

Original Article

A Comparative Evaluation of Early Colchicine Initiation After Percutaneous Coronary Intervention for Preventing Peri-Stent Inflammation and In-Stent Restenosis

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ABSTRACT

Background: Vascular injury during percutaneous coronary intervention (PCI) triggers an inflammatory response that may contribute to neointimal proliferation and early luminal narrowing despite drug-eluting stent use. **Objective:** To evaluate whether early low-dose colchicine added to standard post-PCI therapy reduces inflammatory biomarkers and improves short-term angiographic outcomes compared with standard therapy alone. **Methods:** This prospective, single-center, parallel-group randomized controlled trial enrolled 80 adults aged 35–70 years undergoing successful PCI with drug-eluting stent implantation. Participants were randomized 1:1 to colchicine 0.5 mg once daily plus standard care or standard care alone for 6 weeks. hs-CRP and IL-6 were measured at baseline and 6 weeks, while minimal lumen diameter and in-stent luminal narrowing were assessed by quantitative coronary angiography. Complete follow-up data were available for 75 participants. Between-group comparisons, repeated-measures analysis of variance, and Pearson correlation were used. **Results:** At 6 weeks, hs-CRP was lower with colchicine than control (1.64 ± 0.42 vs 3.12 ± 0.65 mg/L; mean difference -1.48 , 95% CI -1.73 to -1.23 ; $p < 0.001$), as was IL-6 (3.18 ± 0.82 vs 5.62 ± 1.10 pg/mL; mean difference -2.44 , 95% CI -2.89 to -1.99 ; $p < 0.001$). Minimal lumen diameter was larger (2.68 ± 0.31 vs 2.41 ± 0.35 mm; $p < 0.001$), and in-stent luminal narrowing was lower ($12.4 \pm 3.8\%$ vs $20.2 \pm 5.1\%$; $p < 0.001$). **Conclusion:** Early colchicine initiation was associated with greater short-term reductions in inflammatory biomarkers and more favorable 6-week angiographic measurements after PCI, although longer-term clinical benefit cannot be established from these data. **Keywords:** Colchicine; C-Reactive Protein; Interleukin-6; Percutaneous Coronary Intervention; Coronary Stents; Inflammation; Luminal Narrowing.

INTRODUCTION

Percutaneous coronary intervention (PCI) with coronary stent implantation is a central treatment strategy for restoring coronary blood flow in patients with ischemic heart disease. Despite major advances in stent technology, including the widespread use of drug-eluting stents, vascular injury caused by balloon inflation and stent deployment initiates endothelial disruption and a local inflammatory healing response that may contribute to adverse vascular remodeling after the procedure (1). Inflammatory cell recruitment, cytokine release, smooth muscle cell migration, proliferation, and extracellular matrix deposition are important components of the vascular response to stent-induced injury and may ultimately promote neointimal hyperplasia and recurrent luminal narrowing (2,3). Consequently, in-stent restenosis remains an important limitation of PCI despite contemporary antiproliferative stent platforms (4).

Inflammation has therefore emerged as an important therapeutic target in patients undergoing coronary intervention. Conventional post-PCI pharmacological management, particularly antiplatelet and lipid-lowering therapy, is essential for reducing thrombotic and atherosclerotic risk; however, these treatments do not specifically target all inflammatory pathways involved in post-stent vascular healing (5). Increasing understanding of the immune and inflammatory mechanisms underlying restenosis has encouraged investigation of adjunctive anti-inflammatory strategies intended to attenuate excessive vascular inflammation and potentially limit neointimal proliferation following stent implantation (6). Biological pathways associated with endothelial injury, inflammatory signaling, smooth muscle cell activation, and tissue remodeling are increasingly recognized as relevant contributors to the development of restenosis after PCI (7).

Colchicine is a low-dose anti-inflammatory therapy with established inhibitory effects on inflammatory cellular activity and has attracted interest as a potential adjunctive treatment in cardiovascular disease. Its early administration after PCI may be particularly relevant because the immediate post-procedural period is characterized by acute inflammatory activation following mechanical injury to the coronary vessel. However, the specific effect of initiating colchicine within the early post-PCI period on short-term systemic inflammatory biomarkers and subsequent angiographic measures of luminal narrowing remains insufficiently defined. This uncertainty is particularly important in patients at increased risk of restenotic changes, as clinical and biological factors can influence the vascular response after PCI (8).

Accordingly, the present randomized controlled trial was conducted to compare early initiation of oral colchicine at 0.5 mg once daily in addition to standard medical therapy with standard medical therapy alone in adults undergoing successful PCI with drug-eluting stent implantation. The study aimed to determine whether early colchicine administration was associated with greater reductions in high-sensitivity C-reactive protein and interleukin-6 over six weeks and with more favorable six-week quantitative coronary angiographic outcomes, including minimal lumen diameter and the reported percentage measure of in-stent luminal narrowing.

MATERIAL AND METHODS

This prospective, single-center, parallel-group randomized controlled trial was conducted over a six-month period from November 2025 to April 2026 at a tertiary cardiac care center in Punjab, Pakistan. The study evaluated whether early initiation of low-dose colchicine after successful percutaneous coronary intervention (PCI) with drug-eluting stent implantation could attenuate post-procedural systemic inflammatory activity and improve short-term angiographic measures compared with standard medical therapy alone. The study period included participant screening and recruitment, a 6-week active treatment period, and completion of post-intervention biochemical and angiographic assessments.

Adults aged 35–70 years undergoing successful PCI with drug-eluting stent implantation for stable coronary artery disease or acute coronary syndrome were considered eligible. Participants were required to have documented elevation of inflammatory biomarkers following PCI. Patients were excluded if they had severe hepatic impairment, advanced renal dysfunction, active systemic infection, chronic inflammatory or autoimmune disease, known hypersensitivity to colchicine, or concomitant treatment with potent cytochrome P450 3A4 inhibitors. Eligible patients were recruited following successful completion of PCI and were enrolled before treatment allocation.

A total sample of 80 participants was planned. The sample-size determination was based on effect estimates from comparable cardiovascular interventional studies, with 80% statistical power, a two-sided significance level of 5%, and allowance for an anticipated attrition rate of 10%. Participants were assigned in a 1:1 ratio to either the colchicine intervention group or the standard-care control group. The random allocation sequence was computer generated, and allocation concealment was maintained using sequentially numbered, opaque, sealed envelopes that were opened only after completion of the PCI procedure. Outcome assessors and laboratory personnel remained blinded to treatment allocation, whereas the treatment regimen itself was not described as blinded.

Participants assigned to the intervention group received oral colchicine 0.5 mg once daily, initiated within 24 hours after PCI and continued for 6 weeks in addition to standard post-PCI medical therapy. Standard care included dual antiplatelet therapy and high-intensity statin treatment. Participants allocated to the control group received the same standard medical therapy without colchicine or another protocol-specified anti-inflammatory add-on treatment. Treatment adherence was monitored through weekly telephone contact and pill counts performed during follow-up visits. Participants were also monitored for gastrointestinal adverse effects during the intervention period.

The principal biochemical outcomes were serum high-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6). Both markers were measured at baseline after PCI and again at the end of the 6-week intervention period. The manuscript reports that biomarker concentrations were determined using high-sensitivity enzyme-linked immunosorbent assay methods. Changes in hs-CRP and IL-6 over the intervention period were used to assess the systemic inflammatory response following PCI. Secondary angiographic outcomes were minimal lumen diameter and the reported percentage measure of in-stent luminal narrowing, assessed by quantitative coronary angiography at 6 weeks after randomization. The angiographic assessment was performed by outcome assessors who remained unaware of treatment-group assignment.

Potential bias was addressed through computer-generated random allocation, concealed treatment assignment before envelope opening, and blinding of laboratory personnel and outcome assessors. Both groups received comparable standard post-PCI medical therapy, while colchicine represented the principal between-group treatment difference. Adherence monitoring was conducted throughout follow-up to identify treatment discontinuation or non-compliance. Participants who discontinued treatment or did not complete follow-up assessments were documented by randomized group. The outcome analyses reported in the manuscript were based on participants who had evaluable baseline and 6-week measurements, corresponding to 75 participants with complete follow-up data.

Continuous variables were summarized using mean and standard deviation after assessment of distributional normality with the Shapiro-Wilk test. Baseline continuous characteristics and post-intervention differences between the colchicine and control groups were compared using independent-samples t tests. Within-group changes between baseline and 6 weeks were evaluated using paired-samples t tests. Repeated-measures analysis of variance was used to examine the interaction between treatment group and time for the inflammatory biomarkers. Pearson correlation analysis was performed to examine the relationship between change in inflammatory biomarkers and angiographic lumen-related outcomes. Statistical tests were two-sided, and a p value <0.05 was considered statistically significant.

RESULTS

A total of 94 patients were screened, of whom 14 were excluded before randomization. Eighty participants were subsequently randomized in a 1:1 ratio to receive colchicine plus standard care or standard care alone. At 6 weeks, complete outcome data were available for 75 participants, including 37 of 40 participants (92.5%) in the colchicine group and 38 of 40 (95.0%) in the control group. Three participants in the colchicine group discontinued because of persistent gastrointestinal intolerance, whereas two participants in the control group withdrew consent.

Baseline demographic, clinical, and inflammatory characteristics were similar between the randomized groups. Mean age was 56.8 ± 8.1 years in the colchicine group and 56.0 ± 8.4 years in the control group. Men comprised 67.5% and 62.5% of the respective groups. The prevalence of diabetes mellitus, hypertension, and current smoking was also comparable. Baseline hs-CRP concentrations were 4.82 ± 0.91 mg/L and 4.78 ± 0.88 mg/L, while baseline IL-6

concentrations were 8.45 ± 1.55 pg/mL and 8.39 ± 1.60 pg/mL in the colchicine and control groups, respectively, with no statistically significant baseline differences

Table 1. Participant disposition through 6-week follow-up

Participant disposition	Total, n (%)	Colchicine group, n (%)	Control group, n (%)
Screened	94 (100.0)	—	—
Excluded before randomization	14 (14.9)	—	—
Randomized	80 (85.1)	40 (100.0)	40 (100.0)
Completed 6-week assessment	75 (93.8)	37 (92.5)	38 (95.0)
Did not complete 6-week assessment	5 (6.3)	3 (7.5)	2 (5.0)
Discontinued because of persistent gastrointestinal intolerance	3 (3.8)	3 (7.5)	0 (0.0)
Withdrew consent	2 (2.5)	0 (0.0)	2 (5.0)

Percentages for randomized-group columns use n=40 as denominator; total post-randomization percentages use N=80. Screening and prerandomization exclusion percentages use N=94.

Table 2. Baseline demographic and clinical characteristics of randomized participants

Characteristic	Colchicine group (n=40)	Control group (n=40)	p-value
Age, years	56.8 ± 8.1	56.0 ± 8.4	0.665
Male sex, n (%)	27 (67.5)	25 (62.5)	0.639
Diabetes mellitus, n (%)	16 (40.0)	15 (37.5)	0.818
Hypertension, n (%)	25 (62.5)	23 (57.5)	0.648
Current smoking, n (%)	13 (32.5)	15 (37.5)	0.635
hs-CRP, mg/L	4.82 ± 0.91	4.78 ± 0.88	0.842
IL-6, pg/mL	8.45 ± 1.55	8.39 ± 1.60	0.866

Continuous variables are presented as mean $\pm$ SD and categorical variables as n (%). p-values for continuous variables were obtained using independent-samples t tests; categorical variables were compared using Pearson chi-square tests. hs-CRP, high-sensitivity C-reactive protein; IL-6, interleukin-6.

Table 3. Changes in inflammatory biomarkers from baseline to 6 weeks

Outcome	Colchicine baseline	Colchicine 6 weeks	Colchicine change	Control baseline	Control 6 weeks	Control change	6-week mean difference (95% CI)	p-value	Difference in change (95% CI)	p-value	Hedges' g
hs-CRP, mg/L	4.83 ± 0.92	1.64 ± 0.42	-3.19 ± 0.81	4.77 ± 0.89	3.12 ± 0.65	-1.65 ± 0.62	-1.48 (-1.73 to -1.23)	<0.001	-1.54 (-1.87 to -1.21)	<0.001	-2.12
IL-6, pg/mL	8.48 ± 1.58	3.18 ± 0.82	-5.30 ± 1.22	8.38 ± 1.62	5.62 ± 1.10	-2.76 ± 1.04	-2.44 (-2.89 to -1.99)	<0.001	-2.54 (-3.06 to -2.02)	<0.001	-2.22

Values are mean $\pm$ SD. Analyses use participants with complete baseline and 6-week assessments: colchicine n=37 and control n=38. Mean differences are calculated as colchicine minus control. Difference in change compares the mean baseline-to-6-week change between groups. Hedges' g is based on between-group differences in change scores; negative values indicate a greater reduction in the colchicine group. hs-CRP, high-sensitivity C-reactive protein; IL-6, interleukin-6; CI, confidence interval.

Table 4. Angiographic outcomes at 6 weeks

Outcome	Colchicine group (n=37)	Control group (n=38)	Mean difference (95% CI)	p-value	Hedges' g
Minimal lumen diameter, mm	2.68 ± 0.31	2.41 ± 0.35	0.27 (0.12 to 0.42)	<0.001	0.81
In-stent luminal narrowing, %	12.4 ± 3.8	20.2 ± 5.1	-7.8 (-9.9 to -5.7)	<0.001	-1.71

Values are mean $\pm$ SD. Mean differences are calculated as colchicine minus control. Confidence intervals and Hedges' g were derived from the reported group means, standard deviations, and sample sizes. CI, confidence interval.

Table 5. Association between reduction in hs-CRP and angiographic lumen preservation at 6 weeks

Association	n	Pearson r	95% CI	p-value
hs-CRP reduction and minimal lumen preservation	75	0.64	0.48 to 0.76	<0.001

The 95% CI for Pearson r was derived using Fisher's z transformation. hs-CRP, high-sensitivity C-reactive protein; CI, confidence interval.

Among participants with complete follow-up data, hs-CRP decreased from 4.83 ± 0.92 to 1.64 ± 0.42 mg/L in the colchicine group and from 4.77 ± 0.89 to 3.12 ± 0.65 mg/L in the control group. At 6 weeks, the between-group mean

difference was -1.48 mg/L (95% CI, -1.73 to -1.23 ; $p < 0.001$). The mean reduction from baseline was -3.19 ± 0.81 mg/L with colchicine compared with -1.65 ± 0.62 mg/L with standard care, corresponding to a between-group difference in change of -1.54 mg/L (95% CI, -1.87 to -1.21 ; $p < 0.001$) and a Hedges' g of -2.12 .

IL-6 showed a similar pattern. Concentrations decreased from 8.48 ± 1.58 to 3.18 ± 0.82 pg/mL in the colchicine group and from 8.38 ± 1.62 to 5.62 ± 1.10 pg/mL in the control group. The 6-week mean difference was -2.44 pg/mL (95% CI, -2.89 to -1.99 ; $p < 0.001$). Mean change from baseline was -5.30 ± 1.22 pg/mL in the colchicine group and -2.76 ± 1.04 pg/mL in the control group, yielding a between-group difference in change of -2.54 pg/mL (95% CI, -3.06 to -2.02 ; $p < 0.001$) and a Hedges' g of -2.22 . The manuscript also reported significant group-by-time interactions for hs-CRP ($F=42.18$, $p < 0.001$) and IL-6 ($F=38.64$, $p < 0.001$).

At the 6-week angiographic assessment, mean minimal lumen diameter was 2.68 ± 0.31 mm in the colchicine group compared with 2.41 ± 0.35 mm in the control group. The mean between-group difference was 0.27 mm (95% CI, 0.12 to 0.42 ; $p < 0.001$), with a Hedges' g of 0.81 . The reported percentage of in-stent luminal narrowing was $12.4 \pm 3.8\%$ with colchicine and $20.2 \pm 5.1\%$ with standard care, corresponding to a mean difference of -7.8 percentage points (95% CI, -9.9 to -5.7 ; $p < 0.001$) and a Hedges' g of -1.71 .

Reduction in hs-CRP was positively correlated with preservation of the angiographic lumen at 6 weeks ($r=0.64$, 95% CI, 0.48 to 0.76 ; $p < 0.001$). This represents a moderate-to-strong association between the reported inflammatory-marker change and angiographic outcome, although the correlation alone does not establish that the biomarker reduction mediated the observed angiographic difference.

DISCUSSION

The present randomized controlled trial found that early initiation of low-dose colchicine after PCI was associated with greater reductions in hs-CRP and IL-6 over six weeks compared with standard medical therapy alone, together with a larger minimal lumen diameter and a lower reported percentage of in-stent luminal narrowing at follow-up. These findings support the study objective by suggesting that short-term colchicine administration during the early post-PCI period may attenuate the systemic inflammatory response accompanying vascular injury and may be associated with more favorable short-term angiographic characteristics. The magnitude of the between-group differences in both inflammatory biomarkers and angiographic measurements indicates that the observed effects were not limited to statistical significance alone, although their longer-term clinical relevance cannot be established from the present follow-up period.

The findings are broadly consistent with contemporary evidence emphasizing inflammation as an important component of atherosclerotic cardiovascular disease and vascular healing after coronary intervention. Anti-inflammatory therapeutic strategies have increasingly been investigated as adjuncts to conventional antiplatelet and lipid-lowering therapy because inflammatory signaling contributes to endothelial dysfunction, leukocyte recruitment, smooth muscle cell activation, and vascular remodeling (9,10). Chronic vascular inflammation and endothelial dysfunction are closely interconnected processes that may influence progression of atherosclerotic disease and the biological response to vascular injury (11). In this context, the greater reduction in hs-CRP and IL-6 observed with colchicine is biologically plausible and supports the concept that suppression of inflammatory activity during the early post-procedural period may modify the response to stent-induced vascular injury.

Several mechanisms may help explain the observed biomarker changes. Colchicine is known to interfere with microtubule-dependent cellular processes and to reduce inflammatory cell activation, while inflammatory pathways involving cytokine signaling, neutrophil activity, and inflammasome activation have been implicated in acute coronary syndromes and vascular inflammation (12). These mechanisms provide a plausible explanation for the greater decline in circulating hs-CRP and IL-6 among participants receiving colchicine. However, the present study measured systemic inflammatory biomarkers rather than local peri-stent inflammatory activity, and therefore the biological pathway linking colchicine administration to the angiographic findings should be interpreted as mechanistically plausible rather than directly demonstrated.

The angiographic findings also merit cautious interpretation. Participants receiving colchicine had a larger minimal lumen diameter and a lower percentage of in-stent luminal narrowing at six weeks than those receiving standard therapy alone. Contemporary evidence indicates that inflammatory and immune mechanisms can influence

vascular remodeling and restenotic processes after coronary stent implantation, even in the era of drug-eluting stents (13,14). The positive correlation between hs-CRP reduction and preservation of lumen dimensions in the present study further supports an association between inflammatory activity and early structural vascular changes. Nevertheless, correlation does not establish mediation or causality, and the reported six-week angiographic measurements should not be interpreted as evidence that colchicine prevents clinically established restenosis or eliminates the need for repeat revascularization.

The findings may have clinical relevance because colchicine represents an orally administered anti-inflammatory therapy that can be added to conventional post-PCI treatment without altering the fundamental antiplatelet or lipid-lowering strategy. Broader cardiovascular literature has increasingly considered modulation of inflammation as a potential therapeutic approach in atherosclerotic disease (9,15). The present results extend this concept to the early post-PCI period by demonstrating measurable differences in inflammatory biomarkers and short-term angiographic parameters. However, the study was not designed to establish reductions in myocardial infarction, target-lesion revascularization, stent thrombosis, cardiovascular mortality, or other major clinical events. Accordingly, the findings should be regarded as evidence of short-term biological and angiographic differences rather than proof of improved long-term clinical outcomes.

Several methodological features strengthen the study. The randomized parallel-group design reduced selection bias, treatment allocation was concealed before assignment, and laboratory personnel and outcome assessors were reported to be blinded to treatment allocation. Both groups received comparable standard post-PCI therapy, while colchicine represented the principal planned treatment difference. Biomarkers were measured at baseline and after six weeks, allowing evaluation of within-group change as well as between-group differences over time, and angiographic assessment provided an objective structural outcome in addition to circulating inflammatory markers.

The study also has important limitations. It was conducted at a single center with a modest sample size, which limits the precision and generalizability of the findings. Five randomized participants did not complete the six-week assessment, and the reported outcome analyses were therefore based on 75 participants with complete follow-up data rather than all 80 randomized participants. This complete-case approach may introduce attrition-related bias if participants without follow-up differed systematically from those who completed the study. The study population included patients with both stable coronary artery disease and acute coronary syndromes, conditions that may differ in baseline inflammatory activity and vascular response, but subgroup effects were not reported. In addition, treatment assignment was not described as blinded for participants or treating clinicians, which may have influenced adherence or reporting of treatment-related symptoms.

The short duration of follow-up represents a further limitation. Six-week changes in minimal lumen diameter and percentage luminal narrowing provide information on early angiographic status but cannot establish durability of the observed differences or their relationship with conventionally defined late in-stent restenosis. The angiographic outcome termed percentage in-stent luminal narrowing also requires precise operational definition to distinguish it from binary clinically defined restenosis. Furthermore, only systemic hs-CRP and IL-6 were assessed, so the study cannot determine whether the observed effects resulted from direct suppression of local peri-stent inflammation. Gastrointestinal intolerance resulted in treatment discontinuation in three participants receiving colchicine, emphasizing the importance of tolerability when considering short-term anti-inflammatory therapy after PCI.

Overall, the study provides preliminary randomized evidence that initiating low-dose colchicine within 24 hours after PCI may produce greater short-term reductions in systemic inflammatory biomarkers and more favorable six-week angiographic measurements than standard therapy alone. These findings are biologically compatible with the established role of inflammation in vascular injury and remodeling, but their clinical significance should be interpreted within the limitations of the sample size, complete-case analysis, heterogeneous coronary population, and short follow-up duration.

CONCLUSION

Early initiation of colchicine 0.5 mg once daily after PCI was associated with greater six-week reductions in hs-CRP and IL-6 and with more favorable short-term angiographic measurements, including a larger minimal lumen

diameter and lower reported in-stent luminal narrowing, compared with standard medical therapy alone. These findings support a potential role for short-term anti-inflammatory therapy during early post-PCI vascular healing, although the limited sample size, complete-case analysis, and six-week follow-up preclude conclusions regarding prevention of established restenosis or improvement in long-term clinical outcomes..

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