IMMUNE CELLS AS KEY MEDIATORS OF DILATED CARDIOMYOPATHY

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Background: Dilated cardiomyopathy (DCM) is a complex myocardial disorder characterized by left ventricular dilation and systolic dysfunction. Beyond genetic and toxic causes, accumulating evidence points to a pivotal role of immune cells—particularly B cells, T lymphocytes, and macrophages—in disease onset, progression, and tissue remodeling. Chronic inflammation, mediated by these cells, contributes significantly to myocardial dysfunction and poor clinical outcomes.
Objective: To delineate the role of specific immune cell populations in the immunopathogenesis of DCM and assess their diagnostic and therapeutic relevance.
Methods: A structured review of recent experimental and clinical studies was performed, focusing on the role of $CD4^+$ and $CD8^+$ T cells, Th1/Th17 subtypes, B cells, and macrophages. The analysis included their cytokine profiles, mechanisms of antigen recognition, and involvement in myocardial injury and fibrosis.
Results: $CD4^+$ T cells, particularly Th1 and Th17 subsets, contribute to myocardial damage through secretion of IFN-γ and IL-17A, enhancing inflammation and fibrosis. $CD8^+$ cytotoxic T cells induce cardiomyocyte apoptosis via perforin, granzyme, and Fas-ligand pathways. B cells produce autoantibodies targeting β_1-adrenergic receptors, M_2 muscarinic receptors, cardiac myosin, and mitochondrial proteins. These antibodies not only serve as biomarkers but also act pathophysiologically by disrupting intracellular signaling, calcium handling, and inducing apoptosis. Macrophages and dendritic cells orchestrate the immune environment by presenting antigens and producing cytokines such as IL-6 and TNF-α. The persistent activation of these immune cells forms a self-sustaining cycle of myocardial injury.
Conclusions: Immune cells are not merely bystanders but central mediators in the pathogenesis of DCM. Their diagnostic value and potential as therapeutic targets—through immunosuppression, immunoadsorption, and biologic agents—underline the need for a paradigm shift in managing DCM as an immune-mediated disease. Personalized immunological profiling could pave the way for precision medicine in affected patients.