



## Cardio Immunology: Autoimmunity in Acute Myocarditis

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### DESCRIPTION

The most common and self-limited result of a systemic infection with cardiotropic viruses is myocarditis. Patients, however, may experience a temporary or permanent impairment of their ability to pump blood through their hearts, such as acute cardiomyopathy with hemodynamic compromise or severe arrhythmias. In this situation, distinct sources of inflammation are linked to varying dangers from transplantation and mortality. Depending on the degree of cardiac involvement, acute myocarditis is an acute inflammatory myocardial illness with a wide range of clinical symptoms, from asymptomatic to life-threatening presentations. In acute myocarditis, there is compelling evidence of an autoimmune pathogenic process.

Immune Checkpoint Inhibitor (ICI) induced myocarditis is a brand-new kind of myocarditis. In advanced cancer, ICIs are used to "disinhibit" the immune system and increase its vigilance against the disease. With a danger of autoimmune disorders such as myocarditis brought on by an excessively active immune system, this innovative medication class has considerably boosted progression-free survival in advanced non-small-cell lung cancer and quadrupled life expectancy in metastatic melanoma. An inflammatory condition of the heart known as myocarditis has a significant influence on public health. For a precise diagnosis and successful therapy, it is essential to have a thorough grasp of the immunopathogenesis [1].

Our knowledge of how immune cells contribute to inflammatory cardiomyopathy is developing. Experimental animal models have shown the significance of immune cells in the etiology of viral inflammatory cardiomyopathy and viral myocarditis. Conceptually, the pathogenic process of viral inflammatory cardiomyopathy can be broken down into three phases: an acute phase of viral entry into the cell and activation of the innate immune response (last 1–7 days), a subacute phase with activation of the adaptive immune response (last 1–4 weeks), and a chronic phase that can last from months to years and involves delayed or ineffective viral clearance along with chronic inflammation and cardiac remodeling [2].

### Mechanism of autoimmunity in myocarditis

This section will concentrate on the pathophysiology of myocarditis following an acute viral infection, which has been conceived as a multiphase model and recently reviewed by Heymans and colleagues. Viral infection is regarded to be the most researched initiating cause of myocarditis. When viruses or other pathogens directly damage the myocardium, acute injury may result. However, inflammatory chemicals such as cytokines generated during the immune response trigger a cascade of cytolysis, further recruitment of inflammatory cells, and remodeling. The classical innate and adaptive immune system, as well as other cellular and extracellular compartments of the heart, is thought to contribute to the effector and regulatory factors that determine clinical presentation [3].

The innate immune system is triggered by infection. Pattern recognition receptors, such as Toll-Like Receptors (TLRs) and nucleotide-binding oligomerization domain-like receptors, recognize specific molecular patterns of pathogens (referred to as Pathogen-Associated Molecular Patterns, or PAMPs), as well as patterns released from endogenous damaged cells, such as released ATP, S100A8, and other molecules (referred to as Damage-Associated Molecular Patterns, or DAMPs), to activate innate immune. Depending on the pathogen or DAMP, a different type of pattern recognition receptor and downstream signaling may be used. Mast cells, neutrophils, dendritic cells, monocytes, and macrophages are among the innate immune cells that become activated and migrate to the heart as a result. These cells also produce *cytokines*, *chemokines*, *interferons*, and *alarmins*. The two primary inflammatory cell subsets observed in experimental and human myocarditis are monocytes and macrophages. Although the heart's innate immune response is advantageous to the host because of its antiviral properties, excessive or persistent innate immune system activation can result in an exaggerated and/or chronic inflammatory process that causes myocardial deterioration and remodeling, which ultimately results in cardiac dysfunction [4].

CT4<sup>+</sup> T cells have a significant impact on how monocytes differentiate into M1 macrophages, which have proinflammatory

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properties. IFN- $\gamma$ , which is generated by Th1 cells, enhances antigen presentation and macrophages' ability to kill microbes. This process of enhancing the immune response is probably helpful to some extent, but it can be detrimental and result in "collateral damage" if it results in an excessive immunological response, a condition frequently seen in autoimmune illnesses. By activating Ly6Clow, M2 macrophages, on the other hand, encourage fibrosis and repair in the heart while reducing inflammatory response. Additionally, they can be stimulated by Th2-cells that secrete IL-4 and -13. These macrophages are replaced by myofibroblast with profibrotic characteristics when the acute myocarditis develops into a chronic pathological remodeling [5]. There is considerable interest in altering macrophages for therapeutic purposes in cardiovascular illness, particularly in myocarditis and following myocardial infarction, because to their crucial involvement in heart repair with regard to cardiomyocytes mortality and remodeling.

## REFERENCES

1. Richardson P. Report of the 1995 World Health Organization/ International Society and Federation of Cardiology Task Force on the definition and classification of cardiomyopathies. *Circulation*. 1996;93:841-842.
2. Ammirati E, Cipriani M, Moro C, Raineri C, Pini D, Sormani P, et al. Clinical presentation and outcome in a contemporary cohort of patients with acute myocarditis: multicenter Lombardy registry. *Circulation*. 2018;138(11):1088-99.
3. Schultheiss HP, Kuhl U, Cooper LT. The management of myocarditis. *Eur Heart J*. 2011;32(21):2616-2625.
4. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2013;34(33):2636-48.
5. Heidecker B, Ruedi G, Baltensperger N, Gresser E, Kottwitz J, Berg J, et al. Systematic use of cardiac magnetic resonance imaging in MINOCA led to a five-fold increase in the detection rate of myocarditis: a retrospective study. *Swiss Med Wkly*. 2019;149:w20098.