Review

Colchicine in coronary heart disease: from inflammatory biology to secondary prevention

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Received on April 26, 2026; accepted in revised form on May 28, 2026.

Clin Exp Rheumatol 2026; 44: 1306-1311.

Key words: colchicine, atherosclerosis, inflammation, prevention

Competing interests: none declared.

ABSTRACT

Inflammation is now recognised as a central mechanism in atherosclerosis and its clinical complications, shifting coronary heart disease management beyond exclusive lipid lowering and antithrombotic therapy. In this setting, colchicine is a potential low-cost anti-inflammatory candidate for cardiovascular prevention. While the CANTOS trial provided proof of principle that targeting inflammation can reduce recurrent cardiovascular events independently of lipid lowering, randomised colchicine trials such as COLCOT and LoDoCo2 showed reductions in major adverse cardiovascular events in patients with recent myocardial infarction and chronic coronary disease, respectively. Later meta-analyses generally confirmed benefit for major adverse cardiovascular events, myocardial infarction, stroke, and coronary revascularisation, although without a consistent mortality reduction and with gastrointestinal intolerance as the most common adverse effect. This review summarises the biological rationale, clinical trial evidence, safety profile, practical use, limitations, and current place of colchicine in coronary heart disease highlighting that colchicine is a real available cheap option for secondary prevention at least in chronic coronary syndromes.

Introduction

For decades, the management of coronary heart disease (CHD) has been built on control of thrombosis, ischaemia, and lipids. Yet atherosclerosis is not merely a passive process of cholesterol accumulation. It is a chronic inflammatory disease of the arterial wall in which innate immune activation, cytokine signalling, endothelial dysfunction, and plaque destabilisation all interact to produce clinical events. This understanding has transformed cardiovascular medicine by identifying 'residual inflammatory risk' as a target distinct from residual cholesterol risk. This review is firmly aligned with this paradigm and argues that colchicine, a very old anti-inflammatory drug, may have a modern role in coronary prevention (1).

The modern inflammatory model of atherosclerosis places the NLRP3 inflammasome and interleukin-1/interleukin-6 signalling at the centre of plaque progression and destabilisation. Activation of innate immune pathways promotes leukocyte recruitment, cytokine release, matrix degradation, necrotic core expansion, and thrombogenic plaque rupture. Inflammasome activation is relevant not only to pericarditis and myocarditis but also to ischaemic heart disease and heart failure (2). This biological framework explains why anti-inflammatory agents, once considered peripheral to cardiology, are now legitimate candidates for event prevention (3).

The strongest proof that inflammation is causally involved in recurrent atherosclerotic events came from CANTOS. In that trial, canakinumab, a monoclonal antibody against interleukin-1β, reduced recurrent cardiovascular events without lowering LDL cholesterol. CANTOS was therefore a landmark not because canakinumab became routine therapy, but because it ended the long debate over whether inflammation in atherosclerosis was only a marker or a true therapeutic target (4). Once that proof of principle was established, attention turned toward cheaper and more practical drugs, among which colchicine quickly became the most attractive candidate (5).

Colchicine is an ancient drug, long used in gout and later incorporated into the management of pericarditis (6). Its car-

diovascular relevance comes from its broad anti-inflammatory actions: inhibition of tubulin polymerisation, reduction of neutrophil chemotaxis and adhesion, modulation of inflammasome activation, and attenuation of interleukin-1β and downstream interleukin-6 signalling. In practical terms, colchicine acts upstream of many inflammatory processes implicated in plaque instability. The appeal is obvious: oral administration, once-daily low-dose use, global availability, and relatively low cost compared with biologics (7, 8).

Why inflammation matters in coronary heart disease

Atherosclerotic plaque formation begins with endothelial injury and lipoprotein retention, but progression to unstable plaque depends heavily on inflammation. Monocytes migrate into the intima, differentiate into macrophages, ingest lipids, and contribute to foam cell formation. Cholesterol crystals and danger-associated molecular signals activate the NLRP3 inflammasome, amplifying release of interleukin-1β and other cytokines. This cascade promotes smooth muscle cell dysfunction, matrix metalloproteinase activity, fibrous cap thinning, and prothrombotic transformation. The result is not only a larger plaque burden but also greater plaque vulnerability (1).

Inflammation also helps explain the persistent risk observed in many patients despite excellent LDL-C control. A collaborative analysis of PROMINENT, REDUCE-IT, and STRENGTH found that among statin-treated patients, high-sensitivity C-reactive protein was a stronger predictor of future cardiovascular events and death than LDL-C. This does not make lipid lowering less important; rather, it suggests that optimal prevention often requires both aggressive lipid reduction and attenuation of inflammation. The presentation explicitly echoes this point, framing colchicine as a strategy to address residual inflammatory risk in patients already receiving standard preventive therapy (9).

This shift is conceptually important. Traditional secondary prevention targets platelets, coagulation, heart rate, blood pressure, and cholesterol. Anti-inflammatory therapy introduces a different axis of prevention. That is especially relevant because many recurrent events occur in patients who appear well treated according to conventional benchmarks. Residual inflammatory risk, usually approximated clinically by elevated hsCRP, provides one explanation for this gap between optimal guideline-based therapy and persistent event rates (9).

Why colchicine?

Colchicine is particularly interesting because it is mechanistically plausible, clinically familiar, and economically accessible. Unlike selective biologics, it has pleiotropic anti-inflammatory effects and can be given orally at low dose. In cardiovascular studies, the commonly tested regimen has been 0.5 mg once daily, a dose chosen to balance tolerability with chronic use. In contrast with immunosuppressive agents that raise major infection concerns, low-dose colchicine has generally shown an acceptable safety profile, with gastrointestinal intolerance as the main limitation (10-13).

A second reason for enthusiasm is that colchicine had already proven useful in an adjacent cardiovascular inflammatory condition: pericarditis (6). That experience established cardiologists' familiarity with dosing, contraindications, and expected adverse effects. Although pericarditis and atherosclerosis are distinct diseases, the success of colchicine in suppressing sterile inflammation in the pericardium made the hypothesis of benefit in coronary disease more plausible and clinically palatable. The presentation briefly recalls this history, using it as a bridge between inflammatory cardiology and coronary prevention.

Early clinical evidence

Before the large contemporary outcome trials, the Low-Dose Colchicine (LoDoCo) study provided an early signal that colchicine might improve outcomes in stable coronary disease (10). Published in 2013, LoDoCo suggested that adding colchicine 0.5 mg/day to standard therapy could reduce cardiovascular events in patients with stable coronary disease. Although smaller and less definitive than later trials, it established the feasibility of long-term low-dose treatment and provided the rationale for larger randomised studies (10).

This early signal was important because it shifted colchicine from a mechanistic curiosity to a candidate preventive therapy. The drug appeared capable of addressing a process not fully controlled by statins or antiplatelet agents. At the same time, questions remained about generalisability, long-term safety, the best patient population, and whether benefit would be seen in both stable and unstable coronary settings. These questions set the stage for the major trials that followed.

COLCOT: recent myocardial infarction

The COLCOT trial was the first major modern success for colchicine in coronary disease. In patients enrolled within 30 days after myocardial infarction, colchicine 0.5 mg daily significantly reduced ischaemic cardiovascular events compared with placebo. The trial therefore supported the concept that targeting inflammation soon after infarction can improve secondary prevention on top of contemporary therapy. Importantly, COLCOT did not propose colchicine as an alternative to standard treatment, but as an adjunct to statins, antiplatelets, and other evidence-based therapies (11).

A later PubMed-indexed analysis of COLCOT suggested that earlier treatment initiation after myocardial infarction may be associated with greater benefit. This finding is biologically plausible, because the inflammatory response is especially intense in the early post-infarction period. It also raises a practical question: if colchicine is used after myocardial infarction, should it be started during hospitalisation or soon after discharge rather than deferred until later outpatient review? While not definitively settled, the available evidence supports the idea that timing matters (12).

COLCOT helped establish several recurring themes in colchicine research. First, the benefits appeared more consistent for nonfatal ischaemic events

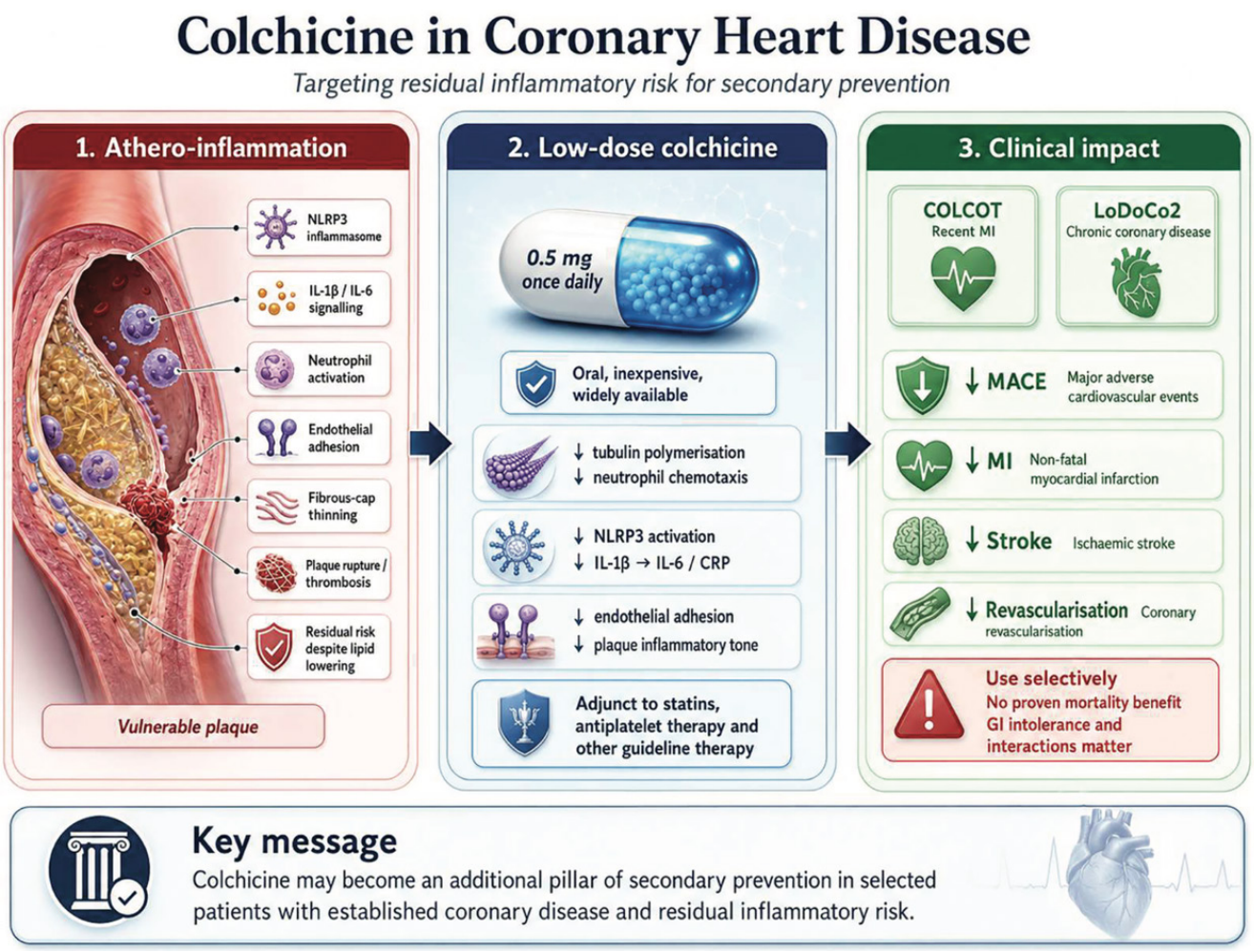

Fig. 1. Colchicine in coronary heart disease.

than for mortality. Second, the drug was broadly usable in real-world secondary prevention populations. Third, tolerability rather than serious toxicity was the main barrier to adherence. Finally, the absolute benefit of colchicine is most meaningful when baseline risk is sufficiently high, which favours its use in patients with established atherosclerotic disease rather than primary prevention (11).

LoDoCo2: chronic coronary disease

If COLCOT was the key post-myocardial infarction study, LoDoCo2 was the decisive trial in chronic coronary disease (13). Published in 2020, LoDoCo2 randomised patients with chronic coronary disease to colchicine 0.5 mg daily or placebo and showed a significant reduction in cardiovascular events. This trial was especially important because it demonstrated benefit outside the immediate post-infarction inflammatory surge, suggesting that chronic low-grade arterial inflammation is also modifiable and clinically relevant (13).

LoDoCo2 broadened the scope of colchicine from a drug potentially useful after recent myocardial infarction to one with possible value across the spectrum of established coronary disease. In other words, benefit was not confined to the unstable plaque phase after infarction. It could also be achieved in ambulatory patients with chronic coronary syndromes already receiving modern preventive care. This is one of the main reasons why recent reviews and the attached presentation frame colchicine as a possible additional 'pillar' of secondary prevention (14-16).

The mechanistic interpretation of LoDoCo2 is attractive. Standard preventive therapies reduce thrombosis, LDL burden, and ischaemic demand; colchicine appears to dampen the inflammatory substrate that predisposes plaques to rupture or erosion. Seen this way, its role is complementary, not competitive. Yet that same complementarity raises an implementation issue: clinicians must decide which patients derive enough incremental benefit to justify long-term addition of another drug.

COPS and conflicting signals

Not all studies have been uniformly positive. The COPS trial in acute coronary syndrome did not show a statistically significant reduction in cardiovascular outcomes at 12 months (17). This reminds us that the colchicine story is favourable overall but not perfectly consistent across all trials and populations. Differences in sample size, timing of enrolment, outcome definitions, adherence, dosing exposure, and statistical power may all contribute to variable individual trial results (17).

The existence of neutral or less convincing trials is important for balanced in-

terpretation. It argues against simplistic overstatements and supports the need for meta-analytic synthesis rather than reliance on one or two prominent studies. It also suggests that patient selection matters. Colchicine may be more effective in some inflammatory phenotypes or risk strata than in others, an issue that remains incompletely resolved.

Meta-analyses and the overall evidence base

Meta-analyses have generally strengthened the case for colchicine. The 2021 European Heart Journal meta-analysis by Fiolet and colleagues concluded that low-dose colchicine reduced major adverse cardiovascular events, myocardial infarction, stroke, and coronary revascularisation across a broad range of patients with coronary disease. At the same time, that analysis did not show an all-cause mortality benefit and noted a possible trade-off between fewer cardiovascular deaths and more non-cardiovascular deaths, although this signal has remained uncertain (14).

A 2024 meta-analysis indexed in PubMed similarly found that colchicine has favourable effects in secondary prevention among patients with established coronary artery disease. The consistency between earlier and later syntheses strengthens confidence that the reduction in recurrent ischaemic events is real, even if the magnitude varies across analyses. By contrast, the absence of a robust mortality reduction is also consistent, suggesting that colchicine should be viewed primarily as an event-reduction strategy rather than a therapy with proven survival benefit (18).

Recent 2025 PubMed-indexed reviews and meta-analyses continue to support this general pattern. Updated evidence suggests lower rates of major adverse cardiovascular events and nonfatal ischaemic complications, but no clear reduction in all-cause or cardiovascular mortality. This matches the message of the review, which emphasises reductions in myocardial infarction, stroke, and revascularisation while acknowledging no significant effect on total or cardiovascular death (19).

From a clinician's perspective, this evidence profile is not trivial. Preventing nonfatal myocardial infarction, ischaemic stroke, and urgent coronary revascularisation is highly meaningful for patients and health systems. Yet the lack of proven mortality benefit influences enthusiasm, reimbursement, and the threshold for routine prescription. Therefore, the best current interpretation is that colchicine is useful for reducing recurrent ischaemic events in selected secondary prevention patients, but it is not a substitute for therapies with established mortality effects (18).

Safety and tolerability

The dominant adverse effect of colchicine in cardiovascular trials has been gastrointestinal intolerance, especially diarrhoea, abdominal discomfort, and treatment discontinuation due to poor tolerance. Serious toxicity has been uncommon at the 0.5 mg/day dose used in major trials, but clinicians must remember the drug's narrow therapeutic window and potential for accumulation in renal or hepatic dysfunction. Drug-drug interactions, especially with strong CYP3A4 or P-glycoprotein inhibitors, remain clinically relevant (20).

The question of infection risk has been debated, but low-dose colchicine has generally not shown the degree of immunosuppressive concern associated with biologics. Likewise, myotoxicity and cytopenias are uncommon in properly selected patients, though caution is warranted in frail individuals and in those receiving interacting medications. In practice, the main reason not to continue colchicine is often not danger but poor tolerability (20).

These safety considerations support a selective rather than indiscriminate approach. Colchicine is better suited to patients with preserved renal and hepatic function, without major interaction risks, and with enough recurrent ischaemic risk to justify another long-term drug. The clinical decision is therefore not only "does colchicine work?" but also "in whom does its net benefit clearly exceed its burden?"

Practical patient selection

At present, the strongest candidates for colchicine appear to be patients with established atherosclerotic coronary disease who remain at meaningful residual risk despite contemporary therapy. This includes patients with prior myocardial infarction, multivessel disease, recurrent events, or persistent inflammatory burden (15, 16).

Whether hsCRP should be routinely used to select patients remains uncertain. The biological rationale is compelling, and data on residual inflammatory risk support such an approach, but major colchicine trials have not required biomarker-guided enrolment in the same way that inflammatory biology might suggest. A reasonable contemporary stance is that elevated hsCRP can strengthen the case for treatment, but normal hsCRP does not necessarily exclude benefit. More precision-based approaches may emerge in the future.

Timing is another practical issue. Evidence from COLCOT supports early post-MI initiation, while LoDoCo2 supports chronic outpatient use. This means colchicine can be considered in two windows: shortly after infarction and later in stable secondary prevention. The strongest evidence does not support its use in primary prevention or in very low-risk patients.

Guideline position and contemporary interpretation

The 2024 ESC Guidelines for chronic coronary syndromes reflect the increasing maturity of the anti-inflammatory approach in secondary prevention. While colchicine is not elevated to the same class-defining status as statins or antiplatelet therapy, the guideline acknowledges contemporary evidence in chronic coronary disease and places colchicine within the broader framework of risk reduction for selected patients. This is a more cautious position than the presentation's emphatic "YES!", but it is directionally consistent: colchicine has moved from experimental periphery to serious clinical consideration (21).

The difference between a trial-based and a guideline-based view matter. Trials answer whether a drug can work under defined conditions; guidelines weigh consistency, generalisability, safety, implementation complexity, and alternative treatments. From this standpoint, colchicine is probably best viewed as

an optional adjunct for appropriately selected secondary prevention patients rather than an automatic universal prescription for everyone with coronary disease (21).

Unresolved issues

Several unresolved questions remain. First, the ideal duration of therapy is not fully established. Trials have generally evaluated long-term use, but whether all patients need indefinite treatment is unknown. Second, the optimal timing after acute coronary events, although suggestive in favour of earlier initiation, still requires refinement. Third, not all trials have been concordant, and heterogeneity persists across acute coronary syndrome, recent myocardial infarction, and chronic coronary disease populations.

Fourth, the field still lacks a precision strategy. We do not yet know whether the greatest benefit lies in patients with elevated hsCRP, recurrent plaque instability, diabetes, clonal haematopoiesis, or other inflammatory phenotypes. Fifth, mortality has remained stubbornly unaffected in most analyses. Finally, the 2025 CLEAR SYNERGY/OASIS 9 trial in acute myocardial infarction added complexity to the literature by tempering the assumption that the benefits of colchicine necessarily extend to every post-MI setting or trial design (22); however, its neutral findings should not be regarded as the final word against colchicine, given important contextual limitations. Conducted during the COVID-19 pandemic, it faced several limitations. The colchicine dosing protocol was altered during the study from 0.5 mg twice daily to 0.5 mg once daily, to reduce gastrointestinal side effects, but high doses were not used in previous trials, such as LoDoCo trials and COLCOT trial for acute coronary syndromes. Outcome reporting may have suffered during the pandemic, leading to potential underreporting of MACE. Adherence was based on self-reporting, which is inherently unreliable. Additionally, the possible inclusion of hemorrhagic events in stroke events adds potential confusion. All of which may have influenced treatment effects and limit direct comparability with prior positive trials (23).

These uncertainties do not negate benefit, but they do caution against oversimplification. The most defensible clinical position today is that colchicine reduces recurrent ischaemic events in many secondary prevention patients with chronic coronary disease, but more work is needed to determine who benefits most, when to start, how long to continue, and how to integrate inflammatory biomarkers into decision-making.

Conclusions

Contemporary literature shows that inflammation is not an epiphenomenon in coronary heart disease, but a treatable driver of residual risk. CANTOS established the principle that anti-inflammatory therapy can reduce recurrent cardiovascular events independently of lipid lowering. Colchicine then emerged as the most practical anti-inflammatory candidate for widespread cardiovascular use, with COLCOT and LoDoCo2 providing the pivotal randomised evidence. Subsequent meta-analyses have generally confirmed reductions in major adverse cardiovascular events, myocardial infarction, stroke, and coronary revascularisation, especially for chronic coronary syndromes, nevertheless the mortality benefit remains unproven, and tolerability limits use in some patients.

Accordingly, colchicine should not be seen as a replacement for standard secondary prevention, but as a plausible adjunct in selected patients with acute or chronic coronary syndromes who remain at meaningful residual inflammatory risk despite contemporary therapy. This review supports the concept that colchicine may become a new pillar of secondary prevention of ischaemic heart disease, though current evidence supports a selective, evidence-informed adoption rather than uncritical universal use.

References

1. ABBATE A, TOLDO S, MARCHETTI C, KRON J, VAN TASSELL BW, DINARELLO CA: Interleukin-1 and the inflammasome as therapeutic targets in cardiovascular disease. *Circ Res* 2020;126(9): 1260-80. https://doi.org/10.1161/circresaha.120.315937
2. IMAZIO M, ABBATE A: The inflammasome as a therapeutic target for myopericardial diseases. *Minerva Cardiol Angiol* 2022; 70(2): 238-47. https://doi.org/10.23736/s2724-5683.21.05876-2
3. TAKAHASHI M: NLRP3 inflammasome as a key driver of vascular disease. *Cardiovasc Res* 2022; 118(2): 372-85. https://doi.org/10.1093/cvr/cvab010
4. RIDKER PM, EVERETT BM, THUREN T *et al.*; CANTOS TRIAL GROUP: Antiinflammatory therapy with canakinumab for atherosclerotic disease. *N Engl J Med* 2017; 377(12): 1119-31. https://doi.org/10.1056/nejmoa1707914
5. GIUBILATO S, CILIBERTI G, SCICCHITANO P *et al.*: Colchicine and atherosclerotic coronary artery disease: an updated review. *J Clin Med* 2025; 14(18): 6396. https://doi.org/10.3390/jcm14186396
6. SCHULZ-MENGER J, COLLINI V, GRÖSCHEL J *et al.*; ESC SCIENTIFIC DOCUMENT GROUP: 2025 ESC Guidelines for the management of myocarditis and pericarditis. *Eur Heart J* 2025; 46(40): 3952-4041. https://doi.org/10.1093/eurheartj/ehaf192
7. IMAZIO M, AGRIMI C, CESCON L, PANZOLLI G, COLLINI V, SINAGRA G: Colchicine for the treatment of the spectrum of cardiovascular diseases: current evidence and ongoing perspectives. *J Cardiovasc Med* (Hagerstown) 2024; 25(9): 653-63. https://doi.org/10.2459/jcm.0000000000001647
8. IMAZIO M, NIDORF M: Colchicine and the heart. *Eur Heart J* 2021; 42(28): 2745-60. https://doi.org/10.1093/eurheartj/ehab221
9. RIDKER PM, BHATT DL, PRADHAN AD *et al.*; PROMINENT, REDUCE-IT, and STRENGTH INVESTIGATORS: Inflammation and cholesterol as predictors of cardiovascular events among patients receiving statin therapy: a collaborative analysis of three randomised trials. *Lancet* 2023; 401(10384): 1293-101. https://doi.org/10.1016/S0140-6736(23)00215-5
10. NIDORF SM, EIKELBOOM JW, BUDGEON CA, THOMPSON PL: Low-dose colchicine for secondary prevention of cardiovascular disease. *J Am Coll Cardiol* 2013; 61(4): 404-10. https://doi.org/10.1016/j.jacc.2012.10.027
11. TARDIF JC, KOUZ S, WATERS DD *et al.*: Efficacy and safety of low-dose colchicine after myocardial infarction. *N Engl J Med* 2019; 381(26): 2497-505. https://doi.org/10.1056/nejmoa1912388
12. BOUABDALLAOUI N, TARDIF JC, WATERS DD *et al.*: Time-to-treatment initiation of colchicine and cardiovascular outcomes after myocardial infarction in the Colchicine Cardiovascular Outcomes Trial (COLCOT). *Eur Heart J* 2020; 41(42): 4092-99. https://doi.org/10.1093/eurheartj/ehaa659
13. NIDORF SM, FIOLET ATL, MOSTERD A *et al.*; LODOCO2 TRIAL INVESTIGATORS: Colchicine in patients with chronic coronary disease. *N Engl J Med* 2020; 383(19): 1838-47. https://doi.org/10.1056/nejmoa2021372
14. FIOLET ATL, OPSTAL TSJ, MOSTERD A *et al.*: Efficacy and safety of low-dose colchicine in patients with coronary disease: a systematic review and meta-analysis of randomized trials. *Eur Heart J* 2021; 42(28): 2765-75. https://doi.org/10.1093/eurheartj/ehab115 Erratum in: *Eur Heart J* 2021; 42(33): 3202. https://doi.org/10.1093/eurheartj/ehab265.
15. IMAZIO M, BALLACCI F, GIORDANO F: Colchicine in acute coronary syndromes: the present and the possible future. *Nat Rev Car-*

diol 2025; 22(4): 215-16. https://doi.org/10.1038/s41569-025-01124-3
16. IMAZIO M: Should we start using colchicine for secondary prevention of acute and chronic coronary syndromes after 2024 European society of cardiology guidelines? *J Cardiovasc Med* (Hagerstown) 2025; 26(1): 4-7. https://doi.org/10.2459/jcm.0000000000001688
17. TONG DC, QUINN S, NASIS A *et al*.: Colchicine in patients with acute coronary syndrome: The Australian COPS Randomized Clinical Trial. *Circulation* 2020; 142(20): 1890-900. https://doi.org/10.1161/circulationaha.120.050771
18. AKL E, SAHAMI N, LABOS C *et al*.: Meta-analysis of randomized trials: efficacy and safety of colchicine for secondary prevention of cardiovascular disease. *J Interv Cardiol* 2024; 2024: 8646351. https://doi.org/10.1155/2024/8646351
19. AHMED M, HASHMI TM, SHAFIQ A *et al*.: Efficacy and safety of colchicine in patients with coronary artery disease: an updated meta-analysis of randomized controlled trials. *Am Heart J Plus* 2025; 59: 100610. https://doi.org/10.1016/j.ahjo.2025.100610
20. ROBINSON PC, TERKELTAUB R, PILLINGER MH *et al*.: Consensus statement regarding the efficacy and safety of long-term low-dose colchicine in gout and cardiovascular disease. *Am J Med* 2022; 135(1): 32-38. https://doi.org/10.1016/j.amjmed.2021.07.025
21. VRINTS C, ANDREOTTI F, KOSKINAS KC *et al*.; ESC SCIENTIFIC DOCUMENT GROUP: 2024 ESC Guidelines for the management of chronic coronary syndromes. *Eur Heart J* 2024; 45(36): 3415-537. https://doi.org/10.1093/eurheartj/ehae17 Erratum in: *Eur Heart J* 2025; 46(16): 1565. https://doi.org/10.1093/eurheartj/ehaf079
22. JOLLY SS, D'ENTREMONT MA, LEE SF *et al*.; CLEAR INVESTIGATORS: Colchicine in acute myocardial infarction. *N Engl J Med* 2025; 392(7): 633-42. https://doi.org/10.1056/nejmoa2405922
23. IMAZIO M: Colchicine for cardiovascular prevention: clarity amidst the confusion. *Am J Cardiovasc Drugs* 2026; 26(1): 7-10. https://doi.org/10.1007/s40256-025-00754-9