أ.م.د.اسيل جاسم محمد
ماجستير طب الاطفال

Myocarditis

Acute or chronic inflammation of the myocardium is characterized by infiammatory cell infiltrats,myocyte necrosis,or degeneration&may be caused by infectious,connective tissue,granulomatous,toxic,or idiopathic processes.

There may be associated systemic manifestations of the disease and on occasion the endocardium or pericardium is involved, though coronary pathology is uniformly absent. Patients may be asymptomatic, have nonspecific prodromal symptoms, or present with overt congestive heart failure, compromising arrhythmias, or sudden death.

ETIOLOGY AND EPIDEMIOLOGY

⁕Viral Infections

Coxsackievirus and other enteroviruses, adenovirus, parvovirus, Epstein-Barr virus, parechovirus, influenza virus, and cytomegalovirus are the most common causative agents in children, though most known viral agents have been reported. In Asia, hepatitis C virus appears to be significant as well. The true incidence of viral myocarditis is unknown as mild cases probably go undetected. The disease is typically sporadic but may be epidemic. Manifestations are, to some degree, age dependent: in neonates and young infants, viral myocarditis can be fulminant; in children, it often will occur as an acute, myopericarditis with heart failure; and in older children and adolescents, it may present with signs a

nd symptoms of acute or chronic heart failure or chest pain.

⁕Bacterial Infections

Bacterial myocarditis has become far less common with the advent of advanced public health measures, which have minimized infectious causes such as diphtheria. Diphtheritic myocarditis is unique as bacterial toxin may produce circulatory collapse and toxic myocarditis characterized by atrioventricular block, bundle-branch block, or ventricular ectopy. Any overwhelming systemic bacterial infection can manifest with circulatory collapse and shock with evidence of myocardial dysfunction characterized by tachycardia, galloprhythm, and low cardiac

output. Additional nonviral infectious causes of myocarditis include rickettsia, protozoa, parasitic infections, and fungal disease.other causes,**immune_mediated &toxic myocardiatis**.

PATHOPHYSIOLOGY

Myocarditis is characterized by myocardial inflammation, injury or necrosis, and ultimately fibrosis. Cardiac enlargement and diminished systolic function occur as a direct result of the myocardial damage. Typical signs of congestive heart failure occur and may progress rapidly to shock, atrial or ventricular arrhythmias, and sudden death. Viral myocarditis may also become a chronic process with persistence of viral nucleic acid in the myocardium, and the perpetuation of chronic inflammation.. Some viral proteins share antigenic epitopes with host cells, resulting in autoimmune damage to the antigenically related myocyte. Cytokines such as tumor necrosis factor-α and interleukin-1 are inhibitors of myocyte response to adrenergic stimuli and result in diminished cardiac function. The final result of viral-associated inflammation can be Dilated cardiomyopathy DCM.

CLINICAL MANIFESTATIONS

Manifestations of myocarditis range from asymptomatic or nonspecific generalized illness to acute cardiogenic shock and sudden death. Infants and young children more often have a fulminant presentation with fever, respiratory distress, tachycardia, hypotension, gallop rhythm, and cardiac murmur. Associated findings may include a rash or evidence of end organ involvement such as hepatitis or aseptic meningitis. Patients with acute or chronic myocarditis may present with chest discomfort, fever, palpitations, easy fatigability, or syncope/near syncope. Cardiac findings include overactive precordial impulse, gallop rhythm, and an apical systolic murmur of mitral insufficiency In patients with associated pericardial disease, a rub may be noted. Hepatic enlargement, peripheral edema, and pulmonary findings such as wheezes or rales may be present in patients with decompensated heart failure.

DIAGNOSIS

Electrocardiographic changes are nonspecific and may include sinus tachycardia, atrial or ventricular arrhythmias, heart block, diminished QRS voltages, and nonspecific ST and T-wave changes, often suggestive of acute ischemia.

Chest x-rays in severe, symptomatic cases reveal cardiomegaly, pulmonary vascular prominence, overt pulmonary edema, or pleural effusions. Echocardiography often shows diminished ventricular systolic function, cardiac chamber enlargement, mitral insufficiency, and occasionally, evidence of pericardial infusion. Cardiac MRI is a standard imaging modality for the diagnosis of myocarditis; information on the presence and extent of edema, gadolinium-enhanced hyperemic capillary leak, myocyte necrosis, left ventricular dysfunction, and evidence of an associated pericardial effusion assist in the cardiac MRI diagnosis of myocarditis.

Endomyocardial biopsy may be useful in identifying inflammatory cell infiltrates or myocyte damage and performing molecular viral analysis using polymerase chain reaction techniques. Catheterization and biopsy, although not without risk (perforation and arrhythmias), should be performed by experienced personnel in patients suspected to have myocarditis or if there is strong suspicion for unusual forms of cardiomyopathy such as storage diseases or mitochondrial defects. Nonspecific tests include sedimentation rate, creatine phosphokinase isoenzymes, cardiac troponin I, and brain natriuretic peptide levels.

DIFFERENTIAL DIAGNOSIS

The predominant diseases mimicking acute myocarditis include carnitine deficiency, other metabolic disorders of energy generation, hereditary mitochondrial defects, idiopathic DCM, pericarditis, , and anomalies of the coronary arteries.

TREATMENT

Primary therapy for acute myocarditis is supportive ,Acutely, the use of inotropic agents, preferably milrinone, should be entertained but used with caution because of their proarrhythmic potential.

Diuretics are often required as well. If in extremis, mechanical ventilatory support and mechanical circulatory support with ventricular

assist device implantation or extracorporeal membrane oxygenation may be needed to stabilize the patient's hemodynamic status and serve as a bridge to recovery or cardiac transplantation. Diuretics, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers,are of use in patients with compensated congestive HF outpatient setting but may be contraindicated in those presenting with fulminant heart failure and cardiovascular collapse.

In patients manifesting with significant atrial or ventricular arrhythmias, specific antiarrhythmic agents (for example, amiodarone) should be administered and implantable cardioverter defibrillator placement considered.

Immunomodulation of patients with myocarditis is controversial. Intravenous immune globulin may have a role in the treatment of acute or fulminant myocarditis and corticosteroids have been reported to improve cardiac function, but the data are not convincing in children.. There are no studies to recommend specific antiviral therapies for myocarditis.

PROGNOSIS

The prognosis of symptomatic acute myocarditis in newborns is poor, and a 75% mortality has been reported. The prognosis is better for children and adolescents, although patients who have persistent evidence of DCM often progress to need for cardiac transplantation. Recovery of ventricular function has been reported in 10-50% of patients, however.