Cardiovascular Reviews, 2025, Volume 3, Issue 3
http://ojs.bbwpublisher.com/index.php/CR
Online ISSN: 2981-8109
Print ISSN: 3083-4880

Colchicine and Cardiovascular Disease: Mechanisms, Clinical Evidence, and Therapeutic Perspectives

Zhiyang Lv[1], Xiaodi Chen[2], Qing Lu[3], Shifang Ding[3]*

[1]Department of Cardiology, Yichang Central People's Hospital, The First College of Clinical Medical Science, China Three Gorges University, Yichang 443003, Hubei, China
[2]Department of Ultrasound, Yichang Central People's Hospital, The First College of Clinical Medical Science, China Three Gorges University, Yichang 443003, Hubei, China
[3]Department of Cardiology, General Hospital of Central Theater Command, Wuhan Clinical Medicine College of Southern Medical University, Wuhan, 430070, Hubei, China

***Corresponding author:** *Shifang Ding, dsfmdwh@163.com*

Abstract: Colchicine, a long-established anti-inflammatory agent, has recently drawn growing interest due to its possible therapeutic roles in cardiovascular disorders. By modulating inflammatory pathways, colchicine has been shown to enhance clinical outcomes in patients with pericarditis, coronary artery involvement, as well as postoperative atrial fibrillation, and to lower the risk of recurrent cardiovascular events. Nevertheless, its underlying pharmacological mechanisms and sustained benefits still warrant comprehensive evaluation. This review summarizes current understanding of colchicine's mechanisms, clinical evidence, and prospective applications, offering updated perspectives for its use in cardiovascular medicine.

Keywords: Colchicine; Cardiovascular disease; Inflammation

Online publication: October 17, 2025

1. Introduction

Colchicine remains one of the most enduring therapeutic agents in modern medicine. Extracted from the corms of the autumn crocus (Colchicum species), it has been employed since ancient Egyptian times as a traditional preparation for alleviating articular pain [1]. Owing to its distinctive anti-inflammatory and analgesic actions, colchicine has served as a cornerstone therapy for various rheumatologic conditions, including gout, osteoarthritis, and ankylosing spondylitis. More recently, extensive research has uncovered its wide-ranging potential across oncology, immunology, dermatology, and particularly cardiology. Among these, its application in preventing and managing cardiovascular diseases has drawn increasing clinical attention. This review

outlines the pharmacological mechanisms and contemporary progress of colchicine in cardiovascular medicine, to offer novel insights into its role in cardiovascular prevention and therapy, thereby revitalizing this time-honored compound with renewed therapeutic significance.

2. Overview of colchicine

2.1. Origin and history

Colchicine, a naturally occurring alkaloid obtained from members of the Liliaceae family, was first documented in the ancient Ebers Papyrus (circa 1500 BCE) as a remedy for joint discomfort and inflammation. Even today, it continues to serve as the frontline therapeutic option for gout, familial Mediterranean fever, and their related complications. In recent decades, its pharmacological repertoire has broadened considerably, showing promising roles in oncology, immunology, dermatology, and cardiovascular medicine. For example, the compound has found therapeutic utility across cutaneous vasculitides, Paget's disease, Sweet's syndrome, and recurrent aphthous ulcers[2]. These advances have offered renewed perspectives on the modern clinical relevance of this time-honored agent.

2.2. Pharmacokinetics

Colchicine, a naturally derived alkaloid, is mainly taken up through the jejunal and ileal segments of the small intestine, exhibiting an oral bioavailability of around 30%. Following ingestion, its plasma concentration peaks within approximately 0.5 to 3 hours and subsequently decreases over the ensuing couple of hours, before showing a secondary elevation attributable to enterohepatic recirculation. The compound possesses a relatively extended half-life and can remain detectable in leukocytes for several days post-administration. Within the bloodstream, colchicine displays moderate affinity for albumin (30%–50%) and is efficiently eliminated from circulation, distributing across multiple organs—including the liver, kidneys, and spleen. It also penetrates peripheral leukocytes rapidly, where intracellular concentrations surpass plasma levels, with particularly high accumulation observed in neutrophils [3]. In addition to its interaction with tubulin, colchicine is metabolized by cytochrome P450 3A4 (CYP3A4) and transported via P-glycoprotein (P-gp). Hepatic deacetylation represents a key metabolic pathway, with an elimination half-life of about 12–30 minutes. The drug is primarily excreted through the biliary route (60%–80%) and, to a lesser extent, renally (20%–40%) [4].

2.3. Mechanisms of action

2.3.1. Anti-inflammatory mechanisms

Microtubules, composed of tubulin subunits, are fibrous structures that play a critical role in maintaining cell morphology, cell division, signal transduction, and intracellular transport. Colchicine exerts its effects by binding to tubulin, thereby preventing microtubule polymerization and disrupting their dynamic stability. This process inhibits mitosis and neutrophil migration, while also altering cell morphology, protein trafficking, and ion homeostasis [5].In addition, colchicine suppresses vesicular transport, reduces macrophage expression of TNF-α receptors, and blocks the release of pro-inflammatory mediators, chemokines, and reactive oxygen species during mast cell degranulation [6]. At the molecular level, colchicine downregulates pro-inflammatory cytokines such as interferon-γ, IL-1β, and IL-6, thereby reducing the adhesion of neutrophils to vascular endothelial cells [2,7].

2.3.2. Antifibrotic mechanisms

Fibrosis represents a shared pathological outcome in various organs such as the myocardium, liver, and kidneys. The antifibrotic potential of colchicine arises mainly from its capacity to remodel the cytoskeleton and modulate inflammatory cascades. Through suppression of IL-6 secretion triggered by IL-1β stimulation, colchicine mitigates ischemia–reperfusion injury and dampens the acute inflammatory response after myocardial infarction [5]. Additionally, it diminishes the activity of extracellular matrix–related proteins (e.g., fibronectin) and TGF-β, inhibits RhoA-dependent signaling, and consequently restrains interstitial fibrotic remodeling [8]. By curbing neutrophil infiltration and fibroblast proliferation, colchicine reduces the subsequent accumulation of collagen fibers [9]. Furthermore, it down-modulates fibrosis-associated gene transcription, such as those encoding collagen type I, collagen type III, MMP-2, and MMP-9, thereby limiting extracellular matrix deposition [10,11].

2.3.3. Anti-atherosclerotic mechanisms

Atherosclerotic cardiovascular disease arises from a combination of endothelial dysfunction, abnormalities in smooth muscle cell function, and macrophage dysregulation. Dysfunction of endothelial and smooth muscle cells not only disrupts vascular homeostasis but also activates immune cells, thereby driving lesion progression; meanwhile, neutrophils exacerbate the disease by promoting plaque instability and thrombosis [5]. The anti-atherosclerotic effects of colchicine include: Inhibiting endothelial cell expression of E-selectin, thereby preventing neutrophil adhesion and recruitment; Blocking vascular smooth muscle cell mitosis to limit their proliferation within atherosclerotic plaques [12]; Reducing the aggregation of activated neutrophils, leukocytes, and platelets, inhibiting degranulation mediated by platelet-activating factor and leukotriene B4, and lowering α-defensin levels, thereby preventing thrombus formation [5,12].

3. Advances in the application of colchicine in cardiovascular diseases

3.1. Pericardial diseases

3.1.1. Pericarditis

Acute pericarditis may lead to acute complications and carries a risk of recurrence. Therefore, reducing pericardial inflammation and delaying disease progression have become important clinical goals. By suppressing systemic inflammatory responses, colchicine holds promise as an effective therapeutic strategy to mitigate the progression of acute pericarditis.

The therapeutic use of colchicine in recurrent pericarditis was first reported by Rodríguez et al. (1987), who observed a marked decline in relapse frequency without significant toxicity—an observation that established its clinical relevance in pericardial disorders [13]. Later, Imazio et al. (2013) conducted a prospective, placebo-controlled, double-blind randomized study to evaluate colchicine's preventive efficacy against acute pericarditis recurrence. In that trial involving 240 participants assigned to either colchicine or placebo, those receiving colchicine exhibited substantially lower rates of persistent symptoms at 72 hours, as well as reduced recurrence and hospitalization frequencies [14].

Furthermore, a meta-analysis confirmed that colchicine shortened symptom duration within the initial 72 hours among individuals experiencing recurrent pericarditis (ARR = 0.30; 95% CI 0.13–0.45) and decreased relapse rates over an 18-month follow-up period (ARR = 0.31; 95% CI 0.13–0.46). Collectively, these data support the safety and therapeutic efficacy of colchicine in both acute and recurrent pericarditis, underscoring

its importance in primary and secondary prevention strategies [15].

Consistent with these findings, the 2015 European Society of Cardiology (ESC) guidelines recommend colchicine as a preferred initial treatment for acute and recurrent pericarditis, to be administered alongside aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs) as part of combination therapy [16].

3.1.2. Postpericardiotomy syndrome

Postpericardiotomy syndrome (PPS) represents a frequent postoperative inflammatory condition observed after cardiac surgery, occurring in approximately 10–40% of patients. Its manifestations range from transient, self-limiting symptoms to severe hemodynamic compromise, occasionally progressing to cardiac tamponade or even fatal outcomes. At present, aspirin remains the preferred initial treatment option, whereas indomethacin and corticosteroids are typically reserved for recurrent or treatment-resistant cases. Over the past decade, increasing evidence has highlighted colchicine's potential role in both the prevention and management of PPS.

Imazio et al.(2012) conducted a prospective, double-blind randomized trial including 360 individuals scheduled for cardiac surgery. Their findings indicated that initiating colchicine (0.5 mg twice daily)48–72 hours before the procedure significantly reduced the occurrence of PPS (RR = 10.0%, 95% CI: 1.1%–18.7%) [17].

More recently, Pan et al. (2023) reported outcomes from a randomized controlled investigation showing that, within 48 hours postoperatively, patients receiving low-dose colchicine exhibited markedly decreased concentrations of myocardial injury markers—including troponin T, troponin I, and creatine kinase-MB—as well as lower IL-6 levels compared with placebo ($P < 0.01$). Furthermore, the overall incidence of PPS was substantially lower (3.08% vs. 17.7%, $P < 0.01$). These results collectively indicate that early administration of low-dose colchicine after surgery can effectively reduce myocardial damage and inflammatory activation, thereby diminishing the risk of PPS development [18].

3.1.3. Pericardial effusion

Meurin et al. (2015) carried out a multicenter randomized clinical investigation with blinded treatment allocation, involving 197 high-risk patients across ten French institutions. Participants assigned to the intervention arm received colchicine (1 mg daily) for 14 days, whereas those in the comparator arm were administered a placebo. The analysis revealed no statistically significant difference between groups regarding pericardial effusion volume or the occurrence of cardiac tamponade. The mean change in effusion size was −0.19 (95% CI: −0.55 to −0.16, $P = 0.23$), suggesting that colchicine was ineffective in limiting effusion accumulation or in averting delayed tamponade events [19].

Subsequently, Kim et al. (2020) examined anti-inflammatory therapy in malignant pericardial effusion following pericardiocentesis, enrolling 445 individuals. Their results indicated that colchicine markedly lowered the risk of composite adverse outcomes, including mortality and repeat drainage or surgical intervention (adjusted HR 0.65, $P = 0.003$), as well as overall death (adjusted HR 0.60, $P = 0.001$). These findings imply that post-pericardiocentesis colchicine therapy confers significant prognostic benefit [20].

Future research should further clarify the clinical role and therapeutic impact of colchicine among individuals presenting with pericardial effusion of diverse causes and stages, focusing on defining the most appropriate dose, initiation timing, and treatment duration, as well as its integration with adjunctive regimens to refine management strategies for pericardial effusion.

3.2. Coronary atherosclerotic heart disease

3.2.1. Stable coronary artery disease

Inflammation is a key component in the development of atherosclerosis. Among patients receiving statin therapy, vulnerable coronary plaques and inflammatory markers have been shown to predict major adverse cardiovascular events (MACE) more effectively than low-density lipoprotein cholesterol levels [21]. Therefore, the use of anti-inflammatory agents may contribute to improved cardiovascular outcomes.

The CANTOS trial [22] investigated the cardioprotective effects of canakinumab, a human monoclonal antibody targeting IL-1β. Canakinumab effectively reduced levels of inflammatory biomarkers without significantly affecting lipid profiles or platelet function. Administered at a dose of 150 mg every three months, it significantly lowered the recurrence of cardiovascular events, providing the first evidence that targeting inflammatory pathways can reduce the risk of cardiovascular events.

Nidorf et al. (2013) conducted the LoDoCo study, which was the first to evaluate the use of low-dose colchicine in patients with stable coronary artery disease. A total of 532 patients were enrolled and treated with colchicine (0.5 mg twice daily). The study demonstrated that colchicine effectively prevented the occurrence of cardiac arrest and non-cardioembolic ischemic stroke, potentially through the inhibition of neutrophil function. These findings suggest that low-dose colchicine provides a preventive benefit against cardiovascular events in patients with stable coronary artery disease [23].

The LoDoCo2 trial further validated the efficacy of colchicine in patients with chronic coronary artery disease. This study enrolled 5,522 patients who were randomly assigned to receive either colchicine or a placebo. The results demonstrated that colchicine significantly reduced the risk of cardiovascular events, with effects consistent with those of previous anti-inflammatory therapies and other secondary prevention strategies. Notably, the benefits emerged early and continued to accumulate over time [24].

Currently, both U.S. and European guidelines recommend colchicine as an anti-inflammatory therapy for coronary artery disease [25, 26]; however, its clinical adoption remains lower compared with lipid-lowering therapy. In 2023, the U.S. FDA approved colchicine (Lodoco, 0.5 mg tablets) for the treatment of atherosclerotic cardiovascular disease, marking it as the first anti-inflammatory drug approved by the FDA for cardiovascular indications. The COLPCI trial [27], currently underway in China, aims to evaluate the efficacy and safety of different doses of colchicine in reducing cardiovascular events among patients undergoing PCI, and its results are highly anticipated.

3.2.2. Acute coronary syndrome

In acute coronary syndrome (ACS), the rupture or erosion of vulnerable plaques triggers intense local inflammation and neutrophil infiltration, accelerating plaque destabilization. Persistent inflammatory activity is a major determinant of post–myocardial infarction outcomes, and residual inflammatory burden has been strongly correlated with recurrent cardiovascular events [28,29]. Evidence suggests that a once-daily regimen of 0.5 mg colchicine may enhance plaque stabilization and dampen inflammatory signaling among individuals presenting with ACS. Kaivan et al. (2018) observed that colchicine improved coronary plaque morphology independently of intensive statin therapy [30]. In contrast, Jennife et al. (2020) reported modulation of specific microRNAs that could serve as novel therapeutic biomarkers [31].

Unlike earlier investigations that failed to demonstrate meaningful cardiovascular benefit [32], the COLCOT

trial found that colchicine therapy led to a significant reduction in cardiovascular event rates [33]. Furthermore, Yu et al. (2024), in the COLOCT trial, provided the first imaging-based evidence that colchicine increased fibrous cap thickness and diminished macrophage infiltration via anti-inflammatory mechanisms, thereby reinforcing non-culprit plaque stability and lowering the likelihood of recurrent adverse cardiac outcomes, particularly among patients with optimally managed LDL-C concentrations [34].

Taken together, current findings highlight colchicine's therapeutic promise in ACS; nevertheless, its clinical efficacy and optimal treatment parameters warrant further confirmation through large-scale trials.

3.3. Atrial fibrillation

Atrial fibrillation (AF) represents the most prevalent sustained cardiac arrhythmia, and its postoperative form frequently complicates the course of patients following cardiac surgery. The pathogenesis is believed to involve pericardial inflammation, autonomic dysregulation, and heightened catecholamine activity [17]. AF represents the most prevalent sustained cardiac arrhythmia, and its postoperative form frequently complicates the course of patients following cardiac surgery. The pathogenesis is believed to involve pericardial inflammation, autonomic dysregulation, and heightened catecholamine activity [35]. Wu et al. (2020) reported that colchicine shortened AF duration and reduced its occurrence through suppression of inflammatory cascades and attenuation of atrial fibrotic remodeling [10]. Moreover, a meta-analysis revealed that a low-dose colchicine regimen markedly decreased the incidence of postoperative AF among patients undergoing coronary artery bypass graft surgery [36]. Further research is warranted to clarify its prophylactic value and define the optimal timing and duration of administration.

3.4. Heart failure

Chronic heart failure represents the end stage of cardiovascular pathology and is characterized by progressive loss of cardiomyocytes and compromised myocardial contractility. Crosstalk between pro-inflammatory signaling, apoptotic pathways, and myocardial dysfunction drives disease progression, while targeting inflammation may help attenuate this process and improve long-term outcomes. Spyridon et al. (2014) reported that, although colchicine administration led to a marked reduction in circulating inflammatory mediators, including C-reactive protein (CRP) and interleukin-6 (IL-6) [37], no significant improvements were observed in functional capacity, survival, or hospital stay. Likewise, Pascual et al. (2024) observed that despite lowering systemic inflammatory activity, colchicine did not substantially influence the incidence of acute decompensation or worsening heart failure events [38].

3.5. Adverse cardiovascular events after revascularization

Revascularization procedures, including percutaneous coronary intervention (PCI) and coronary artery bypass grafting, are often accompanied by postoperative complications such as restenosis, myocardial infarction, or arrhythmias, all of which continue to pose considerable clinical challenges. Endothelial trauma and subsequent inflammatory activation play central roles in these adverse outcomes, and accumulating evidence suggests that colchicine may alleviate such inflammatory sequelae. Shah and colleagues observed that administering colchicine prior to surgery attenuated postoperative increases in circulating inflammatory mediators—specifically IL-6 and CRP—although no substantial reduction in myocardial injury was detected [39]. Conversely, Giannopoulos and co-workers found evidence supporting a

modest protective effect of colchicine against postoperative myocardial damage [40]. In a comprehensive meta-analysis, Fu et al. (2021) concluded that colchicine therapy was associated with a lower rate of major adverse cardiovascular events (MACE) among patients undergoing PCI, while having no statistically significant influence on mortality, infarction recurrence, or restenosis [41]. Taken together, these findings indicate that colchicine shows promise as a prophylactic anti-inflammatory agent in mitigating post-revascularization cardiovascular complications, though its therapeutic efficacy requires confirmation in larger, rigorously designed clinical trials.

4. Safety and tolerability

Colchicine is an ancient drug that, despite its widespread use, may cause adverse events when overdosed due to its mitotic toxicity. In a large randomized controlled trial including 8,659 patients, 17.9% of those receiving colchicine experienced diarrhea, 1.9% had hepatic adverse events, and 4.2% developed myotoxicity, while no cases of neuropathy or death were reported [42]. In another trial involving 14,188 patients, the incidence of adverse events was slightly higher in the colchicine group (15.3% vs. 13.9%), with a significant increase in gastrointestinal events (16.1% vs. 12.2%), particularly diarrhea (12.5% vs. 8.1%); however, no significant differences were observed for other adverse outcomes [43] A systematic review found no significant association between colchicine use and infectious adverse events [44], and long-term therapy did not increase the risk of sepsis or cancer [45]. Overall, colchicine has a favorable safety profile, but individualized dose adjustment, close monitoring of hepatic and renal function, and vigilance against overdose remain essential.

5. Summary and outlook

Colchicine, a long-established therapeutic agent with deep roots in traditional medicine, has undergone a remarkable resurgence in clinical relevance over the past decade. Originally utilized for disorders such as gout and familial Mediterranean fever, its medical scope has progressively expanded to include numerous systemic conditions, most notably cardiovascular pathologies. Currently, both its safety characteristics and diverse pharmacological actions are well substantiated, underscoring its growing importance in cardiovascular therapeutics. Owing to its accessibility, affordability, and convenient oral administration, colchicine possesses the potential to significantly influence global cardiovascular care, particularly within resource-limited regions. As ongoing research continues to elucidate its mechanisms and broaden clinical understanding, future advancements are likely to further extend the therapeutic landscape of colchicine in cardiovascular medicine.

Disclosure statement

The authors declare no conflict of interest.

References

[1] Nerlekar N, Beale A, Harper R, 2014, Colchicine – A Short History of an Ancient Drug. Med J Aust, 201(11): 687–688.

[2] Dasgeb B, Kornreich D, Mcguinn K, et al., 2018, Colchicine: An Ancient Drug With Novel Applications. Br J Dermatol, 178(2): 350–356.

[3] Niel E, Scherrmann J, 2006, Colchicine Today. Joint Bone Spine, 73(6): 672–678.

[4] Dixon D, Patel J, Spence R, et al., 2022, Select Drug–Drug Interactions With Colchicine and Cardiovascular Medications: A Review. Am Heart J, 252: 42–50.

[5] Akodad M, Sicard P, Fauconnier J, et al., 2020, Colchicine and Myocardial Infarction: A Review. Arch Cardiovasc Dis, 113(10): 652–659.

[6] Reyes A, Hu K, Teperman J, et al., 2021, Anti-Inflammatory Therapy for COVID-19 Infection: The Case for Colchicine. Ann Rheum Dis, 80(5): 550–557.

[7] Siak J, Flint N, Shmueli H, et al., 2021, The Use of Colchicine in Cardiovascular Diseases: A Systematic Review. Am J Med, 134(6): 735–744.e1.

[8] Guan T, Gao B, Chen G, et al., 2013, Colchicine Attenuates Renal Injury in a Model of Hypertensive Chronic Kidney Disease. Am J Physiol Renal Physiol, 305(10): F1466–F1476.

[9] Entzian P, Schlaak M, Seitzer U, et al., 1997, Antiinflammatory and Antifibrotic Properties of Colchicine: Implications for Idiopathic Pulmonary Fibrosis. Lung, 175(1): 41–51.

[10] Wu Q, Liu H, Liao J, et al., 2020, Colchicine Prevents Atrial Fibrillation Promotion by Inhibiting IL-1β-Induced IL-6 Release and Atrial Fibrosis in the Rat Sterile Pericarditis Model. Biomed Pharmacother, 129: 110384.

[11] Lv Z, Chen X, Chen P, et al., 2022, Colchicine Prevents Ventricular Arrhythmias Vulnerability in Diet-Induced Obesity Rats. Biochemical and Biophysical Research Communications, 610: 127–132.

[12] Cavallero C, Turolla E, Ricevuti G, 1971, Cell Proliferation in the Atherosclerotic Plaques of Cholesterol-Fed Rabbits. 1. Colchicine and (3H)Thymidine Studies. Atherosclerosis, 13(1): 9–20.

[13] Rodríguez A, Guindo J, Martí V, et al., 1987, Colchicine for Recurrent Pericarditis. Lancet, 2(8574): 1517.

[14] Imazio M, Brucato A, Cemin R, et al., 2013, A Randomized Trial of Colchicine for Acute Pericarditis. N Engl J Med, 369(16): 1522–1528.

[15] Imazio M, Brucato A, Cemin R, et al., 2011, Colchicine for Recurrent Pericarditis (CORP): A Randomized Trial. Ann Intern Med, 155(7): 409–414.

[16] Adler Y, Charron P, Imazio M, et al., 2015, 2015 ESC Guidelines for the Diagnosis and Management of Pericardial Diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) Endorsed by the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J, 36(42): 2921–2964.

[17] Imazio M, Brucato A, Ferrazzi P, et al., 2014, Colchicine for Prevention of Postpericardiotomy Syndrome and Postoperative Atrial Fibrillation: The COPPS-2 Randomized Clinical Trial. JAMA, 312(10): 1016–1023.

[18] Pan T, Jiang C, Zhang H, et al., 2023, The Low-Dose Colchicine in Patients After Non-CABG Cardiac Surgery: A Randomized Controlled Trial. Crit Care, 27(1): 49.

[19] Meurin P, Lelay-Kubas S, Pierre B, et al., 2015, Colchicine for Postoperative Pericardial Effusion: A Multicentre, Double-Blind, Randomised Controlled Trial. Heart, 101(21): 1711–1716.

[20] Kim S, Kim E, Cho J, et al., 2020, Effect of Anti-Inflammatory Drugs on Clinical Outcomes in Patients With Malignant Pericardial Effusion. J Am Coll Cardiol, 76(13): 1551–1561.

[21] Dai N, Hu Y, Tang X, et al., 2025, Cholesterol, Atherosclerotic, and Inflammatory Risks Among Patients With Statin Therapy. JACC Cardiovasc Imaging, 18(2): 242–244.

[22] Ridker P, Everett B, Thuren T, et al., 2017, Antiinflammatory Therapy With Canakinumab for Atherosclerotic Disease. N Engl J Med, 377(12): 1119–1131.

[23] Nidorf S, Eikelboom J, Budgeon C, et al., 2013, Low-Dose Colchicine for Secondary Prevention of Cardiovascular Disease. J Am Coll Cardiol, 61(4): 404–410.

[24] Nidorf S, Fiolet A, Mosterd A, et al., 2020, Colchicine in Patients With Chronic Coronary Disease. N Engl J Med, 383(19): 1838–1847.

[25] Virani S, Newby L, Arnold S, et al., 2023, 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients With Chronic Coronary Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. Circulation, 148(9): e9–e119.

[26] Vrints C, Andreotti F, Koskinas K C, et al., 2024, 2024 ESC Guidelines for the Management of Chronic Coronary Syndromes: Developed by the Task Force for the Management of Chronic Coronary Syndromes of the European Society of Cardiology (ESC) Endorsed by the European Association for Cardio-Thoracic Surgery (EACTS). European Heart Journal, 45(36): 3415–3537.

[27] ICHGCP, 2025, Efficacy and Safety of Colchicine After PCI, visited on July 5, 2025, https://ichgcp.net/clinical-trials-registry/NCT06472908.

[28] Mewton N, Roubille F, Bresson D, et al., 2021, Effect of Colchicine on Myocardial Injury in Acute Myocardial Infarction. Circulation, 144(11): 859–869.

[29] Ridker P, Bhatt D, Pradhan A, et al., 2023, Inflammation and Cholesterol as Predictors of Cardiovascular Events Among Patients Receiving Statin Therapy: A Collaborative Analysis of Three Randomised Trials. Lancet, 401(10384): 1293–1301.

[30] Vaidya K, Arnott C, Martínez G, et al., 2018, Colchicine Therapy and Plaque Stabilization in Patients With Acute Coronary Syndrome: A CT Coronary Angiography Study. JACC Cardiovasc Imaging, 11(2 Pt 2): 305–316.

[31] Barraclough J, Joglekar M, Januszewski A, et al., 2020, A MicroRNA Signature in Acute Coronary Syndrome Patients and Modulation by Colchicine. J Cardiovasc Pharmacol Ther, 25(5): 444–455.

[32] Tong D, Quinn S, Nasis A, et al., 2020, Colchicine in Patients With Acute Coronary Syndrome: The Australian COPS Randomized Clinical Trial. Circulation, 142(20): 1890–1900.

[33] Tardif J, Kouz S, Waters D, et al., 2019, Efficacy and Safety of Low-Dose Colchicine After Myocardial Infarction. N Engl J Med, 381(26): 2497–2505.

[34] Yu M, Yang Y, Dong S, et al., 2024, Effect of Colchicine on Coronary Plaque Stability in Acute Coronary Syndrome as Assessed by Optical Coherence Tomography: The COLOCT Randomized Clinical Trial. Circulation, 150(13): 981–993.

[35] Ho K, Tan J, 2009, Benefits and Risks of Corticosteroid Prophylaxis in Adult Cardiac Surgery: A Dose-Response Meta-Analysis. Circulation, 119(14): 1853–1866.

[36] Kirov H, Caldonazo T, Runkel A, et al., 2024, Colchicine in Patients With Coronary Disease Who Underwent Coronary Artery Bypass Surgery: A Meta-Analysis of Randomized Controlled Trials. Am J Cardiol, 231: 48–54.

[37] Deftereos S, Giannopoulos G, Panagopoulou V, et al., 2014, Anti-Inflammatory Treatment With Colchicine in Stable Chronic Heart Failure: A Prospective, Randomized Study. JACC Heart Fail, 2(2): 131–137.

[38] Pascual-Figal D, Núñez J, Pérez-Martínez M, et al., 2024, Colchicine in Acutely Decompensated Heart Failure: The COLICA Trial. European Heart Journal, 45(45): 4826–4836.

[39] Shah B, Pillinger M, Zhong H, et al., 2020, Effects of Acute Colchicine Administration Prior to Percutaneous Coronary Intervention: COLCHICINE-PCI Randomized Trial. Circ Cardiovasc Interv, 13(4): e008717.

[40] Giannopoulos G, Angelidis C, Kouritas V, et al., 2015, Usefulness of Colchicine to Reduce Perioperative Myocardial Damage in Patients Who Underwent On-Pump Coronary Artery Bypass Grafting. Am J Cardiol, 115(10): 1376–1381.

[41] Fu C, Wang B, 2021, Colchicine Administration for Percutaneous Coronary Intervention: A Meta-Analysis of Randomized Controlled Trials. Am J Emerg Med, 46: 121–125.

[42] Stewart S, Yang K, Atkins K, et al., 2020, Adverse Events During Oral Colchicine Use: A Systematic Review and Meta-Analysis of Randomised Controlled Trials. Arthritis Res Ther, 22(1): 28.

[43] Andreis A, Imazio M, Avondo S, et al., 2021, Adverse Events of Colchicine for Cardiovascular Diseases: A Comprehensive Meta-Analysis of 14,188 Patients From 21 Randomized Controlled Trials. J Cardiovasc Med (Hagerstown), 22(8): 637–644.

[44] Mcewan T, Robinson P, 2021, A Systematic Review of the Infectious Complications of Colchicine and the Use of Colchicine to Treat Infections. Semin Arthritis Rheum, 51(1): 101–112.

[45] Robinson P, Terkeltaub R, Pillinger M, et al., 2022, Consensus Statement Regarding the Efficacy and Safety of Long-Term Low-Dose Colchicine in Gout and Cardiovascular Disease. Am J Med, 135(1): 32–38.