Japanese Multicenter CTO Registry score, CALLY; C-reactive protein–albumin–lymphocyte.

Discussion

In this study, the CALLY index was found to be significantly related to angiographic collateral status in patients with CTO. Patients with poor collateralization had lower CALLY index values, while higher CALLY index values remained independently associated with good collateral development after multivariable adjustment. This finding suggests that collateral formation in CTO may be influenced not only by lesion-related characteristics but also by the patient's systemic inflammatory, nutritional, and immune profile.

Coronary collateral circulation represents an important compensatory mechanism in chronic coronary occlusion. Previous studies have shown that well-developed collaterals may protect the ischemic myocardium and contribute to better clinical outcomes (7–10). In the present cohort, however, anatomical and lesion-related parameters were largely comparable between the poor and good collateral groups. This supports the conclusion that non-anatomical factors may partly explain the variability in collateral development among CTO patients.

The lower CALLY values observed in patients with poor collateralization may reflect a more unfavorable inflammatory background. CRP, one of the components of the CALLY index, is closely related to systemic and coronary inflammation (1,4,11). In our study, CRP levels were higher in the group with poor collateral development, suggesting that inflammatory predominance may be associated with insufficient collateral adaptation. This relationship is biologically plausible because persistent inflammation may impair endothelial function and limit vascular remodeling.

Albumin and lymphocyte count also differed between the groups. Patients with good collateral development had higher albumin levels and lymphocyte counts. Albumin has been associated with cardiovascular prognosis and may reflect nutritional reserve, inflammatory burden, and vascular homeostasis (13). Similarly, lower lymphocyte counts have been linked to adverse outcomes in ischemic heart disease (12). Therefore, the combination of lower albumin, lower lymphocyte count, and higher CRP in patients with poor collateralization may represent a systemic environment that is less favorable for collateral vessel maturation.

The independent association between the CALLY index and collateral status may be explained by the ability of

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this index to capture several non-anatomical pathways in a single laboratory-based variable. Rather than reflecting only inflammation, the CALLY index also incorporates nutritional and immune-related components. This may be particularly relevant in CTO, where collateral formation depends on a coordinated biological response involving endothelial integrity, inflammatory balance, and vascular remodeling.

Although the CALLY index was first investigated mainly in oncological populations, its prognostic relevance has recently been evaluated in cardiovascular conditions (16–19). Previous studies involving patients with ST-segment elevation myocardial infarction showed that lower CALLY index values were associated with worse clinical outcomes after primary percutaneous coronary intervention (16,19). In addition, a population-based study reported an association between the CALLY index and cardiorenal syndrome (18). Our findings add a different perspective by suggesting that this index may also be related to the degree of collateral development in CTO patients.

The clinical value of the CALLY index should be interpreted carefully. Although it was independently associated with good collateral development in this study, its discriminatory performance according to ROC analysis was modest. Therefore, the CALLY index should not be considered a standalone marker for predicting collateral status. Instead, it may provide additional information about the systemic biological background of CTO patients when interpreted together with clinical and angiographic findings.

This study has several limitations. First, the retrospective and single-center design may limit the generalizability of the findings. Second, collateral circulation was evaluated using the Rentrop classification, which is based on angiographic appearance and may not fully represent functional collateral capacity. Third, inflammatory and nutritional parameters were measured at a single time point; therefore, changes over time could not be assessed. Fourth, despite multivariable adjustment, residual confounding cannot be excluded. Finally, the observational design does not allow a causal relationship to be established between the CALLY index and collateral development.

Conclusion

The CALLY index was independently associated with angiographic collateral status in patients with CTO. Although its discriminatory ability was limited, this simple laboratory-based index may help to characterize the inflammatory, nutritional, and immune profile associated with collateral development. Prospective studies are needed to confirm these findings and to determine whether the CALLY index has practical value in the clinical assessment of CTO patients.

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Data Availability
The datasets supporting the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available due to privacy and ethical restrictions.
Ethical Declaration
The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and was approved by the Mardin Training and Research Hospital Scientific Research Ethics Committee (Protocol No: 2026/04-14; Approval Date: April 24, 2026). Due to the retrospective nature of the study, the requirement for written informed consent was waived by the ethics committee.

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