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Original Article

Assessment of periodontitis and its role in viridans streptococcal bacteremia and infective endocarditis

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ABSTRACT

Objectives: To evaluate the role of periodontitis in viridans group streptococci (VGS) bacteremia and Infective Endocarditis.
Methods: A total of 200 subjects including two groups. Group A- 34 subjects undergoing tooth extraction with periodontitis and 46 subjects undergoing tooth extraction without periodontitis and 40 healthy controls. Group B: 40 confirmed cases of infective endocarditis (17 with and 23 without periodontitis) and 40 healthy controls. Subgingival plaque and blood samples were obtained and processed by standard procedures.
Results: A total of 53 blood samples (66.25%) yielded positive cultures after tooth extraction. The relationship between the presence of periodontitis and a positive blood culture was significantly higher ($P = 0.05$) for tooth extraction cases with periodontitis (79.40%) than tooth extraction cases without periodontitis (56.50%). Periodontitis was observed in 42.5% of IE cases. Out of the 40 patients of IE, the blood samples; yielded 40 different isolates, majority were viridans streptococci 15 (37.5%) and staphylococci nine (22.5%). No statistically significant difference was observed between the subgingival plaque and blood isolates of periodontitis in both the groups, indicating similarity of biotypes of viridans streptococci isolated from the blood and the subgingival plaque. Similarity was also observed between the antibiogram profiles of viridans streptococci from both the groups.
Conclusions: Periodontitis enhances viridans streptococcal bacteremia and may be a potential risk factor for infective endocarditis.

1. Introduction

Several studies have established that periodontitis is a risk factor for infective endocarditis.[1] Gingivitis and periodontitis are among the most common human infections. Gingivitis can develop within days and includes inflammatory changes of the gingiva most commonly induced by accumulation of dental plaque. Periodontitis results from a complex interplay between chronic bacterial infection and the inflammatory host response leading to irreversible destruction of tooth-supporting tissues, with tooth loss as a common end point.[2] Periodontitis is associated with elevated inflammation that may contribute to bacteremia associated with infective endocarditis (IE) risk.

It has been reported that patients with periodontitis have inflamed and ulcerated crevicular or pocket epithelium around the teeth, which can act as a portal of entry for bacteria from oral cavity to the blood stream.[3] Lockhart et al. showed that the generalized presence of gingival bleeding after tooth brushing was associated with an almost eightfold increase in bacteremia risk.[4] This is consistent with another study reporting the incidence and the magnitude of bacteremia induced by chewing, tooth brushing and invasive dental procedures to be associated with gingival inflammation induced by periodontitis.[5]

Periodontitis is a potential risk factor for translocation of bacteria from oral cavity into the blood stream via ulcerated inflamed crevice and pocket epithelium and the adjacent gingival

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microcirculation. This may occur following invasive dental procedures and also during normal daily activities.[4–9] Bacteremia and low-grade systemic inflammation induced by periodontal infections may be a risk for systemic conditions like cardiovascular diseases including infective endocarditis, stroke, premature low birth weight delivery and diabetes mellitus.[10]

The earlier study shows that most of the IE cases occur as a result of microorganisms reaching the heart through the blood stream.[11] It is this explanation that has attracted the attention of medical and dental specialists towards this heart condition. The access into the bloodstream and thereby to the heart resulting into cardiovascular ailment is promoted by periodontitis induced inflammation. The present study was carried out to determine the association of periodontitis and viridians group streptococci (VGS) bacteremia in patients of tooth extraction and IE and to compare the biotypes and antimicrobial profiles of VGS in subgingival plaque and blood of these patients.

2. Material and methods

This study was approved by the ethical committee of the Ashwini Rural Medical College, Hospital & Research Centre, Solapur, Maharashtra, India. The study included a total of 200 subjects including 80 healthy controls distributed in two groups as follows:

Group A subjects: In this group, 80 patients (34 with periodontitis and 46 without periodontitis) undergoing tooth extraction and 40 healthy controls were enrolled. Demographic information and medical histories from the participants were obtained and thorough clinical and radiographic examinations of their teeth were conducted after written informed consent. Patients with fewer than 10 teeth; an active viral infection, poorly controlled systemic disease, penicillin allergy, antimicrobial usage within three months prior dental treatment, temperature greater than 100.5 °F or facial cellulitis; or were immune-compromised by virtue of disease or medications were excluded from the study.

Group B subjects: In this group, 40 (17 with periodontitis and 23 without periodontitis) confirmed cases of IE above the age of 18 years and fulfilling Duke diagnostic criteria[12] and 40 controls were enrolled. Informed consent was obtained from each patient. Patients who proved to have any source of infection other than IE were excluded from the study. Patients in whom blood cultures for bacteria turned negative and later on showed fungal growth were also excluded. Pregnant women, patients unable to give informed consent or non-cooperative in the dental examination and known conditions requiring prophylactic antibiotic treatment before dental examinations were also excluded from the study.

2.1. Dental examination (Group A and B subjects)

Assessment of periodontal status was performed by means of clinical attachment loss (CAL), probing pocket depths (PPD), which was measured to the nearest whole millimetre at six sites per tooth by using a William's periodontal probe. Oral hygiene indices such as, papillary bleeding index (PBI),[13] plaque index (PI),[14] and gingival index (GI)[15] were also assessed. All assessments were done by a single trained examiner.

2.2. Sample collection

Subgingival plaque samples of the tooth were collected from the gingival area of buccal and lingual tooth surfaces of affected tooth using sterile curettes into sterile transport media (group A and B).

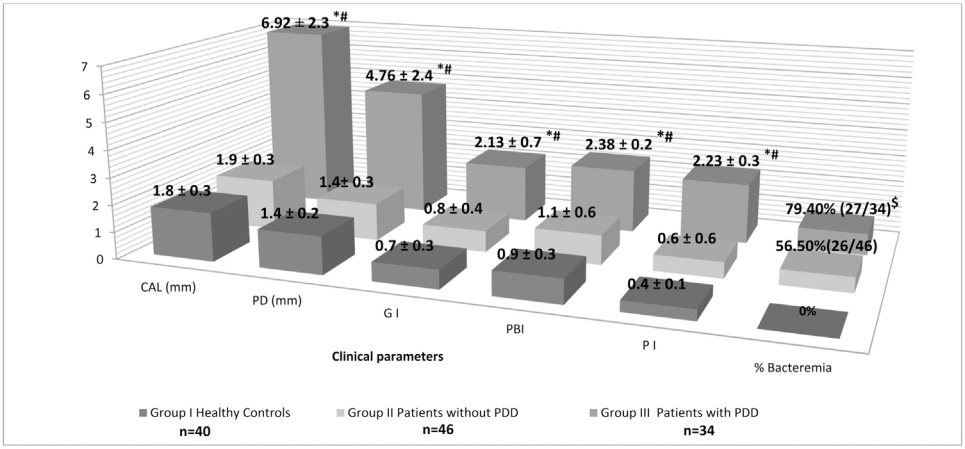
Clinical samples of blood were obtained from healthy controls and patients undergoing tooth extraction. Blood for culture was collected from the site in the antecubital fossa with standard precautions.[16] For each subject 5 ml of venous blood was drawn before and after 3 min of dental extraction (group A).[17–20]

Three blood samples were collected aseptically from healthy controls and patients of IE for aerobic culture from three different sites of the body (right cubital fossa, left cubital fossa and left wrist) at intervals over 24 h (group B).[16]

2.3. Microbiological analysis

Subgingival plaque specimens (group A and B) were inoculated onto special media, tryptone soya blood agar supplemented with strepto supplement (nalidixic acid 3.750 mg, nemomycin sulphate 1.060 mg and polymixin B sulphate 8500 units for 500 ml media) and mutans sanguis agar (Himedia laboratories, Mumbai). Cultures with VGS growth were further subjected to standard biochemical identification using automated Vitek 2 (bioMérieux) system to complete the strain identification. Antimicrobial susceptibilities were measured in MIC by automated Vitek 2 (bioMérieux) system in accordance with CLSI standards.[21]

All the blood samples (group A and B) were screened using automated BD Bactec™ 9050 automated system. Five ml of



* $P < 0.001$ compared with healthy controls
# $P < 0.001$ compared with patients without periodontitis
$ $P = 0.05$ compared with patients without periodontitis
$ $P < 0.0001$ compared with healthy controls

Chart 1. Incidence of post extraction bacteremia related to clinical attachment loss (CAL), periodontal pocket depth (PD), gingival index (GI), papillary bleeding index (PBI) and plaque index (PI). All values are presented in (Mean ± standard deviation).

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