INPLASY

Anti-Inflammatory Effects of Low-Dose Colchicine in Atherosclerotic Cardiovascular Disease: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

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ADMINISTRATIVE INFORMATION

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Conflicts of interest - None declared.

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Amendments - This protocol was registered with the International Platform of Registered Systematic Review and Meta-Analysis Protocols (INPLASY) on 20 February 2026 and was last updated on 20 February 2026.

INTRODUCTION

Review question / Objective P(Population): Adults with atherosclerosis-related cardiovascular disease (e.g., angina pectoris, ischemic cardiomyopathy, acute coronary syndrome, myocardial infarction).
I (Intervention): Low-dose oral colchicine (0.5–1.0 mg/day).
C (Comparator): Placebo or no additional treatment/standard care without colchicine.
O(Outcomes): Post-treatment circulating inflammatory biomarkers, primarily CRP, IL-6 and IL-1β; other reported cytokines (e.g., IL-16, IL-18) will be summarized when extractable.
S (Study design): Randomized controlled trials.
Objective: To systematically review RCTs and quantify the effect of low-dose colchicine on inflammatory biomarkers in atherosclerosis-related cardiovascular disease compared with placebo/standard care.

Rationale Inflammation contributes to the development and progression of atherosclerosis, and serum inflammatory biomarkers (e.g., CRP, IL-6) are commonly used to reflect inflammatory activity. Low-dose colchicine has been tested in multiple cardiovascular settings, but reported effects on inflammatory biomarkers vary across trials and follow-up periods. A synthesis restricted to RCTs can provide a more precise estimate of biomarker changes and explore potential sources of heterogeneity among studies.

Condition being studied Atherosclerosis is characterized by lipid deposition and plaque formation in the arterial wall and is the pathological basis for many ischemic cardiovascular diseases. Plaque progression and destabilization are closely linked to inflammatory pathways, and circulating inflammatory biomarkers are measurable indicators of systemic inflammation related to atherosclerotic disease activity. This review focuses on atherosclerosis-related cardiovascular

diseases in which colchicine has been evaluated for anti-inflammatory effects using serum biomarkers.

METHODS

Search strategy Databases: PubMed, Embase, Web of Science, Cochrane Library, and ClinicalTrials.gov. Timeframe: inception to 7 August 2025. Search terms will combine colchicine with atherosclerosis-related cardiovascular disease terms and inflammatory biomarker terms. Example strategy: (colchicine) AND (acute coronary syndrome OR coronary disease OR myocardial infarction OR heart failure OR ischemic cardiomyopathy) AND (C-reactive protein OR CRP OR interleukin-6 OR IL-6 OR interleukin-1beta OR IL-1β OR cytokine* OR inflammatory marker*).

Participant or population Adults with a confirmed diagnosis of atherosclerosis-related cardiovascular disease (including angina pectoris, ischemic cardiomyopathy, acute coronary syndrome, and myocardial infarction) enrolled in randomized controlled trials.

Intervention Low-dose oral colchicine, typically 0.5–1.0 mg/day, administered according to the regimen and duration specified in each included trial.

Comparator Placebo or no additional treatment/standard care without colchicine.

Study designs to be included Randomized controlled trials comparing low-dose colchicine with placebo or standard care/no additional treatment in atherosclerosis-related cardiovascular disease.

Eligibility criteria Eligible studies must report extractable post-treatment inflammatory biomarker data (e.g., mean/SD or data convertible to effect sizes).
For multiple reports from the same trial/population, the most complete dataset will be retained.
Non-randomized designs (surveys, case reports, reviews) will be excluded.
Studies without essential information (e.g., sample size, follow-up, outcomes) will be excluded.
Studies focusing on cerebrovascular events/stroke populations are excluded in the current protocol.

Information sources Electronic databases: PubMed, Embase, Web of Science, and the Cochrane Library; trial registry: ClinicalTrials.gov. Records will be de-duplicated and screened against prespecified criteria.

Main outcome(s) Primary outcomes are post-treatment levels of CRP, IL-6, and IL-1β. Secondary outcomes include other reported inflammatory biomarkers (e.g., IL-16, IL-18) when data are extractable. Continuous outcomes will be summarized using standardized mean differences (SMDs) with 95% confidence intervals. For CRP, results will also be summarized by follow-up duration (≤7 days vs ≥2 weeks) when applicable.

Data management Search results will be imported into a reference manager for de-duplication. Two reviewers will independently screen titles/abstracts and then full texts. Data will be extracted into a predefined spreadsheet (study characteristics, intervention regimen, follow-up, and biomarker outcomes) and cross-checked. Disagreements will be resolved by discussion.

Quality assessment / Risk of bias analysis Risk of bias will be assessed using the Cochrane risk-of-bias tool for randomized trials. Each study will be evaluated across standard domains (randomization, deviations from intended interventions, missing outcome data, outcome measurement, and selective reporting), with an overall judgment for each trial.

Strategy of data synthesis Continuous outcomes will be pooled as standardized mean differences (SMDs) with 95% confidence intervals. Statistical heterogeneity will be assessed using the Q test and I^2 statistic; heterogeneity will be considered present when $I^2 > 50\%$ or $P < 0.10$. A fixed-effect model will be used when heterogeneity is low; otherwise, a random-effects model will be applied. Robustness will be checked using influence diagnostics. Publication bias will be explored using funnel plots when the number of studies for an outcome is sufficient. Analyses will be conducted in R using the meta, dmetar, and metafor packages.

Subgroup analysis For CRP, subgroup analysis will be performed according to follow-up duration (≤7 days vs ≥2 weeks) to explore whether treatment duration contributes to heterogeneity, provided sufficient studies are available within subgroups.

Sensitivity analysis Sensitivity analyses will assess the robustness of pooled estimates by excluding influential studies identified through influence diagnostics and leave-one-out analyses. Where appropriate, results will be compared before and after excluding influential trials to evaluate the stability of conclusions.

Country(ies) involved China.

Keywords colchicine; atherosclerotic cardiovascular disease; inflammation; C-reactive protein; meta-analysis.

Contributions of each author

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