Infective endocarditis: Perioperative management and surgical principles

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Despite advances in microbial prevention and elimination, the frequency of endocardial infection is still increasing and it remains to be a serious condition. The strategies and aggressiveness of medical and surgical algorithms for managing these patients are evolving and having a significant effect on morbidity and mortality. This review addresses the current understanding of the processes by which the most common and most threatening complications occur, and the current management strategies that cardiologists and cardiac surgeons should be aware of when treating these seriously ill patients. (J Thorac Cardiovasc Surg 2014;147:1133-41)

Despite major advances in diagnostic modalities and antimicrobial therapies, infective endocarditis (IE) remains an extremely ominous infection, with a 1-year mortality rate of up to 40%.[1,2] Management of these critically ill patients requires an understanding of the disease process in its various microbial, hemodymanic, embolic, and immunologic forms. At the time of diagnosis, patients have often progressed to complications through 1 aspect of the disease. An appreciation of the spectrum of the disease processes is crucial for providing a timely and effective intervention. In this article, the processes by which the most common and most threatening complications occur, as well as what every physician ought to know when treating these seriously ill patients are discussed.

OVERVIEW OF COMPLICATIONS

After bacteria seed the endocardium, erosions into various cardiac structures take place though an interplay of direct bacterial invasion, enhanced inflammatory response, and liquifactive enzyme release within cardiac tissues. This is particularly pronounced in IE caused by *Staphylococcus aureus*, especially in the aortic valve position. This is probably due to the less annular fibrous tissue support in the aortic position compared with the mitral position. Unfortunately, IE tends to occur more frequently in the aortic valve than any other valve, and the frequency of *S aureus* as the cause of IE has increased dramatically over the past 2 decades, from 2% in 1990 to 25% in 2009.[3]

Congestive heart failure (CHF) through a sudden volume overload on the ventricles, whether caused by a sudden regurgitant lesion or shunt creation, can complicate IE. Sudden regurgitation usually occurs as a result of chordal rupture in native valve endocarditis (NVE), a valve leaflet perforation in NVE or in bioprosthetic valve IE, or valve dehiscence in prosthetic valve endocarditis (PVE). In NVE and PVE, CHF is the complication with the greatest independent impact on prognosis whether medically or surgically treated.[4,5] Early studies comparing antimicrobial therapy with surgery in patients with IE complicated by CHF demonstrated a clear superiority with surgery (23% vs 71% mortality rates).[6-8] Much less commonly, heart failure can be obstructive in nature when large vegetation obstructs a heart chamber outflow.

A similar pathophysiology also leads to paraannular extension (PAE) of the infection. Given the similar pathophysiology, PAE is also more common with necrotizing organisms, and in the aortic position. In addition, PAE is one of the most frequent complications of IE, occurring in up to 100% of infected prosthetic valves and 40% of infected native valves.[9] The infectious process around the valve weakens the annulus, and eventually leads to tissue destruction, valve dehiscence, abscess formation, and sometimes fistulization. This is associated with an increased occurrence of CHF and a higher mortality rate. A PAE-related fistula has been shown to lead to up to 40% mortality.[10,11]

In addition to the local destruction that complicates IE, systemic embolization is the most common complication. Emboli usually consist of vegetations or friable necrotic and often infected tissues. Unlike local effects, this is more common in the mitral valve position. Although the embolic risk is high (embolic events occur in up to 50% of patients), this risk declines dramatically with initiation of antibiotic treatment, and even more after 2 weeks of effective continuation.[3,12-14] The rate of systemic embolization is significantly higher in patients who have had a previous embolic event, those in the first 2 weeks of antibiotic therapy, those with left-sided IE especially in the mitral position, those with mobile vegetation that is large (10-15 mm) or increasing in size despite antibiotic therapy, and those patients infected with certain pathogens

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Abbreviations and Acronyms

ACC = American College of Cardiology
CHF = congestive heart failure
CT = computed tomography
ESC = European Society of Cardiology
GFP = glutaraldehyde-fixed pericardial
IE = infective endocarditis
MIC = minimum inhibitory concentration
MRI = magnetic resonance imaging
NVE = native valve endocarditis
PAE = paraannular extension
PVE = prosthetic valve endocarditis
TEE = transesophageal echocardiography
TTE = transthoracic echocardiography

(*S aureus*, fungi, enterococci, and HACEK).[3,12,15] No vascular bed is immune, but 65% of these pathogens usually affect the brain, 90% in the middle cerebral artery territory. Renal and splenic vascular beds are also commonly involved.[16] When a septic embolus reaches its target, it ends up causing an infarction, an abscess usually with *S aureus*, or rarely a mycotic aneurysm when the intramural vasa vasora are involved. This is usually followed by wall attenuation and progressive aneurysmal dilatation of the affected vessel.

Less virulent organisms usually have a less pronounced local effect. An indolent subacute course triggers an antibody response that puts the patient at risk of immune complex glomerulonephritis. Acute renal failure complicates up to 30% of patients with IE and is associated with a poor prognosis. This is especially true when it is compounded by septic renal embolism, prerenal failure caused by CHF, and the nephrotoxic effects of gentamicin and vancomycin, commonly used in high doses for treating these patients.[17]

MAKING THE DIAGNOSIS

An accurate diagnosis is crucial. Missing a diagnosis likely leads to a poor outcome or death, and overdiagnosing patients leads to exposure to high doses of potentially toxic antimicrobials that are unnecessary. Several defining criteria have been proposed including those by Pelletier and Petersdorf[18] and Von Reyn and colleagues[19]; however, the more recent Duke criteria and their revisions in 2000 seem to be the most widely used.[20,21] Based on the latter, a diagnosis is made if any of the following is present: (1) pathologically tested tissue; (2) 2 major criteria; (3) 1 major plus 3 minor criteria; or (4) 5 minor criteria (Table 1). A high pretest probability justifies commencing treatment even if the diagnosis is not definite according to the criteria.

Cornerstone major criteria include blood cultures and echocardiographic findings. However, an accurate diagnosis is not always straightforward, given that 25% to 30% of patients in this era present with no previously known cardiac structural abnormality.[22] This is in part due to the more acute rather than subacute disease process taking place with the increased prevalence of *S aureus* as a causative agent. An accurate diagnosis has been made even more difficult by increasing rates of culture-negative IE. In the United States, 79% of cases with culture-negative IE were found to be due to early administration of antimicrobials or faulty culturing technique, emphasizing their relative importance and how seemingly trivial details may alter or complicate the diagnosis.[23]

Echocardiography should be done as soon as IE is suspected. The role of echocardiography goes beyond the diagnosis, and repeat echocardiography is recommended on clinical deterioration or when complications are suspected. Echocardiography guides the decision on whether to operate and when, as discussed later. However, the role of transthoracic echocardiography (TTE) in the assessment of left-sided IE is controversial, because of its significantly lower negative predictive values compared to transesophageal echocardiography (TEE).[24,25] Although TEE has a near 100% negative predictive value with NVE, it is operator dependent and is far less sensitive for PVE and abscesses of the mitral valve associated with posterior annular calcification.[3,26] In these situations, repeat TEE as well as a combination of TTE and TEE might mitigate the limitations caused by acoustic shadowing. Unfortunately, 15% of patients with proven IE were reported to have no echocardiographic findings.[27]

STARTING MANAGEMENT

Managing a patient with IE aims at 2 goals: eradication of infection and restoration of cardiac structures. Eradicating infection, whether medically or surgically, should abort the disease process, and thus prevent further local, hemodynamic, immunologic, or embolic complications. Structural restoration is primarily surgical and is aimed at repairing the damaged tissue in which healing would not be sufficient, and would otherwise have short-term or long-term hemodynamic implications.

Once the diagnosis of IE is suspected, at least 3 sets of blood culture samples should be drawn from different sites and at 30-minute intervals.[28,29] This should be followed immediately by initiation of intravenous empirical antibiotic therapy as shown in Table 2. With no justification for delay, this empirical therapy should be qualitatively bactericidal and quantitatively in high doses for up to 6 weeks. Selecting the best antibiotic for a particular patient should be guided by the presence or absence of previous antibiotic use, whether this is a suspected case of NVE,

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