

MYOCARDIAL HEART DISEASE

Kokand University, Andijan Branch

Medical Faculty, General Medicine Student (Group 24–35)

Ibrokhimjonova Mumtozbeqim Sherzod kizi

Phone: +998 91 607 40 80

Email: mumtozbeqimibrokhimjonova7@gmail.com

Abstract: Pathologies of the Myocardium: Pathophysiology, Classification, and Clinical Consequences This summary addresses the pathologies of the myocardium—the muscular layer of the heart responsible for its pump function. Myocardial diseases are classified based on their underlying etiological and pathophysiological mechanism: ischemic (e.g., myocardial infarction, leading to necrosis and scarring), inflammatory (myocarditis, characterized by immune infiltration and fibrosis), and primary structural (cardiomyopathies—dilated, hypertrophic, restrictive), often with a genetic origin. All these pathological processes inevitably lead to impaired systolic or diastolic function, resulting in the development of Chronic Heart Failure (CHF), arrhythmias (including sudden cardiac death), and thromboembolic complications. Diagnosis requires a comprehensive approach utilizing specific biomarkers (troponins) and imaging modalities (Echocardiography, Cardiac MRI).

Keywords: Myocardium; Myocardial Infarction (MI); Ischemic Heart Disease (IHD); Myocarditis; Cardiomyopathy; Heart Failure (HF); Arrhythmia; Necrosis; Fibrosis; Troponin; Echocardiography (Echo); Ejection Fraction (EF); Cardiac Dysfunction; Coronary Artery Disease; Cardiac Biomarkers; Cardiac Remodeling

Myocardium

The myocardium is the middle, muscular layer of the heart wall responsible for the contraction and pumping function of the heart. Because it generates the force required to circulate blood throughout the body, any disorder affecting the myocardium directly compromises the heart's ability to function effectively.

One of the most significant myocardial conditions is **myocardial infarction**, which represents an acute and life-threatening form of coronary artery disease. It occurs when the blood supply to a particular region of the myocardium is partially or completely blocked, most commonly due to a thrombus in a coronary artery. As a result, the affected cardiac tissue undergoes necrosis. Clinically, myocardial infarction typically presents with severe, pressure-like or burning chest pain radiating to the left arm, shoulder, or jaw, along with shortness of breath, cold sweating, and a pronounced feeling of anxiety or fear.

Another important condition involving the myocardium is **myocarditis**, an inflammatory disease of the heart muscle. It can develop as a complication of viral or bacterial infections, such as influenza or COVID-19. Inflammation weakens the cardiac muscle and disrupts its ability to contract. Patients often experience chest pain, dyspnea, arrhythmias, profound fatigue, and sometimes fever, and in severe cases myocarditis may progress to heart failure.

A broader category of myocardial disorders is **cardiomyopathies**, which represent structural and functional abnormalities of the heart muscle that are not caused by coronary artery disease, hypertension, or valvular defects. The major types include dilated cardiomyopathy, in which the heart chambers become enlarged and the myocardial walls thin out, leading to impaired contractility; hypertrophic cardiomyopathy, characterized by abnormal thickening of the myocardial walls that reduces the filling capacity of the ventricles; and restrictive cardiomyopathy, where the ventricular walls become rigid and less compliant, limiting the heart's ability to fill properly. These conditions commonly manifest with symptoms of heart failure, including shortness of breath, peripheral edema, exercise intolerance, and various forms of arrhythmia.

Classification and Pathophysiological Mechanisms

Pathologies of the Myocardium: A Scientific Overview

Myocardial diseases can be classified according to the primary pathophysiological mechanisms that lead to cardiomyocyte dysfunction and, consequently, to the development of heart failure. These mechanisms include ischemic injury, inflammatory destruction, primary structural abnormalities of the myocardium, and electrical instability.

Ischemic Heart Disease and Myocardial Infarction

The fundamental mechanism in ischemic heart disease is ischemia — an imbalance between the oxygen demand of the myocardium and the oxygen supplied through the coronary arteries. This is most commonly caused by atherosclerotic narrowing of the coronary vessels. An acute myocardial infarction develops when thrombotic occlusion of a coronary artery leads to the complete cessation of blood flow in a specific myocardial region. As a result, the affected cardiomyocytes undergo necrosis. Cell death triggers the release of specific biomarkers, such as cardiac troponins, and the necrotic tissue is later replaced by fibrotic scar tissue, which irreversibly reduces the contractile capacity of the heart muscle.

Myocarditis (Inflammatory Myocardial Injury)

Myocarditis represents an inflammatory process that may be caused by infectious agents—most commonly viruses such as Coxsackievirus, adenoviruses, and parvovirus B19—or by autoimmune mechanisms. Viral invasion of cardiomyocytes initiates an immune response characterized by infiltration of lymphocytes, macrophages, and neutrophils. This inflammatory cascade leads to progressive lysis of myocardial cells, disruption of electrical conduction that increases the risk of arrhythmias, and the development of myocardial edema followed by fibrotic remodeling. Over time, these changes may result in dilated cardiomyopathy and chronic heart failure.

Cardiomyopathies (Primary Myocardial Disorders)

Cardiomyopathies are defined as primary structural and functional disorders of the myocardium that are not caused by ischemic heart disease, hypertension, or valvular defects. They are classified based on characteristic morphological and functional abnormalities:

Type of Cardiomyopathy	Morphological Change	Functional Defect	Etiology (Causes)

Type of Cardiomyopathy	Morphological Change	Functional Defect	Etiology (Causes)
Dilated Cardiomyopathy (DCM)	Marked dilation of the ventricles with thinning of the myocardial walls.	Systolic dysfunction (decreased contractile capacity).	Idiopathic, genetic, post-viral, alcohol- or toxin-induced.
Hypertrophic Cardiomyopathy (HCM)	Pathological thickening of the myocardium, often asymmetric.	Diastolic dysfunction; may include left ventricular outflow tract obstruction.	Primarily genetic mutations in sarcomeric proteins.
Restrictive Cardiomyopathy (RCM)	Increased stiffness and reduced compliance of the ventricular walls, usually with normal thickness.	Severe diastolic dysfunction (impaired filling).	Amyloidosis, sarcoidosis, hemochromatosis, infiltrative diseases.

Arrhythmias (Disorders of Cardiac Rhythm)

Myocardial dysfunction frequently leads to electrical instability of the heart. Damaged or structurally altered cardiomyocytes, along with areas of fibrosis, create a substrate for rhythm disturbances. These include abnormalities of automaticity caused by changes in pacemaker cells or the emergence of ectopic foci. They also involve re-entry mechanisms in which circular pathways of impulse conduction form, often at the border zones between necrotic and viable tissue—such as in post-infarction scars. Such re-entry circuits can trigger life-threatening arrhythmias, including ventricular fibrillation, which is the most common cause of sudden cardiac death.

Conclusion on Myocardial Diseases

The myocardium—the central muscular layer of the heart—plays a fundamental role in maintaining its pumping function. Damage to the myocardium underlies many severe cardiovascular disorders, all of which share a common outcome: a progressive decline in the heart’s ability to pump blood effectively.

Myocardial diseases are generally classified into three major groups based on their underlying pathophysiology.

Ischemic disorders, such as myocardial infarction, arise from impaired blood supply to the myocardium, leading to cardiomyocyte necrosis and the subsequent formation of fibrotic scar tissue.

Inflammatory conditions, most notably myocarditis, occur when the immune system—often activated by an infectious trigger—attacks myocardial cells, resulting in inflammation, edema, and ultimately fibrotic remodeling.

Primary or structural disorders, including dilated, hypertrophic, and restrictive cardiomyopathies, are characterized by intrinsic abnormalities of myocardial structure and function, frequently associated with genetic or idiopathic mechanisms.

Clinical Significance

Despite differing etiologies, these conditions converge on a common and most serious consequence: the development of chronic heart failure. Furthermore, myocardial injury creates a substrate for arrhythmias, including life-threatening ventricular tachyarrhythmias, as well as thromboembolic complications.

Modern diagnostic approaches rely on a combination of imaging modalities—such as echocardiography and cardiac MRI—to evaluate structure and function, and laboratory biomarkers such as cardiac troponins and NT-proBNP to detect cellular damage and assess the degree of cardiac stress.

In conclusion, myocardial health is vital for the overall functioning of the body. Myocardial dysfunction is not only a major cause of global cardiovascular mortality but also a central focus of ongoing research in contemporary cardiology.

Diagnosis of Myocardial Diseases and Their Potential Complications

Diagnosis of Myocardial Pathologies

Accurate identification of myocardial damage requires a comprehensive combination of instrumental and laboratory diagnostic methods.

Electrocardiography (ECG) records the electrical activity of the heart and helps detect ischemia (ST-T segment changes), necrosis (pathological Q wave in myocardial infarction), various arrhythmias, and signs of myocardial hypertrophy typical of certain cardiomyopathies.

Echocardiography (EchoCG) is the key imaging method for assessing cardiac structure and function. It allows evaluation of the ejection fraction (EF), the main parameter of systolic performance; dilation of cardiac chambers, especially in dilated cardiomyopathy; myocardial wall thickness in hypertrophic cardiomyopathy; and regional wall-motion abnormalities, which commonly occur after myocardial infarction.

Laboratory biomarkers are central to myocardial diagnosis. Cardiac troponins I and T are highly specific markers released into the bloodstream during cardiomyocyte necrosis and represent the most important indicators of myocardial infarction and acute myocarditis. NT-proBNP is widely used to detect and monitor heart failure by reflecting ventricular wall stress.

Coronary angiography provides direct visualization of the coronary arteries and is essential for confirming atherosclerotic stenosis that leads to myocardial ischemia and infarction.

Cardiac MRI with contrast enhancement is the “gold standard” for detailed assessment of myocardial tissue characteristics. It enables direct visualization of post-infarction scar tissue, edema, and active inflammation in myocarditis.

Endomyocardial biopsy, though rarely required, is used to obtain histological confirmation in specific cases, such as giant-cell myocarditis or infiltrative cardiomyopathies like amyloidosis or sarcoidosis.

Major Complications of Myocardial Diseases

Myocardial injury can trigger a cascade of life-threatening complications.

Heart failure is the most common consequence. A weakened myocardium is unable to generate adequate cardiac output to meet metabolic demands, resulting in dyspnea, edema, exercise intolerance, and fatigue.

Arrhythmias develop when damaged or inflamed myocardial tissue disrupts the heart's normal electrical conduction. Life-threatening ventricular tachycardia and ventricular fibrillation are leading causes of sudden cardiac death. Atrial fibrillation, although less acute, impairs ventricular filling and significantly increases the risk of thromboembolic events.

Thromboembolic complications arise due to stagnant blood flow within dilated cardiac chambers, particularly in dilated cardiomyopathy or following large myocardial infarctions. Mural thrombi may dislodge and cause ischemic stroke or occlusion of peripheral arteries.

Cardiogenic shock is a critical condition resulting from severely impaired myocardial contractility, often due to extensive myocardial infarction. It leads to inadequate tissue perfusion, profound hypotension, and requires immediate intensive resuscitation.

A rare but catastrophic complication of transmural myocardial infarction is **myocardial wall rupture**, which leads to acute cardiac tamponade caused by blood accumulation in the pericardial cavity, and is almost always fatal without immediate intervention.

Conclusion on Myocardial Diseases

In conclusion, myocardial diseases represent a major group of cardiovascular pathologies that significantly impair the heart's ability to function as an effective pump. The myocardium—the central muscular layer of the heart—is essential for maintaining adequate cardiac output. Damage to this layer underlies many severe and life-threatening cardiac conditions.

Myocardial disorders can be broadly divided into three major groups based on their pathophysiological mechanisms.

Ischemic diseases, such as myocardial infarction, result from impaired coronary blood flow, leading to irreversible cardiomyocyte necrosis and subsequent scar formation.

Inflammatory diseases, including myocarditis, arise when the immune system—often activated by viral infection—attacks myocardial cells, causing inflammation, edema, and progressive fibrosis.

Primary or structural diseases, represented by dilated, hypertrophic, and restrictive cardiomyopathies, involve intrinsic abnormalities of myocardial tissue, frequently associated with genetic or idiopathic origins.

Despite their diverse etiologies, these conditions share common and severe clinical consequences. The most significant of these is the development of chronic heart failure, which reflects the progressive decline of myocardial function. Additionally, myocardial damage creates a substrate for arrhythmias—including life-threatening ventricular tachyarrhythmias—and increases the risk of thromboembolic events.

Modern diagnostic approaches rely on a combination of imaging modalities such as echocardiography and cardiac MRI to assess structure and function, and on laboratory markers such as cardiac troponins and NT-proBNP to detect cellular injury and evaluate hemodynamic stress.

Overall, maintaining myocardial health is crucial for the well-being of the entire organism. Myocardial dysfunction remains a leading cause of morbidity and mortality and continues to be an area of active scientific investigation within contemporary cardiology.

Diagnosis and Complications of Myocardial Diseases

Accurate diagnosis of myocardial damage requires a comprehensive approach combining instrumental and laboratory methods.

Electrocardiography (ECG) records the electrical activity of the heart and allows the detection of ischemia (ST-T segment changes), necrosis (pathological Q waves in myocardial infarction), arrhythmias, and signs of myocardial hypertrophy as seen in cardiomyopathies.

Echocardiography (EchoCG / Cardiac Ultrasound) is a key method for assessing cardiac structure and function. It enables evaluation of ejection fraction, which is the primary indicator of the heart's pumping capacity. Reduced ejection fraction indicates systolic dysfunction, commonly observed in dilated cardiomyopathy or extensive myocardial infarction. Echocardiography also allows assessment of chamber size, wall thickness, and regional wall motion abnormalities.

Laboratory biomarkers include cardiac troponins I and T, which are specific proteins released into the blood during cardiomyocyte necrosis and are the most important markers of acute myocardial infarction and myocarditis. N-terminal pro-B-type natriuretic peptide (NT-proBNP) is used to diagnose and monitor heart failure.

Coronary angiography is an invasive procedure to visualize the coronary arteries and confirm atherosclerotic stenosis causing myocardial infarction.

Cardiac magnetic resonance imaging (MRI) with contrast enhancement is the gold standard for detailed evaluation of myocardial structure and tissue characterization. It can detect post-infarction fibrotic scars, edema, and inflammation in active myocarditis, as well as infiltrative changes in specific cardiomyopathies.

Endomyocardial biopsy involves sampling small fragments of myocardial tissue and is rarely performed, mainly to confirm specific forms of myocarditis, such as giant cell myocarditis, or infiltrative cardiomyopathies.

Damage to the myocardium can lead to a cascade of potentially life-threatening complications.

Heart failure (HF) is the most common consequence. A weakened myocardium cannot generate sufficient cardiac output to meet the body's needs, resulting in dyspnea, peripheral edema, fatigue, and exercise intolerance.

Arrhythmias occur when injured or inflamed myocardial areas disrupt normal electrical conduction. Ventricular tachycardia and fibrillation are the most dangerous, often leading to sudden cardiac death. Atrial fibrillation impairs ventricular filling and increases the risk of thromboembolism.

Thromboembolic complications arise when blood stasis occurs in dilated cardiac chambers, especially in dilated cardiomyopathy or after extensive myocardial infarction. Mural thrombi may detach, causing stroke or peripheral arterial occlusion.

Cardiogenic shock is a critical condition caused by sudden severe decline in myocardial contractility, commonly after large myocardial infarction. It leads to inadequate circulation, hypotension, and requires immediate intensive care.

Myocardial wall rupture is a rare but catastrophic complication of transmural myocardial infarction, resulting in cardiac tamponade due to pericardial blood accumulation and rapid death without emergency intervention.

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