Surgical Treatment of Infective Endocarditis: “BOMBS” and Extreme Procedures on the Critically Ill

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Infective endocarditis is a disease in which a microorganism colonizes a focus in the heart, producing fever, heart murmur, splenomegaly, embolic manifestations, and bacteremia or fungemia. Early diagnosis of this condition is extremely important because it almost invariably leads to devastating complications and death if not treated effectively.

EPIDEMIOLOGY Predisposing factors for infective endocarditis are cardiac abnormalities that disrupt the endocardium by means of a jet injury, as well as the presence of blood-borne microorganisms that colonize these abnormal surfaces. Congenitally bicuspid aortic valve is the most common predisposing lesion for endocarditis of the aortic valve. In addition, congenital abnormalities of the aortic valve, degenerative calcific aortic stenosis, aortic insufficiency secondary to connective tissue disorders, and rheumatic aortic valve disease, are also predisposing lesions for infection.

It is difficult to determine the incidence and prevalence of native aortic valve endocarditis in the general population because this disease is continuously changing. The annual incidence of infective endocarditis is estimated to range from 1.7 to 7.0 episodes per 100,000 person-years in North America.

Patients with prosthetic aortic valves are reported to have an incidence of infective endocarditis of 0.2 to 1.4 episodes per 100 patient-years, which varies with the type of aortic valves. Approximately 1.4% of patients undergoing aortic valve replacement develop prosthetic valve endocarditis during the first postoperative year. Dental extractions have been demonstrated to produce bacteremia; however, even simple mastication, tooth brushing or cleaning, and oral irrigation can produce transient bacteremia. Endoscopic procedures may also produce bacteremia. Intravenous drug users are particularly susceptible to infective endocarditis, which often occurs in structurally normal heart valves.

PATHOGENESIS and PATHOLOGY In 1928, Grant and colleagues theorized that platelet-fibrin thrombi on the heart valve served as a nidus for bacteria adherence. In 1963, Angrist and Oka introduced the term "nonbacterial thrombotic endocarditis" to describe sterile vegetations on a heart valve and provided experimental animal evidence supporting the role of these vegetations in the pathogenesis of endocarditis.

Infective endocarditis of the aortic valve not only causes destruction of the aortic cusps, paravalvular abscess, and cardiac fistulas, but also can cause coronary and systemic embolization of vegetations. Cerebral infarction, either ischemic due to arterial occlusion or hemorrhagic due to rupture of the mycotic aneurysm, is common in these patients. Mycotic aneurysms, infarcts, and abscesses of other organs such as spleen, liver, kidneys, and limbs are also common. Aortic valve endocarditis with a large vegetation that prolapses into the left ventricle and comes in contact with the anterior leaflet of the mitral valve can cause secondary involvement of this valve.

Infection of a mechanical heart valve is usually located in its sewing ring. Infection of a porcine or pericardial valve may involve the cusps, the sewing ring, or both. Infection in aortic valve homografts and pulmonary autografts resembles that of the native aortic valve: it begins in the aortic cusps and destroys them, causing aortic insufficiency, but it may also extend into surrounding structures. Endocarditis after
aortic root replacement with mechanical valves frequently causes dehiscence of the valve from the aortic annulus with consequent false aneurysm.\textsuperscript{8}

**MICROBIOLOGY** The microbiology of infective endocarditis of the aortic valve depends on whether the valve is native or prosthetic, and whether the infection is hospital- or community-acquired. *Staphylococcus aureus* and *Streptococcus viridans* are the most common microorganisms responsible for native aortic valve endocarditis.\textsuperscript{9} *S. aureus* is extremely virulent and able to cause infection in patients with normal aortic valves. *S. viridans* is not as virulent and causes infection that often follows a protracted course. *Staphylococcus epidermidis* and various other streptococci can also cause endocarditis.

Endocarditis due to gram-negative bacteria is uncommon, but it is often resistant to antibiotic therapy and may cause serious complications. *Haemophilus, Actinobacillus, Cardiobacterium, Eikenella*, and *Kingella* (the HACEK group) are gram-negative bacilli grouped together due to their characteristic fastidiousness requiring a prolonged incubation period before growth. Fungal endocarditis is rare but extremely serious. *Candida albicans* and *Aspergillus fumigatus* are the usual agents.

The microbiology of prosthetic aortic valve endocarditis is different from that of the native valve.\textsuperscript{10} Prosthetic valve endocarditis has been arbitrarily classified as *early* when it occurs within the first 2 months after surgery, and *late* when it occurs after 2 months. However, it is possible that many cases of prosthetic valve endocarditis that occur during the first year after surgery are acquired at the time of implantation of the artificial heart valve. This may be particularly true when the infection is caused by the HACEK group of bacteria. Early prosthetic valve endocarditis is caused by contamination of the valve at the time of implantation by perioperative bacteremia. *Staphylococcus epidermidis, S. aureus*, and *Enterococcus faecalis* are among the more common microorganisms responsible for early prosthetic valve endocarditis. The sources of late prosthetic valve endocarditis are more difficult to determine. Bacteremia is probably the principal cause of late endocarditis. Although streptococci and staphylococci are commonly encountered in these patients, a myriad of microorganisms can cause late prosthetic valve endocarditis.\textsuperscript{11}

Nosocomial infections are often caused by *S. aureus* or other staphylococci.

In a small proportion of cases of aortic valve endocarditis, no microorganism can be cultured from either the blood or surgical specimens. This is called "culture-negative endocarditis," but it is important to rule out fastidious microorganisms and every effort should be made to identify them.

**PRESENTATION AND DIAGNOSIS** It is helpful to classify infective endocarditis as acute and subacute because there are major differences between these two clinical presentations. Subacute endocarditis is often caused by less virulent microorganisms such as *S. viridans*. When this organism affects a diseased aortic valve, the clinical course is protracted and antibiotics alone cure most cases. On the other hand, acute endocarditis is frequently caused by a virulent microorganism such as *S. aureus* and may affect a normal aortic valve. The clinical course is acute, and antibiotics alone seldom cure the infection.

The onset of subacute endocarditis in most cases is subtle, with low-grade fever and malaise. Patients think they have the "flu" and are often treated with oral antibiotics for a week to 10 days with improvement of symptoms. However, in most cases the symptoms recur a few days after stopping antibiotics. In the majority of cases no predisposing factor is identified. An aortic valve murmur is present in nearly all patients because they have preexisting aortic valve disease. Splenomegaly is common. Clubbing of the fingers and toes may develop in long-standing cases. Skin and mucous membrane signs occur late in this form of endocarditis. Petechiae appear on any part of the body. Small areas of hemorrhage may be seen in the ocular fundi. Hemorrhages in the nail beds usually have a linear distribution near the distal end, hence the name splinter hemorrhages. Osler nodes are acute, tender, barely palpable nodular lesions in the pulp of the fingers and toes. Bacteria have been cultured from these lesions. Embolization of large vegetation fragments may cause dramatic clinical events such as acute myocardial infarction (AMI), stroke, or splenic or hepatic infarcts. Any other organ also may be involved. Destruction of the aortic cusps causes aortic insufficiency and heart failure. The blood pathology is not distinctive in subacute endocarditis. Anemia
without reticulocytosis develops in patients untreated for more than a few weeks. The leukocyte count is moderately elevated. Blood cultures frequently identify the offending microorganism.

The clinical course of acute endocarditis is often aggressive and patients are often sicker with overwhelming signs of sepsis. Early metastatic infections are common. Acute endocarditis is common in patients with no preexisting aortic valve disease. Early cardiac decompensation due to aortic insufficiency is common. Paravalvular abscess is also common, and depending on the location of the abscess, the electrocardiogram may show an increased PR interval or heart block. The blood picture is one of acute sepsis and blood culture often isolates the infecting agent.

Doppler echocardiography is extremely useful in the diagnosis and management of infective endocarditis and should be considered early. Echocardiography can detect vegetations as small as 1 or 2 mm in size, but it is more reliable in native than in prosthetic valve endocarditis. It is more useful for tissue than for mechanical valves because of the acoustic shadowing of ball, disc, or leaflet motion of mechanical heart valves. Echocardiography is also extremely sensitive for detecting paravalvular abscess and cardiac fistulas.12

Heart catheterization and coronary angiography increase the risk of embolization in patients with aortic valve vegetations and should be avoided. Newer computed tomography (CT) imaging techniques to diagnose coronary artery disease are useful in these patients.

TREATMENT An appropriate antibiotic is the most important aspect of the management of patients with infective endocarditis. Directed antibiotic therapy should be started soon after obtaining several blood cultures. A combination of two or three antibiotics that potentiate each other is often needed in the treatment of endocarditis caused by virulent microorganisms. Intravenous antibiotic therapy is continued for 6 weeks.

Surveillance blood cultures are performed in 48 hours to monitor the efficacy of antibiotic therapy. The patient must be watched closely for signs of CHF, coronary and systemic embolization, and persistent infection. Daily electrocardiograms and frequent echocardiograms are performed during the first 2 weeks of treatment. It is important to operate on patients before they develop intractable heart failure, cardiogenic or septic shock, or extensive aortic root abscesses. Patients with vegetations larger than 10 mm present a clinical problem because they are more likely to develop serious complications and early surgery is justifiable.13

Prosthetic valve endocarditis is best treated by early surgery, particularly in patients with mechanical valves. Acute endocarditis of the aortic valve due to S. aureus is also best treated with early surgery because of the destructive power of the bacteria.14

Patients with neurologic deficits should have CT or magnetic resonance imaging performed to determine if the cerebrovascular accident is ischemic or hemorrhagic. Ischemic damage is far more common than hemorrhagic damage, but both are associated with increased mortality and morbidity. Mycotic aneurysms should be treated before valve surgery. Aortic valve replacement should be postponed for 2 weeks after an ischemic stroke and 4 weeks after a hemorrhagic stroke if possible.14

SURGICAL TREATMENT Patients who need surgery are often very sick and may be in CHF. For this reason and because they often require complex and long surgical procedures, myocardial protection is of utmost importance. When the infection is limited to the cusps of the native aortic valve or a bioprosthetic valve, complete removal of the valve and implantation of a biologic or mechanical valve usually resolves the problem. There is no evidence that bioprostheses are better than mechanical valves in patients with active infective endocarditis.15

Surgery for aortic root abscess and/or cardiac fistulas is challenging. The most important aspect in the surgical treatment of these patients is radical resection of all infected tissues.16 These patients frequently
require replacement of the entire aortic root and reconstruction of the surrounding structures that are also involved by the abscess. Thus, patching of the interventricular septum, dome of the left atrium, intervalvular fibrous body, right atrium, and pulmonary artery may be necessary, as well as repair of the left and/or right coronary arteries.

Postoperative complications are common after surgery for active infective endocarditis. Septic patients may have severe coagulopathy and may bleed excessively after cardiopulmonary bypass. Antifibrinolytic agents, particularly aprotinin, should be used. Transfusion of platelets, cryoprecipitate, and fresh frozen plasma are often necessary to obtain hemostasis. Radical resection of aortic root abscess may cause heart block, for which a permanent pacemaker will be needed postoperatively. Depending on the patient’s clinical condition before surgery, multiorgan failure may develop postoperatively. Neurologic deterioration may occur in patients with preexisting cerebral emboli.  

**Clinical Results** The prognosis of aortic valve endocarditis depends largely on when the disease is diagnosed, on the offending microorganism, and how promptly it is treated. Patients with prosthetic aortic valve endocarditis have a more serious prognosis than patients with native aortic valve endocarditis, and nosocomial infections are associated with higher mortality than community-acquired infections. The results of surgery for infective endocarditis have improved significantly during the past three decades. The operative mortality for patients with infection limited to the cusps of the aortic valve is largely dependent on the patients’ presentation at the time of surgery, age, and comorbidities. Most reports indicate that the operative mortality is under 10%. The operative mortality is higher for prosthetic valve endocarditis and ranges from 20 to 30%. Surgery for aortic root abscess is also associated with higher operative mortality. The 10-year survival after surgery for infective endocarditis is around 50 to 60%.

**REFERENCES:**