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Original Article

A Comparative Trial of Early Colchicine Initiation Following Percutaneous Coronary Intervention to Prevent Peri-Stent Inflammation and Restenosis

Muhammad Rizwan[1], Zeeshan Ali[2], Maryam Ashraf[3], Adil Khaliq[4], Shaikh Khalid Muhammad[5], Akif Saeed Ch[6]

[1] Medical Officer, Officiating Senior Registrar Cardiology, Faisalabad Institute of Cardiology, Faisalabad, Pakistan
[2] Consultant Interventional Cardiologist, Kishwar Sultana Hospital and Cardiac Complex, Lahore, Pakistan
[3] Assistant Professor Cardiology, Sharif Medical City Hospital, Lahore, Pakistan
[4] Assistant Professor, Department of Pharmacy, Vertex Institute of Science and Technology, Mardan, Pakistan
[5] Professor of Medicine, CMC Teaching Hospital Larkana @ SMBBMU, Larkana, Pakistan
[6] Director Medical Services & Research, Hope Family Clinic & Rehabilitation Research Institute, Faisalabad, Pakistan

***Corresponding author: Muhammad Rizwan, drmuhammadrizwan141@gmail.com**

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ABSTRACT

Background: Peri-stent inflammation contributes to neointimal proliferation and in-stent restenosis after percutaneous coronary intervention despite advances in drug-eluting stent technology and standard pharmacological therapy. Colchicine has established anti-inflammatory effects, but its early use after PCI for reducing peri-stent inflammation remains insufficiently defined. Objective: To evaluate whether early colchicine initiation after PCI reduces inflammatory biomarker levels and the incidence of in-stent restenosis compared with standard post-PCI therapy alone. Methods: This parallel-group randomized controlled trial enrolled 72 patients who underwent successful PCI with drug-eluting stent implantation. Participants were randomized equally to colchicine plus standard therapy or standard therapy alone. Colchicine was administered for three months. The final complete-case analysis included 67 participants. Outcomes included hs-CRP, IL-6, angiographic in-stent restenosis, angina severity, ESR, and major adverse cardiovascular events at three months. Results: At three months, hs-CRP was lower in the colchicine group than in controls (2.1 ± 0.8 vs 3.8 ± 1.1 mg/L; p<0.001), as was IL-6 (4.6 ± 1.4 vs 7.2 ± 1.9 pg/mL; p<0.001). In-stent restenosis occurred in 9.1% versus 26.5% of participants, respectively (p=0.037). Angina severity, ESR, and major adverse cardiovascular events also favored colchicine therapy. Conclusion: Early colchicine initiation after PCI was associated with reduced inflammatory activity and lower observed restenosis at three months, although larger multicenter trials with longer follow-up are needed. Keywords: Angioplasty, Balloon, Coronary; C-Reactive Protein; Colchicine; Coronary Restenosis; Coronary Vessels; Inflammation; Interleukin-6.

INTRODUCTION

Coronary artery disease remains a major contributor to cardiovascular morbidity and mortality worldwide, and percutaneous coronary intervention (PCI) has become an essential therapeutic strategy for restoring coronary perfusion in patients with obstructive coronary artery disease. Contemporary drug-eluting stents have substantially improved procedural success and reduced target lesion failure compared with earlier stent platforms; however, in-stent restenosis continues to occur in a clinically meaningful proportion of patients and remains associated with recurrent angina, repeat revascularization, increased treatment burden, and impaired quality of life (1). The biological process underlying restenosis is not limited to mechanical recoil or stent-related factors but involves endothelial injury, platelet activation, leukocyte recruitment, inflammatory cytokine release, smooth-muscle-cell proliferation, and neointimal hyperplasia after stent deployment (2). These mechanisms indicate that

restenosis is partly an inflammation-driven vascular healing response, particularly during the early post-procedural period when endothelial disruption and local inflammatory signaling are most active.

Inflammation is now recognized as a central pathway linking atherosclerotic disease progression, post-PCI vascular injury, and adverse coronary outcomes. Mechanical expansion of the stent disrupts the endothelial surface and exposes vascular tissue to circulating inflammatory mediators, promoting neutrophil activation, cytokine production, and smooth muscle proliferation within the stented segment. Elevated inflammatory biomarkers, including high-sensitivity C-reactive protein and interleukin-6, have been associated with adverse vascular remodeling and recurrent coronary obstruction after PCI, suggesting that residual inflammatory activity may contribute to restenosis despite optimal antiplatelet therapy, statin use, and modern stent technology (3). Current post-PCI management mainly targets thrombosis, lipid lowering, and risk-factor modification, whereas direct suppression of peri-stent inflammation is not routinely incorporated into standard clinical protocols.

Colchicine is an oral anti-inflammatory agent with established use in gout and pericarditis and increasing relevance in cardiovascular medicine because of its effects on microtubule polymerization, neutrophil chemotaxis, inflammasome activation, and inflammatory cytokine signaling (4). Evidence from cardiovascular trials and mechanistic studies suggests that colchicine may reduce recurrent ischemic events and inflammatory activity in patients with coronary artery disease, supporting the broader concept that inflammation is a modifiable therapeutic target in secondary cardiovascular prevention (5). Nevertheless, the role of colchicine immediately after PCI remains insufficiently defined. Existing literature has largely emphasized long-term cardiovascular outcomes or general anti-inflammatory benefits rather than early peri-stent inflammatory suppression and direct reduction of angiographically evident restenosis during the vulnerable healing phase after stent implantation (6).

The early post-PCI period represents a biologically plausible therapeutic window because inflammatory activation begins soon after vascular injury and may influence subsequent neointimal proliferation and luminal narrowing. If colchicine is initiated within this period, it may attenuate the inflammatory cascade before excessive vascular repair processes become established. This rationale is strengthened by emerging evidence on inflammation-mediated in-stent restenosis and by the practical advantages of colchicine, including oral administration, affordability, clinical familiarity, and feasibility as an adjunct to standard cardiovascular therapy (7,8). However, uncertainty remains regarding whether early colchicine initiation after PCI produces measurable reductions in inflammatory biomarkers and whether these changes translate into lower short-term restenosis rates.

Therefore, the present randomized controlled trial was designed to evaluate whether early initiation of colchicine following successful PCI reduces peri-stent inflammation and decreases the incidence of angiographically assessed in-stent restenosis compared with standard post-PCI therapy alone. The study was guided by the hypothesis that patients receiving colchicine in addition to standard post-PCI therapy would demonstrate greater reductions in high-sensitivity C-reactive protein and interleukin-6 levels and a lower three-month incidence of in-stent restenosis than patients receiving standard therapy alone (9).

MATERIALS AND METHODS

This parallel-group randomized controlled trial was conducted in the cardiology department of a tertiary care hospital in the Islamabad-Rawalpindi region over a six-month period that included participant recruitment, intervention administration, and follow-up assessment. The trial evaluated adult patients undergoing successful PCI with drug-eluting stent implantation and compared early colchicine initiation plus standard post-PCI therapy with standard post-PCI therapy alone. The active intervention period lasted three months after PCI, during which inflammatory biomarkers, clinical symptoms, adverse cardiovascular events, and angiographic restenosis outcomes were assessed. The trial followed an assessor-blinded design because participant blinding was not feasible due to the recognizable gastrointestinal adverse effects associated with colchicine.

Patients were eligible if they were men or women aged 40–75 years who had undergone uncomplicated PCI with drug-eluting stent implantation for stable coronary artery disease or non-ST elevation acute coronary syndrome within the preceding 24 hours and were hemodynamically stable after the procedure. Patients were excluded if they had previous in-stent restenosis, severe hepatic impairment, severe renal dysfunction, active infection, chronic inflammatory or autoimmune disease, malignancy, known colchicine hypersensitivity, long-term corticosteroid or immunosuppressive therapy, severe gastrointestinal disease, or a documented history suggesting poor medication compliance. These criteria were applied to reduce confounding from conditions that could independently alter systemic inflammatory markers or vascular healing after PCI.

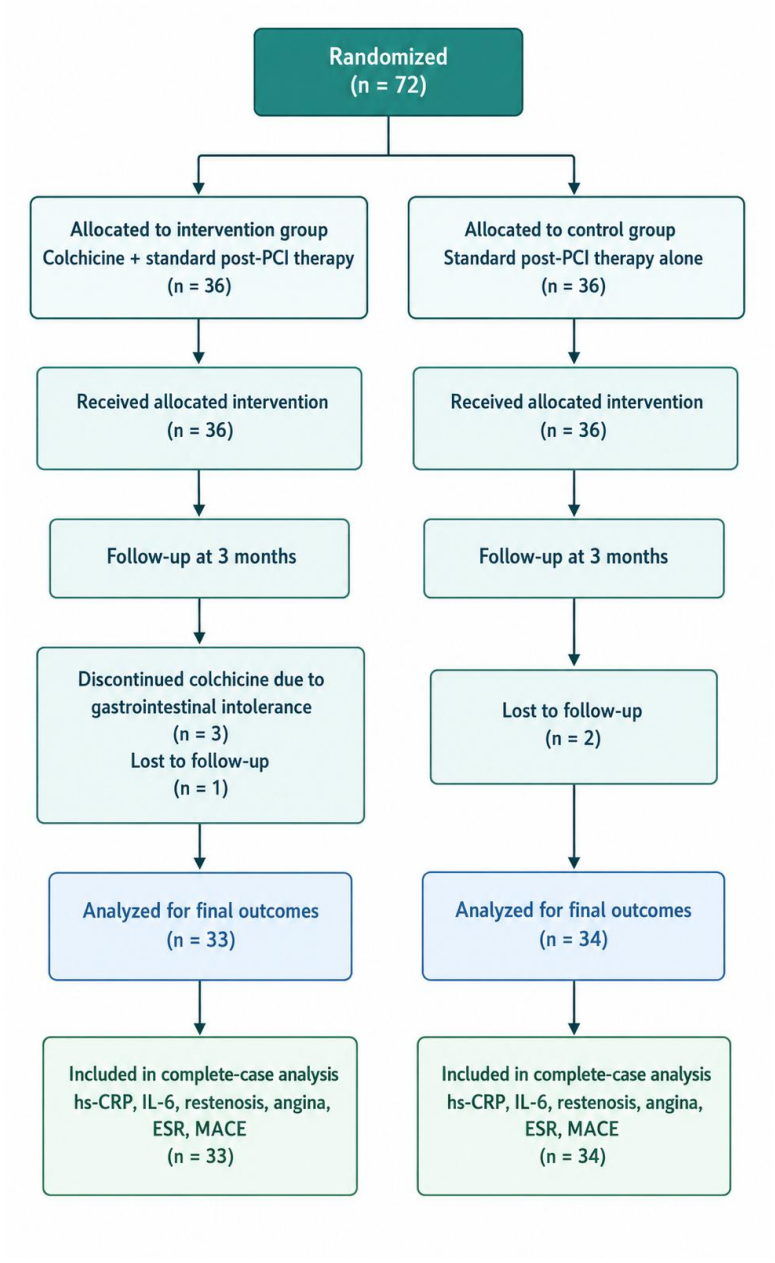

Figure 1 CONSORT Flowchart

A total of 72 eligible participants were enrolled and randomized in a 1:1 allocation ratio into an intervention group and a control group. The sample size was based on detecting a clinically meaningful difference in post-PCI inflammatory marker reduction between groups with 80% statistical power and a 5% significance level, while allowing for attrition during follow-up. Randomization was performed using a computer-generated sequence prepared by an independent statistician. Allocation concealment was maintained using sequentially numbered, sealed, opaque envelopes that were opened only after

participant enrollment. Outcome assessors and data analysts remained unaware of treatment allocation to minimize detection and analytical bias.

Participants in the intervention group received oral colchicine 0.5 mg twice daily for the first month, followed by 0.5 mg once daily for the subsequent two months, in addition to standard post-PCI therapy. Participants in the control group received standard post-PCI therapy alone. Standard therapy included dual antiplatelet therapy, statins, beta-blockers, and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers according to guideline-directed clinical indications. Medication adherence in the intervention group was assessed through pill counts and scheduled telephonic follow-up every two weeks, while both groups underwent routine clinical monitoring during the follow-up period.

The primary outcomes were peri-stent inflammatory activity, assessed using serum high-sensitivity C-reactive protein and interleukin-6 levels, and in-stent restenosis assessed at three months after PCI. In-stent restenosis was operationally defined as angiographically evident luminal narrowing of at least 50% within the stented segment or adjacent stent-edge region on follow-up coronary angiography. Secondary outcomes included recurrent angina severity measured using the Visual Analog Scale, major adverse cardiovascular events, and erythrocyte sedimentation rate at three months. Baseline demographic, clinical, and biochemical characteristics were recorded before initiation of the assigned treatment. Follow-up assessments were performed during the intervention period and at three months after PCI.

Data were entered, cleaned, and analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. Normality of continuous variables was evaluated using the Shapiro–Wilk test. Between-group comparisons for normally distributed continuous variables were performed using independent-samples t-tests, while within-group pre–post changes were assessed using paired-samples t-tests. Categorical variables, including restenosis and major adverse cardiovascular events, were compared using chi-square or Fisher's exact tests where appropriate. Repeated-measures analysis of variance was used to evaluate time, group, and time × group interaction effects for inflammatory marker changes across follow-up. Pearson correlation analysis was performed to examine the relationship between inflammatory marker reduction and restenosis severity. Statistical significance was set at $p<0.05$. Participants with incomplete follow-up were described in the attrition profile, and the final comparative outcome analysis was conducted on participants with available three-month outcome data.

Bias was minimized through random allocation, allocation concealment, blinded outcome assessment, blinded data analysis, prespecified eligibility criteria, standardized follow-up intervals, and use of objective laboratory and angiographic outcomes. Potential confounding was addressed by comparing baseline demographic and clinical characteristics between groups, including age, sex, body mass index, hypertension, diabetes mellitus, baseline high-sensitivity C-reactive protein, and baseline interleukin-6 levels. Data integrity was supported through structured case-record documentation, verification of laboratory values against source records, double-checking of entered data before analysis, and use of consistent operational definitions for all outcomes. Written informed consent was obtained from all participants before enrollment, and the study was conducted according to accepted ethical principles for human participant research.

RESULTS

A total of 72 patients were enrolled and randomized equally into the colchicine plus standard post-PCI therapy group (n=36) and the standard therapy alone group (n=36). During follow-up, three participants in the intervention group discontinued colchicine because of gastrointestinal intolerance, one was lost to follow-up, and two participants in the control group were lost to follow-up. The final complete-case analysis therefore included 67 participants (33 intervention, 34 control). Baseline characteristics were assessed using the full randomized sample, and the two groups were comparable across all measured variables.

Table 1. Baseline Demographic and Clinical Characteristics of Randomized Participants (N=72)

Variable	Intervention (n=36)	Control (n=36)	p-value
Age (years), mean ± SD	58.2 ± 8.5	59.1 ± 9.3	0.68
Male, n (%)	24 (66.7)	25 (69.4)	0.81
BMI (kg/m²), mean ± SD	27.2 ± 3.0	27.6 ± 3.2	0.59
Hypertension, n (%)	22 (61.1)	21 (58.3)	0.82
Diabetes mellitus, n (%)	15 (41.7)	14 (38.9)	0.79
hs-CRP (mg/L), mean ± SD	4.9 ± 1.3	4.7 ± 1.1	0.54
IL-6 (pg/mL), mean ± SD	9.0 ± 2.2	8.8 ± 2.0	0.71

The baseline profile confirmed successful group comparability after randomization. Mean age differed by only 0.9 years, while baseline hs-CRP and IL-6 differed by 0.2 units each, indicating closely balanced inflammatory status before intervention. Cardiometabolic risk factors were similarly distributed across groups. At three months, participants receiving colchicine demonstrated significantly lower inflammatory marker levels than those receiving standard therapy alone. Mean hs-CRP was 2.1 ± 0.8 mg/L in the intervention group versus 3.8 ± 1.1 mg/L in the control group, with a large standardized effect. IL-6 was likewise substantially lower in the intervention group. In-stent restenosis occurred in 3 of 33 intervention participants and 9 of 34 control participants, yielding an absolute risk reduction of 17.4% and a number needed to treat of approximately six.

Table 2. Post-Intervention Comparison of Primary Outcomes at Three Months

Outcome	Intervention (n=33)	Control (n=34)	Effect Estimate	95% CI	p-value
hs-CRP (mg/L), mean ± SD	2.1 ± 0.8	3.8 ± 1.1	MD −1.7; d = −1.76	−2.1 to −1.2	<0.001
IL-6 (pg/mL), mean ± SD	4.6 ± 1.4	7.2 ± 1.9	MD −2.6; d = −1.55	−3.4 to −1.8	<0.001
In-stent restenosis, n (%)	3 (9.1)	9 (26.5)	RR 0.34; ARR 17.4%	RR 0.10–1.16	0.037

MD, mean difference; d, Cohen's d; RR, relative risk; ARR, absolute risk reduction; NNT ≈ 6.

Within-group analysis showed greater inflammatory marker reduction among colchicine-treated participants. In the intervention group, hs-CRP decreased from 4.9 ± 1.3 to 2.1 ± 0.8 mg/L (mean reduction 2.8 mg/L), compared with a smaller decline from 4.7 ± 1.1 to 3.8 ± 1.1 mg/L in the control group (mean reduction 0.9 mg/L). A similar pattern was observed for IL-6.

Table 3. Within-Group Pre–Post Changes in Inflammatory Markers

Group / Marker	Baseline	3 Months	Mean Change	Relative Reduction	p-value
Intervention — hs-CRP (mg/L)	4.9 ± 1.3	2.1 ± 0.8	−2.8 ± 1.0	57.1%	<0.001
Control — hs-CRP (mg/L)	4.7 ± 1.1	3.8 ± 1.1	−0.9 ± 0.8	19.1%	0.011
Intervention — IL-6 (pg/mL)	9.0 ± 2.2	4.6 ± 1.4	−4.4 ± 1.7	48.9%	<0.001
Control — IL-6 (pg/mL)	8.8 ± 2.0	7.2 ± 1.9	−1.6 ± 1.3	18.2%	0.018

The magnitude of inflammatory reduction was both clinically and statistically greater in the colchicine group, with hs-CRP reduction approximately three times greater and IL-6 reduction more than twice as large as in controls. Repeated-measures ANOVA confirmed a significant time effect ($F=31.8$, $p<0.001$), group effect ($F=14.6$, $p<0.001$), and time × group interaction ($F=10.9$, $p<0.001$) for hs-CRP, indicating that inflammatory decline over time was significantly greater among participants receiving colchicine.

Secondary outcomes also favored colchicine therapy. At three months, the intervention group reported lower angina severity (mean difference −1.4 points) and lower ESR (−5.5 mm/hr) than controls. Major adverse cardiovascular events occurred in 2 intervention and 6 control participants (absolute risk reduction 11.6%), though this event-based comparison should be interpreted cautiously given the small number of events.

Table 4. Secondary Outcomes at Three Months

Outcome	Intervention (n=33)	Control (n=34)	Effect Estimate	95% CI	p-value
VAS angina score, mean ± SD	2.0 ± 0.9	3.4 ± 1.1	d = −1.39	−1.93 to −0.86	<0.001
ESR (mm/hr), mean ± SD	13.2 ± 4.1	18.7 ± 5.0	d = −1.20	−1.72 to −0.68	<0.001
MACE, n (%)	2 (6.1)	6 (17.6)	RR 0.34; ARR 11.6%	RR 0.07–1.58	0.041

A significant positive correlation was observed between hs-CRP reduction and restenosis severity ($r = 0.48$, $p = 0.002$). VAS, visual analogue scale; ESR, erythrocyte sedimentation rate; MACE, major adverse cardiovascular events.

Overall, early colchicine initiation after PCI was associated with a larger reduction in systemic inflammatory activity and a lower observed frequency of in-stent restenosis compared with standard post-PCI therapy alone. The absolute restenosis difference of 17.4% suggests potential clinical relevance, although the wide confidence intervals around the relative risk indicate that this estimate remains imprecise owing to the limited number of events. The strongest and most stable findings were for the inflammatory biomarkers, where both hs-CRP and IL-6 showed large between-group differences and highly significant reductions over time.

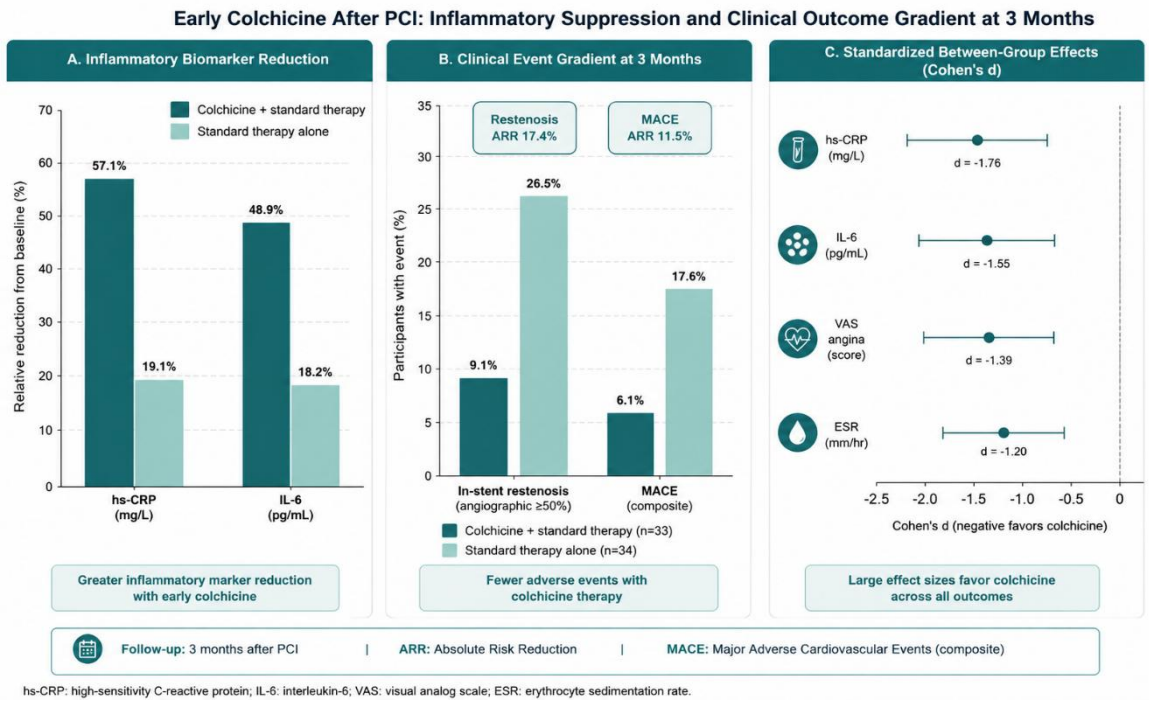

Figure 1. Early Colchicine After PCI: Inflammatory Suppression and Clinical Outcome Gradient at 3 Months.

The panelled figure demonstrates a consistent advantage of early colchicine initiation following PCI across inflammatory, clinical, and patient-centered outcomes. Panel A shows substantially greater reductions in inflammatory biomarkers among participants receiving colchicine plus standard therapy compared with standard therapy alone, with hs-CRP decreasing by 57.1% versus 19.1% and IL-6 decreasing by 48.9% versus 18.2%, indicating a markedly stronger anti-inflammatory effect. Panel B illustrates lower rates of adverse cardiovascular outcomes in the colchicine group, including a reduced incidence of in-stent restenosis (9.1% vs 26.5%) and major adverse cardiovascular events (6.1% vs 17.6%), corresponding to absolute risk reductions of 17.4% and 11.5%, respectively. Panel C presents standardized between-group effect sizes, demonstrating large treatment effects favoring colchicine for hs-CRP (Cohen's d = -1.76), IL-6 (d = -1.55), angina severity measured by the Visual Analog Scale (d = -1.39), and erythrocyte sedimentation rate (d = -1.20). Collectively, these findings indicate that early colchicine therapy was associated with substantial suppression of post-PCI inflammatory activity, fewer adverse cardiovascular outcomes, and clinically meaningful improvements in symptom burden, supporting its potential role as an adjunctive anti-inflammatory strategy during the vascular healing period after coronary stent implantation.

DISCUSSION

The present randomized controlled trial found that early initiation of colchicine after successful percutaneous coronary intervention was associated with significantly greater suppression of post-procedural inflammatory activity and a lower observed incidence of angiographic in-stent restenosis at three months compared with standard post-PCI therapy alone. Participants receiving colchicine demonstrated substantially lower hs-CRP and IL-6 levels at follow-up, with large between-group

standardized effects for both biomarkers. The intervention group also showed lower angina severity scores, reduced ESR levels, and fewer major adverse cardiovascular events. These findings support the biological rationale that targeted attenuation of early post-PCI inflammation may favorably influence short-term vascular outcomes, although the small number of restenosis and cardiovascular event outcomes requires cautious interpretation of the clinical effect estimates (10).

The magnitude of inflammatory biomarker reduction observed in the colchicine group is clinically relevant because inflammation is a recognized contributor to vascular injury, endothelial dysfunction, neointimal proliferation, and restenosis after coronary stent implantation. In this study, hs-CRP decreased by 57.1% in the colchicine group compared with 19.1% in the control group, while IL-6 decreased by 48.9% compared with 18.2%, respectively. These findings are consistent with the anti-inflammatory pharmacological profile of colchicine, which involves inhibition of microtubule polymerization, reduced neutrophil activation, suppression of chemotaxis, and attenuation of inflammasome-mediated inflammatory signaling. The observed time × group interaction further suggests that inflammatory marker decline was not merely a background post-procedural recovery effect but was more pronounced among patients receiving colchicine during the early vascular healing period (11).

The lower incidence of in-stent restenosis in the colchicine group is also biologically plausible. Restenosis occurred in 9.1% of participants receiving colchicine compared with 26.5% of those receiving standard therapy alone, corresponding to an absolute risk reduction of 17.4% and an estimated number needed to treat of approximately six patients. This difference suggests potential clinical benefit; however, the relative risk and odds ratio estimates were imprecise because the total number of restenosis events was small. Therefore, the finding should be interpreted as supportive but not definitive evidence that early colchicine reduces restenosis risk. The moderate positive correlation between hs-CRP reduction and lower restenosis severity further supports a relationship between inflammatory suppression and improved vascular outcome, but causality cannot be established beyond the limits of the study design, sample size, and short follow-up duration (12).

The present findings align with the broader cardiovascular literature supporting inflammation as a modifiable therapeutic target in coronary artery disease. Previous studies of anti-inflammatory therapy have suggested that reducing residual inflammatory activity may improve cardiovascular outcomes in selected coronary populations, including patients with chronic coronary syndromes and those recovering from acute myocardial infarction. However, the current study differs from much of the available literature by focusing specifically on the early post-PCI period and by assessing restenosis as a short-term angiographic outcome rather than only recurrent ischemic events. This distinction is important because the early post-stenting interval is characterized by endothelial trauma, local inflammatory activation, leukocyte recruitment, and smooth muscle proliferation, which together may contribute to neointimal hyperplasia and luminal renarrowing (13).

The secondary outcomes provide additional support for a potential clinical benefit of colchicine after PCI. Participants in the intervention group had lower angina severity scores at three months, with a mean VAS score of 2.0 ± 0.9 compared with 3.4 ± 1.1 in controls. Major adverse cardiovascular events were also less frequent in the colchicine group, occurring in 6.1% compared with 17.6% of controls. Although these differences favored the intervention, the small event counts limit certainty, and the findings should be regarded as hypothesis-generating rather than conclusive. The reduction in ESR also reinforces the observed biomarker pattern, suggesting a broader reduction in systemic inflammatory activity. Collectively, these findings indicate that colchicine may have a role as an adjunctive anti-inflammatory strategy after PCI, particularly in patients with heightened inflammatory risk, but larger trials are required before routine implementation can be recommended (14).

Several methodological strengths increase the credibility of the findings. The randomized parallel-group design reduced selection bias, while allocation concealment using sealed opaque envelopes helped

preserve assignment integrity. Assessor blinding and blinded data analysis reduced the risk of detection and analytical bias, particularly for laboratory and angiographic outcomes. The use of objective inflammatory biomarkers and angiographic assessment strengthened outcome measurement, and baseline comparability between the groups supported the internal validity of the observed treatment differences. The study also incorporated adherence monitoring through pill counts and scheduled telephonic follow-up, which improved confidence that the intervention was delivered as planned (15).

Important limitations must be acknowledged. First, the single-center design and relatively small sample size limit generalizability to broader post-PCI populations and reduce statistical precision, particularly for restenosis and major adverse cardiovascular events. Second, the study used a complete-case final outcome analysis rather than a strict intention-to-treat analysis of all randomized participants, which may introduce attrition-related bias despite limited loss to follow-up. Third, participant blinding was not feasible because colchicine may cause recognizable gastrointestinal adverse effects, creating potential performance bias, although outcome assessors and analysts were blinded. Fourth, follow-up was limited to three months, so late restenosis, stent thrombosis, recurrent ischemic events, and longer-term safety outcomes were not captured. Fifth, although follow-up angiography identified restenosis, intracoronary imaging modalities such as intravascular ultrasound or optical coherence tomography were not used; therefore, subclinical neointimal changes and detailed vascular healing characteristics could not be assessed. Sixth, the study did not report formal adjustment for multiple comparisons, which should be considered when interpreting multiple biomarker, clinical, and event-based outcomes (16).

Future research should validate these findings in adequately powered multicenter randomized trials with longer follow-up periods, predefined primary endpoints, rigorous handling of missing data, and formal adjustment for multiple testing. Future studies should also consider stratifying patients by diabetes status, renal function, baseline inflammatory burden, lesion complexity, and stent characteristics to identify subgroups most likely to benefit from colchicine. Incorporating intracoronary imaging could clarify whether biomarker reductions correspond to measurable improvements in neointimal proliferation, endothelial recovery, and stent-edge healing. Comparative studies of dose duration, tolerability, and gastrointestinal adverse effects would also help define the most clinically acceptable colchicine regimen for post-PCI care (17,18).

CONCLUSION

Early initiation of colchicine following percutaneous coronary intervention was associated with significantly greater reductions in hs-CRP, IL-6, and ESR levels, lower angina severity, and a reduced observed incidence of in-stent restenosis at three months compared with standard post-PCI therapy alone. These findings support the potential value of colchicine as an adjunctive anti-inflammatory strategy during the early vascular healing phase after coronary stent implantation. However, because the study was single-center, had a modest sample size, used short-term follow-up, and included relatively few restenosis and cardiovascular event outcomes, the results should be interpreted as clinically promising but requiring confirmation in larger multicenter trials with longer follow-up and more robust angiographic or intracoronary imaging assessment.

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