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Original Article / Özgün Araştırma

How does colchicine use affect neutrophil/lymphocyte, monocyte/lymphocyte, and platelet/lymphocyte ratios in coronary artery disease?

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Abstract

Objective: It is an established medical fact that the reduction of inflammation plays a pivotal role in the development of atherosclerosis. This reduction of inflammation is an accepted treatment strategy for coronary artery disease (CAD). The objective of the present study was to demonstrate the effect of colchicine, a commonly used anti-inflammatory agent, on inflammatory markers.

Method: The study comprised a sample of 122 patients diagnosed with CAD, whose medical records were meticulously reviewed. Ratios of neutrophil/lymphocyte, monocyte/lymphocyte, and platelet/lymphocyte were calculated and recorded. Following a period of six months during which colchicine was administered, a recalculation of the values was performed.

Results: Prior to the commencement of treatment, the neutrophil/lymphocyte ratio (NLR) (3.99 ± 4.72), monocyte/lymphocyte ratio (MLR) (0.41 ± 0.42), and platelet/lymphocyte ratio (PLR) (129.6 ± 74.1) were observed. Following treatment, a decrease was observed in the neutrophil-to-lymphocyte ratio (NLR) (3.5 ± 3), MLR (0.38 ± 0.28), and PLR (122.6 ± 65). In the post-treatment period, statistically significant decreases were observed NLR, MLR, and PLR compared to pre-treatment levels (p<0.001 for all values).

Conclusion: In the present study, a significant decrease in the levels of NLR, MLR and PLR was observed in patients with CAD following the initiation of colchicine treatment.

Keywords: colchicine, neutrophil/lymphocyte ratios, monocyte/lymphocyte ratios, platelet/lymphocyte ratios, coronary artery disease

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Kolşisin kullanımı koroner arter hastalığında nötrofil/lenfosit, monosit/lenfosit ve trombosit/lenfosit oranlarını nasıl etkiler?

Öz

Amaç: İnflamasyonun azaltılmasının ateroskleroz gelişiminde çok önemli bir rol oynadığı tıbbi olarak kanıtlanmış bir gerçektir. İnflamasyonun azaltılması, koroner arter hastalığı (KAH) için kabul görmüş bir tedavi stratejisidir. Bu çalışmanın amacı, yaygın olarak kullanılan bir anti-inflamatuar ajan olan kolşisinin inflamatuar belirteçler üzerindeki etkisini göstermektir.

Yöntemler: Çalışma, tıbbi kayıtları incelenen KAH tanısı konmuş 122 hastadan oluşan bir örneklemden oluşmaktadır. Nötrofil/lenfosit, monosit/lenfosit ve trombosit/lenfosit oranları gibi inflamasyon belirteçleri hesaplanmış ve kaydedilmiştir. Kolşisin uygulanan altı aylık bir dönemin ardından, değerler yeniden hesaplanmıştır.

Sonuçlar: Tedaviye başlamadan önce nötrofil/lenfosit oranı (NLR) (3,99 ± 4,72), monosit/lenfosit oranı (MLR) (0,41 ± 0,42) ve trombosit/lenfosit oranı (PLR) (129,6 ± 74,1), tedaviden sonra NLR'de (3,5±3), MLR'de (0,38±0,28) ve trombosit/lenfosit oranında (PLR) (122,6±65) azalma gözlemlendi. Tedavi sonrası dönemde, tedavi öncesi seviyelere kıyasla nötrofil-lenfosit oranında (NLR), monosit-lenfosit oranında (MLR) ve trombosit-lenfosit oranında (PLR) istatistiksel olarak anlamlı düşüşler gözlemlendi (tüm değerler için p<0,001).

Sonuç: Bu çalışmada, koroner arter hastalığı olan hastalarda kolşisin tedavisine başlanmasının ardından NLR, MLR ve PLR düzeylerinde anlamlı bir düşüş gözlemlenmiştir.

Anahtar kelimeler: kolşisin - nötrofil lenfosit oranı - monosit lenfosit oranı - platelet lenfosit oranı - koroner arter hastalıpğı.

INTRODUCTION

Inflammation and the development of large atherosclerotic plaques are considered the primary mechanisms for the development and progression of coronary artery disease (CAD)[1]. Colchicine is a well-known anti-inflammatory drug, but it is primarily considered a treatment for gout[2]. Colchicine, a substance with microtubule-inhibiting and anti-inflammatory properties, has been shown to reduce vascular inflammation and plaque progression by suppressing monocyte and neutrophil activation. This, in turn, prevents the recruitment of these cells to atherosclerotic plaques[3].

In recent years, scientific literature has demonstrated an escalating prevalence of research focusing on the neutrophil/lymphocyte ratio (NLR) as a potential diagnostic and prognostic marker for cardiovascular diseases[4]. This ratio is calculated by dividing the absolute value of neutrophils by the absolute value of lymphocytes.

The monocyte/lymphocyte ratio (MLR) is defined as the absolute number of monocytes divided by the number of lymphocytes. As postulated by a considerable number of researchers, the ratio in question is reflective of the contrasting, yet complementary, balance between non-specific inflammatory procedures and adaptive immune responses in the body. Consequently, the use of MLR as a competitive biomarker has been proposed, with the potential to compare it to other biomarkers such as NLR or C-reactive protein (CRP)[5].

It is becoming increasingly evident that inflammation, thrombosis and inflammatory markers have a causative role in the pathogenesis of cardiovascular diseases[6]. It is widely accepted that platelets and leukocytes are the primary inflammatory mediators. The division of platelets into lymphocytes gives rise to a novel inflammatory index, known as the platelet/lymphocyte ratio (PLR)[7]. It has been reported that this index has practical benefits in

the diagnosis and treatment of a wide range of conditions, including kidney disorders, cardiovascular diseases, and cancers[8].

The objective of the present study was to demonstrate the impact of colchicine treatment on inflammation markers in patients diagnosed with coronary artery disease (CAD).

METHODS

The study comprised 122 patients diagnosed with CAD who were treated at the Kdz Echomar Hospital between 2020 and 2025, following a comprehensive review of their medical records. Ethical approval for the study was obtained from the ethics committee of Zonguldak Bülent Ecevit University (meeting date:january 28 2026, number:2026/02). Patients who were pregnant, had acute coronary syndrome, fever or infection of unknown origin that would affect inflammation values, had arrhythmias, renal failure, chronic liver disease, familial hyperlipidemia, or malignancy were excluded from the study.

The administration of colchicine was exclusively oral, with a dosage of 0.5 mg administered once daily.

The present study incorporated all cases with complete data regarding age, sex, and white blood cell (WBC) counts, including determination of neutrophil and lymphocyte counts. The measurement and subsequent expression of all white blood cell counts, including neutrophil, monocyte, platelet, and lymphocyte counts, were conducted and articulated as 1000 cells/mm3. NLR was calculated by dividing the absolute value of neutrophils by the absolute value of lymphocytes. MLR was calculated by dividing the absolute value of monocytes by the absolute value of lymphocytes. PLR was calculated by dividing the absolute value of platelets by the absolute value of lymphocytes.

All participants in the study underwent electrocardiography, their medications were reviewed, and their physical examinations were examined. Echocardiography was performed on each individual to assess mechanical myocardial function. The echocardiographic examination was conducted following a minimum of 15 minutes of rest in the left lateral position (2-dimensional, M-mode, Doppler echocardiography) utilising a Vivid E9 device with an X5-1 transthoracic probe (Vivid 9 Pro, General Electric Medical Systems, Milwaukee, Wisconsin) via parasternal and apical windows. Echocardiography was performed on each participant according to the standard images and techniques outlined in the European Society of Echocardiography (AEC) guidelines.

A retrospective analysis of patient records was conducted for subjects who had undergone coronary angiography a minimum of three months prior to the study and had not undergone any surgical procedures within the preceding three months. Patients who had been prescribed colchicine for the purpose of reducing inflammation during outpatient follow-up and had used it for a period of at least six months were included in the present study. Participants who had previously undergone surgical procedures during their drug use were excluded from the study. It is noteworthy that none of the participants in the study had undergone a second coronary intervention.

Statistical Methods

The statistical analysis of the data obtained in the study was performed using IBM Statistical Package for the Social Sciences (SPSS) for MacOS, version 30.0 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as numbers and percentages [n(%)], while continuous variables were presented as mean ± standard deviation or median (interquartile range, IQR) according to their distribution characteristics. The distribution of continuous variables was evaluated using the Kolmogorov–Smirnov test. Furthermore, skewness–kurtosis values within the ±2 range were considered as

criteria supporting the assumption of normal distribution. The factors affecting the changes in the NLR, MLR and PLR ratios were evaluated by applying multiple linear regression analyses. In the regression models, the difference between post-treatment and pre-treatment measurements (Δ=post-pre) was designated as the dependent variable. The following independent variables were included in the models: age, gender, pre-treatment value of the relevant parameter, and concomitant cardiovascular treatments (ARNI, SGLT2 inhibitors, beta-blockers, diuretics, and mineralocorticoid receptor antagonists). It was hypothesised that antithrombotic therapies (acetylsalicylic acid, P2Y12 receptor inhibitors, and non-vitamin K oral anticoagulants) would be excluded from multivariate analyses due to the prediction that their contribution to the model would be limited. The adequacy of the model fit was evaluated employing the coefficient of determination (R^2), the adjusted R^2, and the F-test. The regression results were reported as the coefficient (B), the standard error, and the p-value. Multicollinearity was assessed using the variance inflation factor (VIF), with a VIF > 5 value being considered indicative of multicollinearity. In the course of all statistical analyses, a two-sided $p < 0.05$ value was considered to be statistically significant.

RESULTS

The demographic and clinical characteristics of patients are outlined in Table 1. The study comprised a total of 122 patients. The demographic composition of the patient population is noteworthy, with 42.6% of subjects being male and 57.4% female. The mean age of the sample was found to be 61 years, with a corresponding standard deviations of ±14 years. The prevalence of smoking was documented in 39.3% (48 individuals). With regard to the presence of comorbidities, 39.3% (48 individuals) of the patients exhibited coronary artery disease, 47.5% (58 individuals) demonstrated hypertension, and 32.8% (40 individuals) presented with diabetes mellitus.

Treatment characteristics were evaluated, revealing that 41.8% (51 people) of patients were using angiotensin receptor blockers (ARB)/angiotensin-converting enzyme (ACE) inhibitors, 58.2% (71 people) were using angiotensin receptor neprilysin inhibitors (ARNI), 78.7% (96 people) were using sodium-glucose cotransporter 2 (SGLT2) inhibitors, 74.6% (91 people) were using beta-blockers, 31.1% (38 people) were using non-vitamin K oral anticoagulants (NOACs), 65.6% were using acetylsalicylic acid, 41.8% (51 people) were using P2Y12 receptor inhibitors, 65.6% (80 people) were using diuretics, and 58.2% (71 people) were using spironolactone.

Table I: Distribution of demographic and clinical findings of patients.

Variables (N=122)	n (%)	Mean ± SD	Median (IQR)
Sex			
Male	52 (42.6)		
Female	70 (57.4)		
Age (year)		61±14	60 (51-71)
Smoxing	48 (39.3)		
Additional illnesses			
HT	58 (47.5)		
DM	40 (32.8)		
CAD	48 (39.3)		
laboratory			
LDL		121.3±30.1	126.1 (103-142.4)
HDL		35.8±10.8	33 (29-40.4)
Uric acid		8.6±2.9	9.5 (5.6-11.4)
Glucose		121.8±54	100 (90-134)
ALT		30.8±35.8	21 (15-38)
AST		40.4±37.7	29 (19-41.4)
Creatinine		1.1±0.8	0.9 (0.8-1.2)
Potassium		4.4±0.7	4.4 (4-4.7)
Sodium		139.5±4	139 (137-142)
Chlorine		104.1±5.3	105 (100-108)
PLT		225.3±68.1	218 (186-261)
HGB		13.5±1.6	13.6 (12.5-14,5)
EF		35.9±11.3	35 (25-45)
Treatments			
ARB/ACEi	51 (41.8)		
ARNİ	71 (58.2)		
SGLT2İ	96 (78.7)		
BBLOc	91 (74.6)		
NOAK	38 (31.1)		
ASA	80 (65.6)		
P2Y12	51 (41.8)		
Diuretic (furosemide)	80 (65.6)		
Spironolactone	71 (58.2)		

The data are presented in tabular form as numbers and percentages [n(%)] for categorical variables, mean ± standard deviation for continuous variables showing a normal distribution, and median (interquartile range, IQR) for continuous variables not showing a normal distribution.CAD: coronary artery disease, HT: hypertension, DM: Diabetes mellitus, LDL: Low Density Lipoprotein, HDL: High Density Lipoprotein; EF: Ejection fraction, ARNİ:angiotensin receptor neprilysin inhibitör(Sacubitril/valsartan), SGLT-2, sodium-glucose cotransporter, ACEİ, Angiotensin-converting enzyme inhibitors; ARB, Angiotensin receptor blocker; Betbloc, Beta blocker; NOAK, Non-Vitamin K Antagonist Oral Anticoagulant; ASA, acetylsalicylic acid

The distribution of inflammatory indices and electrocardiographic findings of patients before and after treatment is shown in Table 2. In the post-treatment period, a statistically significant decrease was observed in NLR, MLR, and PLR compared to pre-treatment levels (p<0.001 for all).

Table II: Inflammatory indices of patients before and after treatment.

	before treatment	after treatment		p-value
NLR			**<0,001**	**r=0,480**
Median (IQR)	**2,89 (1,83-4,54)**	**2,7 (1,8-4)**		
MLR			**<0,001**	**r=0,420**
Median (IQR)	**0,29 (0,23-0,47)**	**0,28 (0,21-0,44)**		
PLR			**<0,001**	**r=0,400**
Median (IQR)	**117,8 (86,2-146)**	**106,6 (87,5-138,3)**		

Continuous variables that do not exhibit a normal distribution are presented as the median (interquartile range, IQR), while continuous variables that exhibit a normal distribution are presented as the mean ± standard deviation. The paired t-test was employed to compare paired

measurements that exhibited a normal distribution, whilst the Wilcoxon signed-rank test was utilised to compare paired measurements that did not demonstrate a normal distribution. NLR:neutrophil/lymphocyte ratio, MLR: monocyte/lymphocyte ratio, PLR: platelet/lymphocyte ratio

The factors affecting the change in neutrophil/lymphocyte ratio (ΔNLR) in patients are presented in Table 3. The model created according to multivariate linear regression analysis was found to be statistically significant (F=43.957; p<0.001) and explains approximately 74% of the ΔNLR variance (adjusted R^2=0.740). The analysis revealed that only the baseline NLR value (preNLR) was significantly associated with the change in NLR (B=−0.406; p<0.001). Consequently, a more pronounced decrease in NLR was identified in patients with higher baseline NLR levels during the follow-up period. No independent significant association was identified between age, gender, and concomitant cardiovascular treatments (ARNI, SGLT2 inhibitors, beta-blockers, diuretics, and spironolactone) and ΔNLR (p>0.05).

Table III: Factors affecting the Neutrophil/Lymphocyte ratio in patients after treatment.

	B	Standard error	t	p-değeri
Age	0.007	0.008	0.844	0.400
Sex (male=1, female=2)	0.089	0.214	0.414	0.680
ARNI	-0.076	0.206	-0.368	0.714
SGLT2i	0.145	0.273	0.531	0.596
BBLOC	-0.250	0.264	-0.946	0.346
Diuretic (furosemide)	0.296	0.225	1.317	0.191
Spironolactone	-0.255	0.221	-1.151	0.252
Model Summary: R=0.936; R²=0.876; Adjusted R²=0.867; F=99.592; p<0.001				

The dependent variable was defined as the difference between post-treatment and pre-treatment measurements (Δ=post-pre). A negative B coefficient is indicative of factors associated with a greater decrease in the relevant parameter. ARNİ:angiotensin receptor neprilysin inhibitör(Sacubitril/valsartan), SGLT-2, sodium-glucose cotransporter, ACEİ, Angiotensin-converting enzyme inhibitors; ARB, Angiotensin receptor blocker; Bbloc, Beta blocker;

The factors affecting the change in the monocyte/lymphocyte ratio (ΔMLR) of patients are presented in Table 4. A multivariate linear regression model was found to be statistically significant (F=35.859; p<0.001) and explains approximately 70% of the ΔMLR variance (adjusted R^2=0.697). The baseline MLR (preMLR) was found to be independently and significantly associated with the change in MLR (ΔMLR) (B=−0.390; p<0.001). Consequently, patients with elevated baseline MLR levels exhibited a more substantial decline in MLR during the follow-up period. In addition, the utilisation of beta-blockers was identified as an independent predictor of ΔMLR (B=−0.067; p=0.007). This finding suggests that patients using beta-blockers experience a more significant decrease in MLR levels. No significant association was identified between age, gender, ARNI, SGLT2 inhibitors, diuretics, and spironolactone use and ΔMLR (p>0.05).

Table IV: Factors affecting the monocyte/lymphocyte ratio of patients after treatment.

	B	Standart hata	t	p-değeri
Age	0.001	0.001	1.419	0.159
Sex (male=1, female=2)	0.001	0.020	0.025	0.980
ARNI	0.004	0.019	0.235	0.814
SGLT2i	0.002	0.025	0.099	0.921
BBLOC	**-0.067**	**0.025**	**-2.728**	**0.007**
Diuretic (furosemide)	0.039	0.021	1.890	0.061
Spironolactone	-0.019	0.020	-0.917	0.361
Model Summary: R=0.938; R²=0.880; Adjusted R²=0.871; F=103.390; p<0.001				

The dependent variable was defined as the difference between post-treatment and pre-treatment measurements (Δ=post-pre). A negative B coefficient is indicative of factors associated with a greater decrease in the relevant parameter. ARNİ:angiotensin receptor neprilysin inhibitör(Sacubitril/valsartan), SGLT-2, sodium-glucose cotransporter, ACEİ, Angiotensin-converting enzyme inhibitors; ARB, Angiotensin receptor blocker; Bbloc, Beta blocker;

The factors that affect the change in platelet/lymphocyte ratio (ΔPLR) in patients are presented in Table 5. A multivariate linear regression model was found to be statistically significant (F=7.333; p<0.001) and explains approximately 30% of the ΔPLR variance (adjusted R^2=0.295). The analysis revealed that only the baseline PLR value (prePLR) was independently and significantly associated with the change in PLR (ΔPLR) (B=−0.163; p<0.001). Consequently, a more pronounced decrease in PLR was observed in patients with higher baseline PLR levels during the follow-up period. No independent significant association was identified between age, gender, and concomitant cardiovascular treatments (ARNI, SGLT2 inhibitors, beta-blockers, diuretics, and spironolactone) and ΔPLR (p > 0.05).

Table V: Factors affecting platelet/lymphocyte ratio after treatment in patients.

	B	Standart hata	t	p-değeri
Age	0.039	0.126	0.309	0.758
Sex (male=1, female=2)	4.975	3.427	1.452	0.149
ARNI	-1.815	3.317	-0.547	0.585
SGLT2i	1.467	4.350	0.337	0.737
BBLOC	-6.912	4.177	-1.655	0.101
Diuretic (furosemide)	6.520	3.606	1.808	0.073
Spironolactone	-0.229	3.524	-0.065	0.948
Model Summary: R=0.965; R²=0.931; Adjusted R²=0.926; F=190.958; p<0.001				

The dependent variable was defined as the difference between post-treatment and pre-treatment measurements (Δ=post-pre). A negative B coefficient is indicative of factors associated with a greater decrease in the relevant parameter. ARNİ:angiotensin receptor neprilysin inhibitör(Sacubitril/valsartan), SGLT-2, sodium-glucose cotransporter, ACEİ, Angiotensin-converting enzyme inhibitors; ARB, Angiotensin receptor blocker; Bbloc, Beta blocker;

DISCUSSION

For a period exceeding three decades, fundamental advancements within the domain of laboratory research have demonstrated that low-grade systemic inflammation, driven by both innate and acquired immune systems, plays a pivotal role in the development of atherosclerosis. The initiation of this process is marked by the accumulation of foam cells in otherwise unharmed arteries. This is followed by the progression of various physiological phenomena, including the formation of plaque, its subsequent rupture, the resultant occlusion, and the narrowing of the affected arteries[9]. At low doses, colchicine has been shown to inhibit microtubule growth, while at high doses it has been demonstrated to promote microtubule depolymerisation. The anti-inflammatory properties of colchicine are attributed to its ability to inhibit tubulin degradation, thereby preventing the formation of inflammasomes. This, in turn, results in a reduction in the production of pro-inflammatory cytokines, as well as an impairment in neutrophil function[10,11]. The positive effect of colchicine on coronary artery disease is a subject that has recently been the focus of intensive research. Colchicine, which is included in European guidelines with a class 2A recommendation level for the treatment of coronary artery disease, is a versatile drug that requires further study[12]. The objective of the present study was to demonstrate the impact of colchicine utilisation on essential and readily quantifiable blood test parameters in patients diagnosed with coronary artery disease.

Neutrophils and lymphocytes represent pivotal components of the immune system. Neutrophils represent a type of innate immune cell that has

the capacity for chemokine production, in addition to the production of cytokines, endothelial growth factor, and matrix metalloproteinase, thereby powering the primary phase of the immune response. Lymphocytes, a type of adaptive immune cell, are the regulatory agents that modulate this specific immune response[13]. Specifically, an increase in neutrophil count has been demonstrated to reduce lymphocyte activity[14]. In recent years, scientific literature has demonstrated an increasing prevalence of the neutrophil-lymphocyte ratio (NLR) in the diagnosis and prognosis of cardiovascular diseases. The NLR is calculated by dividing the absolute value of neutrophils by the absolute value of lymphocytes. The literature suggests that the NLR is effective in the prognosis of cardiovascular diseases[4,15].

Colchicine, a substance derived from the Theautumncrocus (Colchicum autumnale) plant, is among the most venerable pharmaceuticals, with a history of utilisation that dates back to antiquity and continues to the present day[16]. Colchicine exerts a plethora of cellular effects, including the suppression of tubulin polymerization and the subsequent dissolution of the cytoskeleton, as well as mitosis and intracellular transport activities. Colchicine has been found to accumulate in neutrophils, with the result that neutrophil activity is significantly affected. In particular, colchicine has been shown to hinder the directed migration of neutrophils to an inflammatory focus and to reduce neutrophil adhesion to inflamed endothelium. Furthermore, colchicine has been demonstrated to inhibit the adhesion of leukocytes to inflamed endothelium[17]. In light of the aforementioned data, the present study also observed a decrease in the NLR values in patients following the initiation of colchicine treatment. The anti-inflammatory effect of colchicine lends support to the hypothesis that the administration of the drug could positively impact the treatment of coronary artery disease.

Comprising three functionally heterogeneous subsets, monocytes' capacity for versatility in various environments has the potential to determine the advancement of systemic inflammation by combining inherent and derived immunity through phagocytosis, cytokine and chemokine production, antigen presentation, and lymphocyte activation[18]. It has been posited by numerous researchers that the MLR is a comparable competitive biomarker. This is due to the fact that the ratio reflects the contrasting yet compatible balance between non-specific inflammatory processes and appropriate immune responses in the body[5,19]. Recent studies have provided new evidence regarding the clinical significance of MLR in the severity, propagation and results of a variety of diseases, including atherosclerotic cardiovascular diseases, malignant neoplasias, septicemia and pulmonary tuberculosis[20-23]. In the present study, as in previous research, the MLR value was elevated; however, it was noted that the significance of this value decreased with colchicine treatment.

Inflammation, thrombosis, and inflammatory markers have been demonstrated to have a role in the pathogenesis of cardiovascular diseases[6]. It has been established that platelets are capable of interacting with two distinct cell types, namely leukocytes and endothelial cells. The function of these factors is to induce the production of inflammatory substances, which in turn result in monocyte transmigration. The role of these cells in the pathogenesis of atherosclerosis, as well as their association with thrombosis and inflammation, is a subject of ongoing research. It is evident that platelets and leukocytes function as primary mediators of inflammation. The differentiation of platelets into lymphocytes leads to a novel inflammation index, referred to here as PLR[7]. It has been reported that the index has practical benefits in

a wide range of conditions, including renal disease, cardiovascular disease, and cancer[8,24,25]. In a 2016 meta-analysis, Weng Li et al. evaluated the prognostic value of the PLR in acute coronary syndrome. The findings of the study indicated that patients exhibiting elevated PLR levels demonstrated a considerably elevated probability of experiencing unfavourable hospital and longitudinal outcomes[26]. A further study encompassing 2,230 patients diagnosed with acute coronary syndrome revealed a positive correlation between PLR and the GRACE risk score. The integration of PLR and GRACE risk score yielded a more efficacious approach in predicting cardiovascular disease (CVD) events[27]. In summary, it has been hypothesised that PLR may serve as an indicator of unfavourable prognoses in cardiovascular diseases[25,28,29]. However, the findings of contemporary research are contentious, with certain studies failing to establish a correlation between PLR and adverse outcomes in patients with acute myocardial infarction[30]. In the present study, we also examined PLR levels in light of this information and found that PLR levels decreased with anti-inflammatory treatment in our patients.

In conclusion, the reduction of inflammation, a pivotal step in the atherosclerotic process, may have a role in the reduction or prevention of atherosclerosis. In the present study, a significant decrease in NLT, MLR, and PLR values was observed following the initiation of colchicine treatment. Given the established literature consensus regarding these values as inflammatory indicators and, consequently, the potential for atherosclerosis, the observed reduction in NLR, MLR, and PLR by colchicine can be interpreted as contributing to the hypothesis that it may have a regulatory effect on atherosclerosis. In addition, the present study found that beta-blocker use was associated with MLR, but not with NLR and PLR. Further, more extensive studies incorporating a greater number of patients are required on this topic.

Limitation

The present study is limited in its reliability due to the relatively small number of participants, and a longer-term follow-up is required to provide more robust results. Whilst the absence of a control group without colchicine can be regarded as a limitation, the objective was to examine the effect of colchicine, a new addition to a group already receiving treatment for coronary artery disease. This limitation was attempted to be overcome by conducting a statistical comparison of the effects of other treatments. The continuation of treatment with a solitary, minimal dose hinders our ability to respond to inquiries such as whether escalation of dosage is imperative on the basis of bioavailability or patient mass. In order to ascertain the appropriate dose and effect of colchicine, further research is required in the form of larger-scale studies. Moreover, the duration of the study is inadequate in demonstrating the impact on recurrent coronary artery events. The patients included in the study were generally those receiving intensive treatment and had multi-vessel disease, and colchicine was added to their treatment, which compromises the homogeneity of the study. The absence of a control group is also a limitation of the present study.

Ethics Committee Approval: Ethical approval for the study was obtained from the ethics committee of Zonguldak Bülent Ecevit University (meeting date:january 28 2026, number:2026/02).

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