Infective Endocarditis in Dentistry: A Review

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Abstract

Infective endocarditis is a serious infection occurring on the endothelial surfaces of the heart, especially at the valves. It has been classified as "acute" or "sub acute –chronic" on the basis of the severity of the clinical presentation and the progression of the untreated disease[1]. Oral commensal bacteria are the important etiologic agents in this disease. Common dental procedures, even non-surgical dental procedures, can often cause bacteremia of oral commensals. Periodontally diseased patients are at risk from bacteremia even after brushing the teeth. Bacteremia itself rarely affect healthy people but they can result in mortal infective endocarditis in those who have a predisposed risk for this disease, such as those with heart valve diseases, pacemaker implantation, etc[2]. The incidence is reported to vary from 1.5 to six cases per 100,000 persons per year in adults.[3] Antibiotics have to be adequately used to prevent this infection, however, their frequent uses generates drug-resistant mutant bacteria, which is a serious social problem.

The aim of this paper was to provide a comprehensive knowledge of IE along with its various guidelines.

Keywords:- infective endocarditis, dental procedures, oral commensal bacteria, viridans streptococci, Splinter hemorrhages,Conjunctival petechiae, Osler's nodes, Janeway's lesions

Introduction

Infective endocarditis is a life-threatening disease. The characteristic lesion, a vegetation is composed of a collection of platelets, fibrin, microorganisms, and inflammatory cells. It most commonly involves heart valves but may also occur at the site of a septal defect, on the chordae tendineae, or on the mural endocardium.[4]It is always fatal if untreated, and it continues to cause substantial morbidity and mortality despite modern antimicrobial and surgical treatments[5]

Substantial morbidity and mortality results from this infection,despite improvements in the outcome due to advances in antimicrobial therapy and an enhanced ability to diagnose and treat complications.Primary prevention is thus the most important. It was first suggested, at the beginning of the twentieth century, that oral bacteria are related to infective endocarditis[6]. Since then, interest has grown in the relationship between dental procedures and infective endocarditis, and bacteremia as a result of dental treatments has thus continued to be a subject of research interest. Antimicrobial chemotherapy is now widely advocated to protect at-risk patients, however, their frequent uses generates drug-resistant mutant bacteria, which has become a serious social problem[7]. This short review describes the knowledge of infective endocarditis in dentistry.

Definitions

IE is an endovascular, microbial infection of intracardiac structures facing the blood including infections of the large intrathoracic vessels and of intracardiac foreign bodies.[8]

Incidence :-The incidence of IE ranges from one country to another within 3–10 episodes/100 000 person-years.[6] This may reflect methodological differences between surveys rather than true variation. In all epidemiological studies of IE, the male: female ratio is _2:1, although this higher proportion of men is poorly understood. Furthermore, female patients may have a worse prognosis and undergo valve surgery less frequently than their male counterparts.[9]

Prognosis

IE represents a life-threatening, ongoing complication that may impair long-term outcomes in patients with Chronic Heart Disease(CHD) Data from the literature have shown IE to be less severe in children, which might be due to the higher proportion of right-sided IE in patients with CHD, particularly in those with a ventricular septal defect (VSD)-located infection.[10]

Pathognenesis

The adherence of microorganisms to wounded cardiac surfaces and their proliferation at the local site results establishment of infective endocarditis. Damaged heart valves as a sequela of rheumatic fever or previous endocarditis, acquired valve lesions, roughened cardiac surfaces as a result of a jet stream effect from blood crossing congenital cardiac lesions, such as a septal defect, and prosthetic heart valves are the usual predisposing clinical conditions for infective endocarditis. Initially, on the damaged endothelial surfaces, sterile vegetations composed of platelet-fibrin clot and thrombus areformed and then, if bacteria are introduced into the bloodstream, the lesion can trap bacterial cells and thus act as an incubator. Subsequent growth of the infective vegetation causes local myocardial abscess that inhibits the valvular function, and finally results in congestive heart failure.[2]

Besides of local problems of heart, infective vegetation pieces may break off and travel through the patient's body through the blood circulation. These infective emboli can cause cerebral infraction and aneurysms, and thus producing infections in remote organs, such as kidney and spleen.

Etiologic Agents of Infective Endocarditis: - Among the wide variety of bacteria, the leading cause of infective endocarditis is viridans streptococci [11], particularly as the cause for the subacute form of this diseases Viridans streptococci comprise the largest group in the member of streptococci, and they are the most dominant commensals in the oral cavity.

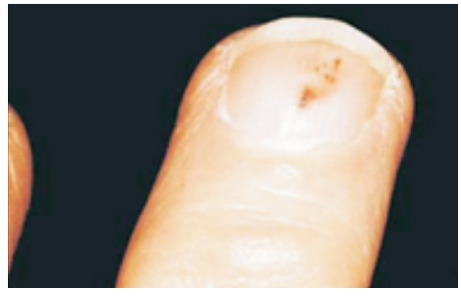
Induction of Bacteremia by Dental Procedures

The blood stream is sterile under normal healthy conditions. The introduction of bacteria into the bloodstream is necessary for an intracardiac infection to occur. Such bacteremia occurs after tooth extractions and other oral surgery procedures. Bacteremia as a result of dental treatments has been a subject of research interest since the first indication at the start of the 20th century that oral bacteria may be related to infective endocarditis .Non surgical dental procedures have also been reported to cause bacteremia at significant frequencies. These include the administration of local anesthesia, orthodontic bandplacement, periodontal probing, dental prophylaxis, scaling and root-planing and even after daily tooth brushing and flossing.[2]

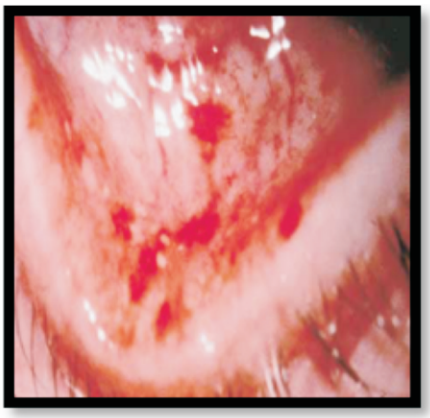
Clinical sign & symptoms

The presentation of infective endocarditis often includes extra cardiac manifestations or findings that are associated with intra-cardiac extension of infection.[1]

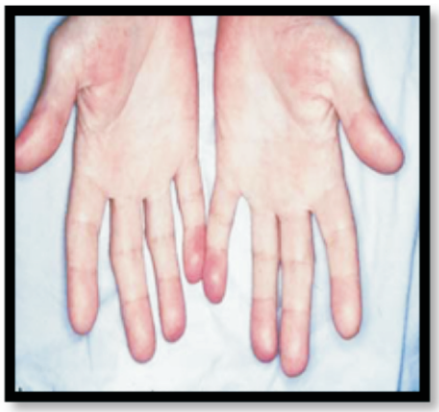
1. Splinter hemorrhages



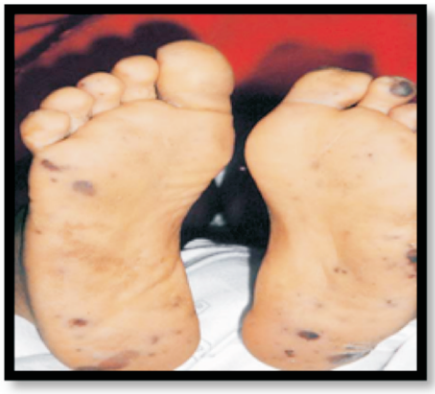
2. Conjunctival petechiae



3. Osler's nodes



4. Janeway's lesions



Clinical features

The clinical history of IE is highly variable according to the causative microorganism, the presence or absence of pre-existing cardiac disease, and the mode of presentation. Thus, IE must be suspected in the following situations:-[12]

1.**Fever** : the most frequent sign of IE IE should be suspected if fever is associated with :- a. Intracardiac prosthetic material (e.g.prosthetic valve, pacemaker,implantable defibrillator,surgical baffle/conduit) b. Previous history of IE c. Previous valvular or congenital heart disease d. Other predisposition for IE (e.g.immunocompromised state,IVDA) e. Predisposition and recent intervention with associated bateraemia f. Evidence of congestive heart failure g. New conduction disturbance h. Positive blood cultures with typical IE causative organism or positive serology for chronic Q fever (microbiological findings may precede cardiac manifestations) i. Vascular or immunologic phenomena:embolic event ,Roch spots,splinter haemorrhages,Janeway lesions,Osler's nodes j. Focal or non specific neurological symptoms and signs k. Evidence of pulmonary embolism/infiltration (right-sided IE)
1. Peripheral abscesses(renal,splenic,cerebral,vertebral)of unknown cause
2. New regurgitant **heart murmur**
3. **Embolic** events of unknown origin
4. **Sepsis** of unknown origin (especially if associated with IE causative organism)

Periodontal Disease and Infective Endocarditis

Several reports indicate that bacteremia is more frequently inducible in patients with severe periodontal diseases than those who have healthier periodontal tissue after tooth brushing and periodontal pocket probing. [13]Gum inflammation loosens the gingival epithelial tissues and it often becomes ulcerative at the inner part of periodontal pockets. It can thus provide oral bacteria the route for getting into the host circulation. Furthermore, some periodontopathic bacteria such as A. actionmycetemcomitans and P. gingivalis have the capacity to invade the host epithelial and connective tissues, and these characteristics are considered to be involved in the enlargement of inflammatory lesions. Periodontopathic bacteria which seem to be less important etiologic agents of infective endocarditis may play important roles in the induction of this disease by causing gum inflammation and opening a route toward the blood circulation for endocardiopathic viridans streptococci.[14]

Diagnosis:-[12]

The diverse nature and evolving epidemiological profile of IE ensure it remains a diagnostic challenge. The clinical history of IE is highly variable according to the causative microorganism, the presence or absence of pre-existing cardiac disease, and the mode of presentation.

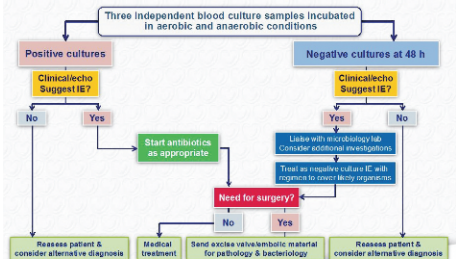
1-Echocardiography diagnosis

Transthoracic and transoesophageal echo-cardiography (TTE/TEE) are importance in diagnosis, management, and follow-up.

- TTE/TEE is recommended as the first-line imaging in suspected IE.
- TEE is recommended in patients with high clinical suspicion of IE and normal TTE.
- Repeat TTE/TEE within 7-10 days in case of negative intial examination and if clinical suspicion of IE persists.
- TEE should be considered in most of adult patients with suspected IE,even in case of positive TTE.
- TEE is not indicatd in patients with a good quality negative TTE and low suspicion of IE

2-Microbiological diagnosis

Positive blood cultures remain the cornerstones of diagnosis and provide live bacteria for susceptibility testing.



Diagnostic criteria [15]

In 1994, Durak et.al proposed criteria for the diagnosis of infective endocarditis ; these criteria are now refered to as the Duke criteria . Duke criteria are a set of clinical criteria set forward for the diagnosis of infective endocarditis.

For diagnosis the requirement is:

- 2 major and 1 minor criteria or
  1 major and 3 minor criteria or
- 5 minor criteria

The Duke criteria, based upon clinical, echocardiographic, and microbiological findings provide high sensitivity and specificity for the diagnosis of IE. Recent amendments recognize the role of Q-fever (a worldwide zoonosis caused by Coxiella burnetii), increasing prevalence of staphylococcal infection, and widespread use of TEE, and the resultant so-called modified Duke criteria are now recommended for diagnostic classification.(~80% overall)for the diagnosis of IE. Recent amendments recognize the role of Q-fever (a worldwide zoonosis caused by Coxiella burnetii), increasing prevalence of staphylococcal infection, and widespread use of TEE, and the resultant so-called **modified Duke criteria** are now recommended for diagnostic classification.

Modified Duke criteria for the diagnosis of IE
(Adapted from Li & al)

MAJOR CRITERIA

Blood culture positive for IE

- Typical microorganisms consistent with IE from 2 separate blood cultures:
  Viridans streptococcus, Streptococcus bovis, HACEK group, Staphylococcus aureus or community acquired enterococci in the absence of a primry focus.
- Microorganisms consistent with IE from 2 persistely positive blood cultures:
  At least 2 positive blood cultures of blood samples drawn > 12 h apart or all of 3 or a majority of ≥ 4 separate cultures of blood with first & last sample drawn at least 1 h apart.
- Single positive blood culture for *Coxiela burneti* or phase I IgG antibody titer > 1:800.

Evidence of endocardial involvement

- Echocardiogram positive for IE. *(Vegetation, Abscess, New partial dehiscence of prosthetic valve).*
- New valvular regurgitation.

MINOR CRITERIA

- Predisposition: Predisposing heart condition, injection drug use.
- Fever: temperature > 38°C.
- Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysms.
- Intracranial haemorrhages, conjunctival haemorrhages, Janeway lesions.
- Immunologic phenomena: glomerulonephritis Osler' s node, Roth' s spot, rheumatoid factor.
- Microbiological evidence: positive blood culture but does not meet a major criterion or serological evidence of active infection with organism consistent with IE.

In summary, echocardiography and blood cultures are the cornerstone of diagnosis of IE. The Duke criteria are useful for the classification of IE but do not replace clinical judgement.

Prevention of Infective Endocarditis: - For dental,oral ,respiratory and oesophageal procedures , prophylaxis of IE aims primarily at viridians,streptococci and HACEK organisms .[5] Various Recommendation and guidelines are proposed for prophylaxes of IE are:-

A) Recommendations by the American Heart Association (1997) [19]

For Dental procdures :-

Recommended prophylaxis	Not recommended prophylaxis
➢ Dental Extraction	❖ *Restorative dentistry (operative and prosthodontic) with/without retraction cord;* Fluoride treatments
➢ Tooth Reimplantation	❖ *Local anesthetic injections (nonintraligamentary)*
➢ Dental Implants	❖ *Intracanal endodontic treatment; post placement and build up*
➢ Scaling and Root planning	❖ *Placement of rubber dams*
➢ Intraligamentary anesthesia	❖ *Postoperative suture removal*
➢ Orthodontic bands	❖ *Placement of removable prosthodontic/orthodontic appliances;* Orthodontic appliance adjustment
➢ Cleaning of teeth and implants where bleeding is anticipated	❖ *Taking of oral impressions ;* Taking of oral radiographs
➢ Ret beyond apex of tooth	❖ Shedding of primary teeth

Antibiotic regime:-

Dental, oral, respiratory, and esophageal procedures
1-not allergic to penicillin ➢ amoxillin 2.0 g (children 50 mg/kg) p.o. 1 h before procedure ➢ **unable to take oral** medication: amoxicillin or ampicillin 2.0 g (children 50 mg/kg) i.v. 1/2 –1 h before procedure **2-allergic to penicillin**: ➢ clindamycin 600 mg (children 20 mg/kg) or azithromycin/clarithromycin 500 mg (children15 mg/kg) 1 h before procedure

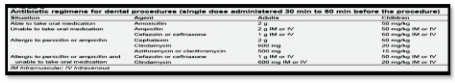
B) Revised Recommendations by the American Heart Association (2007) [20]

American Heart Association (AHA) looked at published research and other scientific articles. They found that fewer conditions were associated with IE. As a result, a smaller group of patients needs to premedicate before dental treatments. The current guidelines recommend

use of preventive antibiotics before certain dental procedures for people with:

- artificial heart valves
- a history of infective endocarditis
- a cardiac transplant that develops a heart valve problem
- congenital (present from birth) heart conditions such as :-
- unrepaired or incompletely repaired cyanotic congenital heart disease, including those with palliative shunts and conduits
- a completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure
- Any repaired congenital heart defect with residual defect at the site or adjacent to the site of a prosthetic patch or a prosthetic device.

Revised antibiotic regiem:-



c) Recommendations by European Society of Cardiology (ESC)for Dental procedures; 2009 [5]

Single dose 30-60 min before procedure

Situation	Antibiotic	Adults	Children
No allergy to penicillin or ampicillin	Amoxicillin or Ampicillin	2 g p.o. or i.v.	50 mg/kg p.o. or i.v.
Allergy to penicillin or ampicillin	Clindamycin	600mg p.o. or i.v.	20 mg/kg p.o. or i.v.

Note:-

Cephalosporins should not be used in patients with anaphylaxis, angio-oedema, or urticaria after intake of penicillin and ampicillin.

Alternatively cephalexin 2 g i.v. or 50 mg/kg i.v. for children, cefazolin or ceftriaxone 1 g i.v. for adults or 50 mg/kg i.v. for children.

Treatment

Successful treatment of IE relies on microbe eradication by antimicrobial drugs. Three sets of blood cultures should be drawn at 30 min intervals before initiation of antibiotics.[16] The initial choice of empirical treatment depends on several considerations:

(i) whether the patient has received prior antibiotic therapy or not

(ii) whether the infection affects a native valve or a prosthesis (and, if so, when surgery was performed (early vs. late PVE) and

(iii)knowledge of local epidemiology, especially for antibiotic resistance and specific genuine culture-negative pathogens

Surgery is the treatment of choice in complicated PVE [17]. Sterilization and "cure" by medical treatment alone are rarely possible in active NVE[18] .Surgery contributes by removing infected material and draining abscesses.

Proposed antibiotic regimens for initial empirical treatment of infective endocarditis (before or without pathogen identification) is :- [5]

Antibiotic	Dosage & route	Duration	Comments
Native valves			
Ampicillin-Sulbactam or Amoxicillin-Clavulanate with Gentamicin	12 g/day i.v. in 4 doses 12 g/day i.v. in 4 doses 3 mg/kg/day i.v. or i.m. in 2 or 3 doses	4-6 weeks 4-6 weeks 4-6 weeks	Patients with blood-culture negative should be treated in consultation with an infectious disease specialist
Vancomycin with Gentamicin with Ciprofloxacin	30 mg/kg/day i.v. in 2 doses 3 mg/kg/day i.v. or i.m. in 2 or 3 doses 1000 mg/day orally in 2 doses or 800 mg/day i.v. in 2 doses	4-6 weeks 4-6 weeks 4-6 weeks	For patients unable to tolerate β-lactams Ciprofloxacin is not uniformly active on Bartonella spp. Addition of Doxycycline is an option if Bartonella spp. is likely
Prosthetic valves (early < 12 months post surgery)			
Vancomycin with Gentamicin with Rifampin	30 mg/kg/day i.v. in 2 doses 3 mg/kg/day i.v. or i.m. in 2 or 3 doses 1200 mg/day i.v. or orally in 2 doses	6 weeks 2 weeks	If no clinical response, surgery and perhaps extension of the antibiotic spectrum to gram-negative pathogens must be considered
Prosthetic valves (late > 12 months post surgery)			
Same as Native valves			

Complications of Infective Endocarditis (IE):

1. Heart failure (HF) - the most frequent complication of IE which represents the most frequent indication for surgery in IE.[21] It is observed in 50–60% of cases overall and is more often present when IE affects the aortic (29%) rather than the mitral (20%) valve.[22] It can be caused by severe aortic or mitral insufficiency, intracardiac fistulae, or, more rarely, by valve obstruction.
2. Uncontrolled infection- second most frequent cause for surgery[21] and encompasses persisting infection (.7–10 days), infection due to resistant organisms, and locally uncontrolled infection.
3. Systemic embolism-very frequent in IE, complicating 20–50% of cases of IE, falling to 6–21% after initiation of antibiotic therapy. The risk of embolism is the highest during the first 2 weeks of antibiotic therapy and is clearly related to the size and mobility of the vegetation.[23]
4. Neurological complications-develops in 20–40% of all patients with IE and are mainly the consequence of vegetation embolism.[24]
5. Infectious aneurysms - Infectious (mycotic) aneurysms (IAs) result from septic arterial embolism to the intraluminal space or vasa vasorum, or from subsequent spread of infection through the intimal vessels[25]
6. Renal failure - common complication of IE which occurs in 30% of patients and predicts poor prognosis. Causes are often multifactorial as:-[26]
   - ü Immune complex and vasculitic glomerulonephritis
   - ü Renal infarction
   - ü Haemodynamic impairment in cases with HF or severe sepsis,or after cardiac surgery
   - ü Antibiotic toxicity
   - ü Nephrotoxicity
7. Rheumatic complications - Musculoskeletal symptoms (arthralgia, myalgia, and back pain) are frequent during IE, and rheumatic complications may be the first manifestations of the disease.MRI or CT of the spine should be performed in IE patients with back pain. Prolonged antibiotic therapy is generally required in definite spondylodiscitis[27]
8. Splenic abscess -Splenic abscess is rare. Persistent or recurrent fever and bacteraemia suggest the diagnosis. Treatment consists of appropriate antibiotic regimens. Splenectomy may be considered for splenic rupture or large abscesses which respond poorly to antibiotics alone.[28]
9. Myocarditis, pericarditis - Cardiac failure may also be due to myocarditis which is frequently associated with abscess formation. Regional myocardial infarction may be caused by coronary embolism or compression. Pericarditis may be associated with an abscess, myocarditis, or bacteraemia often as a result of S. aureus infection. Purulent pericarditis is rare and may necessitate surgical drainage.[29]

Conclusions:

Infective endocarditis is a serious infection occurring on the endothelial surfaces of the heart, especially at the valves. Common dental procedures, even non-surgical dental procedures, often cause bacteremia that can result in infective endocarditis in people who have a predisposing risk for this disease, such as valvular heart diseases including prosthetic valves, congenital heart diseases, cardiomyopathy, coronary artery disease, pacemaker implantation, etc. Common dental procedures often cause bacteremia and periodontally diseased patients may even suffer from bacteremia after tooth brushing. The incidence and severity of IE has not decreased significantly over the years. Treatment of this infection requires adequate use of antibiotics; however their frequent use can also generate drug-resistant mutant bacteria.

Reference

References are available on request at editor@healtalkht.com