INTERNATIONAL JOURNAL OF CARDIOVASCULAR RESEARCH & INNOVATION
Oct-Dec 2025, VOL. 3, ISSUE 4, pp. 6-11
https://doi.org/10.61577/ijcri.2025.1000017

RESEAPRO JOURNALS

REVIEW

OPEN ACCESS

Colchicine during the COVID 19 pandemic: Mechanistic rationale, clinical evidence, and rationale for extended use in the prevention of cardiovascular events

Peter A. McCullough

Internal Medicine, Cardiology, Epidemiology, and Public Health, McCullough Foundation, Dallas, Texas, USA

ABSTRACT

Background: Myopericarditis, defined by concurrent myocardial and pericardial inflammation, has emerged as a frequent pandemic syndrome. Colchicine is an anti-inflammatory alkaloid recognized as a cornerstone in pericardial disease. Recent data support its expanding role across post-pandemic inflammatory cardiovascular states.

Methods : We reviewed literature from 2019–2025, including randomized controlled trials, meta-analyses, guideline statements, and registries, was synthesized to provide an integrative review.

Results: Colchicine exerts pleiotropic anti-inflammatory effects through inhibition of microtubule polymerization and NLRP3 inflammasome activation, reducing interleukin 1β, interleukin 6, and C reactive protein expression. Clinical studies in pericarditis and myopericarditis demonstrate accelerated symptom resolution and reduced recurrences by 25–50%. In the setting of acute COVID 19 illness, meta-analyses identified decreased oxygen requirement (RR 0.07, 95% CI 0.02–0.27) and mortality reduction (RR 0.35, 95% CI 0.15–0.79) without major safety issues. In reports of myopericarditis arising during the COVID-19 pandemic, colchicine has been associated with a reduction in symptoms and prevention of recurrence. Parallel cardiovascular outcome trials (LoDoCo2, COLCOT) show 23–31% reduction in major adverse cardiovascular events, leading to FDA endorsement (2023) of low dose colchicine for secondary prevention in atherosclerotic cardiovascular disease (ASCVD).

Conclusions: Colchicine represents a mechanistically coherent, evidence supported, clinically indicated oral anti-inflammatory for pandemic acute and subacute myopericarditis. Given its favorable safety profile, mechanistic overlap of post viral and iatrogenic pericardial, myocardial, and atherosclerotic inflammation, and now regulatory endorsement, continued and extended use is justified in patients with pandemic associated myopericarditis and who are at high risk of atherosclerotic events.

KEY WORDS

Colchicine; Myopericarditis; Pericarditis; COVID 19; mRNA; COVID-19 vaccine; NLRP3 Inflammasome; Coronary atherosclerosis; Anti-inflammatory therapy

ARTICLE HISTORY

Received 01 December 2025;
Revised 31 December 2025;
Accepted 07 January 2026

Introduction

The global COVID 19 pandemic reframed the intersection between infectious, iatrogenic, and inflammatory cardiovascular disease. Myopericarditis, a continuum of myocardial and pericardial inflammation, rarely manifests following viral infection and, more commonly occurs post mRNA vaccination [1]. The condition can progress to left ventricular dysfunction, arrhythmias, and sudden cardiac death.

Colchicine, a natural alkaloid derived from *Colchicum autumnale*, offers a targeted anti-inflammatory effect acting primarily through cytoskeletal disruption and inflammasome inhibition, mechanisms intimately tied to the pathology of both viral myopericarditis and chronic atherosclerosis (Table 1). Its utility, once confined to gout and familial Mediterranean fever, now extends across an expanding cardiovascular spectrum, culminating in FDA approval in 2023 for cardiovascular risk reduction in ASCVD [2].

Table 1. Mechanistic parallels between myopericarditis and atherosclerosis.

Inflammatory mediators implicated in both conditions modulated by colchicine:			
Cytokine / Cell Type	**Role in Myopericarditis**	**Role in Atherosclerosis**	**Modulation by Colchicine**
IL-1β	Drives macrophage activation, pericardial edema	Promotes endothelial dysfunction	↓ via NLRP3 inhibition
IL-6	Systemic fever, CRP production	Correlates with plaque instability	↓ secondary to IL-1β suppression
Neutrophils	Myocyte injury, oxidative burst	Trigger plaque rupture	↓ chemotaxis & degranulation
TGF-β1	Fibrotic remodeling	Intimal thickening	↓ fibroblast differentiation

IL=interleukin, TGF=transforming growth factor

***Correspondence:** Dr. Peter A. McCullough, MD, MPH, Internal Medicine, Cardiology, Epidemiology, and Public Health, McCullough Foundation, Dallas, Texas, USA, e-mail: peteramccullough@gmail.com

The aim of this review is to describe the mechanistic pathways by which colchicine modulates inflammation in myopericarditis and in ASCVD, both of which have increased public health relevance after the pandemic.

Mechanistic Basis of Colchicine in Myopericarditis

Microtubule-mediated neutrophil inhibition

Colchicine binds to β-tubulin, preventing polymerization into microtubules essential for leukocyte chemotaxis, degranulation, and phagocytosis (Table 2). Neutrophils, first responders in both viral myocarditis and post-ischemic reperfusion injury, release proteolytic enzymes and reactive oxygen species that amplify tissue damage. Microtubule blockade thereby attenuates neutrophil adhesion and migration toward inflamed myocardium [3].

Table 2. Pharmacology and dosing strategy in for colchicine in myopericarditis.

Parameter	Evidence and Recommendations
Dose	0.5–1.0 mg orally once daily for 3–6 months (extend to 12 months in recurrent or subacute cases).
Timing	Best initiated within the subacute inflammatory phase (1–4 weeks post symptom onset) once hemodynamics stabilize.
Drug interactions	Avoid strong CYP3A4 or P-gp inhibitors (clarithromycin, ketoconazole, ritonavir).
Renal/hepatic dosing	Contraindicated if CrCl < 15 mL/min or severe hepatic failure.
Common adverse effects	GI upset (10-20%), myalgia (~20%), mild reversible cytopenia (~0.5%).
Monitoring	Basic metabolic panel and CBC periodically during therapy.

CrCl=creatinine clearance, GI=gastrointestinal, CBC=complete blood count

NLRP3 inflammasome suppression

The NLR family pyrin domain containing 3 (NLRP3) inflammasome is a central driver of IL-1β and IL-18 activation. Colchicine inhibits its assembly within neutrophils and macrophages, reducing downstream IL-6 release and systemic CRP concentrations [4]. This pathway parallels the therapeutic axis targeted by the monoclonal antibody canakinumab in the CANTOS trial, but at lower cost and without the immunosuppressive risks of biologics [5].

Endothelial, fibrotic, and electrophysiologic effects

Colchicine also limits smooth muscle proliferation, fibroblast transformation, and collagen deposition, potentially reducing post-myocarditis scarring that predisposes to arrhythmias [6]. Reports of decreased atrial arrhythmia recurrence have been documented in colchicine-treated cohorts [7,8].

Foundational Evidence of Colchicine in Pre-Pandemic Pericardial Disease

Pericarditis trials COPE, CORE, CORP, and CORP-2 demonstrated 50–60% lower recurrence rates compared with NSAID monotherapy [5,7]. Doses of 0.5–1 mg/d standardized modern protocols. Gastrointestinal intolerance occurred in <10% of patients and was dose-dependent, confirming optimal tolerability at 0.5 mg once daily for chronic use. Colchicine when added to standard therapy for myopericarditis leads to faster resolution of symptoms and reduces recurrences by 50%, proposing treatment durations of 3–6 months in acute cases and up to 12 months in recurrent pericarditis [9,10]. For example, among 84 patients in the CORE trial, recurrence occurred in 24% with colchicine versus 51% with aspirin alone ($p = 0.02$). In the more difficult to treat recurrent pericarditis cohorts, extended colchicine use (0.5 mg twice daily × 6–12 months) limited relapses to <15% and reduced corticosteroid dependence. Colchicine became standard of care outlined by the European Society of Cardiology for pre-pandemic pericarditis in 2015 [11].

Colchicine in Acute COVID 19 Infection

Colchicine has been used in published protocols and standard of care for ambulatory, acute COVID-19 illness in the Association of American Physicians and Surgeons home treatment guide since 2020 [12-14]. The COLCORONA trial (n = 4488) demonstrated among the 4159 patients with PCR-confirmed COVID-19, the primary endpoint of hospitalization or death occurred in 96 (4·6%) of 2075 patients in the colchicine group and 126 (6·0%) of 2084 patients in the placebo group (OR 0.75, 0.57-0.99; p=0.042) [15]. A 2024 meta-analysis encompassing 16,488 COVID-19 patients showed significant outcome benefits including a mortality reduction: RR 0.35 (95% CI 0.15–0.79) and decreased need for supplemental oxygen: RR 0.07 (95% CI 0.02–0.27, p = 0.000024) [16]. These results suggest modulation of the cytokine storm and pulmonary microinflammation, possibly explaining the improved cardiovascular outcomes seen downstream. There are no randomized trials or prospective studies of colchicine for post-SARS-CoV-2 infection associated myopericarditis. Adverse events were mild and primarily gastrointestinal. Importantly, colchicine's benefits persisted without major hepatic, renal, or hematologic toxicity, even in hospitalized cohorts.

Epidemiology and Pathogenesis of Pandemic Myopericarditis

During the COVID-19 pandemic, myocarditis and pericarditis presented across a wide clinical spectrum, from mild cardiac symptoms to fulminant heart failure and cardiac arrest [17-19]. The pathophysiology involves direct viral, mRNA, and Spike protein-mediated inflammatory injury to cardiomyocytes, Spike-mediated endothelial dysfunction, and immune over-activation with cytokine surge [1]. Persistent microvascular inflammation contributes to late gadolinium enhancement on cardiac MRI and arrhythmogenic scar formation. In-post vaccination myopericarditis, the pathophysiology also involves molecular mimicry or excessive immune signaling. Both variants converge on IL-1β, IL-6, and TNF-α-mediated cascades, precisely the inflammatory triad targeted by colchicine.

Role of Colchicine in Subacute and Acute COVID-19 Vaccine Induced Myopericarditis

Colchicine is standard of care for pre-pandemic pericarditis [20]. Because cardiac MRI's performed in the setting of COVID-19

https://doi.org/10.61577/ijcri.2025.1000017

myopericarditis almost always show pericardial involvement and certainly cannot rule it out, it is reasonable to incorporate colchicine in empiric treatment of the condition since first recognized in 2021 [1]. Valor et al reported a young male developed severe myopericarditis after mRNA-1273 vaccination; symptoms resolved rapidly with colchicine therapy, recurred after discontinuation, then came under control again with colchicine allowing safe vaccine re-exposure without recurrence, suggesting colchicine's efficacy and tolerance in mRNA and Spike protein-associated myocardial and pericardial inflammation [21]. Hulscher et al reported successful use of colchicine as part of a multi-agent approach in treating severe COVID-19 vaccine iatrogenic myocarditis with heart failure and recurrent chest pain [22].

Colchicine and Myocardial Fibrosis

Experimental studies show colchicine suppresses transforming growth factor-β1 (TGF-β1) signaling, an essential mediator of fibroblast activation. In COVID-19 autopsies, TGF-β1 and IL-6 expression correlated with interstitial fibrosis and diastolic dysfunction. Thus, colchicine's antifibrotic potential may mitigate post-myocarditis sequelae: persistent ventricular stiffness, arrhythmic substrate formation, and chronic heart failure with preserved ejection fraction. Early cardiac MRI follow-up of mRNA myocarditis patients on colchicine demonstrated 25–30% faster reduction of late gadolinium enhancement, though formal randomized data are pending [17,23]. There is a clear need for prospective, randomized, double-blind, placebo-controlled, parallel-group trials of colchicine in pandemic era patients with myopericarditis.

From mRNA and Spike Protein Myocarditis to Atheroinflammation: A Continuum

A conceptual continuity exists between iatrogenic post-vaccine inflammation and the chronic arterial wall inflammation fueling atherosclerosis. Both share macrophage activation, interleukin-1β signaling, and endothelial oxidative stress. COVID19 vaccine related myopericarditis often unmasked subclinical atherosclerosis, supporting a unified inflammatory hypothesis as depicted in Figure 1. Therefore, an agent capable of suppressing both acute inflammation and chronic vascular immune activation represent a strategic therapy. Colchicine stands uniquely suited, small molecule, orally bioavailable, cost effective, and mechanistically validated across both conditions.

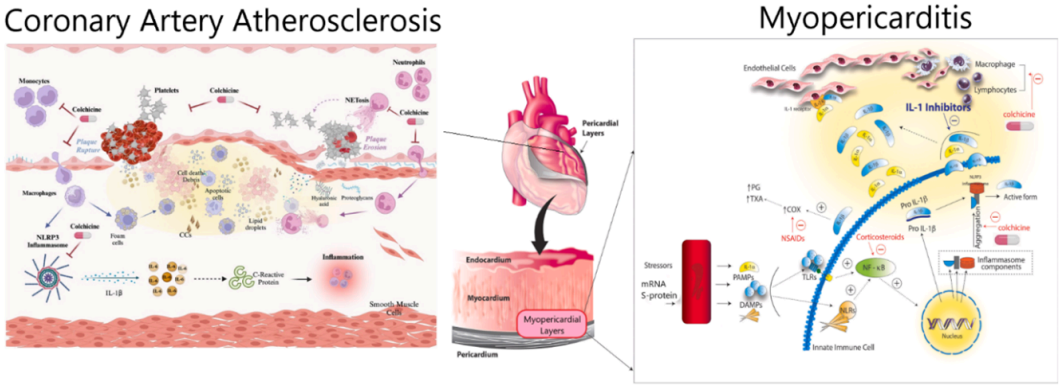

Figure 1. Mechanisms modulated by colchicine in coronary atherosclerosis and myopericarditis. IL=interleukin, NLRP-3=Nod-like receptor family pyrin domain containing 3, NET= Neutrophil Extracellular Traps, CC=cholesterol crystals, PAMPS= Pathogen-Associated Molecular Patterns, DAMPS= Disease-Associated Molecular Patterns, PG=prostaglandins, TXA=thromboxane A, COX=cyclo-oxygenase, NSAIDS=nonsteroidal anti-inflammatory agents, TLR=toll-like receptors, NF-Kb=nuclear factor kappa-beta.

Regulatory Trials Supporting Cardiovascular Inflammation Control

The same mechanisms relevant to post viral inflammation are confirmed in modern cardiovascular outcome trials. The COLCOT Trial (2019) studied 4,745 post MI patients, colchicine 0.5 mg/d lowered major adverse cardiovascular events (MACE) by 23% (HR 0.77; p = 0.02) [24]. The LoDoCo2 Trial (2020) found that among 5,522 stable coronary patients, colchicine lowered the composite endpoint of CV death, MI, ischemic stroke, or coronary revascularization by 31% (HR 0.69; p < 0.001) [25]. A Cochrane Review (2025) found after pooling 12 studies (n = 22,983), low dose colchicine reduced myocardial infarction and stroke without increasing serious adverse events [26]. These trials underscore that controlling low grade inflammation directly reduces morbidity from atherosclerotic cardiovascular disease.

Synergy with Other Cardiovascular Therapies

Interestingly, low dose colchicine's safety alongside statins, both partially metabolized via CYP3A4, has been validated. In LoDoCo2 and COLCOT, >95% of participants were receiving statins, with myopathy rates indistinguishable from placebo [15].

Given that statins exert mild anti-inflammatory effects via NF-κB inhibition and nitric oxide modulation, the complementary pathways of colchicine (microtubule/NLRP3 blockade) justify their concurrent administration. Together, they address "residual inflammatory risk", a dominant driver of recurrent events despite lipid control.

Emerging Clinical Perspectives

Timing and duration

Recent analyses propose that colchicine initiation after hemodynamic stabilization yields better outcomes than in the hyperacute phase of myocardial necrosis. Early trials such as CLEAR SYNERGY (2024) found neutral benefit when colchicine was given within 72 hours of MI (HR 0.99; p = 0.93), whereas delayed initiation (≥13 days post event) manifested substantial reductions in inflammation and events (COLCOT, LoDoCo2) [27].

https://doi.org/10.61577/ijcri.2025.1000017

This temporal response parallels myocarditis treatment, where excessive interference with early immune responses may impair viral clearance, while suppression of subacute inflammation reduces fibrosis and recurrence. Optimal myopericarditis management therefore aligns with subacute initiation, once acute viral replication has subsided, but before fibrotic remodeling dominates.

Safety and monitoring in real-world use

Low dose (0.5-0.6 mg/d) colchicine has a well-characterized safety profile. In the pooled ASCVD population (~23,000 participants), no increase in serious infection, cytopenia, or mortality was observed [20]. Non cardiovascular death signals in isolated trials (LoDoCo2) did not achieve statistical significance and likely reflected confounding. Colchicine's most important drug interactions occur with strong CYP3A4 or P-glycoprotein inhibitors (clarithromycin, ketoconazole, ritonavir, cyclosporine), which markedly increase colchicine levels (when given twice daily), increasing risks for diarrhea, myopathy, and bone marrow suppression. Moderate inhibitors (verapamil, diltiazem, fluconazole) require dose reduction, while concurrent use with statins or fibrates heightens the risk of rhabdomyolysis. Patients with renal or hepatic impairment face greatly amplified toxicity. Administration with grapefruit juice should also be avoided. Diarrhea often signals toxicity, warranting immediate discontinuation. Key precautions include avoidance in advanced renal or hepatic failure and dose reduction in elderly or frail individuals. Myopathy risk with statin co administration remains below 0.2%, comparable to baseline [19]. Once daily dosing of colchicine markedly reduces the cumulative exposure that amplifies drug–drug interactions, thus reducing toxicity and risk of adverse events.

Public Health Implications and Regulatory Trajectory

Following the LoDoCo2 and COLCOT trials, the U.S. FDA granted approval in June 2023 for colchicine 0.5 mg daily which has been widely adapted to the commonly available 0.6 mg generic tablet. This indication for an anti-inflammatory small molecule signifies recognition that chronic vascular inflammation, not just cholesterol, is central to the progression of ASCVD. Given this paradigm, colchicine extended use for myopericarditis and ASCVD is not only mechanistically coherent but consistent with the scientific literature recognizing inflammation control as a legitimate cardiovascular target.

Ethical and Economical Considerations

Emerging from the global COVID-19 pandemic, where low dose anti-inflammatory therapy could avert complications from myopericarditis and ASCVD makes colchicine a public health priority. Transparency in pharmacoeconomic evaluation is essential. Modeling studies estimate that widespread adoption in U.S. coronary artery disease could prevent ~226,000 major adverse events over three years, an immense potential cost savings exceeding billions in acute care expenditure [28].

Integrating Colchicine into Clinical Practice

It is reasonable in acute COVID-19 vaccine induced myopericarditis to begin colchicine immediately combined with NSAID for pain control briefly, tapering NSAID within 7–10 days, continuing colchicine for at least 12 months. For subacute/chronic myopericarditis colchicine 0.5-0.6 mg daily for is indicated for at least 12 months with tapering after normalization of CRP and imaging resolution. This represents a significant extension beyond the 2015/2025 ESC Guideline recommendation of 3 months for acute episodes of pericarditis. While this 12-month duration exceeds traditional guidelines, it is intended to address the unique persistence of Spike-mediated endothelial dysfunction and to prevent the transition from acute inflammation to permanent arrhythmic scarring in the myocardium [11,29]. For the goal of long term prevention of ASCVD events (FDA endorsed, 2023), it is reasonable to use chronic colchicine 0.5-0.6 mg daily maintenance indefinitely in high risk or those with established ASCVD, targeting "residual inflammatory risk." Longitudinal RCTs in post-pandemic myopericarditis are warranted to determine optimal timing and duration beyond 12 months. Such trials should investigate synergy between colchicine, Spike detoxification regiments (nattokinase, bromelain, curcumin), statins, and other anti inflammatories listed in Table 3.

Table 3. Comparison anti-inflammatory strategies for cardiovascular inflammation.

Therapeutic	Mechanism	CV Outcomes	Limitations
NSAIDs	COX inhibition	Symptom relief only	GI/renal toxicity; rebound recurrences
Corticosteroids	Broad immunosuppression	Rapid efficacy	Promotes recurrence, hyperglycemia
Canakinumab	IL-1β monoclonal antibody	↓ MACE in CANTOS	High infection risk; cost >$200,000/yr
Colchicine	Microtubule & NLRP3 inhibition	↓ MACE 23–31%; ↓ pericarditis relapse 50%	Transient GI intolerance; interaction vigilance

NSAIDS=nonsteroidal anti-inflammatory agents, COX=cyclooxygenase, MACE=major adverse cardiovascular events, NLRP=Nod-like receptor family pyrin, IL=interleukin, CANTOS=Canakinumab Anti-Inflammatory Thrombosis Outcome Study

Conclusions

The association between periodontitis and cardiovascular health is well-established, with inflammation serving as a critical link between the two conditions. Periodontal pathogens, systemic inflammatory markers, and endothelial dysfunction all contribute to the increased cardiovascular risk seen in patients with periodontitis. While periodontal therapy has shown promise in reducing systemic inflammation and improving cardiovascular biomarkers, further research is needed to confirm its long-term impact on cardiovascular outcomes. Clinicians should consider periodontal health as part of the comprehensive management of cardiovascular risk, emphasizing the importance of collaborative care between dental and medical professionals.

Disclosure Statement

No potential conflict of interest was reported by the author.

https://doi.org/10.61577/ijcri.2025.1000017

References

1. Mead MN, Rose J, Makis W, Milhoan K, Hulscher N, McCullough PA. Myocarditis after SARS-CoV-2 infection and COVID-19 vaccination: Epidemiology, outcomes, and new perspectives. Int J Cardiovasc Res Innov. 2025;3(1):1-43. https://doi.org/10.61577/ijcri.2025.100001
2. Ostroff JL, BCACP B. Colchicine Use in Atherosclerotic Cardiovascular Disease. 2024. Available at: https://www.uspharmacist.com/article/colchicine-use-in-atherosclerotic-cardiovascular-disease
3. Deftereos SG, Beerkens FJ, Shah B, Giannopoulos G, Vrachatis DA, Giotaki SG, et al. Colchicine in cardiovascular disease: in-depth review. Circulation. 2022;145(1):61-78. https://doi.org/10.1161/CIRCULATIONAHA.121.056171
4. Lavanya N, Sathyamurthy I. Colchicine in Cardiovascular Diseases. J Indian Coll Cardiol. 2023;13(4):135-140. https://doi.org/10.4103/jicc.jicc_23_23
5. Ridker PM, Everett BM, Thuren T, MacFadyen JG, Chang WH, Ballantyne C, et al. Antiinflammatory therapy with canakinumab for atherosclerotic disease. N Engl J Med. 2017;377(12):1119-1131. https://doi.org/10.1056/NEJMoa1707914
6. Andreis A, Imazio M, De Ferrari GM. Colchicine for the treatment of cardiovascular diseases: old drug, new targets. J Cardiovasc Med. 2021;22(1):1-8. https://doi.org/10.2459/JCM.0000000000001079
7. Deftereos S, Giannopoulos G, Kossyvakis C, Efremidis M, Panagopoulou V, Kaoukis A, et al. Colchicine for prevention of early atrial fibrillation recurrence after pulmonary vein isolation: a randomized controlled study. J Am Coll Cardiol. 2012;60(18):1790-1796. Available at: https://www.jacc.org/doi/abs/10.1016/j.jacc.2012.07.031
8. Deftereos S, Giannopoulos G, Efremidis M, Kossyvakis C, Katsivas A, Panagopoulou V, et al. Colchicine for prevention of atrial fibrillation recurrence after pulmonary vein isolation: mid-term efficacy and effect on quality of life. Heart rhythm. 2014;11(4):620-628. https://doi.org/10.1016/j.hrthm.2014.02.002
9. Imazio M, Brucato A, Cemin R, Ferrua S, Maggiolini S, Beqaraj F, et al. A randomized trial of colchicine for acute pericarditis. N Engl J Med. 2013;369(16):1522-1528. https://doi.org/10.1056/NEJMoa1208536
10. Bayes-Genis A, Adler Y, De Luna AB, Imazio M. Colchicine in pericarditis. Eur Heart J. 2017;38(22):1706-1709. https://doi.org/10.1093/eurheartj/ehx246
11. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, et al. 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. 2015;36(42):2921-2964. Available at: https://pubmed.ncbi.nlm.nih.gov/26320112/
12. Physician List & Guide to Home-Based COVID Treatment. Guide to Home-Based COVID Treatment PDF e-Booklet. 2023. https://aapsonline.org/covidpatientguide/
13. McCullough PA, Kelly RJ, Ruocco G, Lerma E, Tumlin J, Wheelan KR, et al. Pathophysiological basis and rationale for early outpatient treatment of SARS-CoV-2 (COVID-19) infection. Am J Med. 2021;134(1):16-22. https://doi.org/10.1016/j.amjmed.2020.07.003
14. McCullough PA, Alexander PE, Armstrong R, Arvinte C, Bain AF, Bartlett RP, et al. Multifaceted highly targeted sequential multidrug treatment of early ambulatory high-risk SARS-CoV-2 infection (COVID-19). Rev Cardiovasc Med. 2020;21(4):517. http://doi.org/10.31083/j.rcm.2020.04.264
15. Tardif JC, Bouabdallaoui N, L'allier PL, Gaudet D, Shah B, Pillinger MH, et al. Colchicine for community-treated patients with COVID-19 (COLCORONA): a phase 3, randomised, double-blinded, adaptive, placebo-controlled, multicentre trial. Lancet Respir Med. 2021;9(8):924-932. https://doi.org/10.1016/S2213-2600(21)00222-8
16. Elshiwy K, Amin GE, Farres MN, Samir R, Allam MF. The role of colchicine in the management of COVID-19: a Meta-analysis. BMC Pulm Med. 2024;24(1):190. https://doi.org/10.1186/s12890-024-03001-0
17. McCullough PA, Hulscher N. Risk stratification for future cardiac arrest after COVID-19 vaccination. World J Cardiol. 2025;17(2):103909. https://doi.org/10.4330/wjc.v17.i2.103909
18. McCullough PA, Mead MN, Hulscher N. COVID-19 Vaccine-Induced Subclinical Myopericarditis: Pathophysiology, Diagnosis, and Clinical Management. Med Res Arch. 2025;13(11). https://doi.org/10.18103/mra.v13i11.7078
19. Hulscher N, Hodkinson R, Makis W, McCullough P. Autopsy proven fatal COVID-19 vaccine-induced myocarditis. 2023. https://www.preprints.org/manuscript/202307.1198
20. Lutschinger LL, Rigopoulos AG, Schlattmann P, Matiakis M, Sedding D, Schulze PC, et al. Meta-analysis for the value of colchicine for the therapy of pericarditis and of postpericardiotomy syndrome. BMC Cardiovasc Disord. 2019;19(1):207. https://doi.org/10.1186/s12872-019-1190-4
21. Valore L, Junker T, Heilmann E, Zuern CS, Streif M, Drexler B, et al. Case report: mRNA-1273 COVID-19 vaccine-associated myopericarditis: Successful treatment and re-exposure with colchicine. Front Cardiovasc Med. 2023;10:1135848. https://doi.org/10.3389/fcvm.2023.1135848
22. Hulscher N, Vickery A, McCullough PA. Resolution of Refractory COVID-19 Vaccine-Induced Myopericarditis with Adjunctive Rapamycin. Med Res Arch. 2024;12(11). https://doi.org/10.18103/mra.v12i11.6099
23. Colchicine Versus Placebo in Acute Myocarditis Patients (ARGO). Available at: https://clinicaltrials.gov/study/NCT05855746
24. Tardif JC, Kouz S, Waters DD, Bertrand OF, Diaz R, Maggioni AP, et al. Efficacy and safety of low-dose colchicine after myocardial infarction. New England journal of medicine. 2019;381(26):2497-2505. https://doi.org/10.1056/NEJMoa1912388
25. Nidorf SM, Eikelboom JW, Budgeon CA, Thompson PL. Low-dose colchicine for secondary prevention of cardiovascular disease. J Am Coll Cardiol. 2013;61(4):404-410. Available at: https://www.jacc.org/doi/abs/10.1016/j.jacc.2012.10.027
26. Beer M, Schönenberger CM, Ewald H, Briel M, Janiaud P. Colchicine for the secondary prevention of cardiovascular events. Cochrane Database Syst Rev. 2025(11). https://doi.org/10.1002/14651858.CD014808.pub2
27. Anum Zehra, Abhishek Gami, Jaideep Patel. The Evolving Role of Colchicine in Coronary Atherosclerosis Management: Timing May Be Everything. American College of Cardiology. 2025. Available at: https://www.acc.org/latest-in-cardiology/articles/2025/04/07/11/00/the-evolving-role-of-colchicine-in-coronary-atherosclerosis-management

https://doi.org/10.61577/ijcri.2025.1000017

28. Buckley LF, Chebrolu B, Al Zaria M, Blankstein R, Libby P, Weber BN. Potential impact of colchicine on atherosclerotic cardiovascular disease in the United States. JACC: Advances. 2025;4(3):101622. Available at: https://www.jacc.org/doi/abs/10.1016/j.jacadv.2025.101622

29. Schulz-Menger J, Collini V, Gröschel J, Adler Y, Brucato A, Christian V, et al. 2025 ESC Guidelines for the management of myocarditis and pericarditis: Developed by the task force for the management of myocarditis and pericarditis of the European Society of Cardiology (ESC) Endorsed by the Association for European Paediatric and Congenital Cardiology (AEPC) and the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2025;46(40):3952-4041. https://doi.org/10.1093/eurheartj/ehaf192

https://doi.org/10.61577/ijcri.2025.1000017