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IS MYOCARDITIS LIKE A HOT KNIFE THROUGH BUTTER

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ABSTRACT

Warm countries are the worm countries. We are living in the "wormy world" But yet parasites are ignored by cardiologists, clinicians and scientists.

Heart disease is often missed in women. The myth of the 'widowmaker. Approximately seven times more women will die from heart disease than breast cancer. Even in women with breast cancer, dying from heart disease is a leading cause of death. Is there any silver bullet?

INTRODUCTION

Inflammation of cardiac myocytes, associated with necrosis followed by degeneration. It can occur at any age, but more common in children (1) 1 to 10 years. One of the few heart diseases that can cause Acute Heart Failure in a previously healthy heart (in children, adolescents and young adults). Myocarditis may be Infectious and Non infectious. Mostly idiopathic, but viral etiology is suspected strongly and the evidence is usually circumstantial. In Europe and North America, viruses are common pathogens and worldwide the most common cause is Chagas Disease, an illness endemic to Central and South America due to Trapnosoma cruzi. It is by bacterial, Viral, Fungal, Non-infectious like Hypersensitivity, immunologically related diseases like S.L.E, Scleroderma, Rheumatic fever, Drugs like Penicillin, Sulfa and radiation, Sarcoidosis, Ureamia.etc. Most common viruses to infect cardiac myocytes are Coxsackie, Adeno viruses, and they are termed as cardiotrophic viruses. The cardiotrophic viruses have direct viral cytotoxicity and generate cell mediated immune reactions against infected myocytes. Both Coxsackie and Adenovirus enter cardiac myocytes after binding the same cell surface receptor the COXSACKIE_ADENO VIRUS RECEPTOR (CAR), which is abundant in children, which may explain why Coxsackie and Adeno viral myocarditis is so common in children (1).Exact incidence of myocarditis is not known. However in series of routine autopsies, 1-9% of all patients had evidence of myocardial inflammation in young adults, upto 20% of all cases of sudden death are due to Myocarditis. Among patients with HIV, Myocarditis is the most common pathologic finding at autopsy, with a prevalence of 50% or more. Hearts of patients with myocarditis, who have developed clinical heart failure, show the following during active inflammatory phase. Global myocardial hypokinesia and bi-venticular dilation.

Viralcardiomyopathy occurs when viral infections cause myocarditis with a resulting thickening of the myocardium and dilation of the ventricles. These viruses include Coxsackie B and adenovirus, echoviruses, influenza H1N1, Epstein-Barr virus, rubella (German measles virus), varicel(chickenpoxvirus), mumps, measles, parvoviruses, yellow fever, dengue fever, polio, rabies and the viruses that cause hepatitis A and C (2,3,4)

Myopericarditis is a combination of both myocarditis and pericarditis appearing in a single individual, namely inflammation of both the pericardium and the heart muscle. It can involve the presence of fluid in the heart (5) Myopericarditis refers primarily to a pericarditis with lesser myocarditis, as opposed to a perimyocarditis, though the two terms are often used interchangeably. Both will be reflected on an ECG. Myo-pericarditis usually involves inflammation of the pericardium, or the sac covering the heart. The

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ACAM2000 smallpox vaccine has been known to cause myopericarditis in some people (6,7)

Eosinophilic myocarditis is inflammation in the heart muscle that is caused by the infiltration and destructive activity of a type of white blood cell, the eosinophil. Typically, the disorder is associated with hypereosinophilia, i.e. an eosinophil blood cell count greater than 1,500 per microliter (normal 100 to 400 per microliter). It is distinguished from the other form of myocarditis which is caused by other types of white blood cell, lymphocytes and monocytes, as well as two respective descendants of these cells, NK cells and macrophages. This distinction is important because the eosinophil-based disorder is due to a particular set of underlying diseases and preferred treatments that differ from those for non-eosinophilic myocarditis (8, 9)

Eosinophilic myocarditis is often viewed as a disorder that has three progressive stages. The first is an inflammatory stage of eosinophilic myocarditis, a thrombotic stage wherein the endocardium (i.e. interior wall) of the diseased heart forms blood clots which break off and travel to another part of the body through the arteries to dominate the clinical presentation. The second stage is a fibrotic stage wherein scarring replaces damaged heart muscle tissue to cause a clinical presentation dominated by a poorly contracting heart and cardiac valve disease (10,11,12) Perhaps less commonly, eosinophilic myocarditis, eosinophilic thrombotic myocarditis, and eosinophilic fibrotic myocarditis are viewed as three separate but sequentially linked disorders in a spectrum of disorders termed (8) The focus here is on eosinophilic myocarditis as a distinct disorder separate from its thrombotic and fibrotic sequelae.

Cardiomyopathy is a group of diseases that affect the heart muscle (16) Early on there may be few or no symptoms.[1] Some people may have shortness of breath, feel tired, or have swelling of the legs due to heart failure. (13) An irregular heart beat may occur as well as fainting Those affected are at an increased risk of sudden cardiac death (14) Types of cardiomyopathy include hypertrophic cardiomyopathy, dilated cardiomyopathy, restrictive cardiomyopathy, arrhythmogenic right ventricular dysplasia, and takotsubo cardiomyopathy (broken heart syndrome) (15). In hypertrophic cardiomyopathy the heart muscle enlarges and thickens (15) In dilated cardiomyopathy the ventricles enlarge and weaken. In restrictive cardiomyopathy the ventricle stiffens.

The cause is frequently unknown. Hypertrophic cardiomyopathy usually is inherited, while dilated cardiomyopathy is inherited in a third of cases.[4] Dilated cardiomyopathy may also result from alcohol, heavy metals, coronary heart disease, cocaine use, and viral infections (13) Restrictive cardiomyopathy may be caused by amyloidosis, hemochromatosis, and some cancer treatments (15) Broken heart syndrome is caused by extreme emotional or physical stress (13)

Many diseases can involve the heart and blood vessels (16) A common underlying factor in infective endocarditis is a structural heart defect, especially faulty heart valves. The heart has a rough lining or abnormal valves, the invading parasites can attach and multiply in the heart. Parasitic infections previously seen only in developing tropical settings can be currently diagnosed worldwide due to travel and population migration. Some parasites may directly or indirectly affect various anatomical structures of the heart, with infections manifested as myocarditis, pericarditis, or pulmonary hypertension (16A) Trypanosoma cruzi, Trypanosoma Gambians, Dinofilariae are the most relevant parasitic infections involving the heart. A variety of protozoan, helminthic parasites may cause a direct effect on various structures of heart like Myocardium, endocardium, cardiac vasculature, and resulting in congestive heart failure, cardiomyopathy, tachycardia, cardiac murmurs, muffled heart sounds. Parasites damage nerve supply to heart. Most of the parasites shows impact on aortic valve or mitral valve and produce rapid beats, irregular beats, angina, chest pain, arm pain, upper back pain, shortness of breath and neck pain.(16B) Infective endocarditis is the most serious of all infections, is characterized by colonization or invasion of heart valves or mural endocardium by a microbe leading to the formation of bulky, friable vegetation's, associated with destruction of underlying cardiac tissue (17) Empoisnnement to this organ by various parasites their toxic metabolites results in progressive myocardial dysfunction in cardiac disease with hypertrophy.(18)

Chagas' cardiomyopathy is the hallmark of a disease that is currently considered a global parasitic disease (20) *Toxoplasma gondii, a protozoan parasite also causes myocarditis. Experimental* myocarditis occurs when toxoplasma cysts rupture within the heart; therefore clinical symptoms may occur sporadically during a chronic infection.(21) Indeed, toxoplasmosis is the most commonly reported parasitic disease occurring after heart transplantation and may simulate organ rejection (22) Disseminated toxoplasmosis with associated myocarditis can lead to a fatal outcome if no prior prophylaxis is given to transplant patients (23, 24). Trichinosis myocarditis may initially manifest with chest pain and mimic an acute myocardial infarction. Reports suggest that ECG evidence of myocardial involvement may be found for up to 75% of patients. Complications such cardiac arrhythmias are considered the most common cause of death associated with trichinellosis (25,26).

Helminthic Parasites- like (ascaris, *Echinococcus granulosus*, *Paragonimus westermani*, schistosoma, *Taeniaa solium*, visceral larva migrans,In particular, Dirofilaria (Dog worm) Trichinella spiralis, lives in heart muscle and other muscle of the body. and *Wuchereria Bancroft*) are causing myocarditis.The parasites will continue to emerge leading to unpredictable epidemics and challenges for the clinicians and scientists. Hence there is an urgent need of surveillance and control. Advance diagnostics, tests, vaccines, therapeutics and development of new drugs are needed.

Both Corynebacterium diptheriae and Staphylococcus aureus have been associated with myocarditis. Both of these bacteria are widely distributed and found in nature and are generally innocuous. Corynebacterium diptheriae is the bacteria that cause diphtheria, an acute, contagious infection that causes the cells of the tonsils and throat to die, resulting in pseudomembranes to form in the back of the throat. **C.** diptheriae occurs primarily in un-immunized school-aged children and in the elderly and immunocompromised. There are vaccines against diptheriae that are readily available. Myocarditis caused by Staphylococcus aureus is generally seen in the face of overwhelming bacteria growing in the blood (sepsis). This used to be a fairly common complication of

sepsis before antibiotics were widely available. Myocarditis caused by **S. aureus** can result in abscesses (pockets of bacteria/pus) to form on the heart itself. (28)

History

The term "Myocarditis", implying inflammatory process of the myocardium was coined and introduced by German Physician-Joseph Friedrich Sobemheim in 1837.Cases of myocarditis have been documented as early as the 1600s,[1] but the term "myocarditis", implying an inflammatory process of the myocardium, was introduced by German physician Joseph Friedrich Sobernheim in 1837(29) However, the term has been confused with other cardiovascular conditions, such as hypertension and ischemic heart disease (30) Following admonition regarding the indiscriminate use of myocarditis as a diagnosis from authorities such as British cardiologist Sir Thomas Lewis and American cardiologist and a co-founder of the American Heart Association Paul White, myocarditis was under-diagnosed (31)

Although myocarditis is clinically and pathologically clearly defined as "inflammation of the myocardium", its definition, classification, diagnosis, and treatment are subject to continued controversy, but endomyocardial biopsy has helped define the natural history of myocarditis and clarify clinicopathological correlations (32)

Mechanism

Once coxsackie and Adeno viruses affect myocytes, they produce Protease-2A, that causes viral replication, myocardial dysfunction and also cleaves myocyte protein such as dystrophin, which may be involved in virus exiting from myocytes(intracellular viral load is higher if dystrophin is absent).Cardiac myocytes have powerful innate antiviral defenses mediated by Janus Kinase(JAK) and STAT pathways activated by interferons, Alpha, Beta. Gamma and Interleukin-6(IL-6). However, these actions can be inhibited by suppressors of cytokine signaling (SOCS), proteins that limit potentially deleterious actions of cytokine signaling in cardiac myocytes. Levels of SOCS profoundly affect susceptibility to coxsackieevirus infection. Thus, the highly variable clinical and pathological manifestations of viral myocarditis depend on the dynamic interplay between mechanisms that determine viral entry, replication and release, and immune mechanisms, of host responsiveness.

Most forms of myocarditis involve the infiltration of heart tissues by one or two types of pro-inflammatory blood cells, lymphocytes and macrophages plus two respective descendants of these cells, NK cells and macrophages. Eosinophilic myocarditis is a subtype of myocarditis in which cardiac tissue is infiltrated by another type of pro-inflammatory blood cell, the eosinophil. Eosinophilic myocarditis is further distinguished from non-eosinophilic myocarditis by having a different set of causes and recommended treatments (33,34)Myocarditis is an inflammatory disease of the heart frequently resulting from viral infections and/or post-viral immune-mediated responses. It is one of the important causes of dilated cardiomyopathy worldwide. The diagnosis is presumed on clinical presentation and noninvasive diagnostic methods such as cardiovascular magnetic resonance imaging. Endomyocardial biopsy remains the gold standard for in vivo diagnosis of myocarditis. The therapeutic and prognostic benefits of endomyocardial biopsy results have recently been demonstrated in several clinical trials. Although remarkable advances in diagnosis, understanding of pathophysiological mechanisms, and treatment of acute myocarditis were gained during the last years, no standard treatment strategies could be defined as yet, apart from standard heart failure therapy and physical rest. In severe cases, mechanical support or heart transplantation may become necessary. There is some evidence that immunosuppressive and immunomodulating therapy are effective for chronic, virus-negative inflammatory cardiomyopathy. Further investigations by controlled, randomized studies are needed to definitively determine their role in the treatment of myocarditis.

Significance Gap in Research

Exact incidence of myocarditis is not known. However in series of routine autopsies, 1-9% of all patients had evidence of myocardial inflammation in young adults, upto 20% of all cases of sudden death are due to Myocarditis .Among patients with HIV, Myocarditis is the most common pathologic finding at autopsy, with a prevalence of 50% or more. Hearts of patients with myocarditis, who have developed clinical heart failure, show the following during active inflammatory phase. Global myocardial hypokinesia and bi-venticular dilation. Histologically with mononuclear inflammatory infiltrate mainly of T lymphocytes and macrophages. Pathologically defined as Myocarditis, its definitions, classifications, diagnosis and treatment are subject to controversy, But endomyocardial biopsy (EMB) has helped to define the natural history of myocarditis and clarify clinicopathological correlations. Endomyocardial Biopsy is useful Confirmation of myocarditis still requires histologic evidence. An AHA, ACCF, ESC joint statement in 2007made a class 1 recommendation for biopsy under the following situations (35) Heart disease is often missed in women: The myth of the 'widowmaker. Approximately seven times more women will die from heart disease than breast cancer. Even in women with breast cancer, dying from heart disease is a leading cause of death.Despite considerable advances in our understanding of myocarditis pathogenesis, the clinical management of myocarditis has changed relatively little in the last few years. This review aims to help bridge the widening gap between recent mechanistic insights, which are largely derived from animal models, and their potential impact on disease burden. (36)

Different strategies including pharmacological and gene therapeutic approaches directed at blocking viral replication or stimulating the antiviral-directed immune response, are under investigation in experimental and clinical studies.(37)Because of the low rate of diagnosis, multi-centre collaborations with standardized evaluations and treatment protocols, mechanistically oriented registries, and core molecular diagnostic facilities will be needed.

Ideas Where Research go Next

Cardiovascular magnetic resonance imaging (CMR), sometimes known as cardiac MRI, is a medical imaging technology for the non-invasive assessment of the function and structure of the cardiovascular system. It is derived from and based on the same basic principles as magnetic resonance imaging (MRI) but with optimization for use in the cardiovascular system. These optimizations are principally in the use of ECG gating and rapid imaging techniques or sequences. By combining a variety of such techniques into

protocols, key functional and morphological features of the cardiovascular system can be assessed.(27) A large number of causes of myocarditis have been identified, The most common cause is Chagas' disease, an illness endemic to Central and South America that is due to infection by the protozoan *Trypanosoma cruzi*.(19) chronic infection, of Chagas disease particularly the heart, can be severely affected and manifest as heart failure. Chagas disease usually causes impairment in the electrical activity of the heart and heart failure

In patients with heart failure, a normal –sized heart or dilated LV> 2 weeks after the onset of symptoms and hemodynamic compromise (or) In patients with a dilated LV, 2 weeks to 3 months after the onset of symptoms, new ventricular arrhythmia or AV nodal block, or Who do not response to usual care after 1-2 weeks. There are no specific laboratory studies. Troponin I levels are increased in about 1/3 of patients, but CPK-MB elevated only in 10%.Echocardiography provides the most convenient way of evaluating cardiac function and can exclude many other processes.MRI with gadolinium enhancement reveals spotty areas of injury throught the myocardium, but correlation with endomyocardial biopsy results have been poor. Usually have no symptoms and if symptoms occur usually a few weeks after viral infection.Most patients recover, although a few die of congestive heart failure. Or arrhythmias. Disease is unusually severe in infants and pregnant women. No specific treatment, only supportive measures are the rule. Despite resolution of the active phase of this disease, functional impairment may persist for years and progress to cardiomyopathy.

Despite considerable advances in our understanding of myocarditis pathogenesis, the clinical management of myocarditis has changed relatively little in the last few years. This review aims to help bridge the widening gap between recent mechanistic insights, which are largely derived from animal models, and their potential impact on disease burden. (36)Different strategies including pharmacological and gene therapeutic approaches directed at blocking viral replication or stimulating the antiviral-directed immune response, are under investigation in experimental and clinical studies.(37)

Current Debate

Currently available data show that immunosuppressive therapy in patients with biopsy-proven, virus-negative inflammatory cardiomyopathy is an effective and safe option in addition to supportive treatment for recovery of cardiac failure. However, larger studies powered to detect a difference in clinical endpoints such as heart failure hospitalization, transplantation, and deaths are still needed.

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