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MYOCARDITIS IS THE MOST COMMON TYPE OF ETHYLOGIC AND MEASURES TO TREAT IT

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Annotation: Myocarditis is inflammation of the myocardium with necrosis of cardiac myocytes. Myocarditis may be caused by many disorders (eg, infection, cardiotoxins, drugs, and systemic disorders such as sarcoidosis) but is often idiopathic. Symptoms can vary and can include fatigue, dyspnea, edema, palpitations, and sudden death. Diagnosis is based on symptoms and clinical findings of abnormal electrocardiography (ECG), cardiac biomarkers, and cardiac imaging in the absence of cardiovascular risk factors. Endomyocardial biopsy confirms clinical diagnosis of myocarditis. Treatment depends on the cause, but general measures include drugs to treat heart failure and arrhythmias and rarely surgery (eg, intra-aortic balloon pump, left ventricular assist device, transplantation). Immunosuppression is of use in certain types of myocarditis (eg, hypersensitivity myocarditis, giant cell myocarditis, myocarditis caused by sarcoidosis).

Key words: myocarditis, blood, heart, necrosis, myocyte.

Myocarditis is inflammation of myocardium with necrosis of cardiac myocyte cells. Biopsy-proven myocarditis typically demonstrates inflammatory infiltrate of the myocardium with lymphocytes, neutrophils, eosinophils, giant cells, granulomas, or a mixture.

The pathophysiology of myocarditis remains a subject of research. Potential mechanisms that lead to myocardial injury include

- Direct cardiomyocyte injury caused by an infectious or other cardiotoxic agent
- Myocardial injury caused by an autoimmune reaction to an infectious or other cardiotoxic agent

Myocardial inflammation can be diffuse or focal. Inflammation can extend into the pericardium causing myopericarditis. The extent of myocardial involvement and extension into adjacent pericardium can determine the type of symptoms. Diffuse involvement can lead to heart failure, arrhythmias and sometimes sudden cardiac death. Focal involvement is less likely to cause heart failure but can lead to arrhythmias and sudden cardiac death. Involvement of the pericardium leads to chest pain and other symptoms typical of pericarditis. Some patients remain asymptomatic whether myocardial involvement is focal or diffuse.

Infectious myocarditis is most often viral in the US and other high income nations. The most common viral causes in the US are parvovirus B19 and human herpes virus 6. In lower income nations, infectious myocarditis is most often associated with rheumatic carditis, Chagas disease, or AIDS. Direct myocardial injury due to SARS-CoV-2 infection, with symptoms ranging from mild chest discomfort to fulminant myocarditis, is uncommon in patients with COVID-19, but the risk of myocarditis is 16 times higher in those with infection than in those not infected.

Noninfectious causes include cardiotoxins, certain drugs, and some systemic disorders. Myocarditis caused by drugs is termed hypersensitivity myocarditis. Myocarditis after mRNA-based COVID-19 vaccination is rare and far less common than COVID-associated myocarditis It occurs mostly in adolescent and young adult males, usually within a week of vaccination, and is generally mild.

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Giant cell myocarditis

Giant cell myocarditis is a rare form of myocarditis with a fulminant course. The etiology is unclear but may include an autoimmune mechanism. Biopsy shows characteristic multinucleated giant cells. Patients with giant cell myocarditis present in cardiogenic shock and frequently have intractable ventricular arrhythmias or complete heart block. Giant cell myocarditis has a poor prognosis but is important to rule out in the setting of an otherwise healthy patient presenting in fulminant heart failure or with intractable arrhythmias because immunosuppressive therapy can help improve survival.

The clinical presentation of myocarditis is variable. Patients may be minimally symptomatic or have fulminant heart failure and fatal arrhythmias. Symptoms depend on the etiology of the myocarditis as well as the extent and severity of myocardial inflammation.
Heart failure symptoms may include fatigue, dyspnea, and edema. Patients may show signs of fluid overload with crackles, elevated jugular venous pulses, and edema. Cardiac examination may be significant for a third (S3) or fourth (S4) heart sound. Systolic murmurs of mitral regurgitation and tricuspid regurgitation may be present in patients with ventricular enlargement.
Sudden cardiac death due to a fatal arrhythmia is sometimes the presenting feature. Patients may experience preceding palpitations or syncope.
When patients have concomitant pericardial inflammation, they may present with chest pain typical of pericarditis. Dull or sharp precordial or substernal pain may radiate to the neck, trapezius ridge (especially the left), or shoulders. Pain ranges from mild to severe. Unlike ischemic chest pain, pain due to pericarditis is usually aggravated by thoracic motion, cough, breathing, or swallowing food; it may be relieved by sitting up and leaning forward. A pericardial friction rub may be auscultated in patients with a pericardial effusion.

Certain clinical findings may be indicative of a specific cause of myocarditis. Infectious myocarditis may be preceded by symptoms of fever, myalgias, and other symptoms depending on the exact pathogen. Drug-related or hypersensitivity myocarditis may be accompanied by a rash. Enlarged lymph nodes may be indicative of sarcoidosis as the underlying etiology. Fulminant heart failure and arrhythmias may be indicative of giant cell myocarditis.

Myocarditis can be acute, subacute, or chronic. There are no set time-frames for each phase. The acute phase typically lasts a few days while the subacute phase lasts weeks to months. If myocarditis does not resolve after a few months, it is called chronic myocarditis. In some cases, myocarditis can lead to dilated cardiomyopathy.

- Electrocardiography (ECG) and cardiac enzymes
- Cardiac imaging
- Sometimes, endomyocardial biopsy
- Tests to identify cause

Myocarditis should be suspected when otherwise healthy patients with no cardiac risk factors present with symptoms of heart failure or arrhythmias. ECG, cardiac enzymes, and cardiac imaging are not specific for myocarditis but can be diagnostic in the appropriate clinical setting.

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ECG can be normal or abnormal in patients with myocarditis. ST segment abnormalities are common and can mimic myocardial ischemia. ST segment elevation is sometimes seen but more common findings include nonspecific ST-T wave changes. Patients may experience conduction delays and atrial or ventricular arrhythmias, including sinus tachycardia, ventricular tachycardia, and ventricular fibrillation.

Cardiac enzymes can be abnormal in patients with acute myocarditis. Cardiac troponin and CK-MB (creatine kinase muscle band isoenzyme) can both be elevated due to necrosis of cardiac myocytes.

Cardiac imaging can be abnormal in patients with myocarditis. Echocardiogram can be normal in early or mild myocarditis, but segmental wall motion abnormalities (mimicking myocardial ischemia) can be seen. Left ventricular dilation and systolic dysfunction can also be seen as in dilated cardiomyopathy. Diastolic relaxation parameters are often abnormal on echocardiography.

Cardiac MRI is becoming increasingly important in the diagnosis of myocarditis. Cardiac MRI of patients with myocarditis may show a characteristic pattern of late gadolinium enhancement in the subepicardial and mid-myocardial walls (in contrast to ischemia where late gadolinium enhancement is usually subendocardial with extension to mid-myocardial and epicardial walls). Other diagnostic features of myocarditis on cardiac MRI are the presence of myocardial edema and myocardial hyperemia relative to skeletal muscle.

Endomyocardial biopsy showing inflammatory infiltrate of the myocardium with necrosis of adjacent myocytes is the gold standard for diagnosis of myocarditis. However, this test has low sensitivity for diagnosis for myocarditis due to sampling error. Therefore, a positive biopsy result is diagnostic for myocarditis, but a negative result does not rule it out. In addition, the biopsy carries a risk of complications, including myocardial rupture and death, so is not routinely done. Endomyocardial biopsy should be done in cases of fulminant heart failure, ventricular arrhythmias, or heart block or if results would affect management (eg, if there is suspicion for giant cell myocarditis where prompt treatment can decrease mortality rates).

Diagnosis of cause

After myocarditis is diagnosed, tests to determine etiology are done. In a young, previously healthy adult who presents with symptoms of a viral infection and myocarditis, an extensive evaluation is usually unnecessary. Differentiating viral from idiopathic myocarditis is difficult, expensive, and generally of little practical importance.

A complete blood count (CBC) is helpful to assess for peripheral eosinophilia, which is present in hypersensitivity myocarditis.

Cardiac catheterization may be useful for ruling out ischemia since myocarditis can mimic myocardial infarction or myocardial ischemia.

In other cases, a biopsy of myocardial tissue may be needed to establish a diagnosis. Acid-fast stains of myocardial tissue are essential if tuberculosis (TB) is considered possible (TB myocarditis can be aggressive and can worsen rapidly with corticosteroid therapy). Myocardial samples are examined for giant cells, which are characteristic of giant cell myocarditis, and the granulomas that occur in sarcoidosis.

Other tests include acute-phase reactants, routine chemistry tests, cultures, autoimmune tests, and, when appropriate, tests for HIV, SARS-CoV-2 infection, histoplasmosis complement fixation or Lyme disease titers (in endemic areas), and antibody tests for coxsackievirus, influenza virus, and streptococcus.

- Treatment of heart failure and arrhythmias

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- Treatment of underlying disorder

Treatment of heart failure includes diuretics and nitrates for symptomatic relief. In cases of fulminant heart failure, intraaortic balloon pump (IABP), left ventricular assist device (LVAD), or transplantation may be necessary. Long-term drug treatment of heart failure involves angiotensin-converting enzyme (ACE) inhibitors, beta-blockers, aldosterone antagonists, angiotensin II receptor blockers (ARBs), or angiotensin receptor/neprilysin inhibitors (ARNIs). Atrial and ventricular arrhythmias are treated with antiarrhythmic therapy. Heart block can be treated with temporary pacing but may require insertion of permanent pacemaker if conduction abnormalities persist.

Infectious myocarditis is generally treated with supportive therapy for associated heart failure and arrhythmias. Antiviral therapy has not been shown to be helpful in the treatment of most viral etiologies, but nirmatrelvir and ritonavir can help treat myocarditis due to SARS-CoV2 infection, and oseltamivir can help treat myocarditis due to influenza. Corticosteroids may also help treat myocarditis due to SARS-CoV2 infection. Bacterial etiologies may be treated with antibiotics, but this has not been shown to be effective except possibly in the acute infectious phase. Parasitic infection should be treated with appropriate antiparasitic drugs.

Hypersensitivity myocarditis is treated by immediate discontinuation of the causative drug or cardiotoxin and, for hypersensitivity myocarditis, corticosteroid therapy. Patients with giant cell myocarditis have improved survival when treated with immunosuppressants, usually corticosteroids and cyclosporine. Myocarditis caused by sarcoidosis can be treated with corticosteroids.

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