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Myocarditis: A Clinical and Diagnostic Conundrum

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Abstract

Inflammation of the cardiac muscle is myocarditis. Frequently, the exact etiology leading to myocarditis may not be found although viruses are a common cause. Myocarditis is often underdiagnosed as individuals may be asymptomatic. Patients with myocarditis may complain of chest pain, which may sometimes mimic acute myocardial infarction or acute coronary syndrome-like picture. Dilated cardiomyopathy from myocarditis may lead to heart failure. Serious complications of myocarditis include cardiac arrest, sudden death. Myocarditis has variable clinical presentation which may even lead to misdiagnosis. The current understanding of myocarditis is limited due to its insidious nature. Diagnosis of myocarditis is often challenging due to varied clinical features and lack of specific diagnostic testing for definitive diagnosis with a broad differential. For most cases of myocarditis, supportive care is provided.

Keywords: Myocarditis, Heart failure, Dilated cardiomyopathy, Inflammatory cardiomyopathy.

INTRODUCTION

Myocardial inflammation is termed as myocarditis. There may be necrosis or degeneration along with inflammatory infiltration in the myocardium [1,2]. Myocarditis is also known as inflammatory cardiomyopathy. Myocarditis is often underdiagnosed because individuals affected may be asymptomatic. It commonly affects younger individuals with a median age in the 40s [1,3]. Myocarditis is clinically classified as fulminant myocarditis, acute myocarditis, chronic active myocarditis, chronic persistent myocarditis. Fulminant myocarditis may lead to complete resolution or rapid deterioration and death. Acute myocarditis can result in cardiac dysfunction from incomplete recovery [2,3]. Chronic active myocarditis may progress to mild or moderate cardiac dysfunction. Chronic persistent myocarditis has usually no cardiovascular compromise [1-4].

Etiology

Table 1: Etiology of myocarditis*:* [Ref: 1,3-7,15-18,21,24-26]
Infectious: 1. Viral agents such as coxsackievirus A and B, Epstein-Barr virus, cytomegalovirus, human immunodeficiency virus, adenovirus, mumps, rubella, rubeola, poliovirus, echovirus, influenza virus, hepatitis B or C virus, parvovirus B19, herpesviruses. 2. Bacterial agents such as diphtheria, Staphylococcus aureus, Clostridium perfringens, mycoplasma, Legionella, streptococci, salmonella, Shigella. 3. Fungal agents such as candida, Mucor Aspergillus, Blastocystis hominis, Cryptococcus, Histoplasma, coccidioidomycosis. 4. Parasitic agents such as Echinococcus, Amoeba, Toxoplasma, Trichinella, Trypanosoma cruzi, schistosomiasis. 5. Rickettsial agents such as Rickettsia rickettssi. 6. Spirochetal agents such as Treponema pallidum, Borrelia burgdorferi.
Radiation
Lymphoma
Metastatic Kaposi's sarcoma
Sarcoidosis
Collagen vascular disease: Kawasaki syndrome, scleroderma, systemic lupus erythematosus, polymyositis, dermatomyositis, Wegener's granulomatosis, Churg-Strauss syndrome
Toxins such as lead, arsenic, carbon monoxide, cobalt, hydrocarbons
Postpartum
Rheumatic fever
Drug-induced: lithium, tricyclic antidepressants, cyclophosphamide, interferon-alpha, interleukin two, 5-fluorouracil, phenothiazines, isoniazid, amphotericin B, tetracycline, doxorubicin, sulfonamides, cocaine, amphetamines anthracyclines, streptomycin, trastuzumab, catecholamines, chemotherapy, anthracyclines
Alcohol

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Half of the cases may not have any clear underlying etiology [4-6]. Hypersensitivity reaction with eosinophilic myocarditis; venoms of scorpions, snakes, wasps; heatstroke; hypothermia; post-transplant cell rejection are other noninfectious causes of myocarditis [6,7].

Pathophysiology

The exact pathogenesis of myocarditis is not very well understood. Three phases have been described in the pathogenesis of myocarditis [6,8].

Phase I or acute phase of myocarditis involves cytotoxicity from inciting factor or causative agent leading to focal necrosis. Phase II or subacute phase involves an immune response by the host which leads to myocyte injury. Phase III or chronic phase is characterized by widespread myocardial fibrosis with cardiac dysfunction leading to dilated cardiomyopathy and contractile dysfunction [6-10].

Clinical features

Myocarditis may be completely asymptomatic or may present with mild to severe symptoms [6,7,10]. Precordial discomfort, myalgias, fatigue, palpitations, dyspnea are common symptoms [9]. A physical exam may reveal persistent tachycardia out of proportion to fever, faint S1, and S4, a murmur of mitral regurgitation [10,11]. Patients may have signs of biventricular failure such as hypotension, hepatomegaly, peripheral edema, distended neck veins, presence of S3. Acute decompensated heart failure is a common presentation [11-13]. A pericardial friction rub may be audible in case of myopericarditis. Patients may develop cardiac arrhythmias [8,12,13]. Arrhythmias may present in the form of syncope, patients may have palpitations from heart block such as Stokes- Adams attack, ventricular tachyarrhythmias. Severe myocarditis may rapidly progress to acute heart failure and cardiogenic shock [11-15].

Diagnostic studies

Table 2: Diagnostic evaluation in myocarditis*:* [Ref:2,5,7,9-4,17,18]	
1.	A chest x-ray may show enlargement of the cardiac silhouette
2.	Electrocardiogram (ECG) findings include sinus tachycardia, interventricular conduction defect, and bundle branch blocks. Lyme disease and diphtheria can lead to heart block. Associated focal necrosis may lead to changes of acute myocardial infarction.
3.	An echocardiogram may show dilated, hypokinetic chambers and segmental wall motion abnormalities.
4.	Lab evaluation findings: elevated creatine kinase with elevated MB fraction, lactate dehydrogenase, aspartate transaminase levels due to myocardial necrosis. Elevated troponin T or I, erythrocyte sedimentation rate, white blood cell count may be found.
5.	Cardiac catheterization is often done to rule out coronary artery and valvular disease.

Leukocytosis with the lymphocyte predominance is common, the finding of eosinophilia suggests eosinophilic myocarditis [16,17]. Acute phase reactants are helpful to monitor clinical progression or response to therapy. An ECG may sometimes show changes suggestive of acute myocardial infarction or even ST-segment elevation usually in more than one vessel distribution. Other ECG findings include long QTc, atrioventricular block, low voltage [14,17-19]. Echocardiogram in the case of fulminant myocarditis has findings of increased septal wall thickness with near-normal diastolic dimensions. Acute myocarditis echocardiographic features include increased diastolic dimensions with normal septal wall thickness [18-20]. Coronary angiography is often required to rule out coronary artery disease as the clinical picture of myocarditis may sometimes mimic myocardial infarction. Cardiac magnetic resonance imaging (MRI) may have a role to diagnose myocarditis by visual assessment of myocardial inflammatory markers. Nuclear imaging may show diffuse uptake suggestive of myocyte necrosis [9,18,20,21].

Right ventricular endomyocardial biopsy may be done to confirm the diagnosis, however, a negative biopsy cannot definitively rule out myocarditis, and the myocardial biopsy maybe even sometimes unnecessary [14,18,21-23].

Treatment

The treatment of myocarditis is directed towards treating the underlying cause such as the use of antibiotics for bacterial infection [20,22,24]. Restriction of physical activity is often recommended to decrease cardiac work. If congestive heart failure is present, treatment is aimed towards maximal medical management of heart failure with diuretics, angiotensin-converting enzyme inhibitors, beta-blockers, aldosterone antagonists, restriction of fluid and salt [22-25]. Cases of myocarditis with apical aneurysm with thrombus such as that in the setting of Chagas disease, atrial fibrillation, or a history of embolic episode requires anticoagulation to prevent thromboembolism [11,16,19,21,24]. Ventricular arrhythmias secondary to myocarditis may require antiarrhythmic treatment such as that with beta-blockers, amiodarone, sotalol, quinidine or procainamide. Preload and afterload reducing agents are often used to treat cardiac decompensation. Inotropic agents may be required in cases of severe hemodynamic compromise [20-22, 25,26]. Aggressive mechanical and surgical interventions such as intra-aortic balloon counter pulsation for hemodynamic support, left ventricular assist device, extracorporeal membrane oxygenation may be required in cases of severe hemodynamic instability. Patients with progressive biopsy-proven myocarditis should be considered for early cardiac transplantation [6,11,19,23,25].

Steroid use is generally contraindicated in early infectious myocarditis. No clear benefits of antiviral regimen or nonsteroidal anti-inflammatory agents have been established [20-22,25]. Immunosuppressants such as prednisone, cyclosporine, azathioprine do not alter the prognosis of myocarditis except in the cases due to collagen vascular disease [17,20,22,24]. About half of the patients with myocarditis may die in five years of diagnosis. A better prognosis has been noted in cases of fulminant myocarditis. Many patients with myocarditis may have complete spontaneous clinical recovery. Half patients may develop subsequent dilated cardiomyopathy [19,21,24-26]. Close clinical follow-up with the serial echocardiographic assessment of left ventricular function and structure is often recommended [22-24,26]. It is important to identify myopericarditis, myocarditis and pericarditis as management can be directed towards a specific entity with improved outcomes [27].

CONCLUSION

Dilated cardiomyopathy from inflammation and necrosis of myocardial tissue occurs in myocarditis. The etiology of myocarditis is often clinically unknown though there are several known causative factors. Various infectious agents and noninfectious factors have been identified as etiology of myocarditis; viral agents are the most common causative factors. Myocarditis often goes underdiagnosed and most cases may not have a clear etiology. Younger patients with usually fewer typical risk factors for the coronary disease may present with chest pain mimicking acute myocardial infarction or new-onset congestive heart failure which may often lead to misdiagnosis. It is important to be vigilant of myocarditis in the differential diagnosis especially in the setting of new-onset heart failure in young patient populations with no obvious contributing factors and need to reinstate the varied clinical presentations of myocarditis. Diagnosis of myocarditis is difficult due to varied presentations, lack of specific diagnostic modalities, and often presentations mimicking some other diagnosis. Further research is required to make an early diagnosis and better treatment modalities to prevent the progression to chronic heart failure and thereby decrease morbidity and mortality from complications of myocarditis.

Conflict of interest

The authors declare no conflict of interest.

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