Immunological Disorders & Immunotherapy
ISSN: 2593-8509
OPEN ACCESS Freely available online
Opinion Article

HLA Gene Variants and Genetic Predisposition in Giant Cell Myocarditis

Selena Jonas*

Department of Immunology, Nilai University, Negeri Sembilan, Malaysia

ABOUT THE STUDY

Giant Cell Myocarditis (GCM) is a rare and severe inflammatory heart disease that poses significant challenges for both diagnosis and treatment. This condition is characterized by the infiltration of the heart tissue by large, multinucleated cells, leading to inflammation and subsequent damage. Although GCM is uncommon, its impact on cardiac function can be devastating. The symptoms of GCM can vary, but they often include heart failure symptoms such as shortness of breath, fatigue, chest pain, and irregular heartbeats.

Mechanisms of giant cell myocarditis

Immune system dysregulation: The exact cause of GCM remains unclear, but evidence suggests that it may be linked to an autoimmune response. In individuals with GCM, the immune system erroneously identifies the heart tissue as foreign and mounts an attack. This immune response leads to the infiltration of inflammatory cells, including giant cells, into the myocardium.

Inflammatory cascade: The production of several pro-inflammatory cytokines and immune cells is a part of the inflammatory cascade in GCM. These mediators contribute to the destruction of cardiac tissue and the formation of giant cells. Interleukin-1 (IL-1), Tumor Necrosis Factor-Alpha (TNF-α), and other cytokines play crucial roles in amplifying the inflammatory response, contributing to the pathogenesis of GCM.

Giant cell formation: Large, multinucleated cells known as giant cells are a defining feature of GCM. These cells result from the fusion of macrophages, which are immune cells responsible for engulfing and digesting foreign substances. In GCM, the fusion of macrophages creates giant cells that contribute to the ongoing inflammation and tissue damage within the heart.

Role of *HLA* genes: Genetic factors may also play a role in predisposing individuals to GCM. *Human Leukocyte Antigen* (*HLA*) genes, which are involved in regulating the immune system, have been implicated in the development of autoimmune diseases. Certain *HLA* gene variants may contribute to the susceptibility of GCM, although more research is needed to fully understand these genetic associations.

Diagnosis challenges

Clinical presentation: GCM often presents with non-specific symptoms such as fatigue, shortness of breath, chest pain, and heart failure. This can make it challenging to differentiate GCM from other heart conditions based solely on clinical symptoms.

Histopathological examination: Definitive diagnosis typically requires a biopsy of heart tissue, obtained through an endomyocardial biopsy. This invasive procedure is not without risks and may not always be feasible, especially in critically ill patients. Additionally, the distribution of giant cells within the myocardium can be patchy, further complicating the diagnostic process.

Management

Immunosuppressive therapy: Given the autoimmune nature of GCM, immunosuppressive therapy is a cornerstone of treatment. Corticosteroids, along with other immunosuppressive agents such as azathioprine and cyclosporine, are commonly used to suppress the aberrant immune response and reduce inflammation.

Heart transplantation: In severe cases where GCM has led to significant cardiac dysfunction and heart failure, heart transplantation may be considered as a life-saving option. However, the scarcity of donor hearts and the challenges associated with transplantation limit this approach.

Giant cell myocarditis remains a rare and enigmatic condition with potentially devastating consequences for cardiac function. Understanding the intricate mechanisms involved in the pathogenesis of GCM is essential for developing targeted therapies and improving outcomes for affected individuals. Advances in diagnostic techniques and treatment modalities are crucial for addressing the challenges associated with this rare cardiac disorder.

Correspondence to: Selena Jonas, Department of Immunology, Nilai University, Negeri Sembilan, Malaysia, E-mail: selenajonas@crc.gov.my

Received: 17-Nov-2023, Manuscript No. IDIT-23-28799; **Editor assigned:** 20-Nov-2023, PreQC No. IDIT-23-28799 (PQ); **Reviewed:** 05-Dec-2023, QC No. IDIT-23-28799; **Revised:** 12-Dec-2023, Manuscript No. IDIT-23-28799 (R); **Published:** 19-Dec-2023, DOI: 10.35248/2593-8509.23.8.164

Citation: Jonas S (2023) *HLA* Gene Variants and Genetic Predisposition in Giant Cell Myocarditis. Immunol Disord Immunother. 8: 164.