Current Concepts of Prophylactic Antibiotics for Dental Patients

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KEYWORDS

• Antibiotic prophylaxis • Infective endocarditis • Prosthetic joint infection

KEY POINTS

- There is no evidence to support the routine use of antibiotic prophylaxis before dental procedures to prevent infective endocarditis.
- There is no evidence to support the routine use of antibiotic prophylaxis before dental procedures to prevent prosthetic joint infection.
- There is no evidence to support an association between the bacteremia after dental procedures and incidence of IE or PJI.

INTRODUCTION

The theoretic need for the use of antibiotic prophylaxis is summarized in the context of bacteremia, presumably from the oral cavity. Oral organisms entering the bloodstream, via invasive dental procedures, can potentially colonize vulnerable areas, such as defective heart valves, prosthetic joints, and implanted devices, such as cardiac stents or hemodialysis shunts. The colonization of these vulnerable sites can result in various sequlae, such as valvular damage, infective endocarditis (IE), and failure of prosthesis or implanted devices. Despite the theoretic context, there are multitudes of variables that often are not included into the equation. These variables include, but are not limited to, the extent of bacteremia, species of bacteria, host susceptibility, presence of comorbidities, type of implanted devices, type of antibiotics used, bacteria response to the antibiotics, and the nature of dental procedures. Practitioners often balance these variables with the perceived benign nature of antibiotics, and elect to use antibiotics rather than considering the risks and benefits of not using them. This is further complicated by an unrealistic fear of legal reprisal and whether a practitioner can justify their decision to an unrelenting legal team.

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HISTORY OF ANTIBIOTIC PROPHYLAXIS GUIDELINES FOR INFECTIVE ENDOCARDITIS

In 1955, the American Heart Associations (AHA) published its first recommendations for prevention of infective endocarditis.[1] These guidelines have evolved over the past decades by work of the AHA and American College of Cardiology Task Force groups. International societies have also published their own recommendations and guidelines, further contributing to the evolution of most recent guidelines.

The updates in the available guidelines, from 1955 through 2007, have taken several factors into consideration. Such factors as drug resistance bacteria, risk stratification of the patient population, etiology of bacteremia, and the complexity of the prophylaxis regiment have been included in development of these guidelines. The 1997 guidelines were the first to acknowledge that IE is often not associated with invasive procedures, and more frequently caused by random bacteremia from routine activities. The rational for these guidelines was largely based on expert opinion and what seemed prudent practice to prevent a life-threatening infection.[2] The evidence used to develop these guidelines could be scored as class IIB, and level of evidence C.

In 2007, the most recent guidelines were developed based on the publications and data questioning the efficacy of antibiotics therapy in prevention of IE, and in an attempt to reduce the complexity of the previous guidelines. These new guidelines have significantly reduced the use of antibiotic prophylaxis to prevent IE.

Justification for Antibiotic Prophylaxis in Prevention of Infective Endocarditis

Despite advances in diagnosis and treatment of IE, it continues to be a dangerous disease. Morbidity and mortality are 50% in high-risk patients, such as those with prosthetic valves, congenital heart disease, and previous history of IE.[3]

Development of IE is the net result of a complex set of circumstances involving bloodstream pathogens interacting with the tissue matrix and platelets at the site of endothelial cell damage. This process is summarized in the stages noted next. It is also important to note that the clinical manifestations of IE are further affected by the host immune system.[4]

1. Formation of nonbacterial thrombotic endocarditis (NBTE): Some cardiac anomalies, congenital or acquired, can result in turbulent blood flow, which can cause endothelial injury. Adhesion of platelets and fibrin to the site of trauma can potentially lead into NBTE.
2. Transient bacteremia: Trauma to oral mucosal surfaces can result in transient bacteremia from the site of injury, populating the bloodstream with viridans group streptococci and other common oral microflora
3. Bacterial adhesion: The bacteria within the bloodstream can adhere to the site of endothelial injury and NBTE. Some microorganisms, such as viridans group streptococci, have surface components that allow their adhesions to various surfaces. This surface characteristic can serve as a virulence factor in development of IE.[5] The adhesion of other organisms, such as staphylococci, is facilitated either by surface components or formation of a biofilm, particularly on the surface of implanted devices. There has been some work on vaccines directed to the adhesion characteristics of viridian group streptococci and staphylococci resulting in some protection against IE in experimental models.[6,7]
4. Proliferation of bacteria: On adherence to NBTE, microorganisms rapidly proliferate forming bacterial vegetation within the damaged endothelial surface. These isolated foci can be potentially unaffected by the host immune system, allowing their further growth and invasion. More than 90% of these vegetations are metabolically inactive rendering them less responsive to antibiotics.[8] The bacterial vegetation