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REVIEW ARTICLE

Bacteraemia – A Transient Blood Invasion after Periodontal Procedures

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ABSTRACT

Transient bacteremias are detected frequently following dental manipulations. Infective endocarditis (IE) can be seen in susceptible individuals and antibiotic prophylaxis can be prescribed for certain procedures considered to be at risk of IE. There are evidences that periodontal disease may be a significant risk factor for the development of certain systemic diseases, such as cardiovascular disease. Initiated by the bacteria into the bloodstreams, these systemic conditions are detrimentally influenced by the bacterial ingrowth.

Keywords: Infective endocarditis, Periodontal disease, Transient bacteremia.

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INTRODUCTION

The bloodstream remains sterile under normal conditions. Transient bacteremia starts when bacteria enters the bloodstream. It is unavoidable. Complications of these bacteremias depend upon the bacterial species, general health care of the patients, and the type of dental procedures. Bacteremias are not only restricted to minimal dental procedures like extractions but also associated with periodontal disease. In a healthy person, bacteremia is countered by our own defense mechanisms. The patients with cardiac anomalies or poor immunity are susceptible to bacteremia. Infective endocarditis (IE) is an infection of endocardium. Valvular damage following rheumatic fever, previous endocarditis, a ventricular septal defect, prosthetic heart valves, or valvular stenosis can lead to changes in blood flow or damage in cardiac endothelium. Changes in blood flow or damaged endothelium surfaces lead to precipitation of platelets and fibrin, where and when the bacteria enters the blood and can colonize with the platelet and fibrin meshwork. They formed into an infective vegetation. These vegetations can result in local myocardial abscesses that inhibit valvular function and lead to congestive heart failure. Vegetations may separate from the surface and may reach distant tissues and cause damage in organs, such as brain, lungs, kidneys, and spleen. An array of pathogens can cause bacteremia. The most common causative bacteria are *Streptococci*, *Staphylococci*, and *Enterobacteria*.[1]

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BACTERIAL ENTRY INTO THE BLOODSTREAM

Bacteria gains entry into the bloodstream from oral niches through a number of mechanisms and different variety of portals. When there is a tissue trauma induced by procedures, such as periodontal probing, scaling and instrumentation beyond the root apex, tooth extractions, a breakage in capillaries, and other small blood vessels that are located in the vicinity of the plaque biofilms may lead to spillage of bacteria into the systemic circulation. A higher microbial load would facilitate such dissemination, as it is known that individuals with poor oral hygiene maintenance are at higher risk of developing bacteremia during oral manipulative procedures. Innate microbial factors may play a role in the latter phenomenon, because only a few species are detected in experimental bacteremias despite the multitude of diverse bacteria residing within the periodontal biofilms. Species which are commonly found in the bloodstream have virulence factors that could be linked to vascular invasion.[2]

NATURE OF ODONTOGENIC BACTEREMIAS

Odontogenic bacteremias are transient in nature. It has been summarized that in healthy individuals, bacterias are scavenged from the bloodstream relatively quickly by the innate and adaptive defense machanisms. Some studies indicate that an episode of odontogenic bacteremia could last as long as 1 hour and most studies report the presence of bacterial species in blood for up to 30 minutes. This is observed in all forms of bacteremias irrespective of the triggering factors, underlying predisposing conditions, or detection methods. From the cumulative data,

it can be deduced that the bacteremic incidence peaks within the first few minutes and then gradually declines after 10 to 20 minutes. Depending upon the threshold sensitivity of the detection method, the total reduction of the bacteremic load may vary from 10 to 90% at 30 and from 2 to 20% at 60 minutes. A few bacteria survive, despite an initial steep fall, in the circulation. The role of these survivors and how they survive host defenses need to be evaluated further, as they may be the ones that initially evade the host immune burst and have the propensity to seed target organs and cause systemic and distant infections.[2]

BACTERIAL DIVERSITY IN ODONTOGENIC BACTEREMIA

Eight bacterial taxonomic families are represented in different oral niches. Of these, the members of the family *Firmicutes* occupy the bulk of microbiome in terms of number of genera and bacterial species. Phylogenetic studies of the oral microbiome have shown that a large proportion of the oral bacteria comprises of the genus *Streptococcus*. A different number of controlled clinical trials have been done where it was shown that Streptococci were the predominant organisms isolated, ranging from 40 to 65% of isolates. Other genera of *Firmicutes* have been isolated from the bloodstreams, to a lesser extent than *Streptococcus* and include species of *Abiotrophia*, *Dialister*, *Selenomonas*, and *Solobacterium* genera in descending order. The fusobacteria family is the second most commonly isolated bacterial species from the bloodstream and includes genera, such as *Actinobacteria*, *Synergistes*, and *Bacteriodetes* are far less common. There are no data in the literature describing the dissemination of either spirochetes or recently detected families, such as the Obsidian pool and TM7 bacteria from the mouth in experimental odontogenic bacteremias or systemic infections secondary to dental treatment procedures. The last two families are uncultivable.[2]

BACTEREMIA DURING PERIODONTAL DRESSING CHANGE

In a study done by Scott Wampole et al, five cases of bacteremia are noted and it was found that all the five cases have bacteremia during periodontal dressing and suture removal. Thus, the potential of bacteremia was evident. However, other cases exhibiting hemorrhage during dressing change shows no evidence of bacteremia. Detection of bacteria in blood depends on many factors. The number of bacteria present in the blood may be extremely small and appears to be related to the degree of trauma applied to the tissue. Amount of bacteria is seemed to be insufficient to gain entrance into the bloodstream as periodontal dressing and suture removal are much less traumatic than the extraction. However, the results of the study show that the bacteria has the potential to gain entry into the bloodstream through direct channels when hemorrhage occurs during the postoperative dressing change. There were sufficient numbers of bacteria in the vasculature so that detection was possible in 25% of the patients studied.[3]

INCIDENCE OF BACTEREMIA AFTER CHEWING, TOOTHBRUSHING, AND SCALING

Bacteremia is usually characterized by adequate concentrations of bacteria in the blood, so the sensitivity of the blood culture methods are crucial. The lysis-filtration method used in the study has shown increased sensitivity compared with BACTEC growth bottles. In a study by Lone Foner et al following chewing, 20% of the periodontitis patients were bacteremic whereas none of the periodontally healthy individuals or gingivitis patients showed evidence of bacteremia. One of the periodontitis patient was bacteremic immediately after toothbrushing. After scaling, bacteremia occurred in two of the healthy participants, in four of the gingivitis participants and in 15 of the periodontitis patients. Duration decreased after 30 minutes of the procedures. This study demonstrates that patients with periodontitis as compared with healthy individuals and gingivitis patients are at increased risk of experiencing bacteremia in association with scaling.[4]

BACTEREMIA DUE TO PERIODONTAL DRESSING

In a study by Christopher et al, patients with untreated periodontitis were at significant risk of experiencing bacteremia because of periodontal probing. Compared with patients with chronic gingivitis, periodontitis group is more susceptible towards bacteremia. When periodontal probing is done, the probe tip is known to penetrate into the epithelium lining of the pocket when inflammation is present and bleeding on probing is indicative of the inflammation beneath the underlying connective tissue. Thus, through this bleeding epithelium, the microorganisms, dislodged from the plaque biofilm, may have had the chance to spread into the vascular connective tissue and thence into the circulatory system. This explains why the number of sites that bled on probing was significantly associated with bacteremia.[5]

BACTEREMIA AFTER PERIODONTAL PROCEDURES

During periodontal surgery, the microbial challenge to the patients are significantly high. The amount of postsurgical trauma varies with amount of trauma inflicted. Periodontal procedures, particularly involving surgical

trauma, may be associated with a high percentage of transient bacteremia. In a study by Kanwarjit et al, it was concluded that the incidence of postoperative bacteremia following periodontal flap surgery is not as high as previously reported. The cause for this finding could be the age group of the patients, preoperative preparation of the patients, antibiotic prophylaxis, and the vasoconstriction at the operative site owing to the adrenalin content of the local anesthetic used. It is concluded that preoperative prophylactic administration of antibiotic is a prerequisite to prevent the postoperative bacteremia and its possible sequelae following periodontal therapy. The clinical results show that amoxicillin is highly effective in reducing postoperative bacteremia in periodontal flap surgery and thus in preventing the possible sequelae (IE and other systemic maladies) in susceptible patients.[6]

BACTEREMIA AFTER IMPLANT SURGERY

There is a little evidence of bacteremia present after implant surgery. Studies have revealed that after implant procedures bacterias are found to be minimal in the blood of the patients. After periodontal flap elevation during implant procedure, there is no evidence of bacteremia present but whenever there is periimplant infections around the implant areas, these inflammations may lead to transient invasion of the bacterias into the blood causing bacteremia. In the patients with prosthetic valves more susceptibility towards the bacteremia increases leading to IE. Patients with IE are under anticoagulant therapy which may cause hematomas raising the risk of infections. Antiseptic implant placement therapy followed by meticulous oral hygiene procedures should not be underestimated because this may minimize the risk of bacteremia arising from implants. To assess the risk associated with implant placement, major efforts should be directed to identify the outcomes of patients at high risk who have undergone dental implant surgery, and the prevalence of patients with dental implants among those hospitalized with IE.[7]

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