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Autoimmune Myocarditis Symptoms in Children and its Prevention

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Abstract

Autoimmune myocarditis is an autoimmune disease that affects the heart. The condition is characterized by inflammation of the heart muscle (myocardium). Autoimmune models of myocarditis were developed in late 1980s to 199s by deveral laboratories. Two main approaches were used to induce disease; either complete Freund's adjuvant supplemented with Mycobacterioum tuberculosis or a mildly infectious virus injected with cardiac myosin or other self-peptides. Further description of the variant of myocarditis can be made based on the equality of the inflammatory cell infiltrate (eosinophilic, polymorphous, lymphocytic or granulomatous).

Keywords: Autoimmune diseases; Prevention; Children; Myocarditis

Study Description

Characterization of the models revealed that both experimental autoimmune myocarditis. and the viral-self peptide models involve two phases; Acute myocarditis around day 7-14 after inoculation or infection and a second, much less severe phase of myocarditis associated with fibrosis and dilated cardiomyopathy staring around day 35-42. Originally, murine cytomegalovirus infection was thought to be the virus model of myocarditis that induced autoimmune myocarditis as a secondary feature of the pathogenesis of disease. Later it was realized that the viral infection acts like an adjuvant to activate the innate immune response and promote autoimmune disease in the presence of self-peptides, similar to the role of complete Freund's advance in autoimmune myocarditis. The viral autoimmune myocarditis models produce a disease remarkably similar to autoimmune myocarditis and to human disease. In serious cases, the signs and symptoms of myocarditis vary, depending on the cause of the disease. Common myocarditis signs and symptoms include Chest pain, Rapid or abnormal heart rhythms (arrhythmias), Shortness of breath, at rest or during physical activity, Fluid retention with swelling of your legs, ankles and feet, Fatigue,

The incidence of pediatric myocarditis is estimated to be 1.95 of 100,000 patients/year. In all three models, autoantibodies against cardiac myosin are found early after self-peptide injection and remain throughout the illness course. Microarray analysis of hearts with myocarditis compared to undismayed control revealed that the genes/proteins that lead to remodeling and fibrosis are unregulated during the first phase of disease during acute myocarditis and that it takes around 3 weeks before fibrosis appears in the heart resulting in dilation that can be detected using echocardiography. Nevertheless, the true incidence is difficult to undermine. Indeed, on the one hand, disease presentation in often with no symptoms. On the other hand, imaging and laboratory tests are not so specific and endocardial biopsy is not performed in most cases. Moreover, symptoms are frequently not specific, sometimes masquerading as respiratory and gastrointestinal infections.

When children develop myocarditis, they might have signs and symptoms including Fever, Fainting, Breathing difficulties Rapid breathing, Rapid or abnormal heart rhythms (arrhythmias). Myocarditis is a broad term and can be classified based upon several parameters, such as etiology, stage and histopathology. Based on etiology, several agents may lead to development of myocarditis, including infectious agents (with viruses showing the higher incidence and prevalence), physical (radiation), and pharmacologic, hematologic, and autoimmune disorders. Unfortunately, different etiologies might lead to similar histopathological characteristics. For example, viral, toxic, ration-associated and autoimmune myocarditis might lead to acute lymphocytic myocarditis with similar pathologic findings. Based on the disease stage, the inflammation may be acute, subacute and chronic, with tissue remodeling and fibrosis that are eventually similar regardless of the initial subtype of myocarditis. It is possible that, with disease progression, several etiologic subtypes merge into a common pathogenic process, characterized by tissue remodeling with chronic inflammation, fibrosis, myocyte damage and eventually leading to a dilated cardiomyopathy phenotype, Although the incidence of dilated cardiomyopathy as a sequela of previous myocarditis is not known, retrospective studies report that up to 50% of dilated cardiomyopathy cases have histological evidence of myocarditis, suggesting a persistent inflammatory process. Ongoing disease may be due to either persistence of virus in the myocardium or an autoimmune process.