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Review Paper

Contemporary Pharmacotherapy of Myocardial Infarction: Recent Advances, Precision Medicine, and a Clinical Pharmacy Perspective Contemporary Pharmacotherapy of Myocardial Infarction

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ABSTRACT

Background: Myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide despite significant advances in cardiovascular prevention and treatment. Contemporary management has evolved beyond conventional pharmacotherapy through the incorporation of novel lipid-lowering agents, anti-inflammatory therapies, precision medicine, and multidisciplinary clinical pharmacy services, all of which contribute to improved patient outcomes. **Objectives:** This review aims to critically summarize current evidence on guideline-directed pharmacotherapy for myocardial infarction, recent therapeutic advances, precision medicine approaches, and the expanding role of clinical pharmacists in optimizing treatment and secondary prevention. **Methods:** A narrative review of the literature was conducted using PubMed/MEDLINE, Scopus, Embase, Web of Science, and Google Scholar. Relevant randomized controlled trials, systematic reviews, meta-analyses, observational studies, and contemporary international clinical practice guidelines published in English were evaluated to provide an evidence-based overview of current and emerging pharmacological strategies for myocardial infarction management. **Results:** Guideline-directed medical therapy, including dual antiplatelet therapy, anticoagulants, beta-blockers, renin-angiotensin-aldosterone system inhibitors, mineralocorticoid receptor antagonists, and high-intensity statins, remains the foundation of myocardial infarction management. Recent therapeutic advances, including PCSK9 inhibitors, inclisiran, bempedoic acid, anti-inflammatory agents, and emerging factor XI/XIa inhibitors, have demonstrated potential to reduce residual cardiovascular risk in selected patients. Precision medicine approaches, particularly CYP2C19 genotype-guided antiplatelet therapy, support individualized treatment selection. Clinical pharmacists play an essential role in medication optimization, medication reconciliation, adverse drug

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reaction monitoring, pharmacogenomic implementation, patient counseling, medication adherence, and secondary prevention, thereby enhancing medication safety and improving long-term cardiovascular outcomes. Conclusion: The pharmacological management of myocardial infarction continues to evolve through the integration of innovative therapies, precision medicine, and multidisciplinary care. Clinical pharmacists are integral to evidence-based cardiovascular management, contributing to safer medication use, personalized therapeutic decision-making, and improved patient outcomes. Continued research focusing on implementation strategies, pharmacogenomics, digital health technologies, and emerging therapeutics is expected to further optimize myocardial infarction care.

INTRODUCTION

Myocardial infarction (MI) is a major manifestation of acute coronary syndrome (ACS) and remains one of the leading causes of death and long-term disability worldwide. It occurs when myocardial oxygen demand exceeds oxygen supply because of a sudden reduction or complete interruption of coronary blood flow, resulting in irreversible myocardial injury if prompt reperfusion is not achieved. Despite substantial advances in cardiovascular medicine, MI continues to impose a significant clinical, social, and economic burden due to its high incidence, recurrent cardiovascular events, prolonged hospitalization, and long-term complications.¹⁻⁴

The underlying mechanism in most cases involves rupture or erosion of an atherosclerotic plaque, followed by platelet activation, thrombus formation, and partial or complete occlusion of a coronary artery. Over the past two decades, improvements in early diagnosis, rapid reperfusion strategies, and evidence-based pharmacotherapy have markedly reduced mortality and improved survival. Nevertheless, recurrent ischemic events, heart failure, arrhythmias, and medication-related adverse effects continue to present important therapeutic challenges.^{2, 5, 6}

Current international recommendations from the American College of Cardiology/American Heart

Association (ACC/AHA) and the European Society of Cardiology (ESC) advocate individualized treatment strategies that incorporate the patient's clinical presentation, ischemic and bleeding risks, comorbidities, and overall cardiovascular profile. Guideline-directed medical therapy, combined with timely coronary revascularization and comprehensive secondary prevention, forms the cornerstone of contemporary MI management and has substantially improved both short- and long-term clinical outcomes.^{1, 2, 7, 8} Recent therapeutic developments have expanded the range of available treatment options beyond conventional antithrombotic and lipid-lowering therapies. Novel agents targeting platelet activation, lipid metabolism, inflammation, and coagulation, together with advances in pharmacogenomics and biomarker-guided treatment, are facilitating more individualized approaches to patient care. In parallel, the role of clinical pharmacists has evolved considerably, extending beyond medication dispensing to include evidence-based medication optimization, prevention of medication-related problems, patient education, adherence support, pharmacogenomic interpretation, and multidisciplinary cardiovascular care.

This review summarizes current evidence on guideline-directed pharmacotherapy and recent therapeutic advances in the management of myocardial infarction. It also highlights emerging precision medicine strategies and examines the expanding contribution of clinical pharmacists in optimizing medication use, enhancing patient safety, and improving cardiovascular outcomes.

2. Epidemiology and Burden of Myocardial Infarction

2.1 Global Burden

Cardiovascular diseases remain the foremost cause of mortality worldwide, with myocardial



infarction (MI) representing one of the principal contributors to premature death, disability, and healthcare expenditure. Despite notable reductions in case-fatality rates in many developed countries owing to improvements in cardiovascular prevention, early diagnosis, reperfusion strategies, and evidence-based pharmacotherapy, MI continues to impose a substantial global health burden. In contrast, many low- and middle-income countries are experiencing a rising incidence of MI, largely driven by population ageing, rapid urbanization, lifestyle transitions, and the increasing prevalence of cardiometabolic risk factors. Consequently, myocardial infarction continues to account for a considerable proportion of hospital admissions, recurrent cardiovascular events, and long-term healthcare costs worldwide.^{9–11}

2.2 Risk Factors

The occurrence of myocardial infarction is influenced by the interaction of both modifiable and non-modifiable risk factors. Major modifiable determinants include hypertension, dyslipidemia, diabetes mellitus, cigarette smoking, obesity, physical inactivity, unhealthy dietary habits, and psychosocial stress, all of which contribute to the initiation and progression of atherosclerosis. Non-modifiable risk factors include advancing age, male sex, family history of premature cardiovascular disease, and inherited genetic

susceptibility. Comprehensive risk-factor assessment and aggressive management through lifestyle modification and evidence-based pharmacological interventions remain fundamental strategies for reducing both first and recurrent myocardial infarction.^{12–14}

2.3 Clinical Classification

The Fourth Universal Definition of Myocardial Infarction classifies MI into five distinct categories according to the underlying pathophysiological mechanism. Type 1 MI results from spontaneous rupture or erosion of an atherosclerotic plaque leading to coronary thrombosis and represents the most frequently encountered subtype. Type 2 MI arises from an imbalance between myocardial oxygen supply and demand in the absence of acute coronary thrombosis. Type 3 MI is characterized by sudden cardiac death suggestive of myocardial ischemia before biomarker confirmation, whereas Types 4 and 5 are procedure-related and occur following percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG), respectively. From a clinical perspective, myocardial infarction is further categorized as ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), a distinction that guides reperfusion strategies, antithrombotic therapy, and overall management.^{15, 16}

Table 1. Classification of Myocardial Infarction

Type	Mechanism	Clinical Example
Type 1	Plaque rupture with thrombosis	STEMI, NSTEMI
Type 2	Oxygen supply-demand mismatch	Severe anemia, sepsis
Type 3	Sudden cardiac death	Cardiac arrest before biomarkers
Type 4a	PCI-related MI	After coronary intervention
Type 4b	Stent thrombosis	Definite stent thrombosis
Type 4c	Restenosis	In-stent restenosis
Type 5	CABG-related MI	Following bypass surgery



3. Pathophysiology of Myocardial Infarction

Myocardial infarction results from a sequence of pathological events initiated by disruption of coronary atherosclerotic plaques, followed by thrombus formation and prolonged myocardial ischemia. If coronary perfusion is not restored promptly, sustained oxygen deprivation leads to irreversible cardiomyocyte death, ventricular remodeling, and progressive impairment of cardiac function. The complex interplay among vascular inflammation, platelet activation, coagulation pathways, and ischemia–reperfusion injury forms the biological basis for current pharmacological treatment strategies.^{17–24}

3.1 Atherosclerotic Plaque Rupture

Most cases of myocardial infarction originate from rupture or superficial erosion of vulnerable atherosclerotic plaques within the coronary arteries. Progressive lipid deposition, infiltration of inflammatory cells, and degradation of the fibrous cap weaken plaque stability over time. Once the protective cap is disrupted, highly thrombogenic plaque components are exposed to circulating blood, triggering platelet adhesion, activation, and aggregation while simultaneously activating the coagulation cascade. These events culminate in the formation of an occlusive thrombus that abruptly compromises coronary blood flow.^{17–19}

3.2 Coronary Thrombosis

Following plaque disruption, activated platelets release potent mediators that amplify platelet recruitment and thrombin generation. The coagulation cascade subsequently promotes fibrin formation, stabilizing the developing thrombus and further obstructing coronary perfusion. Depending on the extent of arterial occlusion and collateral circulation, myocardial ischemia may

progress rapidly to infarction. This pathophysiological process underpins the routine use of dual antiplatelet therapy and anticoagulant agents during the acute management of myocardial infarction to limit thrombus propagation and preserve myocardial tissue.^{20, 21}

3.3 Ischemia–Reperfusion Injury

Although restoration of coronary blood flow is essential for myocardial salvage, reperfusion itself may paradoxically contribute to additional cellular injury. Reoxygenation of previously ischemic myocardium can trigger excessive generation of reactive oxygen species, intracellular calcium overload, mitochondrial dysfunction, endothelial injury, and an exaggerated inflammatory response. Collectively, these mechanisms promote cardiomyocyte death, microvascular dysfunction, infarct expansion, and adverse left ventricular remodeling. Consequently, considerable research has focused on developing cardioprotective interventions capable of attenuating ischemia–reperfusion injury and further improving long-term clinical outcomes following successful reperfusion therapy.^{22–24}

4. Guideline-Directed Pharmacotherapy

Guideline-directed pharmacotherapy is the cornerstone of contemporary management of myocardial infarction and should be initiated promptly following diagnosis unless contraindications exist. The primary therapeutic objectives are to restore coronary perfusion, prevent thrombus progression, limit infarct size, preserve left ventricular function, reduce recurrent ischemic events, and improve long-term survival. Current recommendations from the ACC/AHA and ESC advocate an integrated treatment strategy that combines antiplatelet agents, anticoagulants, reperfusion therapy, beta-blockers, renin–angiotensin–aldosterone system (RAAS)



inhibitors, high-intensity statins, and selected adjunctive therapies according to the patient's clinical presentation, bleeding risk, and comorbid conditions.^{1, 25, 26}

4.1 Antiplatelet Therapy

Platelet activation plays a pivotal role in the initiation and propagation of coronary thrombosis, making antiplatelet therapy a fundamental component of acute myocardial infarction management. In the absence of contraindications, current international guidelines recommend dual antiplatelet therapy (DAPT), consisting of aspirin combined with an oral P2Y₁₂ receptor inhibitor, for most patients presenting with acute coronary syndrome. Aspirin exerts its antithrombotic effect through irreversible inhibition of cyclooxygenase-1 (COX-1), thereby suppressing thromboxane A₂ production and reducing platelet aggregation. P2Y₁₂ receptor inhibitors complement this mechanism by blocking adenosine diphosphate (ADP)-mediated platelet activation and stabilizing thrombus formation.^{25, 26}

Among currently available oral P2Y₁₂ inhibitors, ticagrelor and prasugrel produce more rapid, potent, and predictable platelet inhibition than clopidogrel. Their superior pharmacodynamic profile has translated into improved clinical outcomes in large randomized trials. In the PLATO trial, ticagrelor significantly reduced the composite endpoint of cardiovascular death, myocardial infarction, and stroke compared with clopidogrel without a significant increase in overall major bleeding.²⁷ Likewise, the TRITON–TIMI 38 trial demonstrated that prasugrel lowered the incidence of ischemic complications and stent thrombosis in patients undergoing percutaneous coronary intervention, although this benefit was accompanied by an increased risk of major bleeding in selected high-risk populations.²⁸

Selection of an appropriate P2Y₁₂ inhibitor should therefore be individualized after consideration of

ischemic risk, bleeding risk, patient age, previous cerebrovascular disease, need for urgent surgery, and concomitant medications. Consistent with contemporary guideline recommendations, ticagrelor or prasugrel is generally preferred over clopidogrel for patients without contraindications because of their greater efficacy in preventing recurrent ischemic events. Clopidogrel remains an appropriate alternative for patients with contraindications to potent P2Y₁₂ inhibitors, intolerance, excessive bleeding risk, or when pharmacogenomic and clinical considerations support its use.^{25–28}

From a clinical pharmacy perspective, pharmacists contribute substantially to optimizing antiplatelet therapy through assessment of contraindications, evaluation of potential drug–drug interactions, monitoring for bleeding complications, reinforcement of medication adherence, patient counseling regarding dual antiplatelet therapy, and identification of candidates for CYP2C19 genotype-guided antiplatelet selection where such testing is available.

4.2 Anticoagulant Therapy

Anticoagulant therapy is an essential component of the acute management of myocardial infarction, particularly in patients undergoing primary percutaneous coronary intervention (PCI) or those receiving fibrinolytic therapy. The primary objective of anticoagulation is to inhibit thrombin generation and fibrin formation, thereby preventing further thrombus propagation and reducing the risk of recurrent ischemic complications. The selection of an anticoagulant should be individualized according to the reperfusion strategy, renal function, bleeding risk, and patient-specific clinical characteristics.^{29, 30}

Unfractionated heparin (UFH) remains one of the most widely used anticoagulants because of its rapid onset of action, predictable reversal with protamine sulfate, and extensive clinical



experience. However, its variable pharmacokinetics require careful dose adjustment and activated clotting time or activated partial thromboplastin time monitoring. Low-molecular-weight heparins, particularly enoxaparin, provide more predictable anticoagulant effects, improved bioavailability, and a lower incidence of heparin-induced thrombocytopenia, making them suitable alternatives in many clinical settings.^{30, 31}

Direct thrombin inhibition with bivalirudin represents another therapeutic option during PCI, particularly for patients at increased risk of bleeding. Clinical trials have demonstrated that bivalirudin provides effective anticoagulation while reducing bleeding complications compared with conventional heparin-based regimens in appropriately selected patients. More recently, fondaparinux, a selective factor Xa inhibitor, has shown favorable efficacy and safety in patients with non-ST-segment elevation myocardial infarction, although additional anticoagulation is required during PCI because of the risk of catheter thrombosis.^{29–32}

Clinical pharmacists contribute significantly to the safe administration of anticoagulants by ensuring appropriate dose selection according to body weight and renal function, monitoring laboratory parameters when indicated, identifying clinically important drug interactions, preventing medication errors, and implementing strategies to minimize bleeding complications throughout hospitalization.

4.3 Fibrinolytic Therapy

Prompt restoration of coronary blood flow is essential for limiting myocardial damage in patients with ST-segment elevation myocardial infarction (STEMI). Although primary PCI remains the preferred reperfusion strategy, fibrinolytic therapy continues to play a critical role in healthcare settings where timely PCI is unavailable. Current guidelines recommend

administration of fibrinolytic agents within the recommended therapeutic window for eligible patients presenting early after symptom onset when primary PCI cannot be performed within the guideline-recommended timeframe.^{25, 26}

Contemporary fibrinolytic agents, including tenecteplase, alteplase, and reteplase, promote conversion of plasminogen to plasmin, resulting in fibrin degradation and dissolution of the occlusive coronary thrombus. Early administration has been consistently associated with improved coronary reperfusion, reduced infarct size, preservation of ventricular function, and lower mortality. Nevertheless, the benefits of fibrinolysis must be balanced against the potential risk of major bleeding, including intracranial hemorrhage, making careful patient selection essential.^{33, 34}

Following successful fibrinolytic therapy, patients should receive adjunctive antiplatelet and anticoagulant therapy in accordance with current clinical guidelines and undergo routine assessment for rescue or pharmacoinvasive PCI when indicated. Continuous clinical monitoring is required to evaluate reperfusion success, detect recurrent ischemia, and identify bleeding complications at the earliest opportunity.

From a clinical pharmacy perspective, pharmacists play an important role in verifying eligibility for fibrinolytic therapy, screening for absolute and relative contraindications, ensuring accurate weight-based dosing, coordinating adjunctive antithrombotic therapy, monitoring treatment response, and educating healthcare professionals and patients regarding the recognition and management of hemorrhagic complications.

4.4 Beta-Blockers

Beta-blockers are an integral component of guideline-directed therapy following myocardial infarction because they reduce myocardial oxygen consumption by lowering heart rate, systemic blood pressure, and myocardial contractility. In



hemodynamically stable patients, early initiation of beta-blocker therapy decreases recurrent ischemia, limits infarct expansion, suppresses ventricular arrhythmias, and improves long-term cardiovascular outcomes. Their benefits are particularly evident in patients with left ventricular systolic dysfunction and those at increased risk of recurrent ischemic events.³³

The COMMIT trial demonstrated that early administration of metoprolol reduced the incidence of reinfarction and ventricular fibrillation in patients with acute myocardial infarction. However, an increased occurrence of cardiogenic shock was observed among patients with hemodynamic instability, emphasizing the importance of careful patient selection before treatment initiation. Consequently, beta-blockers should be started only after stabilization of blood pressure and assessment for contraindications such as cardiogenic shock, severe bradycardia, advanced atrioventricular block, or acute decompensated heart failure.³³

Clinical pharmacists contribute to optimal beta-blocker therapy by evaluating patient eligibility, monitoring heart rate and blood pressure, identifying contraindications and clinically significant drug interactions, titrating doses according to therapeutic response, and counseling patients regarding medication adherence and recognition of potential adverse effects.

4.5 Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers

Inhibition of the renin–angiotensin–aldosterone system (RAAS) is fundamental to preventing adverse ventricular remodeling after myocardial infarction. Angiotensin-converting enzyme (ACE) inhibitors reduce afterload, improve left ventricular function, limit progressive myocardial fibrosis, and decrease the risk of recurrent myocardial infarction, heart failure, and cardiovascular mortality. Current clinical

guidelines recommend early initiation of ACE inhibitors, particularly in patients with anterior myocardial infarction, reduced left ventricular ejection fraction, heart failure, hypertension, or diabetes mellitus.³⁴

For patients who are unable to tolerate ACE inhibitors because of cough, angioedema, or other adverse effects, angiotensin receptor blockers (ARBs) provide an effective alternative with comparable cardiovascular benefits. Appropriate selection between these agents should consider renal function, serum potassium concentration, blood pressure, and individual tolerability.

The Survival and Ventricular Enlargement (SAVE) trial established the long-term benefits of captopril in patients with left ventricular dysfunction following myocardial infarction, demonstrating significant reductions in mortality, recurrent myocardial infarction, and progression to heart failure. These findings have firmly established RAAS inhibition as a cornerstone of secondary prevention after myocardial infarction.³⁴

Clinical pharmacists play a vital role in optimizing RAAS inhibitor therapy through individualized dose titration, routine monitoring of renal function and serum electrolytes, identification of drug-related adverse effects, prevention of clinically significant interactions, and patient education regarding treatment adherence and laboratory monitoring.

4.6 Mineralocorticoid Receptor Antagonists

Mineralocorticoid receptor antagonists (MRAs), including eplerenone, provide additional inhibition of aldosterone-mediated myocardial fibrosis, ventricular remodeling, and sodium retention following myocardial infarction. Current guidelines recommend MRAs for patients with reduced left ventricular ejection fraction accompanied by heart failure symptoms or



diabetes mellitus despite receiving optimal guideline-directed medical therapy.³⁵

Evidence from the EPHEsus trial demonstrated that eplerenone significantly reduced cardiovascular mortality and hospitalizations for heart failure in patients with left ventricular dysfunction after myocardial infarction, supporting its routine use in appropriately selected patients. Careful monitoring of renal function and serum potassium is essential because hyperkalemia remains the principal treatment-related adverse effect.³⁵

Clinical pharmacists contribute by identifying eligible patients, reviewing concomitant medications that increase hyperkalemia risk, monitoring renal function and electrolyte concentrations, educating patients regarding dietary potassium intake when appropriate, and ensuring safe long-term use of MRAs.

4.7 High-Intensity Statin Therapy

Intensive lipid-lowering therapy represents a fundamental component of secondary prevention after myocardial infarction. High-intensity statins should be initiated as early as possible irrespective of baseline low-density lipoprotein cholesterol (LDL-C) concentrations unless contraindicated. Beyond their LDL-C lowering effect, statins stabilize atherosclerotic plaques, improve endothelial function, reduce vascular inflammation, and decrease the likelihood of recurrent cardiovascular events.³⁶

The PROVE IT-TIMI 22 trial demonstrated that intensive lipid lowering with atorvastatin was superior to moderate-intensity statin therapy in reducing recurrent cardiovascular events following acute coronary syndrome. These findings support current guideline recommendations advocating aggressive lipid lowering as part of comprehensive post-myocardial infarction management.³⁶

Clinical pharmacists support lipid management by promoting adherence to high-intensity statin therapy, monitoring liver function and statin-associated muscle symptoms when clinically indicated, identifying drug-drug interactions, and recommending additional lipid-lowering therapies such as ezetimibe or PCSK9 inhibitors for patients who fail to achieve recommended LDL-C targets. Although conventional guideline-directed pharmacotherapy has substantially improved survival following myocardial infarction, residual cardiovascular risk remains an important clinical challenge. Continued advances in cardiovascular pharmacology have therefore focused on developing novel therapies that further reduce recurrent ischemic events, improve lipid management, target vascular inflammation, and support individualized treatment strategies. The following section summarizes recent therapeutic innovations that complement established evidence-based management.

5. Recent Advances in Pharmacotherapy for Myocardial Infarction

Although contemporary guideline-directed medical therapy has substantially improved survival following myocardial infarction (MI), a considerable proportion of patients continue to experience recurrent cardiovascular events despite optimal treatment. Persistent residual cardiovascular risk has stimulated the development of novel therapeutic agents that target dyslipidemia, inflammation, thrombosis, and metabolic dysfunction. These emerging therapies complement established treatment strategies and have the potential to further improve long-term cardiovascular outcomes.

5.1 PCSK9 Inhibitors

Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors have transformed lipid



management in patients at very high cardiovascular risk by producing profound reductions in low-density lipoprotein cholesterol (LDL-C). Monoclonal antibodies such as evolocumab and alirocumab inhibit PCSK9-mediated degradation of hepatic LDL receptors, thereby enhancing LDL-C clearance from the circulation.

Large cardiovascular outcome trials have demonstrated that the addition of PCSK9 inhibitors to maximally tolerated statin therapy significantly reduces recurrent myocardial infarction, ischemic stroke, and the need for coronary revascularization. These agents are particularly beneficial in patients with persistent hypercholesterolemia despite intensive lipid-lowering therapy or those with familial hypercholesterolemia. Their excellent safety profile and sustained lipid-lowering efficacy have established PCSK9 inhibition as an important component of secondary prevention in carefully selected patients.

Clinical pharmacists contribute by identifying eligible patients, facilitating access to therapy, monitoring treatment adherence, educating patients regarding administration techniques, and evaluating long-term lipid goal attainment.

5.2 Inclisiran

Inclisiran is a first-in-class small interfering RNA (siRNA) therapy that selectively inhibits hepatic PCSK9 synthesis. Unlike monoclonal antibodies, inclisiran provides prolonged LDL-C reduction with a twice-yearly maintenance dosing schedule following the initial loading doses, offering a practical option for patients requiring sustained lipid lowering.

Clinical studies have consistently demonstrated substantial reductions in LDL-C when inclisiran is added to standard lipid-lowering therapy. Its infrequent dosing schedule may improve treatment adherence and persistence, making it an attractive

therapeutic option for long-term secondary prevention following myocardial infarction.

Pharmacists play an important role in coordinating administration schedules, reinforcing adherence to concomitant lipid-lowering therapy, monitoring therapeutic response, and counseling patients regarding expected benefits and potential adverse effects.

5.3 Bempedoic Acid

Bempedoic acid is an oral adenosine triphosphate citrate lyase inhibitor that reduces hepatic cholesterol synthesis upstream of HMG-CoA reductase. Because the drug is activated primarily within the liver, it has a lower potential for skeletal muscle-related adverse effects compared with statins, making it a valuable alternative for patients with statin intolerance.

Recent clinical evidence has demonstrated meaningful reductions in LDL-C and favorable cardiovascular outcomes when bempedoic acid is incorporated into comprehensive lipid-lowering strategies. Its complementary mechanism of action allows combination therapy with statins, ezetimibe, and PCSK9 inhibitors in patients who fail to achieve recommended lipid targets.

5.4 Anti-inflammatory Therapy

Increasing evidence supports the concept that persistent vascular inflammation contributes to recurrent cardiovascular events even after successful revascularization and optimal lipid control. This recognition has led to renewed interest in anti-inflammatory therapies as an adjunctive strategy for secondary prevention.

Clinical trials evaluating colchicine and interleukin-1 β inhibition have demonstrated reductions in recurrent ischemic events among appropriately selected patients, supporting inflammation as a therapeutic target independent of cholesterol lowering. Nevertheless, patient



selection, safety considerations, and long-term effectiveness require further investigation before widespread implementation.

Clinical pharmacists can assist in identifying suitable candidates, monitoring adverse effects, evaluating potential drug interactions, and promoting adherence during long-term anti-inflammatory treatment.

5.5 Emerging Anticoagulant Strategies

Factor XI and factor XIa inhibitors represent a promising class of anticoagulants designed to reduce thrombotic events while minimizing bleeding complications associated with conventional anticoagulant therapy. By selectively inhibiting components of the intrinsic coagulation pathway, these agents aim to preserve physiological hemostasis while preventing pathological thrombosis.

Although several phase II and phase III clinical trials are ongoing, preliminary findings suggest that factor XI/XIa inhibition may provide an improved balance between efficacy and safety in patients requiring long-term antithrombotic therapy after myocardial infarction.

5.6 Cardiometabolic Therapies

Beyond glucose control, sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists have demonstrated significant cardiovascular benefits in patients with diabetes mellitus and established cardiovascular disease. These agents reduce hospitalization for heart failure, improve cardiovascular outcomes, and may contribute to comprehensive secondary prevention in selected patients with myocardial infarction and associated metabolic disorders.

5.7 Precision Pharmacotherapy

Precision medicine is reshaping the management of myocardial infarction by enabling treatment decisions based on individual genetic, clinical, and biomarker profiles. CYP2C19 genotype-guided selection of P2Y₁₂ inhibitors represents one of the most clinically relevant applications, allowing optimization of antiplatelet therapy while balancing ischemic protection against bleeding risk.

Future advances integrating pharmacogenomics, artificial intelligence, predictive analytics, and digital health technologies are expected to facilitate increasingly individualized cardiovascular care and improve therapeutic outcomes across diverse patient populations.

Table 2. Precision Pharmacotherapy

Approach	Application	Clinical Benefit
CYP2C19 genotyping	Clopidogrel response	Personalized antiplatelet therapy
High-sensitivity troponin	Early MI diagnosis	Rapid diagnosis
LDL-C monitoring	Lipid therapy	Achieve LDL targets
Biomarker-guided therapy	Risk stratification	Better treatment selection
Pharmacogenomics	Drug selection	Reduced adverse reactions

6. Clinical Pharmacy Perspective in Myocardial Infarction Management

The expanding role of clinical pharmacists has significantly influenced the management of myocardial infarction by promoting evidence-

based medication use, improving patient safety, and supporting multidisciplinary cardiovascular care. As integral members of the healthcare team, clinical pharmacists contribute throughout the continuum of care, from emergency management



and hospitalization to discharge planning and long-term secondary prevention. Their expertise in pharmacotherapy optimization, medication safety, patient counseling, and therapeutic monitoring enhances both clinical outcomes and healthcare quality.

6.1 Medication Optimization

Selection of appropriate pharmacological therapy following myocardial infarction requires careful consideration of clinical presentation, comorbidities, renal and hepatic function, concomitant medications, and patient-specific risk factors. Clinical pharmacists assist in optimizing treatment by evaluating the appropriateness of evidence-based therapies, recommending dose adjustments, minimizing therapeutic duplication, and ensuring adherence to current clinical practice guidelines. Individualized pharmacotherapy helps maximize therapeutic benefit while reducing the likelihood of medication-related complications.

6.2 Medication Reconciliation and Transition of Care

Medication discrepancies frequently occur during hospital admission, interdepartmental transfer, and discharge, increasing the risk of preventable adverse drug events and hospital readmissions. Clinical pharmacists perform comprehensive medication reconciliation by reviewing pre-admission medications, identifying omissions or duplications, resolving prescribing discrepancies, and ensuring continuity of evidence-based therapy throughout hospitalization. Structured discharge counseling and communication with primary healthcare providers further facilitate safe transitions of care and improve long-term medication adherence.

6.3 Prevention of Drug-Related Problems

Patients with myocardial infarction commonly receive multiple cardiovascular medications, increasing the potential for drug–drug interactions, contraindications, dosing errors, and adverse drug reactions. Clinical pharmacists play an essential role in identifying and resolving medication-related problems through routine prescription review, therapeutic monitoring, and collaboration with physicians and nursing staff. Continuous evaluation of bleeding risk, renal function, electrolyte balance, and medication tolerability supports safer pharmacotherapy and timely intervention when treatment-related complications occur.

6.4 Patient Education and Medication Adherence

Long-term adherence to secondary prevention therapy remains one of the greatest challenges following myocardial infarction. Clinical pharmacists provide individualized patient education regarding medication purpose, dosing schedules, expected therapeutic benefits, potential adverse effects, lifestyle modification, and the importance of adherence to prescribed treatment. Educational interventions improve patient understanding, encourage shared decision-making, and reduce premature discontinuation of essential cardiovascular medications.

6.5 Pharmacogenomics and Precision Medicine

The integration of pharmacogenomic testing into clinical practice has expanded opportunities for individualized cardiovascular therapy. CYP2C19 genotype-guided selection of P2Y12 inhibitors enables identification of patients who may have reduced responsiveness to clopidogrel and supports selection of alternative agents when appropriate. Clinical pharmacists are well positioned to interpret pharmacogenomic results,



integrate genetic information into therapeutic decision-making, educate healthcare professionals regarding precision medicine, and facilitate implementation of genotype-guided prescribing within multidisciplinary care pathways.

6.6 Secondary Prevention and Long-Term Follow-up

Successful long-term management of myocardial infarction extends beyond the acute hospitalization period. Clinical pharmacists actively participate in cardiovascular risk reduction by monitoring achievement of blood pressure and lipid targets, supporting smoking cessation, promoting healthy lifestyle modifications, encouraging participation in cardiac rehabilitation programs, and reinforcing adherence to evidence-based pharmacotherapy. Regular follow-up enables early identification of

medication-related problems, optimization of therapy, and sustained reduction of recurrent cardiovascular events.

6.7 Future Directions for Clinical Pharmacy Practice

Advances in digital health technologies, telepharmacy, electronic clinical decision support systems, artificial intelligence, and remote patient monitoring are expanding the scope of clinical pharmacy services in cardiovascular care. Future pharmacist-led interventions are expected to incorporate pharmacogenomics, predictive analytics, and personalized medicine to further optimize therapeutic decision-making, improve medication safety, and enhance long-term outcomes in patients recovering from myocardial infarction.

Table 3. Role of Clinical Pharmacists in Myocardial Infarction Management

Stage of Care	Clinical Pharmacist Intervention	Expected Clinical Benefit
Emergency Department	Verify antithrombotic therapy and contraindications	Faster evidence-based treatment
Hospitalization	Medication reconciliation and dose optimization	Reduced medication errors
PCI/Fibrinolysis	Monitor anticoagulant and antiplatelet therapy	Lower bleeding and thrombotic risk
Discharge	Patient counseling and medication review	Improved adherence
Follow-up	Lipid management and adverse-effect monitoring	Better secondary prevention
Long-term Care	Pharmacogenomic support and telepharmacy	Personalized treatment and improved outcomes

7. Special Populations in Myocardial Infarction Management

Management of myocardial infarction requires individualized therapeutic strategies in special patient populations because physiological changes, comorbidities, and differences in drug response may substantially influence treatment efficacy and safety. Careful assessment of

ischemic risk, bleeding risk, organ function, and life expectancy is essential to optimize pharmacotherapy while minimizing treatment-related complications.

7.1 Older Adults

Older adults represent a large proportion of patients presenting with myocardial infarction and frequently have multiple comorbidities, frailty,



and polypharmacy. Although guideline-directed medical therapy remains beneficial, advanced age is associated with an increased risk of bleeding, renal impairment, and adverse drug reactions. Therapeutic decisions should therefore balance the anticipated cardiovascular benefits against potential treatment-related risks. Individualized dose adjustment, careful monitoring, and shared decision-making are essential to optimize outcomes in this population.

Clinical pharmacists play an important role by reviewing medication appropriateness, identifying potentially inappropriate medications, monitoring for drug interactions, simplifying complex treatment regimens, and promoting medication adherence.

7.2 Patients with Diabetes Mellitus

Diabetes mellitus substantially increases the risk of recurrent cardiovascular events following myocardial infarction and is associated with poorer clinical outcomes. Intensive management of cardiovascular risk factors, including lipid abnormalities, hypertension, and hyperglycemia, is therefore essential. Contemporary evidence supports the incorporation of cardioprotective glucose-lowering therapies, particularly sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists, in appropriately selected patients because of their demonstrated cardiovascular benefits.

Clinical pharmacists contribute by optimizing antidiabetic therapy, identifying potential interactions between glucose-lowering and cardiovascular medications, educating patients regarding medication adherence, and monitoring for adverse effects such as hypoglycemia and renal dysfunction.

7.3 Patients with Chronic Kidney Disease

Chronic kidney disease (CKD) complicates the management of myocardial infarction because impaired renal function alters drug pharmacokinetics and increases the risk of both thrombotic and bleeding events. Many cardiovascular medications, including anticoagulants and selected antidiabetic agents, require dose adjustment according to renal function. Continuous assessment of kidney function and electrolyte balance is therefore essential during treatment.

Clinical pharmacists assist by recommending appropriate dose modifications, monitoring renal function throughout hospitalization, preventing nephrotoxic drug combinations, and ensuring safe use of medications in accordance with current prescribing recommendations.

7.4 Patients at High Bleeding Risk

Patients with previous bleeding episodes, advanced age, concomitant anticoagulant therapy, thrombocytopenia, or other predisposing factors require careful selection of antithrombotic therapy. Current treatment strategies emphasize individualized assessment of ischemic and bleeding risks to determine the optimal duration and intensity of dual antiplatelet therapy. Regular clinical review and early recognition of bleeding complications are essential to maintain a favorable balance between therapeutic efficacy and patient safety.

Clinical pharmacists support individualized antithrombotic management by evaluating bleeding risk, reviewing concomitant medications, recommending gastroprotective therapy when indicated, and educating patients regarding recognition of bleeding symptoms.



7.5 Future Considerations for High-Risk Populations

Advances in precision medicine, pharmacogenomics, and digital health technologies are expected to further improve individualized management of special patient populations. Integration of genetic testing, biomarker-guided therapy, and clinical decision support systems may enable more accurate selection of pharmacotherapy while reducing preventable adverse drug events. Future research should continue to evaluate personalized treatment strategies for older adults, patients with chronic kidney disease, diabetes mellitus, and other high-risk populations frequently encountered in clinical practice.

FUTURE PERSPECTIVES

The management of myocardial infarction is expected to evolve further through the integration of precision medicine, innovative pharmacological therapies, and digital healthcare technologies. Although guideline-directed medical therapy has substantially improved survival, recurrent cardiovascular events remain a significant clinical challenge, highlighting the need for more individualized and mechanism-based treatment strategies.

One of the most promising developments is the expansion of precision cardiovascular medicine. Pharmacogenomic testing, particularly CYP2C19 genotype-guided selection of P2Y₁₂ inhibitors, has demonstrated the potential to optimize antiplatelet therapy by identifying patients who are less likely to respond to clopidogrel and may benefit from alternative agents. Future incorporation of broader genetic profiling, circulating biomarkers, and predictive risk models is expected to facilitate individualized therapeutic decision-making and improve both efficacy and safety.

Continued development of novel lipid-lowering therapies, including PCSK9 inhibitors, inclisiran, and bempedoic acid, is likely to further reduce residual cardiovascular risk among patients who fail to achieve recommended low-density lipoprotein cholesterol targets despite intensive statin therapy. Similarly, emerging anticoagulants targeting factor XI and factor XIa offer the possibility of maintaining antithrombotic efficacy while reducing bleeding complications, although confirmation from ongoing large-scale clinical trials is required before widespread implementation.

Inflammation has emerged as an important therapeutic target in atherosclerotic cardiovascular disease. Future investigations should focus on identifying patients most likely to benefit from anti-inflammatory therapies while balancing long-term safety, treatment costs, and overall clinical effectiveness. Advances in cardiovascular imaging and biomarker-guided therapy may further support individualized patient selection for these emerging interventions.

Rapid progress in digital health technologies is also expected to transform post-myocardial infarction care. Artificial intelligence, clinical decision support systems, wearable devices, remote patient monitoring, and telemedicine have the potential to improve medication adherence, facilitate early recognition of clinical deterioration, and support proactive management of cardiovascular risk factors. Integration of these technologies into routine clinical practice may enhance long-term patient outcomes while improving healthcare efficiency.

The contribution of clinical pharmacists is anticipated to expand alongside these therapeutic innovations. Pharmacists will play an increasingly important role in implementing pharmacogenomic-guided prescribing, optimizing complex medication regimens, monitoring treatment effectiveness and safety, leading



medication stewardship initiatives, supporting telepharmacy services, and promoting patient-centered cardiovascular care. Their active participation within multidisciplinary healthcare teams will be essential for translating emerging evidence into routine clinical practice.

Future research should prioritize pragmatic clinical trials, real-world effectiveness studies, pharmacoeconomic evaluations, and implementation research to determine the optimal integration of novel therapies into diverse healthcare settings. Collaborative efforts among clinicians, pharmacists, researchers, and policymakers will be essential to improve access to innovative treatments, reduce healthcare disparities, and further decrease the global burden of myocardial infarction.

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Conflict of Interest

The authors declare that there are no conflicts of interest regarding the publication of this manuscript.

Ethical Approval

This article is a narrative review based exclusively on previously published literature. Ethical approval and informed consent were not required.

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