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INFECTIVE ENDOCARDITIS AND DENTISTRY: AN ARTICLE REVIEW

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ABSTRACT

This review summarizes the studies about association of dentistry with infective endocarditis and emphasizes the need for dental professional to provide antibiotic prophylaxis prior to dental procedures to prevent the dentistry related infective endocarditis and also to avoid the risk associated with the overprescribing of antibiotics. Infective endocarditis is a life threatening condition if untreated and continues to cause significant morbidity and mortality in defiance of modern antimicrobial and surgical treatment. Therefore prevention is the key to reduce the number of cases of Infective endocarditis**.** The main objective of this article is to provide a critical review of the current evidence that links dental procedures to infective endocarditis and also emphasizing the appropriate use of antibiotics. To prevent the infective endocarditis, current guidelines recommend the antibiotic prophylaxis prior to dental procedure for a relatively small subset of patients. The overprescribing of the antibiotics has been increased in general and this issue encourages further research on chemoprophylaxis in dentistry to prevent infective endocarditis. Current evidence on dental induced bacteraemia and the prevelance of infective endocarditis in dentistry raises further questions on the need to provide antibiotic cover in at risk patients.

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INTRODUCTION

Infective endocarditis is a serious infection of the heart occurring on the endothelial surfaces of the heart, most commonly involving the heart valves. It is mainly caused by bacteria but Fungi, Chalamydia, Rickettsia etc. can also lead to infective endocarditis. Oral commensal bacteria are the important etiologic agents in this disease. Among the wide variety of bacteria, the leading cause of infective endocarditis is viridians streptococci, particularly as the cause for the subacute form of this disease. Viridians streptococci comprise the largest group in the member of streptococci, and they are the most dominant commensals in the oral cavity. The most frequently isolated viridians streptococci from the infective endocarditis patients is S. Sanguis (31.9%) followed by S. Oralis (29.8%), the mutans group of streptococci, which is notorious for the cariogenicity, can also cause infective endocarditis. On the other hand, periodontopathic bacteria have been less frequently isolated from patients with infective endocarditis. Actinobacillus actinomycetcomitans has also been reported to be a causative agent but it is not as common as viridians streptococci. The most important pathogen of adult periodontitis is Porphyromonas gingivalis which has never been isolated from patients with infective endocarditis. Bacteraemia can also occur because of non-surgical and common dental procedures. Infective endocarditis is always fatal if untreated and leads to significant morbidity and mortality despite modern antimicrobial and surgical treatment. Infective endocarditis progresses with the adherence of microorganisms to wounded cardiac surfaces and their proliferation at the local site. Damaged heart valves as a sequel of rheumatic fever or previous endocarditis, acquired valve lesions, roughened cardiac surfaces as a result of a jet stream affect from blood crossing congenital cardiac lesions, such as septal defect and prosthetic heart valves are the usual predisposing clinical conditions for infective endocarditis. In addition to the local problems of the heart, pieces of the infective vegetation may break off and travel through the

patient's body through the blood circulation. Cerebral infarction and aneurysm can be caused by these infective emboli, and thus producing infections in the remote organs, such as kidney and spleen. The clinical feature of this complex infection have changed in the past several decades. It is now an infection of older people, and mitral valve prolapsed is the most common predisposing cardiovascular problem in developed countries. In contrast, rheumatic heart disease is an important predisposing problem in developing countries. Since Infective endocarditis caries a high risk of morbidity and mortality, so, to have a good patient outcome, prompt diagnosis, effective treatment, and instant recognition of complications are critical. Infective endocarditis has been classified into acute and subacute types according to the natural history of the disease. Recently as the number of cases with valve replacement surgery is increasing, the number of endocarditis cases associated with the prosthetic valve is increasing. Therefore, these diseases are also classified into prosthetic valve endocarditis and native valve endocarditis.

Bacteremia in Dentistry

Dental treatment was deemed to be the probable cause of Infective Endocarditis in 26% of patients who sought litigation. In the majority of legal cases, clinicians did not follow recognized guidelines or keep adequate clinical notes. The three main factors which link dental procedures legally were the dental operation, the isolation from the dental blood of an oral microorganisms and a short incubation period. (H.Harvie, 2007). One study shows an estimated annual incidence ranging from 2 to 7.9 per 100,000 individuals per year and a short term mortality of 10% to 30% through the breakdown of mucocutaneous barriers and induction of bacteremia, dental therapy and other invasive procedures have been linked to seeding of heart valves and the development of Infective endocarditis. Since the publication of the American Heart Association (AHA) guidelines in 1955, it has been conventionally considered appropriate to prevent infective endocarditis by prophylactic administration of antibiotics before procedures believed to cause bacteraemia. However, the evidence supporting the effectiveness of antibiotic prophylaxis was poor, deriving solely from animal studies, case series and assessments of bacteraemia risk. Notably, the AHA guidelines in 1997 did acknowledge that most Infective endocarditis cases are not attributable to bacteraemia resulting from certain invasive procedures, but rather random bacteraemia from routine daily activities such as tooth brushing or chewing, and thus suggesting that prophylaxis may only prevent small number of cases of infective endocarditis. These guidelines also recognized the potential adverse effects and medical-legal risks associated with prophylaxis. In the absence of a robust evidence base, growing doubts with respect to this widely accepted practice led to a major revision of the AHA guidelines in 2007, narrowing the indication for antibiotic prophylaxis to a smaller population of at risk individuals. Furthermore, the 2008 guidelines from the National Institute of Health and Clinical Excellence (NICE) recommends that antibiotic prophylaxis be abandoned in most situations.(Pei-chunchen *et al*, 2015).

Further studies showed that, it was in the early 20th century that bacteria in the oral cavity were first implicated in infective endocarditis (Horder 1908). Since then, interest has grown in the association amongst dental procedures, subsequent bacteremia and infective endocarditis. It has been found that the reported incidence of bacteraemia in dentistry ranges from 17% to 94%; these varying results have been attributed to patient selection, type of procedure performed and the microbiological techniques used (Heimdahl *et al*. 1990). This study reported on 13 dental operative procedures used routinely in paediatric dentistry to find out the association of bacteraemia in dentistry. General anesthesia was given to the children in each procedure group and an 8-mL blood sample was taken from each patient 30s after each procedure. For the baseline group, blood sample was taken after anaesthetizing the patients but prior to any dental procedure was performed. Two commercial blood culture systems were used and the results were expressed as the percentage of samples that yielded bacteria; no investigations had been done to assess the microbial load following these dental procedures. Four of these conservative dental procedures caused bacteraemia more frequently than the baseline value of 9.4%. In contrast, toothbrushing alone, a daily routine procedure usually, caused a bacteraemia in 38.5% of occasions. In one another study (Roberts *et al*. 2000), the procedures involved in a two surface restorations were investigated, to determine if bacteraemia occurs following these procedures. These included rubber dam placement, use of high speed and slow speed dental handpieces and placement of a matrix band and wedge. In this study, along with the broth culture system, the researcher also calculated the number of colony forming units (CFU) per millilitre of blood in each procedure group. It was found that the placement of matrix band and wedge caused a percentage prevalence of bacteraemia significantly greater than the other procedures.

However, there were no statistically significant differences in the microbial load between these groups. It has been concluded from this study that the placement of rubber dam and a matrix band with a wedge resulted in a bacteraemia comparable with that encountered following a tooth extraction, thus providing evidence that such procedures should be covered by antibiotic prophylaxis. However, this similarity was related to the percentage incidence of samples that yielded bacteria and did not take the number of CFU into consideration in the original sample. Roberts (2004) recommends antibiotic prophylaxis only for procedures, where there is a considerable difference in bacteraemia between pre- and post-procedure blood samples. "This study showed that the dental procedures which involves bleeding are no longer exclusively indicated for antibiotic procedure as many procedures cause bacteraemia without discernible bleeding. (Roberts 2004)." The cumulative exposure to bacteraemia is significantly greater from procedures such as tooth brushing and chewing when compared with dental operative procedures. It was believed that such everyday procedures are the cause of bacterial endocarditis caused by oral organisms because the cumulative exposure is often up to 10^6 times greater than those occurring following surgical procedures such as extraction (Roberts 1999). Bacteraemia which occurred during dental procedures usually contain not that much concentration of bacteria in blood. (Everett & Hirschmann 1977).

This is in contrast to animal studies linking bacteraemia and infective endocarditis, where the concentration of organisms is artificially high, typically in the region of 10^5–10^8 CFU mL^{-1} (Glauser & Francioli 1987). This microbial load of bacteraemia has been shown to be an important factor in the genesis of experimental animal endocarditis (Roberts 1999) and thus extrapolation of experimental animal data to the

clinical setting is difficult. In one another study which was conducted by Al-Karaawi *et al*. in (2001), the cumulative exposure to bacteraemia from dental procedures currently recommended for antibiotic prophylaxis in the American Heart Association (AHA) Guidelines 1997 was compared with the cumulative exposure for dental procedures for which antibiotic prophylaxis was not recommended. High cumulative exposures were obtained for dento-gingival manipulative procedures not currently recommended for antibiotic prophylaxis. A number of studies have also been carried out to determine whether root canal treatment produces significant bacteraemia. Many of the early clinical reports of the link between endodontic treatment and bacteraemia are anecdotal, lack the use of an aseptic technique during treatment and do not match the organisms isolated from the bloodstream to those in the root canal (Ross & Rogers 1943, Bender *et al*. 1960, Trivedi 1984, Bender & Montgomery 1986, Green & Haisch 1988). In other studies, the laboratory procedures used were deficient in that samples were cultured only aerobically and in one such study (Robinson *et al*. 1950), no bacteria were detected in the bloodstream following preparation and filling of seven root canals. Endodontic procedures with instrumentation beyond the apex were shown by Bender *et al*. (1963) to produce detectable bacteraemia in 31% of cases, but, when instrumentation was confined within the tooth, blood cultures were negative. (M. Brincat *et al*, 2006).

The problem of whether patients with periodontal diseases are at higher risk from infective endocarditis than people who have healthy gingiva has not yet been fully addressed. However, several reports indicate that bacteraemia is more frequently inducible in patients with severe periodontal diseases than those who have healthier periodontal tissue after tooth brushing and periodontal pocket probing. Gum inflammation loosens the gingival epithelial tissues and it often becomes ulcerative at the inner part of periodontal pockets. It can thus provide the oral bacteria the route for getting into the host circulation. Direct evidence for the relationship between the prevalence of periodontal disease and the incidence of infective endocarditis remains to be investigated.

DISCUSSION

The antibiotics are being misused very commonly in general and this has focused attention on antibiotic prophylaxis in dentistry to prevent infective endocarditis. New evidence on dental related bacteraemia and the increased incidence of infective endocarditis in association with dental procedures raises further questions on the recommendation of antibiotic cover in at risk patients. More prescriptive guidelines to define who is at risk from infective endocarditis and what procedures require cover will help to decrease overprescribing of antibiotics and reduce the risks of their undesired effects. Dental procedures, especially those that leads to bacteraemia, are frequently responsible for infective endocarditis, hence the need to premedicate the patients with antibiotics for such procedures in at risk patients. Recent evidence from the USA and studies from the Netherlands have presented further data which challenges the practice of prescribing antibiotics before dental procedures to prevent endocarditis. This information also needs to be considered in tandem with the increasing concern over the unnecessary use of antibiotics. Thus, it seems imperative to reassess the topic of antibiotic prophylaxis, dental treatment and infective endocarditis. General dental professionals often express concerns as to what dental procedures produce bacteraemia and who is at risk from such bacteraemia. Poor oral health, mainly periodontal condition, is an important risk factor for infective endocarditis. Gingival inflammation is positively associated with the prevalence and magnitude of bacteraemia. However, bleeding is a poor predictor of dental induced bacteraemia. Some dental procedures cause bacteraemia, although the magnitude will vary. By contrast, various oral hygiene practices and mastication also increases the prevalence of bacteraemia. It has been suggested that routine oral hygiene practices and chewing are responsible for so-called random cases of bacteraemia. Such bacteraemia either from dental treatment or oral hygiene practices etc. are of a low grade intensity and of short duration. Oral health care providers and dental procedures are often regarded as the culprit for infective endocarditis. In many instances the occurrence of endocarditis does not relate to the so-called dental-induced bacteraemia. It may well transpire that random bacteraemia may be more causative in infective endocarditis than dental surgeons carrying out treatment. This view is supported in a recent review article which has evaluated the evidence of dental-induced bacteraemia and infective endocarditis. The three main conclusions are as follows: bleeding is a poor predictor of dental-induced bacteraemia; the intensity of bacterial inoculae arising from dental operative procedures is low when compared to the high intensity needed for ID90 in experimental animals: the procedures most often regarded as requiring antibiotic prophylaxis do not carry the greatest risk of cumulative bacteraemia. The latter arise from chewing and various oral hygiene practices. Further evidence to support this finding comes from an analysis of cases of infective endocarditis where dental treatment has been implicated as the cause. Oral streptococci cause approximately 50% of all such cases. Similarly, only 15% of patients where infective endocatrditis has been diagnosed report medical or dental treatment within the previous 3 months. It has been estimated that 4% or less of all IE cases are related to dental treatment-induced bacteraemia. Whether such bacteraemia arise from dental treatment or were spontaneous is not discernible. (RA Seymour, 2000).

For infective endocarditis prophylaxis, current guidelines support premedication for a relatively small subset of patients. This is based on a review of scientific evidence, which showed that the risk of adverse reactions to antibiotics generally overweigh the benefits of prophylaxis for many patients who would have been considered eligible for prophylaxis. (ADA,2017).

Standard general prophylaxis for high and moderate risk patient:

"According to the ADA chairside guide, for patients with a history of complications associated with their joint replacement surgery who are undergoing dental procedures that include gingival manipulation or mucosal incision, prophylactic antibiotics should only be considered after consultation with the patient and orthopedic surgeon; in cases where antibiotics are deemed necessary, it is most appropriate that the orthopedic surgeon recommend the appropriate antibiotic regimen and, when reasonable, write the prescription." In patients with prosthetic joint implants, a January 2015 ADA clinical practice guidelines, based on 2014 systematic review states, "In general, for patients with the

Table 1. Regimen: Single dose 30 to 60 min before procedure

Situation	Agent	Adults	Children
1. Oral	Amoxicillin	2g	50 mg/kg
2. Unable to take Oral medication	Ampicillin or Cefazolin or Ceftriaxone	2g IM or IV 1g IM or IV	50 mg/kg IM or IV 50 mg/kg IM or IV
3. Allergic to penicillins Or Ampicillin- oral	Cephalexin or Clindamycin or Azithromycin or Clarithromycin	2g 600 mg 500mg	50 mg/kg 20 mg/kg 15 mg/kg
4. Allergic to penicillins and ampicillins and unable to take oral medication	Cefazolin or Ceftriaxone or Clindamycin	1g IM or IV 600 mg IM or IV	50 mg /kg IM or IV 20 mg /kg IM or IV

IM= Intramuscular
IV= Intravenous

prosthetic joint implants, prophylactic antibiotics are not recommended prior to dental procedures to prevent prosthetic joint infection." (ADA, 2017).

Patient selection

The current infective endocarditis/valvular heart disease guidelines state that use of antibiotics prior to certain dental procedures is only recommended for patients with :

- Prosthetic cardiac valves, including transcathetric-implanted prosthesis and homografts
- Prosthetic material used for cardiac valve repair, such as annuloplasty rings and chords
- A previous history of infective endocarditis
- A cardiac transplant with valve regurgitation due to a structurally abnormal valve
- The congenital heart disease such as the unrepaired cyanotic congenital heart disease, any repaired congenital heart defect with residual shunts or valvular regurgitation at the site of or adjacent to the site of a prosthetic patch or a prosthetic device.

Infective endocarditis occurs more commonly in patients with heart transplant than the general population, the risk of infective endocarditis is greater in the first 6 months following transplant because of disruption of endothelium, high intensity immunosuppressive drugs, frequent central venous catheter use, and frequent endomyocardial biopsies. Except the above mentioned conditions, antibiotics prophylaxis is no longer recommended for any other form of congenital heart disease. (ADA, 2017). "In addition to antibiotic prophylaxis of infective endocarditis, other methods of reducing bacteraemia from an oral origin have been sought. The use of pre-surgical 1% povidone iodine has been demonstrated to cause significant reduction in bacteraemia from oral sources (Rise *et al*.1969, Scopp & Orvieto 1971), although routine use may provoke the selection of resistant microorganisms (Park & Hart 1994). In a double-blind study of 60 patients who participated in pre-extraction rinsing with 1% (v/v) chlorhexidine, 1% (v/v) povidone-iodine and a control of NaCl, a significant reduction in bacteraemia between both antimicrobials and the control was observed. (MacFarlane *et al*. 1984). However, there was no difference between the two antiseptics. A positive bacteraemia was reported in 40% and 25% of the povidone-iodine and chlorhexidine rinsers, respectively."

Conclusions

With the input from the ADA, the American Heart Association released guidelines for the prevention of infective endocarditis in 2007 which were approved by the CSA as they relate to dentistry in 2008. In 2017, the AHA, and American college of cardiology (ACC) published a focused update to their 2014 guidelines on the management of valuable heart disease that reinforce the previous recommendations. These current guidelines recommend antibiotic prophylaxis for a relatively small subset of patients. This is based on a systematic review of scientific evidence, which showed that the risk of unwanted effects of antibiotics generally outweigh the benefits of prophylaxis for many patients who would have been considered eligible for prophylaxis in previous versions of the guidelines. Development of drug-resistant bacteria was also a factor to be concerned. The valvular disease management guidelines recommend the persons at risk of developing bacterial endocarditis establish and maintain the best possible oral health to decrease the potential sources of bacterial seeding. They state, "optimal oral health is maintained through regular professional dental care and the use of appropriate dental products such as manual, powered, and ultrasonic toothbrushes, dental floss, and other plaque removal devices."(ADA, 2017).

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