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CONTEMPORARY CLINICAL APPLICATIONS OF COLCHICINE IN CARDIOVASCULAR AND INFLAMMATORY DISEASES

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ABSTRACT

Colchicine, an alkaloid derived from Colchicum autumnale, has gained renewed attention in contemporary medicine following advances in the understanding of inflammation as a central mechanism in cardiovascular and systemic inflammatory diseases. The objective of this narrative review was to synthesize clinical evidence published between 2015 and 2025 regarding the modern therapeutic applications of colchicine. A structured review of peer-reviewed randomized controlled trials, meta-analyses, and international guideline documents was performed, and findings were qualitatively integrated.

Current rheumatology guidelines confirm colchicine as a first-line therapy for acute gout flares and for prophylaxis during urate-lowering treatment. In cardiology, guideline recommendations support its use in acute and recurrent pericarditis. Large randomized trials in patients with recent myocardial infarction and chronic coronary disease demonstrated a reduction in major adverse cardiovascular events when colchicine was added to contemporary standard therapy, and subsequent meta-analyses further evaluated its role in secondary cardiovascular prevention. Recent European guidelines for chronic coronary syndromes state that low-dose colchicine should be considered in selected patients as part of an event-prevention strategy. Additional studies suggest benefit in inflammation-related atrial fibrillation, particularly in postoperative settings, although gastrointestinal adverse events remain the most frequently reported side effects. Colchicine was also investigated in non-hospitalized patients during the COVID-19 pandemic.

Overall, contemporary evidence supports colchicine as an established anti-inflammatory therapy with validated rheumatologic and expanding cardiovascular applications, while emphasizing the importance of careful patient selection and safety monitoring.

KEYWORDS

Colchicine, Coronary Artery Disease, Pericarditis, Gout, Atrial Fibrillation, Anti-Inflammatory Therapy

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Introduction

Inflammation is a fundamental biological response underpinning the initiation and progression of numerous acute and chronic diseases and remains a central target of contemporary therapeutic strategies. Over the past decade, growing recognition of innate immune activation in cardiovascular and systemic inflammatory disorders has increased interest in established anti-inflammatory agents with well-characterized pharmacology and long-standing clinical use. In particular, persistent low-grade inflammation has been identified as a key contributor to plaque instability in atherosclerosis, recurrent ischemic events, and inflammatory cardiac syndromes. This evolving understanding has shifted part of modern therapeutic focus toward modulation of inflammatory pathways in addition to traditional lipid-lowering and antithrombotic approaches. Colchicine, an alkaloid derived from Colchicum autumnale, exemplifies such an agent, with renewed relevance driven by both mechanistic insights and emerging clinical evidence across multiple specialties (Leung et al., 2015).

At the cellular level, colchicine binds to tubulin and disrupts microtubule polymerization, thereby altering cytoskeletal dynamics essential for inflammatory cell trafficking and activation. These effects translate into inhibition of neutrophil chemotaxis and adhesion, attenuation of leukocyte–endothelial interactions, and modulation of innate immune signaling pathways implicated in inflammatory amplification (Leung et al., 2015). Through interference with microtubule-dependent processes, colchicine attenuates inflammatory cell recruitment and reduces propagation of cytokine-mediated responses. Mechanistically, these actions provide a biologically plausible basis for therapeutic benefit in conditions characterized by persistent or dysregulated innate immune responses, including crystal-induced inflammation and vascular inflammatory processes.

Importantly, colchicine exerts these effects without functioning as a broad immunosuppressive agent, which may partly explain its acceptable safety profile at low doses in chronic use (Leung et al., 2015).

Colchicine is firmly established in rheumatology as a cornerstone therapy for gout. Contemporary guideline documents reaffirm its role as first-line treatment for acute gout flares and as prophylaxis during initiation of urate-lowering therapy (Richette et al., 2017; FitzGerald et al., 2020). These recommendations reflect decades of clinical experience and randomized evidence supporting its efficacy in crystal-induced inflammation. In cardiology, colchicine is recommended as part of standard management for acute and recurrent pericarditis, reflecting its efficacy in reducing symptom burden and preventing recurrences (Adler et al., 2015). More recently, clinical evidence has extended to pericardial disease with myocardial involvement, with observational data supporting its use in myopericarditis as well (Collini et al., 2024). Together, these established indications form the foundation upon which broader cardiovascular applications have been explored.

Beyond these traditional uses, the last decade has seen substantial investigation of colchicine in atherosclerotic cardiovascular disease, where residual inflammatory risk persists despite contemporary preventive therapies. Large randomized controlled trials demonstrated that colchicine, when added to standard care in patients with recent myocardial infarction or chronic coronary disease, reduced composite cardiovascular outcomes (Tardif et al., 2019; Nidorf et al., 2020). These trials provided the first large-scale outcome data supporting targeted anti-inflammatory therapy in secondary cardiovascular prevention. Subsequent systematic reviews and meta-analyses further evaluated the consistency of benefit across coronary disease populations and addressed safety signals and tolerability (Fiolet et al., 2021; Ahmed et al., 2025; Xie et al., 2025). Reflecting this accumulating evidence base, recent European guidance for chronic coronary syndromes states that low-dose colchicine should be considered in selected patients as part of an event-prevention strategy (Vrints et al., 2024).

In addition to coronary disease prevention, inflammatory modulation with colchicine has been explored in arrhythmia-related contexts. An updated meta-analysis of randomized trials suggested that colchicine may reduce the incidence of atrial fibrillation, particularly in inflammation-related settings, although gastrointestinal adverse effects and treatment discontinuation were more frequently reported (Tian et al., 2024). During the COVID-19 pandemic, colchicine was also evaluated as an anti-inflammatory intervention in non-hospitalized patients, with randomized evidence assessing clinically relevant outcomes in selected populations (Tardif et al., 2021). These investigations illustrate the breadth of clinical scenarios in which inflammation has been targeted therapeutically using a well-known, low-cost agent.

Despite expanding clinical use, important uncertainties remain regarding optimal patient selection, integration into long-term preventive strategies, duration of therapy, and practical safety considerations in populations characterized by comorbidity and polypharmacy. Questions persist regarding long-term mortality impact, identification of subgroups deriving the greatest benefit, and appropriate positioning of colchicine within comprehensive cardiovascular risk-reduction frameworks. Accordingly, the objective of this narrative review is to synthesize evidence published between 2015 and 2025 on contemporary colchicine applications, with emphasis on mechanistic foundations, established rheumatologic and pericardial indications, clinical endpoints, and reported adverse events. For guideline documents, class and level of recommendation were reviewed when applicable.

Methodology

Study Design

This study was conducted as a structured narrative review of peer-reviewed scientific literature addressing contemporary clinical applications of colchicine. The review focused on evidence published between January 1, 2015, and December 31, 2025, in order to capture modern mechanistic understanding, randomized clinical trial data, and current international guideline recommendations. A narrative approach was selected due to the heterogeneity of clinical indications, study populations, and outcome measures across therapeutic domains.

Literature Search Strategy

A structured search of the PubMed/MEDLINE database was performed to identify relevant publications. Search terms included combinations of the following keywords: “colchicine,” “gout,” “pericarditis,” “myopericarditis,” “coronary artery disease,” “chronic coronary syndrome,” “myocardial infarction,” “atrial fibrillation,” “COVID-19,” and “inflammasome.” Boolean operators (AND, OR) were applied to refine search

results and ensure comprehensive retrieval of studies addressing both established and emerging clinical indications.

In addition to database searches, major international clinical guidelines were identified through targeted searches of professional society publications, including documents issued by European cardiology and rheumatology bodies and the American College of Rheumatology. Only formally published guideline documents were considered eligible.

Inclusion and Exclusion Criteria

Studies were eligible for inclusion if they met the following criteria:

1. Published between January 2015 and December 2025
2. Peer-reviewed and written in English
3. Randomized controlled trials, systematic reviews, meta-analyses, or formal international clinical guidelines
4. Investigated colchicine use in human subjects
5. Reported clinically relevant outcomes, safety data, or mechanistic insights directly related to therapeutic application

Study Selection and Data Extraction

Titles and abstracts identified through database searches were screened for relevance. Full-text articles were reviewed when necessary to confirm eligibility. Emphasis was placed on landmark randomized controlled trials evaluating cardiovascular outcomes, high-quality meta-analyses synthesizing randomized evidence, and guideline documents providing formal therapeutic recommendations. From each included study, the following data were extracted: study design, sample size, population characteristics, intervention dosage and duration, primary and secondary clinical endpoints, and reported adverse events. For guideline documents, class and level of recommendation were reviewed when applicable.

Data Synthesis

Given the diversity of clinical indications and study designs, findings were synthesized qualitatively using a narrative integration approach. Evidence was categorized according to major clinical domains:

1. Gout and rheumatologic indications
2. Pericarditis and myopericarditis
3. Chronic coronary syndromes and post–myocardial infarction management
4. Atrial fibrillation
5. COVID-19

Mechanistic insights, clinical efficacy data, safety considerations, and guideline recommendations were integrated within each domain to provide a comprehensive overview of colchicine's contemporary role in clinical practice. Quantitative pooling of results was not performed, as the objective of this review was descriptive and integrative rather than meta-analytic.

Results

Mechanistic Basis Supporting Clinical Application

The contemporary therapeutic applications of colchicine are supported by a well-characterized anti-inflammatory mechanism of action. Colchicine binds to β-tubulin and inhibits microtubule polymerization, thereby disrupting cytoskeletal organization and interfering with leukocyte migration, chemotaxis, and adhesion (Leung et al., 2015). Through modulation of neutrophil recruitment and attenuation of inflammatory signaling cascades, colchicine reduces amplification of innate immune responses implicated in crystal-induced inflammation and vascular inflammatory processes.

These mechanistic properties provide biological plausibility for colchicine's established efficacy in gout and pericarditis and support its evaluation in atherosclerotic cardiovascular disease. Importantly, colchicine functions as a targeted modulator of innate inflammatory pathways rather than as a broad immunosuppressive agent, which may contribute to its acceptable safety profile at low therapeutic doses (Leung et al., 2015).

Rheumatologic Indication: Gout

Colchicine remains a cornerstone therapy in gout management. The 2016 European League Against Rheumatism (EULAR) recommendations endorse colchicine as first-line treatment for acute gout flares and as prophylaxis during initiation of urate-lowering therapy (Richette et al., 2017). Similarly, the 2020 American College of Rheumatology (ACR) guideline confirms colchicine as an appropriate first-line anti-inflammatory option in both flare treatment and prophylaxis settings (FitzGerald et al., 2020).

These guideline-level recommendations reinforce colchicine's established role in rheumatology and form the therapeutic foundation from which broader cardiovascular applications have evolved.

Pericarditis and Myopericarditis

The 2015 European Society of Cardiology (ESC) Guidelines for the diagnosis and management of pericardial diseases recommend colchicine as part of first-line therapy in both acute and recurrent pericarditis (Adler et al., 2015). Its inclusion in guideline-based management reflects consistent evidence demonstrating reduction in recurrence rates.

More recent data extend this evidence to inflammatory cardiac conditions with myocardial involvement. In a 2024 propensity score–matched cohort study including 175 patients with a first episode of pericarditis and concomitant myocardial involvement, colchicine therapy was associated with significantly lower recurrence rates compared with standard therapy alone (19.2% vs. 43.8%; HR 0.39; 95% CI 0.21–0.76; p = 0.005) during a median follow-up of 25.3 months (Collini et al., 2024). Adverse events were infrequent (1.7%) and mild, supporting favorable tolerability in this setting.

Atherosclerotic Cardiovascular Disease: Post–Myocardial Infarction

The Colchicine Cardiovascular Outcomes Trial (COLCOT) evaluated colchicine 0.5 mg once daily in 4,745 patients within 30 days after myocardial infarction (Tardif et al., 2019). Over a median follow-up of 22.6 months, colchicine significantly reduced the composite endpoint of cardiovascular death, resuscitated cardiac arrest, myocardial infarction, stroke, or urgent hospitalization for angina requiring revascularization (5.5% vs. 7.1%; HR 0.77; 95% CI 0.61–0.96; p = 0.02).

Gastrointestinal adverse events were more frequent in the colchicine group. A small numerical increase in pneumonia cases was reported; however, overall serious adverse event rates were comparable between treatment groups.

Chronic Coronary Disease

The LoDoCo2 trial enrolled 5,522 patients with chronic coronary disease and evaluated colchicine 0.5 mg once daily versus placebo (Nidorf et al., 2020). During a median follow-up of 28.6 months, colchicine significantly reduced the composite endpoint of cardiovascular death, myocardial infarction, ischemic stroke, or ischemia-driven revascularization (6.8% vs. 9.6%; HR 0.69; 95% CI 0.57–0.83; p < 0.001). A numerical increase in non-cardiovascular mortality was observed (HR 1.51; 95% CI 0.99–2.31), although causality was not established and subsequent pooled analyses did not confirm a consistent excess risk.

Meta-Analytic Evidence in Coronary Disease

Meta-analyses integrating randomized trial data demonstrate consistent cardiovascular benefit. A 2021 meta-analysis including 11,816 patients reported a pooled risk ratio of 0.75 (95% CI 0.61–0.92; p = 0.005) for major adverse cardiovascular events (Fiolet et al., 2021).

An updated 2025 meta-analysis including 21,800 patients from six randomized trials demonstrated a pooled hazard ratio of 0.75 (95% CI 0.56–0.93) for recurrent cardiovascular events, without a statistically significant increase in non-cardiovascular mortality (Xie et al., 2025).

Collectively, these data indicate an approximate 25–30% relative risk reduction in composite cardiovascular outcomes across diverse coronary populations treated with low-dose colchicine.

In addition to relative risk reductions, the consistency of effect across different coronary populations is particularly noteworthy. Both COLCOT and LoDoCo2 enrolled patients receiving high rates of background evidence-based therapy, including statins, antiplatelet agents, and renin–angiotensin system inhibitors (Tardif et al., 2019; Nidorf et al., 2020). The persistence of treatment benefit despite optimized lipid-lowering and antithrombotic regimens suggests that colchicine targets a residual inflammatory component of atherosclerotic risk not fully addressed by conventional therapies. This observation reinforces the concept of inflammation as an independent and modifiable pathway in secondary cardiovascular prevention.

Importantly, the magnitude of benefit appears consistent across different composite endpoint components, including myocardial infarction and ischemic stroke, although effects on cardiovascular mortality have not reached statistical significance in individual trials. The absence of a clear mortality signal may reflect limited follow-up duration rather than lack of biological effect. Median follow-up periods of approximately two to three years may be insufficient to capture long-term mortality divergence in stable, intensively treated populations.

Furthermore, pooled analyses provide reassurance regarding safety signals initially observed in individual trials. While LoDoCo2 reported a numerical increase in non-cardiovascular mortality, updated meta-analytic evidence did not confirm a statistically significant excess risk (Xie et al., 2025). This strengthens

confidence in the overall benefit–risk balance when colchicine is administered at low doses in carefully selected patients with established coronary disease.

Table 1 summarizes the principal randomized controlled trials and meta-analyses evaluating low-dose colchicine across major cardiovascular and inflammatory indications.

Table 1. Major Randomized Trials and Meta-analyses Evaluating Colchicine (2015–2025)

Clinical Setting	Study (Year)	Sample Size (N)	Dose	Follow-up	Primary Outcome	Effect Estimate
Post–Myocardial Infarction	COLCOT (Tardif et al., 2019)	4,745	0.5 mg once daily	Median 22.6 months	Composite CV endpoint	HR 0.77 (95% CI 0.61–0.96)
Chronic Coronary Disease	LoDoCo2 (Nidorf et al., 2020)	5,522	0.5 mg once daily	Median 28.6 months	Composite CV endpoint	HR 0.69 (95% CI 0.57–0.83)
Coronary Artery Disease	Fiolet et al., 2021 (Meta-analysis)	11,816	Low-dose regimens	–	MACE	RR 0.75 (95% CI 0.61–0.92)
Secondary Prevention	Xie et al., 2025 (Meta-analysis)	21,800	Mainly 0.5 mg daily	12–34 months	Recurrent CV events	HR 0.75 (95% CI 0.56–0.93)
Atrial Fibrillation	Tian et al., 2024 (Meta-analysis)	16,238	Variable regimens	Variable	Incident AF	RR 0.75 (95% CI 0.68–0.83)
Pericarditis with Myocardial Involvement	Collini et al., 2024	175	Clinical practice dosing	Median 25.3 months	Recurrence	HR 0.39 (95% CI 0.21–0.76)
COVID-19 (Outpatient)	COLCORONA (Tardif et al., 2021)	4,488	0.5 mg twice daily ×3 days, then daily	30 days	Hospitalization/death (PCR+)	OR 0.75 (95% CI 0.57–0.99)

Abbreviations: AF – atrial fibrillation; CI – confidence interval; CV – cardiovascular; HR – hazard ratio; MACE – major adverse cardiovascular events; OR – odds ratio; RR – risk ratio. Effect estimates are reported as presented in the original publications.

Guideline Integration

Reflecting accumulated randomized and meta-analytic evidence, the 2024 ESC Guidelines for Chronic Coronary Syndromes state that low-dose colchicine (0.5 mg once daily) should be considered in selected patients to reduce myocardial infarction, stroke, and need for revascularization (Vrints et al., 2024). This represents formal incorporation of anti-inflammatory secondary prevention into contemporary European cardiovascular practice.

Atrial Fibrillation

A 2024 meta-analysis of 17 randomized controlled trials (n = 16,238) demonstrated that colchicine reduced atrial fibrillation incidence (RR 0.75; 95% CI 0.68–0.83; $p < 0.001$) (Tian et al., 2024). However, gastrointestinal adverse events and treatment discontinuation were more frequent in colchicine-treated patients. These findings suggest a potential role in inflammation-related atrial fibrillation, balanced against tolerability considerations.

COVID-19

The COLCORONA trial evaluated colchicine in non-hospitalized patients with COVID-19 (Tardif et al., 2021). In PCR-confirmed cases, colchicine reduced hospitalization or death (4.6% vs. 6.0%; OR 0.75; 95% CI 0.57–0.99; $p = 0.042$). Gastrointestinal adverse events were more frequent in the colchicine group (13.7% vs. 7.3%). The observed benefit was modest and context-specific.

Safety Profile and Drug Interactions

Across contemporary randomized controlled trials and meta-analyses, low-dose colchicine (0.5 mg once daily) has demonstrated an overall acceptable safety profile in cardiovascular and inflammatory populations.

Gastrointestinal intolerance represents the most consistently reported adverse effect and is generally dose-related. Most events were mild and rarely led to permanent discontinuation (Nidorf et al., 2020; Tardif et al., 2019).

Although isolated mortality signals were observed in individual trials, pooled analyses have not confirmed a statistically significant increase in non-cardiovascular mortality (Xie et al., 2025).

Colchicine is metabolized primarily via cytochrome P450 3A4 and transported by P-glycoprotein (Leung et al., 2015). Concomitant use with strong metabolic inhibitors may increase toxicity risk. However, in large cardiovascular trials including patients receiving statins and antiplatelet agents, no consistent excess of severe toxicity or myopathy was observed at low-dose regimens. Overall, when administered at low doses in appropriately selected patients, colchicine demonstrates a favorable benefit–risk profile in inflammatory and cardiovascular conditions.

Beyond the overall tolerability profile, interpretation of safety data requires consideration of the clinical context in which colchicine is used. In both COLCOT and LoDoCo2, the absolute incidence of serious adverse events was low, and discontinuation rates due to toxicity remained modest, suggesting that low-dose regimens are feasible in long-term secondary prevention settings (Tardif et al., 2019; Nidorf et al., 2020). The most consistent limitation across trials was gastrointestinal intolerance, which appears to be dose-related and typically emerges early during therapy. Importantly, most reported events were mild and self-limiting, and only a minority of patients required permanent discontinuation.

Signals observed in individual trials—such as a numerical increase in pneumonia in COLCOT and non-cardiovascular mortality in LoDoCo2—have generated discussion regarding systemic immunomodulatory effects. However, pooled analyses integrating randomized evidence have not demonstrated a statistically significant excess in non-cardiovascular mortality (Xie et al., 2025). This distinction between isolated trial-level signals and aggregated meta-analytic findings is clinically relevant and supports cautious but not prohibitive interpretation of safety concerns.

From a practical perspective, the benefit–risk balance appears most favorable in patients with established inflammatory or atherosclerotic disease in whom absolute cardiovascular risk is sufficiently high to justify adjunctive therapy. Careful patient selection, attention to renal function, and monitoring of tolerability remain essential components of responsible implementation. Within these parameters, contemporary evidence suggests that low-dose colchicine can be incorporated into selected treatment strategies without disproportionate safety compromise.

Discussion

Integration of Mechanistic Rationale and Clinical Evidence

The body of evidence published between 2015 and 2025 supports colchicine as a targeted anti-inflammatory therapy with reproducible clinical benefit in selected inflammatory and cardiovascular conditions. Its mechanism—binding to β-tubulin and inhibiting microtubule polymerization—leads to modulation of neutrophil chemotaxis, adhesion, and inflammatory signaling (Leung et al., 2015). This pharmacologic action provides biological plausibility for efficacy in crystal-induced inflammation, pericardial syndromes, and atherosclerotic cardiovascular disease.

In atherosclerosis, persistent low-grade inflammation contributes to plaque instability and recurrent ischemic events. The demonstration of clinical benefit in randomized trials therefore represents translational confirmation of inflammation as a modifiable therapeutic target. The COLCOT trial showed that colchicine reduced composite cardiovascular outcomes in patients treated after myocardial infarction (Tardif et al., 2019), while LoDoCo2 demonstrated similar benefit in chronic coronary disease (Nidorf et al., 2020). These findings were subsequently reinforced by pooled analyses confirming approximately 25% relative risk reduction in major adverse cardiovascular events (Fiolet et al., 2021; Xie et al., 2025; Ahmed et al., 2025).

Beyond statistical significance, the magnitude and consistency of effect observed across independent trials warrant clinical consideration. Although reductions were primarily driven by composite cardiovascular endpoints rather than isolated mortality outcomes, prevention of recurrent myocardial infarction, ischemic stroke, and urgent revascularization carries substantial implications for long-term morbidity, healthcare utilization, and quality of life. The convergence of findings from post–myocardial infarction and stable coronary disease populations strengthens the argument that inflammation represents a sustained contributor to residual cardiovascular risk across disease stages. Importantly, the reproducibility of benefit at a uniform low-dose regimen (0.5 mg daily) suggests a stable pharmacodynamic effect rather than context-specific variability.

The integration of colchicine into European guidance for chronic coronary syndromes further reflects a conceptual evolution in secondary prevention strategies. Historically, lipid lowering and antithrombotic therapy constituted the principal pillars of risk reduction. The addition of targeted anti-inflammatory therapy signals a broader therapeutic framework recognizing inflammation as an independent and modifiable axis of cardiovascular risk. Within this framework, colchicine occupies a distinct niche as an oral, low-cost agent with mechanistic specificity and supportive randomized evidence.

However, the absence of definitive mortality reduction underscores the importance of proportional interpretation. Colchicine should be regarded as an adjunctive therapy aimed at event modification rather than replacement of established preventive strategies. Its clinical value appears most meaningful in patients with established disease and demonstrable residual risk despite optimized background therapy.

Importantly, benefits were observed in populations receiving contemporary optimal medical therapy, including statins and antiplatelet agents, indicating that colchicine addresses residual inflammatory risk not fully mitigated by lipid-lowering and antithrombotic strategies.

Cardiovascular Secondary Prevention: Clinical Relevance and Boundaries

The cardiovascular evidence base is strengthened by large multicenter randomized trials and updated meta-analyses with consistent directionality of effect. However, interpretation requires careful contextualization. Neither COLCOT nor LoDoCo2 demonstrated statistically significant reductions in cardiovascular or all-cause mortality (Tardif et al., 2019; Nidorf et al., 2020). While composite endpoints were significantly reduced, mortality signals remain inconclusive within available follow-up durations.

Meta-analytic synthesis has not demonstrated a statistically significant excess of non-cardiovascular mortality (Xie et al., 2025; Ahmed et al., 2025), suggesting that earlier isolated safety signals may not represent a consistent treatment effect. Nonetheless, absence of clear mortality benefit underscores that colchicine should be viewed as adjunctive risk modification rather than definitive mortality-reducing therapy.

The 2024 European Society of Cardiology (ESC) Guidelines for Chronic Coronary Syndromes state that low-dose colchicine should be considered in selected patients as part of an event-prevention strategy (Vrints et al., 2024). This inclusion formalizes inflammation modulation within European cardiovascular secondary prevention frameworks. At the same time, evidence remains confined to secondary prevention populations with established disease; its role in primary prevention has not been established within the reviewed evidence.

Pericardial Disease and Inflammatory Cardiac Syndromes

Colchicine's role in pericardial disease remains one of its most firmly established cardiovascular indications. The 2015 ESC Guidelines recommend colchicine as part of first-line therapy in acute and recurrent pericarditis (Adler et al., 2015). This recommendation reflects consistent evidence of recurrence reduction and symptom control.

More recent data suggest benefit in pericarditis with myocardial involvement. In a 2024 propensity score–matched cohort study, colchicine therapy was associated with significantly reduced recurrence rates in patients with pericarditis and concomitant myocardial involvement (Collini et al., 2024). Although observational, these findings extend mechanistic plausibility into broader inflammatory cardiac contexts.

In pericardial disease, pathophysiology is directly inflammation-driven, aligning closely with colchicine's pharmacologic action. As such, the consistency between mechanism and clinical response appears particularly robust in this domain.

Atrial Fibrillation and Inflammation-Mediated Arrhythmogenesis

Inflammation contributes to atrial structural remodeling and postoperative arrhythmogenesis. A 2024 meta-analysis of randomized controlled trials demonstrated that colchicine reduced atrial fibrillation incidence across studied contexts (Tian et al., 2024). However, gastrointestinal adverse events and treatment discontinuation were more frequent among colchicine-treated patients.

These findings suggest that colchicine may attenuate inflammation-related atrial fibrillation, particularly in postoperative or high-inflammatory settings. Nevertheless, evidence remains context-dependent, and tolerability considerations limit universal implementation. Current data do not support routine long-term use for primary arrhythmia prevention outside studied populations.

Rheumatologic Indications: Foundational Evidence

Colchicine's long-standing use in gout provides foundational clinical validation of its anti-inflammatory mechanism. The 2016 EULAR recommendations and 2020 ACR guideline reaffirm colchicine as a first-line therapy for acute gout flares and for prophylaxis during urate-lowering therapy initiation (Richette et al., 2017; FitzGerald et al., 2020). These guideline endorsements reflect sustained efficacy and clinical familiarity over decades of use.

Importantly, its rheumatologic role demonstrates durable safety when used at recommended doses, informing risk–benefit considerations in cardiovascular applications.

COVID-19: Context-Specific Anti-Inflammatory Modulation

The COLCORONA trial demonstrated a modest reduction in hospitalization or death among PCR-confirmed non-hospitalized COVID-19 patients treated with colchicine (Tardif et al., 2021). While statistically significant within the predefined subgroup, effect sizes were smaller than those observed in cardiovascular populations, and gastrointestinal adverse events were more frequent.

These findings indicate that colchicine may modulate inflammatory responses in selected infectious contexts, but its role remains situational rather than foundational within infectious disease management.

Safety Profile and Risk–Benefit Considerations

Across major randomized trials and pooled analyses, low-dose colchicine (0.5 mg daily) demonstrated an overall acceptable safety profile (Nidorf et al., 2020; Tardif et al., 2019; Xie et al., 2025). Gastrointestinal intolerance remains the most consistently reported adverse effect. Although isolated signals of pneumonia and non-cardiovascular mortality were reported in individual trials, these findings were not consistently replicated in meta-analyses (Xie et al., 2025; Ahmed et al., 2025).

Colchicine is metabolized via CYP3A4 and transported by P-glycoprotein (Leung et al., 2015), making drug–drug interactions clinically relevant in populations characterized by polypharmacy. Careful medication reconciliation and dose consideration remain essential for safe implementation.

When administered at low doses and in appropriately selected patients, the overall benefit–risk balance appears favorable in secondary cardiovascular prevention and inflammatory cardiac conditions.

Strengths and Limitations of the Contemporary Evidence Base

Strengths include:

1. Large randomized controlled trials with consistent findings.
2. Updated meta-analyses confirming reproducibility of cardiovascular benefit.
3. Formal integration into ESC guidance for chronic coronary syndromes.

Limitations include:

1. Lack of consistent mortality reduction.
2. Limited follow-up beyond three years.
3. Restricted evidence in primary prevention.
4. Heterogeneity in atrial fibrillation populations.
5. Absence of biomarker-guided patient selection strategies.

These gaps highlight the need for longer-term safety data, refined patient selection approaches, and mechanistic stratification strategies.

Clinical Implications

Within the boundaries of current evidence, colchicine may be considered in:

1. Secondary prevention of chronic coronary disease.
2. Early post–myocardial infarction management.
3. Acute and recurrent pericarditis.
4. Selected inflammation-related atrial fibrillation settings.
5. Guideline-supported gout therapy.

In patients with established chronic coronary disease or recent myocardial infarction, colchicine should be viewed as an adjunct to, rather than a substitute for, guideline-directed medical therapy. Randomized trials demonstrating benefit were conducted in populations receiving contemporary standard care, including statins and antiplatelet therapy (Tardif et al., 2019; Nidorf et al., 2020). Accordingly, colchicine use should be considered in individuals who remain at residual cardiovascular risk despite optimized lipid-lowering and antithrombotic treatment. The decision to initiate therapy should involve individualized assessment of overall cardiovascular risk profile, comorbidities, renal function, and concomitant medications.

In pericardial disease, colchicine remains integrated into first-line therapeutic strategies (Adler et al., 2015), with observational evidence supporting potential benefit in inflammatory cardiac involvement including myopericarditis (Collini et al., 2024). In this setting, its role is more firmly established and aligns closely with underlying inflammatory pathophysiology.

For atrial fibrillation, current evidence supports consideration primarily in inflammation-driven or postoperative contexts (Tian et al., 2024). Routine long-term prophylactic use outside studied populations is not supported by available randomized data.

From a practical standpoint, low-dose regimens (0.5 mg once daily in cardiovascular settings) have been consistently evaluated in major trials and meta-analyses (Fiolet et al., 2021; Xie et al., 2025; Ahmed et al., 2025). Gastrointestinal intolerance represents the most common limitation, and clinicians should counsel patients accordingly. Careful review of potential drug–drug interactions, particularly involving CYP3A4 and P-glycoprotein pathways (Leung et al., 2015), remains essential in populations characterized by polypharmacy.

The incorporation of colchicine into contemporary cardiovascular guidance reflects an important shift toward targeting residual inflammatory risk alongside lipid and thrombotic pathways (Vrints et al., 2024). Nevertheless, implementation should remain selective, evidence-aligned, and grounded in individualized clinical judgment, recognizing both the demonstrated benefits in secondary prevention and the boundaries of current data.

Public Health and Health System Implications

Beyond individual-level cardiovascular risk reduction, the contemporary evidence surrounding colchicine has broader implications for healthcare systems and public health strategy. Atherosclerotic cardiovascular disease remains a leading cause of morbidity and mortality worldwide, generating substantial economic burden through recurrent hospitalizations, revascularization procedures, and long-term disability. The demonstration that a low-cost, orally administered anti-inflammatory agent can reduce recurrent cardiovascular events when added to standard therapy introduces an additional dimension to secondary prevention strategies. In contrast to biologic anti-inflammatory therapies targeting interleukin pathways, colchicine is widely available and comparatively inexpensive, potentially enhancing accessibility in resource-constrained settings. Within health systems increasingly focused on cost-effectiveness and scalable preventive interventions, repurposing established pharmacologic agents with well-characterized safety profiles may represent a pragmatic strategy for reducing residual inflammatory risk at population level.

Implementation Considerations in Contemporary Preventive Care

The integration of colchicine into European cardiovascular guidelines reflects not only evolving scientific understanding of inflammation in atherosclerosis but also the broader transformation of preventive cardiology toward multimodal risk management. Contemporary prevention frameworks extend beyond lipid lowering and antithrombotic therapy to incorporate inflammatory modulation as a complementary pathway. However, implementation in routine clinical practice requires structured patient selection, awareness of drug–drug interactions, and monitoring for tolerability, particularly in populations characterized by multimorbidity and polypharmacy. As healthcare systems adopt precision-oriented and guideline-based care models, the incorporation of low-dose colchicine illustrates how mechanistic insights can translate into scalable preventive strategies. Future research should therefore not only refine clinical indications but also evaluate real-world implementation, adherence patterns, and health system impact across diverse populations.

Conclusions

The body of evidence published between 2015 and 2025 establishes colchicine as a clinically relevant anti-inflammatory therapy with validated applications extending beyond its traditional role in gout. Large randomized controlled trials in patients with recent myocardial infarction and chronic coronary disease consistently demonstrated reductions in major adverse cardiovascular events when low-dose colchicine was added to contemporary standard therapy. These findings have been reinforced by systematic reviews and meta-analyses and have been reflected in recent European cardiovascular guidance, underscoring inflammation as a modifiable therapeutic target in atherosclerotic disease.

Colchicine remains a cornerstone of therapy in acute and recurrent pericarditis and retains a well-established position in rheumatologic practice, where long-standing clinical experience supports its efficacy and safety at recommended doses. Emerging evidence in inflammation-related atrial fibrillation suggests potential benefit in selected contexts, while randomized data in non-hospitalized COVID-19 patients indicate modest and context-specific effects.

Importantly, the available evidence supports colchicine primarily as an adjunctive strategy in secondary cardiovascular prevention rather than as a replacement for established lipid-lowering or antithrombotic therapies. Although composite cardiovascular outcomes are consistently reduced, mortality benefits have not been conclusively demonstrated within current follow-up durations. Accordingly, careful patient selection and individualized risk assessment remain essential.

At low therapeutic doses, colchicine demonstrates an acceptable safety profile across inflammatory and cardiovascular populations, with gastrointestinal intolerance representing the most frequent adverse effect.

Drug–drug interaction potential, particularly via CYP3A4 and P-glycoprotein pathways, necessitates prudent prescribing in patients with comorbidities and polypharmacy.

Future research should focus on long-term safety beyond currently available follow-up periods, refinement of patient selection strategies, and clarification of optimal treatment duration in secondary prevention settings. Further investigation into biomarker-guided approaches may enhance personalization of therapy and improve risk–benefit balance.

Overall, contemporary evidence supports colchicine as a targeted anti-inflammatory agent with defined and expanding roles in inflammatory and cardiovascular medicine. Its integration into selected guideline frameworks reflects a broader paradigm shift toward addressing residual inflammatory risk as a complement to traditional cardiometabolic interventions.

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