Recent Modalities in the Management of Infective Endocarditis in Pediatric Patient

Essay

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Abstract

Recent Modalities in the Management of Infective Endocarditis in Pediatric Patient

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Infective endocarclitis (IE) is an infection of the endocardium. It is not a common disease in pediatric but it because a high rate of morbidity and mortality. Modified Duke criteria are used for diagnosis of (IE) depending on major and minor criteria. Diagnosis of (IE) depends mainly on blood culture and echocardiography. Two major modalities are used in the treatment of (IE):- Antimicrobial therapy and surgical treatment.

Recently, in 2007, the American heart association had made recommendation for prevention of (IE) that so different from recommendation promoted over 50 years ago.

Key words:

Infective endocarditis (IE) – Duke critiera – Blood cultu Echocardiography.

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List of abbreviation

ABE	Acute bacterial endocarditis
AHA	American heart assoscation
ASD	Atrial septal defect
BCNIE	Blood culture negative IE
BSAC	British Society for Antimicrobial Chemotherapy
CHD	Congenital heart disease
CHF	Congestive heart failure
CNS	Central nervous system
CoNS	Coagulase-negative staphylococci
ELIFA	Enzyme linked immunofluorescent assays
ELISA	Enzyme linked immunosorbent assays
ERCP	Endoscopic retrograde cholangiopancreatography
GI	Gastrointestinal
GU	Genitourinary tract
HACEK	Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae
HCM	Hypertrophic cardiomyopathy
IDUS	Intravenous drug users
IE	Infective endocarditis
INR	International normalized ratio
IVDA	Intravenous drug abusers
MIC	Minimum inhibitory concentration
MRSA	Methicillin resistant staphylococci
MVP	Mitral valve prolapse

NBTE	Nonbacterial thrombotic endocarditis
NIE	Nosocomial infective endocarditis
NVE	Native valve endocarditis
ORSA	Oxacillin-resistant S aureus
OSSA	Oxacillin-susceptible S aureus
PAS	Periodic acid-Schiff
PCR	Polymerase chain reaction
PDA	Patent ductus arteriosis
PVE	Prosthetic valve endocarditis
RHD	Rheumatic heart disease
SBE	Subacute bacterial endocarditis
TEE	Transoesophageal echocardiography
TTE	Transthoracic echocardiography
VSD	Ventricular septal defect

Introduction

Infective endocarditis (IE) is an infection of the endocardial surface of the heart. The intracardiac effects of this infection include severe valvular insufficiency, which may lead to intractable congestive heart failure and myocardial abscesses. IE not only affects the heart but also produces a wide variety of systemic signs and symptoms through several mechanisms, including both sterile and infected emboli and various immunological phenomena. ***(Brusch, 2007)***

The incidence of infective endocarditis in the general population is 2-6 cases/ 100,000 patient. It is higher in patient with underlying valvular heart diseases and in intravenous drug abusers (IVDA).

(Mylonakis and Calderwood,2001)

Risk factors has been completely modified during the last 10 years.Rheumatic valve disease is no longer the main risk factor but others as: intracardiac device, IVDA , hemodialysis and nosocomial infection is also more frequent**.** ***(Habib,2006)***

Despite advances in medical, surgical, and critical care interventions, infective endocarditis remains a disease that is associated with considerable morbidity and mortality. Early diagnosis and active treatment are critical for a better clinical outcome. However**,** infective endocarditis is difficult to diagnose because of the atypical clinical manifestations and frequent negative results from blood culture. Echocardiography plays an important role in the diagnosis and management of suspected or known infective endocarditis . ***(Larry,2005)***

Chapter 1

Infective endocarditis

Infective endocarditis(IE) is an infection of the endocardium that usually involves the valves and adjacent structure . It can damage the valves and rings of connective tissues that surround the valves. As the valves of the heart do not actually receive any blood supply of their own, defense mechanisms (such as white blood cells) cannot enter. So if an organism (such as bacteria) establishes a hold on the valves, the body cannot get rid of them.Normally, blood flows smoothly through these valves. If they have been damaged , bacteria can have a chance to take hold. ***(Morellion and Que, 2004)***

Classification

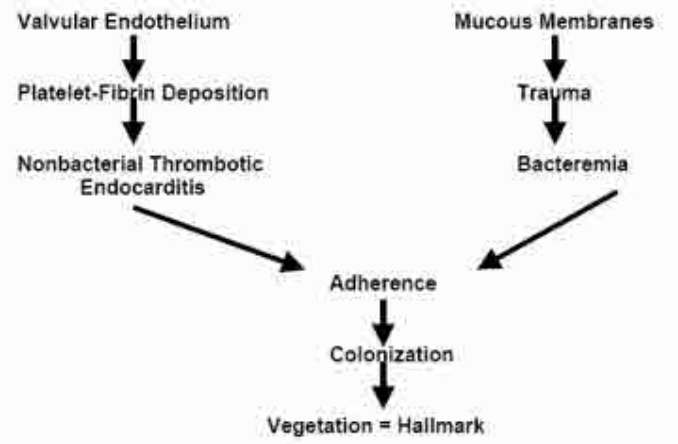
Traditionally, IE has been clinically divided into acute and subacute (because the patients tend to live longer in subacute as opposed to acute) endocarditis. This classifies both the rate of progression and severity of disease. The subacute bacterial endocarditis (SBE) is often due to streptococci of low virulence and mild to moderate illness which progresses slowly over weeks and months, while acute bacterial endocarditis (ABE) is a fulminant illness over days to weeks, and is more likely due to *Staphylococcus aureus* which has much greater virulence, or disease-producing capacity.This terminology is now discouraged. The terms short incubation (meaning less than about six weeks), and long incubation (greater than about six weeks) are preferred. ***(Cunha et al.,1996)***

IE may also be classified as culture-positive or culture-negative. Culture-negative endocarditis is due to prior antibiotic administration or micro-organisms that require a longer period of time to be identified in the laboratory. Such organisms are said to be 'fastidious' because they have demanding growth requirements. Some pathogens responsible for culture-negative endocarditis include Aspergillus species, Brucella species, *Coxiella burnetii*, Chlamydia species, and HACEK bacteria (ie, *Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, Kingella kingae)* ***(Houpikian and Raoult ,2005)***

The distinction between native-valve endocarditis and prosthetic-valve endocarditis is clinically important. Native valve endocarditis (NVE) is a microbial infection involving one or more of the natural heart valves.Streptococci, especially *viridans streptococci*, are responsible for the largest percentage of cases of (NVE). Prosthetic valve endocarditis (PVE) is an endovascular microbial infection occurring on parts of a valve prosthesis or on reconstructed native heart valves. (PVE) develops in 2 to 3% of patients within 1 yr after valve replacement and in 0.5%/yr thereafter. It is more common after aortic than after mitral valve replacement. Early-onset infections (< 2 mo after surgery) are caused mainly by contamination during surgery with antimicrobial-resistant bacteria (eg, *S. epidermidis* is the usual cause of early-onset PVE nearly 30% of cases, diphtheroids, coliform bacilli, Candida sp, Aspergillus sp). Late-onset infections are caused mainly by contamination with low-virulence organisms during surgery or by transient asymptomatic bacteremias, most often with streptococci, *S. epidermidis*,diphtheroids, the fastidious gram-negative bacilli and Haemophilus species . ***(Piper et al.,2001)***

Patients who are injected intravenously may introduce infection which will travel to the right side of the heart. In other patients without a history of intravenous exposure, endocarditis is more frequently left-sided. ***(Jennifer,2000)***

Figure 1:pathogenesis of infective endocarditis



(Morellion and Que, 2004)

Intact cardiac endothelium is a poor stimulator of blood coagulation and is weakly receptive to bacterial attachment.Damaged or denuded endothelium is a potent inducer of thrombogenesis and provides a nidus to which bacteria can adhere and eventually form an infected vegetation. In children with heart disease, the shear force associated with an abnormal high-velocity jet stream of blood can damage the endothelium. Thrombogenesis at such a site results in the deposition of sterile clumps of platelets, fibrin, and occasionally red blood cells, and the formation of nonbacterial thrombotic endocarditis (NBTE). NBTE also can be produced in children with indwelling intravenous catheters positioned in the right side of the heart. Such catheters may traumatize the endocardium or valvular endothelium, exposing the subendothelial collagen. ***(Baltmore,1990)***