

Antibiotic Prophylaxis for Infective Endocarditis

A.Shathariya

Savitha Dental College , Chennai

Abstract:

Some cardiac conditions require antibiotic prophylaxis for some types of dental treatment to reduce the risk of infective endocarditis. Infective endocarditis remains a major medical concern because of its mortality and its incidence is not decreasing. The preventing of this serious condition relies mainly on the administration of prophylactic antibiotics to patients at risk before they undergo a procedure considered to carry risk. All medical and dental practitioners are familiar with this practice but tend to use different regimens in apparently similar circumstances. This review article analyses and discusses the treatment regimen in the prevention of infective endocarditis.

Keywords: Antibiotic prophylaxis, endocarditis, dental treatment.

INTRODUCTION:

Infective endocarditis is a microbial infection of the endocardial surfaces usually involving the cardiac valves. Infective endocarditis remains a major medical concern because of its mortality and costs, and because its incidence is not decreasing [1,2].

The prevention of this severe infection has long been considered desirable and possible. This has relied mainly on the administration of antibiotics to cardiac patients at risk before they undergo a procedure considered to carry risk. However, the indications for preventive treatment and the design of antibiotic regimens are not based on well-established scientific evidence and recent studies have questioned the benefit of such treatment[3-6]. It has been recommended that antibiotic prophylaxis should be given for some types of cardiac conditions which may be susceptible to the development of infective endocarditis.

PATHOGENESIS OF INFECTIVE ENDOCARDITIS:

In pathogenesis of infective endocarditis, the usual process was divided into the following steps:

Endocardial damage;

Establishment and persistence of bacteria within the endocardium;

Bacterial growth with local tissue damage;

Both cardiac and peripheral non-cardiac manifestations.

The normal endothelium is a non-thrombogenic surface and is resistant to colonization by circulating bacteria[7,8]. Endothelial cell injury or dysfunction activates metabolic and synthetic events associated with many vascular disorders[9]. The most common causes of endothelial cell damage are hemodynamic and occur in regions of extreme mechanical or shear stress[10]. These are the flow surfaces of valves along the lines of closure on the systemic side of the circulation. Thus the most common sites are the mitral and aortic valves, ventricular septal defects and complex congenital abnormalities.

The bacteria are usually rapidly destroyed in the blood and if they reach the heart they pass through without settling. Sometimes, however when there is a combination of a high load of bacteria within the blood, more virulent bacteria with a greater capacity to adhere to damaged valves, or altered haemodynamics and thrombus formation on damaged valves or foreign bodies, then bacteria may settle and commence colonization.

The valve is colonized the bacteria must survive and avoid the host defences. Maturation of the vegetation is a key event in the process, where the micro-organisms become fully enveloped and thus protected from cellular and soluble host defence systems[11]. Both staphylococci and streptococci can trigger tissue factor production from local monocytes and induce platelet aggregation. Bacterially-induced platelet activation both helps to envelop the bacteria and to release proteins which are bactericidal. Thus bacteria associated with infective endocarditis need to be resistant to platelet-induced factors.

CAUSES OF BACTERAEMIA:

All surface coverings of the body are colonized by a unique microflora. Any bacteraemia may be of skin, gut, airway, genito-urinary or oral origin. Bacteria from these sources frequently enter the blood on a physiologic basis as a transient bacteraemia, and are dealt with by the host defences. Oral manipulations including dental treatment will produce a greater bacteraemia than physiologic function. The frequency of bacteraemia associated with dental procedures are extraction, periodontal surgery, periodontal scaling, periodontal disease etc[12].

MECHANISM OF PREVENTION:

Antibiotics may prevent endocarditis either by killing bacteria or by damaging them to extent that the host defences can they destroy them. Therefore, the antibiotic may work before the bacteria enter the bloodstream, after they enter the bloodstream or on colonies of bacteria. The primary mechanism by which antibiotic prophylaxis could occur has not been established but a number of studies show that bacteraemia is reduced both in quantity and time in the presence of antibiotics [13].

ADVERSE REACTIONS TO ANTIBIOTICS:

The risks associated with antibiotics are side effects such as gastro-intestinal tract upset, colonization of resistant or fungal strains, drug interactions, allergic reactions (including anaphylaxis) and death. Mild reactions, including urticaria occurs in penicillin courses. Allergic reactions are rare with gentamicin and vancomycin[14].

ANTIBIOTIC RECOMMENDATIONS FOR PAEDIATRIC:

Amoxycillin 50mg/kg (oral up to 2g 1 hour before procedure)

Amoxycillin 1.5g

Highest risk give 1g Amoxycillin IV/+Gentamicin 1.5mg/kg followed by

IV Amoxycillin 1g or Oral 1g[15].

If allergic to penicillin:

Clindamycin 20mg/kg

Cephalosporin 50mg/kg

Vancomycin 20mg/kg IV

Gentamicin 2.5mg/kg IV per day

ANTIBIOTIC REGIMEN:

Standard prophylaxis

Amoxycillin 2g orally as a single dose 1 hour before procedure

If hypersensitive to penicillin

Clindamycin 600mg orally as a single dose 1 hour before procedure

CONCLUSION:

The decision to administer antibiotic prophylaxis is an individual matter and a careful assessment needs to be made on an evidence-based risk assessment, traditional belief ,or what medical and dental practitioners were taught as undergraduates.The cardiac risk should be assessed medically by cardiologist and prophylactic regimen can be given for patients.

REFERENCES:

1. Jaunay T,Sambrook P,Goss A. Antibiotic prescribing practices by South Australian General dental practitioners.Aust Dent J 2000;45:179-186.
2. Dajani AS,Taubert KA,Wilson W,et al. prevention of bacterial endocarditis: recommendations by the American Heart Foundation.J Am Dent Assoc 1997;277:1794-1801.
3. American Dental Association.Position Statement on Antibiotic Prophylaxis.JADA 1998;129:761-769.
4. Ramsdale DR,Roberts GJ,Lucas VS.Dental aspects of endocarditis prophylaxis:new recommendations from a working group of the British cardiac society clinical practice Committee and Royal College of Physicians Clinical Effectiveness and Evaluation Unit 2004.
5. Thompson J.Guidelines for endocarditis prophylaxis.1996 Revision on behalf of the Netherlands Heart Foundation Endocarditis Prophylaxis Committee.Cardiologic 1997;3:75-78.
6. Australian Prescriber Wall Chart. Antibiotic prophylaxis of endocarditis insert Australian Prescriber.Vol 1
7. Patient Information Sheet.Prevention of heart infection or prosthetic joint infection following dental treatment.Edition 1:1-2.Australian Dental Association.
8. Royal Adelaide Hospital Drug Committee.Antibiotic prophylaxis guidelines.Revision 2005.
9. Writing Group for Therpeutic Guidelines:Antibiotic Therpeutic guidelines:Antibiotic Version 12. Melbourne:Therpeutic Guidelines Ltd,2003
10. Spicer WJ,Locke D.Comment in letters to the editor.Aust Prescr 2001;24:51-55.
11. Oliver R,Roberts GJ, Hooper L.Penicillins for the prophylaxis of bacterial endocarditis in dentistry.Cochrane Database of Syst Rev 2004;CDOO3813
12. Mylonakis E,Calderwood SB.Infective endocarditis in adults.New Eng J Med 2001;345:1318-1330.
13. Woods R.Control of infection. In: A guide to the use of drugs in dentistry.Sydney:Australian Dental Association,1996:43-116.
14. Martin MV,Butterworth ML,Longman LP.Infective endocarditis and the dental practitioner: A review of 53cases involving litigation. Br Dent J 1997;182:464-468.
15. Bor DH,Himmelstein DU.Endocarditis prophylaxis for patients with mitral valve prolapse: a quantitative analysis.Am J Med 1984;76:711-717.