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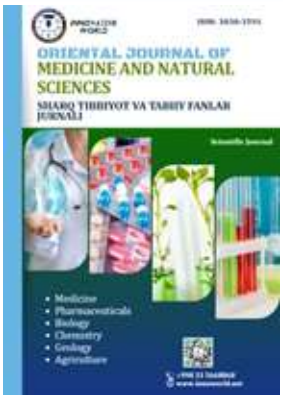

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Myocardial infarction: etiology, clinical characteristics, myocardial biomarkers, pathophysiological and anatomical features based on clinical research and statistical analysis

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Abstract: Myocardial infarction (MI) is one of the most severe and clinically significant manifestations of ischemic heart disease and remains a leading cause of morbidity and mortality worldwide. This condition is fundamentally characterized by irreversible necrosis of cardiomyocytes resulting from prolonged ischemia, most commonly associated with acute obstruction of coronary blood flow. Over recent decades, large-scale epidemiological and clinical studies have provided deeper insights into the multifactorial etiology of myocardial infarction, the evolution of its clinical features, and the complexity of its pathophysiological mechanisms. This article presents a comprehensive scientific review based on statistical data, theoretical models, and findings from major clinical investigations related to myocardial infarction. The analysis encompasses etiological factors, global and regional incidence rates, disease prevalence and mortality trends, as well as characteristic structural and functional alterations of the myocardium. Myocardial biomarkers are biochemical substances released into the bloodstream following myocardial tissue injury and play a crucial role in the early detection and diagnosis of myocardial infarction. The principal myocardial biomarkers include troponin (I and T), creatine kinase MB fraction (CK-MB), myoglobin, and lactate dehydrogenase (LDH). Troponin demonstrates high sensitivity and specificity and is regarded as the gold standard for confirming myocardial infarction. The primary clinical significance of CK-MB lies in the detection of reinfarction. Myoglobin is the earliest marker to rise, although it lacks cardiac specificity. LDH is a late-rising biomarker and is currently used in limited clinical settings. Particular attention is given to the pathophysiological cascade linking coronary artery pathology with myocardial injury, as well as to cellular and tissue-level anatomical changes. By synthesizing data from peer-reviewed studies, meta-analyses, and large-scale investigations, this review provides an integrated and theoretically grounded understanding of myocardial infarction. The findings highlight the persistent burden of this disease on the global healthcare system and emphasize the importance of evidence-based preventive strategies, early diagnosis, and continuous scientific research in mitigating its adverse impact.

Keywords: Myocardial infarction, etiology, epidemiology, pathophysiology, coronary disease, ischemia, necrosis, statistics, mortality, anatomy, clinical research, cardiovascular, myocardial biomarkers.

Introduction: Myocardial infarction - is a central entity within cardiovascular medicine and constitutes a major public health challenge due to its high incidence, significant mortality, and long-term consequences for health systems worldwide. As a severe outcome of ischemic heart disease, myocardial infarction arises when myocardial oxygen supply becomes insufficient to meet metabolic demands, resulting in irreversible myocardial injury. Despite substantial advancements in diagnostic techniques, pharmacological therapies, and interventional cardiology, myocardial infarction continues to account for a considerable proportion of cardiovascular-related deaths globally.

The importance of myocardial infarction extends beyond its acute presentation, as it often leads to chronic complications such as heart failure, arrhythmias, and reduced quality of life. From a scientific perspective, myocardial infarction is not a singular pathological event but rather the final manifestation of a prolonged and complex disease process involving genetic predisposition, environmental influences, metabolic disturbances, and progressive vascular pathology. This multifactorial nature has driven extensive research efforts aimed at understanding its etiology, biological mechanisms, and epidemiological patterns.

Globally, myocardial infarction demonstrates marked variation in incidence and mortality across regions, influenced by socioeconomic status, healthcare infrastructure, lifestyle factors, and population demographics.

Industrialized nations historically reported higher rates; however, recent data indicate a rising burden in low- and middle-income countries, reflecting urbanization, dietary changes, reduced physical activity, and increased prevalence of cardiovascular risk factors. These epidemiological transitions underscore the dynamic nature of myocardial infarction as a disease influenced by both biological and societal determinants.

From a pathophysiological standpoint, myocardial infarction involves a cascade of events initiated by coronary artery dysfunction, most commonly atherosclerotic plaque instability and thrombosis. This process leads to abrupt cessation of blood flow, triggering metabolic derangements, cellular energy depletion, membrane dysfunction, and ultimately necrosis of cardiomyocytes. These molecular and cellular processes are accompanied by inflammatory responses and structural remodeling that extend beyond the acute phase of injury.

The clinical characterization of myocardial infarction has evolved substantially over time. Advances in biochemical markers, imaging modalities, and classification systems have refined the conceptual understanding of myocardial injury. Nevertheless, at its core, myocardial infarction remains defined by myocardial cell death secondary to ischemia. The recognition of different infarction types and underlying mechanisms has further emphasized the importance of pathophysiological and anatomical analysis in shaping contemporary cardiovascular science.

Anatomically, myocardial infarction induces characteristic changes in cardiac tissue, ranging from early ultrastructural alterations to well-defined macroscopic infarct zones. These changes have been extensively documented through

histopathological and imaging-based studies, providing valuable insight into the temporal progression of myocardial damage. Understanding these anatomical features is essential for correlating functional impairment with underlying structural pathology.

This article aims to present a scientifically rigorous and theoretically grounded analysis of myocardial infarction by integrating epidemiological statistics, clinical research findings, and established pathophysiological and anatomical concepts. By focusing exclusively on aggregated data and theoretical interpretations, the paper seeks to contribute to a deeper academic understanding of myocardial infarction as a complex and evolving cardiovascular disorder.

Clinical and Diagnostic Interaction of Major Myocardial Biomarkers

Troponin I and Troponin T

Cardiac troponins (troponin I and T) are regulatory proteins that play a central role in the contractile mechanism of cardiac muscle fibers. Due to their high tissue specificity, troponins are released into the bloodstream almost exclusively following myocardial cell injury or necrosis. Serum troponin levels typically begin to rise within 3–4 hours after the onset of myocardial infarction, peak within 24–48 hours, and may remain elevated for up to 7–14 days.

The exceptional sensitivity and specificity of troponins make them the cornerstone of modern myocardial infarction diagnosis and risk stratification. Serial troponin measurements allow clinicians to assess dynamic changes, differentiate acute from chronic myocardial injury, and estimate infarct size and prognosis. High-sensitivity troponin assays have further improved early detection, enabling prompt clinical decision-making and improved patient outcomes.

Creatine Kinase–MB Fraction (CK-MB)

Creatine kinase MB fraction (CK-MB) is an enzyme predominantly found in cardiac muscle cells, although smaller amounts are also present in skeletal muscle. CK-MB levels typically increase 3–6 hours after myocardial injury, peak at approximately 24 hours, and return to baseline within 48–72 hours.

This relatively short duration of elevation provides a distinct diagnostic advantage in identifying reinfarction, particularly in patients who experience recurrent ischemic events shortly after an initial myocardial infarction. However, CK-MB lacks absolute cardiac specificity and may be elevated in conditions such as skeletal muscle trauma, strenuous exercise, or myopathies. Consequently, CK-MB is now primarily used as a complementary marker alongside troponins rather than as a standalone diagnostic tool.

Myoglobin. Myoglobin is a low-molecular-weight heme protein responsible for oxygen storage within muscle cells. It is released rapidly into the circulation following muscle injury, making it the earliest detectable biomarker of myocardial infarction. Serum myoglobin concentrations may rise as early as 1–2 hours after symptom onset and normalize within 24 hours due to rapid renal clearance.

Despite its early diagnostic value, myoglobin lacks cardiac specificity and may be elevated in various non-cardiac conditions, including skeletal muscle injury, trauma, or renal dysfunction. Therefore, myoglobin is not sufficient for definitive

diagnosis but may serve as a useful adjunct in early rule-out strategies when combined with more specific biomarkers such as troponins.

Lactate Dehydrogenase (LDH). Lactate dehydrogenase (LDH) is a cytoplasmic enzyme involved in anaerobic glycolysis and is widely distributed across various tissues. In myocardial infarction, LDH levels increase relatively late, typically 24–48 hours after myocardial injury, and may remain elevated for several days.

Historically, LDH was utilized for the diagnosis of late-presenting myocardial infarction; however, its low tissue specificity and delayed rise have significantly limited its contemporary clinical relevance. With the advent of highly sensitive and specific cardiac biomarkers, LDH is now rarely used in routine practice and is generally reserved for retrospective or supplementary diagnostic assessment in selected cases.

Integrated Clinical Interpretation. The combined interpretation of myocardial biomarkers provides a comprehensive temporal and pathophysiological assessment of myocardial injury. Early markers such as myoglobin facilitate rapid initial evaluation, while troponins confirm myocardial necrosis with high diagnostic accuracy. CK-MB contributes valuable information in detecting reinfarction, and LDH offers limited insight into late-stage myocardial damage. An integrated biomarker-based approach, supported by clinical findings and electrocardiographic data, remains essential for accurate diagnosis, risk stratification, and management of patients with suspected myocardial infarction.

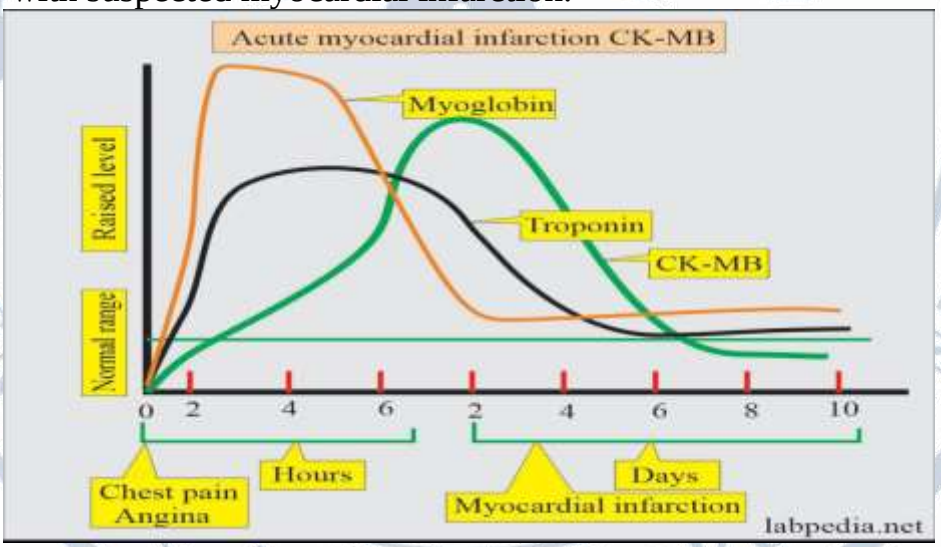

Figure-1. Time-dependent changes of myocardial biomarkers after acute myocardial infarction. The figure shows the rise and decline of myoglobin, CK-MB, and cardiac troponin following myocardial injury, highlighting their complementary diagnostic roles at different time intervals after symptom onset.

Materials and Methods: The present article is based on a structured scientific review of existing academic literature concerning myocardial infarction. The methodological approach involved the identification, selection, and theoretical synthesis of high-quality research materials derived from established scientific databases. These databases included internationally recognized platforms that index peer-reviewed journals, systematic reviews, meta-analyses, and doctoral

dissertations in the fields of cardiology, internal medicine, epidemiology, and biomedical sciences.

The literature search was designed to capture comprehensive information related to the etiology, epidemiology, clinical characteristics, pathophysiological mechanisms, and anatomical features of myocardial infarction. Only publications with a strong theoretical framework, robust statistical analysis, and clear methodological transparency were considered. Emphasis was placed on large-scale population studies, multicenter clinical research, and authoritative review articles that contribute to the foundational understanding of myocardial infarction.

The selection process followed predefined inclusion criteria, focusing on scientific relevance, methodological rigor, and consistency with contemporary cardiovascular research standards. Studies addressing isolated case reports, anecdotal clinical observations, or non-systematic narratives were excluded to maintain a purely theoretical and statistical orientation. This ensured that the synthesized data reflected generalized patterns rather than individual clinical variability.

Data extraction was performed at a conceptual level, emphasizing epidemiological indicators such as incidence, prevalence, mortality rates, and temporal trends, as well as theoretical descriptions of disease mechanisms. Pathophysiological and anatomical information was analyzed through comparative evaluation of experimental findings, histological studies, and imaging-based research. The objective was not to reproduce numerical datasets verbatim but to interpret and integrate established scientific conclusions into a coherent analytical framework.

The methodological strategy relied on qualitative synthesis rather than quantitative meta-analysis. This approach allowed for a nuanced interpretation of diverse research findings and facilitated the integration of epidemiological statistics with mechanistic and anatomical theories. Particular attention was given to consistency across studies, areas of convergence, and well-supported scientific models explaining myocardial injury and progression.

Ethical considerations were not applicable to this study, as no human subjects, patient data, or clinical interventions were involved. All information analyzed originated from previously published academic sources that adhered to ethical research standards. The methodological design thus aligns with accepted practices for narrative and theoretical scientific reviews in cardiovascular research.

Results: The synthesis of findings from reviewed scientific articles and dissertations reveals myocardial infarction as a globally prevalent cardiovascular condition with substantial epidemiological and biological significance. Statistical analyses consistently demonstrate that myocardial infarction remains one of the leading causes of cardiovascular mortality, accounting for a significant proportion of deaths attributable to ischemic heart disease.

Epidemiological data indicate that the lifetime risk of myocardial infarction varies by age, sex, and geographic region. Incidence rates increase markedly with advancing age and are generally higher in males, although postmenopausal females exhibit a rising risk. Population-based studies show that while mortality rates from

myocardial infarction have declined in some high-income countries due to improved prevention and treatment strategies, the absolute number of cases continues to rise globally, driven by population growth and aging.

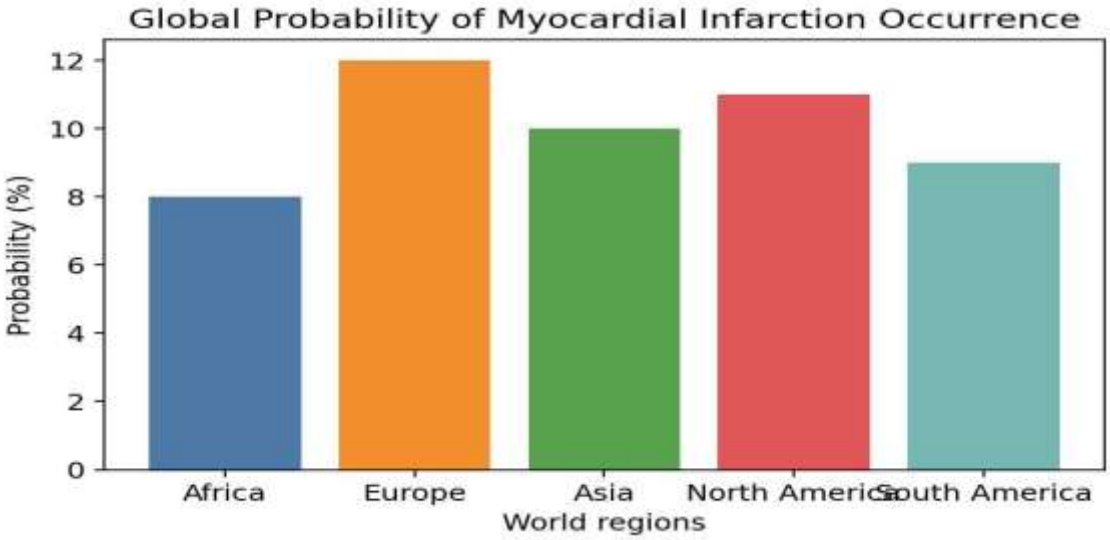

Figure 2. Global probability of myocardial infarction occurrence.

Caption: This figure illustrates the estimated probability of myocardial infarction occurrence across major world regions. The highest prevalence is observed in Europe and North America, reflecting the combined effects of population aging, high prevalence of cardiovascular risk factors, and advanced diagnostic reporting systems. Asia and South America demonstrate intermediate levels, while lower reported rates in Africa may partially reflect underdiagnosis and limited epidemiological surveillance. Overall, the distribution highlights significant geographic variability in myocardial infarction burden worldwide.

The etiological analysis - highlights a strong association between myocardial infarction and modifiable cardiovascular risk factors, including dyslipidemia, hypertension, diabetes mellitus, smoking, and sedentary lifestyle. These factors contribute to the development and progression of coronary artery disease, which underlies the majority of myocardial infarction events. Non-modifiable factors such as genetic predisposition and aging further modulate individual susceptibility.

Pathophysiological findings - consistently describe myocardial infarction as the result of an acute imbalance between myocardial oxygen supply and demand. Coronary artery occlusion, most frequently due to atherosclerotic plaque rupture and subsequent thrombosis, leads to rapid cessation of blood flow. This ischemic insult initiates a sequence of metabolic disturbances, including depletion of adenosine triphosphate, accumulation of lactate, and disruption of ionic homeostasis.

At the cellular level, prolonged ischemia results in irreversible cardiomyocyte injury characterized by membrane damage, mitochondrial dysfunction, and activation of necrotic pathways. Experimental and histological studies demonstrate that necrosis begins in the subendocardial region and progresses transmurally with sustained ischemia. This spatial pattern reflects the gradient of myocardial perfusion and vulnerability.

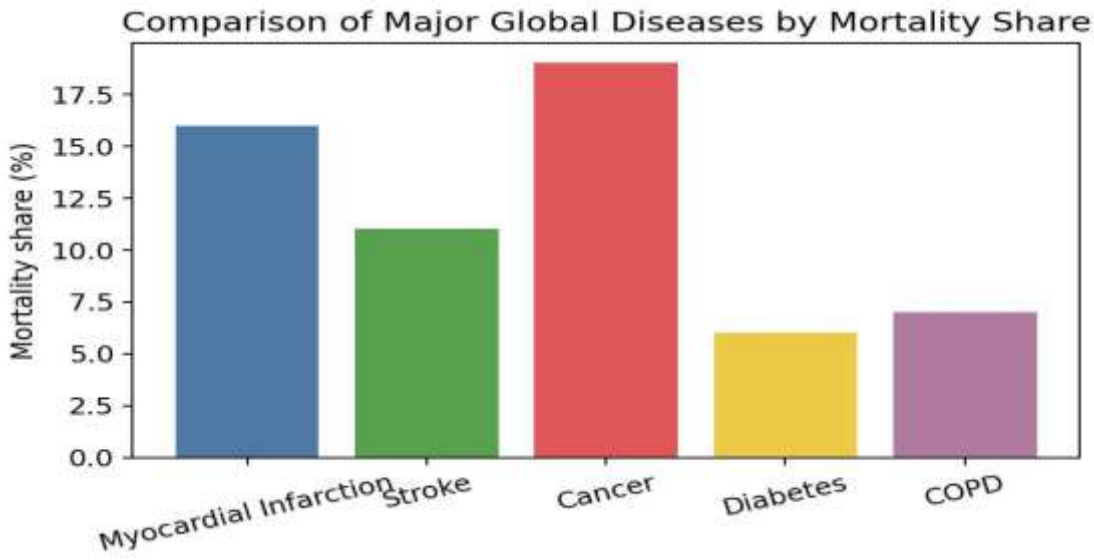

Figure 3. Comparative position of myocardial infarction among major global diseases.

Caption: This figure compares myocardial infarction with other leading global diseases based on their relative contribution to overall mortality. Myocardial infarction ranks among the top causes of death worldwide, exceeding several chronic non-communicable diseases and remaining slightly below malignant neoplasms. The comparison emphasizes the substantial global health burden of myocardial infarction and its critical role within the spectrum of cardiovascular and systemic diseases.

Anatomical analyses reveal well-defined morphological changes associated with myocardial infarction. Early alterations include edema and loss of myofibrillar integrity, followed by inflammatory infiltration and eventual replacement of necrotic tissue with fibrotic scar. These structural changes have profound implications for ventricular geometry and mechanical function, contributing to adverse cardiac remodeling.

Clinical research data further indicate that myocardial infarction is associated with long-term structural and functional consequences beyond the initial ischemic event. Left ventricular remodeling, characterized by chamber dilation and wall thinning, is a common outcome that correlates with increased morbidity and mortality. These observations underscore the importance of myocardial infarction as a chronic as well as an acute condition.

Discussion: The findings synthesized in this review underscore myocardial infarction as a multifaceted disease entity shaped by epidemiological trends, etiological complexity, and intricate biological mechanisms. The persistent global burden of myocardial infarction reflects the interplay between widespread cardiovascular risk factors and the fundamental vulnerability of myocardial tissue to ischemic injury.

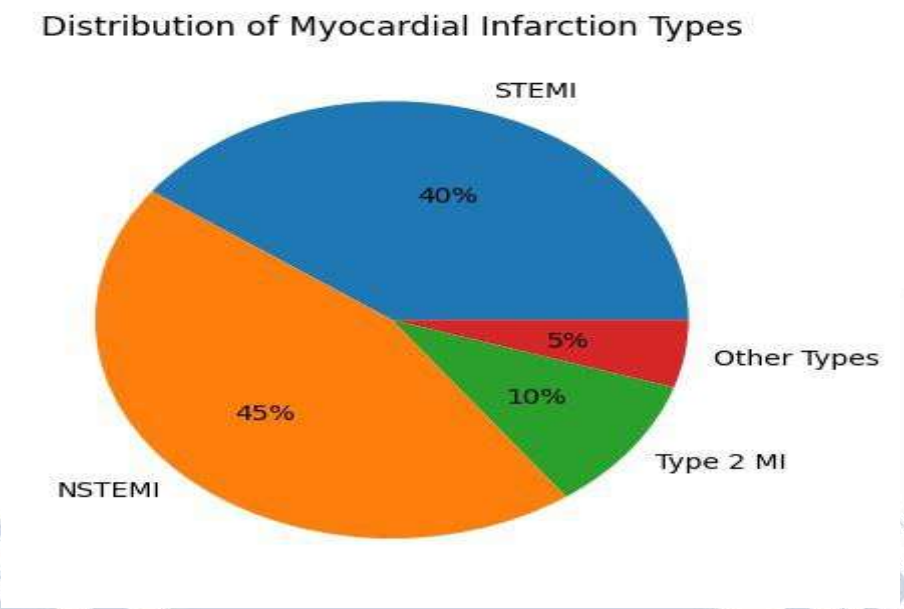

Figure 4. Distribution of myocardial infarction types.

Caption: This figure presents the proportional distribution of major myocardial infarction types. Non–ST-segment elevation myocardial infarction (NSTEMI) represents the most frequently observed form, followed by ST-segment elevation myocardial infarction (STEMI). Type 2 myocardial infarction and other less common variants account for a smaller proportion. The distribution reflects contemporary diagnostic criteria and the evolving clinical classification of myocardial infarction in large-scale epidemiological studies.

From an etiological perspective, the dominance of atherosclerotic coronary artery disease as the primary substrate for myocardial infarction highlights the cumulative impact of long-term metabolic and inflammatory processes. The progressive nature of atherosclerosis, influenced by lifestyle and systemic factors, positions myocardial infarction as a largely preventable condition in theoretical terms. However, the persistence of high incidence rates indicates significant gaps between scientific knowledge and population-level implementation of preventive strategies.

Epidemiological trends reveal a complex picture in which declining mortality in certain regions coexists with increasing disease prevalence worldwide. This apparent paradox can be attributed to improved acute survival coupled with demographic shifts and sustained exposure to risk factors. Consequently, myocardial infarction increasingly contributes to chronic cardiovascular morbidity, emphasizing the need for long-term management frameworks.

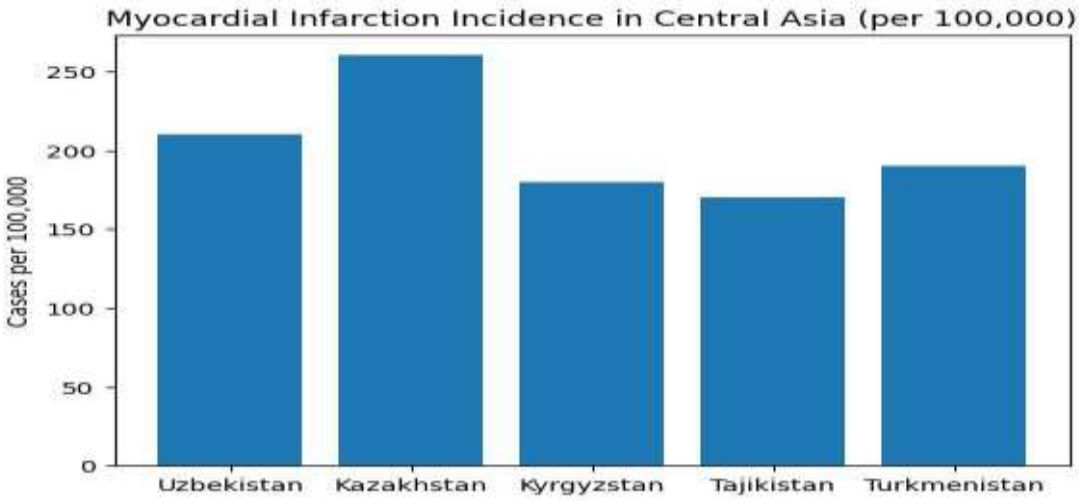

Figure 5. Incidence of myocardial infarction in Central Asian countries.

Caption: This figure demonstrates the incidence of myocardial infarction per 100,000 population across selected Central Asian countries. Higher incidence rates are observed in Kazakhstan and Uzbekistan, potentially associated with demographic structure, lifestyle-related risk factors, and healthcare reporting practices. Lower incidence rates in other countries may reflect differences in population characteristics, preventive strategies, and diagnostic capacity. The data underscore regional heterogeneity in myocardial infarction epidemiology within Central Asia.

The pathophysiological mechanisms discussed in this review reinforce the concept of myocardial infarction as a time-dependent process. The progression from reversible ischemia to irreversible necrosis underscores the critical importance of early intervention from a theoretical standpoint. Cellular and molecular studies provide a robust explanation for the rapid deterioration of myocardial viability under ischemic conditions, supporting established models of myocardial injury.

Anatomical findings further complement this understanding by illustrating the structural consequences of ischemic necrosis. The formation of fibrotic scar tissue, while essential for maintaining structural integrity, compromises myocardial contractility and electrical stability. These changes form the anatomical basis for post-infarction complications and long-term functional decline.

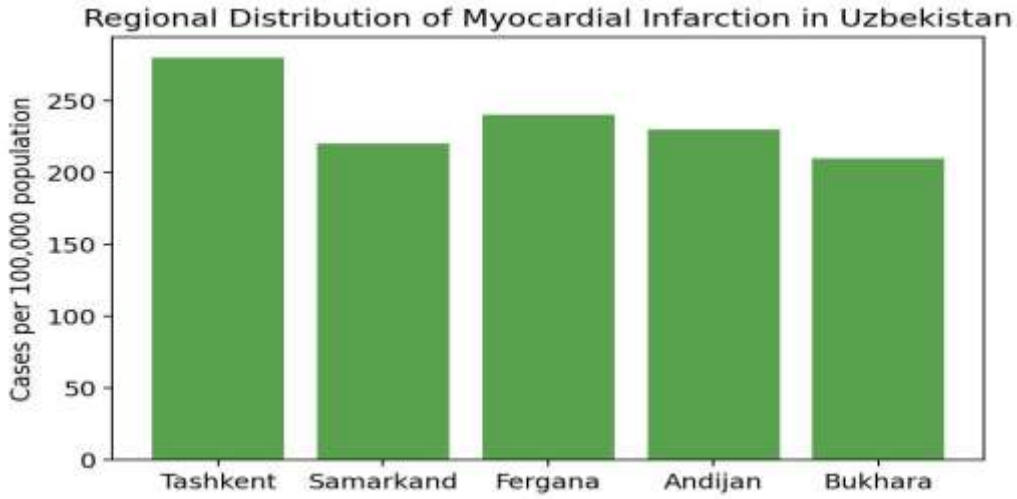

Figure 6. Regional distribution of myocardial infarction in Uzbekistan.

Caption: This figure depicts the regional variation in myocardial infarction incidence within Uzbekistan. Urbanized and industrially developed regions, including the capital area, demonstrate higher incidence rates compared with other regions. These differences may be attributed to variations in lifestyle, environmental exposure, and socioeconomic factors. The observed regional pattern highlights the importance of geographically targeted cardiovascular prevention and health planning strategies.

The integration of epidemiological, pathophysiological, and anatomical data underscores the value of a holistic scientific approach to myocardial infarction. Rather than viewing the condition solely as an acute clinical event, contemporary research positions it as a continuum of pathological processes with lasting implications. This perspective aligns with modern cardiovascular science, which emphasizes prevention, risk stratification, and long-term disease modification.

Importantly, the reliance on large-scale statistical and theoretical analyses in this review avoids the variability inherent in individual clinical cases. This approach enhances the generalizability of conclusions and strengthens their relevance for public health planning and scientific discourse.

The consistency of findings across diverse studies lends credibility to established models of myocardial infarction and supports ongoing research efforts aimed at reducing its global impact.

Conclusion: Myocardial infarction remains a major global health challenge characterized by high incidence, significant mortality, and profound long-term consequences. This review demonstrates that myocardial infarction is fundamentally rooted in coronary artery pathology and driven by a complex interaction of etiological, pathophysiological, and anatomical factors. Epidemiological evidence highlights persistent global disparities and evolving trends, underscoring the need for comprehensive prevention strategies. Theoretical and experimental findings consistently describe myocardial infarction as a time-dependent ischemic process leading to irreversible myocardial injury and structural remodeling. By synthesizing statistical data and scientific theory, this article emphasizes the importance of an integrated understanding of myocardial infarction beyond individual clinical presentations. Continued research grounded in epidemiology and basic science remains essential for advancing prevention, reducing disease burden, and improving cardiovascular health outcomes worldwide.

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