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Infective Endocarditis

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Summary (Abstract)

In this report we are going to discuss mechanism of infective endocarditis , the major and minor criteria (diagnose) and the way of treatment.

Introduction

Infective endocarditis (IE) is an infection of the heart valves. A large number of different microorganisms are capable of causing this disease. Depending on the particular pathogen, endocarditis can be indolent, progressing slowly over weeks to months, or fulminant, presenting with a very toxic picture over several days. In the preantibiotic era, this disease was almost uniformly fatal. While endocarditis can now be treated medically, it is a classic example of an infectious disease where "bactericidal" antibiotics are absolutely necessary because the local and systemic host defense mechanisms are of limited benefit.

Discussion

Lewis and Grant (Heart 10:21, 1923) were the first to clearly describe the pathogenesis of IE. They recognized that the disease often followed a transient bacteremia in individuals with an anatomically deformed cardiac valve. We now understand that infection results from a sequence of events: transient bacteremia, seeding of a valvular surface and formation of a mature vegetation. The factors associated with these events are summarized below. a) Transient bacteremia. Individuals with preexistent valvular disease are at risk of subacute IE resulting from transient bacteremias. Numerous studies have demonstrated that minor trauma (tooth brushing, use of oral irrigation devices, urethral catheterization) may be associated with a low grade bacteremia. In acute IE the bacteremia may also be from an inapparent focus (i.e. uninfected), from another site of infection, or from direct intravenous injection. b) Site of bacterial seeding on the cardiac valve i) Nonbacterial thrombus (NBT). In subacute infective endocarditis bacteria seed sites of previous micro or macroscopic damage. These sites are characterized by the deposition of a platelet-fibrin thrombus. This was first recognized by Angrist and called a nonbacterial thrombus. These lesions form as a result of mechanical stress or antecedent valvular disease. In acute IE, the NBT may not be necessary, the more virulent organisms appear capable of colonizing normal cardiac valvular surfaces. ii) Hemodynamic factors. Infective endocarditis develops on one or more cardiac valves; more often on the left side than on the right. The proportion of cases involving the tricuspid valve is greater with ABE (acute bacterial endocarditis) and with drug addicts. Rodbard demonstrated that the following hemodynamic features predicted the anatomic site of vegetation formation: a. the presence of a high-pressure source (e.g. left ventricle). b. high velocity flow through a narrow orifice (such as an insufficient mitral or aortic valve). c. a low-pressure chamber or "sink" beyond the orifice (such as left atrium, or left ventricle during diastole). He subsequently showed that infected vegetations from cases of endocarditis generally exist on the low-pressure side of the high pressure narrow orifice system: i.e. atrial surface of mitral valve leaflets in mitral insufficiency. Satellite vegetations may also develop

due to the jet stream from the primary vegetation. c) Bacterial factors. Endocarditis is predominantly a Gram positive bacterial infection. Gram positive bacteria adhere to cardiac valvular surfaces more avidly than Gram negatives. This is due to the presence of surface components (adhesins) such as the extracellular polysaccharide, dextran, for streptococci that mediates bacterial binding to and colonization of valvular surfaces. Other factors include the serum resistance of Gram positives and their capacity to interact with platelets. d) Vegetation formation. Once bacteria have colonized the valvular surface a vegetation forms. This consists of bacteria encased in a meshwork of platelets and fibrin. The vegetation serves as a barrier to host defenses. It is not vascularized, has few mononuclear or PMNs and is therefore not easily sterilized by host factors or antimicrobials. e) Pathology. The gross appearance of vegetations is variable. There may be single or multiple lesions. Destruction of the underlying valve is often present. There is often greater necrosis and friability of the lesions associated with acute IE. Adjoining structures e.g. chordae, myocardial abscesses may also be involved. This was about the mechanism of the disease for the diagnose we use a constellation of physical, laboratory and historical findings. A set of criteria known as the Duke Criteria have been used to help establish a diagnosis of IE as definite or probable. It relies on pathological, clinical and laboratory data and is modeled after the Jones criteria (used in the diagnosis of Rheumatic) Blood culture is the most important initial laboratory test in the workup of IE. Bacteraemia is usually continuous and the majority of patients with IE have positive blood cultures. If antibiotic therapy has been administered prior to the collection of blood cultures, the rate of positive cultures declines. Modern blood culture techniques now enable isolation of most pathogens that cause IE. For this reason, practices that were traditionally used to facilitate isolation of fastidious pathogens, such as the use of specific blood culture bottles or extending incubation beyond 5 days, are no longer generally recommended In cases of suspected IE that are culture-negative other microbiological testing approaches may be useful. For example, serological testing is necessary for the diagnosis of Q fever, murine typhus and psittacosis. In addition, *Bartonella* can be isolated with special culture techniques and serological studies may also be helpful for identifying this pathogen. Culture of valvular tissue may yield a causative organism when blood cultures are negative and microscopy for fastidious or intracellular pathogens may also be diagnostic. Molecular techniques to recover specific DNA or 16S ribosomal RNA from valve tissue and blood by using PCR Technique. Echocardiography is the second cornerstone of diagnostic efforts and should be performed in all patients in whom IE is suspected and finally modified duke criteria a collection of major and minor criteria , major blood culture positive for characteristic organism echocardiographic identification of valve related or mass or pus with minor fever predisposing of heart lesion or IV drug abuse vascular lesion or immunologic phenomena finally for the treatment Local host defenses at the infected vegetation are unable to control the infection. Therefore antimicrobial therapy is necessary to eliminate all organisms. This requires the following: a) Use of bactericidal antimicrobial agents. Agents that are primarily bacteriostatic will often lead to disappearance of many symptoms and signs of the disease but will be associated with relapse almost all of the time because the vegetation is not sterilized. This infection requires the use of bactericidal antibiotics administered over a relatively prolonged time period. b) Timing of initiation of antimicrobial therapy. Treatment should be started early, but great urgency for the initiation of therapy is primarily limited to cases of acute endocarditis where rapid valve destruction is likely.

Conclusions and Recommendations

Infective endocarditis remains a challenging disease with relevant early and late mortality Recent changes in the epidemiology of IE make the diagnosis a challenge, and traditional diagnostic criteria are insufficient. Despite modern medical and surgical therapy, IE is still associated with a high rate of complications and increased mortality. Early surgery is becoming more common and should be used for all patients. IE is resource-consuming, and a multidisciplinary approach is essential to provide efficient and cost-effective treatment.

Bibliography (References)

1] A graphic description of the disease by a medical student with endocarditis in the pre-antibiotic era.

[2] 188. Iversen K, et al. Partial oral treatment of endocarditis. American heart journal. 2013;165:116–122.[PubMed]

[3] Modified from Durack DT et al: Am J Med, 96:200, 1994 and Karchmer AW: In Braunwald E, Zipes DP, Libby P (eds): Heart Disease. A Textbook of Cardiovascular Medicine, 6th ed. Philadelphia, WB Saunders, 2001, p 1723.