nodes), and funduscopic findings of cotton-wool spots or Roth's spots. Conjunctival and splinter hemorrhages are rare in SLE but common in SBE. Microscopic hematuria is the usual renal manifestation of SBE (i.e., focal glomerulonephritis), but full-blown nephritis with proteinuria and hematuria are typical of SLE renal involvement. Although clinical findings of SLE and SBE may overlap, SBE is ruled out by the absence of high-grade or continuous bacteremia.

Atrial myxomas may mimic SBE with fever, murmurs, and embolic phenomena (e.g., splinter hemorrhages). Highly elevated ESR levels are common with atrial myxomas, but biologically false-positive results on Venereal Disease Research Laboratory (VDRL) testing, elevated rheumatoid factors, and renal involvement are not seen. On TTE or TEE, atrial myxomas appear as masses or vegetations on the atrial surface rather than on a valve as in IE. SBE is ruled out by the absence of bacteremia.

Besides clinical mimics of SBE, there are also echocardiographic mimics, including papillary fibromas, thrombi, calcified valves, myxomatous degeneration, and marantic endocarditis. These disorders are usually unaccompanied by fever or bacteremia. The term *marantic endocarditis* refers to uninfected vegetations with a murmur and negative blood cultures that occur secondary to malignancy. Patients with marantic endocarditis are afebrile unless fever is caused by the underlying malignancy (e.g., lymphoma). The patient with marantic endocarditis due to lymphoma may have fever, splenomegaly, and other manifestations of SBE. Negative blood cultures effectively rule out IE. Infectious CNE (e.g., Q fever) may show little or no visible vegetations. Infectious CNE should be considered if fever, murmur, and vegetation are present along with peripheral manifestations of IE.

Treatment

Effective treatment of IE depends on the antibiotic susceptibility of the pathogen, the penetration of the antibiotic into the vegetation, and the appropriate duration of antibiotic therapy. Antibiotics selected for IE preferably should be bactericidal. In IE, the organisms are deeply embedded in the vegetation, and prolonged therapy is necessary for penetration and sterilization of the vegetation. Early in IE therapy, blood cultures rapidly become negative, but treatment is continued because infection in the vegetation has not been eradicated. Multiplication of bacteria, which is required for bactericidal activity of antibiotics, is reduced within vegetations and is one reason for the requirement for prolonged antibiotics. It is important to note that in cases of *Staphylococcus aureus* endocarditis, blood cultures may not clear rapidly and may remain positive for days despite appropriate antibiotic therapy. Penetration into the vegetation is critical; for example, viridans streptococci are highly susceptible to β-lactam antibiotics but require a prolonged course of antimicrobial therapy to eradicate the pathogens in the vegetation.

Whereas some cases of uncomplicated IE may be treated with 2 weeks of antimicrobial therapy, the usual duration of monotherapy or combination therapy is 4 to 6 weeks, depending on the pathogen. Effective antimicrobial therapy does not eliminate supportive or embolic complications of endocarditis. Therapeutic failure is usually related to valvular destruction, a complication that may require valve replacement. Suppurative intracardiac or extracardiac complications usually require drainage for cure of IE. The overarching principles of IE therapy are presented in Table 93-6, and Table 93-7 provides an outline of specific antibiotic regimens that may be used to treat IE.

TABLE 93-6 PRINCIPLES OF THERAPY FOR INFECTIVE ENDOCARDITIS

1. Antibiotic selection initially is made empirically on the basis of physical examination and clinical history.
2. Bactericidal antibiotics are prescribed.
3. The MIC and MBC are measured to insure adequate dosing of the agent.
4. Intermittent dosing provides superior penetration into the thrombus compared with continuous infusion; penetration is directly related to peak serum level.
5. The patient should be treated in a health care facility for the first 1-2 wk.
6. The usual duration of therapy is 4-6 wk.
7. A 4-wk course is appropriate for an uncomplicated case of NVE (a shorter course of 2 wk may be appropriate in some cases); a 6-wk course is required for the treatment of PVE and those infections with large vegetations (i.e., infection by HACEK organisms*).

Modified from Brusch JL: Diagnosis of infective endocarditis. In Brusch JL, editor: Infective endocarditis, New York, 2007, Informa Healthcare, pp 241–254.
MBC, Minimal bactericidal concentration; MIC, minimal inhibitory concentration.
*HACEK organisms: *Haemophilus* spp, *Actinobacillus actinomycetcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella kingae*.

Complications of endocarditis may be intracardiac or extracardiac, and they may also be classified by damage mechanism (i.e., immunologic versus infectious). The infectious intracardiac complications of IE include purulent pericarditis and paravalvular abscess; they manifest clinically with persistent fever or persistent bacteremia despite appropriate antibiotic therapy. Complications may be septic or immunologic; for example, splenic involvement may be immunologic (splenic infarct) or septic (splenic abscess). Embolic events are related to vegetation size. Bland central nervous system emboli (e.g., aseptic meningitis) may complicate SBE, whereas septic emboli (e.g., acute bacterial meningitis) may complicate ABE. Particularly with ABE, there may be valvular perforation or destruction resulting in acute congestive heart failure. It is often these complications that dictate whether and when surgery will occur. The indications for surgical intervention are shown in Table 93-8. As a general principle, paravalvular abscess or intractable congestive heart failure requires urgent surgical intervention. Persistent vegetations or embolic disease that occurs after 1 week of appropriate antibiotic therapy should also prompt surgical consideration.

Prognosis

The prognosis of all forms of IE depends directly on any complications related to the infection. Consequently, early diagnosis and initiation of appropriate antibiotic therapy is the key to limiting mortality. Recent studies have supported the role of early surgical intervention, when appropriate, as a significant aid to decreasing morbidity and mortality, specifically in relation to having fewer embolic events. If treated in a timely fashion and with appropriate antibiotics, the cure rate for viridans streptococci and *S. bovis* is estimated to be 98% in NVE and up to 88% in PVE. Right-sided endocarditis in intravenous drug abusers is usually caused by *S. aureus* and typically has a cure rate of 90% in NVE and 75% to 80% in PVE. However, among non–intravenous drug abusers, cure rates in IE involving *S. aureus* are far lower: 60% to 70% in NVE and 50% in PVE. When gram-negative bacilli or fungal organisms are the causative agent,