

MYOCARDITIS: ETIOLOGY, PATHOPHYSIOLOGY, CLINICAL FEATURES, AND MANAGEMENT**Abduraimova Chinora Mirzaqul qizi**

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Abstract: Myocarditis is an inflammatory disease of the myocardium that can result from a wide range of infectious, autoimmune, toxic, and idiopathic causes. The clinical presentation of myocarditis is highly variable, ranging from mild, self-limiting symptoms to severe acute heart failure, life-threatening arrhythmias, and sudden cardiac death. Viral infections are considered the most common etiological factor, although autoimmune mechanisms and drug-induced myocardial injury are also increasingly recognized. The pathophysiology involves direct myocardial injury and immune-mediated inflammatory responses leading to myocyte necrosis and ventricular dysfunction. Early diagnosis remains challenging due to nonspecific clinical manifestations and limitations of diagnostic tools. This article provides a comprehensive overview of the etiology, pathophysiological mechanisms, clinical features, and current approaches to the management of myocarditis, emphasizing the importance of early recognition and individualized treatment strategies.

Keywords: myocarditis, myocardial inflammation, cardiomyopathy, heart failure, viral infection, autoimmune disease, arrhythmia, cardiac MRI, endomyocardial biopsy.

Myocarditis is a heterogeneous inflammatory disorder of the myocardium characterized by myocyte injury and interstitial inflammatory infiltration. The disease may occur as an isolated cardiac condition or as part of systemic inflammatory or infectious processes. Its clinical importance lies in its unpredictable course, ranging from complete spontaneous recovery to progression toward dilated cardiomyopathy and end-stage heart failure. The etiology of myocarditis is multifactorial. Viral

infections remain the leading cause in developed countries, with common pathogens including Coxsackievirus B, adenovirus, parvovirus B19, and human herpesvirus 6. In recent years, SARS-CoV-2 has also been identified as an important trigger of myocardial inflammation. Bacterial causes are less frequent but may include *Borrelia burgdorferi* (Lyme disease), *Corynebacterium diphtheriae*, and *Mycobacterium tuberculosis*. Protozoal and parasitic infections such as *Trypanosoma cruzi* (Chagas disease) are significant in endemic regions. Non-infectious causes include autoimmune diseases such as systemic lupus erythematosus, rheumatoid arthritis, and giant cell myocarditis. Drug-induced myocarditis may occur due to hypersensitivity reactions to antibiotics, antipsychotics, or chemotherapy agents such as immune checkpoint inhibitors. Toxic exposures including alcohol, cocaine, and heavy metals may also contribute to myocardial inflammation. The pathophysiological process of myocarditis involves a complex interaction between direct myocardial injury and immune-mediated responses. Initially, pathogens or toxins cause cardiomyocyte damage, triggering the activation of innate and adaptive immune systems. This leads to infiltration of inflammatory cells such as macrophages, T lymphocytes, and neutrophils into the myocardium. Cytokine release, including tumor necrosis factor-alpha (TNF- α), interleukins, and interferons, amplifies the inflammatory cascade, resulting in further myocardial injury. Persistent inflammation leads to myocyte necrosis, interstitial edema, and disruption of normal myocardial architecture. Over time, ventricular remodeling may occur, characterized by dilation, reduced contractility, and fibrosis. This structural remodeling is a key mechanism in the progression from acute myocarditis to chronic dilated cardiomyopathy. The clinical presentation of myocarditis is highly variable. Some patients remain asymptomatic, while others present with acute or fulminant heart failure. Common symptoms include chest pain, dyspnea, fatigue, palpitations, and reduced exercise tolerance. In some cases, myocarditis mimics acute coronary syndrome with chest pain and elevated cardiac biomarkers such as troponin. Arrhythmias, including ventricular tachycardia and atrial fibrillation, may be the first manifestation of disease. In severe cases, cardiogenic shock or sudden cardiac death may occur. Physical examination findings are often nonspecific but may include tachycardia, hypotension, signs of heart failure, and new-onset heart murmurs due to functional mitral or tricuspid regurgitation. Diagnosis of myocarditis is challenging due to its nonspecific presentation. Electrocardiography may show ST-segment changes, T-wave inversion, or arrhythmias. Elevated serum cardiac biomarkers indicate myocardial injury but are not specific. Echocardiography is useful for assessing ventricular function and detecting wall motion abnormalities.

CONCLUSION

Myocarditis is a complex and potentially life-threatening inflammatory disease of the myocardium with a wide spectrum of clinical manifestations. Its etiology is diverse, with viral infections representing the most common cause, although autoimmune, toxic, and drug-induced factors also play a significant role. The pathophysiological process involves a combination of direct myocardial injury and immune-mediated inflammatory responses, which may lead to ventricular dysfunction, arrhythmias, and progression to dilated cardiomyopathy. Early diagnosis remains challenging due to nonspecific clinical features; however, advances in cardiac imaging, particularly cardiac magnetic resonance imaging, have significantly improved diagnostic accuracy. Endomyocardial biopsy remains the gold standard in selected cases. Management is primarily supportive and directed toward heart failure treatment, arrhythmia control, and, when appropriate, immunosuppressive or advanced mechanical support therapies. Overall, early recognition, accurate diagnosis, and timely management are essential to improve patient outcomes and reduce complications associated with myocarditis. Further research is needed to better understand its molecular mechanisms and to develop targeted therapeutic strategies.

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