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Colchicine in Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA): A Narrative Review

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ABSTRACT

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is a heterogeneous clinical syndrome, accounting for 5–15% of myocardial infarction cases, characterized by myocardial injury in the absence of significant epicardial coronary obstruction. Unlike classical myocardial infarction caused by plaque rupture, MINOCA arises from diverse mechanisms, including coronary vasospasm, microvascular dysfunction, thromboembolism, spontaneous coronary artery dissection, Takotsubo cardiomyopathy, and myocarditis. Despite unobstructed coronary arteries, MINOCA carries substantial morbidity, recurrent cardiovascular events, and mortality, with inflammation playing a central role in its pathophysiology. Colchicine, a well-established anti-inflammatory agent, has emerged as a promising therapeutic option due to its ability to inhibit the NLRP3 inflammasome, modulate the AMPK/SIRT1 signaling pathway, and regulate neutrophil function, including chemotaxis, adhesion, and neutrophil extracellular trap formation. These mechanisms suppress pro-inflammatory cytokine release, reduce pyroptosis, and mitigate microvascular injury, ultimately limiting myocardial damage and improving cardiac function. While most clinical evidence derives from obstructive coronary syndromes, experimental and clinical studies suggest that colchicine's anti-inflammatory and cardioprotective effects may extend to patients with MINOCA, particularly those with microvascular dysfunction or ongoing inflammation. This narrative review synthesizes current knowledge on MINOCA pathophysiology, elucidates the mechanistic basis for colchicine therapy, and highlights its potential role as a targeted anti-inflammatory intervention in this unique patient population.

Keywords: colchicine; inflammation; literature review; MINOCA

1. INTRODUCTION

Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA), which accounts for 5–15% of all myocardial infarction patients, is becoming more widely acknowledged as a separate clinical condition. MINOCA results from a variety of processes, such as coronary vasospasm, microvascular dysfunction, thromboembolism, and spontaneous coronary artery dissection, in contrast to conventional myocardial infarction, which is caused by atherosclerotic plaque rupture and substantial coronary blockage [3,5]. MINOCA is clinically relevant since it is linked to severe morbidity, recurrent cardiovascular events, and mortality even in the absence of significant epicardial stenosis. Elevated biomarkers, including interleukin-6 and high-sensitivity C-reactive protein, show that inflammation is a key component of its etiology [1,3,8].

The complicated, frequently non-thrombotic mechanisms causing MINOCA may not be adequately addressed by conventional myocardial infarction management, especially antiplatelet therapy, which is mostly derived from obstructive coronary artery disease. This restriction has increased interest in treatment approaches that target inflammation, which is becoming more widely acknowledged as a major cause of cardiac damage and microvascular injury in this population. Because it can disrupt microtubule assembly, inhibit the NLRP3 inflammasome, and lower pro-inflammatory cytokine production mechanisms closely related to the pathophysiology of MINOCA, colchicine, a well-known anti-inflammatory drug, has shown promise [2,4,12,15].

Colchicine lowers significant adverse cardiovascular events, such as myocardial infarction and stroke, without raising cardiovascular mortality, according to clinical studies and meta-analyses in individuals with coronary artery disease. Its anti-inflammatory and cardioprotective activities indicate prospective benefits in MINOCA, especially in individuals with microvascular dysfunction or persistent inflammation, even if the majority of the evidence comes from obstructive coronary syndromes [4,7,12,16]. The purpose of this narrative review is to summarize the current understanding of the pathophysiology of MINOCA, clarify the mechanistic basis of colchicine therapy, and assess its possible use in conjunction with traditional antiplatelet tactics.

2. REVIEW CONTENT

2.1 Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA)

A clinical presentation of acute myocardial infarction (MI) without considerable obstructive coronary artery disease, which is characterized as less than 50% stenosis on coronary angiography, is referred to as Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) [6,17]. While ruling out other non-ischemic causes, including myocarditis, Takotsubo cardiomyopathy, or pulmonary embolism, the diagnosis must satisfy the universal criteria for acute MI, which include an increase and/or fall in cardiac troponin with evidence of myocardial ischemia [6,9,17]. MINOCA is regarded as a tentative diagnosis that requires additional assessment in order to identify the underlying mechanism [11]. Advanced imaging, especially cardiac magnetic resonance, optical coherence tomography, and intravascular ultrasound, is frequently necessary to clarify the etiology and direct tailored treatment because traditional angiography may overlook subtle plaque disruption, microvascular dysfunction, or vasomotor disorders [14].

In the absence of significant epicardial coronary artery stenosis, a variety of atherosclerotic and non-atherosclerotic processes contribute to the pathophysiology of myocardial infarction with non-obstructive coronary arteries (MINOCA), which is complicated and multifactorial [20].

(1) *Atherosclerotic Mechanism*

Atherosclerotic processes, specifically plaque disruption (rupture or erosion) in the absence of severe coronary artery stenosis, are linked to a subset of MINOCA instances. Optical coherence tomography (OCT) and intravascular ultrasound (IVUS) are examples of advanced intracoronary imaging that have shown that subtle atherosclerotic changes that are not visible on traditional angiography can cause thrombus formation, distal embolization, or temporary occlusion, which can cause myocardial damage without long-term obstruction [14,8,28]. Because patients with atherosclerotic [28] MINOCA are more likely to experience adverse cardiac events than those with non-atherosclerotic causes, these atherosclerotic mechanisms are clinically significant. Determining these pathways is essential for directing secondary preventive and antiplatelet measures [18,28].

(2) *Non-Atherosclerotic Mechanism*

A significant portion of the etiologies underpinning MINOCA are non-atherosclerotic, with coronary vasospasm and coronary microvascular dysfunction (CMD) being two of the most important factors [26,31]. Despite angiographically normal or nearly normal vessels, coronary vasospasm causes temporary, localized, or diffuse constriction of the epicardial coronary arteries, resulting in reversible ischemia. These episodes can happen on their own or be brought on by endothelium dysfunction, mental stress, exposure to cold, or stimulant usage. In individuals with suspected MINOCA, provocative testing, most frequently with acetylcholine or ergonovine, is crucial in detecting vasospastic angina [5,18,26]. On the other hand, CMD, which frequently disproportionately affects women, is indicative of decreased vasodilatory capacity or elevated microvascular resistance within the coronary microcirculation. Microvascular function can be understood through diagnostic evaluation utilizing coronary flow reserve or the index of microvascular resistance, and new data that connects CMD to systemic inflammation and a worse prognosis emphasizes the significance of mechanism-specific treatment [5,18,30].

Myocarditis and Takotsubo cardiomyopathy are significant non-atherosclerotic disorders that can resemble MINOCA. Takotsubo cardiomyopathy is a temporary stress-induced illness that is defined by reversible left ventricular systolic dysfunction brought on by mental or physical stress. It presents with sudden chest pain, ECG abnormalities, and troponin increase comparable to MI. To distinguish Takotsubo from actual ischemia injury, cardiac magnetic resonance imaging is crucial [5,18,26]. The symptoms and biomarker patterns of myocarditis, an inflammatory condition of the heart, are similar to those of MI. Cardiac magnetic resonance imaging that shows myocardial edema and late gadolinium enhancement is used to diagnose it; biopsy is only used in certain cases where there is still doubt. It is important to distinguish these entities from ischemic types of MINOCA because their prognostic implications and therapeutic methods are quite different [3,5,26].

2.2 Colchicine's Mechanism of Action in MINOCA

(1) *Inhibition of NLRP3 Inflammasome Activation*

Colchicine mainly targets the NLRP3 inflammasome, a crucial mediator of sterile inflammation and pyroptosis in cardiovascular damage, to provide its anti-inflammatory actions in MINOCA [21]. There are multiple interrelated processes in the mechanism. To stop microtubule polymerization, which is essential for the trafficking and assembly of inflammasome components, especially the approximation of NLRP3 and ASC (apoptosis-associated speck-like protein containing a CARD), colchicine first binds to tubulin.

Colchicine prevents ASC from assembling into the NLRP3 inflammasome complex by interfering with microtubule dynamics. This prevents caspase-1 from being recruited and activated, which is necessary for the maturation and release of pro-inflammatory cytokines such as IL-1β and IL-18 [15,19,24,31]. Colchicine inhibits the activation of the NLRP3 inflammasome, which results in decreased production of IL-1β and IL-18, decreased leukocyte recruitment, and suppression of pyroptosis, a type of inflammatory cell death linked to cardiac damage and microvascular dysfunction [15,24,31,37]. Colchicine enhances heart function and lessens myocardial damage by inhibiting these pathways, according to experimental studies. Colchicine's anti-inflammatory and cardioprotective actions are further enhanced by its modulation of other intracellular signaling pathways, such as AMPK/SIRT1 and NF-κB [21,29,31].

(2) *Modulation of AMPK/SIRT1/NLRP3 Signaling Pathway*

Colchicine modulates the AMPK/SIRT1/NLRP3 signaling pathway to give cardioprotective benefits in MINOCA, particularly in myocardial damage caused by microembolization. In cardiac tissue, it stimulates SIRT1 expression and phosphorylates AMPK, which functions as an upstream regulator of SIRT1. Together, they minimize myocardial damage and enhance heart function by suppressing NLRP3 inflammasome activation, which lowers pyroptosis and the production of pro-inflammatory cytokines. Colchicine's protective effects are diminished when the AMPK/SIRT1 pathway is inhibited, underscoring the significance of this signaling axis and bolstering its potential therapeutic application in patients with microvascular or microembolic-related cardiac damage [27,34].

(3) *Effects on Neutrophil Function*

By altering neutrophil activity, a key factor in cardiac inflammation, colchicine has strong cardioprotective benefits in MINOCA. Colchicine reduces neutrophil infiltration and activation inside the myocardium and limits future tissue damage by binding to tubulin and interfering with microtubule dynamics, which hinders neutrophil chemotaxis, adhesion, and recruitment to sites of vascular injury [2, 13]. Colchicine specifically reduces the release of neutrophil granule proteins, including NGAL, which are linked to inflammation and plaque susceptibility, and L-selectin (CD62L) expression on neutrophils, which is necessary for endothelium attachment and migration [2, 25].

Inhibition of neutrophil extracellular trap (NET) production is a crucial mechanism. NETs, which are made of granular proteins and DNA, worsen cardiac damage and cause microvascular blockage. Colchicine inhibits peptidylarginine deiminase 4, an essential enzyme in NETosis, and restores cytoskeletal structure to decrease NET release [36,42,39]. Following myocardial injury, this activity lessens microvascular blockage, attenuates cardiac remodeling, and enhances cardiac function [40,39].

Furthermore, colchicine reduces the pool of circulating neutrophils that can spread inflammatory damage by inhibiting neutrophil proliferation in the bone marrow through the S100A8/A9-NLRP3-IL-1β pathway [40]. Colchicine routinely shows strong tissue- and molecular-level modulation of neutrophil activity, which is probably crucial for its therapeutic effectiveness in MINOCA, even if some clinical investigations report only slight changes in systemic neutrophil counts or inflammatory indicators [40,43,25].

2.3 Colchicine versus Antiplatelet Therapy in MINOCA: Comparative Insights

A key component of the treatment of obstructive myocardial infarction is antiplatelet medication, which prevents thrombotic events by preventing platelet aggregation. However, the benefits of traditional antiplatelet medication are less certain because MINOCA is characterized by varied and frequently non-thrombotic causes. Because routine antiplatelet use is mostly derived from obstructive MI data rather than directly supported by MINOCA-specific trials, observational studies, and expert consensus indicate that routine antiplatelet use in MINOCA may not significantly enhance clinical outcomes [33, 35]. Colchicine, on the other hand, is the only anti-inflammatory medication that is presently authorized for secondary prevention in coronary artery disease and mainly targets inflammation, which is a major cause of atherothrombosis and microvascular damage [38,44,45].

Although gastrointestinal side effects are more frequent, clinical trials and meta-analyses in larger coronary populations show that colchicine successfully lowers major adverse cardiovascular events (MACE), myocardial infarction, stroke, and the need for coronary revascularization without appreciably raising cardiovascular mortality [44,45,46]. In contrast, increased antiplatelet medication in MINOCA may increase the risk of bleeding and has not demonstrated any extra benefits [35]. Notably, combination therapy with colchicine and antiplatelet agents seems safe and may further lower inflammatory biomarkers, like high-sensitivity C-reactive protein, while having little effect on platelet reactivity. This suggests that colchicine may be used in targeted therapy for patients with MINOCA [23,38,41].

CONCLUSION

The complicated and diverse disease known as Myocardial Infarction with Non-Obstructive Coronary Arteries (MINOCA) is characterized by myocardial damage even in the absence of substantial epicardial coronary obstruction. Plaque disruption, coronary vasospasm, microvascular dysfunction, Takotsubo cardiomyopathy, and myocarditis are examples of both atherosclerotic and non-atherosclerotic causes. The development of the disease and its unfavorable consequences are mostly dependent on inflammation and microvascular damage, underscoring the necessity of focused treatment approaches.

Colchicine inhibits the NLRP3 inflammasome, modifies the AMPK/SIRT1 signaling pathway, and regulates neutrophil function, including chemotaxis, adhesion, and neutrophil extracellular trap formation, to have cardioprotective effects in MINOCA. Colchicine may enhance myocardial recovery and reduce tissue damage in MINOCA patients by inhibiting inflammatory signaling, lowering pyroptosis, and minimizing microvascular injury. In addition to highlighting areas for further study to maximize its clinical use, this narrative review emphasizes the mechanistic justification for colchicine as a viable anti-inflammatory medication in this particular patient population.

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