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Infective Endocarditis

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Infective Endocarditis

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I. INTRODUCTION

Infective endocarditis (IE) has been recognized as a separate clinical entity for more than 100 years. Despite advances in both the diagnosis and treatment of this challenging disease, the morbidity and mortality associated with IE remain high. IE is defined as the presence of an active infection on the endocardium that usually involves 1 or more valve surfaces. The infection is not necessarily confined to the endocardial surface but can involve other cardiac structures such as myocardium, chordae, pericardium, and walls of adjacent blood vessels as well as endovascular devices. Accurate and early diagnosis is crucial because prompt management can have profound consequences on clinical outcome. However, the diagnosis of endocarditis can be challenging as signs and symptoms may be subtle and vague. The evolution of diagnostic criteria, along with developments in laboratory tests and advances in imaging technology, has substantially enhanced the diagnosis and, equally important, has helped prevent prolonged and unnecessary medical or surgical therapy.

II. EPIDEMIOLOGY

An estimated 10,000 to 15,000 new cases of endocarditis are diagnosed annually in the United States.¹ The true incidence of IE has been difficult to ascertain because the diagnostic criteria have changed throughout the past 3 decades. Nonetheless, over this time the incidence has been increasing, with survey estimates ranging from 1.7 to 6.2 cases per 100,000 persons annually.² This increase is probably related to the growing numbers of elderly in the population and the occurrence of age-related degenerative valvular disease among them as well as to increases in the number of injection drug abusers, nosocomial infections with resistant organisms, immunocompromised hosts, and prosthetic implantations. Rheumatic heart disease (RHD) historically has been a significant predisposing factor for developing IE (and is still so in developing countries) but now is related to less than 20% of cases.² A

report evaluating endocarditis cases between 1938 and 1967 showed RHD to be present in 39% of cases, whereas a recent report showed an RHD rate of 6%.^{3,4}

There are a number of risk factors for developing IE. Structural heart disease accounts for approximately three quarters of IE cases.^{2,3} The most significant risk factor for right-sided IE is injection drug abuse, although left-sided IE is equally prevalent in this high-risk group. Breaks in colonized mucosal surfaces confers risk for IE with certain organisms. *Streptococcus bovis* IE, for example, is highly associated with presence of colonic lesions due to inflammatory bowel disease, polyps, or malignancy. Nosocomial IE frequently is related to indwelling vascular devices or focal infections (eg, respiratory tract or an infected surgical site). Although more than 100,000 heart valves are implanted in the United States annually, IE of prosthetic valves is infrequent, comprising only 7% of cases in one report.⁵ Prosthetic-valve endocarditis (PVE) within the first 2 months after implantation (early PVE) is usually caused by nosocomial staphylococcal infections, while PVE occurring more than 1 year after implantation (late PVE) is usually caused by the organisms that cause community-acquired native valve IE.⁶ PVE occurs in 1% to 4% of valve recipients in the first year after implantation, and this rate increases by 1% per year thereafter.⁵ The type of prosthetic valve does not appear to alter the risk of developing IE.

III. PATHOGENESIS AND MICROBIOLOGY

Damage to the endocardial lining in the heart must be present for an infection to establish a vegetative lesion on a valvular surface because the endothelial lining is normally highly resistant to infection.⁷ Damage to the endocardial surface can arise from many etiologies, either congenital or acquired, and damage frequently occurs as a result of abnormal blood jets from high to low pressure regions inside the heart (eg, valvular disease that creates areas of turbulent flow). Patients with ventricular septal defects develop vegetative lesions on the right ventricular side of the defect.⁸ Patients with aortic regurgitation who develop IE tend to form vegetative lesions on the chordae