narra j

Original Article

Impact of colchicine on hs-CRP, neutrophil levels, neutrophil-to-lymphocyte ratio and major adverse cardiac events (MACEs) in Thai patients with acute coronary syndrome undergoing percutaneous coronary intervention

Wittawat Wattanasiriporn[1,2*], Paruj Rattanasidha[1], Haris Munirwan[3] and Chantisa Arayangkoon[4]

[1]Division of Cardiology, Department of Internal Medicine, Rajavithi Hospital, Bangkok, Thailand; [2]Division of Cardiology, Department of Internal Medicine, College of Medicine, Rangsit University, Bangkok, Thailand; [3]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia; [4]Division of Nephrology, Department of Medicine, Rajavithi Hospital, College of medicine, Rangsit University, Bangkok, Thailand

*Corresponding author: bankwit@gmail.com

Abstract

Acute coronary syndrome (ACS) is strongly associated with inflammation, which contributes to plaque instability, thrombosis, and adverse cardiovascular outcomes. High-sensitivity C-reactive protein (hs-CRP), neutrophil count, and neutrophil-to-lymphocyte ratio (NLR) are inflammatory markers that have been associated with poor prognosis in patients with ACS. Colchicine has emerged as a potential adjunctive anti-inflammatory therapy in cardiovascular disease. This study aimed to evaluate the effects of colchicine on inflammatory markers and clinical outcomes in Thai patients with ACS undergoing percutaneous coronary intervention (PCI). This single-center, retro-prospective observational cohort study included adult patients with ACS who underwent PCI at Rajavithi Hospital, Bangkok, Thailand, in 2024. Patients were classified into colchicine and non-colchicine groups based on treatment exposure after PCI. Hs-CRP, neutrophil count, and NLR were assessed at baseline, 1 month, and 3 months. Major adverse cardiac events (MACE) during follow-up were also recorded. A total of 56 patients were included, comprising 38 in the colchicine group and 18 in the non-colchicine group. Compared with the non-colchicine group, the colchicine group showed significantly greater reductions from baseline to 3 months in hs-CRP levels (2.29±3.37 vs 0.45±1.03; p=0.044), neutrophil count (21.86±10.62 vs 4.13±12.92; p=0.001), and NLR (2.98±2.93 vs 1.68±3.60; p=0.025). No significant differences in MACE were observed between the two groups. This study highlighted that colchicine was associated with greater reductions in inflammatory markers during the early post-PCI period, although no significant difference in short-term clinical outcomes was identified. Larger prospective studies are needed to confirm these findings.

Keywords: Colchicine, acute coronary syndrome, acute myocardial infarction, percutaneous coronary intervention, C-reactive protein

Introduction

Acute coronary syndrome (ACS), encompassing conditions such as unstable angina and myocardial infarction, represents a leading cause of morbidity and mortality worldwide [1].

https://narraj.org/

Received: March 7, 2026 - Accepted: May 1, 2026 - Published online: May 11, 2026

Characterized by the abrupt onset of chest pain and other cardiovascular symptoms, ACS is primarily driven by the rupture of atherosclerotic plaques, leading to thrombosis and subsequent ischemia [1]. Inflammation plays a pivotal role in the pathophysiology of ACS, contributing to both plaque instability and the thrombotic process [2-5]. Elevated levels of high sensitivity C-reactive protein (hs-CRP), a well-established biomarker of systemic inflammation, alongside neutrophil-lymphocyte ratio (NLR) have been consistently linked with adverse cardiovascular outcomes in patients with ACS [6-8]. Higher hs-CRP levels and higher NLR level are associated with an increased risk of recurrent events and poor prognosis [6-8], highlighting the need for effective strategies to manage inflammation in this high-risk population.

Colchicine, an ancient therapeutic agent derived from the *Colchicum autumnale* plant, has gained renewed interest due to its potent anti-inflammatory properties. Traditionally utilized for the treatment of gout, its applications have been expanded to various inflammatory diseases [9]. Colchicine exerts its effects by inhibiting the NOD-like receptor pyrin domain-containing protein 3 (NLRP3) inflammasome, thereby reducing the production of pro-inflammatory cytokines and subsequently lowering hs-CRP levels [10]. The anti-inflammatory mechanisms of colchicine suggest its potential role as a novel therapeutic option in the management of coronary artery disease, where inflammation is a key driver of disease progression and complications [11, 12].

As of the most recent updates, major cardiovascular guidelines, including those from the American College of Cardiology (ACC) and the American Heart Association (AHA), emphasize the importance of targeting inflammation in cardiovascular disease management. While colchicine is not universally recommended for all patients, it is considered a viable option for individuals with a history of myocardial infarction [13], particularly those at high risk for recurrent events [14]. The guidelines suggest that colchicine can be used in conjunction with standard therapy, especially in patients who may benefit from additional anti-inflammatory treatment [14]. Furthermore, the European Society of Cardiology (ESC) guidelines also recognize the role of inflammation in atherogenesis and suggest that therapies like colchicine could be considered in specific patient populations, particularly those with recurrent events or elevated inflammatory markers [15]. However, clinicians are advised to weigh the benefits against potential side effects, such as gastrointestinal disturbances and interactions with other medications. The evolving role of colchicine in cardiovascular care reflects a broader trend toward personalized medicine, where treatment strategies are tailored to individual patient risk profiles and inflammatory states [16].

Despite increasing evidence supporting the anti-inflammatory effects of colchicine in cardiovascular disease, data from Asian populations, particularly Thai patients with ACS undergoing PCI, remain limited. In addition, real-world observational evidence on the effects of colchicine on inflammatory biomarkers during the early post-PCI period is scarce, and its short-term impact on clinical outcomes in routine practice remains unclear. Therefore, the aim of this study was to evaluate the effects of colchicine on inflammatory markers and clinical outcomes in Thai patients with ACS undergoing PCI. Specifically, hs-CRP levels were measured at baseline, 1 month, and 3 months after treatment to assess the relationship between colchicine use and inflammatory response in this population. The findings of this study may provide further insight into the potential role of colchicine in reducing inflammation and improving clinical management in patients with ACS after PCI.

Methods

Study design and setting

This single-center, retro-prospective observational cohort study was conducted at Rajavithi Hospital, Bangkok, Thailand, in 2024. The study evaluated the effects of colchicine on inflammatory markers and clinical outcomes among patients with ACS who underwent PCI during the early post-PCI period. The study was conducted after approved by the Institutional Review Board of Rajavithi Hospital.

Patients and criteria

The study included patients aged 18 years and older who were diagnosed with ACS, including ST-elevation myocardial infarction, non-ST-elevation myocardial infarction, or unstable angina, and

underwent PCI within 45 days of diagnosis. The 45-day window was selected to capture patients during the early post-ACS inflammatory phase while reflecting real-world variation in the timing of PCI. Patients were excluded if they had contraindications to colchicine, were pregnant or lactating, had severe renal or hepatic impairment, had received long-term colchicine therapy for other indications, or were scheduled for coronary artery bypass graft surgery after coronary angiography.

Sample and sampling

Patients were retrospectively identified from hospital medical records and included using a consecutive sampling approach based on eligibility criteria. They were then classified into two exposure groups according to post-PCI treatment: those who received colchicine 0.6 mg once daily and those who did not receive colchicine. Due to the retrospective nature of the study and the limited number of eligible patients, no formal sample size calculation was performed; therefore, this study should be considered exploratory.

Data collection

Baseline data were obtained from medical records, including demographic characteristics, ACS diagnosis, comorbidities, coronary angiography findings, PCI-related data, and relevant laboratory results. Inflammatory markers were assessed using blood samples collected within 3 days after admission and during follow-up at 1 month and 3 months. Clinical outcomes during follow-up, including recurrent cardiovascular events, hospitalizations, mortality, and treatment-related complications, were also recorded. Missing data were handled using complete-case analysis, and the number of patients included in each analysis is presented in the tables. Missing values were mainly due to incomplete laboratory measurements or loss to follow-up.

Study variables

The main independent variable was colchicine exposure after PCI. The primary outcome variables were changes in inflammatory markers, including hs-CRP, neutrophil count, absolute neutrophil count, white blood cell count, and NLR, measured at baseline, 1 month, and 3 months. Clinical outcomes assessed during follow-up included major adverse cardiac events, such as stroke, repeat revascularization, complications, and death. Other variables collected included age, sex, ACS subtype, comorbidities, statin use, coronary angiography findings, troponin I, and left ventricular ejection fraction.

Statistical analysis

Descriptive statistics are presented as frequency and percentage for categorical variables, and as mean±SD or median with interquartile range for continuous variables, as appropriate. Differences between the colchicine and non-colchicine groups were analyzed using the Chi-square test or Fisher's exact test for categorical variables, and the independent t-test or Mann-Whitney U test for continuous variables, depending on data distribution. Multivariable regression analysis was performed to adjust for potential confounders, including age, sex, baseline hs-CRP, and coronary angiography findings. A two-sided $p<0.05$ was considered statistically significant. No formal correction for multiple comparisons was applied; therefore, the findings should be interpreted as exploratory. Data were analyzed using SPSS Statistics version 26 (IBM, Armonk, NY, USA).

Results

Baseline characteristics of the patients

A total of 56 patients with ACS undergoing coronary angiography were included, of whom 38 received colchicine and 18 did not. Their baseline demographic and clinical characteristics are presented in **Table 1**. The sex and age distributions were comparable between the two groups, with no significant differences. Male patients predominated in both groups, and the mean age was 58.63±12.14 years in the colchicine group and 61.89±13.84 years in the non-colchicine group. The distribution of ACS diagnoses, including non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI), was also similar between the groups. Comorbidities, including hypertension and diabetes mellitus, were common but did not differ

significantly. A significant difference was observed in coronary angiography findings, with single-vessel disease being more frequent in the non-colchicine group. LVEF data were unavailable for three patients because of incomplete echocardiographic records (**Table 1**).

Effect of colchicine on inflammatory markers (hs-CRP, neutrophil and NLR) in patients with ACS undergoing PCI

The comparison of changes in hs-CRP levels between baseline and 3 months showed a significant difference between the two groups (**Figure 1A**). The colchicine group had a greater reduction in hs-CRP levels than the non-colchicine group (2.29±3.37 vs 0.45±1.03, p=0.044) (**Table 2**). These findings indicate that hs-CRP levels decreased more markedly in patients who received colchicine during the 3-month follow-up period, suggesting a greater reduction in inflammatory response.

Similarly, the mean change in neutrophil count from baseline to 3 months differed significantly between the two groups (**Figure 1B**). The colchicine group had a greater reduction in neutrophil count than the non-colchicine group (21.86±10.62 vs 4.13±12.92, p=0.001), indicating a more pronounced decrease over the 3-month follow-up period (**Table 2**). A significant difference was also observed in the change in NLR from baseline to 3 months (**Figure 1C**). The reduction in NLR was greater in the colchicine group than in the non-colchicine group (2.98±2.93 vs 1.68±3.60, p=0.025), suggesting a stronger anti-inflammatory effect in patients who received colchicine (**Table 2**).

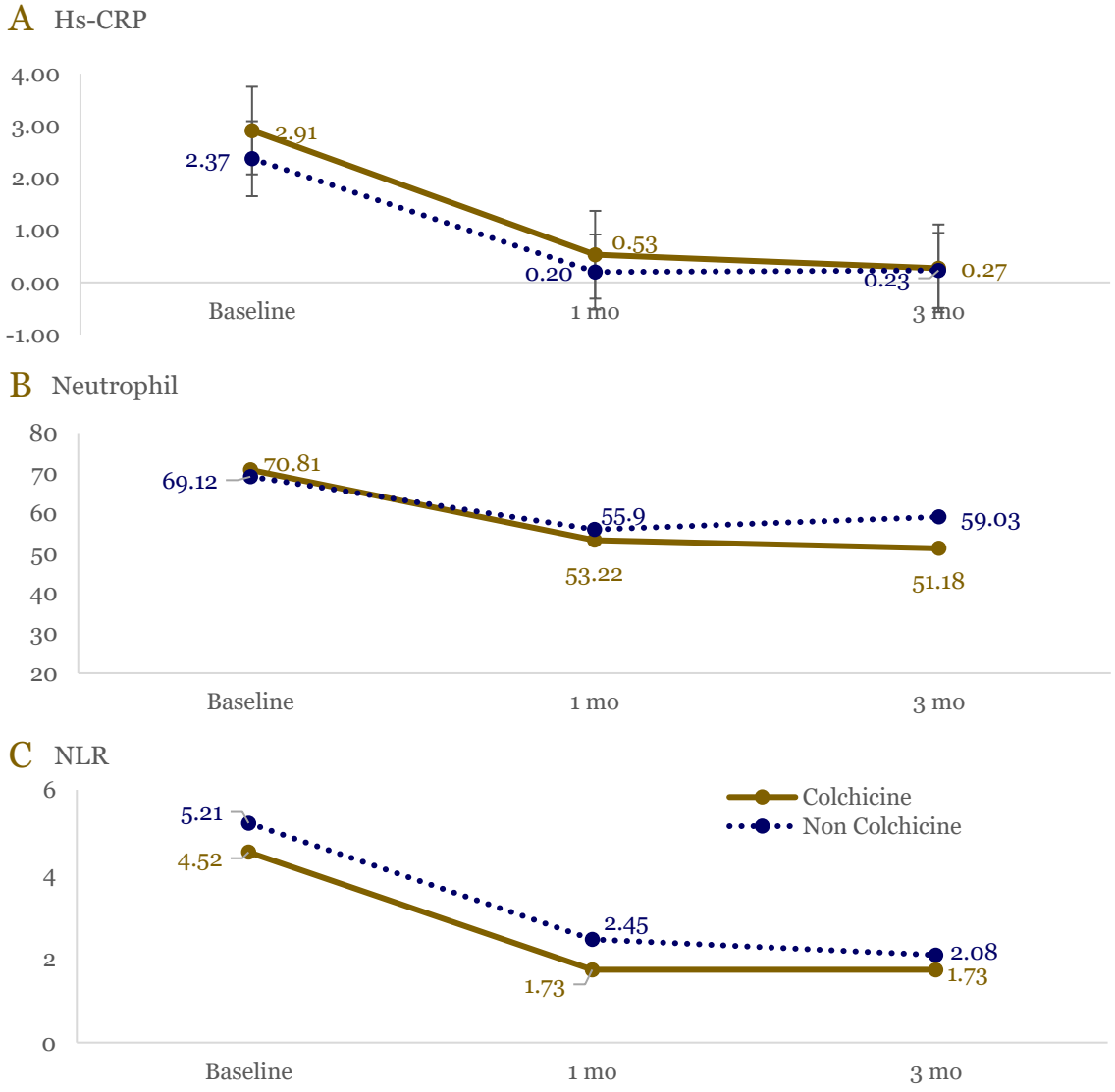

Figure 1. Effects of colchicine on inflammatory markers in patients with acute coronary syndrome undergoing percutaneous coronary intervention (PCI). (A) Effects of colchicine on high-sensitivity C-reactive (hs-CRP) protein levels. (B) Effects of colchicine on neutrophil count. (C) Effects of colchicine on neutrophil-to-lymphocyte ratio (NLR).

Table 1. Demographic and clinical characteristics of patients (n=56)

Characteristics	Total (n=56)		Colchicine (n=38)		Non-colchicine (n=18)		*p*-value
	n	%	n	%	n	%	
Gender							0.305 [a]
Male	45	80.4	32	84.2	13	72.2	
Female	11	19.6	6	15.8	5	27.8	
Age (years)							0.211 [a]
<50	12	21.4	8	21.1	4	22.2	
50–59	16	28.6	14	36.8	2	11.1	
60–69	15	26.8	9	23.7	6	33.3	
≥70	13	23.2	7	18.4	6	33.3	
Mean±SD	59.68±12.68		58.63±12.14		61.89±13.84		0.374 [b]
Diagnosis							0.175 [a]
NSTEMI	18	32.1	10	26.3	8	44.4	
STEMI	38	67.9	28	73.7	10	55.6	
Comorbidity							0.418 [a]
Yes	33	58.9	21	55.3	12	66.7	
No	23	41.1	17	44.7	6	33.3	
Hypertension	28	50.0	16	42.1	12	66.7	0.086 [a]
Diabetes	17	30.4	11	28.9	6	33.3	0.739 [a]
Prior stroke	4	7.1	2	5.3	2	11.1	0.587 [a]
Hearth failure	2	3.6	1	2.6	1	5.6	0.544 [a]
Other	5	8.9	3	7.9	2	11.1	0.652 [a]
Smoke	19	33.9	14	36.8	5	27.8	0.560 [a]
Statin							1.000 [a]
Atorvastatin	55	98.2	37	97.4	18	100	
Rosuvastatin	1	1.8	1	2.6	0	0	
CAG result (n=54)							0.014 [a*]
SVD	17	31.5	9	24.3	8	47.1	
DVD	18	33.3	17	45.9	1	5.9	
TVD	19	35.2	11	29.7	8	47.1	
Stent (n=54)							1.000 [a]
DES	52	96.3	35	94.6	17	100	
POBA	2	3.7	2	5.4%	0	0	
Troponin I (n=53), mean±SD	14,329.01±44,520.19		17,159.75±53,208.36		8,824.79±18,758.90		0.895 [b]
LVEF (n=53)							0.416 [a]
<40%	13	24.5	10	28.6	3	16.7	
40–49%	10	18.9	5	14.3	5	27.8	
≥50%	30	56.6	20	57.1	10	55.6	
Mean±SD	49.52±11.54		48.52±11.55		51.46±11.59		0.385 [b]

CAG: coronary angiography; DVD: double-vessel disease; LVEF: left ventricular ejection fraction; NSTEMI: non-ST-elevation myocardial infarction; STEMI: ST-elevation myocardial infarction; SVD: single-vessel disease; TVD: triple-vessel disease.

[a] Analyzed with Chi-square test or Fisher's exact test

[b] Analyzed with independent t-test or Mann-Whitney U-test

* Statistically significant at $p<0.05$

Table 2. Comparison of laboratory biomarkers between the colchicine and non-colchicine groups at baseline, 1 month, and 3 months

Biomarkers	Colchicine		Non-colchicine		Difference		*p*-value [a]
	n	Mean±SD	n	Mean±SD	Mean	95%CI	
High-sensitivity C-reactive protein							
0 month	37	2.91±4.25	17	2.37±4.28	0.540	-1.964–3.043	0.075
1 month	25	0.53±1.10	9	0.20±0.29	0.329	-0.434–1.092	0.433
3 months	23	0.27±0.84	8	0.23±0.35	0.045	-0.588–0.678	0.982
Diff 0–3	22	2.29±3.37	8	0.45±1.03	1.839	0.186–3.491	0.044*
% change 0–3	22	72.41±62.99	8	23.60±105.32	48.804	15.322–112.930	0.039*
White blood cell count							
0 month	37	10,357.57±3,623.46	17	10,239.41±3,658.77	118.156	-2018.679–2254.990	0.912
1 month	17	6,718.24±1,898.71	9	7,858.89±1,647.85	-1140.654	-2688.211–406.904	0.141
3 months	22	6,986.82±2,388.10	8	7,228.75±2,110.39	-241.932	-2205.486–1721.622	0.708
Diff 0–3	22	3,258.64±3,905.95	8	2,268.75±2,502.50	989.886	-2060.293–4040.066	0.512
% change 0–3	22	25.58±33.20	8	17.30±26.56	8.278	-18.509–35.064	0.532
Neutrophil							
0 month	37	70.81±12.13	17	69.12±13.26	1.693	-5.648–9.034	0.645
1 month	17	53.22±8.50	9	55.90±16.66	-2.676	-12.769–7.416	0.589
3 months	22	51.18±9.98	8	59.03±8.68	-7.848	-16.025–0.329	0.059
Diff 0–3	22	21.86±10.62	8	4.13±12.92	17.739	8.236–27.241	0.001*
% change 0–3	22	29.35±12.81	8	4.00±20.11	25.353	12.690–38.016	<0.001*
Absolute neutrophil count							
0 month	37	7,549.80±3,403.96	17	7,343.26±3,759.94	206.533	-1861.494–2274.560	0.842
1 month	17	3,592.84±1,253.89	9	4,402.32±1,646.60	-809.471	-1998.140–379.198	0.173
3 months	22	3,638.19±1,687.83	8	4,303.04±1,426.73	-664.851	-2040.384–710.683	0.174
Diff 0–3	22	3,992.88±3,264.25	8	1,932.52±2,334.27	2060.360	-526.141–4646.862	0.114
% change 0–3	22	46.35±27.47	8	19.07±38.83	27.273	1.306–53.241	0.031*
Neutrophil-to-lymphocyte ratio							
0 month	37	4.52±3.02	18	5.21±5.54	-0.694	-3.593–2.205	0.524
1 month	17	1.73±0.48	11	2.45±1.20	-0.717	-1.546–0.111	0.041*
3 months	24	1.73±0.88	12	2.08±0.71	-0.345	-0.941–0.250	0.247
Diff 0–3	24	2.98±2.93	12	1.68±3.60	1.302	-0.970–3.574	0.025*
% change 0–3	24	49.87±46.25	12	11.06±56.74	38.804	2.963–74.646	0.011*

[a] Analyzed using independent t-test or Mann-Whitney U-test
* Statistically significant at $p<0.05$

Effect of colchicine on major adverse cardiac event (MACE) incidence in patients with ACS undergoing PCI

MACEs were uncommon in both groups during follow-up. Overall, stroke and death each occurred in one patient (1.8%), while repeat revascularization and complications each occurred in two patients (3.6%). No statistically significant differences were observed between the colchicine and non-colchicine groups for any of the assessed events, suggesting comparable short-term clinical outcomes between the two groups (**Table 3**).

Table 3. Comparison of major adverse cardiac events (MACE) between the colchicine and non-colchicine groups of patients with ACS undergoing PCI (n=56)

Clinical outcomes	Total (n=56)		Colchicine (n=38)		non-colchicine (n=18)		*p*-value
	n	%	n	%	n	%	
Stroke	1	1.8	0	0	1	5.6	0.321
Re-vascular	2	3.6	1	2.6	1	5.6	0.544
Complication	2	3.6	2	5.3	0	0	1.000
Death	1	1.8	0	0	1	5.6	0.321

[a] Analyzed using Fisher's exact test

Discussion

The present study evaluated the effects of colchicine on inflammatory markers and clinical outcomes in Thai patients with ACS undergoing PCI. The findings showed that patients who received colchicine had greater reductions in hs-CRP levels, neutrophil count, and NLR over the 3-month follow-up period than those who did not receive colchicine. However, no significant differences were observed in major adverse cardiac events between the two groups. These findings suggest that colchicine may have a beneficial anti-inflammatory effect in the early post-PCI period, although its short-term effect on clinical outcomes remains uncertain.

Inflammation has a central role in the pathophysiology of ACS, contributing to plaque instability, thrombus formation, and recurrent ischemic events. Among the inflammatory biomarkers assessed in the present study, hs-CRP is one of the most widely used indicators of systemic inflammation and has been consistently associated with adverse cardiovascular outcomes. In a previous study, CRP levels in patients with acute myocardial infarction decreased from a median of 1.89 mg/L during hospitalization to 1.24 mg/L at 1 month after discharge [17]. In the present study, the reduction in hs-CRP from baseline to 3 months was significantly greater in the colchicine group than in the non-colchicine group. This finding suggests that colchicine may contribute to a more pronounced attenuation of systemic inflammation after ACS and PCI. This result is in line with previous studies that have shown anti-inflammatory effects of colchicine in cardiovascular disease [9,11-13].

The biological plausibility of this finding is supported by the known mechanism of colchicine, which suppresses inflammatory activity through inhibition of microtubule polymerization and modulation of inflammasome-mediated pathways [18,19]. By binding to tubulin, colchicine disrupts cytoskeletal functions that are essential for neutrophil chemotaxis, adhesion, and activation, thereby attenuating leukocyte-mediated inflammatory responses [18]. In addition, colchicine has been shown to suppress NLRP3 inflammasome activation and downstream caspase-1 signaling in patients with ACS, providing a mechanistic basis for its anti-inflammatory effect in the setting of myocardial ischemia [19]. Clinical studies have also shown that low-dose colchicine can reduce inflammatory biomarkers, including hs-CRP, in patients with coronary artery disease and after myocardial infarction [20,21]. Therefore, the greater reduction in hs-CRP observed in the colchicine group in the present study may reflect more effective suppression of residual inflammatory activity during recovery after myocardial ischemia and PCI.

In addition to hs-CRP, the present study also found a significantly greater reduction in neutrophil count in patients who received colchicine. Neutrophils are among the earliest inflammatory cells recruited to sites of vascular injury and myocardial damage. They contribute to endothelial dysfunction, oxidative stress, protease release, and amplification of the inflammatory response, which may exacerbate tissue injury and promote atherosclerotic progression [22,23]. Therefore, the greater decline in neutrophil count in the colchicine group

may indicate that colchicine helps suppress the acute inflammatory response following ACS. This observation further supports the anti-inflammatory effect of colchicine in this clinical setting.

A similar pattern was observed for NLR, which decreased more markedly in the colchicine group than in the non-colchicine group. NLR has been increasingly recognized as a useful marker of systemic inflammation and an accessible prognostic indicator in patients with ACS. Elevated NLR has been associated with worse outcomes, including higher mortality, greater risk of adverse cardiovascular events, and longer hospital stay [7,8]. The greater reduction in NLR in the colchicine group, therefore, provides additional evidence that colchicine may attenuate inflammatory burden after PCI. Although the present study was not designed to evaluate prognostic performance of NLR, the observed reduction may still be clinically relevant because it reflects improvement in an inflammatory marker that has been linked to poorer cardiovascular prognosis.

Despite the significant reductions in inflammatory biomarkers, the present study did not show significant differences in clinical outcomes, including stroke, repeat revascularization, complications, and death. Several explanations may account for this finding. First, the number of clinical events was very low, which limited the ability to detect meaningful between-group differences. Second, the follow-up period of 3 months may have been too short to observe differences in hard cardiovascular outcomes, which often require longer observation. Third, the study sample was relatively small, and therefore, the study may have been underpowered to detect differences in infrequent clinical events. Consequently, the absence of significant differences in MACE should not be interpreted as evidence of no clinical benefit, but rather as an indication that the present study was primarily able to detect biomarker changes rather than definitive outcome differences.

The findings of this study should also be interpreted in light of several limitations. The observational design limits the ability to establish a causal relationship between colchicine use and the observed reductions in inflammatory markers. Because treatment allocation was not randomized, residual confounding may have remained despite comparable baseline characteristics in many variables. The study was conducted in a single center with a relatively small number of patients, which may limit the generalizability of the findings to other populations and healthcare settings. Some follow-up laboratory data were missing, and complete-case analysis was used. This approach may have introduced bias if patients with missing data differed systematically from those with complete data. The follow-up period was relatively short and may not have been sufficient to evaluate the longer-term impact of colchicine on recurrent cardiovascular events or mortality.

Despite these limitations, this study has important strengths. It provides real-world data on the anti-inflammatory effects of colchicine in Thai patients with ACS undergoing PCI, a population for which evidence remains limited. The serial assessment of inflammatory biomarkers at baseline, 1 month, and 3 months also allowed evaluation of changes over time during the early post-PCI period. These findings add to the growing body of evidence supporting the anti-inflammatory role of colchicine in cardiovascular disease and suggest that this effect may also be observed in routine clinical practice in an Asian population.

Conclusion

Colchicine use in Thai patients with ACS undergoing PCI was associated with significant reductions in hs-CRP levels, neutrophil count, and NLR during the 3-month follow-up period, suggesting a beneficial anti-inflammatory effect in the early post-PCI phase. However, no significant differences were observed in major adverse cardiac events between the colchicine and non-colchicine groups. These findings support the potential role of colchicine as an adjunctive anti-inflammatory therapy in patients with ACS undergoing PCI. Nevertheless, the results should be interpreted with caution because of the observational design, small sample size, and short follow-up duration. Larger prospective studies are needed to confirm these findings and to determine whether the observed reductions in inflammatory markers translate into meaningful clinical benefit.

Ethics approval

The study was approved by the Institutional Review Board of Rajavithi Hospital (approval number: 154/2567).

Acknowledgments

The authors thank the Departments of Nutrition and Dietetics, Research and Technology Assessment, Information Technology, and Nephrology, Rajavithi Hospital, as well as all participating patients.

Competing interests

There were no identified potential conflicts of interest related to this article.

Funding

This study received no external funding.

Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

This study used artificial intelligence (AI) tool, ChatGPT, in the language refinement to improve grammar, sentence structure, and readability of the manuscript. We confirm that all AI-assisted processes were critically reviewed by the authors to ensure the integrity and reliability of the results.

How to cite

Wattanasiriporn W, Rattanasidha P, Munirwan H, Arayangkoon C. Impact of colchicine on hs-CRP, neutrophil levels, neutrophil-to-lymphocyte ratio and major adverse cardiac events (MACEs) in Thai patients with acute coronary syndrome undergoing percutaneous coronary intervention. Narra J 2026; 6 (2): e3079 - http://doi.org/10.52225/narra.v6i2.3079.

References

1. Fihn SD, Blankenship JC, Alexander KP, *et al.* 2014 ACC/AHA/AATS/PCNA/SCAI/STS focused update of the guideline for the diagnosis and management of patients with stable ischemic heart disease: A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines, and the American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. J Am Coll Cardiol 2014;64(18):1929-1949.
2. Libby P. Inflammation in atherosclerosis. Arterioscler Thromb Vasc Biol 2012;32(9):2045-2051.
3. Ross R. Atherosclerosis - An inflammatory disease. N Engl J Med 1999;340:115-126.
4. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med 2005;352:1685-1695.
5. Momiyama Y, Adachi H, Fairweather D, *et al.* Inflammation, atherosclerosis and coronary artery disease. Clin Med Insights Cardiol 2016;8(Suppl 3):67-70.
6. Kaptoge S, Seshasai SRK, Gao P, *et al.* C-reactive protein and incidence of cardiovascular diseases: A meta-analysis of prospective studies. Eur Heart J 2010;31(18):2286-2293.
7. Tamhane UU, Aneja S, Montgomery D, *et al.* Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute coronary syndrome. Am J Cardiol 2008;102(6):653-657.
8. Sawant AC, Adhikari P, Narra SR, *et al.* Neutrophil to lymphocyte ratio predicts short-and long-term mortality following revascularization therapy for ST elevation myocardial infarction. Cardiol J 2014;21(5):500-508.
9. Tardif JC, Kouz S, Waters DD, *et al.* Efficacy of colchicine for prevention of cardiac events in patients with prior myocardial infarction: The COLCOT randomized trial. J Am Coll Cardiol 2019;73(24):2884-2893.
10. Zhang X, Liu C, Zhang X, *et al.* Colchicine prevents acute coronary syndrome by inhibiting the NLRP3 inflammasome. J Cell Physiol 2018;233(12):9461-9470.

11. Nidorf SM, Eikelboom JW, Budgeon CA, Thompson PL. Low-dose colchicine for secondary prevention of cardiovascular disease. J Am Coll Cardiol 2013;61:404-10.
12. Nidorf SM, Fiolet ATL, Mosterd A, *et al*. Colchicine in patients with chronic coronary disease. N Engl J Med 2020;383:1838-1847.
13. Mewton N, Roubille F, Bresson D, *et al*. Effect of colchicine on myocardial injury in acute myocardial infarction. Circulation 2021;144:859-869.
14. Arnett DK, Blumenthal RS, Albert MA, *et al*. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. Circulation 2019;140(11):596-646.
15. Catapano AL, Graham I, De Backer G, *et al*. 2019 ESC/EAS guidelines for the management of dyslipidaemias: Lipid modification to reduce cardiovascular risk. Eur Heart J 2019;41(1):111-188.
16. Byrne RA, Rossello X, Coughlan JJ, *et al*. 2023 ESC guidelines for the management of acute coronary syndromes. Eur Heart J 2023;44(38):3720-3826.
17. Vanhaverbeke M, Veltman D, Pattyn N, *et al*. C-reactive protein during and after myocardial infarction in relation to cardiac injury and left ventricular function at follow-up. Clin Cardiol 2018;41:1201-1206.
18. Leung YY, Yao Hui LL, Kraus VB. Colchicine - Update on mechanisms of action and therapeutic uses. Semin Arthritis Rheum 2015;45(3):341-350.
19. Robertson S, Martínez GJ, Payet CA, *et al*. Colchicine therapy in acute coronary syndrome patients acts on caspase-1 to suppress NLRP3 inflammasome monocyte activation. Clin Sci 2016;130(14):1237-1246
20. Fiolet ATL, Silvis MJM, Opstal TSJ, *et al*. Short-term effect of low-dose colchicine on inflammatory biomarkers, lipids, blood count and renal function in chronic coronary artery disease and elevated high-sensitivity C-reactive protein. PLoS One 2020;15(8):e0237665.
21. Sun M, Dubé MP, Hennessy T, *et al*. Low-dose colchicine and high-sensitivity C-reactive protein after myocardial infarction: A combined analysis using individual patient data from the COLCOT and LoDoCo-MI studies. Int J Cardiol 2022;363:20-22.
22. Ma Y. Role of neutrophils in cardiac injury and repair following myocardial infarction. Cells 2021;10(7):1676.
23. Jordan JE, Zhao ZQ, Vinten-Johansen J. The role of neutrophils in myocardial ischemia-reperfusion injury. Cardiovasc Res 1999;43(4):860-878.