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Commentary

Etiopathogenesis of Myocarditis-Induced Dilated Cardiomyopathy

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DESCRIPTION

Patients with myocarditis exhibit 55-80% 5-year transplantation-free survival as determined by biopsies. Not only during the acute phase but also throughout the follow-up, mortality is seen. There are two post-acute clinical situations for the progressive illness myocarditis. In the first case, the inflammation clears up, and then there is a full recovery accompanied by better heart health. In between 40% and 60% of instances, myocarditis is said to cure on its own. In the second scenario, a stable Dilated Cardiomyopathy (DCM) develops after the initial phase. Follow-up clinical studies on patients with histologically established myocarditis revealed that 14-52% of them developed DCM pathology over the course of many years. In the absence of coronary artery disease, DCM is referred to as left ventricular dilatation coupled with systolic dysfunction. Histologically, DCM is characterized by the substantial fibrotic tissue and collagen deposition in place of the heart muscle cells. Patients with DCM experience heart failure and failure of other organs due to heart and heart valve issues, blood clots, and arrhythmias in addition to the weakening of the heart pump. Children and adults with DCM have a transplantation-free survival rate of 50-60% over 5 years, however new medications have the potential to raise this number as high as 80%. Patients with DCM who have Chagas disease have been observed to have a very high fatality rate. Myocardial inflammation and DCM may be related. Myocarditis and DCM that co-occur are known as Inflammatory DCM (iDCM).

In fact, myocardial inflammation is immune-histochemically evident in 16-30% of patients with chronic DCM. In the follow-up myocarditis cohort, cardiac fatalities were primarily linked to DCM traits like systolic dysfunction and left ventricular dilatation. Accordingly, patients with myocarditis have a worse prognosis when they have ventricular dilatation and systolic dysfunction.

Disease etiology

In North America and Europe, idiopathic myocarditis is common. Cardiotropic entero virus infections, like Coxsackie Virus B3 (CVB3), have been linked to the condition and are thought to be the causative agent. Patients with myocarditis who had cardiac biopsies were found to have entero viruses present at a rate of 14-57%. In biopsies from myocarditis patients, other viruses such parvovirus B19, adenoviruses, or herpes viruses have also been found. There has been a long-term shift in the identification of herpes viruses and parvovirus B19 from entero-virus and adenovirus. The causal role of viruses found in myocarditis patients is not immediately clear, though. For instance, a very high prevalence of parvovirus B19 has been found in patients with myocarditis, indicating the virus may be harmful in the condition.

However, myocarditis-negative hearts also had a significant frequency of parvovirus B19. Therefore, it is still unknown whether certain viral infections play a causal or concomitant role in the pathophysiology of myocarditis. Patients with Lyme disease are also diagnosed with myocarditis in North America and Europe (borreliosis). The bacteria *Borrelia burgdorferi*, which causes the illness, is spread *via* the bite of a tick that has the infection. Up to 10% of Lyme disease patients are thought to develop myocarditis.

Inflammatory heart disease is most frequently brought on in Latin America by infections with the protozoan *Trypanosoma cruzi* (Chagas disease). The cause of Chagas disease has a relatively well-established aetiology. The infectious forms of the parasite are transferred through the bites of blood-sucking triatomines, often known as kissing bugs. Trypanosome infection in humans causes the illness, which has two clinically different phases. The acute phase lasts for several weeks and is typically asymptomatic, however it can also be accompanied by fever, local edoema, or skin lesions. About one-third of those infected go on to develop a chronic form of the disease 10 to 30 years later, which is primarily characterised by DCM or iDCM but may also be accompanied by neurological and/or gastrointestinal tract diseases.

Chagas disease's chronic stage typically progresses, resulting in irreversible heart failure. The most prevalent and severe clinical manifestation of Chagas disease, which is linked to a poor prognosis and high mortality rates, is cardiac dysfunction brought on by myocarditis and iDCM. Systemic autoimmune illnesses and certain drugs are the main non-infectious causes of

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myocarditis. For instance, patients with myasthenia gravis and systemic lupus erythematosus have both experienced myocarditis. Numerous fatal myocarditis instances have recently been documented in cancer patients immediately after they began taking immune checkpoint inhibitors. Immune checkpoint inhibitors are a class of medications (antibodies) that target T cell response negative regulators like Cytotoxic T-Lymphocyte Associated Protein-4 (CTLA-4), Programmed Cell Death Protein-1 (PD-1), and PD-1 ligand (PD-L1).

Accordingly, 30% of patients with myocarditis and DCM develop high titers of heart-specific auto-antibodies. Cardiac Myosin Heavy Chain (MyHC) has been identified as the most prominent auto-antigen for circulating heart autoantibodies in myocarditis and cardiomyopathy patients. In fact, the presence of anti-MyHC autoantibodies has been associated with worse left ventricular systolic function and diastolic stiffness in patients with chronic myocarditis. For example, histological analysis demonstrated increased levels of Major Histocompatibility Complex (MHC) class I and II, known as Human Leukocyte Antigen (HLA) complexes and co-stimulatory molecules B7-1, B7-2, and CD40 in hearts of myocarditis patients. More recent data pointed also to the importance of the humoral response in myocarditis.