MORE ABOUT . . . CARDIOVASCULAR DISEASES

HOW DO I RECOGNISE MYOCARDITIS: CURRENT CONCEPTS

J KER, *MB ChB, MMed (Int)*
Senior Lecturer: *Department of Physiology, University of Pretoria*

Myocarditis is a focal or diffuse inflammatory process of the myocardium.[1,2] The clinical manifestation of this process can follow one of four patterns. However, the majority of patients will be asymptomatic, as the process is often subclinical and self-limiting.[3] At the other end of the spectrum patients may present with sudden cardiac death, with myocarditis as a postmortem diagnosis.[4-6] We can divide the symptomatic group into those with cardiac and those with non-cardiac symptoms. The fourth presenting pattern of myocarditis is that of a chronic, progressive disease, presenting as dilated cardiomyopathy.[2,3]

CARDIAC SYMPTOMS IN MYOCARDITIS

- Chest pain with a clinical syndrome mimicking myocardial infarction
- Syncope
- Palpitations
- Symptoms of cardiac failure, either acute and fulminant or following the pattern of a chronic, dilated cardiomyopathy.

NON-CARDIAC SYMPTOMS IN MYOCARDITIS

- Flu-like symptoms
- Fever
- Arthralgias
- Malaise.

Another important complication of myocarditis is a disturbance of the coagulation pathway with systemic and/or pulmonary thromboemboli as a result, and as many as 15% of patients will develop a detectable left ventricular thrombus.[7-9]

ELECTROCARDIOGRAPHIC CHANGES IN MYOCARDITIS

ECG changes are common in myocarditis and include the following:[3]

- Diffuse ST-segment and T-wave abnormalities
- Prolonged QT interval
- Bundle-branch block
- Complete heart block
- Supraventricular and ventricular tachyarrhythmias.

Based on aetiology and histopathological appearance several distinct types of myocarditis have been identified in the mammalian heart.[2,10]

CAUSES OF MYOCARDITIS

Heavy metals: iron, lead, copper, mercury.
Drugs:

- Toxic — catecholamines, arsenic, anthracyclines
- Hypersensitivity reactions — penicillins, sulphonamides, streptomycin
- Physical agents: radiation, electrocution, hyperthermia.

Infectious causes

- Bacterial — Brucella, Staphylococcus, mycoplasma, mycobacteria
- Viral — Coxsackie, cytomegalovirus, dengue
- Rickettsial
- Protozoal — toxoplasma
- Parasitic — Ascaris, Echinococcus, Taenia, Trichinella.

Systemic diseases — autoimmune, vasculitides.
Miscellaneous — bee and wasp stings, carbon monoxide, phosphorus, scorpion, snake and spider bites.

HISTOPATHOLOGICAL SUBCLASS OF MYOCARDITIS

- Lymphocytic type.
- Eosinophilic type.
- Neutrophilic type.
- Mixed inflammatory cell infiltrate type.
- Giant cell type.
- Granulomatous type.

PATHOPHYSIOLOGY

Our current understanding of the pathophysiology of myocarditis is the result of studies of animal models, where animals are infected with a cardiotropic virus, such as Coxsackie B.[2] There are two phases in the virus-induced myocarditis process: the first is virus-dependent and the second is the result of the host's immune response.[11] The first phase consists of virus-induced myocytolysis and the second is a postviral immune reaction — this can be primary, directed at virus-infected myocytes or it can be a secondary autoimmune reaction against cardiac myocytes, beta receptors, calcium channels and/or mitochondria. This second phase is characterised by the infiltration of the myocardial interstitium by inflammatory cells, which persist long after the disappearance of viral particles[10] and the production of various proinflammatory cytokines. These inflammatory cytokines activate endothelial cells, recruit more inflammatory cells to the myocardial interstitium and some of them have significant negative inotropic effects.[2] Recently, it has been suggested that molecular mimicry may play a role in the development of an autoimmune myocarditis after cardiac insults, such as viral infection, drug exposure and various miscellaneous causes of car-

diac injury.[2] Currently it appears that the host's immunological response is the main culprit in the myocardial inflammatory process and furthermore that this host response perpetuates inflammation in the myocardium long after the initial insult has disappeared.

The only accepted light microscopic criteria for the diagnosis of myocarditis are the Dallas criteria.[10,11] According to this set of criteria myocardial biopsy specimens are considered diagnostic of active myocarditis if light microscopy reveals infiltrating, interstitial, inflammatory cells with myocytolysis. Borderline (or ongoing) myocarditis is present if there is inflammatory cell infiltration but no myocytolysis. The biopsy is negative for myocarditis if there is neither infiltration of the cardiac interstitium by inflammatory cells nor myocytolysis.

TREATMENT

Treatment of myocarditis consists of three aspects:

- identification and removal of the cause (if possible)
- treatment of cardiac failure
- management of ventricular dysrhythmias.

Investigations should be made for removable causes of myocarditis (e.g. drugs). Currently, studies suggest that immunosuppression should not be used in the routine treatment of patients with myocarditis with the important exception of patients with myocarditis due to systemic, autoimmune disease, such as lupus, polymyositis and systemic sclerosis.[2]

References available on request.