29.9B: Myocarditis and Endocarditis

Endocarditis and myocarditis are driven by inflammation of the heart.

LEARNING OBJECTIVES

Distinguish between myocarditis and endocarditis and their effects on the heart

KEY TAKEAWAYS

Key Points

- Inflammation of the inner layer of the heart wall, the endocardium, often including the heart valves, is called endocarditis.
- Endocarditis may be infective or non-infective. Diagnosis of endocarditis includes clinical identification of the vegetation, echocardiogram, and blood cultures to demonstrate the infective microorganisms involved in infective endocarditis.
- In subacute infective endocarditis, the vegetation may include granulomatous tissue which can become fibrotic or calcify.
- In non-infective endocarditis such as nonbacterial thrombic endocarditis (NBTE), also called marantic endocarditis, vegetations are typically small and sterile (do not contain microorganisms) and typically do not engender an inflammatory response.
- Inflammation of the myocardium, the heart muscle, is called myocarditis, typically characterized by infection of the heart, an inflammatory infiltrate, and heart muscle damage with or without necrosis (tissue death).
Myocarditis most commonly has an infectious cause, such as the parovirus B19, lyme disease, or other viral and nonviral pathogens, but may also be an autoimmune reaction.

Key Terms

- **inflammation**: A condition of any part of the body, consisting in congestion of the blood vessels, with obstruction of the blood current, and growth of morbid tissue. It is manifested outwardly by redness and swelling, attended with heat and pain.

Endocarditis is an inflammation of the inner layer of the heart, the endocardium. It usually involves the heart valves (native or prosthetic valves). Other structures that may be involved include the interventricular septum, the chordae tendineae, the mural endocardium, or even on intracardiac devices.

Endocarditis is characterized by a prototypic lesion, the vegetation, which is a mass of platelets, fibrin, microcolonies of microorganisms, and scant inflammatory cells. In the subacute form of infective endocarditis, the vegetation may also include a center of granulomatous tissue, which may fibrose or calcify.

Classification and Diagnosis

There are multiple ways to classify endocarditis. The simplest classification is based on etiology: either infective or non-infective, depending on whether a microorganism is the source of the inflammation or not. Regardless, diagnosis of endocarditis is based on the clinical features, investigations such as echocardiogram, as well as any blood cultures demonstrating the presence of endocarditis-causing microorganisms.

Since the valves of the heart do not receive any dedicated blood supply, defensive immune mechanisms (such as white blood cells) cannot directly reach the valves via the bloodstream. If an organism (such as bacteria) attaches to a valve surface and forms a vegetation, the host immune response is blunted. The lack of blood supply to the valves also has implications on treatment, since drugs also have difficulty reaching the infected valve. Normally, blood flows smoothly through these valves. If they have been damaged (from rheumatic fever, for example) the risk of bacteria attachment is increased.

Types of Endocarditis

Nonbacterial thrombic endocarditis (NBTE) or marantic endocarditis is most commonly found on previously undamaged valves. As opposed to infective endocarditis, the vegetations in NBTE are small, sterile, and tend to aggregate along the edges of the valve or the cusps. Also unlike infective endocarditis, NBTE does not cause an inflammation response from the body. NBTE usually occurs during a hypercoagulable state such as system wide bacterial infection, or pregnancy, though it is also sometimes seen in patients with venous catheters.

Another form of sterile endocarditis is termed Libman-Sacks endocarditis; this form occurs more often in patients with lupus erythematosus and is thought to be due to the deposition of immune complexes. Like NBTE, Libman-Sacks endocarditis involves small vegetations, while infective endocarditis is composed of large vegetations. These immune complexes precipitate an inflammation reaction, which helps to differentiate it from NBTE. Also unlike NBTE, Libman-
Sacks endocarditis does not seem to have a preferred location of deposition and may form on the undersurfaces of the valves or even on the endocardium.

**Myocarditis**

Myocarditis or inflammatory cardiomyopathy is inflammation of heart muscle (myocardium). Myocarditis is most often due to infection by common viruses, such as parvovirus B19, less commonly nonviral pathogens such as Borrelia burgdorferi (Lyme disease) or Trypanosoma cruzi, or as a hypersensitivity response to drugs.

Myocarditis is often an autoimmune reaction. Streptococcal M protein and coxsackievirus B have regions (epitopes) that are immunologically similar to cardiac myosin. After the virus is gone, the immune system may attack cardiac myosin.

**Symptoms and Outcomes**

The consequences of myocarditis thus also vary widely. It can cause a mild disease without any symptoms that resolves itself, or it may cause chest pain, heart failure, or sudden death. An acute myocardial infarction-like syndrome with normal coronary arteries has a good prognosis. Heart failure, even with dilated left ventricle, may have a good prognosis. Ventricular arrhythmias and high-degree heart block have a poor prognosis. Loss of right ventricular function is a strong predictor of death.

Symptoms in infants and toddlers tend to be more nonspecific, with generalized malaise, poor appetite, abdominal pain, and/or chronic cough. Later stages of the illness will present with respiratory symptoms with increased work of breathing and is often mistaken for asthma.

Since myocarditis is often due to a viral illness, many patients give a history of symptoms consistent with a recent viral infection, including fever, rash, diarrhea, joint pains, and easy fatigueability.

A large number of causes of myocarditis have been identified, but often a cause cannot be found. In Europe and North America, viruses are common culprits. Worldwide, however, 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.